chapter 23

# Management of Patients With Chest and Lower Respiratory Tract Disorders

## LEARNING OBJECTIVES

***On completion of this chapter, the learner will be able to:***

1. Identify patients at risk for atelectasis and nursing interventions related to its prevention and management.
2. Compare the various pulmonary infections with regard to causes, clinical manifestations, nursing management, complications, and prevention.
3. Use the nursing process as a framework for care of the patient with pneumonia.
4. Describe nursing measures to prevent aspiration.
5. Relate pleurisy, pleural effusion, and empyema to pulmonary infection.
6. Describe smoking and air pollution as causes of pulmonary disease.
7. Relate the therapeutic management techniques of acute respiratory distress syndrome to the underlying pathophysiology of the syndrome.
8. Describe risk factors and measures appropriate for prevention and management of pulmonary embolism.
9. Describe preventive measures appropriate for controlling and eliminating occupational lung disease.
10. Discuss the modes of therapy and related nursing management for patients with lung cancer.
11. Describe the complications of chest trauma and their clinical manifestations and nursing management.

## GLOSSARY

**acute lung injury:** an umbrella term for hypoxemic, respiratory failure; acute respiratory distress syndrome is a severe form of acute lung injury

**acute respiratory distress syndrome:** nonspecific pulmonary response to a variety of pulmonary and nonpulmonary insults to the lung; characterized by interstitial infiltrates, alveolar hemorrhage, atelectasis, decreased compliance, and refractory hypoxemia

**asbestosis:** diffuse lung fibrosis resulting from exposure to asbestos fibers

**atelectasis:** collapse or airless condition of the alveoli caused by hypoventilation, obstruction to the airways, or compression

## GLOSSARY *(Continued)*

**central cyanosis:** bluish discoloration of the skin or mucous membranes due to hemoglobin carrying reduced amounts of oxygen

**consolidation:** lung tissue that has become more solid in nature due to collapse of alveoli or infectious process (pneumonia)

**cor pulmonale:** "heart of the lungs"; enlargement of the right ventricle from hypertrophy or dilation or as a secondary response to disorders that affect the lungs

**empyema:** accumulation of purulent material in the pleural space

**fine-needle aspiration:** insertion of a needle through the chest wall to obtain cells of a mass or tumor; usually performed under fluoroscopy or chest computed tomography guidance

**hemoptysis:** the coughing up of blood from the lower respiratory tract

**hemothorax:** partial or complete collapse of the lung due to blood accumulating in the pleural space; may occur after surgery or trauma

**induration:** an abnormally hard lesion or reaction, as in a positive tuberculin skin test

**nosocomial:** pertaining to or originating from a hospitalization; not present at the time of hospital admission

**open lung biopsy:** biopsy of lung tissue performed through a limited thoracotomy incision

**orthopnea:** shortness of breath when reclining or in the supine position

**pleural effusion:** abnormal accumulation of fluid in the pleural space

**pleural friction rub:** localized grating or creaking sound caused by the rubbing together of inflamed parietal and visceral pleurae

**pleural space:** the area between the parietal and visceral pleurae; a potential space

**pneumothorax:** partial or complete collapse of the lung due to positive pressure in the pleural space

**pulmonary edema:** increase in the amount of extravascular fluid in the lung

**pulmonary embolism:** obstruction of the pulmonary vasculature with an embolus; embolus may be due to blood clot, air bubbles, or fat droplets

**purulent:** consisting of, containing, or discharging pus

**restrictive lung disease:** disease of the lung that causes a decrease in lung volumes

**tension pneumothorax:** pneumothorax characterized by increasing positive pressure in the pleural space with each breath; this is an emergency situation and the positive pressure needs to be decompressed or released immediately

**thoracentesis:** insertion of a needle into the pleural space to remove fluid that has accumulated and decrease pressure on the lung tissue; may also be used diagnostically to identify potential causes of a pleural effusion

**transbronchial:** through the bronchial wall, as in a transbronchial lung biopsy

**ventilation–perfusion ratio:** the ratio between ventilation and perfusion in the lung; matching of ventilation to perfusion optimizes gas exchange

Conditions affecting the lower respiratory tract range from acute problems to chronic disorders. Many of these disorders are serious and often life-threatening. Patients with lower respiratory tract disorders require care from nurses with astute assessment and clinical management skills who understand the impact of the particular disorder on the patient's quality of life and ability to carry out usual activities of daily living. Patient and family teaching is an important nursing intervention in the management of all lower respiratory tract disorders.

## ATELECTASIS

**Atelectasis** refers to closure or collapse of alveoli and often is described in relation to x-ray findings and clinical signs and symptoms. Atelectasis may be acute or chronic and may cover a broad range of pathophysiologic changes, from microatelectasis (which is not detectable on chest x-ray) to macroatelectasis with loss of segmental, lobar, or overall lung volume. The most commonly described atelectasis is acute atelectasis, which occurs most often in the postoperative setting or in people who are immobilized and have a shallow, monotonous breathing pattern. Excess secretions or mucous plugs may also cause obstruction of airflow and result in atelectasis in an area of the lung. Atelectasis also is observed in patients with a chronic airway obstruction that impedes or blocks air flow to an area of the lung (eg, obstructive atelectasis in the patient with lung cancer that is invading or compressing the airways). This type of atelectasis is more insidious and slower in onset.

### Pathophysiology

Atelectasis may occur in adults as a result of reduced ventilation or any blockage that obstructs passage of air to and from the alveoli, thus reducing alveolar ventilation. After the trapped alveolar air is absorbed into the bloodstream, no additional air can enter into the alveoli because of the blockage. As a result, the affected portion of the lung becomes airless and the alveoli collapse. Possible causes are altered breathing patterns, retained secretions, pain, alterations in small airway function, prolonged supine positioning, increased abdominal pressure, reduced lung volumes due to musculoskeletal or neurologic disorders, restrictive defects, and specific surgical procedures (eg, upper abdominal, thoracic, or open heart surgery).

Patients are at high risk for atelectasis postoperatively because of several factors. A monotonous, low tidal breathing pattern may cause small airway closure and alveolar collapse. This can result from the effects of anesthesia or analgesic agents, supine positioning, splinting of the chest wall because of pain, or abdominal distention. Secretion retention, airway obstruction, and an impaired cough reflex may also occur, or patients may be reluctant to cough because of pain. Figure 23-1 shows the mechanisms and consequences of acute atelectasis in postoperative patients.

Atelectasis resulting from bronchial obstruction by secretions may also occur in patients with impaired cough mechanisms (eg, musculoskeletal or neurologic disorders) as well as in those who are debilitated and bedridden. In addition, atelectasis may develop because of excessive pressure on the lung tissue, which restricts normal lung expansion on inspiration. Such pressure can be produced by fluid accumulating within the pleural space (**pleural effusion**), air in the pleural space (**pneumothorax**), or blood in the pleural space (**hemothorax**). The **pleural space** is the area between the parietal and the visceral pleurae. Pressure may also be produced by a pericardium distended with fluid (pericardial effusion), tumor growth within the thorax, or an elevated diaphragm.

### Clinical Manifestations

The development of atelectasis usually is insidious. Signs and symptoms include increasing dyspnea, cough, and sputum production.

In acute atelectasis involving a large amount of lung tissue (lobar atelectasis), marked respiratory distress may be observed. In addition to the previously mentioned signs and symptoms, tachycardia, tachypnea, pleural pain, and **central cyanosis** (a bluish skin hue that is a late sign of hypoxemia) may be anticipated. Patients characteristically have difficulty breathing in the supine position and are anxious.

In chronic atelectasis, signs and symptoms are similar to those of acute atelectasis. The chronic nature of the alveolar collapse predisposes patients to infection distal to the obstruction. Therefore, the signs and symptoms of a pulmonary infection also may be present.

### Assessment and Diagnostic Findings

When clinically significant atelectasis develops, it is generally characterized by increased work of breathing and hypoxemia. Decreased breath sounds and crackles are heard over the affected area. A chest x-ray may suggest a diagnosis of atelectasis before clinical symptoms appear; the x-ray may reveal patchy infiltrates or consolidated areas. Depending on the degree of hypoxemia, pulse oximetry ($SpO_2$) may demonstrate a low saturation of hemoglobin with oxygen (less than 90%) or a lower-than-normal partial pressure of arterial oxygen ($PaO_2$).

*NURSING ALERT*

**Tachypnea, dyspnea, and mild to moderate hypoxemia are hallmarks of the severity of atelectasis.**

### Prevention

Nursing measures to prevent atelectasis include frequent turning, early mobilization, and strategies to expand the lungs and to manage secretions. Voluntary deep-breathing maneuvers (at least every 2 hours) assist in preventing and treating atelectasis. The performance of these maneuvers requires the patient to be alert and cooperative. Patient education and reinforcement are key to the success of these interventions. The use of incentive spirometry or voluntary deep breathing enhances lung expansion, decreases the potential for airway closure, and may generate a cough. Secretion management techniques include directed cough, suctioning, aerosol nebulizer treatments followed by chest physical therapy (postural drainage and chest percussion), and bronchoscopy. In some settings, a metered-dose inhaler is used to dispense a bronchodilator rather than an aerosol nebulizer. Chart 23-1 summarizes measures used to prevent atelectasis.

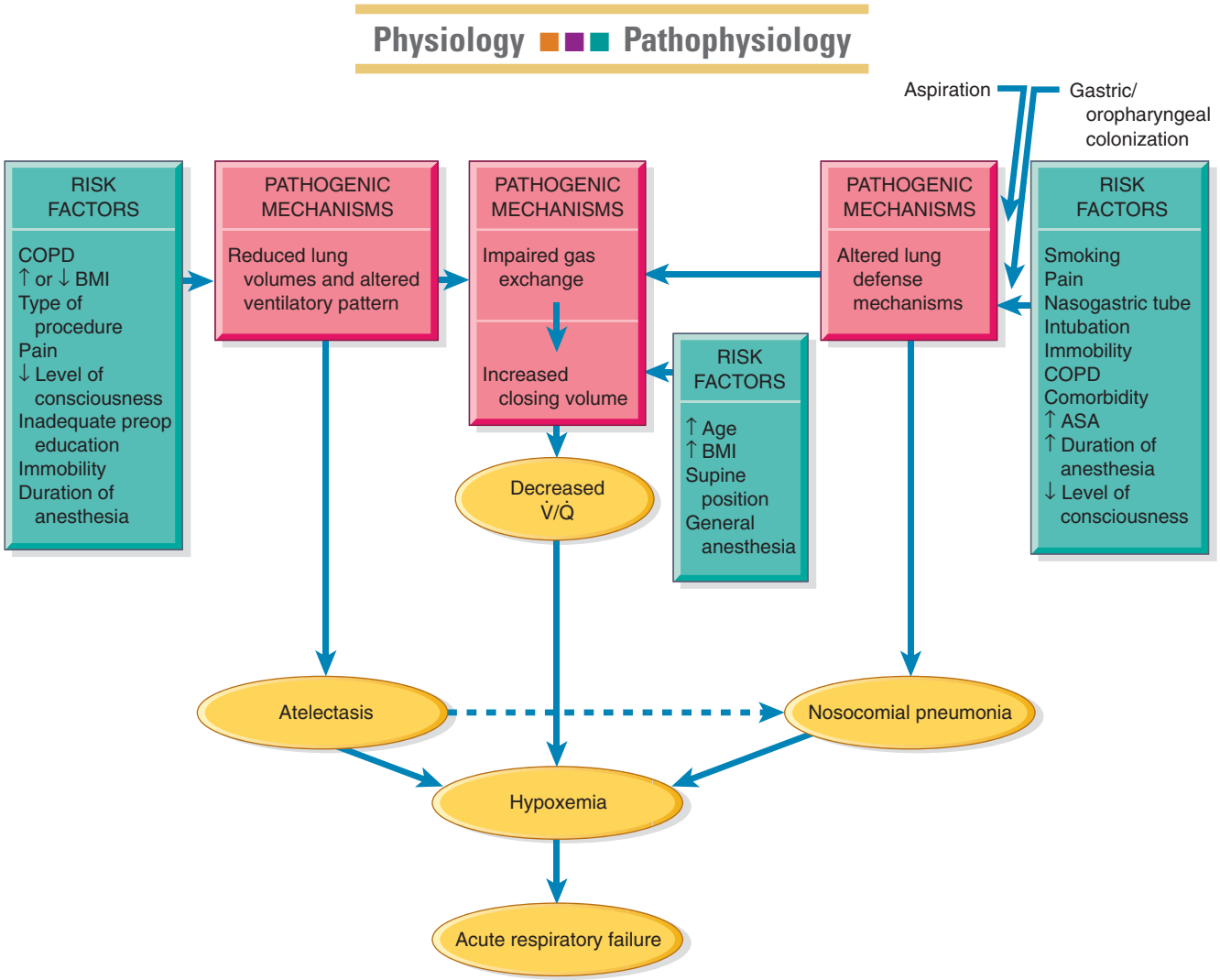


**Figure 23-1** Relationship of risk factors, pathogenic mechanisms, and consequences of acute atelectasis in the postoperative patient. ASA, acetylsalicylic acid; COPD, chronic obstructive pulmonary disease; BMI, body mass index; (V̇/Q̇), ventilation–perfusion ratio. From the work of Jo Ann Brooks-Brunn, DNS, RN, FAAN, FCCP, Indiana University Medical Center, Indianapolis.

## Management

The goal of treatment is to improve ventilation and remove secretions. Strategies to prevent atelectasis, which include frequent turning, early ambulation, lung volume expansion maneuvers (eg, deep-breathing exercises, incentive spirometry), and coughing, also serve as the first-line measures to minimize or treat atelectasis by improving ventilation. In patients who do not respond to first-line measures or who cannot perform deep-breathing exercises, other treatments such as positive end-expiratory pressure (PEEP; a simple mask and one-way valve system that provides varying amounts of expiratory resistance, usually 10 to 15 cm $H_2O$), continuous positive pressure breathing (CPPB), or bronchoscopy may be used. Before initiating more complex, costly, and labor-intensive therapies, the nurse should ask several questions:

- Has the patient been given an adequate trial of deep-breathing exercises?
- Has the patient received adequate education, supervision, and coaching to carry out the deep-breathing exercises?
- Have other factors been evaluated that may impair ventilation or prevent a good patient effort (eg, lack of turning, mobilization; excessive pain; excessive sedation)?

### Chart 23-1 • *Preventing Atelectasis*

- Change patient's position frequently, especially from supine to upright position, to promote ventilation and prevent secretions from accumulating.
- Encourage early mobilization from bed to chair followed by early ambulation.
- Encourage appropriate deep breathing and coughing to mobilize secretions and prevent them from accumulating.
- Teach/reinforce appropriate technique for incentive spirometry.
- Administer prescribed opioids and sedatives judiciously to prevent respiratory depression.
- Perform postural drainage and chest percussion, if indicated.
- Institute suctioning to remove tracheobronchial secretions, if indicated.

If the cause of atelectasis is bronchial obstruction from secretions, the secretions must be removed by coughing or suctioning to allow air to reenter that portion of the lung. Chest physical therapy (chest percussion and postural drainage) may also be used to mobilize secretions. Nebulizer treatments with a bronchodilator or sodium bicarbonate may be used to assist patients in the expectoration of secretions. If respiratory care measures fail to remove the obstruction, a bronchoscopy is performed. Although bronchoscopy is an excellent measure to acutely remove secretions and increase ventilation, it is imperative for the nurse to assist the patient with maintaining the patency of the airways after bronchoscopy, using the traditional techniques of deep breathing, coughing, and suctioning. Severe or massive atelectasis may lead to acute respiratory failure, especially in patients with underlying lung disease. Endotracheal intubation and mechanical ventilation may be necessary.

If the cause of atelectasis is compression of lung tissue, the goal is to decrease the compression. With a large pleural effusion that is compressing lung tissue and causing alveolar collapse, treatment may include **thoracentesis** (removal of the fluid by needle aspiration) or insertion of a chest tube. The measures to increase lung expansion described previously also are used.

Management of chronic atelectasis focuses on removing the cause of the obstruction of the airways or the compression of the lung tissue. For example, bronchoscopy may be used to open an airway obstructed by lung cancer or a nonmalignant lesion, and the procedure may involve cryotherapy or laser therapy. If the atelectasis is a result of obstruction caused by lung cancer, an airway stent or radiation therapy to shrink a tumor may be used to open the airways and provide ventilation to the collapsed area. However, in patients who have experienced chronic, long-term collapse, it may not be possible to reopen the airways and reaerate the area of the lung. In some cases, surgical management may be indicated.

# RESPIRATORY INFECTIONS

## *Acute Tracheobronchitis*

Acute tracheobronchitis, an acute inflammation of the mucous membranes of the trachea and the bronchial tree, often follows infection of the upper respiratory tract (see Chapter 22). Patients with viral infections have decreased resistance and can readily develop a secondary bacterial infection. Adequate treatment of upper respiratory tract infection is one of the major factors in the prevention of acute bronchitis.

### Pathophysiology

In acute tracheobronchitis, the inflamed mucosa of the bronchi produces mucopurulent sputum, often in response to infection by *Streptococcus pneumoniae*, *Haemophilus influenzae*, or *Mycoplasma pneumoniae*. In addition, a fungal infection (eg, *Aspergillus*) may also cause tracheobronchitis. A sputum culture is essential to identify the specific causative organism. In addition to infection, inhalation of physical and chemical irritants, gases, or other air contaminants can also cause acute bronchial irritation.

### Clinical Manifestations

Initially, the patient has a dry, irritating cough and expectorates a scanty amount of mucoid sputum. The patient may report sternal soreness from coughing and have fever or chills, night sweats, headache, and general malaise. As the infection progresses, the patient may be short of breath, have noisy inspiration and expiration (inspiratory stridor and expiratory wheeze), and produce **purulent** (pus-filled) sputum. In severe tracheobronchitis, blood-streaked secretions may be expectorated as a result of the irritation of the mucosa of the airways.

### Medical Management

Antibiotic treatment may be indicated depending on the symptoms, sputum purulence, and results of the sputum culture. Antihistamines usually are not prescribed, because they can cause excessive drying and make secretions more difficult to expectorate. Expectorants may be prescribed, although their efficacy is questionable. Fluid intake is increased to thin the viscous and tenacious secretions. Copious, purulent secretions that cannot be cleared by coughing place patients at risk for increasing airway obstruction and the development of more severe lower respiratory tract infections, such as pneumonia. Suctioning and bronchoscopy may be needed to remove secretions. Rarely, endotracheal intubation may be necessary in cases of acute tracheobronchitis leading to acute respiratory failure, such as in patients who are severely debilitated or who have coexisting diseases that also impair the respiratory system.

In most cases, treatment of tracheobronchitis is largely symptomatic. Increasing the vapor pressure (moisture content) in the air reduces airway irritation. Cool vapor therapy or steam inhalations may help relieve laryngeal and tracheal irritation. Moist heat to the chest may relieve the soreness and pain. Mild analgesic or antipyretic agents may be indicated.

### Nursing Management

Acute tracheobronchitis is usually treated in the home setting. A primary nursing function is to encourage bronchial hygiene, such as increased fluid intake and directed coughing to remove secretions. The nurse encourages and assists the patient to sit up frequently to cough effectively and to prevent retention of mucopurulent sputum. If the patient is taking antibiotics for an underlying infection, it is important to emphasize the need to complete the full course of antibiotics prescribed. Fatigue is a consequence of tracheobronchitis; therefore, the nurse cautions the patient against overexertion, which can induce a relapse or exacerbation of the infection. The patient is advised to rest.

## *Pneumonia*

Pneumonia is an inflammation of the lung parenchyma caused by various microorganisms, including bacteria, mycobacteria, fungi, and viruses. *Pneumonitis* is a more general term that describes an inflammatory process in the lung tissue that may predispose or place the patient at risk for microbial invasion. Pneumonia and influenza are the most

common causes of death from infectious diseases in the United States. Together they account for nearly 60,000 deaths annually and rank as the eighth leading cause of death in the United States (Minino, Heron, Murphy, et al., 2007).

## Classification

Pneumonias are classified as community-acquired pneumonia (CAP), hospital-acquired (nosocomial) pneumonia (HAP), pneumonia in the immunocompromised host, and aspiration pneumonia (Table 23-1). There is overlap in how specific pneumonias are classified, because they may occur in differing settings.

### *Community-Acquired Pneumonia*

CAP occurs either in the community setting or within the first 48 hours after hospitalization or institutionalization. The need for hospitalization for CAP depends on the severity of the pneumonia. The causative agents for CAP that requires hospitalization are most frequently *S. pneumoniae, H. influenzae, Legionella, Pseudomonas aeruginosa,* and other gram-negative rods. The specific etiologic agent is identified in about 50% of cases. It is estimated that more than 915,000 episodes of CAP occur in adults 65 years of age and older each year in the United States (Mandell, Wunderink, Anzueto, et al., 2007).

*S. pneumoniae* (pneumococcus) is the most common cause of CAP in people younger than 60 years of age without comorbidity and in those 60 years and older with comorbidity. *S. pneumoniae,* a gram-positive organism that resides naturally in the upper respiratory tract, colonizes the upper respiratory tract and can cause disseminated invasive infections, pneumonia and other lower respiratory tract infections, and upper respiratory tract infections, such as otitis media and rhinosinusitis. It may occur as a lobar or bronchopneumonic form in patients of any age and may follow a recent respiratory illness.

*H. influenzae* causes a type of CAP that frequently affects elderly people and those with comorbid illnesses (eg, chronic obstructive pulmonary disease [COPD], alcoholism, diabetes mellitus). The presentation is indistinguishable from that of other forms of bacterial CAP and may be subacute, with cough or low-grade fever for weeks before diagnosis.

