LEARNING OBJECTIVES

1. Describe the pathophysiology, types, clinical manifestations, and collaborative care of pneumonia.
2. Explain the nursing management of the patient with pneumonia.
3. Describe the pathogenesis, classification, clinical manifestations, complications, diagnostic abnormalities, and nursing and collaborative management of tuberculosis.
4. Identify the causes, clinical manifestations, and nursing and collaborative management of pulmonary fungal infections.
5. Explain the pathophysiology, clinical manifestations, and nursing and collaborative management of lung abscesses.
6. Identify the causative factors, clinical features, and management of environmental lung diseases.
7. Describe the causes, risk factors, pathogenesis, clinical manifestations, and nursing and collaborative management of lung cancer.
8. Identify the mechanisms involved and the clinical manifestations of pneumothorax, fractured ribs, and flail chest.
9. Explain the purpose, methods, and nursing responsibilities related to chest tubes.
10. Explain the types of chest surgery and appropriate preoperative and postoperative care.
11. Compare and contrast extrapulmonary and intrapulmonary restrictive lung disorders in terms of causes, clinical manifestations, and collaborative management.
12. Describe the pathophysiology, clinical manifestations, and management of pulmonary embolism, pulmonary hypertension, and cor pulmonale.
13. Discuss the use of lung transplantation as a treatment for pulmonary disorders.

KEY TERMS

- acute bronchitis, p. ***
- atelectasis, p. ***
- chylothorax, p.
- community-acquired pneumonia, p. ***
- cor pulmonale, p. ***
- empyema, p. ***
- flail chest, p. ***
- hemothorax, p. ***
- hospital-acquired pneumonia, p. ***
- lung abscess, p. ***
- pleural effusion, p. ***
- pleurisy (pleuritis), p. ***
- pneumoconiosis, p. ***
- pneumonia, p. ***
- pneumothorax, p. ***
- pulmonary edema, p. ***
- pulmonary embolism, p. ***
- pulmonary hypertension, p. ***
- tension pneumothorax, p. ***
- thoracentesis, p. ***
- thoracotomy, p. ***
- tuberculosis, p. ***

Electronic Resources

Supplemental content related to Chapter 28 can be found . . .

Companion CD

- contents
- contents

Evolve Website

http://evolve.elsevier.com/Lewis/medsurg
- Content Updates
- Key Points
- Concept Map Creator

- Expanded Audio Glossary
- Key Term Flash Cards
- Electronic Calculators
- WebLinks

A wide variety of problems affect the lower respiratory system. Lung diseases that are characterized primarily by an obstructive disorder, such as asthma, emphysema, chronic bronchitis, and cystic fibrosis, are discussed in Chapter 29. All other lower respiratory tract diseases are discussed in this chapter.

Respiratory tract infections are common. Lower respiratory tract infections are the most common cause of death in the world. Chronic lower respiratory disease is the fourth leading cause of death in the United States, and pneumonia ranks as the seventh leading cause of death despite the availability of antimicrobial

Reviewed by Christine L. Willis, RN, MSN, ANP, CS, Adult Nurse Practitioner, Pulmonary and Critical Care Medicine, Duke University Medical Center, Durham, N.C.
agents. Tuberculosis, although potentially curable and preventable, is a worldwide public health threat.

**ACUTE BRONCHITIS**

Acute bronchitis is an inflammation of the bronchi in the lower respiratory tract, usually due to infection. It is one of the most common conditions seen in primary care. It usually occurs as a sequel to an upper respiratory tract infection. A type of acute bronchitis seen in chronic obstructive pulmonary disease (COPD) is acute exacerbation of chronic bronchitis (AECB). AECB represents an acute infection superimposed on chronic bronchitis. AECB is a potentially serious condition that may lead to respiratory failure. (Chronic bronchitis is discussed in Chapter 29.)

The cause of most cases of acute bronchitis is viral (rhinovirus, influenza). However, bacterial causes are also common both in smokers (e.g., Streptococcus pneumoniae, Haemophilus influenzae) and in nonsmokers (e.g., Mycoplasma pneumoniae, Chlamydia pneumoniae). In acute bronchitis, persistent cough following an acute upper airway infection (e.g., rhinitis, pharyngitis) is the most common symptom. Cough is often accompanied by production of clear, mucoid sputum, although some patients produce purulent sputum. Associated symptoms include fever, headache, malaise, and shortness of breath on exertion. Physical examination may reveal mildly elevated temperature, pulse, and respiratory rate with either normal breath sounds or rhonchi and expiratory wheezing. Chest x-rays can differentiate acute bronchitis from pneumonia because there is no evidence of consolidation or infiltrates on x-ray with bronchitis.

Acute bronchitis is usually self-limiting, and the treatment is generally supportive, including fluids, rest, and antiinflammatory agents. Cough suppressants or bronchodilators may be prescribed for symptomatic treatment of nocturnal cough or wheezing. Antibiotics generally are not prescribed unless the person has a prolonged infection associated with constitutional symptoms. If this is an acute bronchitis due to an influenza virus, treatment with antiviral drugs—either zanamivir (Relenza) or oseltamivir (Tamiflu)—can be started, but the antiviral drug must be initiated within 48 hours of the onset of symptoms.

The COPD patient with severe AECB is usually treated empirically with broad-spectrum antibiotics for 5 to 7 days. Early initiation of antibiotic treatment in COPD patients has resulted in a decrease in relapses and a decrease in hospital admissions. (See Chapter 29 for a discussion of COPD.)

**PNEUMONIA**

Pneumonia is an acute inflammation of the lung parenchyma caused by a microbial organism. Until 1936, pneumonia was the leading cause of death in the United States. The discovery of sulfa drugs and penicillin was pivotal in the treatment of pneumonia. Since that time, there has been remarkable progress in the development of antibiotics to treat pneumonia. However, despite the new antimicrobial agents, pneumonia is still common and is associated with significant morbidity and mortality. Pneumonia and influenza are the seventh leading cause of death in the United States. Data indicate that the mortality from pneumonia and influenza is increasing (3.2%). Pneumonia is currently the leading cause of death from an infectious disease in the United States.

**Etiology**

**Normal Defense Mechanisms.** Normally, the airway distal to the larynx is sterile because of protective defense mechanisms. These mechanisms include the following: filtration of air, warming and humidification of inspired air, epiglottis closure over the trachea, cough reflex, mucociliary escalator mechanism, secretion of immunoglobulin A, and alveolar macrophages (see Chapter 26).

**Factors Predisposing to Pneumonia.** Pneumonia is more likely to result when defense mechanisms become incompetent or are overwhelmed by the virulence or quantity of infectious agents. Decreased consciousness depresses the cough and epiglottal reflexes, which may allow aspiration of oropharyngeal contents into the lungs. Tracheal intubation interferes with the normal cough reflex and the mucociliary escalator mechanism. It also bypasses the upper airways, in which filtration and humidification of air normally take place. The mucociliary mechanism is impaired by air pollution, cigarette smoking, viral upper respiratory infections (URIs), and normal changes of aging. In cases of malnutrition, the functions of lymphocytes and polymorphonuclear (PMN)[AU1] leukocytes are altered. Diseases such as leukemia, alcoholism, and diabetes mellitus are associated with an increased frequency of gram-negative bacilli in the oropharynx. (Gram-negative bacilli are not normal flora in the respiratory tract.) Altered oropharyngeal flora can also occur secondary to antibiotic therapy given for an infection elsewhere in the body. The factors predisposing to pneumonia are listed in Table 28-1.1

**Acquisition of Organisms.** Organisms that cause pneumonia reach the lung by three methods:

1. **Aspiration** from the nasopharynx or oropharynx. Many of the organisms that cause pneumonia are normal inhabitants of the pharynx in healthy adults.
2. **Inhalation** of microbes present in the air. Examples include Mycoplasma pneumoniae and fungal pneumonias.
3. **Hematogenous spread** from a primary infection elsewhere in the body. An example is Staphylococcus aureus.

**TABLE 28-1 Factors Predisposing to Pneumonia**

<table>
<thead>
<tr>
<th>Factor</th>
</tr>
</thead>
<tbody>
<tr>
<td>Aging</td>
</tr>
<tr>
<td>Air pollution</td>
</tr>
<tr>
<td>Altered consciousness: alcoholism, head injury, seizures, anesthetics, drug overdose, stroke</td>
</tr>
<tr>
<td>Altered oropharyngeal flora secondary to antibiotics</td>
</tr>
<tr>
<td>Bed rest and prolonged immobility</td>
</tr>
<tr>
<td>Chronic diseases: chronic lung disease, diabetes mellitus, heart disease, cancer, end-stage renal disease</td>
</tr>
<tr>
<td>Debilitating illness</td>
</tr>
<tr>
<td>Human immunodeficiency virus (HIV) infection</td>
</tr>
<tr>
<td>Immunosuppressive drugs (corticosteroids, cancer chemotherapy, immunosuppressive therapy after organ transplant)</td>
</tr>
<tr>
<td>Inhalation or aspiration of noxious substances</td>
</tr>
<tr>
<td>Intestinal and gastric feedings via nasogastric or nasointestinal tubes</td>
</tr>
<tr>
<td>Malnutrition</td>
</tr>
<tr>
<td>Smoking</td>
</tr>
<tr>
<td>Tracheal intubation (endotracheal intubation, tracheostomy)</td>
</tr>
<tr>
<td>Upper respiratory tract infection</td>
</tr>
</tbody>
</table>
Types of Pneumonia

Pneumonia can be caused by bacteria, viruses, *Mycoplasma*, fungi, parasites, and chemicals. Although pneumonia can be classified according to the causative organism, a clinically effective way is to classify pneumonia as community-acquired or hospital-acquired pneumonia. Classifying pneumonia is important because of differences in the likely causative organisms and the selection of appropriate antibiotics (Table 28-2).

### Community-Acquired Pneumonia

Community-acquired pneumonia (CAP) is defined as a lower respiratory tract infection of the lung parenchyma with onset in the community or during the first 2 days of hospitalization. More than 4 million adults are diagnosed with CAP yearly in the United States, and more than 1 million are hospitalized. The number of cases of pneumococcal pneumonia yearly in the United States, and more than 1 million are hospitalized.

#### Organisms Associated with Pneumonia

<table>
<thead>
<tr>
<th>Category</th>
<th>Organisms</th>
</tr>
</thead>
<tbody>
<tr>
<td>Community-Acquired Pneumonia</td>
<td><em>Streptococcus pneumonia</em></td>
</tr>
<tr>
<td></td>
<td><em>Mycoplasma pneumoniae</em></td>
</tr>
<tr>
<td></td>
<td><em>Haemophilus influenzae</em></td>
</tr>
<tr>
<td></td>
<td>Respiratory viruses</td>
</tr>
<tr>
<td></td>
<td><em>Chlamydia pneumoniae</em></td>
</tr>
<tr>
<td></td>
<td><em>Legionella pneumophila</em></td>
</tr>
<tr>
<td></td>
<td>Oral anaerobes</td>
</tr>
<tr>
<td></td>
<td><em>Moraxella catarrhalis</em></td>
</tr>
<tr>
<td></td>
<td><em>Staphylococcus aureus</em></td>
</tr>
<tr>
<td></td>
<td><em>Nocardia</em></td>
</tr>
<tr>
<td></td>
<td>Enteric aerobic gram-negative bacteria (e.g., <em>Klebsiella</em>)</td>
</tr>
<tr>
<td></td>
<td><em>Mycobacterium tuberculosis</em></td>
</tr>
<tr>
<td>Hospital-Acquired Pneumonia</td>
<td><em>Pseudomonas aeruginosa</em></td>
</tr>
<tr>
<td></td>
<td><em>Enterobacter</em></td>
</tr>
<tr>
<td></td>
<td><em>Escherichia coli</em></td>
</tr>
<tr>
<td></td>
<td><em>Proteus</em></td>
</tr>
<tr>
<td></td>
<td><em>Klebsiella</em></td>
</tr>
<tr>
<td></td>
<td><em>Staphylococcus aureus</em></td>
</tr>
<tr>
<td></td>
<td><em>Streptococcus pneumoniae</em></td>
</tr>
<tr>
<td></td>
<td>Oral anaerobes</td>
</tr>
</tbody>
</table>

*Most common cause of community-acquired pneumonia (CAP).

#### Step 1: Assessment of the ability to treat the patient at home

*Evaluate comorbidities, hemodynamic stability, etc.*

#### Step 2: Calculation of the pneumonia PORT (Pneumonia Patient Outcomes Research Team) Severity Index (PSI), with recommendations for home care and clinical judgment.

This scale, produced by the Agency for Healthcare Research and Quality (AHRQ), is based on multiple factors and stratifies the patient into a risk class (Table 28-3). (The PSI calculator is available for PDA download at [http://pda.ahrq.gov](http://pda.ahrq.gov))

### Hospital-Acquired Pneumonia

Hospital-acquired pneumonia (HAP) is defined only 50% of the time. Organisms that are identified and site to treat is indicated and site to treat is indicated

#### Step 3: Clinician judgment in the final decision to treat, either as an outpatient or in the hospital.

Specific antibiotics for empiric treatment have been recommended by the ATS, the IDSA (Table 28-4 presents the IDSA comprehensive guide), and the Canadian Infectious Disease Society/Canadian Thoracic Society (CIDS/CTS). These guidelines provide an evidence-based consensus approach to initial empiric management of CAP for both outpatients and inpatients. The IDSA CAP guidelines are available online at [www.AHRQ.gov](http://www.AHRQ.gov).

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*A risk score (total point score) for a given patient is obtained by summing the patient age in years (age = 10 for females) and the points for each applicable patient characteristic.*

*BUN: Blood urea nitrogen.*
Therapy for pneumonia is empiric because specific pathogens are not identified at the time treatment is initiated. The specific antibiotics recommended from the IDSA, ATS, and CIDS/CID for empiric treatment are all fairly similar and start with either a macrolide (erythromycin, azithromycin [Zithromax], clarithromycin [Biaxin]; respiratory fluoroquinolones: moxifloxacin [Avelox], gatifloxacin [Tequin], levofloxacin [Levaquin], gemifloxacin [Factive]; advanced macrolides: azithromycin, clarithromycin; β-lactams: high-dose amoxicillin, amoxicillin/clavulanate [Augmentin], cefpodoxime (Vantin), cefprozil [Ceftizil], cefuroxime (Ceftin); antipseudomonal agents: piperacillin (Pipracil), imipenem/cilastatin [Primaxin], meropenem [Merrem IV], cefepime [Maxipime], piperacillin/tazobactam [Zosyn]. COPD, Chronic obstructive pulmonary disease; HF, heart failure; ICU, intensive care unit.

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### TABLE 28-4 Initial Therapy for Suspected Bacterial Community-Acquired Pneumonia in Immunocompetent Adults

<table>
<thead>
<tr>
<th>Patient Variable</th>
<th>Preferred Treatment Options*</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Outpatient</strong></td>
<td>A macrolide or doxycycline</td>
</tr>
<tr>
<td>Previously healthy</td>
<td>A respiratory fluoroquinolone alone, an advanced macrolide plus high-dose amoxicillin, an advanced macrolide plus high-dose amoxicillin/clavulanate</td>
</tr>
<tr>
<td>No recent antibiotic therapy</td>
<td>A respiratory fluoroquinolone alone, an advanced macrolide plus high-dose amoxicillin/clavulanate</td>
</tr>
<tr>
<td>Recent antibiotic therapy (last 3 months)</td>
<td>A respiratory fluoroquinolone alone, an advanced macrolide plus high-dose amoxicillin/clavulanate</td>
</tr>
<tr>
<td>Comorbidities (COPD, diabetes, renal disease, HF, malignancy)</td>
<td>A respiratory fluoroquinolone alone, an advanced macrolide plus high-dose amoxicillin/clavulanate</td>
</tr>
<tr>
<td>No recent antibiotic therapy</td>
<td>A respiratory fluoroquinolone alone, an advanced macrolide plus high-dose amoxicillin/clavulanate</td>
</tr>
<tr>
<td>Recent antibiotic therapy</td>
<td>A respiratory fluoroquinolone alone, an advanced macrolide plus high-dose amoxicillin/clavulanate</td>
</tr>
<tr>
<td><strong>Inpatient</strong></td>
<td>A respiratory fluoroquinolone alone, an advanced macrolide plus a β-lactam antibiotic selected based on previous antibiotic history</td>
</tr>
<tr>
<td>Medical Unit</td>
<td>A respiratory fluoroquinolone alone, an advanced macrolide plus a β-lactam antibiotic selected based on previous antibiotic history</td>
</tr>
<tr>
<td>No recent antibiotic therapy</td>
<td>A respiratory fluoroquinolone alone, an advanced macrolide plus a β-lactam antibiotic selected based on previous antibiotic history</td>
</tr>
<tr>
<td>Recent antibiotic therapy</td>
<td>A respiratory fluoroquinolone alone, an advanced macrolide plus a β-lactam antibiotic selected based on previous antibiotic history</td>
</tr>
<tr>
<td>ICU</td>
<td>Either an antipseudomonal agent plus ciprofloxacin or an antipseudomonal agent plus an aminoglycoside plus a respiratory fluoroquinolone or a macrolide</td>
</tr>
<tr>
<td>Pseudomonas infection not an issue</td>
<td>Either an antipseudomonal agent plus ciprofloxacin or an antipseudomonal agent plus an aminoglycoside plus a respiratory fluoroquinolone or a macrolide</td>
</tr>
<tr>
<td>Pseudomonas infection not an issue but patient has β-lactam allergy</td>
<td>Either an antipseudomonal agent plus ciprofloxacin or an antipseudomonal agent plus an aminoglycoside plus a respiratory fluoroquinolone or a macrolide</td>
</tr>
<tr>
<td>Pseudomonas is an isue</td>
<td>Either an antipseudomonal agent plus ciprofloxacin or an antipseudomonal agent plus an aminoglycoside plus a respiratory fluoroquinolone or a macrolide</td>
</tr>
<tr>
<td>Pseudomonas is an issue but patient has β-lactam allergy</td>
<td>Either an antipseudomonal agent plus ciprofloxacin or an antipseudomonal agent plus an aminoglycoside plus a respiratory fluoroquinolone or a macrolide</td>
</tr>
</tbody>
</table>

*Macrolides: erythromycin, azithromycin (Zithromax), clarithromycin (Biaxin); respiratory fluoroquinolones: moxifloxacin (Avelox, Vigamox), gatifloxacin (Tequin), levofloxacin (Levaquin), gemifloxacin (Factive); advanced macrolides: azithromycin, clarithromycin; β-lactams: high-dose amoxicillin, amoxicillin/clavulanate (Augmentin), cefpodoxime (Vantin), cefprozil (Ceftizil), cefuroxime (Ceftin); antipseudomonal agents: piperacillin (Pipracil), imipenem/cilastatin (Primaxin), meropenem (Merrem IV), cefepime (Maxipime), piperacillin/tazobactam (Zosyn). COPD, Chronic obstructive pulmonary disease; HF, heart failure; ICU, intensive care unit.

The IDSA recommends hospital discharge criteria for patients with CAP. During the 24 hours prior to discharge to home, the patient should have no more than one of the following (unless it represents the patient’s baseline status): temperature >100.4°F (37.8°C); pulse >100 beats/minute; respiratory rate >24 breaths/minute; systolic blood pressure (BP) <90 mm Hg; oxygen saturation in arterial blood by pulse oximetry (SpO2) <90%; and inability to maintain oral intake.4

**Hospital-Acquired, Ventilator-Associated, and Health Care–Associated Pneumonia.** Hospital-acquired pneumonia (HAP) is pneumonia occurring 48 hours or longer after hospital admission and not incubating at the time of hospitalization.5 Ventilator-associated pneumonia (VAP) refers to pneumonia that occurs more than 48 to 72 hours after endotracheal intubation. Health care–associated pneumonia (HCAP) includes any patient with a new-onset pneumonia who (1) was hospitalized in an acute care hospital for 2 or more days within 90 days of the infection; (2) resided in a long-term care facility; (3) received recent intravenous antibiotic therapy, chemotherapy, or wound care within the past 30 days of the current infection; or (4) attended a hospital or hemodialysis clinic. HAP is estimated to occur at a rate of 5 to 15 cases per 1000 hospital admissions, with the rate increasing by 6 to 20 times in patients requiring mechanical ventilation.6 HAP is the second most common nosocomial infection, second only to urinary tract infection. It costs $1.3 billion annually in hospital charges.9

The microorganisms responsible for HAP, VAP, and HCAP are usually bacterial and rarely viral or fungal (see Table 28-2). Many of the organisms enter the lungs after aspiration of particles from the patient’s own oropharynx. Immunosuppressive therapy, general debility, and endotracheal intubation are risk factors predisposing to pneumonia (see Table 28-1), while contaminated health...
care devices and the general environment are potential sources of pathogens.

Once the diagnosis is made, empiric treatment of the pneumonia is initiated based on known risk factors, early versus late onset, and disease severity. A major problem in treating infectious diseases is multidrug-resistant (MDR) organisms, which means that the pathogen has developed resistance to multiple antibiotics. MDR organisms are identified by antibiotic susceptibility tests. The virulence of these organisms can severely limit the available and appropriate antimicrobial therapy. In addition, MDR organisms can increase the morbidity and mortality associated with pneumonia. (MDR organisms are discussed in Chapter 15.)

**Fungal Pneumonia.** Fungi may also be the cause of pneumonia (see the section on Pulmonary Fungal Infections later in this chapter).

**Aspiration Pneumonia.** Aspiration pneumonia refers to the sequelae occurring from abnormal entry of secretions or substances into the lower airway. It usually follows aspiration of material from the mouth or stomach into the trachea and subsequently the lungs. The person who has aspiration pneumonia usually has a history of loss of consciousness (e.g., as a result of seizure, anesthesia, head injury, stroke, or alcohol intake). With loss of consciousness, the gag and cough reflexes are depressed, and aspiration is more likely to occur. Another risk factor is tube feedings. The dependent portions of the lung are most often affected, primarily the superior segments of the lower lobes and the posterior segments of the upper lobes, which are dependent in the supine position.

The aspirated material—food, water, vomitus, or toxic fluids—is the triggering mechanism for the pathology of this type of pneumonia. There are three distinct forms of aspiration pneumonia: If the aspirated material is an inert substance (e.g., barium), the initial manifestation is usually caused by mechanical obstruction of airways. When the aspirated materials contain toxic fluids such as gastric juices, there is chemical injury to the lung with infection as a secondary event, usually 48 to 72 hours later; this is identified as chemical (noninfectious) pneumonitis. The most important form of aspiration pneumonia is bacterial infection. The infecting organism is usually one of the normal oropharyngeal flora, and multiple organisms, including both aerobes and anaerobes, are isolated from the sputum of the patient with aspiration pneumonia. Antibiotic therapy is based on an assessment of the severity of illness, where the infection was acquired (community vs. hospital), and the type of organism present.

**Opportunistic Pneumonia.** Certain patients with altered immune responses are highly susceptible to respiratory infections. Individuals considered at risk include (1) those who have severe protein-calorie malnutrition; (2) those who have immune deficiencies; (3) those who have received transplants and been treated with immunosuppressive drugs; and (4) patients who are being treated with radiation therapy, chemotherapy drugs, and corticosteroids (especially for a prolonged period). The individual has a variety of altered parameters, including altered B- and T-lymphocyte function, depressed bone marrow function, and decreased levels or function of neutrophils and macrophages. In addition to the bacteria and viral causative agents, other agents that cause pneumonia in the immunocompromised patient are *Pneumocystis jiroveci* (formerly *carinii*) and other fungi, and cytomegalovirus (CMV).

*Pneumocystis jiroveci* is an opportunistic pathogen; this fungus rarely causes pneumonia in the healthy individual. *Pneumocystis jiroveci* pneumonia (PCP) has been identified as the most common acquired immunodeficiency syndrome (AIDS)-defining opportunistic infection in the United States and is a common cause of AIDS-associated pneumonia. In this type of pneumonia, the chest x-ray usually shows a diffuse bilateral alveolar pattern of infiltration. In widespread disease, the lungs are massively consolidated.

Clinical manifestations are insidious and include fever, tachypnea, tachycardia, dyspnea, nonproductive cough, and hypoxemia. Pulmonary physical findings are minimal in proportion to the serious nature of the disease. Bacterial and viral pneumonias must first be ruled out because of the vague presentation of PCP. Treatment consists of a course of trimethoprim/sulfamethoxazole (Bactrim) as the primary agent. An alternative medication for the Bactrim-intolerant patient is dapsone.10 In populations at risk for development of PCP (e.g., patients with hematologic malignancies or human immunodeficiency virus [HIV]–positive patients with CD4+ T-lymphocyte counts <200/µl), prophylactic therapy with trimethoprim/sulfamethoxazole may be used. Aerosolized pentamidine (Nebupent), although less commonly used, is an alternative for prophylaxis in Bactrim-intolerant patients. (PCP is also discussed in Chapter 15.)

**Cytomegalovirus (CMV).** CMV is a cause of viral pneumonia in the immunocompromised patient, particularly in transplant recipients. CMV is a member of the herpesvirus family, which includes Epstein-Barr virus, herpes simplex virus, and varicella-zoster virus.8 This virus is not highly contagious, but it is a prevalent virus, with 40% to 100% of the population generally exposed in childhood.11 CMV gives rise to latent infections and reactivation with shedding of infectious virus. It can be a serious lung pathogen in transplant patients. CMV interstitial pneumonia can be a mild disease, or it can result in severe pulmonary insufficiency with high mortality rates. Ganciclovir (Cytovene) is recommended for treatment of CMV pneumonia.

**Pathophysiology**

**Pneumococcal pneumonia** is the most common cause of bacterial pneumonia and is caused by the *Streptococcus pneumoniae* organism. *S. pneumoniae*, also called pneumococcus, can infect the upper respiratory tract, the blood, and the nervous system. The organism is generally found in the nose and throat. When it invades the lung, pneumonia can occur. The Centers for Disease Control and Prevention (CDC) estimates that 40,000 deaths and 500,000 cases of pneumococcal pneumonia occur annually in the United States. There are twice as many cases in African Americans compared to whites.12

The pathophysiology related to this type of pneumonia is discussed here. (The pathophysiology of other types of pneumonia is similar.) There are four characteristic stages of the disease process:

1. **Congestion.** After the pneumococcal organisms reach the alveoli, there is an outpouring of fluid into the alveoli. The organisms multiply in the serous fluid, and the infection is spread. The pneumococci damage the host by their overwhelming growth and by interfering with lung function.