Mycoplasma pneumonia is caused by *M. pneumoniae*. Mycoplasma pneumonia is spread by infected respiratory droplets through person-to-person contact. Patients can be tested for mycoplasma antibodies. The inflammatory infiltrate is primarily interstitial rather than alveolar. It spreads throughout the entire respiratory tract, including the bronchioles, and has the characteristics of a bronchopneumonia. Earache and bullous myringitis are common. Impaired ventilation and diffusion may occur.

Viruses are the most common cause of pneumonia in infants and children but are relatively uncommon causes of CAP in adults. In immunocompromised adults, cytomegalovirus is the most common viral pathogen, followed by herpes simplex virus, adenovirus, and respiratory syncytial virus. The acute stage of a viral respiratory infection occurs within the ciliated cells of the airways, followed by infiltration of the tracheobronchial tree. With pneumonia, the inflammatory process extends into the alveolar area, resulting in edema and exudation. The clinical signs and symptoms of a viral pneumonia are often difficult to distinguish from those of a bacterial pneumonia.

### *Hospital-Acquired Pneumonia*

HAP, also known as **nosocomial** pneumonia, is defined as the onset of pneumonia symptoms more than 48 hours after admission in patients with no evidence of infection at the time of admission. The most lethal nosocomial infection, HAP accounts for approximately 15% of hospital-acquired infections and is the leading cause of death in patients with such infections (File, 2007). The estimated incidence of HAP is 4 to 7 episodes per 1000 hospitalizations (File, 2007). Ventilator-associated pneumonia is considered a type of nosocomial pneumonia that is associated with endotracheal intubation and mechanical ventilation. This type of pneumonia is defined as bacterial pneumonia that develops in patients with acute respiratory failure who have been receiving mechanical ventilation for at least 48 hours.

Certain factors may predispose patients to HAP because of impaired host defenses (eg, severe acute or chronic illness), a variety of comorbid conditions, supine positioning and aspiration, coma, malnutrition, prolonged hospitalization, hypotension, and metabolic disorders. Hospitalized patients are also exposed to potential bacteria from other sources (eg, respiratory therapy devices and equipment, transmission of pathogens by the hands of health care personnel). Numerous intervention-related factors also may play a role in the development of HAP (eg, therapeutic agents leading to central nervous system depression with decreased ventilation, impaired removal of secretions, or potential aspiration; prolonged or complicated thoracoabdominal procedures, which may impair mucociliary function and cellular host defenses; endotracheal intubation; prolonged or inappropriate use of antibiotics; use of nasogastric tubes). In addition, immunocompromised patients are at particular risk. HAP is associated with a high mortality rate, in part because of the virulence of the organisms, their resistance to antibiotics, and the patient's underlying disorder.

The common organisms responsible for HAP include the pathogens *Enterobacter* species, *Escherichia coli, H. influenzae, Klebsiella* species, *Proteus, Serratia marcescens, P. aeruginosa,* methicillin-sensitive or methicillin-resistant *Staphylococcus aureus* (MRSA), and *S. pneumoniae*. Most patients with HAP are colonized by multiple organisms. Pseudomonal pneumonia occurs in patients who are debilitated, those with altered mental status, and those with prolonged intubation or with tracheostomy. Staphylococcal pneumonia can occur through inhalation of the organism or spread through the hematogenous route. It is often accompanied by bacteremia and positive blood cultures. Its mortality rate is high. Specific strains of staphylococci are resistant to all available antimicrobial agents except vancomycin. Overuse and misuse of antimicrobial agents are major risk factors for the emergence of these resistant pathogens. Because MRSA is highly virulent, steps must be taken to prevent its spread. Patients with MRSA are isolated in a private room, and contact precautions (gown, mask, glove, and antibacterial soap) are used. The number of people in contact with affected patients is minimized, and appropriate precautions must be

**Table 23-1 COMMONLY ENCOUNTERED PNEUMONIAS**

| Type (Causal Organism) | Epidemiology | Clinical Features | Treatment | Complications/ Comments |
|---|---|---|---|---|
| **Community-Acquired Pneumonia** | | | | |
| Streptococcal pneumonia (*Streptococcus pneumoniae*) | Most prevalent in winter months<br>More frequent occurrence in African Americans<br>Incidence greatest in the elderly and in patients with COPD, heart failure, alcoholism, asplenia, diabetes, and after influenza<br>Leading infectious cause of illness worldwide among young children, people with underlying chronic health conditions, and the elderly<br>Mortality in hospitalized adults with invasive disease: 14% | Abrupt onset, toxic appearance, pleuritic chest pain. Usually involves one or more lobes<br>Lobar infiltrate common on chest x-ray or bronchopneumonia pattern | Penicillin nonresistant—penicillin G, amoxicillin<br>Penicillin resistant—cefotaxime, ceftriaxone, fluoroquinolone | Shock, pleural effusion, superinfections, pericarditis, and otitis media |
| *Haemophilus influenzae* (*Haemophilus influenzae*) | Incidence greatest in alcoholics, the elderly, patients in long-term care facilities and nursing homes, patients with diabetes or COPD, and children <5 yr of age<br>Accounts for 5–20% of community-acquired pneumonias<br>Mortality rate: 30% | Frequently insidious onset associated with upper respiratory tract infection 2–6 wk before onset of illness. Fever, chills, productive cough. Usually involves one or more lobes. Bacteremia is common. Infiltrate, occasional bronchopneumonia pattern on chest x-ray | Non–beta-lactamase producing—amoxicillin; beta-lactamase producing—second- or third-generation cephalosporin, amoxicillin-clavulanate | Lung abscess, pleural effusion, meningitis, arthritis, pericarditis, epiglottitis |
| Legionnaires' disease (*Legionella pneumophila*) | Highest occurrence in summer and fall<br>May cause disease sporadically or as part of an epidemic<br>Incidence greatest in middle-aged and older men, smokers, patients with chronic diseases, those receiving immunosuppressive therapy, and those in close proximity to excavation sites<br>Accounts for 15% of community-acquired pneumonias<br>Mortality rate: 15–50%. | Flulike symptoms. High fevers, mental confusion, headache, pleuritic pain, myalgias, dyspnea, productive cough, hemoptysis, leukocytosis<br>Bronchopneumonia, unilateral or bilateral disease, lobar consolidation | Fluoroquinolone, azithromycin | Hypotension, shock, and acute renal failure |
| *Mycoplasma pneumoniae* (*Mycoplasma pneumoniae*) | Increase in fall and winter<br>Responsible for epidemics of respiratory illness<br>Most common type of atypical pneumonia<br>Accounts for 20% of community-acquired pneumonias. More common in children and young adults<br>Mortality rate: <0.1% | Onset is usually insidious. Patients not usually as ill as in other pneumonias. Sore throat, nasal congestion, ear pain, headache, low-grade fever, pleuritic pain, myalgias, diarrhea, erythematous rash, pharyngitis. Interstitial infiltrates on chest x-ray | Macrolide, a tetracycline | Aseptic meningitis, meningoencephalitis, transverse myelitis, cranial nerve palsies, pericarditis, myocarditis |
| Viral pneumonia (influenza viruses types A, B adenovirus, parainfluenza, cytomegalovirus, coronavirus, varicella-zoster) | Incidence greatest in winter months<br>Epidemics occur every 2–3 yr.<br>Most common causative organisms in adults. Other organisms in children (eg, cytomegalovirus, respiratory syncytial virus)<br>Accounts for 20% of community-acquired pneumonias | Patchy infiltrate, small pleural effusion on chest x-ray<br>In most patients, influenza begins as an acute upper respiratory infection; others have bronchitis, pleurisy, and so on, and still others develop gastrointestinal symptoms. | Oseltamivir or zanamivir<br>Treated symptomatically<br>Does not respond to treatment with currently available antimicrobials | Superimposed bacterial infection, bronchopneumonia |

*(Continued)*

**Table 23-1 COMMONLY ENCOUNTERED PNEUMONIAS (Continued)**

| Type (Causal Organism) | Epidemiology | Clinical Features | Treatment | Complications/ Comments |
|---|---|---|---|---|
| Chlamydial pneumonia (*Chlamydophilia pneumoniae*) | Reported mainly in college students, military recruits, and the elderly<br>May be a common cause of community-acquired pneumonia or observed in combination with other pathogens<br>Mortality rate is low because the majority of cases are relatively mild. The elderly with coexistent infections, comorbidities, and reinfections may require hospitalization. | Hoarseness, fever, chills, pharyngitis, rhinitis, nonproductive cough, myalgias, arthralgias<br>Single infiltrate on chest x-ray; pleural effusion possible | Fluoroquinolone | Reinfection and acute respiratory failure |
| **Hospital-Acquired Pneumonia** | | | | |
| *Pseudomonas* pneumonia (*Pseudomonas aeruginosa*) | Incidence greatest in those with preexisting lung disease, cancer (particularly leukemia); those with homograft transplants, burns; debilitated people; and patients receiving antimicrobial therapy and treatments such as tracheostomy, suctioning, and in postoperative settings. Almost always of nosocomial origin.<br>Accounts for 15% of hospital-acquired pneumonias<br>Mortality rate: 40–60% | Diffuse consolidation on chest x-ray. Toxic appearance: fever, chills, productive cough, relative bradycardia, leukocytosis | Antipseudomonal beta-lactam plus ciprofloxacin, levofloxacin or aminoglycoside | Lung cavitation. Has capacity to invade blood vessels, causing hemorrhage and lung infarction. Usually requires hospitalization |
| Staphylococcal pneumonia (*Staphylococcus aureus*) | Incidence greatest in immunocompromised patients, IV drug users, and as a complication of epidemic influenza<br>Commonly nosocomial in origin<br>Accounts for 10–30% of hospital-acquired pneumonias<br>Mortality rate: 25–60%<br>Methicillin-resistant *S. aureus* (MRSA) may also cause community-based infection. | Severe hypoxemia, cyanosis, necrotizing infection. Bacteremia is common. | Methicillin susceptible—antistaphylococcal penicillin<br>Methicillin resistant—vancomycin or linezolid | Pleural effusion/ pneumothorax, lung abscess, empyema, meningitis, endocarditis. Frequently requires hospitalization. Treatment must be vigorous and prolonged because disease tends to destroy lung tissue. |
| *Klebsiella* pneumonia (*Klebsiella pneumoniae* [Friedländer's bacillus-encapsulated gram-negative aerobic bacillus]) | Incidence greatest in the elderly; alcoholics; patients with chronic disease, such as diabetes, heart failure, COPD; patients in chronic care facilities and nursing homes<br>Accounts for 2–5% of community-acquired and 10–30% of hospital-acquired pneumonias<br>Mortality rate: 40–50% | Tissue necrosis occurs rapidly. Toxic appearance: fever, cough, sputum production, bronchopneumonia, lung abscess. Lobar consolidation, bronchopneumonia pattern on chest x-ray | Meropenem or levofloxacin or piperacillin/tazobactam plus amikacin | Multiple lung abscesses with cyst formation, empyema, pericarditis, pleural effusion. May be fulminating, progressing to fatal outcome |

*Continued on following page*

**Table 23-1 COMMONLY ENCOUNTERED PNEUMONIAS (Continued)**

| Type (Causal Organism) | Epidemiology | Clinical Features | Treatment | Complications/ Comments |
|---|---|---|---|---|
| **Pneumonia in the Immunocompromised Host** | | | | |
| *Pneumocystis* pneumonia (PCP) *(Pneumocystis jiroveci)* | Incidence greatest in patients with AIDS and patients receiving immunosuppressive therapy for cancer, organ transplantation, and other disorders. Frequently seen with cytomegalovirus infection<br>Mortality rate 15–20% in hospitalized patients and fatal if not treated | Pulmonary infiltrates on chest x-ray. Nonproductive cough, fever, dyspnea | Trimethoprim/ sulfamethoxazole (TMP-SMZ) | Respiratory failure |
| Fungal pneumonia *(Aspergillus fumigatus)* | Incidence greatest in immunocompromised and neutropenic patients<br>Mortality rate: 15–20%. | Cough, hemoptysis, infiltrates, fungus ball on chest x-ray | Voriconazole or anidulafungin or caspofungin<br>Lobectomy for fungus ball | Dissemination to brain, myocardium, and thyroid gland |
| Tuberculosis *(Mycobacterium tuberculosis)* | Incidence increased in indigent, immigrant, and prison populations, people with AIDS, and the homeless<br>Mortality rate <1% (depending on comorbidity) | Weight loss, fever, night sweats, cough, sputum production, hemoptysis, nonspecific infiltrate (lower lobe), hilar node enlargement, pleural effusion on chest x-ray | Isoniazid plus rifampin plus ethambutol plus pyrazinamide (see section on TB) | Reinfection and acute respiratory infection |
| **Pneumonia from Aspiration** | | | | |
| Anaerobic bacteria *(S. pneumonia, H. influenzae, S. aureus)* | Risk: reduced consciousness, dysphagia, disorders of upper GI tract; mechanical disruption of glottic closure (endotracheal tube, tracheostomy, nasogastric feeding) | Abrupt onset of dyspnea, low-grade fever, cough, predisposing condition for aspiration | Clindamycin or beta-lactam antibiotics | Identification of potential aspirate is important for treatment. |

AIDS, acquired immunodeficiency syndrome; COPD, chronic obstructive pulmonary disease; GI, gastrointestinal; TB, tuberculosis.

taken when transporting these patients within or between facilities.

The usual presentation of HAP is a new pulmonary infiltrate on chest x-ray combined with evidence of infection such as fever, respiratory symptoms, purulent sputum, or leukocytosis. Pneumonias from *Klebsiella* or other gram-negative organisms (*E. coli, Proteus, Serratia*) are characterized by destruction of lung structure and alveolar walls, **consolidation** (tissue that solidifies as a result of collapsed alveoli or infectious process such as pneumonia), and bacteremia. Elderly patients and those with alcoholism, chronic lung disease, or diabetes are at particular risk. Development of a cough or increased cough and sputum production are common presentations, along with low-grade fever and general malaise. In debilitated or dehydrated patients, sputum production may be minimal or absent. Pleural effusion, high fever, and tachycardia are common.

### Pneumonia in the Immunocompromised Host

Pneumonia in immunocompromised hosts includes *Pneumocystis* pneumonia (PCP), fungal pneumonias, and *Mycobacterium tuberculosis*. The organism that causes PCP is now known as *Pneumocystis jiroveci* instead of *Pneumocystis carinii*. The acronym PCP still applies because it can be read "**P***neumo***c***ystis* **p**neumonia."

Pneumonia in the immunocompromised host occurs with use of corticosteroids or other immunosuppressive agents, chemotherapy, nutritional depletion, use of broad-spectrum antimicrobial agents, acquired immunodeficiency syndrome (AIDS), genetic immune disorders, and long-term advanced life-support technology (mechanical ventilation). It is seen with increasing frequency because affected patients constitute a growing portion of the population; however, pneumonias that typically occur in immunocompromised people may also occur in immunocompetent people. Patients with compromised immune systems commonly develop pneumonia from organisms of low virulence. In addition, increasing numbers of patients with impaired defenses develop HAP from gram-negative bacilli (*Klebsiella, Pseudomonas, E. coli*, Enterobacteriaceae, *Proteus, Serratia*).

Pneumonia in immunocompromised hosts may be caused by the organisms also observed in CAP or HAP (*S. pneumoniae, S. aureus, H. influenzae, P. aeruginosa, M. tuberculosis*). PCP is rarely observed in immunocompetent hosts and is often an initial AIDS-defining complication. Whether patients are immunocompromised or immunocompetent, the clinical presentation of pneumonia is similar. PCP has a subtle onset, with progressive dyspnea, fever, and a nonproductive cough.

### *Aspiration Pneumonia*

Aspiration pneumonia refers to the pulmonary consequences resulting from entry of endogenous or exogenous substances into the lower airway. The most common form of aspiration pneumonia is bacterial infection from aspiration of bacteria that normally reside in the upper airways. Aspiration pneumonia may occur in the community or hospital setting. Common pathogens are *S. pneumoniae*, *H. influenzae*, and *S. aureus*. Substances other than bacteria may be aspirated into the lung, such as gastric contents, exogenous chemical contents, or irritating gases. This type of aspiration or ingestion may impair the lung defenses, cause inflammatory changes, and lead to bacterial growth and a resulting pneumonia. (See later discussion of aspiration.)

## Pathophysiology

Normally, the upper airway prevents potentially infectious particles from reaching the sterile lower respiratory tract. Pneumonia arises from normal flora present in patients whose resistance has been altered or from aspiration of flora present in the oropharynx; patients often have an acute or chronic underlying disease that impairs host defenses. Pneumonia may also result from bloodborne organisms that enter the pulmonary circulation and are trapped in the pulmonary capillary bed.

Pneumonia affects both ventilation and diffusion. An inflammatory reaction can occur in the alveoli, producing an exudate that interferes with the diffusion of oxygen and carbon dioxide. White blood cells, mostly neutrophils, also migrate into the alveoli and fill the normally air-filled spaces. Areas of the lung are not adequately ventilated because of secretions and mucosal edema that cause partial occlusion of the bronchi or alveoli, with a resultant decrease in alveolar oxygen tension. Bronchospasm may also occur in patients with reactive airway disease. Because of hypoventilation, a ventilation–perfusion mismatch occurs in the affected area of the lung. Venous blood entering the pulmonary circulation passes through the underventilated area and travels to the left side of the heart poorly oxygenated. The mixing of oxygenated and unoxygenated or poorly oxygenated blood eventually results in arterial hypoxemia.

If a substantial portion of one or more lobes is involved, the disease is referred to as *lobar pneumonia*. The term *bronchopneumonia* is used to describe pneumonia that is distributed in a patchy fashion, having originated in one or more localized areas within the bronchi and extending to the adjacent surrounding lung parenchyma. Bronchopneumonia is more common than lobar pneumonia (Fig. 23-2).

## Risk Factors

Being knowledgeable about the factors and circumstances that commonly predispose people to pneumonia helps identify patients at high risk for the disease. Table 23-2 describes risk factors for pneumonia; additional risk factors are travel or exposure to certain environments and residence in a long-term care facility. Increasing numbers of patients who have compromised defenses against infections are susceptible to pneumonia. Some types of pneumonia, such as those caused by viral infections, occur in previously healthy people, often after a viral illness.

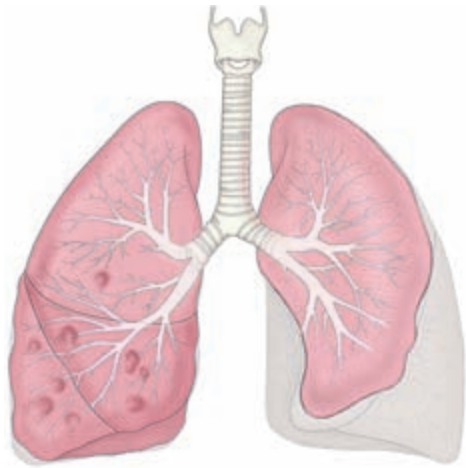


**Figure 23-2** Distribution of lung involvement in bronchial and lobar pneumonia. In bronchopneumonia (*left*), patchy areas of consolidation occur. In lobar pneumonia (*right*), an entire lobe is consolidated.

Pneumonia occurs in patients with certain underlying disorders such as heart failure, diabetes, alcoholism, COPD, and AIDS. Certain diseases also have been associated with specific pathogens. For example, staphylococcal pneumonia has been noted after epidemics of influenza, and patients with COPD are at increased risk for development of pneumonia caused by pneumococci or *H. influenzae*. In addition, cystic fibrosis is associated with respiratory infection caused by pseudomonal and staphylococcal organisms, and PCP has been associated with AIDS. Pneumonias occurring in hospitalized patients often involve organisms not usually found in CAP, including enteric gram-negative bacilli and *S. aureus*.

## Clinical Manifestations

Pneumonia varies in its signs and symptoms depending on the type, causal organism, and presence of underlying disease. However, it is not possible to diagnose a specific form of pneumonia (CAP, HAP, immunocompromised host, or aspiration) by clinical manifestations alone. The patient with streptococcal (pneumococcal) pneumonia usually has a sudden onset of chills, rapidly rising fever (38.5° to 40.5°C [101° to 105°F]), and pleuritic chest pain that is aggravated by deep breathing and coughing. The patient is severely ill, with marked tachypnea (25 to 45 breaths/min), accompanied by other signs of respiratory distress (eg, shortness of breath, use of accessory muscles in respiration). The pulse is rapid and bounding, and it usually increases about 10 bpm for every degree (Celsius) of temperature elevation. A relative bradycardia (a pulse–temperature deficit in which the pulse is slower than that expected for a given temperature) may suggest viral infection, mycoplasma infection, or infection with a *Legionella* organism.

Some patients exhibit an upper respiratory tract infection (nasal congestion, sore throat), and the onset of symptoms of pneumonia is gradual and nonspecific. The predominant symptoms may be headache, low-grade fever, pleuritic pain, myalgia, rash, and pharyngitis. After a few

**Table 23-2 RISK FACTORS AND PREVENTIVE MEASURES FOR PNEUMONIA**

| Risk Factor | Preventive Measure |
|---|---|
| Conditions that produce mucus or bronchial obstruction and interfere with normal lung drainage (eg, cancer, cigarette smoking, chronic obstructive pulmonary disease) | Promote coughing and expectoration of secretions.<br>Encourage smoking cessation. |
| Immunosuppressed patients and those with a low neutrophil count (neutropenic) | Initiate special precautions against infection. |
| Smoking; cigarette smoke disrupts both mucociliary and macrophage activity | Encourage smoking cessation. |
| Prolonged immobility and shallow breathing pattern | Reposition frequently and promote lung expansion exercises and coughing. Initiate suctioning and chest physical therapy if indicated. |
| Depressed cough reflex (due to medications, a debilitated state, or weak respiratory muscles); aspiration of foreign material into the lungs during a period of unconsciousness (head injury, anesthesia, depressed level of consciousness), or abnormal swallowing mechanism | Reposition frequently to prevent aspiration and administer medications judiciously, particularly those that increase risk for aspiration. Perform suctioning and chest physical therapy if indicated. |
| Nothing-by-mouth (NPO) status; placement of nasogastric, orogastric, or endotracheal tube | Promote frequent oral hygiene. Minimize risk for aspiration by checking placement of tube and proper positioning of patient. |
| Supine positioning in patients unable to protect their airway | Elevate head of bed at least 30 degrees. |
| Antibiotic therapy (in very ill people, the oropharynx is likely to be colonized by gram-negative bacteria) | Monitor patients receiving antibiotic therapy for signs and symptoms of pneumonia. |
| Alcohol intoxication (because alcohol suppresses the body's reflexes, may be associated with aspiration, and decreases white cell mobilization and tracheobronchial ciliary motion) | Encourage reduced or moderate alcohol intake (in case of alcohol stupor, position patient to prevent aspiration). |
| General anesthetic, sedative, or opioid preparations that promote respiratory depression, which causes a shallow breathing pattern and predisposes to the pooling of bronchial secretions and potential development of pneumonia | Observe the respiratory rate and depth during recovery from general anesthesia and before giving medications. If respiratory depression is apparent, withhold the medication and contact the physician. |
| Advanced age, because of possible depressed cough and glottic reflexes and nutritional depletion | Promote frequent turning, early ambulation and mobilization, effective coughing, breathing exercises, and nutritious diet. |
| Respiratory therapy with improperly cleaned equipment | Make sure that respiratory equipment is cleaned properly; participate in continuous quality improvement monitoring with the respiratory care department. |
| Transmission of organisms from health care providers | Use strict hand hygiene and gloves.<br>Implement health care provider education. |

days, mucoid or mucopurulent sputum is expectorated. In severe pneumonia, the cheeks are flushed and the lips and nail beds demonstrate central cyanosis (a late sign of poor oxygenation [hypoxemia]).

The patient may exhibit **orthopnea** (shortness of breath when reclining), preferring to be propped up or sitting in bed leaning forward (orthopneic position) in an effort to achieve adequate gas exchange without coughing or breathing deeply. Appetite is poor, and the patient is diaphoretic and tires easily. Sputum is often purulent; however, this is not a reliable indicator of the etiologic agent. Rusty, blood-tinged sputum may be expectorated with streptococcal (pneumococcal), staphylococcal, and *Klebsiella* pneumonia.

Signs and symptoms of pneumonia may also depend on a patient's underlying condition. Different signs occur in patients with conditions such as cancer, and in those who are undergoing treatment with immunosuppressants, which decrease the resistance to infection. Such patients have fever, crackles, and physical findings that indicate consolidation of lung tissue, including increased tactile fremitus (vocal vibration detected on palpation), percussion dullness, bronchial breath sounds, egophony (when auscultated, the spoken "E" becomes a loud, nasal-sounding "A"), and whispered pectoriloquy (whispered sounds are easily auscultated through the chest wall). These changes occur because sound is transmitted better through solid or dense tissue (consolidation) than through normal air-filled tissue; these sounds are described in Chapter 21.

Purulent sputum or slight changes in respiratory symptoms may be the only sign of pneumonia in patients with COPD. It may be difficult to determine whether an increase in symptoms is an exacerbation of the underlying disease process or an additional infectious process.

## Assessment and Diagnostic Findings

The diagnosis of pneumonia is made by history (particularly of a recent respiratory tract infection), physical examination, chest x-ray, blood culture (bloodstream invasion [bacteremia] occurs frequently), and sputum examination. The sputum sample is obtained by having patients do the following: (1) rinse the mouth with water to minimize contamination by normal oral flora, (2) breathe deeply several times, (3) cough deeply, and (4) expectorate the raised sputum into a sterile container.

More invasive procedures may be used to collect specimens. Sputum may be obtained by nasotracheal or orotracheal suctioning with a sputum trap or by fiberoptic bronchoscopy (see Chapter 21). Bronchoscopy is often used in patients with acute severe infection, in patients with chronic or refractory infection, in immunocompromised patients when a diagnosis cannot be made from an expectorated or induced specimen, and in mechanically ventilated patients.

## Prevention

A pneumococcal vaccine provides specific prevention against pneumococcal pneumonia and other infections caused by *S. pneumoniae* (otitis media, other upper respiratory tract infections). Such vaccination has been demonstrated to prevent pneumonia in otherwise healthy patients with an efficiency of 65% to 85%. To reduce or prevent serious complications of CAP in high-risk groups, vaccination against pneumococcal infection is advised for the following:

- People 65 years of age or older
- Immunocompetent people who are at increased risk for illness and death associated with pneumococcal disease because of chronic illness (eg, cardiovascular disease, pulmonary disease, diabetes mellitus, chronic liver disease) or disability
- People with functional or anatomic asplenia
- People living in environments or social settings in which the risk of disease is high
- Immunocompromised people at high risk for infection

The Centers for Disease Control and Prevention (CDC) recommends one-time revaccination after 5 years for people in high-risk categories, including those previously vaccinated up to 5 years ago and those who were older than 65 years of age at the time of primary vaccination (CDC, 2007b).

In addition, the CDC (2004) has identified four specific strategies for the prevention of HAP: (1) staff education and involvement in infection prevention, (2) infection and microbiologic surveillance, (3) prevention of transmission of microorganisms, and (4) modifying host risk for infection. Important nursing measures for prevention of HAP include providing anticipatory interventions and preventive care.

## Medical Management

### Pharmacologic Therapy

The treatment of pneumonia includes administration of the appropriate antibiotic as determined by the results of a Gram stain. However, the causative organism is not identified in half of CAP cases when therapy is started. Guidelines are used to guide antibiotic choice; however, the resistance patterns, prevalence of causative organisms, patient risk factors, treatment setting (inpatient versus outpatient), and costs and availability of newer antibiotic agents must all be considered. Examples of risk factors that may increase the risk of infection with certain types of pathogens appear in Chart 23-2. See Table 23-1 for treatment of patients with pneumonia due to specific pathogens.

Management of CAP includes blood cultures performed quickly for identification of the causal pathogen and prompt administration of antibiotics (within 4 hours) in patients in whom CAP is strongly suspected. In the outpatient setting, empirical treatment of CAP is often used, that is, treatment based on the clinician's estimation of likely causative organisms. In previously healthy people with no risk factors for drug-resistant *S. pneumoniae*, a macrolide antibiotic (azithromycin, clarithromycin, or erythromycin) is recommended. For outpatients with CAP who have cardiopulmonary disease or other modifying factors, treatment should include a respiratory fluoroquinolone (moxifloxacin, gemifloxacin, or levofloxacin) or a beta-lactam agent (cefpodoxime or cefuroxime) plus a macrolide (Mandell, et al., 2007). These are guidelines only; treatment regimens may be modified for individual patients.

For patients with CAP, criteria for hospital admission include age, home environment/caregiver support, severity of illness (eg, pneumonia severity index), and presence of comorbid conditions. For those who are not receiving intensive care, a respiratory fluoroquinolone or a beta-lactam agent plus a macrolide are recommended. Criteria for intensive care unit (ICU) admission include need for mechanical ventilation; presence of septic shock and need for vasopressors; acute respiratory failure; and at least three minor criteria, including increased respiratory rate, multilobar infiltrates, confusion, uremia, leukopenia, and hypothermia (Mandell, et al., 2007). For acutely ill ICU patients, management includes a beta-lactam agent plus azithromycin or a fluoroquinolone. For *Pseudomonas* infection, an antipneumococcal, antipseudomonal beta-lactam is used plus either ciprofloxacin or levofloxacin. Lastly, for community-acquired MRSA, vancomycin or linezolid should be added to the regimen.

Inpatients should be switched from intravenous (IV) to oral therapy when they are hemodynamically stable, are improving clinically, are able to take medications/fluids by mouth, and have a normally functioning gastrointestinal tract. As soon as patients are clinically stable, have no medical problems, and have a safe environment for continued care, they should be discharged from the hospital (Mandell, et al., 2007). Clinical stability is defined as temperature less than or equal to 37.8°C (100°F), heart rate less than or

**CHART 23-2 *Risk Factors for Pathogenic Lung Infections***

**Risk Factors for Infection with Penicillin-Resistant and Drug-Resistant Pneumococci**

- Age over 65 years
- Alcoholism
- Beta-lactam therapy (eg, cephalosporins) in past 3 months
- Immunosuppressive disorders
- Multiple medical comorbidities
- Exposure to a child in a day care facility

**Risk Factors for Infection with Enteric Gram-Negative Bacteria**

- Residency in a long-term care facility
- Underlying cardiopulmonary disease
- Multiple medical comorbidities
- Recent antibiotic therapy

**Risk Factors for Infection with *Pseudomonas aeruginosa***

- Structural lung disease (eg, bronchiectasis)
- Corticosteroid therapy
- Broad-spectrum antibiotic therapy (more than 7 days in the past month)
- Malnutrition

equal to 100 bpm, respiratory rate less than or equal to 24 breaths/min, systolic blood pressure greater than or equal to 90 mm Hg, and oxygen saturation greater than or equal to 90%, with ability to maintain oral intake and normal (baseline) mental status.

In suspected HAP or nosocomial pneumonia, treatment is usually initiated with a broad-spectrum IV antibiotic and may be monotherapy or combination therapy. For patients with no known multidrug resistance, monotherapy with ceftriaxone, ampicillin/sulbactam, levofloxacin, or ertapenem is used. With known multidrug resistance, a three-drug combination therapy may be used; this drug regimen may include an antipseudomonal cephalosporin or ceftazidime or antipseudomonal carbapenem or piperacillin-tazobactam plus antipseudomonal fluoroquinolone or aminoglycoside plus linezolid or vancomycin. The patient's status must be assessed 72 hours after the initiation of therapy, and antibiotics should be discontinued or modified based on the culture results (File, 2007).

Of concern is the rampant rise in respiratory pathogens that are resistant to available antibiotics (Siegel, Rhinehart, Jackson, et al., 2006). Examples include vancomycin-resistant enterococcus (VRE), MRSA, and drug-resistant *S. pneumoniae*. There is a tendency for clinicians to use antibiotics aggressively and inappropriately; they may use broad-spectrum agents when narrow-spectrum agents are more appropriate. Mechanisms to monitor and minimize the inappropriate use of antibiotics are in place. Education of clinicians about the use of evidence-based guidelines in the treatment of respiratory infection is important, and some institutions have implemented algorithms to assist clinicians in choosing the appropriate antibiotics. Monitoring and surveillance of susceptibility patterns for pathogens are also important.

### *Other Therapeutic Regimens*

Antibiotics are ineffective in viral upper respiratory tract infections and pneumonia, and their use may be associated with adverse effects. Treatment of viral infections with antibiotics is a major reason for the overuse of these medications in the United States. Antibiotics are indicated with a viral respiratory infection *only* if a secondary bacterial pneumonia, bronchitis, or rhinosinusitis is present. With the exception of the use of antimicrobial therapy, treatment of viral pneumonia is the same as that for bacterial pneumonia.

Treatment of viral pneumonia is primarily supportive. Hydration is a necessary part of therapy, because fever and tachypnea may result in insensible fluid losses. Antipyretics may be used to treat headache and fever; antitussive medications may be used for the associated cough. Warm, moist inhalations are helpful in relieving bronchial irritation. Antihistamines may provide benefit with reduced sneezing and rhinorrhea. Nasal decongestants may also be used to treat symptoms and improve sleep; however, excessive use can cause rebound nasal congestion. Bed rest is prescribed until the infection shows signs of clearing. If hospitalized, the patient is observed carefully until the clinical condition improves.

If hypoxemia develops, oxygen is administered. Pulse oximetry or arterial blood gas analysis is used to determine the need for oxygen and to evaluate the effectiveness of the therapy. Arterial blood gases may be used to obtain a baseline measure of the patient's oxygenation and acid–base status; however, pulse oximetry is used to continuously monitor the patient's oxygen saturation and response to therapy. More aggressive respiratory support measures include administration of high concentrations of oxygen (fraction of inspired oxygen [$FiO_2$]), endotracheal intubation, and mechanical ventilation. Different modes of mechanical ventilation may be required; see Chapter 25.

## Gerontologic Considerations

Pneumonia in elderly patients may occur as a primary diagnosis or as a complication of a chronic disease. Pulmonary infections in older people frequently are difficult to treat and result in a higher mortality rate than in younger people. General deterioration, weakness, abdominal symptoms, anorexia, confusion, tachycardia, and tachypnea may signal the onset of pneumonia. The diagnosis of pneumonia may be missed because the classic symptoms of cough, chest pain, sputum production, and fever may be absent or masked in elderly patients. Also, the presence of some signs may be misleading. Abnormal breath sounds, for example, may be caused by microatelectasis that occurs as a result of decreased mobility, decreased lung volumes, or other respiratory function changes. Chest x-rays may be needed to differentiate chronic heart failure, which is often seen in the elderly, from pneumonia as the cause of clinical signs and symptoms.

Supportive treatment includes hydration (with caution and with frequent assessment because of the risk of fluid overload in the elderly), supplemental oxygen therapy, and assistance with deep breathing, coughing, frequent position changes, and early ambulation. All of these are particularly important in the care of elderly patients with pneumonia. To reduce or prevent serious complications of pneumonia in the elderly, vaccination against pneumococcal and influenza infections is recommended (Fiore, Shay, Haber, et al., 2007).

## Complications

### *Shock and Respiratory Failure*

Severe complications of pneumonia include hypotension and shock and respiratory failure (especially with gram-negative bacterial disease in elderly patients). These complications are encountered chiefly in patients who have received no specific treatment or inadequate or delayed treatment. These complications are also encountered when the infecting organism is resistant to therapy, when a comorbid disease complicates the pneumonia, or when the patient is immunocompromised.

If the patient is seriously ill, aggressive therapy may include hemodynamic and ventilatory support to combat peripheral collapse, maintain arterial blood pressure, and provide adequate oxygenation. A vasopressor agent may be administered by continuous IV infusion and at a rate adjusted in accordance with the pressure response. Corticosteroids may be administered parenterally to combat shock and toxicity in patients who are extremely ill with pneumonia and at apparent risk for death from the infection. Patients may require endotracheal intubation and mechanical ventilation. Heart failure, cardiac dysrhythmias, pericarditis,

and myocarditis also are complications of pneumonia that may lead to shock.

### Pleural Effusion

Parapneumonic pleural effusions occur in at least 40% of bacterial pneumonias. A parapneumonic effusion is any pleural effusion associated with bacterial pneumonia, lung abscess, or bronchiectasis. After the pleural effusion is detected on a chest x-ray, a thoracentesis may be performed to remove the fluid. The fluid is sent to the laboratory for analysis. There are three stages of parapneumonic pleural effusions based on pathogenesis: uncomplicated, complicated, and thoracic empyema. An **empyema** occurs when thick, purulent fluid accumulates within the pleural space, often with fibrin development and a loculated (walled-off) area where the infection is located (see later discussion). A chest tube may be inserted to treat pleural infection by establishing proper drainage of the empyema. Sterilization of the empyema cavity requires 4 to 6 weeks of antibiotics, which may include clindamycin, meropenem, or piperacillin/tazobactam (Bartlett, 2007). Sometimes surgical management is required.

## NURSING PROCESS

## The Patient With Pneumonia

### Assessment

Nursing assessment is critical in detecting pneumonia. Fever, chills, or night sweats in a patient who also has respiratory symptoms should alert the nurse to the possibility of bacterial pneumonia. Respiratory assessment further identifies the clinical manifestations of pneumonia: pleuritic-type pain, fatigue, tachypnea, use of accessory muscles for breathing, bradycardia or relative bradycardia, coughing, and purulent sputum. The nurse monitors the patient for the following: changes in temperature and pulse; amount, odor, and color of secretions; frequency and severity of cough; degree of tachypnea or shortness of breath; changes in physical assessment findings (primarily assessed by inspecting and auscultating the chest); and changes in the chest x-ray findings.

In addition, it is important to assess elderly patients for unusual behavior, altered mental status, dehydration, excessive fatigue, and concomitant heart failure.

### Diagnosis

#### *Nursing Diagnoses*

Based on the assessment data, the major nursing diagnoses may include the following:

- Ineffective airway clearance related to copious tracheobronchial secretions
- Activity intolerance related to impaired respiratory function
- Risk for deficient fluid volume related to fever and a rapid respiratory rate
- Imbalanced nutrition: less than body requirements
- Deficient knowledge about the treatment regimen and preventive health measures

#### *Collaborative Problems/Potential Complications*

Based on the assessment data, collaborative problems or potential complications that may occur include the following:

- Continuing symptoms after initiation of therapy
- Shock
- Respiratory failure
- Atelectasis
- Pleural effusion
- Confusion

### Planning and Goals

The major goals may include improved airway patency, rest to conserve energy, maintenance of proper fluid volume, maintenance of adequate nutrition, an understanding of the treatment protocol and preventive measures, and absence of complications.

### Nursing Interventions

#### *Improving Airway Patency*

Removing secretions is important because retained secretions interfere with gas exchange and may slow recovery. The nurse encourages hydration (2 to 3 L/day), because adequate hydration thins and loosens pulmonary secretions. Humidification may be used to loosen secretions and improve ventilation. A high-humidity face mask (using either compressed air or oxygen) delivers warm, humidified air to the tracheobronchial tree, helps liquefy secretions, and relieves tracheobronchial irritation. Coughing can be initiated either voluntarily or by reflex. Lung expansion maneuvers, such as deep breathing with an incentive spirometer, may induce a cough. To improve airway patency, the nurse encourages the patient to perform an effective, directed cough, which includes correct positioning, a deep inspiratory maneuver, glottic closure, contraction of the expiratory muscles against the closed glottis, sudden glottic opening, and an explosive expiration. In some cases, the nurse may assist the patient by placing both hands on the lower rib cage (either anteriorly or posteriorly) to focus the patient on a slow deep breath, and then manually assisting the patient by applying constant, external pressure during the expiratory phase.

Chest physiotherapy (percussion and postural drainage) is important in loosening and mobilizing secretions (see Chapter 25). Indications for chest physiotherapy include sputum retention not responsive to spontaneous or directed cough, a history of pulmonary problems previously treated with chest physiotherapy, continued evidence of retained secretions (decreased or abnormal breath sounds, change in vital signs), abnormal chest x-ray findings consistent with atelectasis or infiltrates, and deterioration in oxygenation. The patient is placed in the proper position to drain the involved lung segments, and then the chest is percussed and vibrated either manually or with a mechanical percussor. Other devices, such as the Flutter device (Axcan Pharma), assist in secretion removal. The nurse may consult the respiratory therapist for volume-expansion protocols and secretion-management protocols that help direct the respiratory care of the patient and match the patient's needs with appropriate treatment schedules.

After each position change, the nurse encourages the patient to breathe deeply and cough. If the patient is too weak to cough effectively, the nurse may need to remove the mucus by nasotracheal suctioning (see Chapter 25). It may take time for secretions to mobilize and move into the central airways for expectoration. Therefore, it is important for the nurse to monitor the patient for cough and sputum production after the completion of chest physiotherapy.

The nurse also administers and titrates oxygen therapy as prescribed or via protocols. The effectiveness of oxygen therapy is monitored by improvement in clinical signs and symptoms, patient comfort, and adequate oxygenation values as measured by pulse oximetry or arterial blood gas analysis.

### *Promoting Rest and Conserving Energy*

The nurse encourages the debilitated patient to rest and avoid overexertion and possible exacerbation of symptoms. The patient should assume a comfortable position to promote rest and breathing (eg, semi-Fowler's position) and should change positions frequently to enhance secretion clearance and pulmonary ventilation and perfusion. It is important to instruct outpatients not to overexert themselves and to engage in only moderate activity during the initial phases of treatment.

### *Promoting Fluid Intake*

The respiratory rate of patients with pneumonia increases because of the increased workload imposed by labored breathing and fever. An increased respiratory rate leads to an increase in insensible fluid loss during exhalation and can lead to dehydration. Therefore, it is important to encourage increased fluid intake (at least 2 L/day), unless contraindicated. Note that hydration must be achieved more slowly and with careful monitoring in patients with preexisting conditions such as heart failure.

### *Maintaining Nutrition*

Many patients with shortness of breath and fatigue have a decreased appetite and consume only fluids. Fluids with electrolytes (commercially available drinks, such as Gatorade) may help provide fluid, calories, and electrolytes. Other nutritionally enriched drinks or shakes may be helpful. In addition, IV fluids and nutrients may be administered if necessary.

### *Promoting Patients' Knowledge*

The patient and family are instructed about the cause of pneumonia, management of symptoms, signs and symptoms that should be reported to the physician or nurse, and the need for follow-up. The patient also needs information about factors (both patient risk factors and external factors) that may have contributed to development of pneumonia and strategies to promote recovery and prevent recurrence. If the patient is hospitalized, he or she is instructed about the purpose and importance of management strategies that have been implemented and about the importance of adhering to them during and after the hospital stay. Explanations should be given simply and in language that the patient can understand. If possible, written instructions and information should be provided, and alternative formats should be provided for patients with hearing or vision loss, if necessary. Because of the severity of symptoms, the patient may require that instructions and explanations be repeated several times.