2. **Red hepatization.** There is massive dilation of the capillaries, and alveoli are filled with organisms, neutrophils, red blood cells (RBCs), and fibrin (Fig. 28-1). The lung appears red and granular, similar to the liver, which is why the process is called hepatization.

3. **Gray hepatization.** Blood flow decreases, and leukocytes and fibrin consolidate in the affected part of the lung.
**Clinical Manifestations**

Patients with pneumonia usually have a sudden onset of symptoms, including fever, shaking chills, shortness of breath, cough productive of purulent sputum (rust-colored sputum can be seen in pneumococcal pneumonia), and pleuritic chest pain (in some cases). In the elderly or debilitated patient, confusion or stupor (in pneumococcal pneumonia), and pleuritic chest pain (in some cases) are most commonly caused by infection with viruses, *M. pneumoniae* but can also be due to other bacterial pathogens, such as *H. influenzae*.

Pneumonia may also manifest atypically with a more gradual onset, a dry cough, and extrapulmonary manifestations such as headache, myalgias, fatigue, sore throat, nausea, vomiting, and diarrhea. On physical examination, crackles are often heard. This presentation of manifestations is classically produced by *M. pneumoniae* but can also be caused by *Legionella* and *C. pneumoniae*.

The initial manifestations of viral pneumonia are highly variable. Viruses also cause pneumonia that is usually characterized by an atypical presentation with chills; fever; dry, nonproductive cough; and extrapulmonary symptoms. Primary viral pneumonia can be caused by influenza virus infection. Viral pneumonia is also found in association with systemic viral diseases such as measles, varicella-zoster, and herpes simplex.

**Complications**

Most cases of pneumonia generally run an uncomplicated course. However, complications can occur, and they develop more frequently in individuals with underlying chronic diseases and other risk factors. Complications may include the following:

1. **Pleurisy** (inflammation of the pleura) is relatively common.
2. **Pleural effusion** (transudate fluid in the pleural space) can occur. It develops in 40% of hospitalized patients with pneumococcal pneumonia. Usually the effusion is sterile and is reabsorbed in 1 to 2 weeks; occasionally, effusions require aspiration by means of thoracentesis.
3. **Atelectasis** (collapsed, airless alveoli) of one or part of one lobe may occur. These areas usually clear with effective coughing and deep breathing.
4. **Bacteremia** (bacterial infection in the blood) occurs in 30% of patients with pneumococcal pneumonia and is associated with a 20% mortality rate. The rate can go as high as 60% in elderly patients.12
5. **Lung abscess** is not a common complication of pneumonia. It is seen with pneumonia caused by *S. aureus* and gram-negative pneumonias (see the section on Lung Abscess later in this chapter).
6. **Empyema** (accumulation of purulent exudate in the pleural cavity) is relatively infrequent (occurs in <5% of cases) but requires antibiotic therapy and drainage of the exudate by a chest tube or open surgical drainage.
7. **Pericarditis** results from spread of the infecting organism from an infected pleura or via a hematogenous route to the pericardium (fibroserous sac around the heart).
8. **Meningitis** can be caused by *S. pneumoniae*. The patient with pneumonia who is disoriented, confused, or somnolent should have a lumbar puncture to evaluate the possibility of meningitis.
9. **Endocarditis** can develop when the organisms attack the endocardium and the valves of the heart. The clinical manifestations are similar to those of acute bacterial endocarditis (see Chapter 38).

**Diagnostic Studies**

The common diagnostic measures for pneumonia are presented in Table 28-5. History, physical examination, and chest x-ray often provide enough information to make management decisions without costly laboratory tests.

Chest x-ray often shows a typical pattern characteristic of the infecting organism and is invaluable in the diagnosis of pneumonia. Lobar or segmental consolidation suggests a bacterial cause, usually *S. pneumoniae* or *Klebsiella*. Diffuse pulmonary infiltrates are most commonly caused by infection with viruses, *Legionella*, or pathogenic fungi. Cavitory shadows suggest the presence of a necrotizing infection with destruction of lung tissue commonly caused by *S. aureus*, gram-negative bacteria, and *M. tuberculosis*. Pleural effusions, which can occur in up to 30% of patients with CAP, can also be seen on x-ray.

Sputum Gram stain and cultures are not always obtainable. The interpretation of a Gram stain is not standardized, and atypical pathogens are missed. However, obtaining a lower respiratory tract sputum specimen for culture is recommended before initiating antibiotic therapy in the hospitalized patient.6 If a delay in the time from collecting the sputum to incubation exceeds 2 to 5 hours, results are less reliable. Sputum needs to be sent for culture as soon as possible.
as it is collected. Before antibiotic treatment is begun, two blood cultures may be done for patients who are seriously ill. Although microbial studies are expected before treatment, initiation of antibiotics should not be delayed. Empiric therapy is generally started without culture results in order to maximize treatment effectiveness. Empiric therapy is based on observation and experience, and the exact cause of the infection may not be known. Pulse oximetry is measured routinely and can reveal oxygen desaturation. Arterial blood gases (ABGs) may be obtained and can reveal hypoxemia (partial pressure of oxygen in arterial blood [PaO$_2$] <80 mm Hg), hypercapnia (partial pressure of carbon dioxide in arterial blood [PaCO$_2$] >45 mm Hg), and acidosis. Leukocytosis is found in the majority of patients with bacterial pneumonia, usually with a white blood cell (WBC) count greater than 15,000/μL (15 × 10$^9$/L) with the presence of bands (immature neutrophils).

**Collaborative Care**

Prompt treatment with the appropriate antibiotic almost always cures bacterial and mycoplasma pneumonia. In uncomplicated cases, the patient responds to drug therapy within 48 to 72 hours. Indications of improvement include decreased temperature, improved breathing, and reduced chest pain. Abnormal physical findings can last for more than 7 days.

In addition to antibiotic therapy, supportive measures may be used, including oxygen therapy to treat hypoxemia, analgesics to relieve the chest pain for patient comfort, and antipyretics such as aspirin or acetaminophen for significantly elevated temperature. During the acute febrile phase, the patient’s activity should be restricted, and rest should be encouraged and planned.

Most individuals with mild to moderate illness who have no other underlying disease process can be treated on an outpatient basis. If there is a serious underlying disease or if the pneumonia is accompanied by severe dyspnea, hypoxemia, or other complications, the patient should be hospitalized. Guidelines for hospitalization for CAP are presented in Table 28-3.

Currently, there is no definitive treatment for viral pneumonia. Two antiviral drugs, amantadine (Symmetrel) and rimantadine (Flumadine), are approved for use within 48 hours of onset of symptoms in the treatment of influenza A virus. The neuraminidase inhibitors, zanamivir (Relenza) and oseltamivir (Tamiflu), are active against both influenza A and B (see Chapter 27). During outbreaks of influenza, the CDC encourages the use of amantadine or rimantadine for chemoprophylaxis and the use of oseltamivir or zanamivir for treatment.

Vaccination against influenza is the mainstay of prevention and is recommended annually for use in the individual considered to be at risk (see Table 27-3). Individuals at risk for influenza include the elderly, residents of long-term care facilities, patients with COPD or diabetes mellitus, and health care workers. Both an inactivated influenza vaccine and a live, attenuated virus (LAIV) can be used to reduce the risk of influenza. The LAIV, marketed under the name Flumist, is an intranasally administered vaccine only approved for use among healthy persons ages 5 to 49 years. Inactivated influenza vaccine is approved for persons ages ≥6 months, including those with high-risk conditions. The CDC recommends that all persons ≥65 years, others at risk for influenza complications, health care workers, and household contacts of high-risk persons receive the inactivated influenza vaccine. The inactivated vaccine is modified annually to reflect the anticipated strains in the upcoming season. This vaccine is 30% to 40% effective in preventing clinical illness.

**Pneumococcal Vaccine.** Pneumococcal vaccine is indicated primarily for the individual considered at risk who (1) has chronic illnesses such as lung and heart disease and diabetes mellitus, (2) is recovering from a severe illness, (3) is 65 years of age or older, or (4) is in a long-term care facility. This is particularly important because the rate of drug-resistant *S. pneumoniae* infections is increasing. The vaccine is 50% to 80% effective in preventing bacteremic pneumococcal disease. In the immunosuppressed individual at risk for development of fatal pneumococcal infection (e.g., asplenic patient; patient with nephrotic syndrome, renal failure, or AIDS; transplant recipient), a second dose is recommended after 5 years, although the efficacy of revaccination is unknown. All persons ≥65 years of age who have not received vaccine within 5 years (and were <65 years of age at the time of vaccination) should receive another dose of vaccine. Pneumococcal vaccine and influenza vaccine can be given at the same time in different arms.

**Drug Therapy.** The main problems with the use of antibiotics in pneumonia are the development of multidrug-resistant (MDR) organisms and the patient’s hypersensitivity or allergic reaction to certain antibiotics.

Most cases of CAP in otherwise healthy adults do not require hospitalization. The oral antibiotic therapy administered is frequently empiric treatment with broad-spectrum antibiotics. Once the patient is categorized (see Table 28-3), empiric therapy can be based on the likely infecting organism.

For HAP, VAP, and HCAP, the American Thoracic Society (ATS) recommends that empiric antibiotic therapy be based on whether the patient has risk factors for MDR organisms. The antibiotic regimen needs to be adapted to the local patterns of antibiotic resistance.

When using empiric therapy, it is important to recognize the nonresponding patient. Therapy may require modification based on the patient’s sputum culture results or clinical response. Clinical re-
response is evaluated by factors such as a change in fever, sputum purulence, leukocytosis, oxygenation, and chest x-ray patterns. Clinical improvement after adequate VAP treatment can be seen by day 3 to 5. Patients with VAP may experience rapid deterioration. Patients who deteriorate or fail to respond to therapy will require aggressive evaluation to assess for noninfectious etiologies, complications, other coexisting infectious processes, or pneumonia caused by a drug-resistant pathogen. While monotherapy (one-drug therapy) with selected agents can be used with severe HAP and VAP in the absence of resistant organisms, patients in risk groups initially receive combination therapy until the culture results are known and it is confirmed that one agent can be used. Aerosolized antibiotics have not proven to have value in the treatment of VAP, but they may be considered adjunctive therapy for patients with MDR gram-negative organisms not responding to systemic therapy.

**Nutritional Therapy.** Fluid intake of at least 3 L/day is important in the supportive treatment of pneumonia. If the patient has heart failure, fluid intake must be individualized. If oral intake cannot be maintained, intravenous (IV) administration of fluids and electrolytes may be necessary for the acutely ill patient. Weight loss often occurs in patients with pneumonia because of increased metabolic needs and difficulty eating due to shortness of breath and pleuritic pain. Therefore, it is important to provide nutritional intake to meet the needs of the patient. Small, frequent meals are better tolerated by the dyspeptic patient.

**NURSING MANAGEMENT PNEUMONIA**

**Nursing Assessment**

Subjective and objective data that should be obtained from a patient with pneumonia are presented in Table 28-6.

**Nursing Diagnoses**

Nursing diagnoses for the patient with pneumonia may include, but are not limited to, those presented in Nursing Care Plan (NCP) 28-1.

**Planning**

The overall goals are that the patient with pneumonia will have (1) clear breath sounds, (2) normal breathing patterns, (3) no signs of hypoxia, (4) normal chest x-ray, and (5) no complications related to pneumonia.

**Nursing Implementation**

**Health Promotion.** There are many nursing interventions to help prevent the occurrence of, as well as the morbidity associated with, pneumonia. Teaching the individual to practice good health habits, such as proper diet and hygiene, adequate rest, and regular exercise, can maintain the natural resistance to infecting organisms. If possible, exposure to URIs should be avoided. If a URI occurs, it should be treated promptly with supportive measures (e.g., rest, fluids). If symptoms persist for more than 7 days, the person should obtain medical care. The individual at risk for pneumonia (e.g., the chronically ill, older adult) should be encouraged to obtain both influenza and pneumococcal vaccines.

In the hospital, the nursing role involves identifying the patient at risk (see Tables 28-1 and 28-3) and taking measures to prevent the development of pneumonia. The patient with altered conscious-ness should be placed in positions (e.g., side-lying, upright) that will prevent or minimize the risk of aspiration. The patient should be turned and repositioned at least every 2 hours to facilitate adequate lung expansion and to discourage pooling of secretions. In VAP, a significant reduction in pneumonia incidence is found when patients are placed in a semirecumbent position (45 degrees). The ATS recommends that intubated patients be in a semirecumbent position (30 to 45 degrees), particularly during enteral feeding. In intubated patients, the ATS also recommends continuous aspiration of subglottic secretions above the tracheal tube cuff using a specially designed endotracheal tube to prevent the risk of VAP.

The patient who has a feeding tube generally requires attention to measures to prevent aspiration (see Chapter 40). Although the feeding tube is small, an interruption in the integrity of the lower esophageal sphincter still exists, which can allow reflux of gastric fluids and increase the risk of VAP.

---

**TABLE 28-6 NURSING ASSESSMENT Pneumonia**

<table>
<thead>
<tr>
<th>Subjective Data</th>
<th>Important Health Information</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Past health history:</strong> Lung cancer, COPD, diabetes mellitus, chronic debilitating disease, malnutrition, altered consciousness. AIDS, exposure to chemical toxins, dust, or allergens.</td>
<td></td>
</tr>
<tr>
<td><strong>Medications:</strong> Use of antibiotics; corticosteroids, chemotherapy, or any other immunosuppressants.</td>
<td></td>
</tr>
<tr>
<td><strong>Surgery or other treatments:</strong> Recent abdominal or thoracic surgery, splenectomy, endotracheal intubation, or any surgery with general anesthesia; tube feedings.</td>
<td></td>
</tr>
</tbody>
</table>

**Functional Health Patterns**

**Health perception–health management:** Cigarette smoking, alcoholism; recent upper respiratory tract infection, malaise.

**Nutritional–metabolic:** Anorexia, nausea, vomiting; chills.

**Activity-exercise:** Prolonged bed rest or immobility; fatigue, weakness; dyspnea, cough (productive or nonproductive); nasal congestion.

**Cognitive-perceptual:** Pain with breathing, chest pain, sore throat; headache; abdominal pain, muscle aches.

**Objective Data**

<table>
<thead>
<tr>
<th>General</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fever, restlessness or lethargy; splinting of affected area.</td>
</tr>
</tbody>
</table>

**Respiratory**

Tachypnea; pharyngitis; asymmetric chest movements or retraction; decreased excursion; nasal flaring; use of accessory muscles (neck, abdomen); grunting; crackles, friction rub on auscultation; dullness on percussion over consolidated areas, increased tactile fremitus on palpation; pink, rusty, purulent, green, yellow, or white sputum (amount may be scant to copious).

**Cardiovascular**

Tachycardia.

**Neurologic**

Changes in mental status, ranging from confusion to delirium.

**Possible Findings**

Leukocytosis; abnormal ABGs with ↓ or normal PaO2, ↑ PaCO2, and ↑ pH initially, and later ↓ PaO2, ↑ PaCO2, and ↓ pH; positive sputum Gram stain and culture; patchy or diffuse infiltrates, abscesses, pleural effusion, or pneumothorax on chest x-ray.*

*In the elderly dehydrated patient, chest x-ray may not be indicative of pneumonia until patient is rehydrated.

**ABGs,** Arterial blood gases; **AIDS,** acquired immunodeficiency syndrome; **COPD,** chronic obstructive pulmonary disease.
Section 5  Problems of Oxygenation: Ventilation

NURSING CARE PLAN 28-1

Pneumonia

NURSING DIAGNOSIS  Ineffective breathing pattern related to inflammation and pain as evidenced by dyspnea, tachypnea, nasal flaring, altered chest excursion

PATIENT GOAL  Demonstrates an effective respiratory rate, rhythm, and depth of respirations

OUTCOMES (NOC)  

Respiratory Status: Ventilation

- Respiratory rate ___
- Respiratory rhythm ___
- Ease of breathing ___
- Symmetrical chest expansion ___

Measurement Scale
1 = Severely compromised
2 = Substantially compromised
3 = Moderately compromised
4 = Mildly compromised
5 = Not compromised

PATIENT GOAL  Demonstrates effective coughing and increased air exchange

EXPERIENCES normal breath sounds

OUTCOMES (NOC)  

Respiratory Status: Airway Patency

- Respiratory rate ___
- Ease of breathing ___
- Moves sputum out of airway ___

Measurement Scale
1 = Severely compromised
2 = Substantially compromised
3 = Moderately compromised
4 = Mildly compromised
5 = Not compromised

NURSING DIAGNOSIS  Ineffective airway clearance related to retained secretions and excessive mucous as evidenced by ineffective cough, adventitious breath sounds, dyspnea

PATIENT GOAL  Demonstrates effective coughing and increased air exchange

EXPERIENCES normal breath sounds

OUTCOMES (NOC)  

Airway Management

- Auscultate breath sounds, noting areas of decreased/absent ventilation, and presence of adventitious sounds to obtain ongoing data on patient’s response to therapy.
- Remove secretions by encouraging coughing or by suctioning to clear airway.
- Regulate fluid intake to optimize fluid balance and liquefy secretions.

Cough Enhancement

- Assist patient to a sitting position with head slightly flexed, shoulders relaxed, and knees flexed to improve respiratory status.
- Instruct patient to inhale deeply several times, to exhale slowly, and to cough at the end of exhalation to promote effective coughing.
- Encourage use of incentive spirometry to aid in lung expansion and prevent atelectasis.

and intestinal contents. The ATS recommends that oral tubes be placed, rather than nasal tubes, to prevent nosocomial sinusitis.8

The patient who has difficulty swallowing (e.g., stroke patient) needs assistance in eating, drinking, and taking medication to prevent aspiration. The patient who has recently had surgery and others who are immobile need assistance with turning and deep-breathing measures at frequent intervals (see Chapter 20). The nurse must be careful to avoid overmedication with narcotics or sedatives, which can cause a depressed cough reflex and accumulation of fluid in the lungs. The gag reflex should be present in the individual who has had local anesthesia to the throat before the administration of fluids or food.
Strict medical asepsis and adherence to infection control guidelines should be practiced by the nurse to reduce the incidence of nosocomial infections. Poor hand-washing practices allow spread of pathogens via the hands of the health care worker. Staff should wash their hands each time before and after they provide care to a patient and whenever removing gloves. Respiratory devices can harbor microorganisms and have been associated with outbreaks of pneumonia. Strict sterile aseptic technique should be used when suctioning the trachea of a patient, and caution is required when handling ventilator circuits, tracheostomy tubing, and nebulizer circuits that can become contaminated from patient secretions.

**Acute Intervention.** Although many patients with pneumonia are treated on an outpatient basis, the nursing care plan for a patient with pneumonia (see NCP 28-1) is applicable to both these individuals and in-hospital patients. It is important for the nurse to remember that pneumonia is an acute, infectious disease. Although most cases of pneumonia are potentially completely curable, complications can result. The nurse must be aware of these complications and their manifestations. The essential components of nursing care for patients with pneumonia include monitoring physical assessment parameters, facilitating laboratory and diagnostic tests, providing treatment, and monitoring the patient’s response to treatment (see Table 28-6). Along with physical assessment (including pulse oximetry monitoring), prompt collection of specimens and prompt initiation of antibiotics (within 4 hours of arriving at the hospital) are critical. Oxygen therapy, administration of bronchodilators, hydration, nutritional support, and therapeutic positioning are part of the management of the patient.

Therapeutic positioning identifies the best position for the patient to assure stable oxygenation status. The concepts of therapeutic positioning are based on type of lung disease and patient response to positioning. The “good lung down” position is utilized for patients with unilateral lung disease, in whom better oxygenation is achieved when the unaffected lung (good lung) is placed in the down (lateral) position. For bilateral lung disease, research indicates that positioning the patient with the right lung down provides the best ventilation and perfusion. The use of bronchial hygiene techniques (postural drainage, percussion, vibration) is generally not warranted unless the patient is producing large volumes of sputum (>30 ml/day) or has x-ray evidence of mucous plugging and lobar collapse. Incentive spirometry, turning, coughing, and deep breathing all increase lung volume, mobilize secretions, and prevent atelectasis. Exercise and early ambulation augment bronchial hygiene and are encouraged as tolerated.

**Ambulatory and Home Care.** The patient needs to be reassured that complete recovery from pneumonia is possible. It is extremely important to emphasize the need to take all of the prescribed drug and to return for follow-up medical care and evaluation. The patient needs to be taught about any drug-drug and food-drug interactions for the prescribed antibiotic. Adequate rest is needed to maintain progress toward recovery and to prevent a relapse. The patient should be told that it may be weeks before the usual vigor and sense of well-being are felt. A prolonged period of convalescence may be necessary for the older adult or chronically ill patient.

The patient considered to be at risk for pneumonia should be told about available vaccines and should discuss them with the health care provider. Deep-breathing exercises or incentive spirometry therapy should be practiced for 6 to 8 weeks after the patient is discharged from the hospital.

**HEALTHY PEOPLE**

**Prevention of Respiratory Diseases**

- Avoid cigarette smoking and exposure to environmental smoke.
- Avoid exposure to allergens, indoor pollutants, and ambient air pollutants.
- Wear proper protection when working in an occupation with prolonged exposure to dust, fumes, or gases.
TUBERCULOSIS

Tuberculosis (TB) is an infectious disease caused by Mycobacterium tuberculosis. It usually involves the lungs, but it also occurs in the larynx, kidneys, bones, adrenal glands, lymph nodes, and membranes and can be disseminated throughout the body. TB is the world’s second most common cause of death from infectious disease, after HIV/AIDS. An estimated 2 billion people (one third of the world’s population) are infected with the M. tuberculosis bacteria. There are an estimated 8 to 9 million cases a year, and approximately 2 million people die annually. The highest incidence is seen in the developing countries of Africa and Asia, with a recent increase seen in the former Soviet Union. Although a decline has been seen in TB rates in the United States, over 14,000 new cases of TB per year are still being reported. Despite the decline in TB nationwide, rates have increased in certain states and high rates continue to be reported in certain populations (e.g., foreign-born persons, ethnic minorities [see Cultural and Ethnic Health Disparities Box]). Targeted interventions for these at-risk population and efforts toward a worldwide attack against TB are needed.

The major factors that have contributed to the resurgence of TB have been (1) high rates of TB among patients with HIV infection and (2) the emergence of multidrug-resistant (MDR) strains of M. tuberculosis. Once a strain of M. tuberculosis develops resistance to isoniazid and rifampin, it is defined as multidrug-resistant tuberculosis (MDR-TB). MDR-TB developed because patients had poor compliance with drug therapy, leading to treatment failure; were lost to follow-up treatment; or were placed on drug regimens to which their infections were no longer susceptible.

TB is seen disproportionately in the poor, the underserved, and minorities. Individuals at risk for TB include homeless persons, residents of inner-city neighborhoods, foreign-born persons, older adults, those in institutions (long-term care facilities, prisons), injection drug users, persons at poverty level, and those with poor access to health care. Immunosuppression from any etiology (e.g., HIV infection, malignancy) increases the risk of TB infection. The prevalence of TB is high in areas of the United States where there is a large population of Native Americans, such as Arizona and New Mexico. Higher TB rates are found in non-Hispanic whites in seven southeastern states of the United States compared with the rest of the country. In the United States, African Americans have TB rates 8 times higher than non-Hispanic whites. Health care workers with increased exposure to TB are considered at high risk.

Etiology and Pathophysiology

M. tuberculosis is a gram-positive, acid-fast bacillus that is usually spread from person to person via airborne droplets. These droplets are produced when the infected individual with pulmonary or laryngeal TB coughs, sneezes, speaks, or sings. Brief exposure to a few tubercle bacilli rarely causes an infection. Rather, TB is more commonly spread by repeated close contact (within 6 inches of the person’s mouth) with the infected person. TB is not highly infectious, and transmission usually requires close, frequent, or prolonged exposure. The disease cannot be spread by hands, books, glasses, or dishes.

The very small droplet nuclei, 1 to 5 μm in size, contain M. tuberculosis. Because they are so small in size, the particles remain airborne for minutes to hours. Once inhaled, these small nuclei lodge in alveoli in the small distal airways of the lung. M. tuberculosis replicates slowly and spreads via the lymphatic system. The organisms find favorable environments for growth primarily in the upper lobes of the lungs, kidneys, epiphyses of the bone, cerebral cortex, and adrenal glands.

Cellular immunity limits further multiplication and spread of the infection. After the cellular immune system is activated, a characteristic tissue granuloma, formed from alveolar macrophages, contains the bacteria and prevents further replication. At this point the person has TB infection, which can be detected by using the tuberculin skin test. (It may take 2 to 10 weeks for the infected person to develop a positive reaction to the tuberculin skin test.) The granuloma is usually not viable and, as a result, the infection remains contained and active disease may never occur. Persons who are infected with M. tuberculosis, but who do not have TB disease, cannot spread the infection to other people.

TB infection occurs when the bacteria are inhaling but there is an effective immune response and the bacteria become inactive. The majority of people mount effective immune responses to encapsulate these organisms for the rest of their lives, preventing primary infection from progressing to disease. TB infection in a person who does not have the active TB disease is not considered a case of TB and is often referred to as latent TB infection (LTBI). If the initial immune response is not adequate, control of the organisms is not maintained and active primary disease results. TB disease is defined as active bacteria that multiply and cause clinically active disease. Certain individuals are at a higher risk of active disease, including those who are immunosuppressed for any reason (e.g., patients with HIV infection, those receiving cancer chemotherapy or long-term corticosteroid therapy) or have diabetes mellitus.

Dormant but viable M. tuberculosis organisms persist for years. Once infected with TB, 3% to 5% of individuals develop TB disease within 1 year, and another 3% to 5% develop TB disease within their lifetime. Reactivation of LTBI can occur if the host’s
defense mechanisms become impaired. The reasons for reactivation of latent infection are not well understood, but they are related to decreased resistance found in older adults, individuals with concomitant diseases, and those who receive immunosuppressive therapy.

Classification

The American Thoracic Association and American Lung Association have adopted a classification system that covers the entire population (Table 28-7).

Clinical Manifestations

In the early stages of TB, the person is usually free of symptoms. People with LTBI have a positive skin test but are asymptomatic. Active TB disease may initially present with fatigue, malaise, anorexia, unexplained weight loss, low-grade fevers, and night sweats. A characteristic pulmonary manifestation is a cough that becomes frequent and produces white, frothy sputum. Dyspnea is unusual. Hemothysis is not a common finding and is usually associated with more advanced cases. Sometimes TB has more acute, sudden manifestations; the patient has high fever, chills, generalized flu-like symptoms, pleuritic pain, and a productive cough.