### *Monitoring and Managing Potential Complications*

**CONTINUING SYMPTOMS AFTER INITIATION OF THERAPY.** The patient is observed for response to antibiotic therapy; patients usually begin to respond to treatment within 24 to 48 hours after antibiotic therapy is initiated. If the patient started taking antibiotics before evaluation by culture and sensitivity of the causative organisms, antibiotics may need to be changed once the results are available. The patient is monitored for changes in physical status (deterioration of condition or resolution of symptoms) and for persistent recurrent fever, which may be a result of medication allergy (signaled possibly by a rash); medication resistance or slow response (greater than 48 hours) of the susceptible organism to therapy; pleural effusion; or pneumonia caused by an unusual organism, such as *P. jiroveci* or *Aspergillus fumigatus*. Failure of the pneumonia to resolve or persistence of symptoms despite changes on the chest x-ray raises the suspicion of other underlying disorders, such as lung cancer. As previously described, lung cancers may invade or compress airways, causing an obstructive atelectasis that may lead to pneumonia.

In addition to monitoring for continuing symptoms of pneumonia, the nurse also monitors for other complications, such as shock and multisystem failure and atelectasis, which may develop during the first few days of antibiotic treatment.

**SHOCK AND RESPIRATORY FAILURE.** The nurse assesses for signs and symptoms of shock and respiratory failure by evaluating the patient's vital signs, pulse oximetry values, and hemodynamic monitoring parameters. The nurse reports signs of deteriorating patient status and assists in administering IV fluids and medications prescribed to combat shock. Intubation and mechanical ventilation may be required if respiratory failure occurs. Shock is described in detail in Chapter 15, and care of the patient receiving mechanical ventilation is described in Chapter 25.

**PLEURAL EFFUSION.** If pleural effusion develops and thoracentesis is performed to remove fluid, the nurse assists in the procedure and explains it to the patient. After thoracentesis, the nurse monitors the patient for pneumothorax or recurrence of pleural effusion. If a chest tube needs to be inserted, the nurse monitors the patient's respiratory status (see Chapter 25 for more information on care of patients with chest tubes).

**CONFUSION.** A patient with pneumonia is assessed for confusion and other more subtle changes in cognitive status. Confusion and changes in cognitive status resulting from pneumonia are poor prognostic signs. Confusion may be related to hypoxemia, fever, dehydration, sleep deprivation, or developing sepsis. The patient's underlying comorbid conditions may also play a part in the development of confusion. Addressing the underlying factors and ensuring patient safety are important nursing interventions.

### *Promoting Home and Community-Based Care*

**TEACHING PATIENTS SELF-CARE.** Depending on the severity of the pneumonia, treatment may occur in the hospital or in the outpatient setting. Patient education is crucial regardless of the setting, and the proper administration of antibiotics is important. In some instances, the patient may be initially treated with IV antibiotics as an inpatient and then discharged to continue the IV antibiotics at home. It is important that a seamless system of care be maintained for the patient from hospital to home; this includes communication between the nurses caring for the patient in both settings.

If oral antibiotics are prescribed, it is important to teach the patient about their proper administration and potential side effects. The patient should be instructed about symptoms that require contacting the health care provider: difficulty breathing, worsening cough, recurrent/increasing fever, and medication intolerance.

After the fever subsides, the patient may gradually increase activities. Fatigue and weakness may be prolonged after pneumonia, especially in the elderly. The nurse encourages breathing exercises to promote secretion clearance and volume expansion. A patient who is being treated as an outpatient should be contacted by the health care team or instructed to contact the health care provider 24 to 48 hours after starting therapy. The patient is also instructed to return to the clinic or physician's office for a follow-up chest x-ray and physical examination. Often improvement in chest x-ray findings lags behind improvement in clinical signs and symptoms.

The nurse encourages the patient to stop smoking. Smoking inhibits tracheobronchial ciliary action, which is the first line of defense of the lower respiratory tract. Smoking also irritates the mucous cells of the bronchi and inhibits the function of alveolar macrophage (scavenger) cells. The patient is instructed to avoid stress, fatigue, sudden changes in temperature, and excessive alcohol intake, all of which lower resistance to pneumonia. The nurse reviews with the patient the principles of adequate nutrition and rest, because one episode of pneumonia may make a patient susceptible to recurring respiratory tract infections.

**CONTINUING CARE.** A patient who is severely debilitated or who cannot care for himself or herself may require referral for home care. During home visits, the nurse assesses the patient's physical status, monitors for complications, assesses the home environment, and reinforces previous teaching. The nurse evaluates the patient's adherence to the therapeutic regimen (ie, taking medications as prescribed, performing breathing exercises, consuming adequate fluid and dietary intake, and avoiding smoking, alcohol, and excessive activity). The nurse stresses to the patient and family the importance of monitoring for complications or exacerbation of the pneumonia. The nurse encourages the patient to obtain an influenza vaccination at the prescribed times, because influenza increases susceptibility to secondary bacterial pneumonia, especially that caused by staphylococci, *H. influenzae*, and *S. pneumoniae*. The nurse also urges the patient to seek medical advice about receiving the vaccine (Pneumovax) against *S. pneumoniae*.

## Evaluation

### *Expected Patient Outcomes*

Expected patient outcomes may include the following:

1. Demonstrates improved airway patency, as evidenced by adequate oxygenation by pulse oximetry or arterial blood gas analysis, normal temperature, normal breath sounds, and effective coughing
2. Rests and conserves energy by limiting activities and remaining in bed while symptomatic and then slowly increasing activities
3. Maintains adequate hydration, as evidenced by an adequate fluid intake and urine output and normal skin turgor
4. Consumes adequate dietary intake, as evidenced by maintenance or increase in body weight without excess fluid gain
5. States explanation for management strategies
6. Complies with management strategies
7. Exhibits no complications
   a. Exhibits acceptable vital signs, pulse oximetry, and arterial blood gas measurements
   b. Reports productive cough that diminishes over time
   c. Has absence of signs or symptoms of shock, respiratory failure, or pleural effusion
   d. Remains oriented and aware of surroundings
   e. Maintains or increases weight
8. Complies with treatment protocol and prevention strategies

## Aspiration

Aspiration of stomach contents into the lungs is a serious complication that can cause pneumonia and result in the following clinical picture: tachycardia, dyspnea, central cyanosis, hypertension, hypotension, and finally death. It can occur when the protective airway reflexes are decreased or absent due to a variety of factors (Chart 23-3).

CHART 23-3 *Risk Factors for Aspiration*

- Seizure activity
- Brain injury
- Decreased level of consciousness from trauma, drug or alcohol intoxication, excessive sedation, or general anesthesia
- Nausea and vomiting in the patient with a decreased level of consciousness
- Endotracheal intubation; tube malposition; high residual volumes
- Flat body positioning
- Stroke
- Swallowing disorders
- Cardiac arrest
- Silent aspiration

**NURSING ALERT**

**When a nonfunctioning nasogastric tube allows the gastric contents to accumulate in the stomach, a condition known as silent aspiration may result. Silent aspiration often occurs unobserved and may be more common than suspected. If untreated, massive inhalation of gastric contents develops in a period of several hours.**

## Pathophysiology

The primary factors responsible for death and complications after aspiration of gastric contents are the volume and character of the aspirated gastric contents. For example, a small, localized aspiration from regurgitation can cause pneumonia and acute respiratory distress; a massive aspiration is usually fatal.

A full stomach contains solid particles of food. If these are aspirated, the problem then becomes one of mechanical blockage of the airways and secondary infection. During periods of fasting, the stomach contains acidic gastric juice, which, if aspirated, can be very destructive to the alveoli and capillaries. Fecal contamination (more likely seen in intestinal obstruction) increases the likelihood of death, because the endotoxins produced by intestinal organisms may be absorbed systemically, or the thick proteinaceous material found in the intestinal contents may obstruct the airway, leading to atelectasis and secondary bacterial invasion.

Aspiration pneumonitis may develop from aspiration of substances with a low pH. The aspiration of gastric contents causes a chemical burn of the tracheobronchial tree and pulmonary parenchyma, and an inflammatory response. This leads to the destruction of alveolar–capillary endothelial cells, with a consequent outpouring of protein-rich fluids into the interstitial and intra-alveolar spaces. As a result, surfactant is lost, which in turn causes the airways to close and the alveoli to collapse. Finally, the impaired exchange of oxygen and carbon dioxide causes respiratory failure.

Aspiration pneumonia develops after inhalation of colonized oropharyngeal material. The pathologic process involves an acute inflammatory response to bacteria and bacterial products. Most commonly, the bacteriologic findings include gram-positive cocci, gram-negative rods, and occasionally anaerobic bacteria (Bartlett, 2006).

## Prevention

Prevention is the primary goal when caring for patients at risk for aspiration. Several preventive interventions, including positioning, dietary changes, drugs, oral hygiene, and tube feeding, have been proposed, especially for elderly patients. However, a systematic review of these measures is insufficient to confirm their effectiveness (Bartlett, 2006).

### Compensating for Absent Reflexes

Aspiration may occur if the patient cannot adequately coordinate protective glottic, laryngeal, and cough reflexes. This hazard is increased if the patient has a distended abdomen, is supine, has the upper extremities immobilized by IV infusions or hand restraints, receives local anesthetic agents to the oropharyngeal or laryngeal area for diagnostic procedures, has been sedated, or has had long-term intubation.

When vomiting, people can normally protect their airway by sitting up or turning on the side and coordinating breathing, coughing, gag, and glottic reflexes. If these reflexes are active, an oral airway should not be inserted. If an airway is in place, it should be pulled out the moment the patient gags so as not to stimulate the pharyngeal gag reflex and promote vomiting and aspiration. Suctioning of oral secretions with a catheter should be performed with minimal pharyngeal stimulation.

### Assessing Feeding Tube Placement

When a patient is intubated, aspiration may occur even with a nasogastric tube in place and may result in nosocomial pneumonia. Assessment of nasogastric tube placement is key to prevention of aspiration. The best method for determining tube placement is via x-ray. Other methods have been studied; research results are inconsistent, although observation of the aspirate and testing of its pH are the most reliable (Bourgault, Ipe, Weaver, et al., 2007; Metheny, Meert & Clouse, 2007). Inadvertent tube placement in the respiratory tract is only one source of complications related to tube insertion. A tube with feeding ports in the esophagus significantly increases the risk of aspiration.

Patients who receive continuous or timed-interval tube feedings must be positioned properly. Patients receiving continuous infusions are given small volumes under low pressure in an upright position, which helps prevent aspiration. Patients receiving tube feedings at timed intervals are maintained in an upright or semirecumbent position (elevation of the head of the bed to a 30- to 45-degree angle) during the feeding (Bourgault, et al., 2007). Tube feedings must be given only when it is certain that the feeding tube is positioned correctly in the stomach. Many patients today receive enteral feeding directly into the duodenum through a small-bore flexible feeding tube or surgically implanted tube. Correct placement is confirmed by chest x-ray. Feedings are given slowly and are regulated by a feeding pump.

### Identifying Delayed Stomach Emptying

A full stomach can cause aspiration because of increased intragastric or extragastric pressure. Situations that delay emptying of the stomach include intestinal obstruction; increased gastric secretions in gastroesophageal reflex disease; increased gastric secretions during anxiety, stress, or pain; and abdominal distention because of paralytic ileus, ascites, peritonitis, use of opioids or sedatives, severe illness, or vaginal delivery.

When a feeding tube is present, contents are aspirated, usually every 4 hours, to determine the amount of the last feeding left in the stomach (residual volume). Preliminary evidence suggests that gastric residuals are insensitive and sometimes unreliable markers of tolerance to tube feedings. Patients should be assessed for indications of intolerance to feedings if gastric residual volume is greater than 200 to 250 mL. Prokinetic agents and small bowel feeding should be considered if gastric residual volumes remain high. Stopping enteral nutrition because of a single elevated gastric residual volume should be avoided (Bourgault, et al., 2007).

### Managing Effects of Prolonged Intubation

Prolonged endotracheal intubation or tracheostomy can depress the laryngeal and glottic reflexes because of disuse. Patients with prolonged tracheostomies are encouraged to phonate and exercise their laryngeal muscles. For patients who have had long-term intubation or tracheostomies, it may be helpful to have a speech therapist experienced in swallowing disorders work with the patient to address swallowing problems.

## Severe Acute Respiratory Syndrome

Severe acute respiratory syndrome (SARS) is a viral respiratory illness caused by a coronavirus, called SARS-associated coronavirus. It was first reported in Asia in 2003 and quickly spread to countries in North America, South America, Europe, and Asia. The World Health Organization (WHO) reported that 8422 people worldwide became sick with SARS during the 2003 outbreak, and 916 died (Hirsch, 2007).

SARS develops in people who either have close contact with a person who has been diagnosed with the disease or a history of travel or residence in an area with known cases. The SARS-associated coronavirus is transmitted via respiratory droplets when an infected person coughs or sneezes; the droplets may be deposited on the mucous membranes (mouth, nose, eyes) of a nearby person. The virus may also be spread when a person touches a surface or object contaminated by the droplets and then touches his or her mucous membranes. The virus may be transmitted in other ways, including sewage and water, but these methods of transmission are unclear at this time (Hirsch, 2007).

Characteristic symptoms of SARS are a fever (greater than 38.0°C [100.4°F]), coughing, and difficulty breathing. The incubation period is usually 2 to 7 days; however, longer periods have been reported. About 95% of patients develop symptoms within 10 days (Hirsch, 2007). Risk factors associated with poor outcomes include older age, comorbid conditions (eg, diabetes, chronic hepatitis B, COPD), atypical symptoms, elevated serum lactate dehydrogenase on admission, and acute renal failure. Currently, no treatment except supportive care is recommended (Hirsch, 2007). Antibiotics, antiviral agents, and corticosteroids have been given, but there are no data to support their efficacy.

Infection control measures designed to limit transmission of SARS are a priority. In health care settings, the general CDC guidelines for infection control in health care facilities should be followed; in addition, specific strategies for SARS should be in place including use of negative pressure isolation rooms, personal protective equipment, hand hygiene, environmental cleaning and disinfection techniques, and source control measures to contain patients' secretions (Hirsch, 2007). Additional and updated information is available on the CDC Web site (www.cdc.gov/ncidod/sars).

## Pulmonary Tuberculosis

Tuberculosis (TB) is an infectious disease that primarily affects the lung parenchyma. It also may be transmitted to other parts of the body, including the meninges, kidneys, bones, and lymph nodes. The primary infectious agent, M. *tuberculosis*, is an acid-fast aerobic rod that grows slowly and is sensitive to heat and ultraviolet light. *Mycobacterium bovis* and *Mycobacterium avium* have rarely been associated with the development of a TB infection.

TB is a worldwide public health problem that is closely associated with poverty, malnutrition, overcrowding, substandard housing, and inadequate health care. Mortality and morbidity rates continue to rise; M. *tuberculosis* infects an estimated one third of the world's population and remains the leading cause of death from infectious disease in the world. According to the WHO, an estimated 1.6 million deaths resulted from TB in 2005 (WHO, 2007).

In the United States, almost 15,000 cases of TB are reported annually to the CDC (2005a). Factors that prevent elimination of TB in the United States are the prevalence of TB among foreign-born residents, delays in detecting and reporting cases of TB, lack of protection of contacts of people with infections cases of TB, presence of substantial number of people with latent TB, and maintaining clinical and public health expertise in TB (CDC, 2005a).

### Transmission and Risk Factors

TB spreads from person to person by airborne transmission. An infected person releases droplet nuclei (usually particles 1 to 5 μm in diameter) through talking, coughing, sneezing, laughing, or singing. Larger droplets settle; smaller droplets remain suspended in the air and are inhaled by a susceptible person. Chart 23-4 lists risk factors for TB. Chart 23-5 summarizes the CDC's recommendations for prevention of TB transmission in health care settings.

CHART 23-4 *Risk Factors for Tuberculosis (TB)*

- Close contact with someone who has active TB. Inhalation of airborne nuclei from an infected person is proportional to the amount of time spent in the same air space, the proximity of the person, and the degree of ventilation.
- Immunocompromised status (eg, those with HIV infection, cancer, transplanted organs, and prolonged high-dose corticosteroid therapy)
- Substance abuse (IV/injection drug users and alcoholics)
- Any person without adequate health care (the homeless; impoverished; minorities, particularly children under age 15 year and young adults between ages 15 and 44 year)
- Preexisting medical conditions or special treatment (eg, diabetes, chronic renal failure, malnourishment, selected malignancies, hemodialysis, transplanted organ, gastrectomy, jejunoileal bypass)
- Immigration from countries with a high prevalence of TB (southeastern Asia, Africa, Latin America, Caribbean)
- Institutionalization (eg, long-term care facilities, psychiatric institutions, prisons)
- Living in overcrowded, substandard housing
- Being a health care worker performing high-risk activities: administration of aerosolized pentamidine and other medications, sputum induction procedures, bronchoscopy, suctioning, coughing procedures, caring for the immunosuppressed patient, home care with the high-risk population, and administering anesthesia and related procedures (eg, intubation, suctioning)

**Chart 23-5 • *CDC Recommendations for Preventing Transmission of Tuberculosis (TB) in Health Care Settings***

1. Early identification and treatment of persons with active TB
   a. Maintain a high index of suspicion for TB to identify cases rapidly.
   b. Promptly initiate effective multidrug anti-TB therapy based on clinical and drug-resistance surveillance data.
2. Prevention of spread of infectious droplet nuclei by source control methods and by reduction of microbial contamination of indoor air
   a. Initiate acid-fast bacilli (AFB) isolation precautions immediately for all patients who are suspected or confirmed to have active TB and who may be infectious. AFB isolation precautions include use of a private room with negative pressure in relation to surrounding areas and a minimum of six air exchanges per hour. Air from the room should be exhausted directly to the outside. Use of ultraviolet lamps and/or high-efficiency particulate air filters to supplement ventilation may be considered.
   b. Persons entering the AFB isolation room should use disposable particulate respirators that fit snugly around the face.
   c. Continue AFB isolation precautions until there is clinical evidence of reduced infectiousness (ie, cough has substantially decreased, and the number of organisms on sequential sputum smears is decreasing). If drug resistance is suspected or confirmed, continue AFB precautions until the sputum smear is negative for AFB.
   d. Use special precautions during cough-inducing procedures.
3. Surveillance for TB transmission
   a. Maintain surveillance for TB infection among health care workers (HCWs) by routine, periodic tuberculin skin testing. Recommend appropriate preventive therapy for HCWs when indicated.
   b. Maintain surveillance for TB cases among patients and HCWs.
   c. Promptly initiate contact investigation procedures among HCWs, patients, and visitors exposed to an untreated, or ineffectively treated, patient with infectious TB for whom appropriate AFB procedures are not in place. Recommend appropriate therapy or preventive therapy for contacts with disease or TB infection without current disease. Therapeutic regimens should be chosen based on the clinical history and local drug-resistance surveillance data.

## Pathophysiology

TB begins when a susceptible person inhales mycobacteria and becomes infected. The bacteria are transmitted through the airways to the alveoli, where they are deposited and begin to multiply. The bacilli also are transported via the lymph system and bloodstream to other parts of the body (kidneys, bones, cerebral cortex) and other areas of the lungs (upper lobes). The body's immune system responds by initiating an inflammatory reaction. Phagocytes (neutrophils and macrophages) engulf many of the bacteria, and TB-specific lymphocytes lyse (destroy) the bacilli and normal tissue. This tissue reaction results in the accumulation of exudate in the alveoli, causing bronchopneumonia. The initial infection usually occurs 2 to 10 weeks after exposure.

Granulomas, new tissue masses of live and dead bacilli, are surrounded by macrophages, which form a protective wall. They are then transformed to a fibrous tissue mass, the central portion of which is called a Ghon tubercle. The material (bacteria and macrophages) becomes necrotic, forming a cheesy mass. This mass may become calcified and form a collagenous scar. At this point, the bacteria become dormant, and there is no further progression of active disease.

After initial exposure and infection, active disease may develop because of a compromised or inadequate immune system response. Active disease also may occur with reinfection and activation of dormant bacteria. In this case, the Ghon tubercle ulcerates, releasing the cheesy material into the bronchi. The bacteria then become airborne, resulting in further spread of the disease. Then the ulcerated tubercle heals and forms scar tissue. This causes the infected lung to become more inflamed, resulting in further development of bronchopneumonia and tubercle formation.

Unless this process is arrested, it spreads slowly downward to the hilum of the lungs and later extends to adjacent lobes. The process may be prolonged and is characterized by long remissions when the disease is arrested, followed by periods of renewed activity. Approximately 10% of people who are initially infected develop active disease. Some people develop reactivation TB (also called adult-type TB). This type of TB results from a breakdown of the host defenses. It most commonly occurs in the lungs, usually in the apical or posterior segments of the upper lobes or the superior segments of the lower lobes.

## Clinical Manifestations

The signs and symptoms of pulmonary TB are insidious. Most patients have a low-grade fever, cough, night sweats, fatigue, and weight loss. The cough may be nonproductive, or mucopurulent sputum may be expectorated. Hemoptysis also may occur. Both the systemic and the pulmonary symptoms are chronic and may have been present for weeks to months. Elderly patients usually present with less pronounced symptoms than younger patients. Extrapulmonary disease occurs in up to 16% of cases in the United States. In patients with AIDS, extrapulmonary disease is more prevalent.

## Assessment and Diagnostic Findings

A complete history, physical examination, tuberculin skin test, chest x-ray, acid-fast bacillus smear, and sputum culture are used to diagnose TB. If the patient is infected with TB, the chest x-ray usually reveals lesions in the upper lobes, and the acid-fast bacillus smear contains mycobacteria.

Clinical manifestations of fever, anorexia, weight loss, night sweats, fatigue, cough, and sputum production prompt a more thorough assessment of respiratory function, for example, assessing the lungs for consolidation by evaluating breath sounds (diminished, bronchial sounds; crackles), fremitus, and egophony.

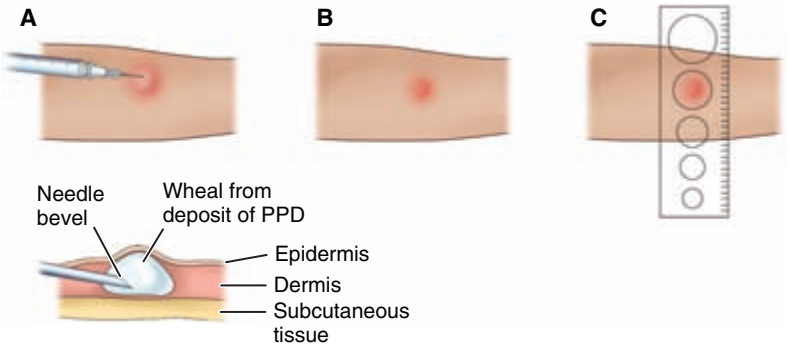


**Figure 23-3** The Mantoux test for tuberculosis. **A,** Correct technique for inserting the needle involves depositing the purified protein derivative (PPD) subcutaneously with the needle bevel facing upward. **B,** The reaction to the Mantoux test usually consists of a wheal, a hivelike, firm welt. **C,** To determine the extent of the reaction, the wheal is measured using a commercially prepared gauge. Interpretation of the Mantoux test is discussed in the text.

### *Tuberculin Skin Test*

The Mantoux method is used to determine whether a person has been infected with the TB bacillus. The Mantoux method is a standardized, intracutaneous injection procedure and should be performed only by those trained in its administration and reading. Tubercle bacillus extract (tuberculin), purified protein derivative (PPD), is injected into the intradermal layer of the inner aspect of the forearm, approximately 4 inches below the elbow (Fig. 23-3). Intermediate-strength PPD, in a tuberculin syringe with a half-inch 26- or 27-gauge needle, is used. The needle, with the bevel facing up, is inserted beneath the skin. Then 0.1 mL of PPD is injected, creating an elevation in the skin, a well-demarcated wheal 6 to 10 mm in diameter. The site, antigen name, strength, lot number, date, and time of the test are recorded. The test result is read 48 to 72 hours after injection. Tests read after 72 hours tend to underestimate the true size of **induration** (hardening). A delayed localized reaction indicates that the person is sensitive to tuberculin.

A reaction occurs when both induration and erythema (redness) are present. After the area is inspected for induration, it is lightly palpated across the injection site, from the area of normal skin to the margins of the induration. The diameter of the induration (not erythema) is measured in millimeters at its widest part (see Fig. 23-3), and the size of the induration is documented. Erythema without induration is not considered significant.

The size of the induration determines the significance of the reaction. A reaction of 0 to 4 mm is considered not significant. A reaction of 5 mm or greater may be significant in people who are considered to be at risk. It is defined as positive in patients who are human immunodeficiency virus (HIV) positive or have HIV risk factors and are of unknown HIV status, in those who are close contacts of someone with active TB, and in those who have chest x-ray results consistent with TB. An induration of 10 mm or greater is usually considered significant in people who have normal or mildly impaired immunity. A significant reaction indicates past exposure to *M. tuberculosis* or vaccination with bacille Calmette-Guérin (BCG) vaccine. The BCG vaccine is given to produce a greater resistance to development of TB. It is effective in up to 76% of people who receive it. The BCG vaccine is used in Europe and Latin America but not routinely in the United States.

A significant (positive) reaction does not necessarily mean that active disease is present in the body. More than 90% of people who are tuberculin-significant reactors do not develop clinical TB. However, all significant reactors are candidates for active TB. In general, the more intense the reaction, the greater the likelihood of an active infection.

A nonsignificant (negative) skin test does not exclude TB infection or disease, because patients who are immunosuppressed cannot develop an immune response that is adequate to produce a positive skin test. This is referred to as anergy.

### *QuantiFERON-TB Gold Test*

In 2005, the U.S. Food and Drug Administration approved a new test for the detection of TB. The QuantiFERON-TB Gold (QFT-G) test is an enzyme-linked immunosorbent assay (ELISA) that detects the release of interferon-gamma by white blood cells when the blood of a patient with TB is incubated with peptides similar to those in *M. tuberculosis*. The results of the QFT-G test are available in less than 24 hours and are not affected by prior vaccination with BCG. A positive tuberculin skin test or QFT-G only indicates that a person has been infected with TB. It does not indicate whether or not the person has active progression of the disease (CDC, 2005c).

## Classification

Data from the history, physical examination, TB test, chest x-ray, and microbiologic studies are used to classify TB into one of five classes. A classification scheme provides public health officials with a systematic way to monitor epidemiology and treatment of the disease.

- Class 0: no exposure; no infection
- Class 1: exposure; no evidence of infection
- Class 2: latent infection; no disease (eg, positive PPD reaction but no clinical evidence of active TB)
- Class 3: disease; clinically active
- Class 4: disease; not clinically active
- Class 5: suspected disease; diagnosis pending

## Gerontologic Considerations

TB may have atypical manifestations in elderly patients, whose symptoms may include unusual behavior and altered mental status, fever, anorexia, and weight loss. In many elderly patients, the tuberculin skin test produces no reaction (loss of immunologic memory) or delayed reactivity for up to 1 week (recall phenomenon). A second skin test is performed in 1 to 2 weeks.

## Medical Management

Pulmonary TB is treated primarily with antituberculosis agents for 6 to 12 months. A prolonged treatment duration is necessary to ensure eradication of the organisms

and to prevent relapse. The continuing and increasing resistance of *M. tuberculosis* to TB medications is a worldwide concern and challenge in TB therapy. Several types of drug resistance must be considered when planning effective therapy:

- Primary drug resistance: resistance to one of the first-line antituberculosis agents in people who have not had previous treatment
- Secondary or acquired drug resistance: resistance to one or more antituberculosis agents in patients undergoing therapy
- Multidrug resistance: resistance to two agents, isoniazid (INH) and rifampin. The populations at greatest risk for multidrug resistance are those who are HIV positive, institutionalized, or homeless.

The increasing prevalence of drug resistance points out the need to begin TB treatment with four or more medications, to ensure completion of therapy, and to develop and evaluate new anti-TB medications.

### Pharmacologic Therapy

In current TB therapy, four first-line medications are used (Table 23-3), INH, rifampin (Rifadin), pyrazinamide, and ethambutol (Myambutol). Combination medications, such as INH and rifampin (Rifamate) or INH, pyrazinamide (PZA), and rifampin (Rifater) and medications administered twice a week (eg, rifapentine [Priftin]) are available to help improve patient adherence. Capreomycin (Capastat), ethionamide (Trecator), para-aminosalicylate sodium, and cycloserine (Seromycin) are second-line medications. Additional potentially effective medications include other aminoglycosides, quinolones, rifabutin, and clofazimine (Lamprene).

Recommended treatment guidelines for newly diagnosed cases of pulmonary TB (CDC, 2003) have two parts: an initial treatment phase and a continuation phase. The initial phase consists of a multiple-medication regimen of INH, rifampin, pyrazinamide, and ethambutol. This initial intensive-treatment regimen is administered daily for 8 weeks, after which options for the continuation phase of treatment include INH and rifampin or INH and rifapentine. The continuation regimen lasts for an additional 4 or 7 months. The 4-month period is used for the large majority of patients (CDC, 2003). The 7-month period is recommended for patients with cavitary pulmonary TB whose sputum culture after the initial 2 months of treatment is positive, for those whose initial phase of treatment did not include PZA, and for those being treated once weekly with INH and rifapentine whose sputum culture is positive at the end of the initial phase of treatment. People are considered noninfectious after 2 to 3 weeks of continuous medication therapy. Vitamin B (pyridoxine) is usually

**Table 23-3 FIRST-LINE ANTITUBERCULOSIS MEDICATIONS**

| Commonly Used Agents | Adult Daily Dosage* | Most Common Side Effects | Drug Interactions† | Remarks* |
|---|---|---|---|---|
| Isoniazid (INH) | 5 mg/kg (300 mg maximum daily) | Peripheral neuritis, hepatic enzyme elevation, hepatitis, hypersensitivity | Phenytoin—synergistic<br>Antabuse<br>Alcohol | Bactericidal<br>Pyridoxine is used as prophylaxis for neuritis.<br>Monitor AST and ALT. |
| Rifampin (Rifadin) | 10 mg/kg (600 mg maximum daily) | Hepatitis, febrile reaction, purpura (rare), nausea, vomiting | Rifampin increases metabolism of oral contraceptives, quinidine, corticosteroids, coumarin derivatives and methadone, digoxin, oral hypoglycemics; PAS may interfere with absorption of rifampin. | Bactericidal<br>Orange urine and other body secretions<br>Discoloring of contact lenses<br>Monitor AST and ALT |
| Rifabutin (Mycobutin) | 5 mg/kg (300 mg maximum daily) | | | |
| Rifapentine (Priftin) | 10 mg/kg (600 mg twice weekly) | Hepatotoxicity, thrombocytopenia | Avoid protease inhibitors. | Orange-red coloration of body secretions, contact lenses, dentures<br>Use with caution in elderly or in those with renal disease. |
| Pyrazinamide | 15–30 mg/kg (2.0 g maximum daily)* | Hyperuricemia, hepatotoxicity, skin rash, arthralgias, GI distress | | Bactericidal<br>Monitor uric acid, AST, ALT |
| Ethambutol (Myambutol) | 15–25 mg/kg (no maximum daily dose, but base on lean body wt)* | Optic neuritis (may lead to blindness; very rare at 15 mg/kg), skin rash | | Bacteriostatic<br>Use with caution with renal disease or when eye testing is not feasible. Monitor visual acuity, color discrimination.‡ |
| Combinations: INH + rifampin (eg, Rifamate) | 150-mg & 300-mg caps (2 caps daily) | | | |

*Check product labeling for detailed information on dose, contraindications, drug interactions, adverse reactions, and monitoring.
†Refer to current literature, particularly on rifampin, because it increases hepatic microenzymes and therefore interacts with many drugs.
‡Initial examination should be performed at start of treatment.

administered with INH to prevent INH-associated peripheral neuropathy (see Table 23-3). The total number of doses taken, not simply the duration of treatment, more accurately determines whether a course of therapy has been completed.

INH also may be used as a prophylactic (preventive) measure for people who are at risk for significant disease, including:

- Household family members of patients with active disease
- Patients with HIV infection who have a PPD test reaction with 5 mm of induration or more
- Patients with fibrotic lesions suggestive of old TB detected on a chest x-ray and a PPD reaction with 5 mm of induration or more
- Patients whose current PPD test results show a change from former test results, suggesting recent exposure to TB and possible infection (skin test converters)
- Users of IV/injection drugs who have PPD test results with 10 mm of induration or more
- Patients with high-risk comorbid conditions and a PPD result with 10 mm of induration or more

Other candidates for preventive INH therapy are those 35 years or younger who have PPD test results with 10 mm of induration or more and one of the following criteria:

- Foreign-born individuals from countries with a high prevalence of TB
- High-risk, medically underserved populations
- Institutionalized patients

Prophylactic INH treatment involves taking daily doses for 6 to 12 months. Liver enzymes, blood urea nitrogen (BUN), and creatinine levels are monitored monthly. Sputum culture results are monitored for acid-fast bacillus to evaluate the effectiveness of treatment and the patient's adherence to the treatment regimen.

## Nursing Management

Nursing management includes promoting airway clearance, advocating treatment regimen, promoting activity and nutrition, and preventing transmission.

### *Promoting Airway Clearance*

Copious secretions obstruct the airways in many patients with TB and interfere with adequate gas exchange. Increasing the fluid intake promotes systemic hydration and serves as an effective expectorant. The nurse instructs the patient about correct positioning to facilitate airway drainage (postural drainage); this is described in Chapter 25.

### *Advocating Adherence to Treatment Regimen*

The multiple-medication regimen that the patient must follow can be quite complex. Understanding of the medications, schedule, and side effects is important. The nurse teaches the patient that TB is a communicable disease and that taking medications is the most effective means of preventing transmission. The major reason treatment fails is that patients do not take their medications regularly and for the prescribed duration. This may be due to side effects or the complexity of the treatment regimen.

The nurse instructs the patient to take the medication either on an empty stomach or at least 1 hour before meals because food interferes with medication absorption (although taking medications on an empty stomach frequently results in gastrointestinal upset). Patients taking INH should avoid foods that contain tyramine and histamine (tuna, aged cheese, red wine, soy sauce, yeast extracts), because eating them while taking INH may result in headache, flushing, hypotension, lightheadedness, palpitations, and diaphoresis.

In addition, rifampin can alter the metabolism of certain other medications, making them less effective. These medications include beta-blockers, oral anticoagulants such as warfarin (Coumadin), digoxin, quinidine, corticosteroids, oral hypoglycemic agents, oral contraceptives, theophylline, and verapamil (Calan, Isoptin). This issue should be discussed with the physician and pharmacist so that medication dosages can be adjusted accordingly. The nurse informs the patient that rifampin may discolor contact lenses and that the patient may want to wear eyeglasses during treatment. The nurse monitors for other side effects of anti-TB medications, including hepatitis, neurologic changes (hearing loss, neuritis), and rash. Liver enzymes, BUN, and serum creatinine levels are monitored to detect changes in liver and kidney function. Sputum culture results are monitored for acid-fast bacilli to evaluate the effectiveness of the treatment regimen and adherence to therapy.

The nurse instructs the patient about the risk of drug resistance if the medication regimen is not strictly and continuously followed. The nurse carefully monitors vital signs and observes for spikes in temperature or changes in the patient's clinical status. Caregivers of patients who are not hospitalized are taught to monitor the patient's temperature and respiratory status. Changes in the patient's respiratory status are reported to the primary health care provider.

### *Promoting Activity and Adequate Nutrition*

Patients with TB are often debilitated from prolonged chronic illness and impaired nutritional status. The nurse plans a progressive activity schedule that focuses on increasing activity tolerance and muscle strength. Anorexia, weight loss, and malnutrition are common in patients with TB. The patient's willingness to eat may be altered by fatigue from excessive coughing; sputum production; chest pain; generalized debilitated state; or cost, if the patient has few resources. Identifying facilities (eg, shelters, soup kitchens, Meals on Wheels) that provide meals in the patient's neighborhood may increase the likelihood that the patient with limited resources and energy will have access to a more nutritious intake. A nutritional plan that allows for small, frequent meals may be required. Liquid nutritional supplements may assist in meeting basic caloric requirements.

### *Preventing Spreading of Tuberculosis Infection*

In an effort to prevent transmission of TB to others, the nurse carefully instructs the patient about important hygiene measures, including mouth care, covering the mouth and nose when coughing and sneezing, proper disposal of tissues, and handwashing. TB is a disease that must be reported to the health department so that people who have been in contact with the affected patient during the infectious stage can undergo screening and possible treatment, if indicated.

In addition to the risk of transmission of TB infection to other people, it can also be spread to other parts of the body of affected patients. Spread or dissemination of TB infection to nonpulmonary sites of the body is known as miliary TB. It is the result of invasion of the bloodstream by the tubercle bacillus. Usually it results from late reactivation of a dormant infection in the lung or elsewhere. The origin of the bacilli that enter the bloodstream is either a chronic focus that has ulcerated into a blood vessel or multitudes of miliary tubercles lining the inner surface of the thoracic duct. The organisms migrate from these foci into the bloodstream, are carried throughout the body, and disseminate throughout all tissues, with tiny miliary tubercles developing in the lungs, spleen, liver, kidneys, meninges, and other organs.

The clinical course of miliary TB may vary from an acute, rapidly progressive infection with high fever to a slowly developing process with low-grade fever, anemia, and debilitation. At first, there may be no localizing signs except an enlarged spleen and a reduced number of leukocytes. However, within a few weeks, the chest x-ray reveals small densities scattered diffusely throughout both lung fields; these are the miliary tubercles, which gradually grow.

The possibility of spread to nonpulmonary sites in the body requires careful monitoring for this very serious form of TB. The nurse monitors vital signs and observes for spikes in temperature as well as changes in renal and cognitive function. Few physical signs may be elicited on physical examination of the chest, but at this stage, the patient has a severe cough and dyspnea. Treatment of miliary TB is the same as for pulmonary TB.

## Lung Abscess

A lung abscess is necrosis of the pulmonary parenchyma caused by microbial infection (Bartlett, 2007). It is generally caused by aspiration of anaerobic bacteria. By definition, in a lung abscess, the chest x-ray demonstrates a cavity of at least 2 cm. Patients who are at risk for aspiration of foreign material and development of a lung abscess include those with impaired cough reflexes who cannot close the glottis and those with swallowing difficulties. Other at-risk patients include those with central nervous system disorders (eg, seizure, stroke), drug addiction, alcoholism, esophageal disease, or compromised immune function; patients without teeth and those receiving nasogastric tube feedings; and patients with an altered state of consciousness due to anesthesia.

### Pathophysiology

Most lung abscesses are a complication of bacterial pneumonia or are caused by aspiration of oral anaerobes into the lung. Abscesses also may occur secondary to mechanical or functional obstruction of the bronchi by a tumor, foreign body, or bronchial stenosis, or from necrotizing pneumonias, TB, pulmonary embolism (PE), or chest trauma.

Most lung abscesses are found in areas of the lung that may be affected by aspiration. The site of the lung abscess is related to gravity and is determined by position. For patients who are confined to bed, the posterior segment of an upper lobe and the superior segment of the lower lobe are the most common areas. However, atypical presentations may occur, depending on the position of the patient when the aspiration occurred.

Initially, the cavity in the lung may or may not extend directly into a bronchus. Eventually, the abscess becomes surrounded, or encapsulated, by a wall of fibrous tissue. The necrotic process may extend until it reaches the lumen of a bronchus or the pleural space and establishes communication with the respiratory tract, the pleural cavity, or both. If the bronchus is involved, the purulent contents are expectorated continuously in the form of sputum. If the pleura is involved, an empyema results. A communication or connection between the bronchus and pleura is known as a bronchopleural fistula.

The organisms frequently associated with lung abscesses are *S. aureus*, *Klebsiella*, and other gram-negative species (Bartlett, 2007). However, anaerobic organisms may also be present. The organisms vary depending on the underlying predisposing factors.

### Clinical Manifestations

The clinical manifestations of a lung abscess may vary from a mild productive cough to acute illness. Most patients have a fever and a productive cough with moderate to copious amounts of foul-smelling, sometimes bloody, sputum. The fever and cough may develop insidiously and may have been present for several weeks before diagnosis. Leukocytosis may be present. Pleurisy or dull chest pain, dyspnea, weakness, anorexia, and weight loss are common.

### Assessment and Diagnostic Findings

Physical examination of the chest may reveal dullness on percussion and decreased or absent breath sounds with an intermittent **pleural friction rub** (grating or rubbing sound) on auscultation. Crackles may be present. Confirmation of the diagnosis is made by chest x-ray, sputum culture, and, in some cases, fiberoptic bronchoscopy. The chest x-ray reveals an infiltrate with an air–fluid level. A computed tomography (CT) scan of the chest may be required to provide more detailed images of different cross-sectional areas of the lung.

### Prevention

The following measures reduce the risk of lung abscess:

- Appropriate antibiotic therapy before any dental procedures in patients who must have teeth extracted while their gums and teeth are infected
- Adequate dental and oral hygiene, because anaerobic bacteria play a role in the pathogenesis of lung abscess
- Appropriate antimicrobial therapy for patients with pneumonia

### Medical Management

The findings of the history, physical examination, chest x-ray, and sputum culture indicate the type of organism and the treatment required. Adequate drainage of the lung abscess may be achieved through postural drainage and chest physiotherapy. Patients should be assessed for an adequate cough. Some patients require insertion of a percutaneous chest catheter for long-term drainage of the abscess. Therapeutic use of bronchoscopy to drain an abscess is uncommon. A diet

high in protein and calories is necessary, because chronic infection is associated with a catabolic state, necessitating increased intake of calories and protein to facilitate healing. Surgical intervention is rare, but pulmonary resection (lobectomy) is performed if massive **hemoptysis** (coughing up of blood) occurs or if there is little or no response to medical management.

#### *Pharmacologic Therapy*

IV antimicrobial therapy depends on the results of the sputum culture and sensitivity and is administered for an extended period. Standard treatment for an anaerobic lung infection is clindamycin (Cleocin). Large IV doses are usually required, because the antibiotic must penetrate the necrotic tissue and the fluid in the abscess. The IV dose is continued until there is evidence of symptom improvement.

Long-term therapy with oral antibiotics replaces IV therapy after the patient shows signs of improvement (usually 3 to 5 days). Improvement is demonstrated by normal temperature, decreased white blood cell count, and improvement on chest x-ray (resolution of surrounding infiltrate, reduction in cavity size, absence of fluid). Oral administration of antibiotic therapy is continued for an additional 4 to 8 weeks and sometimes longer. If treatment is stopped too soon, a relapse may occur.

### Nursing Management

The nurse administers antibiotics and IV treatments as prescribed and monitors for adverse effects. Chest physiotherapy is initiated as prescribed to facilitate drainage of the abscess. The nurse teaches the patient to perform deep-breathing and coughing exercises to help expand the lungs. To ensure proper nutritional intake, the nurse encourages a diet that is high in protein and calories. The nurse also offers emotional support, because the abscess may take a long time to resolve.

#### *Promoting Home and Community-Based Care*

**Teaching Patients Self-Care**

A patient who has had surgery may return home before the wound closes entirely or with a drain or tube in place. In these cases, the nurse teaches the patient or caregivers about how to change the dressings to prevent skin excoriation and odor, how to monitor for signs and symptoms of infection, and how to care for and maintain the drain or tube. The nurse also reminds the patient to perform deep-breathing and coughing exercises every 2 hours during the day and shows caregivers how to perform chest percussion and postural drainage to facilitate expectoration of lung secretions.