The HIV-infected patient with TB often has atypical physical examination and chest x-ray findings. Classic signs such as fever, cough, and weight loss may be attributed to Pneumocystis jiroveci pneumonia (PCP) or other HIV-associated opportunistic diseases. Clinical manifestations of respiratory problems in patients with HIV must be carefully investigated to determine the cause.

Complications

Miliary TB. Large numbers of organisms can invade the bloodstream and spread to all body organs. This hematogenous disseminated spread with involvement of many organs simultaneously is called miliary TB. It can occur as a result of primary disease or reactivation of latent infection. The patient may be either acutely ill with fever, dyspnea, and cyanosis or chronically ill with systemic manifestations of weight loss, fever, and gastrointestinal (GI) disturbance. Hepatomegaly, splenomegaly, and generalized lymphadenopathy may be present.

Pleural Effusion and Empyema. Pleural TB can result from either primary disease or reactivation of latent infection. A pleural effusion is caused by the bacteria in the pleural space triggering an inflammatory reaction and a pleural exudate of protein-rich fluid. Empyema is less common than effusion but may occur from large numbers of tubercular organisms in the pleural space.

Tuberculosis Pneumonia. Acute pneumonia may result when large amounts of tubercle bacilli are discharged from granulomas into the lung or lymph nodes. The clinical manifestations are similar to those of bacterial pneumonia, including chills, fever, productive cough, pleuritic pain, and leukocytosis.

Other Organ Involvement. Although the lungs are the primary site of TB, other body organs may also be involved. The most serious is involvement of the central nervous system with inflammation of the meninges. Bone (Pott’s disease of the spine) and joint tissue may be involved in the infectious disease process. The kidneys, adrenal glands, lymph nodes, and both female and male genital tracts may also be infected.

Diagnostic Studies

TB Skin Test. The intradermal administration of tuberculin as a diagnostic test for tuberculous infection has been used for over 100 years. The tuberculin skin test (TST) (Mantoux test) using purified protein derivative (PPD) is the best way to diagnose latent M. tuberculosis infection. Induration (not redness) at the injection site 48 to 72 hours after the test means the person has been exposed to TB and has developed antibodies. The reaction occurs 2 to 12 weeks after the initial exposure. The induration is measured and, based on the size of the induration and the population risk, an interpretation is made according to diagnostic standards for determining a positive test reaction. The procedure for performing the tuberculin skin test is described in Chapter 26.) If a person has a positive reaction, he or she should not be tested again since the sensitivity to tuberculin tends to persist throughout life.

Guidelines for targeted tuberculin testing emphasize targeting only high-risk groups and discourage testing low-risk individuals. Because the response to TST may be decreased in the immunocompromised patient, smaller induration reactions (≥5 mm) are considered positive. Two-step testing is recommended for initial testing for health care workers, who get repeated testing, and for individuals who have a decreased response to allergens. In these people, a second TST may cause an accelerated response (“booster effect”) that may be misinterpreted as a new conversion. (See Table 26-12 for guidelines in interpreting the TST.)

Chest X-Ray. Although the findings on chest x-ray examination are important, it is not possible to make a diagnosis of TB solely on the basis of this examination. This is because other diseases can mimic the x-ray appearance of TB. The chest x-ray findings suggestive of TB include upper lobe infiltrates, cavitary infiltrates, and lymph node involvement.

### TABLE 28-7 Classification of Tuberculosis (TB)

<table>
<thead>
<tr>
<th>Class</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>No TB exposure</td>
</tr>
<tr>
<td>1</td>
<td>TB exposure, no infection</td>
</tr>
<tr>
<td>2</td>
<td>Latent TB infection, no disease</td>
</tr>
<tr>
<td>3</td>
<td>TB clinically active</td>
</tr>
<tr>
<td>4</td>
<td>TB, but not clinically active</td>
</tr>
<tr>
<td>5</td>
<td>TB suspect</td>
</tr>
</tbody>
</table>

No current disease (history of exposure, negative tuberculin skin test) and clinical or x-ray evidence of current disease)

Positive bacteriologic studies, no x-ray findings compatible with TB, no clinical evidence of TB)

Tuberculosis skin test; no clinical or x-ray evidence of current disease)

Suspicious (diagnosis pending; person should not be in this classification for more than 3 months)

No TB exposure, not infected (no history of exposure, negative tuberculin skin test)

TB exposure, no evidence of infection (history of exposure, negative tuberculin skin test)

TB infection without disease (significant reaction to tuberculin skin test, negative bacteriologic studies, no x-ray findings compatible with TB, no clinical evidence of TB)

TB infection with clinically active disease (positive bacteriologic studies or both a significant reaction to tuberculin skin test and clinical or x-ray evidence of current disease)

No current disease (history of previous episode of TB or abnormal, stable x-ray findings in a person with a significant reaction to tuberculin skin test; negative bacteriologic studies if done; no clinical or x-ray evidence of current disease)

Four drug regimen options have been identified for the 6-month regimen to be maximally effective. If drug resistance to isoniazid (INH), four drugs are necessary in the initial phase (due to liver disease, pregnancy, etc.), option 4 is recommended.

Drug Alert - Isoniazid (INH)
- Alcohol may increase hepatotoxicity of drug. Instruct patient to avoid drinking alcohol during treatment.
- Monitor for signs of liver damage before and while taking drug.

Other drugs are primarily used for treatment of resistant strains or if the patient develops toxicity to the primary drugs. The newer rifamycins, rifabutin and rifapentine, should be considered first line in special situations: rifabutin for patients receiving medications that have interactions with rifampin or who have an intolerance to rifampin, and rifapentine with INH in once-weekly dosing for selected patients.

Directly observed therapy (DOT) involves providing the antituberculous drugs directly to the patient and watching as he or she swallows the medications. It is the preferred strategy for all patients with TB to assure adherence. When DOT is not being used, fixed-dose combination antituberculous drugs may enhance adherence to treatment recommendations. Combinations of isoniazid and rifampin (Rifamate) and of isoniazid, rifapentine, and pyrazinamide (Rifater) are available to simplify therapy. The therapy for persons with HIV follows the same therapy options outlined in Table 28-10 except that alternative regimens that include once-weekly INH plus rifapentine continuation dosing in any HIV-infected patient and twice-weekly INH plus rifapentine or rifabutin should not be used if CD4+ counts are < 100/μL. Health care providers must be alert for possible drug interactions between antiretrovirals and rifamycins.

Many second-line drugs for treatment of TB carry a greater risk of toxicity and require closer monitoring. Newer drugs for the treatment of TB that have been placed in categories of second-line drugs include the quinolones, such as levofloxacin, moxifloxacin, and gatifloxacin.

An important reason for follow-up care in the patient with TB is to ensure adherence to the treatment regimen. Noncompliance is a major factor in the emergence of multidrug resistance and treatment failures. Many individuals do not adhere to the treatment program in spite of understanding the disease process and the value of treatment. DOT is recommended for patients known to be at risk for noncompliance with therapy. DOT is an expensive but essential public health issue. Completing therapy is important because of the danger of reactivation of TB and MDR-TB seen in patients who do not complete the full course of therapy. In many areas, the public health nurse administers DOT at a clinic site.

Teaching patients about the side effects of these drugs and when to seek prompt medical attention is critical. The major side effect of isoniazid, rifampin, and pyrazinamide is nonviral hepatitis. Liver function tests should be monitored. Baseline liver function tests are done at the start of treatment. Monthly monitoring of liver function tests is done if baseline tests are abnormal.

Latent Tuberculosis Infection. Latent TB infection (LTBI) occurs when an individual becomes infected with M. tuberculosis. Drug therapy can be used to prevent a TB infection from developing into active disease. The indications for treatment of LTBI are presented in Table 28-11.

The drug generally used in treatment of LTBI is isoniazid (INH). It is effective and inexpensive and can be administered orally. Isoniazid is usually administered once daily for 6 to 12 months. The therapy is recommended.
### TABLE 28-9
**DRUG THERAPY**
Tuberculosis (TB)

<table>
<thead>
<tr>
<th>Drug</th>
<th>Mechanisms of Action</th>
<th>Side Effects</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>First-Line Drugs</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>• isoniazid (INH)</td>
<td>Bacteriocidal against rapidly dividing cells</td>
<td>Asymptomatic elevation of aminotransferases, clinical hepatitis, fulminant hepatitis, peripheral neurotoxicity, hypersensitivity (skin rash, arthralgia, fever)</td>
<td>Metabolism primarily by liver and excretion by kidneys, pyridoxine (vitamin B6) administration during high-dose therapy as prophylactic measure; routine monthly monitoring of liver tests not necessary unless preexisting liver disease or abnormal liver tests; safe in pregnancy.</td>
</tr>
<tr>
<td>• rifampin (Rifadin)</td>
<td>Bacteriocidal against rapidly dividing cells and against semidormant bacteria</td>
<td>Cutaneous reactions, GI disturbance (nausea, anorexia, abdominal pain), flu-like syndrome, hepatotoxicity, immunologic reactions, orange discoloration of bodily fluids (spumum, urine, sweat, tears); drug interactions</td>
<td>Most common use with isoniazid; safe in pregnancy; low incidence of side effects; suppression of effect of oral contraceptives; possible orange urine.</td>
</tr>
<tr>
<td>• ethambutol (Myambutol)</td>
<td>Bacteriostatic for the tubercle bacillus</td>
<td>Retrobulbar neuritis (decreased red-green color discrimination), peripheral neuritis (rare), skin rash</td>
<td>Side effects uncommon and reversible with discontinuation of drug; most common use as substitute drug when toxicity occurs with isoniazid or rifampin; safe in pregnancy; baseline Selin test and color discrimination and monthly if dose &gt;15-25 mg/kg.</td>
</tr>
<tr>
<td>• rifabutin (Mycobutin)</td>
<td>Bacteriocidal against rapidly dividing cells and against semidormant bacteria</td>
<td>Hematologic toxicity, uveitis (rare), GI symptoms, polyarthralgias, hepatotoxicity (&lt;1%); pseudojaundice (usually resolves), rash (rare), orange discoloration of bodily fluids</td>
<td>Used as a substitute for rifampin; reserved for patients unable to take rifampin. Patients should be warned of the orange discoloration. Soft contact lenses and clothing may be permanently stained. Drug used with caution in pregnancy.</td>
</tr>
<tr>
<td>• pyrazinamide (PZA)</td>
<td>Bacteriostatic effect against dormant or semidormant organisms</td>
<td>Hepatotoxicity, GI symptoms (nausea, vomiting), polyarthralgias, skin rash, hyperuricemia, dermatitis</td>
<td>No data on safety of PZA in pregnancy; World Health Organization recommends it for use in pregnancy. No sufficient data to recommend it in pregnancy; monitoring similar to rifampin.</td>
</tr>
<tr>
<td>• Rifapentine (Priftin)</td>
<td>Bacteriostatic against rapidly dividing cells and against semidormant bacteria</td>
<td>Similar to those of rifampin</td>
<td></td>
</tr>
<tr>
<td><strong>Second-Line Drugs</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>• cycloserine (Seromycin)</td>
<td>Bacteriocidal or bacteriostatic; inhibits cell wall synthesis</td>
<td>Central nervous system effects (headache, restlessness, seizures, psychosis); given with pyridoxine to prevent neurotoxic effects</td>
<td>Neuropsychiatric monitoring monthly; used for drug-resistant organisms that are susceptible to this drug; use in pregnancy only when no other alternative; contraindicated if history of psychosis. Liver function tests should be obtained at baseline and monitored monthly if there is underlying liver disease; thyroid-stimulating hormone should be measured at baseline and at monthly intervals.</td>
</tr>
<tr>
<td>• ethionamide (Trecator)</td>
<td>Exact mechanism of action unknown</td>
<td>Hepatotoxicity, neurotoxicity, GI effects (metallic taste, nausea, vomiting), endocrine effects (hypothyroidism, impotence)</td>
<td>Live function tests should be obtained at baseline and monitored monthly if there is underlying liver disease; thyroid-stimulating hormone should be measured at baseline and at monthly intervals.</td>
</tr>
<tr>
<td>• streptomycin</td>
<td>Bacteriocidal; inhibits protein synthesis</td>
<td>Ototoxicity, neurotoxicity, nephrotoxicity</td>
<td>Contraindicated in pregnancy; baseline hearing, Romberg test, serum creatinine measurements.</td>
</tr>
<tr>
<td>• capestomycin (Capastat)</td>
<td>Bacteriocidal; inhibits protein synthesis</td>
<td>Ototoxicity, nephrotoxicity</td>
<td>Cautious use in older adults; avoid in pregnancy; monitoring as for streptomycin.</td>
</tr>
<tr>
<td>• kanamycin (Kantrex) and amikacin</td>
<td>Bacteriocidal; inhibits protein synthesis</td>
<td>Ototoxicity, nephrotoxicity</td>
<td>Use in selected cases for treatment of resistant strains; contraindicated in pregnancy.</td>
</tr>
<tr>
<td>• para-aminosalicylic acid (PAS)</td>
<td>Interferes with metabolism of tubercle bacillus</td>
<td>Hepatotoxicity, GI distress, malabsorption syndrome, coagulopathy</td>
<td>Use in selected cases for treatment of resistant strains. Preferred oral agent for drug-resistant TB caused by organisms sensitive to this drug; avoid in pregnancy due to teratogenic effects.</td>
</tr>
<tr>
<td>• Fluoroquinolones: levofoxacin (Levaquin), moxifloxacin (Avelox, Vigamox), gatifloxacin (Tequin)</td>
<td>Bacteriocidal</td>
<td>GI disturbance, neurologic effects (dizzy, headache), rash</td>
<td></td>
</tr>
</tbody>
</table>

TABLE 28-10

<table>
<thead>
<tr>
<th>DRUG THERAPY</th>
<th>Regimen Options for the Initial Treatment of Tuberculosis</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Tuberculosis Previously Untreated: Initial Phase and Continuation Phase</strong></td>
<td></td>
</tr>
<tr>
<td><strong>Option 1</strong></td>
<td>Initial phase: 4-drug regimen consisting of INH, rifampin, pyrazinamide, ethambutol. Given daily for 56 doses OR 5 days/wk DOT for 40 doses. Ethambutol may be discontinued if susceptibility to INH or rifampin is documented. Continuation phase: INH, rifampin daily for 126 doses OR 5 days/wk DOT for 90 doses.</td>
</tr>
<tr>
<td><strong>Option 2</strong></td>
<td>Initial phase: 4-drug regimen consisting of INH, rifampin, pyrazinamide, ethambutol. Given daily for 14 doses, followed by twice weekly for 12 doses OR 5 days/wk DOT for 10 doses, then twice weekly for 12 doses. Continuation phase: INH, rifampin twice weekly for 36 doses OR once weekly for 18 doses.</td>
</tr>
<tr>
<td><strong>Option 3</strong></td>
<td>Initial phase: 4-drug regimen consisting of INH, rifampin, pyrazinamide, ethambutol. Given 3 times weekly for 24 doses. Continuation phase: INH, rifampin 3 times weekly for 54 doses.</td>
</tr>
<tr>
<td><strong>Option 4</strong></td>
<td>Initial phase: 3-drug regimen consisting of INH, rifampin, ethambutol. Given daily for 56 doses OR 5 days/wk DOT for 40 doses. Continuation phase: INH, rifampin daily for 217 doses OR 5 days/wk DOT for 155 doses.</td>
</tr>
</tbody>
</table>

Source: Centers for Disease Control and Prevention, MMWR Recomm Rep 52(RR-11), 2003.

DOT, Directly observed therapy; INH, isoniazid.

TABLE 28-11

<table>
<thead>
<tr>
<th>Indications for Treatment of Latent Tuberculosis Infection</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Newly infected patient at high risk</td>
</tr>
<tr>
<td>• Person with known or suspected HIV infection and positive tuberculin skin test</td>
</tr>
<tr>
<td>• Exposure of household members and other close associates to newly diagnosed patient</td>
</tr>
<tr>
<td>• Significant tuberculin skin test reactors with abnormal chest x-ray</td>
</tr>
<tr>
<td>• Significant tuberculin skin test reactors in special clinical situations (immunosuppression therapy, use of corticosteroids, diabetes mellitus, silicosis, gastrectomy, end-stage renal disease, head and neck cancer)</td>
</tr>
<tr>
<td>• Other significant tuberculin skin test converters (10-mm increase within a 2-yr period regardless of age)</td>
</tr>
<tr>
<td>• Other significant tuberculin skin test reactors (persons born outside of the United States from high-prevalence counties; medically underserved low-income populations, including high-risk racial or ethnic populations [e.g., Asian/Pacific Islanders, American Indian/Alaskan Native, African Americans, Hispanics], residents in long-term care facilities, health care workers, mycobacteriology laboratory technicians)</td>
</tr>
</tbody>
</table>


9 months. INH can be given daily or twice weekly with DOT. The 9-month regimen is more effective, but compliance issues may make the 6-month regimen preferable. For HIV patients and those with fibrotic lesions on chest x-ray, INH is given for 9 months. An alternative 4-month therapy with rifampin may be indicated if the patient is resistant to INH.

**Vaccine.** Immunization with bacille Calmette-Guérin (BCG) vaccine, a live, attenuated strain of Mycobacterium bovis, to prevent TB is in use in many parts of the world. Although millions of people have been vaccinated with BCG, the efficacy of the vaccine in preventing TB in adults is not clear. BCG was found to reduce the incidence of TB, particularly fatal disseminated TB, in infants and young children. Thus BCG is being offered at birth for children in high-prevalence areas in developing countries. The BCG vaccination can result in a positive reaction on the TST. This reaction will wane over time. Recent advances in DNA sequencing hold promise for an M. tuberculosis vaccine. The development of a TB vaccine is an urgent worldwide public health priority.

**NURSING MANAGEMENT**

**TUBERCULOSIS**

- **Nursing Assessment**
  - It is important to determine whether the patient was ever exposed to a person with TB. The patient should be assessed for productive cough, night sweats, afternoon temperature elevation, weight loss, pleuritic chest pain, and crackles over the apices of the lungs.
  - If the patient has a productive cough, an early-morning sputum specimen will be required for an acid-fast bacillus (AFB) smear to detect the presence of mycobacteria.

- **Nursing Diagnoses**
  - Nursing diagnoses for the patient with TB may include, but are not limited to, the following:
    - Ineffective breathing pattern related to decreased lung capacity
    - Imbalanced nutrition: less than body requirements related to chronic poor appetite, fatigue, and productive cough
    - Noncompliance related to lack of knowledge of disease process, lack of motivation, and long-term nature of treatment
    - Ineffective health maintenance related to lack of knowledge about the disease process and therapeutic regimen
    - Activity intolerance related to fatigue, decreased nutritional status, and chronic febrile episodes

- **Planning**
  - The overall goals are that the patient with TB will (1) comply with the therapeutic regimen, (2) have no recurrence of disease, (3) have normal pulmonary function, and (4) take appropriate measures to prevent the spread of the disease.

- **Nursing Implementation**
  - **Health Promotion.** The ultimate goal related to TB in the United States is eradication. Selective screening programs in known risk groups are of value in detecting individuals with TB. The person with a positive tuberculin skin test should have a chest x-ray to assess for the presence of TB. Another important measure is to identify the contacts of the individual who has TB. These
contacts should be assessed for the possibility of infection and the need for chemoprophylaxis.

**Acute Intervention.** Acute in-hospital care is seldom required for the patient with TB. If hospitalization is needed, it is usually for a brief period. Patients strongly suspected of having TB should (1) be placed on airborne isolation, (2) receive appropriate drug therapy, and (3) receive an immediate medical workup, including chest x-ray, sputum smear, and culture. Airborne infection isolation is indicated for the patient with pulmonary or laryngeal TB until the patient is considered to be noninfectious (effective drug therapy, improving clinically, three negative AFB smears). Airborne infection isolation refers to isolation of patients infected with organisms spread by the airborne route in a single-occupancy room with negative pressure and an airflow of 6 to 12 exchanges per hour. Ultraviolet radiation of the air in the upper part of the room is another approach to reduce airborne TB organisms. Ultraviolet lights are commonly seen in clinics and homeless shelters. Masks are needed to filter out droplet nuclei. High-efficiency particulate air (HEPA) masks are worn whenever entering the patient’s room because they can remove almost 100% of the small particles >3 mm in diameter. The mask must be molded to fit tightly around the nose and mouth.

The patient should be taught to cover the nose and mouth with paper tissue every time he or she coughs, sneezes, or produces sputum. The tissues should be thrown into a paper bag and disposed of with the trash, burned, or flushed down the toilet. The patient should also be taught careful hand-washing techniques after handling sputum and soiled tissues. If the patient needs to be out of the negative-pressure room, the patient wears a standard isolation mask to prevent coughing tubercular organisms into the environment. Special precautions should be taken during high-risk procedures that induce coughing, such as sputum induction, aerosolized pentamidine treatments, intubation, bronchoscopy, or endoscopy.

**Ambulatory and Home Care.** Patients who have responded clinically are discharged home despite positive smears if their household contacts have already been exposed and the patient is not posing a risk to susceptible persons. Determination of absolute noninfectiousness requires negative cultures. Most treatment failures occur because the patient neglects to take the drug, discontinues it prematurely, or takes it irregularly.

The nurse should teach the patient so that the need for compliance with the prescribed regimen is fully understood by the patient and family. Notification of the public health department is essential if drug compliance is questionable so that follow-up of close contacts can be accomplished. In some cases, the public health nurse will be responsible for DOT. When the chemotherapy regimen has been completed and there is evidence of negative cultures, the patient is improving clinically, and there is radiologic evidence of improvement, most individuals can be considered adequately treated. Follow-up care may be indicated during the subsequent 12 months, including bacteriologic studies and chest x-ray.

Because approximately 5% of individuals experience relapses, the patient should be taught to recognize the symptoms that indicate recurrence of TB. If these symptoms occur, immediate medical attention should be sought. The patient needs to be instructed about certain factors that could reactivate TB, such as immunosuppressive therapy, malignancy, and prolonged debilitating illness. If the patient experiences any of these events, the health care provider must be told so that reactivation of TB can be closely monitored. In some situations, it may be necessary to put the patient on anti-TB therapy.

**Evaluation**

The expected outcomes are that the patient with TB will have

- complete resolution of the disease
- normal pulmonary function
- absence of any complications
- no transmission of TB

**ATYPICAL MYCOBACTERIA**

Pulmonary disease that closely resembles TB may be caused by atypical acid-fast mycobacteria. This type of pulmonary disease is indistinguishable from TB clinically and radiologically but can be differentiated by bacteriologic culture. These organisms are not believed to be airborne and thus are not transmitted by droplet nuclei.

There are many atypical mycobacteria that can affect the lung. *M. avium* complex (MAC), an opportunistic mycobacterium found...
in water, causes pulmonary infection due to exposure to aerosols generated from baths, hot spas, and swimming pools. This is one of the most common of the atypical mycobacteria presently encountered. However, only a small number of people exposed to the organism will actually develop MAC lung disease. These are people who are immunosuppressed (e.g., HIV, cancer) or have underlying lung disease (e.g., cystic fibrosis). Other mycobacteria include M. kansasii, M. scrofulaceum, M. intracellulare, and M. xenopi. Treatment depends on identification of the causative agent and determination of drug sensitivity. Many of the drugs used in treating TB are used in combating infections from atypical mycobacteria.

**PULMONARY FUNGAL INFECTIONS**

Pulmonary fungal infections are increasing in incidence. They are found frequently in seriously ill patients being treated with corticosteroids, antineoplastic and immunosuppressive drugs, or multiple antibiotics. They are also found in patients with AIDS and cystic fibrosis. Community-acquired pulmonary lung infections include aspergillosis, cryptococcosis, and candidiasis. Types of fungal infections are presented in Table 28-12. These infections are not transmitted from person to person, and the patient does not have to be placed in isolation. The clinical manifestations are similar to those of bacterial pneumonia. Skin testing, serology, and biopsy methods are available to assist in identifying the infecting organism.

**Collaborative Care**

Amphotericin B remains the standard therapy for treating serious systemic fungal infections. It must be given intravenously to achieve adequate blood and tissue levels because it is poorly absorbed from the GI tract. Amphotericin B is considered a toxic agent and determination of drug sensitivity. Many of the drugs used in treating TB are used in combating infections from atypical mycobacteria.

<table>
<thead>
<tr>
<th>Infection</th>
<th>Characteristics</th>
</tr>
</thead>
<tbody>
<tr>
<td>Histoplasmosis</td>
<td>Indigenous to soil of North American river valleys, inhalation of mycelia into lungs, infected individual often free of symptoms, generally self-limiting, chronic disease similar to TB</td>
</tr>
<tr>
<td>Cryptococcosis</td>
<td>Indigenous to semiarid regions of southwestern United States, inhalation of arthropores into lungs, suppurative and granulomatous reaction in lungs, symptomatic infection in one third of individuals</td>
</tr>
<tr>
<td>Blastomycosis</td>
<td>Indigenous to southeastern and midwestern United States, inhalation of fungus into lungs, progression of disease often insidious, possible involvement of skin</td>
</tr>
<tr>
<td>Cryptococcus</td>
<td>True yeast, indigenous worldwide in soil and pigeon excreta, inhalation of fungus into lungs, possible meningitis</td>
</tr>
<tr>
<td>Aspergillus</td>
<td>True mold inhabiting mouth, widely distributed, invasion of lung tissue resulting in possible necrotizing pneumonia; in individual with asthma, allergic broncho-pulmonary aspergillosis may require corticosteroid therapy</td>
</tr>
<tr>
<td>Candidiasis</td>
<td>Leading cause of mycotic infections in hospitalized and immunocompromised hosts, ubiquitous and frequent colonization of upper respiratory and GI tracts, infections often following broad-spectrum antibiotic therapy (systemic or inhaled), possible development of localized pulmonary infiltrate to widespread bilateral consolidation with hypoxemia</td>
</tr>
<tr>
<td>Actinomycosis</td>
<td>Not a true fungus, pseudohyphae present; anaerobic; gram-positive, higher bacteria with branching hyphae; presence of necrotizing pneumonia after aspiration; pneumonitis, commonly in lower lobes with abscess or empyema formation</td>
</tr>
<tr>
<td>Nocardiosis</td>
<td>Not a true fungus; aerobic, higher bacteria with branching hyphae; soil saprophyte widely distributed in nature; acquisition of infection from nature; rarely present in sputum without accompanying disease</td>
</tr>
<tr>
<td>Pneumocystis Pneumonia (PCP)</td>
<td>This organism rarely causes pneumonia in the healthy individual; fungus present in the environment; common opportunistic pneumonia in persons with AIDS and impaired immune systems.</td>
</tr>
</tbody>
</table>

*AIDS, Acquired immunodeficiency syndrome.*

TABLE 28-12 Fungal Infections of the Lung
LUNG ABSCESS

Etiology and Pathophysiology

Lung abscess is a pus-containing lesion of the lung parenchyma that gives rise to a cavity. The cavity is formed by necrosis of the lung tissue. In many cases the causes and pathogenesis of lung abscess are similar to those of pneumonia. Most lung abscesses are caused by aspiration of material from the GI tract into the lungs. Risk factors for aspiration include alcoholism, seizure disorders, neuromuscular diseases, drug overdose, general anesthesia, and stroke. The areas of the lung most commonly affected are the superior segments of the lower lobes and the posterior segments of the upper lobes. Fibrous tissue usually forms around the abscess in an attempt to wall it off. The abscess may erode into the bronchial system, causing the production of foul-smelling or sour-tasting sputum. It may grow toward the pleura and cause pleuritic pain. Multiple small abscesses can occur within the lung.