**Continuing Care**

A patient whose condition requires therapy at home may need referral for home care. During home visits, the nurse assesses the patient's physical condition, nutritional status, and home environment, as well as the ability of the patient and family to carry out the therapeutic regimen. Patient teaching is reinforced, and nutritional counseling is provided with the goal of attaining and maintaining an optimal state of nutrition. To prevent relapses, the nurse emphasizes the importance of completing the antibiotic regimen and of following suggestions for rest and appropriate activity. If IV antibiotic therapy is to continue at home, the services of home care nurses may be arranged to initiate IV therapy and to evaluate its administration by the patient or family.

Although most outpatient IV therapy is administered in the home setting, the patient may visit a nearby clinic or physician's office for this treatment. In some cases, patients with lung abscess may have ignored their health. Therefore, it is important to use this opportunity to address health promotion strategies and health screening with the patient.

## PLEURAL CONDITIONS

Pleural conditions are disorders that involve the membranes covering the lungs (visceral pleura) and the surface of the chest wall (parietal pleura) or disorders affecting the pleural space.

## *Pleurisy*

### Pathophysiology

Pleurisy (pleuritis) refers to inflammation of both layers of the pleurae (parietal and visceral). Pleurisy may develop in conjunction with pneumonia or an upper respiratory tract infection, TB, or collagen disease; after trauma to the chest, pulmonary infarction, or PE; in patients with primary or metastatic cancer; and after thoracotomy. The parietal pleura has nerve endings, and the visceral pleura does not. When the inflamed pleural membranes rub together during respiration (intensified on inspiration), the result is severe, sharp, knifelike pain.

### Clinical Manifestations

The key characteristic of pleuritic pain is its relationship to respiratory movement. Taking a deep breath, coughing, or sneezing worsens the pain. Pleuritic pain is limited in distribution rather than diffuse; it usually occurs only on one side. The pain may become minimal or absent when the breath is held. It may be localized or radiate to the shoulder or abdomen. Later, as pleural fluid develops, the pain decreases.

### Assessment and Diagnostic Findings

In the early period, when little fluid has accumulated, a pleural friction rub can be heard with the stethoscope, only to disappear later as more fluid accumulates and separates the inflamed pleural surfaces. Diagnostic tests may include chest x-rays, sputum analysis, thoracentesis to obtain a specimen of pleural fluid for examination, and, less commonly, a pleural biopsy.

### Medical Management

The objectives of treatment are to discover the underlying condition causing the pleurisy and to relieve the pain. As the underlying disease (pneumonia, infection) is treated, the pleuritic inflammation usually resolves. At the same time, it is necessary to monitor for signs and symptoms of pleural effusion, such as shortness of breath, pain, assumption of a position that decreases pain, and decreased chest wall excursion.

Prescribed analgesic agents and topical applications of heat or cold provide symptomatic relief. Indomethacin (Indocin), a nonsteroidal anti-inflammatory agent, may provide pain relief while allowing the patient to take deep breaths and cough more effectively. If the pain is severe, an intercostal nerve block may be required.

### Nursing Management

Because the patient has considerable pain on inspiration, the nurse offers suggestions to enhance comfort, such as turning frequently onto the affected side to splint the chest wall and reduce the stretching of the pleurae. The nurse also teaches the patient to use the hands or a pillow to splint the rib cage while coughing.

## *Pleural Effusion*

Pleural effusion, a collection of fluid in the pleural space, is rarely a primary disease process; it is usually secondary to other diseases. Normally, the pleural space contains a small amount of fluid (5 to 15 mL), which acts as a lubricant that allows the pleural surfaces to move without friction (Fig. 23-4). Pleural effusion may be a complication of heart failure, TB, pneumonia, pulmonary infections (particularly viral infections), nephrotic syndrome, connective tissue disease, pulmonary embolus, and neoplastic tumors. The most common malignancy associated with a pleural effusion is bronchogenic carcinoma.

### Pathophysiology

In certain disorders, fluid may accumulate in the pleural space to a point at which it becomes clinically evident. This almost always has pathologic significance. The effusion can be a relatively clear fluid, or it can be bloody or purulent. An effusion of clear fluid may be a transudate or an exudate. A transudate (filtrate of plasma that moves across intact capillary walls) occurs when factors influencing the formation and reabsorption of pleural fluid are altered, usually by imbalances in hydrostatic or oncotic pressures. The finding of a transudative effusion generally implies that the pleural membranes are not diseased. A transudative effusion most commonly results from heart failure. An exudate (extravasation of fluid into tissues or a cavity) usually results from inflammation by bacterial products or tumors involving the pleural surfaces.

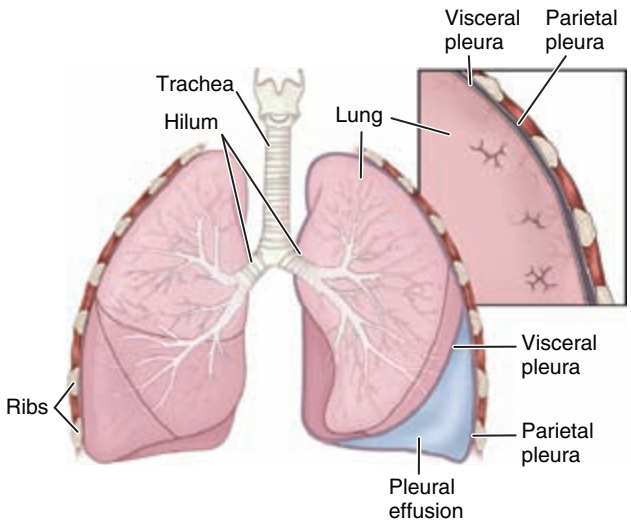


**Figure 23-4** In pleural effusion, an abnormal volume of fluid collects in the pleural space, causing pain and shortness of breath. Pleural effusion is usually secondary to other disease processes.

### Clinical Manifestations

Usually, the clinical manifestations are caused by the underlying disease. Pneumonia causes fever, chills, and pleuritic chest pain, whereas a malignant effusion may result in dyspnea, difficulty lying flat, and coughing. The severity of symptoms is determined by the size of the effusion, the speed of its formation, and the underlying lung disease. A large pleural effusion causes dyspnea (shortness of breath). A small to moderate pleural effusion causes minimal or no dyspnea.

### Assessment and Diagnostic Findings

Assessment of the area of the pleural effusion reveals decreased or absent breath sounds, decreased fremitus, and a dull, flat sound on percussion. In the case of an extremely large pleural effusion, the assessment reveals a patient in acute respiratory distress. Tracheal deviation away from the affected side may also be apparent.

Physical examination, chest x-ray, chest CT, and thoracentesis confirm the presence of fluid. In some instances, a lateral decubitus x-ray is obtained. For this x-ray, the patient lies on the affected side in a side-lying position. A pleural effusion can be diagnosed because this position allows for the "layering out" of the fluid, and an air–fluid line is visible.

Pleural fluid is analyzed by bacterial culture, Gram stain, acid-fast bacillus stain (for TB), red and white blood cell counts, chemistry studies (glucose, amylase, lactate dehydrogenase, protein), cytologic analysis for malignant cells, and pH. A pleural biopsy also may be performed as a diagnostic tool.

### Medical Management

The objectives of treatment are to discover the underlying cause of the pleural effusion; to prevent reaccumulation of fluid; and to relieve discomfort, dyspnea, and respiratory compromise. Specific treatment is directed at the underlying cause (eg, heart failure, pneumonia, cirrhosis). If the pleural fluid is an exudate, more extensive diagnostic procedures are performed to determine the cause. Treatment for the primary cause is then instituted.

Thoracentesis is performed to remove fluid, to obtain a specimen for analysis, and to relieve dyspnea and respiratory compromise (see Chapter 21). Thoracentesis may be performed under ultrasound guidance. Depending on the size of the pleural effusion, the patient may be treated by removing the fluid during the thoracentesis procedure or by inserting a chest tube connected to a water-seal drainage system or suction to evacuate the pleural space and re-expand the lung.

However, if the underlying cause is a malignancy, the effusion tends to recur within a few days or weeks. Repeated thoracenteses result in pain, depletion of protein and electrolytes, and sometimes pneumothorax. Once the

pleural space is adequately drained, a chemical pleurodesis may be performed to obliterate the pleural space and prevent reaccumulation of fluid. Pleurodesis may be performed using either a thoracoscopic approach or a chest tube. A chemically irritating agent (eg, talc or another chemical irritant) is instilled or aerosolized into the pleural space. With the chest tube approach, after the agent is instilled, the chest tube is clamped for 60 to 90 minutes and the patient is assisted to assume various positions to promote uniform distribution of the agent and to maximize its contact with the pleural surfaces. The tube is unclamped as prescribed, and chest drainage may be continued several days longer to prevent reaccumulation of fluid and to promote the formation of adhesions between the visceral and parietal pleurae.

Other treatments for pleural effusions caused by malignancy include surgical pleurectomy, insertion of a small catheter attached to a drainage bottle for outpatient management (Pleurx catheter [Denver Biomedical]), or implantation of a pleuroperitoneal shunt. A pleuroperitoneal shunt consists of two catheters connected by a pump chamber containing two one-way valves. Fluid moves from the pleural space to the pump chamber and then to the peritoneal cavity. The patient manually pumps on the reservoir daily to move fluid from the pleural space to the peritoneal space.

### Nursing Management

The nurse's role in the care of patients with a pleural effusion includes implementing the medical regimen. The nurse prepares and positions the patient for thoracentesis and offers support throughout the procedure. The nurse is responsible for making sure the thoracentesis fluid amount is recorded and sent for appropriate laboratory testing. If a chest tube drainage and water-seal system is used, the nurse is responsible for monitoring the system's function and recording the amount of drainage at prescribed intervals. Nursing care related to the underlying cause of the pleural effusion is specific to the underlying condition. Care of the patient with a chest tube is discussed in Chapter 25.

If a chest tube is inserted for talc instillation, pain management is a priority and the nurse helps the patient assume positions that are the least painful. However, frequent turning and movement are important to facilitate adequate spreading of the talc over the pleural surface. The nurse evaluates the patient's pain level and administers analgesic agents as prescribed and as needed.

If the patient is to be managed as an outpatient with a pleural catheter for drainage, the nurse educates the patient and family about management and care of the catheter and drainage system.

## *Empyema*

An empyema is an accumulation of thick, purulent fluid within the pleural space, often with fibrin development and a loculated (walled-off) area where infection is located.

### Pathophysiology

Most empyemas occur as complications of bacterial pneumonia or lung abscess. They also result from penetrating chest trauma, hematogenous infection of the pleural space, nonbacterial infections, and iatrogenic causes (after thoracic surgery or thoracentesis). At first the pleural fluid is thin, with a low leukocyte count, but it frequently progresses to a fibropurulent stage and, finally, to a stage where it encloses the lung within a thick exudative membrane (loculated empyema).

### Clinical Manifestations

The patient is acutely ill and has signs and symptoms similar to those of an acute respiratory infection or pneumonia (fever, night sweats, pleural pain, cough, dyspnea, anorexia, weight loss). If the patient is immunocompromised, the symptoms may be vague. If the patient has received antimicrobial therapy, the clinical manifestations may be less obvious.

### Assessment and Diagnostic Findings

Chest auscultation demonstrates decreased or absent breath sounds over the affected area, and there is dullness on chest percussion as well as decreased fremitus. The diagnosis is established by chest CT. Usually a diagnostic thoracentesis is performed, often under ultrasound guidance.

### Medical Management

The objectives of treatment are to drain the pleural cavity and to achieve complete expansion of the lung. The fluid is drained, and appropriate antibiotics, in large doses, are prescribed based on the causative organism. Sterilization of the empyema cavity requires 4 to 6 weeks of antibiotics. Drainage of the pleural fluid depends on the stage of the disease and is accomplished by one of the following methods:

- Needle aspiration (thoracentesis) with a thin percutaneous catheter, if the volume is small and the fluid is not too purulent or too thick
- Tube thoracostomy (chest drainage using a large-diameter intercostal tube attached to water-seal drainage [see Chapter 25]) with fibrinolytic agents instilled through the chest tube in patients with loculated or complicated pleural effusions
- Open chest drainage via thoracotomy, including potential rib resection, to remove the thickened pleura, pus, and debris and to remove the underlying diseased pulmonary tissue

With long-standing inflammation, an exudate can form over the lung, trapping it and interfering with its normal expansion. This exudate must be removed surgically (decortication). The drainage tube is left in place until the pus-filled space is obliterated completely. The complete obliteration of the pleural space is monitored by serial chest x-rays, and the patient should be informed that treatment may be long term. Patients are frequently discharged from the hospital with a chest tube in place, with instructions to monitor fluid drainage at home.

### Nursing Management

Resolution of empyema is a prolonged process. The nurse helps the patient cope with the condition and instructs the patient in lung-expanding breathing exercises to restore normal respiratory function. The nurse also provides care specific to the method of drainage of the pleural fluid (eg, needle aspiration, closed chest drainage, rib resection and

drainage). When the patient is discharged home with a drainage tube or system in place, the nurse instructs the patient and family on care of the drainage system and drain site, measurement and observation of drainage, signs and symptoms of infection, and how and when to contact the health care provider. (See Nursing Process: The Patient Undergoing Thoracic Surgery in Chapter 25.)

## Pulmonary Edema

**Pulmonary edema** is defined as abnormal accumulation of fluid in the lung tissue, the alveolar space, or both. It is a severe, life-threatening condition.

### Pathophysiology

Pulmonary edema most commonly occurs as a result of increased microvascular pressure from abnormal cardiac function. The backup of blood into the pulmonary vasculature resulting from inadequate left ventricular function causes an increased microvascular pressure, and fluid begins to leak into the interstitial space and the alveoli. Other causes of pulmonary edema are hypervolemia or a sudden increase in the intravascular pressure in the lung. One example, which may occur in the patient who has undergone pneumonectomy, is sometimes termed "flash" pulmonary edema. When one lung has been removed, all the cardiac output goes to the remaining lung. If the patient's fluid status is not monitored closely, pulmonary edema can quickly develop in the postoperative period as the patient's pulmonary vasculature attempts to adapt. A second example is called re-expansion pulmonary edema. This may result from a rapid reinflation of the lung after removal of air from a pneumothorax or evacuation of fluid from a large pleural effusion.

### Clinical Manifestations

Increasing respiratory distress, characterized by dyspnea, air hunger, and central cyanosis, is present. Patients are usually very anxious and often agitated. As the fluid leaks into the alveoli and mixes with air, a foam or froth is formed. The patient coughs up (or the nurse suctions out) these foamy, frothy, and often blood-tinged secretions. The patient experiences acute respiratory distress and may become confused.

### Assessment and Diagnostic Findings

Auscultation reveals crackles in the lung bases (especially in the posterior bases) that rapidly progress toward the apices of the lungs. These crackles are caused by the movement of air through the alveolar fluid. The chest x-ray reveals increased interstitial markings. The patient may have tachycardia. Pulse oximetry values begin to fall, and arterial blood gas analysis demonstrates worsening hypoxemia.

### Medical Management

Management focuses on correcting the underlying disorder. If the pulmonary edema is cardiac in origin, then improvement in left ventricular function is the goal. Vasodilators, inotropic medications, afterload or preload agents, or contractility medications may be administered. Additional cardiac measures (eg, intra-aortic balloon pump) may be indicated if there is no response. If the problem is fluid overload, diuretics are administered and fluids are restricted. Oxygen is administered to correct the hypoxemia; in some circumstances, intubation and mechanical ventilation are necessary. The patient is extremely anxious, and morphine is prescribed to reduce anxiety and control pain.

### Nursing Management

Nursing management includes assisting with administration of oxygen and intubation and mechanical ventilation if respiratory failure occurs. The nurse also administers medications (eg, morphine, vasodilators, inotropic medications, preload and afterload agents) as prescribed and monitors the patient's responses. Nursing management in pulmonary edema is described in more detail in Chapter 30.

## Acute Respiratory Failure

Respiratory failure is a sudden and life-threatening deterioration of the gas exchange function of the lung and indicates failure of the lungs to provide adequate oxygenation or ventilation for the blood. Acute respiratory failure is defined as a decrease in arterial oxygen tension ($PaO_2$) to less than 50 mm Hg (hypoxemia) and an increase in arterial carbon dioxide tension ($PaCO_2$) to greater than 50 mm Hg (hypercapnia), with an arterial pH of less than 7.35.

It is important to distinguish between acute and chronic respiratory failure. Chronic respiratory failure is defined as deterioration in the gas exchange function of the lung that has developed insidiously or has persisted for a long period after an episode of acute respiratory failure. The absence of acute symptoms and the presence of a chronic respiratory acidosis suggest the chronicity of the respiratory failure. Two causes of chronic respiratory failure are COPD (discussed in Chapter 24) and neuromuscular diseases (discussed in Chapter 65). Patients with these disorders develop a tolerance to the gradually worsening hypoxemia and hypercapnia. However, patients with chronic respiratory failure can develop acute failure. For example, a patient with COPD may develop an exacerbation or infection that causes additional deterioration of gas exchange. The principles of management of acute versus chronic respiratory failure are different; the following discussion is limited to acute respiratory failure.

### Pathophysiology

In acute respiratory failure, the ventilation or perfusion mechanisms in the lung are impaired. Ventilatory failure mechanisms leading to acute respiratory failure include impaired function of the central nervous system (drug overdose, head trauma, infection, hemorrhage, sleep apnea), neuromuscular dysfunction (myasthenia gravis, Guillain-Barré syndrome, amyotrophic lateral sclerosis, spinal cord trauma), musculoskeletal dysfunction (chest trauma, kyphoscoliosis, malnutrition), and pulmonary dysfunction (COPD, asthma, cystic fibrosis).

Oxygenation failure mechanisms leading to acute respiratory failure include pneumonia, acute respiratory distress syndrome, heart failure, COPD, pulmonary embolism, and **restrictive lung diseases**.

In the postoperative period, especially after major thoracic or abdominal surgery, inadequate ventilation and res-

piratory failure may occur because of several factors. During this period, for example, acute respiratory failure may be caused by the effects of anesthetic, analgesic, and sedative agents, which may depress respiration (as described earlier) or enhance the effects of opioids and lead to hypoventilation. Pain may interfere with deep breathing and coughing. A ventilation–perfusion mismatch is the usual cause of respiratory failure after major abdominal, cardiac, or thoracic surgery.

### Clinical Manifestations

Early signs are those associated with impaired oxygenation and may include restlessness, fatigue, headache, dyspnea, air hunger, tachycardia, and increased blood pressure. As the hypoxemia progresses, more obvious signs may be present, including confusion, lethargy, tachycardia, tachypnea, central cyanosis, diaphoresis, and finally respiratory arrest. Physical findings are those of acute respiratory distress, including use of accessory muscles, decreased breath sounds if the patient cannot adequately ventilate, and other findings related specifically to the underlying disease process and cause of acute respiratory failure.

### Medical Management

The objectives of treatment are to correct the underlying cause and to restore adequate gas exchange in the lung. Intubation and mechanical ventilation may be required to maintain adequate ventilation and oxygenation while the underlying cause is corrected.

### Nursing Management

Nursing management of patients with acute respiratory failure includes assisting with intubation and maintaining mechanical ventilation (described in Chapter 25). Patients are usually managed in the intensive care unit. The nurse assesses the patient's respiratory status by monitoring the level of responsiveness, arterial blood gases, pulse oximetry, and vital signs. In addition, the nurse assesses the entire respiratory system and implements strategies (eg, turning schedule, mouth care, skin care, range of motion of extremities) to prevent complications. The nurse also assesses the patient's understanding of the management strategies that are used and initiates some form of communication to enable the patient to express concerns and needs to the health care team.

Finally, the nurse addresses the problems that led to the acute respiratory failure. As the patient's status improves, the nurse assesses the patient's knowledge of the underlying disorder and provides teaching as appropriate to address the disorder.

## Acute Respiratory Distress Syndrome

**Acute respiratory distress syndrome** (ARDS) is a severe form of **acute lung injury**. This clinical syndrome is characterized by a sudden and progressive pulmonary edema, increasing bilateral infiltrates on chest x-ray, hypoxemia unresponsive to oxygen supplementation regardless of the amount of PEEP, and the absence of an elevated left atrial pressure. Patients often demonstrate reduced lung compliance. A wide range of factors are associated with the development of ARDS (Chart 23-6), including direct injury to the lungs (eg, smoke inhalation) or indirect insult to the lungs (eg, shock). ARDS has been associated with a mortality rate ranging from 25% to 58% (Hansen-Flaschen & Siegel, 2007). The major cause of death in ARDS is nonpulmonary multiple-system organ failure, often with sepsis.

**Chart 23-6 • *Etiologic Factors Related to Acute Respiratory Distress Syndrome (ARDS)***

- Aspiration (gastric secretions, drowning, hydrocarbons)
- Drug ingestion and overdose
- Hematologic disorders (disseminated intravascular coagulopathy [DIC], massive transfusions, cardiopulmonary bypass)
- Prolonged inhalation of high concentrations of oxygen, smoke, or corrosive substances
- Localized infection (bacterial, fungal, viral pneumonia)
- Metabolic disorders (pancreatitis, uremia)
- Shock (any cause)
- Trauma (pulmonary contusion, multiple fractures, head injury)
- Major surgery
- Fat or air embolism
- Systemic sepsis

### Pathophysiology

ARDS occurs as a result of diffuse alveolar damage. Inflammatory triggers initiate the release of cellular and chemical mediators, causing injury to the alveolar capillary membrane in addition to other structural damage to the lungs. Severe ventilation–perfusion mismatching occurs. Alveoli collapse because of the inflammatory infiltrate, blood, fluid, and surfactant dysfunction. Small airways are narrowed because of interstitial fluid and bronchial obstruction. Lung compliance may markedly decrease, resulting in decreased functional residual capacity and severe hypoxemia. The blood returning to the lung for gas exchange is pumped through the nonventilated, nonfunctioning areas of the lung, causing shunting. This means that blood is interfacing with nonfunctioning alveoli and gas exchange is markedly impaired, resulting in severe, refractory hypoxemia. Figure 23-5 shows the sequence of pathophysiologic events leading to ARDS.