Infectious agents generally cause lung abscesses. The organisms involved cause infection and necrosis of the lung tissue. Examples include enteric gram-negative organisms (e.g., Klebsiella), S. aureus, and anaerobic bacilli (e.g., Bacteroides). Putrid, offensive sputum is associated with anaerobic bacteria. Lung abscess can also result from malignant growth, TB, and various parasitic and fungal diseases of the lung.

Clinical Manifestations and Complications

The onset of a lung abscess is usually insidious, especially if anaerobic organisms are the primary cause. A more acute onset occurs with aerobic organisms. The most common manifestation is cough-producing purulent sputum (often dark brown) that is foul smelling and foul tasting. Hemoptysis is common, especially at the time that an abscess ruptures into a bronchus. Other common manifestations are fever, chills, prostration, pleuritic pain, dyspnea, cough, and weight loss.

Physical examination of the lungs indicates dullness to percussion and decreased breath sounds on auscultation over the segment of lung involved. There may be transmission of bronchial breath sounds to the periphery if the communicating bronchus becomes patent and drainage of the segment begins. Crackles may also be present in the later stages as the abscess drains. Oral examination often reveals dental caries, gingivitis, and periodontal infection.

Complications that can occur include chronic pulmonary abscess, bronchiectasis, brain abscess as a result of the hematogenous spread of infection, bronchopleural fistula, and empyema from abscess perforation into the pleural cavity.

Diagnostic Studies

A chest x-ray may reveal a solitary cavitary lesion with fluid that is identified as an air-fluid level. Computed tomography (CT) scanning is very helpful if there is a question of cavitation not clearly seen on chest x-ray. Lung abscess, in contrast to other types of abscesses, does not require assisted drainage, as long as there is drainage via the bronchus. Routine sputum cultures can be collected. However, contaminants can confuse the results, and it is difficult to isolate anaerobic bacteria. Pleural fluid and blood cultures may be obtained. Bronchoscopy may be used in cases of abscess in which drainage is delayed or in which there are factors that suggest an underlying malignancy.

NURSING and COLLABORATIVE MANAGEMENT

LUNG ABSCESS

Antibiotics given for a prolonged period (up to 2 to 4 months) are usually the primary method of treatment. Penicillin has historically been the drug of choice because of the frequent presence of anaerobic organisms. However, recent studies suggest that there is β-lactamase production by the anaerobic bacteria involved in abscesses of the lung, and they are resistant to penicillin. Clindamycin (Cleocin) has been shown to be superior to penicillin and is the standard treatment for an anaerobic lung infection. Patients with purulent lung abscesses usually show clinical improvement with decreased fever within 3 to 4 days of beginning antibiotics.

Because of the need for prolonged antibiotic therapy, the patient must be aware of the importance of continuing the medication for the prescribed period. The patient needs to know about untoward side effects that need to be reported to the health care provider. Sometimes the patient is asked to return periodically during the course of antibiotic therapy for repeat cultures and sensitivity tests to ensure that the infecting organism is not becoming resistant to the antibiotic. When antibiotic therapy is completed, the patient is reevaluated.

The patient should be taught how to cough effectively (see Table 29-000). Chest physiotherapy and postural drainage are sometimes used to drain abscesses located in the lower or posterior portions of the lung. Postural drainage to the lung area involved will aid the removal of secretions (see Fig. 29-000). Rest, good nutrition, and adequate fluid intake are all supportive measures to facilitate recovery. If dentition is poor and dental hygiene is not adequate, the patient should be encouraged to obtain dental care.

Surgery is rarely indicated but occasionally may be necessary when reinfection of a large cavitary lesion occurs or to establish a diagnosis when there is evidence of an underlying neoplasm or chronic associated disease. The usual procedure in such cases is a lobectomy or pneumonectomy. An alternative to surgery is percutaneous drainage, but there is a potential risk of contamination of the pleural space.

ENVIRONMENTAL LUNG DISEASES

Environmental or occupational lung diseases are caused or aggravated by workplace or environmental exposure and are preventable. They result from inhaled dust or chemicals. The duration of exposure and the amount of inhalant have a major influence on whether the exposed individual will have lung damage. Occupational and environmental asthma is especially prevalent and is discussed in Chapter 29. The other major groups of diseases are pneumoconiosis, chemical pneumonitis, and hypersensitivity pneumonitis.

Pneumoconiosis is a general term for a group of lung diseases caused by inhalation and retention of dust particles. The literal meaning of pneumoconiosis is “dust in the lungs.” Examples of this condition are silicosis, asbestosis, and berylliosis. The classic response to the inhaled substance is diffuse parenchymal infiltration with phagocytic cells. This eventually results in diffuse pulmonary fibrosis (excess connective tissue). Fibrosis is the result of tissue repair after inflammation. Pneumoconiosis and other environmental lung diseases are presented in Table 28-13. Hantavirus, a potentially fatal disease with outbreaks reported in the United States and Canada, is transmitted by inhalation of aerosolized rodent excreta particles.
Chemical pneumonitis results from exposures to toxic chemical fumes. Acutely, there is diffuse lung injury characterized as pulmonary edema. Chronically, the clinical picture is that of bronchiolitis obliterans (obstruction of the bronchioles due to inflammation and fibrosis), which is usually associated with a normal chest x-ray or one that shows hyperinflation. An example is silo filler’s disease.

Hypersensitivity pneumonitis or extrinsic allergic alveolitis is a form of parenchymal lung disease seen when antigens are inhaled to which an individual is allergic. Examples include bird fancier’s lung and farmer’s lung.

Lung cancer, either squamous cell carcinoma or adenocarcinoma, is the most frequent cancer associated with asbestos exposure. People with more exposure are at a greater risk of disease. There is a minimum lapse of 15 to 19 years between first exposure and development of lung cancer. Mesotheliomas, both pleural and peritoneal, are also associated with asbestos exposure.

Clinical Manifestations
Acute symptoms of pulmonary edema may be seen following early exposures to chemical fumes. However, symptoms of many environmental lung diseases may not occur until at least 10 to 15 years after the initial exposure to the inhaled irritant. Dyspnea and cough are often the earliest manifestations. Chest pain and cough with sputum production usually occur later. Pulmonary function studies often show reduced vital capacity. A chest x-ray will often reveal lung involvement specific to the primary problem. CT scans have been shown to be useful in detecting early lung involvement. Cor pulmonale (described later in this chapter) is a late complication, especially in conditions characterized by diffuse pulmonary fibrosis. Complications that often result are pneumonia, chronic bronchitis, emphysema, and lung cancer. Manifestations of these complications can be the reason the patient seeks health care.

### TABLE 28-13 Environmental Lung Diseases

<table>
<thead>
<tr>
<th>Disease</th>
<th>Agents/Industries</th>
<th>Description</th>
<th>Complications</th>
</tr>
</thead>
<tbody>
<tr>
<td>Asbestosis</td>
<td>Asbestos fibers present in insulation, construction material, shipyards, textiles</td>
<td>Disease appears 15-35 yr after first exposure. Interstitial fibrosis develops. Pleural plaques, which are calcified lesions, develop on pleura. Dyspnea, basal crackles, and decreased vital capacity are early manifestations.</td>
<td>Diffuse interstitial pulmonary fibrosis; lung cancer, especially in cigarette smokers; mesothelioma (rare type of cancer affecting pleura and peritoneal membrane)</td>
</tr>
<tr>
<td>Berylliosis</td>
<td>Beryllium dust present in aircraft manufacturing, metalurgy, rocket fuels</td>
<td>Formation of noncaseating granulomas. Acute pneumonitis occurs after heavy exposure. Interstitial fibrosis can also occur.</td>
<td>Progression of disease possible after removal of stimulating inhalant</td>
</tr>
<tr>
<td>Bird fancier’s, breeder’s, or handler’s lung</td>
<td>Bird droppings or feathers</td>
<td>Hypersensitivity pneumonitis is present.</td>
<td>Progressive fibrosis of lung</td>
</tr>
<tr>
<td>Byssinosis</td>
<td>Cotton, flax, and hemp dust (textile industry)</td>
<td>Airway obstruction is caused by contraction of smooth muscles. Chronic disease results from severe airway obstruction and decreased elastic recoil.</td>
<td>Progression of chronic disease after cessation of dust exposure</td>
</tr>
<tr>
<td>Coalworker’s pneumoconiosis (black lung)</td>
<td>Coal dust</td>
<td>Incidence is high (20%-30%) in coal workers. Deposits of carbon dust cause lesions to develop along respiratory bronchioles. Bronchioles dilate because of loss of wall structure. Chronic airway obstruction and bronchitis develop. Dyspnea and cough are common early symptoms.</td>
<td>Progressive, massive lung fibrosis; increased risk of chronic bronchitis and emphysema with smoking</td>
</tr>
<tr>
<td>Farmer’s lung</td>
<td>Inhalation of airborne material from moldy hay or similar matter</td>
<td>Hypersensitivity pneumonitis occurs. Acute form is similar to pneumonia, with manifestations of chills, fever, and malaise. Chronic, insidious form is type of pulmonary fibrosis.</td>
<td>Progressive fibrosis of lung</td>
</tr>
<tr>
<td>Hantavirus pulmonary syndrome (HPS)</td>
<td>Rodent droppings inhaled while in rodent-infested areas</td>
<td>Acute hemorrhagic fever associated with severe pulmonary and cardiovascular collapse and death. Incubation period is 1-4 wk with prodrome (3-5 days) of flu-like symptoms. No cure or specific treatment exists.</td>
<td>CDC recommends rapid transfer to ICU with supportive therapy and early intervention vital; research on this virus is done in high-level biocontainment facilities</td>
</tr>
<tr>
<td>Silicosis</td>
<td>Silica dust present in quartz rock in mining of gold, copper, tin, coal, lead; also present in sandblasting, foundries, quarries, pottery making, masonry</td>
<td>In chronic disease, dust is engulfed by macrophages and may be destroyed, resulting in fibrotic nodules. Acute disease results from intense exposure in short time period. Within 5 yr, it progresses to severe disability from lung fibrosis.</td>
<td>Increased susceptibility to tuberculosis; progressive, massive fibrosis; high incidence of chronic bronchitis</td>
</tr>
<tr>
<td>Silo filler’s disease</td>
<td>Nitrogen oxides from fermentation of vegetation in freshly filled silo</td>
<td>Chemical pneumonitis occurs.</td>
<td>Progressive bronchiolitis obliterans</td>
</tr>
</tbody>
</table>

CDC, Centers for Disease Control and Prevention; ICU, intensive care unit.
Collaborative Care
The best approach to management of environmental lung diseases is to try to prevent or decrease environmental and occupational risks. Well-designed, effective ventilation systems can reduce exposure to irritants. Wearing masks is appropriate in some occupations. Periodic inspections and monitoring of workplaces by agencies such as the Occupational Safety and Health Administration (OSHA) and the National Institute for Occupational Safety and Health (NIOSH) reinforce the obligations of employers to provide a safe work environment. NIOSH is responsible for workplace safety and health regulations in the United States (www.cdc.gov/niosh/homepage.html).

Active and passive smoking increases the insult to the lungs. Patients need to be advised that they should not smoke and to avoid passive smoke as well. Passive smoke, inhalation of environmental tobacco smoke by a nonsmoker, is an important source of occupational exposure with increased risk for development of lung cancer. This has led to regulations requiring a smoke-free workspace for all employees.

Early diagnosis is essential if the disease process is to be halted. There is no specific treatment for most environmental lung diseases. The best treatment is to decrease or stop exposure to the harmful agent. Strategies are directed toward providing symptomatic relief. If there are coexisting problems, such as pneumonia, chronic bronchitis, emphysema, or asthma, they need to be treated.

LUNG CANCER
Lung cancer is the leading cause of cancer-related deaths in the United States. Lung cancer accounts for 28% of all cancer deaths. There are about 172,570 new cases of lung cancer every year in the United States. It is the leading cause of cancer death among men; 58% of the lung cancer deaths are in men. African Americans have the highest death rate and Hispanics the lowest.30

Lung cancer is now the leading cause of death in women, surpassing breast cancer. The Surgeon General’s report, Women and Smoking: A Report of the Surgeon General—2001, identified a 600% increase in women’s death rates from lung cancer since 1950 and attributes this to smoking.31 The CDC compared lung cancer death rates between the genders. They found that mortality rates increased from 1968 to 1999 for women (266%) versus men (15%), highlighting the need for smoking prevention strategies targeting women.32 In addition, a total of 36.5% of teenagers (male and female) smoke. The overall 5-year survival rate from lung cancer is 15%. Lung cancer most commonly occurs in individuals more than 50 years of age who have a long history of cigarette smoking. The disease is found most in persons 40 to 75 years of age, with peak incidence between 55 and 65 years of age.

Etiology
Cigarette smoking is the most important risk factor in the development of lung cancer. Smoking is responsible for approximately 80% to 90% of all lung cancers. Tobacco smoke contains 60 carcinogens in addition to substances (carbon monoxide, nicotine) that interfere with normal cell development. Cigarette smoking, a lower airway irritant, causes a change in the bronchial epithelium, which usually returns to normal when smoking is discontinued. The risk of lung cancer is gradually lowered when smoking ceases, and continues to decline with time. Ten years following cessation of smoking, lung cancer mortality risk is reduced 30% to 50%. The Memorial Sloan-Kettering Cancer Center has developed a tool that calculates risk for developing lung cancer for older smokers and ex-smokers (www.mskcc.org/PredictionTools/LungCancer).

The risk of developing lung cancer is directly related to total exposure to cigarette smoke, measured by total number of cigarettes smoked in a lifetime, age of smoking onset, depth of inhalation, tar and nicotine content, and the use of unfiltered cigarettes. Sidestream smoke (smoke from burning cigarettes, cigars) contains the same carcinogens found in mainstream smoke (smoke inhaled and exhaled from the smoker). This environmental tobacco smoke inhaled by nonsmokers poses a 35% increased risk of the development of lung cancer in nonsmokers.33 This exposure, called passive smoking, can occur early in life for children of smokers. Children are more vulnerable to environmental smoke than adults because their respiratory and immune systems are not fully developed. Childhood exposure to cigarette smoke is associated with increased prevalence of asthma among adults. Children exposed to cigarette smoke are more likely to become smokers.

Those who smoke pipes and cigars also have an increased risk of developing lung cancer; their risk is slightly higher than that of nonsmokers. Cigar smokers are at higher risk for lung cancer than are pipe smokers. However, heavy smoking of cigars and inhalation of smoke from small cigars correlate with the rates of lung cancer observed in cigarette smokers.

Another major risk factor for lung cancer is inhaled carcinogens. These include asbestos, radon, nickel, iron and iron oxides, uranium, polycyclic aromatic hydrocarbons, chromates, arsenic, and air pollution. Exposure to these substances is common for employees of industries involved in mining, smelting, or chemical or petroleum manufacturing. The cigarette smoker who is also exposed to one or more of these chemicals or to high amounts of air pollution is at significantly higher risk for lung cancer.

**Gender Differences**

<table>
<thead>
<tr>
<th>Lung Cancer</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Men</strong></td>
</tr>
<tr>
<td>- Men with lung cancer have a worse prognosis than women.</td>
</tr>
<tr>
<td>- Lung cancer incidence and deaths are decreasing in men.</td>
</tr>
<tr>
<td><strong>Women</strong></td>
</tr>
<tr>
<td>- Women who smoke are at 2 times greater risk of developing lung cancer than men who smoke.</td>
</tr>
<tr>
<td>- Women:</td>
</tr>
<tr>
<td>- develop lung cancer after fewer years of smoking as compared to men</td>
</tr>
<tr>
<td>- develop lung cancer at a younger age than men</td>
</tr>
<tr>
<td>- are more likely to develop small cell carcinoma than men</td>
</tr>
<tr>
<td>- Nonsmoking women are at greater risk of developing lung cancer than men.</td>
</tr>
</tbody>
</table>
There are marked variations in a person’s propensity to develop lung cancer. To date no genetic abnormality has conclusively been identified for lung cancer. It is known that the carcinogens in cigarette smoke directly damage DNA. One theory is that people have different genetic carcinogen-metabolizing pathways.

Pathophysiology

The pathogenesis of primary lung cancer is not well understood. Lung cancer is thought to arise from bronchial epithelial cells (bronchogenic). These cells grow slowly, and it takes 8 to 10 years for a tumor to reach 1 cm in size, which is the smallest lesion detectable on an x-ray. Lung cancers occur primarily in the segmental bronchi or beyond and have a preference for the upper lobes of the lungs (Fig. 28-2). Pathologic changes in the bronchial system show nonspecific inflammatory changes with hypersecretion of mucus, desquamation of cells, reactive hyperplasia of the basal cells, and metaplasia of normal respiratory epithelium to stratified squamous cells. Pathologic types of lung cancer are presented in Fig. 28-3.

Primary lung cancers are often categorized into two broad subtypes (Table 28-14), non–small cell lung cancer (NSCLC) (80%) and small cell lung cancer (SCLC) (20%). Lung cancers metastasize primarily by direct extension and via the blood circulation and the lymph system. The common sites for metastatic growth are the liver, brain, bones, scalene lymph nodes, and adrenal glands. Paraneoplastic Syndrome. Certain lung cancers cause the paraneoplastic syndrome, which results from production of active substances (e.g., hormones, enzymes, antigens) either by the tumor itself or in response to the tumor. SCLCs are most commonly associated with the paraneoplastic syndrome. The systemic manifestations seen are hormonal, dermatologic, neuromuscular, vascular, hematologic, and connective tissue syndromes. These syndromes can respond temporarily to symptomatic treatment, but they are impossible to control without successful treatment of the underlying lung cancer.

Clinical Manifestations

Lung cancer is clinically silent for most individuals for the majority of its course. Asymptomatic patients whose cancer is found on routine chest x-ray account for about 10% of new cases. The clinical manifestations of lung cancer are usually nonspecific and appear late in the disease process. Manifestations depend on the type of primary lung cancer, its location, and metastatic spread. Persistent pneumonitis that is a result of obstructed bronchi may be one of the earliest manifestations, causing fever, chills, and cough.

One of the most common symptoms, and often the one reported first, is a persistent cough that may be productive of sputum. Blood-tinged sputum may be produced because of bleeding caused by malignancy, but hemoptysis is not a common symptom. Chest pain may be present and localized or unilateral, ranging from mild to severe. Dyspnea and an auscultatory wheeze may be present if there is bronchial obstruction.

Later manifestations may include nonspecific systemic symptoms such as anorexia, fatigue, weight loss, and nausea and vomiting. Hoarseness may be present as a result of involvement of the laryngeal nerve. Unilateral paralysis of the diaphragm, dysphagia, and superior vena cava obstruction may occur because of intratho-
TABLE 28-14  Comparison of the Types of Primary Lung Cancer

<table>
<thead>
<tr>
<th>Cell Type</th>
<th>Risk Factors</th>
<th>Characteristics</th>
<th>Response to Therapy</th>
</tr>
</thead>
<tbody>
<tr>
<td>Non–Small Cell Lung Cancer (NSCLC)</td>
<td>• Adenocarcinoma: Has been associated with lung scarring and chronic interstitial fibrosis; is related to cigarette smoking.</td>
<td>Most common type; Accounts for approximately 30%-40% of lung cancers; more common in women; often has no clinical manifestations until widespread metastasis is present; usually begins in mucous glandular tissue; is most commonly located in peripheral portions of lungs.</td>
<td>Surgical resection is often attempted; cancer does not respond well to chemotherapy.</td>
</tr>
<tr>
<td>• Squamous cell carcinoma: Almost always associated with cigarette smoking; is associated with exposure to environmental carcinogens (e.g., uranium, asbestos).</td>
<td>Second most common type of lung cancer; accounts for 30%-35% of lung cancers; is more common in men; arises from the bronchial epithelium (surface cells) of the lungs or bronchus, slow-growing cancer that usually begins in the bronchial tubes; diseases nodules tend to be clumped together; produces earlier symptoms because of bronchial obstructive characteristics; does not have a strong tendency to metastasize.</td>
<td>Surgical resection is often attempted; life expectancy is better than for small cell lung cancer.</td>
<td></td>
</tr>
<tr>
<td>• Large cell (undifferentiated) carcinoma: High correlation with cigarette smoking and exposure to environmental carcinogens.</td>
<td>The least common form of NSCLC; accounts for 5%-15% of lung cancers; composed of large-sized cells that are anaplastic and often arise in the bronchi; commonly causes cavitation; is highly metastatic via lymphatics and blood; commonly peripheral rather than central location in lungs.</td>
<td>Surgery is not usually attempted because of high rate of metastases; tumor may be radiosensitive but often recurs.</td>
<td></td>
</tr>
<tr>
<td>Small Cell Lung Cancer (SCLC)</td>
<td>• Small cell carcinoma: Associated with cigarette smoking, exposure to environmental carcinogens.</td>
<td>Accounts for 15%-25% of lung cancers; is most malignant form; tends to spread early via lymphatics and bloodstream; is frequently associated with endocrine disturbances; predominantly central and can cause bronchial obstruction and pneumonia.</td>
<td>Cancer has poorest prognosis; however, chemotherapy advances have been substantial; radiation is used as adjuvant therapy as well as palliative measure; average median survival is 16 months.</td>
</tr>
</tbody>
</table>

*See Fig. 28-3.

Diagnostic Studies
Chest x-rays may initially identify a lung mass or infiltrate. The findings may show obstructive features of the tumor such as atelectasis and pneumonia. The x-ray can also show evidence of metastasis to the ribs or vertebrae and the presence of pleural effusion. CT scanning is the single most effective noninvasive technique for evaluating lung cancer. CT scans of the brain and bone scans complete the evaluation for metastatic disease. With CT scans, the location and extent of masses in the chest can be identified, as well as any mediastinal involvement or lymph node enlargement. Magnetic resonance imaging (MRI) may be used in combination with or instead of CT scans. Positron emission tomography (PET) promises to be a useful diagnostic tool in early clinical staging. PET allows measurement of differential metabolic activity in normal and diseased tissues.

Sputum cytology can identify malignant cells, but results are positive in only 20% to 30% of specimens because the malignant cells may not be present in the sputum. Biopsy is necessary for a definitive diagnosis. If the mass is well visualized on CT scan and accessible, cells can be aspirated using guided percutaneous fine-needle biopsy technique. If it is too small or difficult to access, an alternative is a bronchoscopy to obtain transbronchial aspirations, brushings, and washings. Mediastinoscopy is another technique that involves the insertion of a scope via a small anterior chest incision into the mediastinum. This is done to obtain tissue from the mediastinal lymph nodes to assess for metastasis. Video-assisted thoracoscopic surgery (VATS) involves inserting a scope via a small thoracic incision in the chest wall to obtain tissue samples inaccessible by mediastinoscopy. If a thoracentesis is performed to relieve a pleural effusion, the fluid should be analyzed for malignant cells. Table 28-15 summarizes the diagnostic management of lung cancer.

Staging. Staging of non–small cell lung cancer (NSCLC) is performed according to the TNM staging system (see Table 16-5). Assessment criteria are T, which denotes tumor size, location, and degree of invasion; N, which indicates regional lymph node involvement; and M, which represents the presence or absence of distant metastases. Depending on the TNM designation, the tumor is then staged, which assists in estimating prognosis and determining the appropriate therapy. A simplified version of staging of NSCLC is presented in Table 28-16. Patients with stages I, II, and IIIA disease may be surgical candidates. However, stage IIB or IV disease is usually inoperable and has a poor prognosis.

Staging of small cell lung cancer (SCLC) by TNM has not been published as palliative measure; average median survival is 16 months.
Respiratory System

Extensive SCLC means that some of the cancer extends to the chest wall or to other parts of the body. On average, patients with cancer is very controversial. The U.S. Preventive Services Task Force (www.preventiveservices.ahrq.gov) states there is insufficient evidence to recommend screening for lung cancer with chest x-ray or CT and that smoking cessation should be emphasized to the patient as the preferred modality for reducing lung cancer mortality. A new noninvasive blood test that could detect lung cancer in its earliest stages is being studied at Duke University Medical Center.

The nurses’ role in screening for lung cancer includes educating patients on the strengths and limitations of the proposed screening measures for early detection of lung cancer. Smoking cessation and prevention are essential in decreasing morbidity and mortality associated with lung cancer. Nurses play a vital role in counseling patients in tobacco cessation and prevention. (Smoking cessation is discussed in Chapter 12.)

**Collaborative Care**

**Surgical Therapy.** Surgical resection is the treatment of choice in NSCLC stages I and II, because the disease is potentially curable with resection. The 5-year survival in stage I disease with complete resection is 60% to 80%; in patients with stage II disease, it is 40% to 60%. For other NSCLC stages, surgery may be indicated in conjunction with radiation therapy and/or chemotherapy. Fifty percent of all NSCLC lung cancers are not resectable at the time of diagnosis. In limited-stage SCLC (which is rare), surgical resection, chemotherapy, and radiation therapy may be recommended. The surgical procedures that may be performed include pneumonectomy (removal of one entire lung), lobectomy (removal of one or more lobes of the lung), or lung-conserving resection or segmental or wedge resection procedures. When the tumor is considered operable, the patient’s cardiopulmonary status must be evaluated to determine the ability to withstand surgery. This is done by clinical studies of pulmonary function, arterial blood gases (ABGs), and other tests, as indicated by the individual’s status.

**Radiation Therapy.** Radiation therapy may be used with the intent to cure in the individual who is unable to tolerate surgical resection due to comorbidities. It may also be used as adjuvant therapy after resection of the tumor. Recent guidelines indicate that radiation therapy is useful as part of the treatment for locally advanced, unresectable NSCLC. Improved survival and symptom control have been achieved with combination treatments (radiation therapy, surgery, chemotherapy). Complications of radiation therapy include esophagitis, skin irritation, and radiation pneumonitis. Hyperfractionated radiation, given twice daily, has improved response and survival rates in SCLC. However, there is increased toxicity, particularly esophagitis.

Radiation therapy is also used to relieve symptoms dyspnea and hemoptysis resulting from bronchial obstructive tumors and to treat superior vena cava syndrome. It can also be used to treat pain that is caused by metastatic bone lesions or cerebral metastasis. Radiation may be used preoperatively to reduce tumor mass prior to resection or postoperatively as an adjuvant measure.

**Chemotherapy.** Chemotherapy may be used in the treatment of nonresectable tumors or as adjuvant therapy to surgery in NSCLC. A variety of chemotherapy drugs and multidrug regimens (i.e., protocols), including combination chemotherapy, have been used. These

---

**TABLE 28-15**

<table>
<thead>
<tr>
<th>Characteristics</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Stage III</strong></td>
</tr>
<tr>
<td><strong>A</strong> Tumor spread to the nearby structures (lung, mediastinum, malignant pleural effusion, contralateral lymph nodes, scalene or supraclavicular lymph nodes)</td>
</tr>
<tr>
<td><strong>B</strong> Distant metastasis</td>
</tr>
</tbody>
</table>

**TABLE 28-16**

<table>
<thead>
<tr>
<th>Stages</th>
<th>Characteristics</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>I</strong></td>
<td>Tumor is small and localized to lung. No lymph node involvement.</td>
</tr>
<tr>
<td><strong>A</strong></td>
<td>Tumor &lt;3 cm</td>
</tr>
<tr>
<td><strong>B</strong></td>
<td>Tumor &gt;3 cm and invading surrounding local areas</td>
</tr>
<tr>
<td><strong>II</strong></td>
<td>Tumor &lt;3 cm with invasion of lymph nodes on same side of chest</td>
</tr>
<tr>
<td><strong>A</strong></td>
<td>Tumor &gt;3 cm involving the bronchus and lymph nodes on same side of chest and tissue of other local organs</td>
</tr>
<tr>
<td><strong>B</strong></td>
<td>Extensive tumor involving heart, trachea, esophagus, mediastinum, malignant pleural effusion, contralateral lymph nodes, scalene or supraclavicular lymph nodes</td>
</tr>
<tr>
<td><strong>III</strong></td>
<td>Tumor spread to the nearby structures (chest wall, pleura, pericardium) and regional lymph nodes</td>
</tr>
<tr>
<td><strong>A</strong></td>
<td>Extensive tumor involving heart, trachea, esophagus, mediastinum, malignant pleural effusion, contralateral lymph nodes, scalene or supraclavicular lymph nodes</td>
</tr>
<tr>
<td><strong>B</strong></td>
<td>Distant metastasis</td>
</tr>
</tbody>
</table>

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**Collaborative Care**

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**Chemotherapy.** Chemotherapy may be used in the treatment of nonresectable tumors or as adjuvant therapy to surgery in NSCLC. A variety of chemotherapy drugs and multidrug regimens (i.e., protocols), including combination chemotherapy, have been used. These
drugs include etoposide (VePesid), carboplatin (Paraplatin), cisplatin (Platinol), paclitaxel (Taxol), vinorelbine (Navelbine), cyclophosphamide (Cytoxan), ifosfamide (Ifex), docetaxel (Taxotere), gemcitabine (Gemzar), topotecan (Hycaintin), and irinotecan (Camptosar). Chemotherapy has improved survival in patients with advanced NSCLC and is now considered standard treatment. Treatment of limited-stage SCLC includes combination chemotherapy and radiation therapy.

**Biologic and Targeted Therapy.** Targeted therapy uses drugs that specifically block the growth of molecules involved in specific aspects of tumor growth (see Chapter 16). Therefore, there are usually fewer toxicities than with chemotherapy. One type of targeted therapy that is approved for patients with locally advanced and metastatic NSCLC is erlotinib (Tarceva). It inhibits an enzyme, tyrosine kinase, associated with epidermal growth factor receptor. Thus this drug blocks the growth-stimulatory signals in the cancer cells. Erlotinib is used to treat patients whose cancer has progressed despite other treatments. Gefitinib (Iressa), which has a mechanism of action similar to that of erlotinib, is available on a limited basis in the United States to patients who have shown a response to the drug in the past, and to current users who are responding to it now.

**Other Therapies**

**Prophylactic Cranial Radiation.** Intracranial metastasis occurs in up to 39% of patients with SCLC. Most chemotherapy drugs do not adequately penetrate the blood-brain barrier. Prophylactic cranial radiation is effective in preventing metastasis (20%), although it is not known if it increases survival. Toxicities of this therapy may include scalp erythema, fatigue, and alopecia.

**Bronchoscopic Laser Therapy.** Bronchoscopic laser therapy makes it possible to remove obstructing bronchial lesions. The neodymium:yttrium-aluminum-garnet (Nd:YAG) laser is most commonly used for laser resection using either the flexible or the rigid bronchoscope. The thermal energy of the laser is transmitted to the target tissue. It is a safe and effective treatment of endobronchial obstructions from tumors. Relief of the symptoms from airway obstruction as a result of thermal necrosis and shrinkage of the tumor can be dramatic.

**Photodynamic Therapy.** Photodynamic therapy is a safe, bronchoscopic laser therapy for lung cancer. Porfimer (Photofrin) is injected intravenously and selectively concentrates in tumor cells. After a set time period (usually 48 hours), the tumor is exposed to laser light, producing a toxic form of oxygen that destroys tumor cells. Necrotic tissue is removed through a bronchoscope.

**Airway Stenting.** Stents can be used alone or in combination with other techniques for palliation of dyspnea, cough, or respiratory insufficiency. The advantage of an airway stent is that it supports the airway wall against collapse or external compression and can impede extension of tumor into the airway lumen.

**Cryotherapy.** Cryotherapy is a technique in which tissue is destroyed as a result of freezing. Bronchoscopic cryotherapy is used to ablate (destroy) bronchogenic carcinomas, especially polypoid lesions. There is insufficient evidence that supports its success in the treatment of early-stage lung cancer.

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**EVIDENCE-BASED PRACTICE**

**Do Noninvasive Interventions Improve Quality of Life in Patients with Lung Cancer?**

**Clinical Question**

In lung cancer patients (P), does receiving noninvasive interventions (I) compared to receiving no additional treatment (C) improve symptoms, psychologic functioning, and quality of life? (O)

**Best Available Evidence**

Systematic review of randomized control trials (RCTs)

**Critical Appraisal and Synthesis of Evidence**

- Meta-analysis of 9 RCTs ($p = 833$)
- Interventions studied included nursing interventions to manage breathlessness, nursing care programs, nutritional interventions, psychotherapeutic interventions, exercise, and refluxology.

**Conclusions**

- Nursing interventions to manage breathlessness showed improved symptom control, performance status, and emotional functioning.
- Nursing programs were effective in delaying clinical deterioration, dependency, and symptom distress, as well as improving emotional functioning and satisfaction with care.
- Exercise had a beneficial effect on self-empowerment.
- Nutritional interventions only showed positive results in increasing energy.
- Psychologic interventions and refluxology had some short-lasting positive effects on quality of life.

**Implications for Nursing Practice**

- It is important to develop and maintain a supportive and empathetic relationship with the patient with lung cancer.
- Offer patients supportive multidisciplinary interventions that may benefit their emotional, psychologic, and physical well-being.
- These interventions may also help patients with other types of cancers.

**Reference for Evidence**

Sola I, Thompson E, Subirana M, et al: Non-invasive interventions for improving well-being and quality of life in patients with lung cancer, Cochrane Lung Cancer Group, Cochrane Database of Systematic Reviews 4, 2005. $P$, Patient population of interest; $I$, intervention or area of interest; $C$, comparison of interest or comparison group; $O$, outcome(s) of interest (see p. 000).

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**NURSING MANAGEMENT**

**LUNG CANCER**

**Nursing Assessment**

It is important to determine the understanding of the patient and the family concerning the diagnostic tests (those completed as well as those planned), the diagnosis or potential diagnosis, the treatment options, and the prognosis. At the same time, the nurse can assess the level of anxiety experienced by the patient and the support provided and needed by the patient’s significant others. Subjective and objective data that should be obtained from a patient with lung cancer are presented in Table 28-17.

**Nursing Diagnoses**

Nursing diagnoses for the patient with lung cancer may include, but are not limited to, the following:

- Ineffective airway clearance related to increased tracheobronchial secretions and presence of tumor
- Anxiety related to lack of knowledge of diagnosis or unknown prognosis and treatments
- Acute pain related to pressure of tumor on surrounding structures and erosion of tissues
- Imbalanced nutrition: less than body requirements related to increased metabolic demands, increased secretions, weakness, and anorexia

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**NURSING MANAGEMENT**

**LUNG CANCER**

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- Acute pain related to pressure of tumor on surrounding structures and erosion of tissues
- Imbalanced nutrition: less than body requirements related to increased metabolic demands, increased secretions, weakness, and anorexia
ADH, Antidiuretic hormone; COPD, chronic obstructive pulmonary disease; CT, computed tomography; MRI, magnetic resonance imaging; PET, positron emission tomography; TB, tuberculosis.

- Ineffective health maintenance related to lack of knowledge about the disease process and therapeutic regimen
- Ineffective breathing pattern related to decreased lung capacity

Planning

The overall goals are that the patient with lung cancer will have (1) effective breathing patterns, (2) adequate airway clearance, (3) adequate oxygenation of tissues, (4) minimal to no pain, and (5) a realistic attitude toward treatment and prognosis.

Nursing Implementation

Health Promotion. The best way to halt the epidemic of lung cancer is for people to stop smoking. Important nursing activities to assist in the progress toward this goal include promoting smoking cessation programs and actively supporting education and policy changes related to smoking. Important changes have occurred as the result of the recognition that passive smoke is a health hazard; laws require designation of nonsmoking areas or prohibiting smoking in most public places and a ban on smoking on airline flights. Other actions aimed at controlling tobacco use include restrictions on tobacco advertising on television and warning label requirements for cigarette packaging. For the individual who does have a smoking habit, efforts should be made to assist the smoker to stop smoking. The updated evidence-based guideline, *Treating Tobacco Use and Dependence: Clinical Practice Guideline*, describes a framework (the five As) for approaching patients who are willing to attempt to quit smoking. The five As stand for the five strategies: ask, advise, assess, assist, and arrange (see Chapter 12, Table 12-5). The four stages of change identified in smokers attempting to quit include precontemplation (“I want”), contemplation (“I might”), preparation (“I will”), and action (“I am”). (The stages of change in relationship to patient teaching are discussed in Chapter 5 and Table 5-3.) Each stage requires specific actions to progress to the next stage. Nurses working with patients at their individual stage of change will help them progress to the next stage. For patients unwilling to quit, motivational interviewing is recommended (discussed in Chapter 12 on p. 000).

The evidence-based guideline also offers the five Rs strategy for motivating smokers to quit: relevance, risk, reward, roadblocks, and repetition (see Table 12-5). Because some patients relapse months or years after having stopped smoking, nurses need to continually provide assistance to prevent relapse. (Agents and strategies to assist patients to stop smoking are discussed in Chapter 12 and Tables 12-4, 12-5, and 12-6.)

In addition, the 2004 Surgeon General’s Report on Smoking and Health (www.cdc.gov/tobacco/sgr) offers tips to quit, strategies to use, and an online interactive visual animation of the effects of smoking and the benefits in quitting (“The Health Consequences of Smoking on The Human Body”). Nicotine’s addictive properties make quitting a difficult task that requires much support. Nicotine replacement significantly lessens the urge to smoke and increases the percentage of smokers who successfully quit smoking. All patients should be offered some form of nicotine replacement. (Nicotine replacement therapy products are presented in Table 12-4.) Research into smoking behaviors and successful strategies to promote smoking cessation is ongoing. A combination of behavioral techniques and nicotine replacement products is the most effective strategy to help smokers quit. The advice and motivation of health care professionals can be a powerful force in smoking cessation. Nurses are in a unique position to promote smoking cessation because they see large numbers of smokers who may be reluctant to seek help. Support for the smoker includes education that smoking a few cigarettes during a cessation attempt (a slip) is much different than resuming the full smoking habit (a relapse). Despite the slip, smokers should be encouraged to continue the attempt at cessation without viewing the effort as a failure. The nurse needs to be aware of resources in the community to assist the individual who is interested in quitting.

Acute Intervention. Care of the patient with lung cancer will initially involve support and reassurance during the diagnostic evaluation. (Specific nursing measures related to the diagnostic studies are outlined in Chapter 26.)

Another major responsibility of the nurse is to help the patient and family cope with the diagnosis of lung cancer. The patient may feel guilty about cigarette smoking having caused the cancer and need to discuss this feeling with someone who has a nonjudgmental attitude. Counseling from a social worker, psychologist, or member of the clergy may be needed. Nursing research focusing
on the effects of spirituality on the sense of well-being of people with lung cancer found that people with more meaning in their lives had decreased symptom distress. Additionally, prayer was associated positively with psychologic well-being. This validates the impact of spiritual care for these patients and helps guide nurses’ practice in spiritual needs. Research on the role of the family found that family disagreements about treatment decisions for patients with advanced lung cancer were common. For nurses, these findings suggest the need to be aware of differences of opinion in order to facilitate family communication and improve patient satisfaction with treatment decisions.

Specific care of the patient will depend on the treatment plan. Providing symptom management is critical. The nurse needs to monitor for signs and symptoms of progressive or recurrent disease and notify the physician if this is suspected. Postoperative care for the patient having surgery is discussed later in this chapter. Care of the patient undergoing radiation therapy and chemotherapy is discussed in Chapter 16. The nurse has a major role in providing patient comfort, teaching methods to reduce pain, monitoring for side effects of prescribed medications, fostering appropriate coping strategies for the patient and family, assessing smoking cessation readiness, and helping access resources to deal with the illness. The nurse coordinates care with members of the interdisciplinary team and keeps the patient and family informed.

Ambulatory and Home Care. The patient who has had a surgical resection with intent to cure should be followed up carefully for manifestations of metastasis. The patient and family should be told to contact the physician if symptoms such as hemoptysis, dysphagia, chest pain, and hoarseness develop.

For many individuals who have lung cancer, little can be done to significantly prolong their lives. Radiation therapy and chemotherapy can be used to provide palliative relief from distressing symptoms. Constant pain becomes a major problem. (Measures used to relieve pain are discussed in Chapter 10. Care of the patient with cancer is discussed in Chapter 16.)

Evaluation

The expected outcomes are that the patient with lung cancer will have

- adequate breathing patterns
- minimal to no pain
- realistic attitude about prognosis

OTHER TYPES OF LUNG TUMORS

Other types of primary lung tumors include sarcomas, lymphomas, and bronchial adenomas. Bronchial adenomas are small tumors that arise from the lower trachea or major bronchi and are considered malignant because they are locally invasive and frequently metastasize. Clinical manifestations of bronchial adenomas include hemoptysis, persistent cough, localized obstructive wheezing, and pneumonia. Bronchial adenomas can usually be treated successfully with surgical resection.

The lungs are a common site for secondary metastases and are more often affected by metastatic growth than by primary lung tumors. The pulmonary capillaries, with their extensive network, are ideal sites for tumor emboli. In addition, the lungs have an extensive lymphatic network. The primary malignancies that spread to the lungs often originate in the GI or genitourinary (GU) tracts and in the breast. General symptoms of lung metastases are chest pain and nonproductive cough.

Benign tumors of the lung are rare. Hamartomas of the lung are the most common benign tumor. These tumors, composed of fibrous tissue, fat, and blood vessels, are congenital malformations of the connective tissue of the bronchial walls. Hamartomas are slow-growing tumors. Chondromas are rare benign tumors that arise in the bronchial cartilage; leiomyomas are myomas of smooth, nonstriated muscle. Mesotheliomas may be malignant or benign and originate from the visceral pleura. Benign mesotheliomas are localized lesions.

Chest Trauma and Thoracic Injuries

Thoracic injuries are the cause of death in 20% to 25% of all trauma victims; injury to the chest wall is found in 45% of these thoracic trauma victims. Traumatic injuries fall into two major categories: (1) blunt trauma and (2) penetrating trauma. Blunt trauma occurs when the body is struck by a blunt object, such as a steering wheel. The types of forces involved in blunt chest trauma injuries include deceleration, acceleration, shearing, and compression. The external injury may appear minor, but the impact may cause severe, life-threatening internal injuries, such as a ruptured spleen. Contrecoup trauma, a type of blunt trauma, is caused by the impact of parts of the body against other objects. This type of injury differs from blunt trauma primarily in the velocity of the impact. Internal organs are rapidly forced back and forth (acceleration-deceleration injury) within the bony structures that surround them so that internal injury is sustained not only on the side of the body impacted but also on the opposite side, where the organ or organs hit bony structures. If the velocity of impact is great enough, organs and blood vessels can literally be torn from their points of origin. This is the shearing injury that can cause transection of the aorta, hemothorax, and diaphragmatic rupture injuries. Compression injury occurs when the body cannot handle the degree of external pressure during blunt trauma, resulting in contusions, crush injuries, and organ rupture.

Penetrating trauma occurs when a foreign body impales or passes through the body tissues (e.g., gunshot wounds, stabbings). Table 28-18 describes selective traumatic injuries as they

<table>
<thead>
<tr>
<th>Mechanism of Injury</th>
<th>Common Related Injury</th>
</tr>
</thead>
<tbody>
<tr>
<td>Blunt Trauma</td>
<td>Rib fractures, flail chest, pneumothorax, hemopneumothorax, myocardial contusion, pulmonary contusion, cardiac tamponade, great vessel tears</td>
</tr>
<tr>
<td>Shoulder-harness seat belt injury</td>
<td>Fractured clavicle, dislocated shoulder, rib fractures, pulmonary contusion, pericardial contusion, cardiac tamponade</td>
</tr>
<tr>
<td>Crush injury (e.g., heavy equipment, crushing thorax)</td>
<td>Pneumothorax and hemopneumothorax, flail chest, great vessel tears and rupture, decreased blood return to heart with decreased cardiac output</td>
</tr>
<tr>
<td>Penetrating Trauma</td>
<td>Open pneumothorax, tension pneumothorax, hemopneumothorax, cardiac tamponade, esophageal damage, tracheal tear, great vessel tears</td>
</tr>
</tbody>
</table>
Thoracic injuries range from simple rib fractures to life-threatening tears of the aorta, vena cava, and other major vessels. The most common thoracic emergencies and their management are described in Table 28-20.

### PNEUMOTHORAX

A pneumothorax is air in the pleural space. As a result of the air in the pleural space, there is partial or complete collapse of the lung. This condition should be suspected after any blunt trauma to the chest wall. Pneumothorax may be closed or open. Pneumothorax associated with trauma may be accompanied by hemothorax, a condition called hemopneumothorax.

<table>
<thead>
<tr>
<th>Injury</th>
<th>Definition</th>
<th>Clinical Manifestations</th>
<th>Emergency Management</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pneumothorax</td>
<td>Air in pleural space (see Fig. 28-4).</td>
<td>Dyspnea, decreased movement of involved chest wall, diminished or absent breath sounds on the affected side, hyperresonance to percussion</td>
<td>Chest tube insertion with chest drainage system</td>
</tr>
<tr>
<td>Hemothorax</td>
<td>Blood in the pleural space, usually occurs in conjunction with pneumothorax.</td>
<td>Dyspnea, diminished or absent breath sounds, dullness to percussion, shock</td>
<td>Chest tube insertion with chest drainage system; autotransfusion of collected blood, treatment of hypovolemia as necessary</td>
</tr>
<tr>
<td>Tension pneumothorax</td>
<td>Air in pleural space that does not escape. Continued increase in amount of air shifts intrathoracic organs and increases intrathoracic pressure (see Fig. 28-5).</td>
<td>Cyanosis, air hunger, violent agitation, tracheal deviation away from affected side, subcutaneous emphysema, neck vein distention, hyperresonance to percussion</td>
<td>Medical emergency: needle decompression followed by chest tube insertion with chest drainage system</td>
</tr>
<tr>
<td>Flail chest</td>
<td>Fracture of two or more adjacent ribs in two or more places with loss of chest wall stability (see Fig. 28-6).</td>
<td>Paradoxic movement of chest wall, respiratory distress, associated hemothorax, pneumothorax, pulmonary contusion</td>
<td>Stabilize flail segment with intubation in some patients; taping in others; oxygen therapy; treat associated injuries; analgesia</td>
</tr>
<tr>
<td>Cardiac tamponade</td>
<td>Blood rapidly collects in pericardial sac, compresses myocardium because the pericardium does not stretch, and prevents heart from pumping effectively.</td>
<td>Muffled, distant heart sounds, hypotension, neck vein distention, increased central venous pressure</td>
<td>Medical emergency: pericardiocentesis with surgical repair as appropriate</td>
</tr>
</tbody>
</table>

**TABLE 28-19**

**EMERGENCY MANAGEMENT**

**Chest Trauma**

<table>
<thead>
<tr>
<th>Etiology</th>
<th>Assessment Findings</th>
<th>Interventions</th>
</tr>
</thead>
<tbody>
<tr>
<td>Blunt</td>
<td>✷ Dyspnea, respiratory distress</td>
<td>✷ Initial</td>
</tr>
<tr>
<td>Motor vehicle accident</td>
<td>✷ Cough with or without hemoptysis</td>
<td>✷ • Ensure patent airway.</td>
</tr>
<tr>
<td>Pedestrian accident</td>
<td>✷ Cyanosis of mouth, face, nail beds, mucous membranes</td>
<td>✷ • Administer high-flow O2 with non-rebreather mask.</td>
</tr>
<tr>
<td>Fall</td>
<td>✷ Tracheal deviation</td>
<td>✷ • Establish IV access with two large-bore catheters. Begin fluid resuscitation as appropriate.</td>
</tr>
<tr>
<td>Assault with blunt object</td>
<td>✷ Audible air escaping from chest wound</td>
<td>✷ • Remove clothing to assess injury.</td>
</tr>
<tr>
<td>Crush injury</td>
<td>✷ Decreased breath sounds on side of injury</td>
<td>✷ • Cover sucking chest wound with nonporous dressing taped on three sides.</td>
</tr>
<tr>
<td>Explosion</td>
<td>✷ Decreased O2 saturation</td>
<td>✷ • Stabilize impaled objects with bulky dressings. Do not remove.</td>
</tr>
<tr>
<td>Penetrating</td>
<td>✷ Frotty secretions</td>
<td>✷ • Assess for other significant injuries and treat appropriately.</td>
</tr>
<tr>
<td>Knife</td>
<td>✷ Cardiovascular</td>
<td>✷ • Stabilize flail rib segment with hand followed by application of large pieces of tape horizontal across the flail segment.</td>
</tr>
<tr>
<td>Gunshot</td>
<td>✷ Rapid, thready pulse</td>
<td>✷ • Place patient in a semi-Fowler position or position patient on the injured side if breathing is easier after cervical spine injury has been ruled out.</td>
</tr>
<tr>
<td>Stick</td>
<td>✷ Narrowed pulse pressure</td>
<td>✷ • Ongoing Monitoring</td>
</tr>
<tr>
<td>Arrow</td>
<td>✷ Asymmetric BP values in arms</td>
<td>✷ Monitor vital signs, level of consciousness, oxygen saturation, cardiac rhythm, respiratory status, and urinary output.</td>
</tr>
<tr>
<td>Other missiles</td>
<td>✷ Distended neck veins</td>
<td>✷ • Anticipate intubation for respiratory distress.</td>
</tr>
<tr>
<td></td>
<td>✷ Muffled heart sounds</td>
<td>✷ • Release dressing if tension pneumothorax develops after sucking chest wound is covered.</td>
</tr>
<tr>
<td></td>
<td>✷ Chest pain</td>
<td></td>
</tr>
<tr>
<td></td>
<td>✷ Crunching sound synchronous with heart sounds</td>
<td></td>
</tr>
<tr>
<td></td>
<td>✷ Dysrhythmias</td>
<td></td>
</tr>
<tr>
<td></td>
<td>✷ Surface Findings</td>
<td></td>
</tr>
<tr>
<td></td>
<td>✷ Bruising</td>
<td></td>
</tr>
<tr>
<td></td>
<td>✷ Abrasions</td>
<td></td>
</tr>
<tr>
<td></td>
<td>✷ Open chest wound</td>
<td></td>
</tr>
<tr>
<td></td>
<td>✷ Asymmetric chest movement</td>
<td></td>
</tr>
<tr>
<td></td>
<td>✷ Subcutaneous emphysema</td>
<td></td>
</tr>
</tbody>
</table>

**TABLE 28-20**

**EMERGENCY MANAGEMENT**

**Thoracic Injuries**

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<th>Emergency Management</th>
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<td>Dyspnea, decreased movement of involved chest wall, diminished or absent breath sounds on the affected side, hyperresonance to percussion</td>
<td>Chest tube insertion with chest drainage system</td>
</tr>
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<td>Medical emergency: pericardiocentesis with surgical repair as appropriate</td>
</tr>
</tbody>
</table>
Types of Pneumothorax

Closed Pneumothorax. Closed pneumothorax has no associated external wound. The most common form is a spontaneous pneumothorax, which is accumulation of air in the pleural space without an apparent antecedent event. It is caused by rupture of small blebs on the visceral pleural space. The cause of the blebs is unknown. This condition occurs most commonly in underweight male cigarette smokers between 20 and 40 years of age. There is a tendency for this condition to recur.

Other causes of closed pneumothorax include the following:
1. Injury to the lungs from mechanical ventilation
2. Injury to the lungs from insertion of a subclavian catheter
3. Perforation of the esophagus
4. Injury to the lungs from broken ribs
5. Ruptured blebs or bullae in a patient with COPD

Open Pneumothorax. Open pneumothorax occurs when air enters the pleural space through an opening in the chest wall (Fig. 28–4, B). Examples include stab or gunshot wounds and surgical thoracotomy. A penetrating chest wound is often referred to as a sucking chest wound.