### Clinical Manifestations

By definition, ARDS is an acute event that typically develops over 4 to 48 hours (Hansen-Flaschen & Siegel, 2007). Initially, ARDS closely resembles severe hemodynamic pulmonary edema. The acute phase of ARDS is marked by a rapid onset of severe dyspnea that usually occurs 12 to 48 hours after the initiating event. Arterial hypoxemia that does not respond to supplemental oxygen is characteristic. Findings on chest x-ray are similar to those seen with cardiogenic pulmonary edema and are visible as bilateral infiltrates that quickly worsen. The acute lung injury then progresses to fibrosing alveolitis with persistent, severe hypoxemia. The patient also has increased alveolar dead space (ventilation to alveoli, but poor perfusion) and decreased pulmonary compliance ("stiff lungs," which are difficult to ventilate).

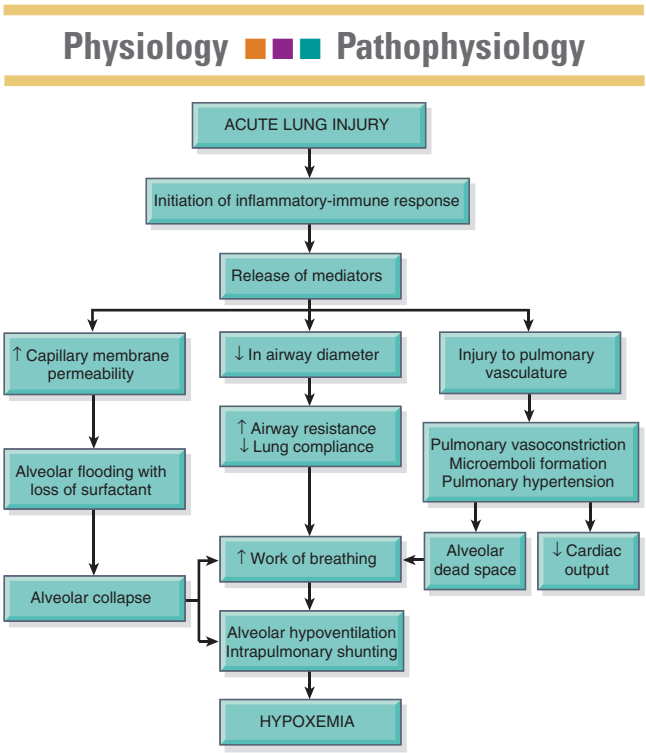


**Figure 23-5** Pathogenesis and pathophysiology of acute respiratory distress syndrome.

Clinically, the patient is thought to be in the recovery phase if the hypoxemia gradually resolves, the chest x-ray improves, and the lungs become more compliant.

## Assessment and Diagnostic Findings

On physical examination, intercostal retractions and crackles may be present as the fluid begins to leak into the alveolar interstitial space. Common diagnostic tests performed in patients with potential ARDS include plasma brain natriuretic peptide (BNP) levels, echocardiography, and pulmonary artery catheterization. The BNP level is helpful in distinguishing ARDS from hemodynamic pulmonary edema. Transthoracic echocardiography may be used if the BNP is not conclusive. Pulmonary artery catheterization is the definitive method to distinguish between hemodynamic (heart failure) and permeability pulmonary edema (ARDS) (Hansen-Flaschen & Siegel, 2007).

## Medical Management

The primary focus in the management of ARDS includes identification and treatment of the underlying condition. Aggressive, supportive care must be provided to compensate for the severe respiratory dysfunction. This supportive therapy almost always includes intubation and mechanical ventilation. In addition, circulatory support, adequate fluid volume, and nutritional support are important. Supplemental oxygen is used as the patient begins the initial spiral of hypoxemia. As the hypoxemia progresses, intubation and mechanical ventilation are instituted. The concentration of oxygen and ventilator settings and modes are determined by the patient's status. This is monitored by arterial blood gas analysis, pulse oximetry, and bedside pulmonary function testing.

PEEP is a critical part of the treatment of ARDS. PEEP usually improves oxygenation, but it does not influence the natural history of the syndrome. Use of PEEP helps increase functional residual capacity and reverse alveolar collapse by keeping the alveoli open, resulting in improved arterial oxygenation and a reduction in the severity of the ventilation–perfusion imbalance. By using PEEP, a lower $FiO_2$ may be required. The goal is a $PaO_2$ greater than 60 mm Hg or an oxygen saturation level of greater than 90% at the lowest possible $FiO_2$. PEEP and modes of mechanical ventilation are discussed in Chapter 25.

Systemic hypotension may occur in ARDS as a result of hypovolemia secondary to leakage of fluid into the interstitial spaces and depressed cardiac output from high levels of PEEP therapy. Hypovolemia must be carefully treated without causing further overload. Inotropic or vasopressor agents may be required. Pulmonary artery pressure catheters are used to monitor the patient's fluid status and the severe and progressive pulmonary hypertension sometimes observed in ARDS.

### Pharmacologic Therapy

There is no specific pharmacologic treatment for ARDS except supportive care. Numerous pharmacologic treatments are under investigation to stop the cascade of events leading to ARDS. These include surfactant replacement therapy, pulmonary antihypertensive agents, and antisepsis agents.

### Nutritional Therapy

Adequate nutritional support is vital in the treatment of ARDS. Patients with ARDS require 35 to 45 kcal/kg/day to meet caloric requirements. Enteral feeding is the first consideration; however, parenteral nutrition also may be required.

## Nursing Management

### General Measures

A patient with ARDS is critically ill and requires close monitoring in the intensive care unit, because the patient's condition could quickly become life-threatening. Most of the respiratory modalities discussed in Chapter 25 are used in this situation (oxygen administration, nebulizer therapy, chest physiotherapy, endotracheal intubation or tracheostomy, mechanical ventilation, suctioning, bronchoscopy). Frequent assessment of the patient's status is necessary to evaluate the effectiveness of treatment.

In addition to implementing the medical plan of care, the nurse considers other needs of the patient. Positioning is important. The nurse turns the patient frequently to improve ventilation and perfusion in the lungs and enhance secretion drainage. However, the nurse must closely monitor the patient for deterioration in oxygenation with changes in position. Oxygenation in patients with ARDS is sometimes improved in the prone position. This position may be evaluated for improvement in oxygenation and used in special circumstances. Devices and specialty beds are available to assist the nurse in placing the patient in a prone position.

The patient is extremely anxious and agitated because of the increasing hypoxemia and dyspnea. It is important to

reduce the patient's anxiety because anxiety increases oxygen expenditure by preventing rest. Rest is essential to limit oxygen consumption and reduce oxygen needs.

### Ventilator Considerations

If the patient is intubated and receiving mechanical ventilation with PEEP, several considerations must be addressed. PEEP, which causes increased end-expiratory pressure, is an unnatural pattern of breathing and feels strange to patients. The patients may be anxious and "fight" the ventilator. Nursing assessment is important to identify problems with ventilation that may be causing the anxiety reaction: tube blockage by kinking or retained secretions; other acute respiratory problems (eg, pneumothorax, pain); a sudden decrease in the oxygen level; the level of dyspnea; or ventilator malfunction. In some cases, sedation may be required to decrease the patient's oxygen consumption, allow the ventilator to provide full support of ventilation, and decrease the patient's anxiety. Sedatives that may be used are lorazepam (Ativan), midazolam (Versed), dexmedetomidine (Precedex), propofol (Diprivan), and short-acting barbiturates.

If the PEEP level cannot be maintained despite the use of sedatives, neuromuscular blocking agents (paralytic agents) may be administered to paralyze the patient. Examples of these agents include pancuronium (Pavulon), vecuronium (Norcuron), atracurium (Tracrium), and rocuronium (Zemuron). The resulting paralysis allows the patient to be ventilated more easily. With paralysis, the patient appears to be unconscious; loses motor function; and cannot breathe, talk, or blink independently. However, the patient retains sensation and is awake and able to hear. The nurse must reassure the patient that the paralysis is a result of the medication and is temporary. Paralysis should be used for the shortest possible time and never without adequate sedation and pain management.

Use of paralytic agents has many dangers and side effects. The nurse must be sure the patient does not become disconnected from the ventilator, because respiratory muscles are paralyzed and the patient will be apneic. Consequently, the nurse ensures that the patient is closely monitored, and all ventilator and patient alarms must be on at all times. Eye care is important as well, because the patient cannot blink, increasing the risk of corneal abrasions. Neuromuscular blockers predispose the patient to deep venous thrombi, muscle atrophy, and skin breakdown. Nursing assessment is essential to minimize the complications related to neuromuscular blockade. The patient may have discomfort or pain but cannot communicate these sensations. Analgesia is usually administered concurrently with neuromuscular blocking agents. The nurse must anticipate the patient's needs regarding pain and comfort. The nurse checks the patient's position to ensure it is comfortable and in normal alignment and talks to, and not about, the patient while in the patient's presence.

In addition, it is important for the nurse to describe the purpose and effects of the paralytic agents to the patient's family. If family members are unaware that these agents have been administered, they may become distressed by the change in the patient's status.

## Pulmonary Arterial Hypertension

Pulmonary arterial hypertension exists when the mean pulmonary artery pressure exceeds 25 mm Hg with a pulmonary capillary wedge pressure of less than 15 mm Hg (Badesch, Abman, Simonneau, et al., 2007). Unlike systemic blood pressure, these pressures cannot be measured indirectly; instead, they must be measured directly during right-sided heart catheterization. In the absence of these measurements, clinical recognition becomes the only indicator of pulmonary hypertension. However, pulmonary arterial hypertension is a condition that is not clinically evident until late in its progression.

There are two types of pulmonary arterial hypertension: idiopathic (or primary) pulmonary arterial hypertension and pulmonary arterial hypertension due to a known cause (Badesch, et al., 2007). It occurs most often in women 20 to 40 years of age, either sporadically or in patients with a family history, and is usually fatal within 5 years of diagnosis. There are several possible causes, but the exact cause is unknown (Chart 23-7). The clinical presentation may occur with no evidence of pulmonary or cardiac disease.

In contrast, pulmonary arterial hypertension due to a known cause is more common and results from existing cardiac or pulmonary disease. The prognosis depends on the severity of the underlying disorder and the changes in the pulmonary vascular bed. A common cause of pulmonary arterial hypertension is pulmonary artery constriction due to hypoxemia from COPD (cor pulmonale), which is discussed below.

### Pathophysiology

Conditions such as collagen vascular disease, congenital heart disease, anorexigens (specific appetite depressants), chronic use of stimulants, portal hypertension, and HIV infection increase the risk of pulmonary arterial hypertension in susceptible patients. Vascular injury occurs with endothelial dysfunction and vascular smooth muscle dysfunction, which leads to disease progression (vascular smooth

**Chart 23-7 • *Causes of Pulmonary Arterial Hypertension***

**Idiopathic (Primary) Arterial Hypertension and Pulmonary Arterial Hypertension Due to a Known Cause**

- Collagen vascular diseases
- Congenital systemic-to-pulmonary shunts
- Portal hypertension
- Altered immune mechanisms (HIV infection)
- Diseases associated with significant venous or capillary involvement
- Chronic thrombotic or embolic disease
- Pulmonary venous hypertension
- Pulmonary vasoconstriction due to hypoxemia
- Chronic obstructive pulmonary disease (COPD), interstitial lung disease, sleep-disordered breathing
- Miscellaneous causes: sarcoidosis, histiocytosis, compression of pulmonary vessels

muscle hypertrophy, adventitial and intimal proliferation [thickening of the wall], and advanced vascular lesion formation). Normally, the pulmonary vascular bed can handle the blood volume delivered by the right ventricle. It has a low resistance to blood flow and compensates for increased blood volume by dilation of the vessels in the pulmonary circulation. However, if the pulmonary vascular bed is destroyed or obstructed, as in pulmonary hypertension, the ability to handle whatever flow or volume of blood it receives is impaired, and the increased blood flow then increases the pulmonary artery pressure. As the pulmonary arterial pressure increases, the pulmonary vascular resistance also increases. Both pulmonary artery constriction (as in hypoxemia or hypercapnia) and a reduction of the pulmonary vascular bed (which occurs with pulmonary emboli) result in increased pulmonary vascular resistance and pressure. This increased workload affects right ventricular function. The myocardium ultimately cannot meet the increasing demands imposed on it, leading to right ventricular hypertrophy (enlargement and dilation) and failure. Passive hepatic congestion may also develop.

## Clinical Manifestations

Dyspnea, the main symptom of pulmonary hypertension, occurs at first with exertion and eventually at rest. Substernal chest pain also is common. Other signs and symptoms include weakness, fatigue, syncope, occasional hemoptysis, and signs of right-sided heart failure (peripheral edema, ascites, distended neck veins, liver engorgement, crackles, heart murmur). Anorexia and abdominal pain in the right upper quadrant may also occur.

## Assessment and Diagnostic Findings

Several tests are used to determine if there is a known cause for the pulmonary hypertension. If the diagnostic tests and thorough evaluation of the patient reveal no known cause, a diagnosis of primary pulmonary hypertension is made. Complete diagnostic evaluation includes a history, physical examination, chest x-ray, pulmonary function studies, electrocardiogram (ECG), echocardiogram, ventilation–perfusion scan, sleep studies, autoantibody tests (to identify diseases of collagen vascular origin), HIV tests, liver function testing, and cardiac catheterization. Pulmonary function studies may be normal or show a slight decrease in vital capacity and lung compliance, with a mild decrease in the diffusing capacity. The $PaO_2$ also is decreased (hypoxemia). The ECG reveals right ventricular hypertrophy, right axis deviation, and tall peaked P waves in inferior leads; tall anterior R waves; and ST-segment depression, T-wave inversion, or both anteriorly. An echocardiogram can assess the progression of the disease and rule out other conditions with similar signs and symptoms. A ventilation–perfusion scan or pulmonary angiography detects defects in pulmonary vasculature, such as pulmonary emboli. Cardiac catheterization of the right side of the heart reveals elevated pulmonary arterial pressure and determines whether there is a vasoactive component to the pulmonary hypertension.

## Medical Management

The goal of treatment is to manage the underlying condition related to pulmonary hypertension of known cause. Recommendations regarding therapy are tailored to the patient's individual situation, functional New York Heart Association class, and specific needs (Badesch, et al., 2007). Anticoagulation should be considered for patients with pulmonary hypertension and patients with an indwelling catheter for administration of medications. Most patients with pulmonary hypertension do not have hypoxemia at rest but require supplemental oxygen with exercise. Diuretics and oxygen should be added as needed. Appropriate oxygen therapy (see Chapter 25) reverses the vasoconstriction and reduces the pulmonary hypertension in a relatively short time.

Different classes of medications are used to treat pulmonary hypertension; these include calcium channel blockers, phosphodiesterase-5 inhibitors, endothelin antagonists, and prostanoids. The choice of therapeutic agents is based on the severity of the disease.

A small number of patients with pulmonary hypertension respond favorably to acute vasodilation and do well with a calcium channel blocking agent. However, because calcium channel blockers are effective in only a small percentage of patients, other treatment options, including prostacyclin, are often necessary (Badesch, et al., 2007).

The oral medication sildenafil (Revatio, Viagra) is a potent specific phosphodiesterase-5 inhibitor that degrades cyclic 3′,5′-guanosine monophosphate (cGMP) and promotes pulmonary vasodilation.

Bosentan (Tracleer), an endothelin receptor antagonist, causes vasodilation and is prescribed for its antihypertensive effects in patients with pulmonary hypertension. It is administered orally twice a day. Liver function must be monitored in patients using bosentan. Other endothelin receptor antagonists are sitaxsentan (Thelin) and ambrisentan (Letairis).

Prostanoids mimic prostaglandin. Prostaglandin (prostacyclin) relaxes vascular smooth muscle by stimulating the production of cyclic 3′,5′-adenosine monophosphate (AMP) and inhibiting the growth of smooth muscle cells. Epoprostenol (Flolan), treprostinil (Remodulin), and iloprost (Ventavis) are examples of prostanoids. Because of its short half-life in the circulation (ie, 3 minutes), epoprostenol can be administered only by continuous IV infusion and requires training of the patient and caregiver. Treprostinil has a longer half-life than epoprostenol and is subcutaneously infused. Iloprost is more stable than the other two prostanoids and is inhaled. The frequency of dosing is six to eight times per day.

Lung transplantation remains an option for all eligible patients who have severe disease and symptoms after 3 months of receiving epoprostenol. Atrial septostomy may be considered for selected patients with severe disease (Badesch, et al., 2007); this procedure results in shunting of blood from the right side of the heart to the left, decreasing the strain on the right side of the heart and maintaining left ventricular output.

## Nursing Management

The major nursing goal is to identify patients at high risk for pulmonary arterial hypertension, such as those with COPD, pulmonary emboli, congenital heart disease, and mitral valve disease. The nurse must be alert for signs and symptoms, administer oxygen therapy appropriately,

and instruct the patient and family about the use of home oxygen therapy. In patients treated with prostanoids (ie, epoprostenol or treprostinil), education about the need for central venous access (epoprostenol), subcutaneous infusion (treprostinil), proper administration and dosing of the medication, pain at the injection site, and potential severe side effects is extremely important. Emotional and psychosocial aspects of this disease must be addressed. Formal and informal support groups for patients and families are extremely valuable.

## *Pulmonary Heart Disease (Cor Pulmonale)*

**Cor pulmonale** is a condition in which the right ventricle of the heart enlarges (with or without right-sided heart failure) as a result of diseases that affect the structure or function of the lung or its vasculature. It is a type of pulmonary arterial hypertension due to a known cause. Any disease affecting the lungs and accompanied by hypoxemia may result in cor pulmonale. The most frequent cause is severe COPD (see Chapter 24), in which changes in the airway and retained secretions reduce alveolar ventilation. Other causes are conditions that restrict or compromise ventilatory function, leading to hypoxemia or acidosis (eg, deformities of the thoracic cage, massive obesity) and conditions that reduce the pulmonary vascular bed (eg, primary idiopathic pulmonary arterial hypertension, pulmonary embolus). Certain disorders of the nervous system, respiratory muscles, chest wall, and pulmonary arterial tree also may be responsible for cor pulmonale.

### Pathophysiology

Pulmonary disease can produce physiologic changes that in time affect the heart and cause the right ventricle to enlarge and eventually fail. Any condition that deprives the lungs of oxygen can cause hypoxemia and hypercapnia, resulting in ventilatory insufficiency. Hypoxemia and hypercapnia cause pulmonary arterial vasoconstriction and possibly reduction of the pulmonary vascular bed, as in emphysema or pulmonary emboli. The result is increased resistance in the pulmonary circulatory system, with a subsequent rise in pulmonary blood pressure (pulmonary hypertension). A mean pulmonary arterial pressure of 45 mm Hg or more may occur in cor pulmonale. Right ventricular hypertrophy may result, followed by right ventricular failure. In short, cor pulmonale results from pulmonary hypertension, which causes the right side of the heart to enlarge because of the increased work required to pump blood against high resistance through the pulmonary vascular system.

### Clinical Manifestations

Symptoms of cor pulmonale are usually related to the underlying lung disease, such as COPD. With right ventricular failure, the patient may develop increasing edema of the feet and legs, distended neck veins, an enlarged palpable liver, pleural effusion, ascites, and heart murmurs. Headache, confusion, and somnolence may occur as a result of increased levels of carbon dioxide (hypercapnia). Patients often complain of increasing shortness of breath, wheezing, cough, and fatigue.

### Medical Management

The objectives of treatment are to improve ventilation and to treat both the underlying lung disease and the manifestations of heart disease. Supplemental oxygen is administered to improve gas exchange and to reduce pulmonary arterial pressure and pulmonary vascular resistance. Improved oxygen transport relieves the pulmonary hypertension that is causing the cor pulmonale.

Continuous, 24-hour oxygen therapy in patients with severe hypoxemia reportedly leads to better survival rates and greater reduction in pulmonary vascular resistance. Substantial improvement may require 4 to 6 weeks of oxygen therapy, usually in the home. Periodic assessment of pulse oximetry and arterial blood gases is necessary to determine the adequacy of alveolar ventilation and to monitor the effectiveness of oxygen therapy.

Chest physical therapy and bronchial hygiene maneuvers as indicated to remove accumulated secretions and the administration of bronchodilators further improve ventilation. Additional measures depend on the patient's condition. If the patient is in respiratory failure, endotracheal intubation and mechanical ventilation may be necessary. If the patient is in heart failure, hypoxemia and hypercapnia must be relieved to improve cardiac function and output. Bed rest, sodium restriction, and diuretic therapy also are instituted judiciously to reduce peripheral edema (ie, to lower pulmonary arterial pressure through a decrease in total blood volume) and the circulatory load on the right side of the heart. Digitalis may be prescribed to relieve pulmonary hypertension if the patient also has left ventricular failure, a supraventricular dysrhythmia, or right ventricular failure that does not respond to other therapy.

ECG monitoring may be indicated because of the high incidence of dysrhythmias in patients with cor pulmonale. Any respiratory infection must be treated promptly to avoid further impaired gas exchange and exacerbations of hypoxemia and pulmonary heart disease. The prognosis depends on whether the pulmonary hypertension is reversible. (See earlier discussion of the management of acute respiratory failure.)