An open pneumothorax should be covered with a vented dressing. (A vented dressing is one secured on three sides with the fourth side left untaped.) This allows air to escape from the vent and decreases the likelihood of tension pneumothorax developing. If the object that caused the open chest wound is still in place, it should not be removed until a physician is present. The impaled object should be stabilized with a bulky dressing.

Tension Pneumothorax. Tension pneumothorax is a pneumothorax with rapid accumulation of air in the pleural space, causing severely high intrapleural pressures with resultant tension on the heart and great vessels. It may result from either an open or a closed pneumothorax (Fig. 28–5). In an open chest wound, a flap may act as a one-way valve; thus air can enter on inspiration but cannot escape. The intrathoracic pressure increases, the lung collapses, and the mediastinum shifts toward the unaffected side, which is subsequently compressed. As the pressure increases, cardiac output is altered because of decreased venous return and compression of the vena cava and aorta. Tension pneumothorax can occur with mechanical ventilation and resuscitative efforts. It can also occur if chest tubes are clamped or become blocked in a patient with a pneumothorax. Unclamping the tube or relief of the obstruction will remedy this situation.

Tension pneumothorax is a medical emergency, with both the respiratory and circulatory systems affected. If the tension in the pleural space is not relieved, the patient is likely to die from inadequate cardiac output or severe hypoxemia. The emergency management is to insert a large-bore needle into the chest wall to release the trapped air.

Hemothorax. Hemothorax is an accumulation of blood in the intrapleural space. It is frequently found in association with open pneumothorax and is then called a hemopneumothorax. Causes of hemothorax include chest trauma, lung malignancy, complications of anticoagulant therapy, pulmonary embolus, and tearing of pleural adhesions.

Chylothorax. Chylothorax is lymphatic fluid in the pleural space due to a leak in the thoracic duct. Causes include trauma, surgical procedures, and malignancy. The thoracic duct is disrupted and the chylous fluid, milky white with high lipid content, fills the pleural space. Total lymphatic flow through the thoracic duct is 1500 to 2400 ml/day. Fifty percent of cases will heal with conservative treatment (chest drainage, bowel rest, and parenteral nutrition). Surgery and pleurodesis are options if conservative therapy fails. Pleurodesis is the artificial production of adhesions between the parietal and visceral pleura, usually done with a chemical sclerosing agent.

Clinical Manifestations

If the pneumothorax (hemothorax or chylothorax) is small, mild tachycardia and dyspnea may be the only manifestations. If the pneumothorax (hemothorax or chylothorax) is large, respiratory distress may be present, including shallow, rapid respirations; dyspnea; air hunger; and oxygen desaturation. Chest pain and a cough with or without hemoptysis may be present. On auscultation, there

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FIG. 28–5 Tension pneumothorax. As pleural pressure on the affected side increases, mediastinal displacement ensues with resultant respiratory and cardiovascular compromise.
are no breath sounds over the affected area. A chest x-ray shows the presence of air or fluid in the pleural space.

If a tension pneumothorax develops, severe respiratory distress, tachycardia, and hypotension occur. Mediastinal displacement can occur with a tracheal shift to the unaffected side. The patient is hemodynamically unstable in this medical emergency.

**Collaborative Care**

Treatment depends on the severity of the pneumothorax (hemothorax or chylothorax) and the nature of the underlying disease. If the patient is stable, and the amount of air and fluid accumulated in the intrapleural space is minimal, no treatment may be needed as the pneumothorax will resolve spontaneously. If the amount of air or fluid is minimal, the pleural space also can be aspirated with a large-bore needle. As a lifesaving measure, needle venting (using a large-bore needle) of the pleural space may be used. A Heimlich valve may also be used to evacuate air from the pleural space. The Heimlich valve is a portable, lightweight, one-way flutter valve device similar to a water-seal drain. The most definitive and common form of treatment of pneumothorax and hemothorax is to insert a chest tube and connect it to water-seal drainage. Repeated spontaneous pneumothorax may need to be treated surgically by a partial pleurectomy, stapling, or pleurodesis to promote adherence of the pleurae to one another.

**FRACUTRED RIBS**

Rib fractures are the most common type of chest injury resulting from trauma. Ribs 5 through 10 are most commonly fractured because they are least protected by chest muscles. If the fractured rib is splintered or displaced, it may damage the pleura and lungs.

Clinical manifestations of fractured ribs include pain (especially on inspiration) at the site of injury. The patient splints the affected area and takes shallow breaths to try to decrease the pain. Because the patient is reluctant to take deep breaths, atelectasis may develop because of decreased ventilation.

The main goal in treatment is to decrease pain so that the patient can breathe adequately to promote good chest expansion. Strapping the chest with tape or using a binder is not generally done. Most physicians believe that these measures should be avoided because they reduce lung expansion and predispose the individual to atelectasis. Narcotic drug therapy must be individualized and used with caution because these drugs can depress respirations.

**FLAIL CHEST**

Flail chest results from multiple rib fractures, causing an unstable chest wall (Fig. 28-6). The diagnosis of flail chest is made on the basis of fracture of two or more ribs, in two or more separate locations, causing an unstable segment. The flail segment usually involves anterior (sternal separation) or lateral rib fractures. The chest wall cannot provide the bony structure necessary to maintain bellows action and ventilation. The affected (flail) area will move paradoxically with respect to the intact portion of the chest during respiration. During inspiration the affected portion is sucked in, and during expiration it bulges out. This paradoxical chest movement prevents adequate ventilation of the lung in the injured area and increases the work of breathing. The underlying lung may have a pulmonary contusion aggravating hypoxemia. The associated pain, fractures, and lung injury give rise to an alteration in breathing pattern and hypoxemia.

A flail chest is usually apparent on visual examination of the unconscious patient. The patient manifests rapid, shallow respirations and tachycardia. A flail chest may not be initially apparent in the conscious patient as a result of splinting of the chest wall. The patient moves air poorly, and movement of the thorax is asymmetric and uncoordinated. Palpation of abnormal respiratory movements, evaluation for crepitus near the rib fractures, chest x-ray, and ABGs all assist in the diagnosis.

Initial therapy consists of airway management, adequate ventilation, supplemental oxygen therapy, careful administration of IV solutions, and pain control. The definitive therapy is to reexpand the lung and ensure adequate oxygenation. Although many patients can be managed without the use of mechanical ventilation, a short period of intubation and ventilation may be necessary until the diagnosis of the lung injury is complete. The lung parenchyma and fractured ribs will heal with time. Some patients continue to experience intercostal pain after the flail chest has resolved.

**CHEST TUBES AND PLEURAL DRAINAGE**

The purpose of chest tubes and pleural drainage is to remove air and fluid from the pleural space and to restore normal intrapleural pressure so that the lungs can reexpand. Small accumulations of air or fluid in the pleural space may not require removal by thoracentesis or chest tube insertion. Instead, the air and fluid may be reabsorbed over time.

Under normal conditions, intrapleural pressure is below atmospheric pressure (approximately 4 to 5 cm H₂O below atmospheric pressure during expiration and approximately 8 to 10 cm H₂O below atmospheric pressure during inspiration). (Intrapleural pres-
since the anterior approach requires dissection of the pectoral mus-

cles. In this instance, the tube is directed apically for air evacuation and inferiorly and posteriorly for fluid removal.45

The tubes are sutured to the chest wall, and the puncture wound is covered with a dressing. Some clinicians prefer to use an airtight seal with petroleum gauze. However, continued use of petroleum gauze or ointment can irritate the skin.49 During insertion, the tubes are kept clamped. After the tubes are in place in the pleural space, they are connected to drainage tubing and pleural drainage and the clamp is removed. Each tube may be connected to a separate drainage system and suction. More commonly, a Y-connector is used to attach both chest tubes to the same drainage system.

**Pleural Drainage**

Most pleural drainage systems have three basic compartments, each with its own separate function (Fig. 28-8). The *first compartment*, or collection chamber, receives fluid and air from the chest cavity. The fluid drains through the 6-foot connecting tube into this collection chamber. The chamber holds up to 2000 ml. The fluid stays in this chamber while the air vents to the second compartment. The *second compartment*, called the water-seal chamber, contains 2 cm of water, which acts as a one-way valve. The incoming air enters the water seal and enters the suction control chamber. The chamber holds up to 2000 ml. The fluid acts as a one-way valve to prevent backflow of air into the patient from the system.) Initial bubbling of air is seen in this chamber while a pneumothorax is evacuated. Intermittent bubbling can also be seen during exhalation, coughing, or sneezing due to an increase in the patient’s intrathoracic pressure. In this chamber fluctuations, or “tidaling,” will be seen that reflect the pressures in the pleural space. If tidalizing is not seen, either the lungs have reexpanded or there is a kink or obstruction in the tubing. The air exits the water seal and enters the suction control chamber.

The *third compartment*, the suction control chamber, applies controlled suction to the chest drainage system. The classic suction control chamber uses a column of water with the top end vented to the atmosphere to control the amount of suction from the wall regulator. The chamber is typically filled with 20 cm of water. When

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**Figure 28-7** Placement of chest tubes.

**Figure 28-8** Chest drainage unit. Both units have three chambers: (1) collection chamber; (2) water-seal chamber; and (3) suction control chamber. Suction control chamber requires a connection to a wall suction source that is dialed up higher than the prescribed suction for the suction to work. A, Water suction. This unit uses water in the suction control chamber to control the wall suction pressure. B, Dry suction. This unit controls wall suction by using a regulator control dial.
the negative pressure generated by the suction source exceeds the set 20 cm, air from the atmosphere enters the chamber through the vent on top and the air bubbles up through the water, causing a suction-breaker effect. As a result, excess pressure is relieved. The amount of suction applied is regulated by the amount of water in this chamber and not by the amount of suction applied to the system. An increase in suction does not result in an increase in negative pressure to the system because any excess suction merely draws in air through the vent on the top of the third chamber. The suction pressure is usually ordered to be ~20 cm H₂O, although higher pressures (~40 cm H₂O) are sometimes necessary to evacuate the pleural space.

Two types of suction control chambers are available on the market: water and dry (see Fig. 28-8). The water suction control chamber system is the classic system outlined previously. Bubbling in this third compartment indicates if the suction is functioning. To start the suction, the vacuum source is turned up until gentle bubbling appears. Turning the vacuum source higher just makes the bubbling more vigorous and makes the water evaporate faster. Even at gentle bubbling, water evaporates in this chamber, and water must be added periodically. The dry suction control chamber system contains no water. It has a visual alert that indicates if the suction is working, so bubbling is not seen in a third chamber. It uses either a restrictive device or a regulator to dial the desired negative pressure; this is internal in the chest drainage system. To increase the suction pressures, the dial is turned on the drainage system. Increasing the vacuum suction source will not increase the pressure.

A variety of commercial disposable plastic chest drainage systems are available. One popular system is the Atrium Chest Drainage Unit shown in Fig. 28-8. The manufacturer’s suggestions for use are included with the equipment. Another chest drainage unit, a portable unit, can be used to allow the patient more mobility. This unit (Fig. 28-9) allows for a maximum of 500 ml drainage and offers a dry seal system to prevent air leaks, and can be used for long-term drainage. If a patient is discharged home with this device, patient and family teaching includes how to use the system and empty the drainage chamber. The Atrium Medical Company offers online educational videos of these products (www.atriummed.com/Products/Chest_Drains/education.asp).

**Heimlich Valves.** Another device that may be used to evacuate air from the pleural space is the Heimlich valve. This device consists of a rubber flutter one-way valve within a rigid plastic tube. It is attached to the external end of the chest tube. The valve opens whenever the pressure is greater than atmospheric pressure and closes when the reverse occurs. The Heimlich valve is a flutter valve, and functions like a water seal. The device can be used for emergency transport, in an emergency pneumothorax kit, when placing small-bore chest drains (pigtail catheters), and for home care or long-term care nursing units.

**Small Chest Tubes.** Small-size (<14 F) chest tubes (“pigtail catheters”) are used in selected patients because they are less traumatic. They drain air and fluid equally well as large-bore chest tubes. The drains may be straight catheters or “pigtail” catheters (curled at the distal end to look like a pig’s tail). Curled catheters are considered to be less traumatic than straight catheters. These catheters, if occluded, can be irrigated using sterile water. Irrigation of these tubes is generally performed by physicians. Chemical pleurodesis (adding an abrasive product into the pleural space to obliterate the pleural space) can be performed using this small-size catheter. This system is not suitable for trauma or for drainage of blood. It is used for a pneumothorax and for pleural effusions.

Chest tubes placed in a pleural space should not be left open to air since a pneumothorax can occur due to the loss of negative pleural pressure. It is possible that this smaller size tube can kink or become occluded by fluid, or the tip can become lodged along the pleural wall, with the potential for a pneumothorax or obstruction of a draining effusion. The nurse needs to assess the functioning of this product. Both the small-bore chest tube and the Heimlich valve should be used with caution in patients on mechanical ventilators since there is the potential for rapid accumulation of air and a tension pneumothorax.

**NURSING MANAGEMENT**

**CHEST DRAINAGE**

Some general guidelines for nursing care of the patient with chest tubes and water-seal drainage systems are presented in Table 28-21. The traditional practice of routine milking and/or stripping of chest tubes to maintain patency is no longer recommended since it can cause dangerously high intrapleural pressure and damage to pleural tissue. Drainage and blood are not likely to clot inside chest tubes because the newer chest tubes are made with a coating that makes them nonthrombogenic. The nurse should remember that insertion of a chest tube, as well as its continued presence, can be painful for the patient. Dislodgment of the tube may occur if the tube is not stabilized.

Clamping of chest tubes during transport or when the tube is accidentally disconnected is no longer advocated. The danger of rapid accumulation of air in the pleural space, causing tension pneumothorax, is far greater than that of a small amount of atmospheric air entering the pleural space. Chest tubes may be momentarily clamped to change the drainage apparatus or to check for air leaks. Clamping for more than a few moments is indicated only in assessing how the patient will tolerate chest tube removal. The physician may want to do this to simulate chest tube removal and identify if there will be clinical problems with tube removal. Generally this is done 4 to 6 hours before the tube is removed, and the patient is monitored closely. If a chest tube becomes disconnected, the most important intervention is reestablishment of the water-seal system immediately and attachment of a new drainage system as soon as possible. In some hospitals, when disconnection occurs, the chest tube is immersed in sterile water (about 2 cm) until the system can be reestablished. It is important for the nurse to know
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6. If there is no bubbling seen in the suction control chamber, this indicates (1) no suction/suction loss, (2) suction not high enough, or (3) pleural air leak.

5. This suction control chamber should have constant bubbling. This indicates the suction is functioning.

4. Dial the wall suction regulator higher than the ordered suction amount until bubbling is seen in the suction control chamber (generally 80-120 mm Hg).

3. Observe for air bubbling in the water-seal chamber and fluctuations (tidaling).
   - If no tidalizing is observed (rising with inspiration and falling with expiration in the spontaneously breathing patient), the drainage system is blocked, the lungs are reexpanded, or the system is attached to suction.
   - If bubbling increases, there may be an air leak in the drainage system or a leak from the patient (bronchopleural leak).

2. Keep all connections between chest tubes, drainage tubing, and the drainage collector tight and tape at connections.

1. Keep all tubing as straight as possible and coiled loosely below chest level. Do not let the patient lie on it.

---

**TABLE 28.21 Clinical Guidelines for Care of Patient with Chest Tubes and Water-Seal Drainage**

**Drainage System**

1. Take and document vital signs, auscultate lungs, observe chest wall. Document pain level.

2. Assess every shift for manifestations of reaccumulation of air and fluid in the chest (for absent breath sounds), significant bleeding (>100 ml/hr), chest drainage site infection (drainage, erythema, fever, WBC), or poor wound healing. Notify physician for management plan. Evaluate for subcutaneous emphysema at chest tube site.

3. Encourage the patient to breathe deeply periodically to facilitate lung expansion and encourage range-of-motion exercises to the shoulder on the affected side. Incentive spirometry every hour while awake may be necessary to prevent atelectasis or pneumonia.

**Patient’s Clinical Status**

1. Monitor the patient’s clinical status.

2. Mark the time of measurement and the fluid level on the drainage bottle according to the prescribed orders. Marking intervals may range from once per hour to every 8 h. Any change in the quantity or characteristics of drainage (e.g., clear yellow to bloody) should be reported to the physician and recorded. Notify physician if >100 ml/hr drainage.

3. Monitor the fluid drainage and evacuate no more than 1000-1200 ml of pleural fluid from the pleural space at one time to prevent rebound hypotension or reexpansion pulmonary edema.

4. Check the position of the chest drainage container. If the drainage system is overturned and the water seal is disrupted, return it to an upright position and encourage the patient to take a few deep breaths, followed by forced exhalations and cough maneuvers.

5. If the drainage system breaks, place the distal end of the chest tubing connection in a sterile water container at a 2-cm level as an emergency water seal.

6. Do not strip or milk chest tubes routinely since this dangerously increases pleural pressures. **Milking:** alternately folding or squeezing and then releasing drainage tubing. Milk only if drainage and evidence of clots/obstruction. Take 15-cm strips of the chest tube and squeeze and release starting close to the chest and repeating down the tube distally.

**Chest Drainage**

1. Never elevate the drainage system to the level of the patient’s chest because this will cause fluid to drain back into the lungs. Secure the unit to the drainage stand. If the drainage chambers are full, change the system. Do not try to empty it.

2. Keep the water-seal chamber at the appropriate water level by adding sterile water as needed due to evaporation of water.

3. High fluid levels in the water seal indicate residual negative pressure.
   - The chest system may need to be vented by using the high-negativity release valve available on the drainage system to release residual pressure from the system.
   - Do not lower water-seal column when wall suction is not operating or when patient is on gravity drainage.

**Monitoring Wet vs. Dry Suction Chest Drainage Systems**

**Suction Control Chamber in Wet Suction System**

1. After connecting patient to system, turn the dial on the chest drainage system to amount ordered (generally 20 cm pressure), connect suction tubing to wall suction source, and increase the suction until all of the orange float valve is seen in the window.

2. The orange float valve is a visual indicator of suction. The orange bellows needs to be extended to the arrow (delta mark) or further. Wall suction must be set from 80 to 120 mm Hg. There is no water, bubbling, or evaporation. This system can be dialed up to 40 cm suction if needed for a patient.

3. If suction is to be decreased, turn the dial down. There will still be high negative pressure in the system, as evidenced by high water-seal manometer pressure, and it needs to be vented by using the high-negativity release valve.
the patient is observed for respiratory distress,} 

**Complications**

Chest tube malposition is the most common complication. Routine monitoring is done by the nurse to evaluate if the chest drainage is successful by observing for tidalizing in the water-seal chamber, listening for breath sounds over the lung fields, and measuring the amount of fluid drainage. Reexpansion pulmonary edema can occur after rapid expansion of a collapsed lung in patients with a pneumothorax or evacuation of large volumes of pleural fluid (>1 to 1.5 L). A vasovagal response with symptomatic hypotension can occur from too rapid removal of fluid.

Infection at the skin site is also possible. Meticulous sterile technique during dressing changes can reduce the incidence of infected sites. Other complications include (1) pneumonia (from not turning, coughing, and taking deep breaths and/or not using incentive spirometers) and (2) shoulder disuse (“frozen shoulder”) from lack of range-of-motion exercises. Poor patient compliance or lack of patient teaching can contribute to these complications. Nurses can make a tremendous impact on preventing these complications.

**Chest Tube Removal**

The patient with a chest tube has frequent chest x-rays to evaluate for reexpansion and for evacuation of drainage. The chest tubes are removed when the lungs are reexpanded and fluid drainage has ceased. Generally suction is discontinued and the patient is placed on gravity drainage 24 hours before the tube is removed. Explain the procedure to the patient and premedicate the patient at least 15 minutes before tube removal. The tube is removed by cutting the sutures; applying sterile petroleum jelly gauze dressing; having the patient take a deep breath, exhale, and bear down (Valsalva maneuver); and then removing the tube. The site is covered with an airtight dressing; the pleura will seal itself off, and the wound is healed in several days. A chest x-ray is done after the chest tube is removed by cutting the procedure to the patient and premedicate the patient at least 15 minutes before tube removal. The tube is removed by cutting the procedure to the patient and premedicate the patient at least 15 minutes before tube removal. The tube is removed by cutting

**TABLE 28-21 Clinical Guidelines for Care of Patient with Chest Tubes and Water-Seal Drainage—cont’d**

<table>
<thead>
<tr>
<th>Chest Tube Dressings</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Change dressing when wet; change routinely Monday, Wednesday, and Friday unless ordered more frequently by physician.</td>
</tr>
<tr>
<td>2. Remove old dressing carefully to avoid removing unsecured chest tube. Evaluate the site and culture site if necessary.</td>
</tr>
<tr>
<td>3. Clean site with sterile normal saline. Apply sterile 4 × 4 gauze and tape to secure the dressing. Vaseline gauze may be used around the tube to prevent air leak. Date the dressing and document dressing change.</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Obtaining a Sample From the Chest Tube</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Form a loop in the tubing in an area to get the most recently drained fluid.</td>
</tr>
<tr>
<td>2. Swab an area of the tubing with alcohol and allow to air dry.</td>
</tr>
<tr>
<td>3. Insert a 20-gauge or smaller syringe needle at an angle and aspirate the sample; do not puncture the other side of the tubing.</td>
</tr>
<tr>
<td>4. Place sample in appropriate, labeled container and send to lab.</td>
</tr>
</tbody>
</table>

**Preoperative Care**

Before chest surgery, baseline data are obtained on the respiratory and cardiovascular systems. Diagnostic studies performed are pulmonary function studies, chest x-rays, electrocardiogram (ECG), ABGs, blood urea nitrogen (BUN), serum creatinine, blood glucose, serum electrolytes, and complete blood count. An anesthesia consult is done preoperatively. Additional studies of cardiac function may be done for the patient who is to undergo a pneumonectomy. A careful physical assessment of the lungs, including percussion and auscultation, should be done. This will allow the nurse to compare preoperative and postoperative findings.

The patient should be encouraged to stop smoking before surgery to decrease secretions and increase O₂ saturation. In the anxious period before surgery, this is not an easy thing for the habitual smoker to do. Preoperative teaching should include exercises for effective deep breathing and incentive spirometry. If the patient practices these techniques before surgery, the techniques will be easier to perform postoperatively. The patient should be told that adequate medication will be given to reduce the pain, and the patient is helped to splint the incision with a pillow to facilitate deep breathing.

For most types of chest surgery, chest tubes are inserted and connected to water-sealed drainage systems. The purpose of these tubes should be explained to the patient. In addition, supplemental oxygen is frequently given the first 24 hours after surgery. Range-of-motion exercises on the surgical side similar to those for the mastectomy patient should be taught (see Chapter 52).

The thought of losing part of a vital organ is frequently frightening. The patient should be reassured that the lungs have a large degree of functional reserve. Even after the removal of one lung, there is enough lung tissue to maintain adequate oxygenation.

The nurse should be available to deal with the questions asked by the patient and the family. Questions should be answered honestly. The nurse should try to facilitate the expression of concerns, feelings, and questions. (General preoperative care and teaching are discussed in Chapter 18.)

**Surgical Therapy**

Thoracotomy (surgical opening into the thoracic cavity) surgery is considered major surgery because the incision is large, cutting into bone, muscle, and cartilage. The two types of thoracic incisions are median sternotomy, performed by splitting the sternum, and lateral thoracotomy. The median sternotomy is primarily used for surgery involving the heart. The two types of lateral thoracotomy are posterolateral and anterolateral. The posterolateral thoracotomy is used for most surgeries involving the lung. The incision
used to gain access to the lung. The anterolateral incision is made necessary to remove the ribs. Strong mechanical retractors are placed posteriorly at the fourth, fifth, or sixth intercostal space. It is rarely necessary to remove the ribs. Strong mechanical retractors are used to gain access to the lung. The anterolateral incision is made in the fourth or fifth intercostal space from the sternal border to the midaxillary line. This procedure is commonly used for surgery or trauma victims, mediastinal operations, and wedge resections of the upper and middle lobes of the lung.

The extensiveness of the thoracotomy incision often results in severe pain for the patient after surgery. Because muscles have been severed, the patient is reluctant to move the shoulder and arm on the surgical side. Chest tubes are placed in the pleural space except in pneumonectomy surgery. In a pneumonectomy, the space from which the lung was removed gradually fills with serosanguineous fluid.

**Video-Assisted Thoracic Surgery (VATS).** VATS is a thorascoscopic surgical procedure that in many cases can avoid the impact of a full thoracotomy. The procedure involves three or four 1-inch incisions made on the chest that allow the thoracoscope (a special fiberoptic camera) and instruments to be inserted and manipulated. Video-assisted thoracoscopy improves visualization because the surgeon can view the thoracic cavity on the video monitor. The thoracoscope is equipped with a camera that magnifies the image on the monitor. Thoracoscopy can be used to diagnose and treat a variety of conditions of the lung, pleura, and mediastinum.

The candidate for this type of procedure should not have a prior history of conventional thoracic surgery because the probability of adhesion formation would make access more difficult. The patient whose lesions are in the lung periphery or the mediastinum is a better candidate because of better accessibility. The patient considered for thorascoscopic surgery should have sufficient pulmonary function preoperatively to allow the surgeon to perform conventional thoracotomy if complications occur. Complications that may occur include bleeding, diaphragmatic perforation, air emboli, persistent pleural air leaks, and tension pneumothorax.

There are many benefits of VATS when compared with a conventional thoracotomy procedure. These include less adhesion formation, minimal blood loss, less time under anesthesia, shorter hospitalization, faster recovery, less pain, and less need for postoperative rehabilitation therapy because of minimal disruption of thoracic structures.

Chest tubes are placed at the end of the procedure through one of the incisions. The incisions are closed with sutures or a wound-approximating adhesive bandage. Nursing assessment and care postoperatively include monitoring respiratory status and lung re-expansion with the chest tubes and checking the incisions for drainage or dehiscence. The most common complication is prolonged air leak. A return to prior activities should be encouraged as quickly as possible. The hospital stay averages from 1 to 5 days, depending on the type of surgery.

**TABLE 28-22 Chest Surgeries**

<table>
<thead>
<tr>
<th>Type and Description</th>
<th>Indication</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lobectomy</td>
<td>Removal of one lobe of lung</td>
<td>Lung cancer, bronchiectasis, TB, emphysematous bullae, benign lung tumors, fungal infections</td>
</tr>
<tr>
<td>Pneumonectomy</td>
<td>Removal of entire lung</td>
<td>Lung cancer (most common)</td>
</tr>
<tr>
<td>Segmental Resection</td>
<td>Removal of one or more lung segments</td>
<td>Lung cancer, bronchiectasis</td>
</tr>
<tr>
<td>Wedge Resection</td>
<td>Removal of small, localized lesion that occupies only part of a segment</td>
<td>Lung biopsy, excision of small nodules</td>
</tr>
<tr>
<td>Decortication</td>
<td>Removal of or stripping of thick, fibrous membrane from visceral pleura</td>
<td>Empyema unresponsive to conservative management</td>
</tr>
<tr>
<td>Exploratory Thoracotomy</td>
<td>Incision into thorax to look for injured or bleeding tissues</td>
<td>Chest trauma</td>
</tr>
<tr>
<td>Thoracotomy Not Involving Lungs</td>
<td>Incision into thorax for surgery on other organs</td>
<td>Hiatal hernia repair, open heart surgery, esophageal surgery, tracheal resection, aortic aneurysm repair</td>
</tr>
<tr>
<td>Video-Assisted Thoracic Surgery (VATS)</td>
<td>VATS under general anesthesia in OR. Procedures performed using VATS include lung biopsy, lobectomy, resection of nodules, repair of fistulas</td>
<td>Video-assisted technique with a rigid scope with a distal lens inserted into the pleura and image shown on a monitor screen; allows surgeon to manipulate instruments passed into the pleural space through separate small intercostal incisions</td>
</tr>
<tr>
<td>Lung Volume Reduction Surgery (LVRS)</td>
<td>Advanced bullous emphysema, a1-antitrypsin emphysema</td>
<td></td>
</tr>
</tbody>
</table>
Restrictive Respiratory Disorders

Restrictive respiratory disorders are characterized by a restriction in lung volume (caused by decreased compliance of the lungs or chest wall) as opposed to obstructive disorders, which are characterized by increased resistance to airflow. Pulmonary function tests are the best means of differentiating between restrictive and obstructive respiratory disorders (Table 28-23). Mixed obstructive and restrictive disorders can be seen together. For example, a patient may have both chronic bronchitis (an obstructive problem) and pulmonary fibrosis (a restrictive problem).

Restrictive problems are generally categorized into extrapulmonary and intrapulmonary disorders. Extrapulmonary causes of restrictive lung disease include disorders involving the central nervous system (CNS) or the neuromuscular system, and chest wall disorders that limit chest wall expansion. (Some of these disorders are discussed in Chapter 20.)
are listed in Table 28-24.) In this group of extrapulmonary causes, the lung tissue is normal. Intrapulmonary causes of restrictive lung disease are intrinsic lung diseases involving the pleura or the lung tissue. This damage can be caused by inflammation and scarring of lung tissue (interstitial lung disease), air spaces (pneumonitis), or pleura (empyema) (Table 28-25).

**PLEURAL EFFUSION**

**Types**
The pleural space lies between the lung and chest wall and normally contains a very thin layer of fluid. **Pleural effusion** is a collection of fluid in the pleural space (see Fig. 28-5, A). It is not a disease but rather a sign of a serious disease. Pleural effusion is frequently classified as transudative or exudative according to whether the protein content of the effusion is low or high, respectively.51 A transudate occurs primarily in noninflammatory conditions and is an accumulation of protein-poor, cell-poor fluid. Transudative pleural effusions (also called hydrothoraces) are caused by (1) increased hydrostatic pressure found in heart failure (HF), which is the most common cause of pleural effusion, or (2) decreased oncotic pressure (from hypoalbuminemia) found in chronic liver or renal disease. In these situations, fluid movement is facilitated out of the capillaries and into the pleural space.

An **exudative effusion** is an accumulation of fluid and cells in an area of inflammation. An exudative pleural effusion results from increased capillary permeability characteristic of the inflammatory reaction. This type of effusion occurs secondary to conditions such as pulmonary malignancies, pulmonary infections, pulmonary embolization, and GI disease (e.g., pancreatic disease, esophageal perforation).

The type of pleural effusion can be determined by a sample of pleural fluid obtained via thoracentesis (a procedure done to remove fluid from the pleural space). Exudates have a high protein content, and the fluid is generally dark yellow or amber.52 Transudates have a low protein content or contain no protein, and the fluid is clear or pale yellow. The fluid can also be analyzed for RBCs and WBCs, malignant cells, bacteria, and glucose.

An **empyema** is a pleural effusion that contains pus. It is caused by conditions such as pneumonia, TB, lung abscess, and infection of surgical wounds of the chest. A complication of empyema is fibrothorax, in which there is fibrous fusion of the visceral and parietal pleurae (see Fig. 28-4, A).

**Clinical Manifestations**
Common clinical manifestations of pleural effusion are progressive dyspnea and decreased movement of the chest wall on the affected side. There may be pleuritic pain from the underlying disease. Physical examination of the chest will indicate dullness to percussion and absent or decreased breath sounds over the affected area. The chest x-ray will indicate an abnormality if the effusion is greater than 250 ml. Manifestations of empyema include the mani-
festations of pleural effusion, as well as fever, night sweats, cough, and weight loss. A thoracentesis reveals an exudate containing thick, purulent material.

**Thoracentesis**

If the cause of the pleural effusion is not known, a diagnostic thoracentesis is needed to obtain pleural fluid for analysis (see Fig. 26-16). If the degree of pleural effusion is severe enough to impair breathing, a therapeutic thoracentesis is done to improve breathing and to remove fluid for analysis.

A thoracentesis is performed by having the patient sit on the edge of a bed and lean forward over a bedside table. The puncture site is determined by chest x-ray, and percussion of the chest is used to assess the maximum degree of dullness. The skin is cleaned with an antiseptic solution and anesthetized locally. The thoracentesis needle is inserted into the intercostal space. Fluid can be aspirated with a syringe, or tubing can be connected to allow fluid to drain into a sterile collecting bottle. After the fluid is removed, the needle is withdrawn, and a bandage is applied over the insertion site.

Usually only 1000 to 1200 ml of pleural fluid are removed at one time. Because high volumes are removed, rapid removal can result in hypotension, hypoxemia, or pulmonary edema. A follow-up chest x-ray should be done to detect a possible pneumothorax that could have been induced by perforation of the visceral pleura. During and after the procedure, vital signs and pulse oximetry are monitored, and the patient should be observed for any manifestations of respiratory distress.
ARDS, the most effective agent for pleurodesis.53 Thoracoscopy can be though doxycycline (Vibramycin) and bleomycin (Blenoxane) tions. The treatment of pleural effusions secondary to malignant uretics and sodium restriction will result in decreased pleural effu- underlycause. For example, adequate treatment of HF with di-
The main goal of management of pleural effusions is to treat the chemical pleurodesis may be used to sclerose the effusions are frequently recurrent and accumulate quickly after thoracentesis. Decisions may be made to rotate the patient’s positions to spread the agent uniformly throughout the pleural space. The decision to rotate the patient from side to side to back depends on physician preference and the patient’s ability to tolerate turning.32 Chest tubes are left in place after pleurodesis until fluid drainage is less than 150 ml/day and no air leaks are noted. A more rapid completion of the pleurodesis procedure, in less than 24 hours, is reported in the literature with good results,54 and this limited admission for symptomatic malignant effusions may become more frequent.

Treatment of empyema is generally with chest tube drainage. Appropriate antibiotic therapy is also needed to eradicate the causative organism. A condition called trapped lung can occur with ef-usions and empyemas. This is a fibrous peel around the pleura that can cause severe pulmonary restriction. A decortication surgical procedure to remove the pleural peel may need to be performed.

PLEURISY

Pleurisy (pleuritis) is an inflammation of the pleura. The most common causes are pneumonia, TB, chest trauma, pulmonary in-
factions, and neoplasms. The inflammation usually subsides with adequate treatment of the primary disease. The pain of pleurisy is typically abrupt and sharp in onset and is aggravated by inspira-
tion. The patient’s breathing is shallow and rapid to avoid unneces-
sary movement of the pleura and chest wall. A pleural friction rub may occur, which is the sound over areas where inflamed visceral pleura and parietal pleura rub over one another during inspiration. This sound is usually loudest at peak inspiration but can be heard during exhalation as well.

Treatment of pleurisy is aimed at treating the underlying dis-
ease and providing pain relief. Taking analgesics and lying on or splinting the affected side may provide some relief. The patient should be taught to splint the rib cage when coughing. Intercostal nerve blocks may be done if the pain is severe.

ATELECTASIS

Atelectasis is a condition of the lungs characterized by collapsed, airless alveoli. The most common cause of atelectasis is airway obstruction that results from retained exudates and secretions. This is frequently observed in the postoperative patient. Normally the pores of Kohn (see Fig. 26-1) provide for collateral passage of air from one alveolus to another. Deep inspiration is necessary to open the pores effectively. For this reason, deep-breathing exercises are important in preventing atelectasis in the high-risk patient (e.g., postoperative, immobilized patient). (The prevention and treat-
ment of atelectasis are discussed in Chapter 19.)

Interstitial Lung Diseases

Many acute and chronic lung disorders with variable degrees of pulmonary inflammation and fibrosis are collectively referred to as interstitial lung diseases (ILD) or diffuse parenchymal lung diseases. ILDs have been difficult to classify because more than 200 known diseases have diffuse lung involvement. The lung in-
volvement may occur either from the primary condition or as a significant part of a multiorgan process, as may occur in connective tissue disorders (e.g., systemic lupus erythematosus, rheu-
matoïd arthritis).

Among the ILDs of known cause, the largest group comprises occupational and environmental exposures, especially the inhalation of dusts and various fumes or gases. The most common ILDs of unknown etiology are idiopathic pulmonary fibrosis and sarcoidosis.

**TABLE 28-25 Intrapulmonary Causes of Restrictive Lung Disease**

<table>
<thead>
<tr>
<th>Disease or Alteration</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Pleural Disorders</strong></td>
<td>Inflammation, scarring, or fluid in the pleural space causing restriction</td>
</tr>
<tr>
<td>• Pleural effusion</td>
<td>Accumulation of fluid in pleural space secondary to altered hydrostatic or oncotic pressure; fluid collection &gt;250 ml showing up on chest x-ray</td>
</tr>
<tr>
<td>• Pleurisy (pleuritis)</td>
<td>Inflammation of pleura; classification as fibronous (dry) or serofibronous (wet); wet pleurisy accompanied by an increase in pleural fluid and possibly resulting in pleural effusion</td>
</tr>
<tr>
<td>• Pneumothorax</td>
<td>Accumulation of air in pleural space with accompanying lung collapse</td>
</tr>
<tr>
<td><strong>Parenchymal Disorders</strong></td>
<td>Inflammation, collapse, or scarring of the lung tissue</td>
</tr>
<tr>
<td>• Atelectasis</td>
<td>Condition of lung characterized by collapsed, airless alveoli; possibly acute (e.g., in postoperative patient) or chronic (e.g., in patient with malignant tumor)</td>
</tr>
<tr>
<td>• Pneumonia</td>
<td>Acute inflammation of lung tissue caused by bacteria, viruses, fungi, chemicals, dusts, and other factors</td>
</tr>
<tr>
<td>• Interstitial lung diseases (ILDs)</td>
<td>General term that includes a variety of chronic lung disorders characterized by some type of injury, inflammation, and scarring (or fibrosis); this process occurs in the interstitium (tissue between the alveoli) and the lung becomes stiff (fibrotic); can be caused by occupational and environmental exposures (see Table 28-13), infections (e.g., TB), and connective tissue disorders (e.g., rheumatoid arthritis); when all known causes of ILDs are ruled out, the condition is termed idiopathic pulmonary fibrosis (IPF)</td>
</tr>
<tr>
<td>• ARDS*</td>
<td>Atelectasis, pulmonary edema, congestion, and hyaline membrane lining the alveolar wall; result of variety of conditions, including shock lung, O2 toxicity, gram-negative sepsis, cardiopulmonary bypass, and aspiration pneumonia</td>
</tr>
</tbody>
</table>

*See Chapter 68 for clinical manifestations and management. ARDS, Acute respiratory distress syndrome.
IDIOPATHIC PULMONARY FIBROSIS

_**Idiopathic pulmonary fibrosis (IPF)**_ is characterized by scar tissue in the connective tissue of the lungs as a sequel to inflammation or irritation. A common risk factor for IPF is environmental or occupational inhalation of organic and inorganic substances (see the section on Environmental Lung Diseases earlier in this chapter). Other risk factors include cigarette smoking and history of chronic aspiration. There also may be genetic risk factors.

Clinical manifestations of IPF include exertional dyspnea, nonproductive cough, and inspiratory crackles with or without clubbing. Chest x-ray shows changes characteristic of IPF. High-resolution CT scan is the most definitive diagnostic study. Pulmonary function tests show a typical pattern characteristic of restrictive lung disease (see Table 28-23). Open lung biopsy using VATS may help to differentiate the specific pathology.

The clinical course is variable and the prognosis poor, with a 5-year survival rate of 30% to 50% after diagnosis. Treatment includes corticosteroids, cytotoxic agents (azathioprine [Imuran], cyclophosphamide [Cytoxan]), and antifibrotic agents (colchicine). However, there is no good evidence that any of these treatments improves survival or quality of life. Lung transplantation is an option that should be considered for those who meet the criteria. (Lung transplantation is discussed later in this chapter.)

SARCOIDOSIS

_Sarcoidosis_ is a chronic, multisystem granulomatous disease of unknown cause that primarily affects the lungs. The disease may also involve the skin, eyes, liver, kidney, heart, and lymph nodes. It is seen worldwide, can be familial, and is 3 to 4 times more common among African Americans in the United States than among non-African Americans. The lifetime risk of sarcoidosis in African Americans in the United States is 2.4%, compared to a risk of 0.85% for whites.55 Spontaneous resolution of the disease is common, but progressive and disabling organ failure can occur in up to 10% of patients. The disease is often acute or subacute and self-limiting, but in other patients it is chronic, with remissions and exacerbations.

Marked pulmonary fibrosis can be present with severe restrictive lung disease. Cor pulmonale (right-sided heart failure) and bronchiectasis can develop in the advanced stages. Corticosteroids are the most commonly used agents for the treatment of pulmonary sarcoidosis. A trial of methotrexate may be considered if the patient does not respond to or cannot tolerate corticosteroid therapy. If this is ineffective or not tolerated, cyclophosphamide (Cytoxan) or azathioprine (Imuran) may be initiated.55 Nonsteroidal antiinflammatory agents, such as ibuprofen (Motrin), may help decrease acute inflammation or relieve symptoms but are not a treatment of sarcoidosis. Disease progression is monitored by pulmonary function tests, chest x-ray, and CT scan.

Vascular Lung Disorders

PULMONARY EDEMA

_Pulmonary edema_ is an abnormal accumulation of fluid in the alveoli and interstitial spaces of the lungs. It is a complication of various heart and lung diseases (Table 28-26). It is considered a medical emergency and may be life threatening.

Normally, there is a balance between the hydrostatic and colloid osmotic pressures in the pulmonary capillaries. If the hydrostatic pressure increases or the colloid osmotic pressure decreases, the net effect will be fluid leaving the pulmonary capillaries and entering the interstitial space. This stage is referred to as **interstitial edema**. At this stage, the lymphatics can usually drain away the excess fluid. If fluid continues to leak from the pulmonary capillaries, it will enter the alveoli. This stage is referred to as **alveolar edema**. Pulmonary edema interferes with gas exchange by causing an alteration in the diffusing pathway between the alveoli and the pulmonary capillaries. The most common cause of pulmonary edema is left-sided HF. (The clinical manifestations and management of pulmonary edema are described in Chapter 35.)

<table>
<thead>
<tr>
<th>TABLE 28-26 Causes of Pulmonary Edema</th>
</tr>
</thead>
<tbody>
<tr>
<td>- Heart failure</td>
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<tr>
<td>- Overhydration with intravenous fluids</td>
</tr>
<tr>
<td>- Hypoalbuminemia: nephrotic syndrome, hepatic disease, nutritional disorders</td>
</tr>
<tr>
<td>- Altered capillary permeability of lungs: inhaled toxins, inflammation (e.g., pneumonia), severe hypoxia, near-drowning</td>
</tr>
<tr>
<td>- Malignancies of the lymph system</td>
</tr>
<tr>
<td>- Respiratory distress syndrome (e.g., O₂ toxicity)</td>
</tr>
<tr>
<td>- Unknown causes: neurogenic condition, narcotic overdose, reex- pansion pulmonary edema, high altitude</td>
</tr>
</tbody>
</table>

PULMONARY EMBOLISM

Etiology and Pathophysiology

_Pulmonary embolism_ (PE) is the blockage of pulmonary arteries by a thrombus, fat or air embolus, or tumor tissue. The word _embolus_ derives from a Greek word meaning “plug” or “stopper.” A pulmonary embolus consists of material that gains access to the venous system and then to the pulmonary circulation. The material eventually reaches a section of the pulmonary arterial vessels, where it lodges, thus obstructing perfusion (Fig. 28-10). More than 500,000 patients a year are diagnosed with PE in the United States, resulting in approximately 200,000 deaths.56 Of the people who die, approximately 13,000 die because of lack of response to treatment. The vast majority die because of failure of diagnosis.57 Without treatment, the mortality rate is 30%; with treatment there is a 2% to 8% mortality rate.58 It is one of the most common causes of preventable death in hospitalized patients.58

Most pulmonary emboli arise from thrombi in the deep veins of the legs. Other sites of origin include the right side of the heart (especially with atrial fibrillation), the upper extremities (rare), and the pelvic veins (especially after surgery or childbirth). Lethal pulmonary embolism most commonly originate in the femoral or iliac veins. _Embolics_ are mobile clots that generally do not stop moving until they lodge at a narrowed part of the circulatory system. The lungs are an ideal location for emboli to lodge because of their extensive arterial and capillary network. The lower lobes are most frequently affected because they have a higher blood flow than the other lobes.

Thrombi in the deep veins can dislodge spontaneously. However, a more common mechanism is jarring of the thrombus by mechanical forces, such as sudden standing, and by changes in the rate of blood flow, such as those that occur with the Valsalva maneuver. The majority of patients with PE due to deep vein thrombosis (DVT) have no leg symptoms at the time of diagnosis.56 In addition to dislodged thrombi, less common causes of PE include fat emboli (from fractured long bones), air emboli (from
improperly administered IV therapy), bacterial vegetations, amniotic fluid, and tumors. Tumor emboli may originate from primary or metastatic malignancies.

The most common risk factors for PE are immobilization, surgery within the last 3 months, stroke, history of DVT, and malignancy. The Nurses’ Health Study reported an increased risk of PE in women associated with obesity, heavy cigarette smoking, and hypertension. More than 95% of pulmonary emboli arise from thrombi in the deep veins of the lower extremity. Generally the DVTs that are below the knee have not been considered a risk factor for PE since they rarely migrate to the pulmonary circulation without first extending above the knee. Upper extremity DVT occasionally occurs in the presence of central venous catheters or cardiac pacing wires. These cases may resolve with removal of the catheter. The highest rate of DVT is seen in spinal cord injury patients (67% to 100%).

Clinical Manifestations

The signs and symptoms of PE are generally subtle and nonspecific, making diagnosis difficult. The classic triad of dyspnea, chest pain, and hemoptyis occurs in only about 20% of patients. The most common manifestations of PE are anxiety and the sudden onset of unexplained dyspnea, tachypnea, or tachycardia. A mild to moderate hypoxemia with a low PaCO₂ is a common onset of unexplained dyspnea, tachypnea, or tachycardia. A chest pain, and hemoptysis occurs in only about 20% of patients. A physical examination may reveal tachycardia and a pleural friction rub. Small emboli frequently are undetected or produce vague, transient symptoms. The exception to this is the patient with underlying cardiopulmonary disease, in whom even small- or medium-sized emboli may result in severe cardiopulmonary com-

promise. However, repeated small emboli gradually cause a reduction in the capillary bed and eventual pulmonary hypertension. An ECG and chest x-ray may indicate right ventricular hypertrophy secondary to pulmonary hypertension.

Complications

Pulmonary infarction (death of lung tissue) is most likely when the following factors are present: (1) occlusion of a large- or medium-sized pulmonary vessel (>2 mm in diameter), (2) insufficient collateral blood flow from the bronchial circulation, or (3) preexisting lung disease. Infarction results in alveolar necrosis and hemorrhage. Occasionally the infarcted tissue becomes infected, and an abscess may develop. Concomitant pleural effusion is frequently found.

Pulmonary hypertension occurs when more than 50% of the area of the normal pulmonary bed is compromised. Pulmonary hypertension also results from hypoxemia. As a single event, an embolus does not cause pulmonary hypertension unless it is massive. However, recurrent small- to medium-sized emboli may result in chronic pulmonary hypertension. Pulmonary hypertension eventually results in dilatation and hypertrophy of the right ventricle. Depending on the degree of pulmonary hypertension and its rate of development, outcomes can vary, with some patients dying within months of the diagnosis and others living for decades.

Diagnostic Studies

A ventilation-perfusion lung scan is the most frequently used test to aid in the diagnosis of PE. The lung scan has two components and is most accurate when both are performed:

1. Perfusion scanning involves IV injection of a radioisotope. A scanning device detects the adequacy of the pulmonary circulation.
2. Ventilation scanning involves inhalation of a radioactive gas such as xenon. Scanning reflects the distribution of gas through the lung. The ventilation component requires the cooperation of the patient and may be impossible to perform in the critically ill patient, particularly if the patient is intubated.

D-dimer testing may be recommended when a PE is initially suspected. D-dimer is a degradation product rarely found in healthy individuals. However, levels of D-dimer are elevated in any condition involving degradation of fibrin (infection, cancer, surgery, heart failure). They are elevated 8 times higher in venous thromboembolism. When D-dimer levels are normal (<250 µg/L), it is highly unlikely that the patient has a PE. Thus a normal or near-normal D-dimer level can rule out a PE. In the event that the D-dimer levels are elevated, a noninvasive venous study (see Table 38-8) is indicated to look for a DVT as the likely source of a PE. If a DVT is located by venous ultrasound, the index of suspicion for PE is very high and anticoagulant treatment should be initiated immediately. Patients with an elevated D-dimer level but normal venous ultrasound need a lung scan or spiral CT scan.

If the lung scan is inconclusive, pulmonary angiography is recommended. Pulmonary angiography is an invasive procedure that involves the insertion of a catheter through the antecubital or femoral vein, advancement to the pulmonary artery, and injection of contrast medium. This allows visualization of the pulmonary vascular system and location of the embolus.

The use of computed tomography (CT) has revolutionized the diagnosis of PE. A spiral (or helical) CT scan, a noninvasive di-
agin test, may also be used to diagnose PE. Conventional CT scans rotate a frame 360 degrees in one direction, stop, make an image (called a slice), and then spin back in the opposite direction to make another slice after again stopping. The spiral CT scan is able to continuously rotate while obtaining slices and does not have to start and stop between each slice. This allows visualization of entire anatomic regions such as the lungs. The data can be computer reconstructed to allow for a three-dimensional picture of the area being imaged and assist in emboli visualization.

ABG analysis is important, but not diagnostic. The PaO₂ is low because of inadequate oxygenation secondary to an occluded pulmonary vasculature. The PaCO₂ is usually low because of hyperventilation. The pH remains normal unless respiratory alkalosis develops as a result of prolonged hyperventilation or to compensate for lactic acidosis caused by shock. Abnormal findings are usually reported on the chest x-ray (atelectasis, pleural effusion) and the ECG (ST segment and T wave changes), but they are not diagnostic for PE. Serum troponin levels are elevated in 30% to 50% of patients with PE, and, although not diagnostic, they are predictive of an adverse prognosis. Serum b-type natriuretic peptide levels, while not diagnostic, may be helpful in identifying the severity of the clinical course.

**Collaborative Care**

When the diagnosis of PE has been made, treatment should be instituted immediately (Table 28-27). The objectives of treatment are to (1) prevent further growth or multiplication of thrombi in the lower extremities, (2) prevent embolization from the upper or lower extremities to the pulmonary vascular system, and (3) provide cardiopulmonary support if indicated.

**TABLE 28-27** Acute Pulmonary Embolism

<table>
<thead>
<tr>
<th>Collaberaive Therpay</th>
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<tbody>
<tr>
<td><strong>Diagnostic</strong></td>
<td>History and physical examination</td>
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<tr>
<td></td>
<td>Chest x-ray</td>
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<tr>
<td></td>
<td>Continuous ECG monitoring</td>
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<tr>
<td></td>
<td>ABGs</td>
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<td></td>
<td>Venous ultrasound</td>
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<td></td>
<td>CBC count with WBC differential</td>
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<tr>
<td></td>
<td>p-dimer level</td>
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<tr>
<td></td>
<td>Troponin level, BNP level</td>
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<tr>
<td></td>
<td>Ventilation-perfusion lung scan</td>
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<tr>
<td></td>
<td>Spiral CT</td>
</tr>
<tr>
<td></td>
<td>Pulmonary angiography</td>
</tr>
<tr>
<td><strong>Collaborative Therapy</strong></td>
<td>Supplemental oxygen, intubation may be necessary</td>
</tr>
<tr>
<td></td>
<td>IV for medications and fluid replacement</td>
</tr>
<tr>
<td></td>
<td>Continuous IV heparin for acute treatments</td>
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<tr>
<td></td>
<td>Warfarin (Coumadin) for long-term therapy</td>
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<tr>
<td></td>
<td>Monitoring of aPTT and INR levels</td>
</tr>
<tr>
<td></td>
<td>Bed rest</td>
</tr>
<tr>
<td></td>
<td>Narcotics for pain relief</td>
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<tr>
<td></td>
<td>Inferior vena cava filter</td>
</tr>
<tr>
<td></td>
<td>Thrombolytic agent may be considered</td>
</tr>
<tr>
<td></td>
<td>Pulmonary embolectomy in life-threatening situation</td>
</tr>
</tbody>
</table>

**Drug Therapy.** Properly managed anticoagulant therapy is effective in the treatment of many patients with PE. Although unfractionated heparin has been traditionally used for PE, low-molecular-weight heparin (e.g., enoxaparin [Lovenox]) is becoming more commonly used in the treatment of PE. Warfarin (Coumadin) should be initiated within the first 24 hours and is typically administered for 3 to 6 months. Factor Xa inhibitors and direct thrombin inhibitors (see Table 38-9) are also being used in the treatment of PE.

The dosage of heparin is adjusted according to the activated partial thromboplastin time (aPTT), while warfarin dose is determined by the international normalized ratio (INR). Frequent changes and titrations of heparin doses are needed initially in order to obtain a therapeutic aPTT level. The heparin works to prevent future clots, but does not dissolve existing clots. Anticoagulant therapy may be contraindicated if the patient has blood dyscrasias, hepatic dysfunction causing alteration in the clotting mechanism, injury to the intestine, overt bleeding, a history of hemorrhagic stroke, or neurologic conditions.

Fibrinolytic agents, such as tissue plasminogen activator (tPA) or alteplase (Activase), dissolve the pulmonary embolus and the source of the thrombus in the pelvis or deep leg veins, thereby decreasing the likelihood of recurrent emboli. Indications for thrombolytic therapy in PE include hemodynamic instability and right ventricular dysfunction. (Fibrinolytic therapy is discussed in Chapter 34.)

**Surgical Therapy.** If the degree of pulmonary arterial obstruction is severe and the patient does not respond to conservative therapy, an immediate embolectomy may be indicated. Pulmonary embolectomy, a rare procedure, has a 50% mortality rate. Preoperative pulmonary angiography is necessary to identify and locate the site of the embolus. When a pulmonary embolectomy is performed, the patient also has placement of a vena cava filter.

To prevent further pulmonary embolization, an inferior vena cava (IVC) filter may be warranted. This device prevents migration of large clots into the pulmonary system, is easily and safely placed percutaneously, is biocompatible, and does not require the patient be anticoagulated. It can be used for patients who have an absolute contraindication to anticoagulant therapy. In addition, it may be used as a prophylactic measure for patients at high risk of PE (e.g., those with spinal cord injury or cor pulmonale). The complications associated with this device are rare and include misplacement, migration, and perforation. The spinal cord injury patient with an IVC filter cannot have assisted cough (“quad cough”) to mobilize secretions since the quad cough procedure can displace the filter.
NURSING MANAGEMENT

PULMONARY EMBOLISM

Nursing Implementation

Health Promotion. Nursing measures aimed at prevention of PE parallel those for prophylaxis of deep vein thrombosis (see Chapter 38, p. 000).

Acute Intervention. The prognosis of a patient with PE is good if therapy is promptly instituted. The patient should be kept on bed rest in a semi-Fowler position to facilitate breathing. An IV line should be maintained for medications and fluid therapy. The nurse should know the side effects of medications and observe for them. Oxygen therapy should be administered as ordered. Careful monitoring of vital signs, cardiac dysrhythmia monitoring, pulse oximetry, ABGs, and lung sounds is critical to assess the patient’s status. Laboratory results are monitored to assure normal ranges of aPTT and INR. Nursing care includes assessing for the complications of anticoagulant therapy (e.g., bleeding, hematomas, bruising) and PE (e.g., atelectasis, pneumonia). The nursing care plan includes interventions related to immobility and fall precautions.

The patient is usually anxious because of pain, sense of doom, inability to breathe, and fear of death. The nurse should carefully explain the situation and provide emotional support and reassurance to help relieve the patient’s anxiety.

Ambulatory and Home Care. The patient affected by thromboembolic processes may require psychological and emotional support. In addition to the thromboembolic problems, the patient may have an underlying chronic illness requiring long-term treatment. To provide supportive therapy, the nurse must understand and differentiate between the various problems caused by the underlying disease and those related to thromboembolic disease. Patient teaching regarding long-term anticoagulant therapy is critical. The anticoagulant therapy continues for at least 3 to 6 months; patients with recurrent emboli are treated indefinitely. Warfarin blood levels are initially drawn monthly, and patients may have follow-up appointments at a nurse-managed anticoagulation clinic to monitor their medication and adjust dosages.

Long-term management is similar to that for the patient with DVT (see discussion of DVT in Chapter 38 on p. 000). Discharge planning is aimed at limiting progression of the condition and preventing complications and recurrence. The nurse must reinforce the need for the patient to return to the health care provider for regular follow-up examination.

Evaluation

The expected outcomes are that the patient who has a PE will have

• adequate tissue perfusion and respiratory function
• adequate cardiac output
• increased level of comfort
• no recurrence of PE

Pulmonary Hypertension

Pulmonary hypertension is elevated pulmonary pressures resulting from an increase in pulmonary vascular resistance to blood flow. The disease commonly presents with shortness of breath and fatigue. Pulmonary hypertension can occur as a primary disease (primary pulmonary hypertension) or as a secondary complication of a respiratory, cardiac, autoimmune, hepatic, or connective tissue disorder (secondary pulmonary hypertension).

PRIMARY PULMONARY HYPERTENSION

Primary pulmonary hypertension (PPH) is a severe and progressive disease. PPH is characterized by mean pulmonary arterial pressure greater than 25 mm Hg at rest (normal, 12 to 16 mm Hg) or greater than 30 mm Hg with exercise in the absence of a demonstrable cause. Until the last decade, this disorder was rapidly progressive with right heart failure and death. The median survival if untreated was 2.8 years. The introduction of epoprostenol (Flolan) therapy in the 1990s has greatly improved survival rates. Unfortunately, the disease remains incurable despite these advances.

Etiology and Pathophysiology

The exact etiology of PPH is unknown. It is a rare and potentially fatal disease. PPH has been linked to the use of fenfluramine in the drug Fen-Phen, which was used as an appetite suppressant to treat obesity. The drug was withdrawn from the market in 1996. PPH affects more women than men; the mean age at diagnosis is 36 years old. It may have a genetic component as the incidence is higher in families.

Normally the pulmonary circulation is characterized by low resistance and low pressure. In pulmonary hypertension, the pulmonary pressures are elevated. Until recently the pathophysiology of PPH was poorly understood. Recently it was discovered that a key mechanism involved in PPH is a deficient release of vasodilator mediators from the pulmonary epithelium, with a resultant cascade of injury (Fig. 28-11). Vasoconstriction, remodeling of the walls of the pulmonary vessels, and thrombosis in situ are the three elements that combine to cause the increased vascular resistance.61

![FIG. 28-11](Pathogenesis of pulmonary hypertension and cor pulmonale.)
The remodeling process is a complex set of events involving endothelial cell injury that results in intimal/medial wall thickening.

**Clinical Manifestations**

Classic symptoms of pulmonary hypertension are dyspnea on exertion and fatigue. Exertional chest pain, dizziness, and exertional syncope are other symptoms. These symptoms are related to the inability of cardiac output to increase in response to increased oxygen demand. Eventually, as the disease progresses, dyspnea occurs at rest. Pulmonary hypertension increases the workload of the right ventricle and causes right ventricular hypertrophy (a condition called cor pulmonale) and eventually heart failure. A chest x-ray generally shows enlarged central pulmonary arteries and clear lung fields. An enlarged right heart may be seen. Echocardiogram usually reveals right ventricular hypertrophy. The mean time between onset of symptoms and the diagnosis is 2 years. By the time patients become symptomatic, the disease is already in the advanced stages and the pulmonary artery pressure is 2 to 3 times normal.

**Collaborative Care**

PPH is a diagnosis of exclusion. All other conditions must be ruled out. Diagnostic evaluation includes ECG, chest x-ray, pulmonary function tests, echocardiogram, and spiral CT. Cardiac catheterization is done to measure pulmonary artery pressure, cardiac output, and left ventricular filling pressure. Early recognition of pulmonary hypertension is essential to interrupt the vicious cycle responsible for the progression of the disease (see Fig. 28-11). Patients are classified using the New York Heart Association functional classification (see Table 35-4).

Although there is no cure for PPH, treatment can relieve symptoms, increase quality of life, and prolong life. Diuretic therapy relieves dyspnea and peripheral edema and may be useful in reducing right ventricular volume overload. Anticoagulation therapy is recommended for patients with severe pulmonary hypertension to prevent in situ thrombus formation and venous thrombosis. Warfarin is given to keep the INR in the 2 to 3 range. Hypoxia is a potent pulmonary vasoconstrictor, and use of low-flow oxygen provides symptomatic relief. The goal is to keep oxygen saturation at \( >90\% \).

Vasodilator therapy is used to reduce right ventricular overload by dilating pulmonary vessels and reversing remodeling. Some patients with pulmonary hypertension can be effectively managed with calcium channel blocker therapy, such as sustained-release nifedipine (Procardia) or diltiazem (Cardizem).

Epoprostenol (Flolan) has revolutionized the management of PPH. It is a prostacyclin that promotes pulmonary vasodilation and reduces pulmonary vascular resistance. Continuous epoprostenol has been shown to result in significant improvement in clinical symptoms and long-term survival. It is now the treatment of choice for selected patients who are unresponsive to calcium channel blockers. Its administration requires the placement of an indwelling central line catheter and continuous infusion pump. The patient and family must be trained to use the portable intravenous infusion pump, mix medications, manage the central line, and monitor for complications (Fig. 28-12). The half-life of the drug is less than 6 minutes. If the central line is disrupted, stopped, or dislodged for any reason, clinical deterioration from abrupt withdrawal of epoprostenol can occur. This is a serious event with potential rebound pulmonary hypertension and clinical deterioration developing within minutes. The patient will have signs and symptoms of right-sided heart failure, including dyspnea, cyanosis, cough, syncope, and weakness. If the patient loses significant weight, the dosage, which is weight based, may become excessive. Symptoms of overdose include flushing, hypotension, and tachycardia. The major problems have been infections related to vascular access and broken central lines.

Epoprostenol has been successful in improving the quality of life of patients with PPH. The drug was developed as a bridge to lung transplantation but is now a standard of care. Although the patient and family teaching is extensive, the patient on continuous epoprostenol therapy can be successfully managed with an interdisciplinary health care team.

Treprostinil (Remodulin), a prostacyclin, is used as a continuous subcutaneous injection. It causes vasodilation of the pulmonary arterial system and inhibits platelet aggregation. The subcutaneous route has drawbacks, including needles that could dislodge and infusion site pain and reactions. The drug has recently been approved for continuous intravenous use. This drug is stable at room temperature and has a longer half-life than epoprostenol. It may be a reasonable alternative to epoprostenol for some patients.

Bosentan (Tracleer), a form of prostacyclin, is the only oral vasodilator currently approved for the treatment of pulmonary hypertension. It is an active endothelin receptor antagonist and works by blocking the hormone endothelin, which causes blood vessels to constrict. Monthly liver function tests are needed since there is a risk of hepatotoxicity. Sildenafil (Revatio) is an oral phosphodiesterase inhibitor that prolongs the vasodilatory effect of nitric oxide and appears to be effective in decreasing pulmonary vascular resistance. It should not be taken by patients using nitrates because severe hypotension may develop. Iloprost (Ventavis) is an inhaled form of prostacyclin in an aerosolized preparation. It is taken 6 to 9 times a day using a disk inserted into a nebulizer. Because of the risk of orthostatic hypotension, iloprost should not be taken by patients with low systolic blood pressure (<75 mm Hg). Beraprost (an oral formulation of prostacyclin) is undergoing clinical trials for the treatment of early-stage pulmonary hypertension.

Surgical interventions for pulmonary hypertension include atrial septostomy (AS), pulmonary thromboendarterectomy (PTE), and lung transplantation. AS involves the creation of an atrial right-to-left shunt to decompress the right ventricle. It is indicated for a select group of patients awaiting lung transplantation. PTE may provide a potential cure for those patients suffering from...
chronic thromboembolic pulmonary hypertension. It is only recommended for patients with operable sites where the emboli can be surgically removed by embolectomy.

Lung transplantation has been the mainstay of treatment for pulmonary hypertension for those patients who do not respond to drug therapy and progress to severe right-sided heart failure. Recurrence of the disease has not been reported in individuals who have undergone transplantation. A patient education and support website for pulmonary hypertension is located at www.phassociation.org.

SECONDARY PULMONARY HYPERTENSION

Secondary pulmonary hypertension (SPH) occurs when a primary disease causes a chronic increase in pulmonary artery pressures. Secondary pulmonary hypertension can develop as a result of parenchymal lung disease, left ventricular dysfunction, intracardiac shunts, chronic pulmonary thromboembolism, or systemic connective tissue disease. The specific primary disease pathology can result in anatomic or vascular changes causing the pulmonary hypertension. Anatomic changes causing the increased pulmonary vascular resistance include (1) loss of capillaries as a result of alveolar wall damage (e.g., COPD), (2) stiffening of the pulmonary vasculature (e.g., pulmonary fibrosis connective tissue disorders), and (3) obstruction of blood flow (chronic emboli).

Vasomotor increases in pulmonary vascular resistance are found in conditions characterized by alveolar hypoxia. Hypoxia causes localized vasoconstriction and shunting of blood away from poorly ventilated alveoli. Alveolar hypoxia can be caused by a wide variety of conditions. It is possible to have a combination of both anatomic changes and vasomotor constriction. This is found in the patient with long-standing chronic bronchitis who has chronic hypoxia in addition to loss of lung tissue.

The symptoms can reflect the underlying disease, but some are directly attributable to SPH, including dyspnea, fatigue, lethargy, and chest pain. The initial physical findings can include right ventricular hypertrophy and signs of right ventricular failure (increased pulmonic heart sound, right-sided fourth heart sound, peripheral edema, hepatomegaly). Diagnosis of SPH is similar to that of PPH. Treatment of SPH consists mainly of treating the underlying primary disorder. When irreversible pulmonary vascular damage has occurred, therapies used for PPH are initiated. The efficacy of treatment for pulmonary hypertension has been primarily evaluated for PPH, and there are limited data on the effectiveness of these therapies for SPH. Epoprostenol is used in the management of SPH. More studies are ongoing into effective therapies for SPH.

COR PULMONALE

Cor pulmonale is enlargement of the right ventricle secondary to diseases of the lung, thorax, or pulmonary circulation. Pulmonary hypertension is usually a preexisting condition in the individual with cor pulmonale. Cor pulmonale may be present with or without overt cardiac failure. The most common cause of cor pulmonale is COPD. Almost any disorder that affects the respiratory system can cause cor pulmonale. The etiology and pathogenesis of pulmonary hypertension and cor pulmonale are outlined in Fig. 28-11.

Clinical Manifestations

Clinical manifestations of cor pulmonale include dyspnea, chronic productive cough, wheezing respiration, retrosternal or substernal pain, and fatigue. Chronic hypoxemia leads to polycythemia and increased total blood volume and viscosity of the blood. (Polycythemia is often present in cor pulmonale secondary to COPD.) Compensatory mechanisms that are secondary to hypoxemia can aggravate the pulmonary hypertension. Episodes of cor pulmonale in a person with underlying chronic respiratory problems are frequently triggered by an acute respiratory tract infection.

If heart failure accompanies cor pulmonale, additional manifestations such as peripheral edema; weight gain; distended neck veins; full, bounding pulse; and enlarged liver will also be found. (Heart failure is discussed in Chapter 35.) A chest x-ray will show an enlarged right ventricle and pulmonary artery.

Collaborative Care

The primary management of cor pulmonale is directed at treating the underlying pulmonary problem that precipitated the heart problem (Table 28-28). Long-term low-flow O2 therapy is used to correct the hypoxemia and reduce vasoconstriction in chronic states of respiratory disorders. If fluid, electrolyte, and acid-base imbalances are present, they must be corrected. Diuretics and a low-sodium diet will help decrease the plasma volume and the load on the heart. Bronchodilator therapy is indicated if the underlying respiratory problem is due to an obstructive disorder. Digoxin use is controversial. However, studies have confirmed a modest effect of digoxin on the failing right ventricle in chronic cor pulmonale. Other treatments include those for pulmonary hypertension, such as vasodilator therapy, calcium channel blockers, and anticoagulants. Theophylline may help due to its weak inotropic effect on the heart. When medical treatment fails, lung transplantation is an option for some patients.

Chronic management of cor pulmonale resulting from COPD is similar to that described for COPD (see Chapter 28). Continuous low-flow O2 during sleep, exercise, and small, frequent meals may allow the patient to feel better and be more active.

LUNG TRANSPLANTATION

Lung transplantation has become an important mode of therapy for patients with a variety of end-stage lung diseases. A variety of pulmonary disorders are potentially treatable with some type of lung transplantation (Table 28-29). Improved patient selection criteria, technical advances, and better methods of immunosuppres-
Respiratory System

• α1-Antitrypsin deficiency
• Bronchiectasis
• Cystic fibrosis
• Emphysema
• Idiopathic pulmonary fibrosis
• Interstitial lung disease
• Pulmonary fibrosis secondary to other diseases (e.g., sarcoidosis)
• Pulmonary hypertension

**TABLE 28-29** Indications for Lung Transplantation

The majority of these transplant recipients are patients with cystic fibrosis, and their parents or relatives are donors.

Single-lung transplantation involves an incision on the side of the chest. The opposite lung is ventilated while the diseased lung is excised. The lung is removed and the donor lung implanted. Three Anastomoses are done: the bronchus, pulmonary artery, and pulmonary veins. In a bilateral lung transplantation, the incision is made across the sternum and the donor lungs are implanted separately. A median sternotomy incision is used for a heart-lung transplant procedure. Chest tubes are placed around the donor lungs to help them reexpand with air. Lobar transplantation from living donors is reserved for candidates who urgently need transplantation and are unlikely to survive until a donor becomes available. The majority of these transplant recipients are patients with cystic fibrosis, and their parents or relatives are donors.

Patients being considered for lung transplantation need to undergo extensive evaluation. The candidate for lung transplantation should not have a malignancy or recent history of malignancy (within the last 2 years), renal or liver insufficiency, or HIV. Typically patients wait an average of 12 to 18 months for a donor lung.

The candidate and the family undergo psychologic screening to determine the ability to cope with a postoperative regimen that requires strict adherence to immunosuppressive therapy, continuous monitoring for early signs of infection, and prompt reporting of manifestations of infection for medical evaluation. Many transplant centers require preoperative outpatient pulmonary rehabilitation to maximize physical conditioning.

Early postoperative care includes ventilatory support, fluid and hemodynamic management, immunosuppression, detection of early rejection, and prevention or treatment of infection. Pulmonary clearance measures, including aerosolized bronchodilators, chest physiotherapy, and deep-breathing and coughing techniques, minimize potential complications. Maintenance of fluid balance is vital in the postoperative phase.

Lung transplant recipients are at high risk for bacterial, viral, fungal, and protozoal infections. Infections are the leading cause of death in the early period after the transplant. Gram-negative bacterial pneumonia is common. Among potential causes of viral infections, cytomegalovirus (CMV) is the most important in lung transplantation patients, usually seen 4 to 8 weeks postoperatively. Clinical manifestations of CMV infection include fever, bone marrow suppression, hepatitis, enteritis, and pneumonitis. Aspergillus is the most common fungal infection.

Immunosuppressive therapy usually includes a three-drug regimen of cyclosporine or tacrolimus, azathioprine (Imuran) or mycophenolate mofetil (CellCept), and prednisone. (The mechanisms of action of these drugs are discussed in Tables 14-18 and Fig. 14.18.) However, because of an array of potential adverse effects and drug interactions, there are limitations to immunosuppressive therapy. Drug levels are monitored on a regular basis. Lung transplant recipients are usually maintained on higher levels of immunosuppressive therapy than other organ recipients. Immunosuppressive drugs are discussed in Chapter 14.

Acute rejection is fairly common in lung transplantation and can be seen as soon as 5 to 7 days after surgery. It is characterized by low-grade fever, fatigue, and oxygen desaturation with exercise. Accurate diagnosis is by transtracheal biopsy. Treatment is high doses of corticosteroids administered IV for 3 days. In patients with persistent or recurrent acute rejection, other strategies may include antilymphocyte antibodies or changing maintenance immunosuppressive drugs.

**TABLE 28-30** Indications for Lung Transplantation

<table>
<thead>
<tr>
<th>Condition</th>
<th>Indications for Lung Transplantation</th>
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</thead>
<tbody>
<tr>
<td>Idiopathic pulmonary fibrosis</td>
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<tr>
<td>Emphysema</td>
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<tr>
<td>Bronchiectasis</td>
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<td>Cystic fibrosis</td>
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<td>Interstitial lung disease</td>
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<td>Pulmonary fibrosis secondary to other diseases (e.g., sarcoidosis)</td>
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<td>Pulmonary hypertension</td>
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**CRITICAL THINKING EXERCISE**

**CASE STUDY**

**Aspiration Pneumonia**

**Patient Profile.** Sam, a 27-year-old African American man, was admitted to the hospital because of an uncontrollable fever. He was transferred from a long-term care facility. He has a history of a gunshot wound to his left chest. Following a cardiac arrest after the accident, he developed hypoxic encephalopathy. He has a tracheostomy and gastrostomy tube. He has a history of methicillin-resistant *Staphylococcus aureus* (MRSA) in his sputum.

**Subjective Data**

- Family says that they visit him regularly and are very devoted to him.
1. In assessing a patient with pneumococcal pneumonia, the nurse recognizes that clinical manifestations of this condition include:
   a. fever, chills, and a productive cough with rust-colored sputum.
   b. a nonproductive cough and night sweats that are usually self-limiting.
   c. a gradual onset of nasal stuffiness, sore throat, and purulent productive cough.
   d. an abrupt onset of fever, nonproductive cough, and formation of lung abscesses.

2. An appropriate nursing intervention for a patient with pneumonia with the nursing diagnosis of ineffective airway clearance related to thick secretions and fatigue would be to:
   a. perform postural drainage every hour.
   b. provide analgesics as ordered to promote patient comfort.
   c. administer oxygen as prescribed to maintain optimal oxygen levels.
   d. teach the patient how to cough effectively to bring secretions to the mouth.

3. A patient with TB has been admitted to the hospital and is placed in an airborne infection isolation room. The patient is taught all the following to prevent spread of the disease except:
   a. expect routine TST to evaluate infection.
   b. take all medications for the full length of time to prevent multidrug-resistant TB.
   c. wear a standard isolation mask if leaving the airborne infection isolation room.
   d. maintain precautions in airborne infection isolation room by coughing into a paper tissue.

4. A patient has been receiving high-dose corticosteroids and broad-spectrum antibiotics for treatment secondary to a traumatic injury and infection. The nurse plans care for the patient knowing that the patient is most susceptible to:
   a. candidiasis.
   b. aspergillosis.
   c. histoplasmosis.
   d. coccidioidomycosis.

5. Which of the following statements best describes the treatment of lung abscess?
   a. It is best treated with surgical excision and drainage.
   b. Antibiotics given for a prolonged period are the usual treatment of choice.
   c. The abscess is difficult to treat and frequently results in pulmonary fibrosis.
   d. Penicillin can effectively eradicate the anaerobic organisms causing the abscess.

6. A common complication of many types of environmental lung diseases is:
   a. pulmonary fibrosis.
   b. liquefactive necrosis.
   c. benign tumor growth.
   d. diffuse airway obstruction.

7. The patient with lung cancer needs to receive influenza vaccine and pneumococcal vaccines. The nurse will:
   a. administer both vaccines at the same time in the same arm.
   b. administer both vaccines at the same time in different arms.
   c. administer the flu shot and tell the patient to come back 1 week later to receive the pneumococcal vaccine.
   d. administer the pneumococcal vaccine and suggest Flumist (nasal vaccine) instead of the influenza injection.

8. The nurse identifies a flail chest in a trauma patient when:
   a. multiple rib fractures are determined by x-ray.
   b. a tracheal deviation to the unaffected side is present.
   c. paradoxic chest movement occurs during respiration.
   d. there is decreased movement of the involved chest wall.

9. The nurse notes tidaling of the water level in the tube submerged in the water-seal chamber in a patient with closed chest tube drainage. The nurse should:
   a. continue to monitor this normal finding.
   b. check all connections for a leak in the system.
   c. lower the drainage collector further from the chest.
   d. clamp the tubing at progressively distal points away from the patient until the tidaling stops.

10. A nursing measure that should be instituted after a pneumonectomy is:
    a. monitoring chest tube drainage and functioning.
    b. positioning the patient on the unaffected side or back.
    c. range-of-motion exercises on the affected upper extremity.
    d. auscultating frequently for lung sounds on the affected side.

11. Guillain-Barré syndrome causes respiratory problems primarily by:
    a. depressing the CNS.
    b. deforming chest wall muscles.
    c. paralyzing the diaphragm secondary to trauma.
    d. interrupting nerve transmission to respiratory muscles.
12. A patient is on a continuous epoprostenol infusion pump. The alarm goes off indicating an obstruction in the intravenous line downstream. The nurse should:
   a. check vital signs and oxygen saturation.
   b. auscultate the lungs for pulmonary congestion.
   c. assess the central line immediately for any obstruction or accidental clamping of tubing.
   d. monitor for flushing and hypotension due to rebound from no medication and the short half-life of the drug.

13. Which of the following statements does not describe the follow-up management of lung transplantation?
   a. The lung is biopsied using a transtracheal method.
   b. High doses of oxygen are administered around the clock.
   c. The use of a home spirometer will help to monitor lung function.
   d. Immunosuppressant therapy usually involves a three-drug regimen.

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RESOURCES

American Society of Clinical Oncology
703-299-0150
www.asco.org

Cancer Care Ontario
Lung Cancer Clinical Practice Guidelines
416-971-9800
www.ccpemhc.ca/lungcpg.html

Cancer Information Service
NCI Public Inquiries Office
800-422-6237
www.cancer.gov

Centers for Disease Control and Prevention
National Center for Chronic Disease Prevention and Health Promotion Tobacco Information and Prevention Source (TIPS)
800-311-3435 www.cdc.gov/tobacco

Centers for Disease Control and Prevention
National Center for Health Statistics
301-458-4000 www.cdc.gov/nchs/fastats

Centers for Disease Control and Prevention
Surgeon General’s Report on Smoking and Health—2005
404-639-3311 www.cdc.gov/tobacco/sgr/sgr

Pulmonary Hypertension Association (PHA)
301-565-3004 www.phassociation.org

Smoking Cessation Consumer Tool Kit
Agency for Healthcare Research and Quality (AHRQ)
301-427-1364 www.ahrq.gov

Try To Stop
Massachusetts Department of Public Health
800-TRY-TO-STOP (800-879-8678)
www.trytostop.org

For additional Internet resources, see the website for this book at http://evolve.elsevier.com/Lewis/medsurg.