### Nursing Management

Nursing care addresses the underlying disorder leading to cor pulmonale as well as the problems related to pulmonary hyperventilation and right-sided cardiac failure. If intubation and mechanical ventilation are required to manage acute respiratory failure, the nurse assists with the intubation procedure and maintains mechanical ventilation. The nurse assesses the patient's respiratory and cardiac status and administers medications as prescribed.

During the patient's hospital stay, the nurse instructs the patient about the importance of close self-monitoring (fluid retention, weight gain, edema) and adherence to the therapeutic regimen, especially the 24-hour use of oxygen. It is important to address factors that affect the patient's adherence to the treatment regimen.

### *Promoting Home and Community-Based Care*

#### Teaching Patients Self-Care

Because cor pulmonale is a chronic disorder, most of the care and monitoring of patients with cor pulmonale is performed by patients and families in the home. If supplemental oxygen is administered, the nurse instructs the patient and family in its safe and correct use. Nutrition counseling is warranted if the patient is on a sodium-restricted diet or is taking diuretics. The nurse teaches the family to monitor for signs and symptoms of right ventricular failure and about emergency interventions and when to call for assistance. Most importantly, the nurse works with the patient to stop smoking.

#### Continuing Care

Referral for home care may be warranted for patients who cannot manage self-care and for those whose physical condition warrants close assessment. During home visits, the home care nurse evaluates the patient's status and the patient's and family members' understanding of the therapeutic regimen and their adherence to it. If oxygen is used in the home, the nurse determines whether it is being administered safely and as prescribed. It is important to assess the patient's progress in stopping smoking and to reinforce the importance of smoking cessation with the patient and family. The nurse identifies strategies to assist with smoking cessation and refers the patient and family to community support groups. In addition, the nurse reminds the patient about the importance of other health promotion and screening practices.

## Pulmonary Embolism

**Pulmonary embolism** (PE) refers to the obstruction of the pulmonary artery or one of its branches by a thrombus (or thrombi) that originates somewhere in the venous system or in the right side of the heart. Deep venous thrombosis (DVT), a related condition, refers to thrombus formation in the deep veins, usually in the calf or thigh, but sometimes in the arm, especially in patients with peripherally inserted central catheters. DVT is discussed in detail in Chapter 31. Venous thromboembolism (VTE) is a term that includes both DVT and PE.

PE is a common disorder and often is associated with trauma, surgery (orthopedic, major abdominal, pelvic, gynecologic), pregnancy, heart failure, age older than 50 years, hypercoagulable states, and prolonged immobility. It also may occur in apparently healthy people. An estimated 237,000 nonfatal and 294,000 fatal cases of PE occur in the United States each year (AlMahmeed & Carman, 2007). Risk factors for PE are identified in Chart 23-8.

### Pathophysiology

Most commonly, PE is due to a blood clot or thrombus. However, there are other types of emboli: air, fat, amniotic fluid, and septic (from bacterial invasion of the thrombus).

When a thrombus completely or partially obstructs a pulmonary artery or its branches, the alveolar dead space is increased. The area, although continuing to be ventilated, receives little or no blood flow. Therefore, gas exchange is impaired or absent in this area. In addition, various substances are released from the clot and surrounding area that cause regional blood vessels and bronchioles to constrict. This results in an increase in pulmonary vascular resistance. This reaction compounds the ventilation–perfusion imbalance.

The hemodynamic consequences are increased pulmonary vascular resistance due to the regional vasoconstriction and reduced size of the pulmonary vascular bed. This results in an increase in pulmonary arterial pressure and, in turn, an increase in right ventricular work to maintain pulmonary blood flow. When the work requirements of the right ventricle exceed its capacity, right ventricular failure occurs, leading to a decrease in cardiac output followed by a decrease in systemic blood pressure and the development of shock. Atrial fibrillation also causes PE. An enlarged right atrium in fibrillation causes blood to stagnate and form clots in this area. These clots are prone to travel into the pulmonary circulation.

A massive PE is best defined by the degree of hemodynamic instability rather than the percentage of pulmonary vasculature occlusion. It is described as an occlusion of the outflow tract of the main pulmonary artery or of the bifurcation of the pulmonary arteries. Multiple small emboli

**CHART 23-8 *Risk Factors for Pulmonary Embolus***

**Venous Stasis (slowing of blood flow in veins)**

- Prolonged immobilization (especially postoperative)
- Prolonged periods of sitting/traveling
- Varicose veins
- Spinal cord injury

**Hypercoagulability (due to release of tissue thromboplastin after injury/surgery)**

- Injury
- Tumor (pancreatic, gastrointestinal, genitourinary, breast, lung)
- Increased platelet count (polycythemia, splenectomy)

**Venous Endothelial Disease**

- Thrombophlebitis
- Vascular disease
- Foreign bodies (IV/central venous catheters)

**Certain Disease States (combination of stasis, coagulation alterations, and venous injury)**

- Heart disease (especially heart failure)
- Trauma (especially fracture of hip, pelvis, vertebra, lower extremities)
- Postoperative state/postpartum period
- Diabetes mellitus
- Chronic obstructive pulmonary disease (COPD)

**Other Predisposing Conditions**

- Advanced age
- Obesity
- Pregnancy
- Oral contraceptive use
- History of previous thrombophlebitis, pulmonary embolism
- Constrictive clothing

can lodge in the terminal pulmonary arterioles, producing multiple small infarctions of the lungs. A pulmonary infarction causes ischemic necrosis of part of the lung.

## Clinical Manifestations

Symptoms of PE depend on the size of the thrombus and the area of the pulmonary artery occluded by the thrombus; they may be nonspecific. Dyspnea is the most frequent symptom; the duration and intensity of the dyspnea depend on the extent of embolization. Chest pain is common and is usually sudden and pleuritic in origin. It may be substernal and may mimic angina pectoris or a myocardial infarction. Other symptoms include anxiety, fever, tachycardia, apprehension, cough, diaphoresis, hemoptysis, and syncope. The most frequent sign is tachypnea (very rapid respiratory rate).

The clinical picture may mimic that of bronchopneumonia or heart failure. In atypical instances, PE causes few signs and symptoms, whereas in other instances it mimics various other cardiopulmonary disorders. Obstruction of the pulmonary artery results in pronounced dyspnea, sudden substernal pain, rapid and weak pulse, shock, syncope, and sudden death.

## Assessment and Diagnostic Findings

Death from PE commonly occurs within 1 hour after the onset of symptoms; therefore, early recognition and diagnosis are priorities. Because the symptoms of PE can vary from few to severe, a diagnostic workup is performed to rule out other diseases. The initial diagnostic workup may include chest x-ray, ECG, arterial blood gas analysis, and ventilation–perfusion ($\dot{V}/\dot{Q}$) scan. The chest x-ray is usually normal but may show infiltrates, atelectasis, elevation of the diaphragm on the affected side, or a pleural effusion. The chest x-ray is most helpful in excluding other possible causes. The ECG usually shows sinus tachycardia, PR-interval depression, and nonspecific T-wave changes. Arterial blood gas analysis may show hypoxemia and hypocapnia (from tachypnea); however, arterial blood gas measurements may be normal even in the presence of PE.

Pulmonary angiography is considered the best method to diagnose PE; however, it may not be feasible, cost-effective, or easily performed, especially with critically ill patients. The pulmonary angiogram allows for direct visualization under fluoroscopy of the arterial obstruction and accurate assessment of the perfusion deficit. A specially trained team must be available to perform the procedure, in which a catheter is threaded through the vena cava to the right side of the heart to inject dye, similar to a cardiac catheterization.

The $\dot{V}/\dot{Q}$ scan continues to be used to diagnose PE, especially in facilities that do not use pulmonary angiography or do not have access to a spiral CT scanner. The $\dot{V}/\dot{Q}$ scan is minimally invasive, involving the IV administration of a contrast agent. This scan evaluates different regions of the lung (upper, middle, lower) and allows comparisons of the percentage of ventilation and perfusion in each area. This test has a high sensitivity but can be more cumbersome than a CT scan and is not as accurate as a pulmonary angiogram.

A high suspicion of PE may warrant a spiral computed CT scan of the lung, D-dimer assay (blood test for evidence of blood clots), and pulmonary arteriogram. Spiral CT of the chest may also assist in the diagnosis. In spiral CT, the examination table advances at a constant rate through the scanner while the x-ray tube rotates continuously around the patient, following a spiral path, thus allowing the gathering of continuous data with no gaps between images. Unlike the traditional CT scan, the spiral CT scan evaluates slices as narrow as 1.0 mm, as compared with 5.0-mm slices obtained by traditional CT scan. This allows for a more accurate visualization of a PE. However, spiral CT has limitations. It cannot be performed at the bedside, so unstable patients must be transported to a CT scanner. In addition, IV infusion of contrast agent is necessary for visualization.

## Prevention

For patients at risk for PE, the most effective approach for prevention is to prevent DVT. Active leg exercises to avoid venous stasis, early ambulation, and use of anti-embolism stockings are general preventive measures. Mechanical prophylaxis can be classified as either static or dynamic. Graduated compression stockings of either thigh or knee length are considered static devices; there is little evidence to support their use. Problematic issues include inappropriate application and inappropriate fit of the stockings. Sequential compression devices (SCDs), plastic sleeves that can be inflated with air, are dynamic and are used to prevent venous stasis through compression and relaxation of the calf muscles, as in muscle contraction. SCDs have been proven to successfully reduce the risk of DVT and have been shown to be an effective therapy when used in combination with pharmacologic prophylaxis (Limpus, Chaboyer, McDonald, et al., 2006). Several types of SCDs are used as sources of compression and use foot, calf, and thigh-high compression, as well as graduated, asymmetric, and circumferential compression. Little evidence supports any particular type of compression. Graduated compression involves the sequential movement of air in the sleeve up the leg, followed by relaxation of the sleeve. The advantage of this therapy is the extended duration of compression compared with standard inflation. Asymmetric compression involves inflating only the area on the back of the leg or foot. Circumferential compression involves even compression of the entire leg. Other interventions for preventing DVT and PE are shown in Chart 23-9.

Several organizations publish suggested regimens for the prevention of VTE in surgical patients. These recommendations include both nonpharmacologic (mechanical) and pharmacologic strategies. Patients are stratified by risk of DVT by type of surgical procedure and risk of VTE. The most widely used guidelines are those from the American College of Chest Physicians (2008). Surgical patients are the primary target for prevention of VTE, although all hospitalized patients are at risk. Anticoagulant therapy may be prescribed for patients whose hemostasis is adequate and who are undergoing major elective abdominal or thoracic surgery.

## Medical Management

Because PE is often a medical emergency, emergency management is of primary concern. After emergency measures have been initiated and the patient is stabilized, the treatment goal is to dissolve (lyse) the existing emboli

CHART 23-9 HOME CARE CHECKLIST

## *Prevention of Recurrent Pulmonary Embolism*

| At the completion of the home care instruction, the patient or caregiver will be able to: | PATIENT | CAREGIVER |
|---|---|---|
| • Describe the underlying process leading to pulmonary embolism. | ✔ | ✔ |
| • Describe the need for continued anticoagulant therapy after the initial embolism. | ✔ | ✔ |
| • Name the anticoagulant prescribed and identify dosage and schedule of administration. | ✔ | ✔ |
| • Describe potential side effects of coagulation such as bruising and bleeding and identify ways to prevent bleeding:<br>  • Avoid the use of sharps (razors, knives, etc.) to prevent cuts; shave with an electric shaver.<br>  • Use a toothbrush with soft bristles to prevent gum injury.<br>  • Do not take aspirin or antihistamines while taking warfarin sodium (Coumadin).<br>  • Always check with health care provider before taking any medicine, including over-the-counter medications.<br>  • Avoid laxatives, because they may affect vitamin K absorption.<br>  • Report the occurrence of dark, tarry stools to the health care provider immediately.<br>  • Wear an identification bracelet or carry a medicine card stating that you are taking anticoagulants. | ✔ | ✔ |
| • Describe strategies to prevent recurrent deep venous thrombosis and pulmonary emboli:<br>  • Continue to wear anti-embolism stockings (compression hose) as long as directed.<br>  • Avoid sitting with legs crossed or sitting for prolonged periods of time.<br>  • When traveling, change position regularly, walk occasionally, and do active exercises of moving the legs and ankles while sitting.<br>  • Drink fluids, especially while traveling and in warm weather, to avoid hemoconcentration due to fluid deficit. | ✔ | ✔ |
| • Describe the signs and symptoms of lower extremity circulatory compromise and potential deep venous thrombosis: calf or leg pain, swelling, pedal edema. | ✔ | ✔ |
| • Describe the signs and symptoms of pulmonary compromise related to recurrent pulmonary embolism. | ✔ | ✔ |
| • Describe how and when to contact the health care provider if symptoms of circulatory compromise or pulmonary compromise are identified. | ✔ | ✔ |

and prevent new ones from forming. Treatment may include a variety of modalities: general measures to improve respiratory and vascular status, anticoagulation therapy, thrombolytic therapy, and surgical intervention.

### *Emergency Management*

Massive PE is a life-threatening emergency. The immediate objective is to stabilize the cardiopulmonary system. A sudden increase in pulmonary resistance increases the work of the right ventricle, which can cause acute right-sided heart failure with cardiogenic shock. Emergency management consists of the following actions:

- Nasal oxygen is administered immediately to relieve hypoxemia, respiratory distress, and central cyanosis.
- Intravenous infusion lines are inserted to establish routes for medications or fluids that will be needed.
- A perfusion scan, hemodynamic measurements, and arterial blood gas determinations are performed. Spiral (helical) CT or pulmonary angiography may be performed.
- Hypotension is treated by a slow infusion of dobutamine (Dobutrex), which has a dilating effect on the pulmonary vessels and bronchi, or dopamine (Intropin).
- The ECG is monitored continuously for dysrhythmias and right ventricular failure, which may occur suddenly.
- Digitalis glycosides, IV diuretics, and antiarrhythmic agents are administered when appropriate.
- Blood is drawn for serum electrolytes, complete blood count, and hematocrit.
- If clinical assessment and arterial blood gas analysis indicate the need, the patient is intubated and placed on a mechanical ventilator.
- If the patient has suffered massive embolism and is hypotensive, an indwelling urinary catheter is inserted to monitor urinary output.
- Small doses of IV morphine or sedatives are administered to relieve patient anxiety, to alleviate chest discomfort, to improve tolerance of the endotracheal tube, and to ease adaptation to the mechanical ventilator.

### *General Management*

Measures are initiated to improve respiratory and vascular status. Oxygen therapy is administered to correct the hypoxemia, relieve the pulmonary vascular vasoconstriction, and reduce the pulmonary hypertension. Use of anti-embolism stockings or intermittent pneumatic leg compression devices reduces venous stasis. These measures compress the superficial veins and increase the velocity of blood

in the deep veins by redirecting the blood through the deep veins. Elevating the leg (above the level of the heart) also increases venous flow.

### Pharmacologic Therapy

#### Anticoagulation Therapy

Anticoagulant therapy (heparin, warfarin sodium [Coumadin]) has traditionally been the primary method for managing acute DVT and PE. Unfractionated and low-molecular-weight heparins may be used to prevent recurrence of emboli, but they have no effect on emboli that are already present. Heparin is generally recommended for patients who have been diagnosed with PE; numerous specific options for treatment are available.

Unfractionated heparin administration is dosed by actual body weight and is associated with several concerns. Because the half-life of heparin is dose dependent, it is difficult and time-consuming to adjust and maintain the IV drip infusion at a therapeutic level; frequent laboratory testing is necessary. With long-term heparin use, there is also the risk of antibody formation and bleeding. Despite the risks, anticoagulation after initial clot formation and dislodgement is necessary because of the high risk for a recurrent thrombus. Therapy may be changed to an oral regimen, such as warfarin, as soon as the patient is able to take oral medications. Heparin must be continued until the international normalized ratio (INR) is within a therapeutic range, typically 2.0 to 2.5. Once the patient starts an oral regimen, it is important that he or she continue to take the same brand of warfarin, because the bioavailability may vary greatly among brands.

High doses of subcutaneous low-molecular-weight heparin (enoxaparin [Lovenox]) or heparinoids may also be used for PE. These include enoxaparin, fondaparinux (Arixtra), dalteparin (Fragmin), inzaparin (Innohep), lepirudin (Refludan), and argatroban (Novastan). These agents are direct thrombin inhibitors; therefore, they require less frequent monitoring and dose adjustment. All medications have contraindications and side effects that the nurse must be aware of before administration. Lepirudin and argatroban are both contraindicated in patients with overt major bleeding and in patients who are hypersensitive to these agents or at high risk for bleeding (eg, recent cerebrovascular accident [CVA, stroke], anomaly of vessels or organs, recent major surgery, recent puncture of large vessels or organ biopsy). Major side effects are bleeding anywhere in the body and anaphylactic reaction resulting in shock or death. Other side effects include fever, abnormal liver function, and allergic skin reaction. All patients must continue to take some form of anticoagulation for at least 3 to 6 months after the embolic event.

#### Thrombolytic Therapy

Thrombolytic therapy (urokinase, streptokinase, alteplase) is used in treating PE, particularly in patients who are severely compromised (eg, those who are hypotensive and have significant hypoxemia despite oxygen supplementation) (Carlbom & Davidson, 2007). Thrombolytic therapy resolves the thrombi or emboli quickly and restores more normal hemodynamic functioning of the pulmonary circulation, thereby reducing pulmonary hypertension and improving perfusion, oxygenation, and cardiac output. However, bleeding is a significant side effect. Contraindications to thrombolytic therapy include a CVA within the past 2 months, other active intracranial processes, active bleeding, surgery within 10 days of the thrombotic event, recent labor and delivery, trauma, or severe hypertension. Consequently, thrombolytic agents are advocated only for PE affecting a significant area of blood flow to the lung and causing hemodynamic instability.

Before thrombolytic therapy is started, INR, partial thromboplastin time (PTT), hematocrit, and platelet counts are obtained. An anticoagulant is stopped prior to administration of a thrombolytic agent. During therapy, all but essential invasive procedures are avoided because of potential bleeding. If necessary, fresh whole blood, packed red cells, cryoprecipitate, or frozen plasma is administered to replace blood loss and reverse the bleeding tendency. After the thrombolytic infusion is completed (which varies in duration according to the agent used), anticoagulant therapy is initiated.

### Surgical Management

A surgical embolectomy is rarely performed but may be indicated if the patient has a massive PE or hemodynamic instability or if there are contraindications to thrombolytic therapy. This invasive procedure involves removal of the actual clot and must be performed by a cardiovascular surgical team with the patient on cardiopulmonary bypass. It may be used for patients who fail to improve with thrombolytic therapy, have contraindications to thrombolytic therapy and have had a massive PE, or must have the clot removed to help reduce right-sided heart failure. Although surgical embolectomy ensures removal of the clot, it is not without risk. It has a mortality rate of 30% (Carlbom & Davidson, 2007).

Transvenous catheter embolectomy is a technique in which a vacuum-cupped catheter is introduced transvenously into the affected pulmonary artery. Suction is applied to the end of the embolus, and the embolus is aspirated into the cup. The surgeon maintains suction to hold the embolus within the cup, and the entire catheter is withdrawn through the right side of the heart and out the femoral vein. Catheters are available that pulverize the clot with high-velocity jets of normal saline solution. An inferior vena cava filter is usually inserted at the time of surgery to protect against a recurrence.

Interrupting the inferior vena cava is another surgical technique used when PE recurs or when the patient does not tolerate anticoagulant therapy. This approach prevents dislodged thrombi from being swept into the lungs while allowing adequate blood flow. The preferred approach is the application of Teflon clips to the inferior vena cava to divide the lumen into small channels without occluding caval blood flow. Also, the use of transvenous devices that occlude or filter the blood through the inferior vena cava is a fairly safe way to prevent recurrent PE. One such technique involves insertion of a filter (eg, Greenfield filter) through the internal jugular vein or common femoral vein (Fig. 23-6). This filter is advanced into the inferior vena

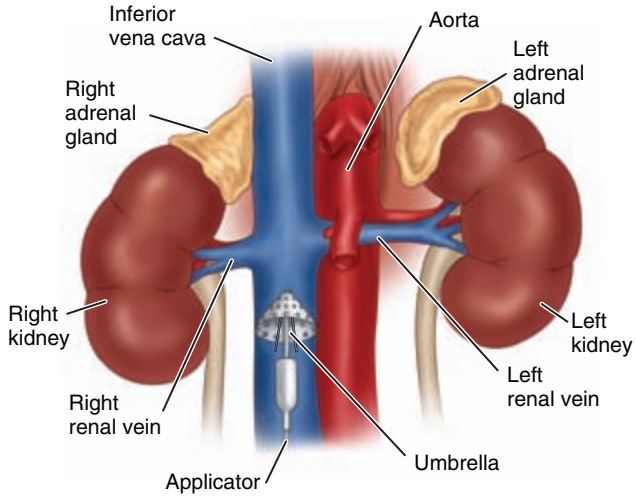


**Figure 23-6** An umbrella filter is in place in the inferior vena cava to prevent pulmonary embolism. The filter (compressed within an applicator catheter) is inserted through an incision in the right internal jugular vein. The applicator is withdrawn when the filter fixes itself to the wall of the inferior vena cava after ejection from the applicator.

cava, where it is opened. The perforated umbrella permits the passage of blood but prevents the passage of large thrombi. It is recommended that anticoagulation be continued in patients with a vena cava filter if there are no contraindications to its use.

## Nursing Management

### Minimizing the Risk of Pulmonary Embolism

A key role of the nurse is to identify the patient at high risk for PE and to minimize the risk of PE in all patients. The nurse must have a high degree of suspicion for PE in all patients, but particularly in those with conditions predisposing to a slowing of venous return (see Chart 23-8).

### Preventing Thrombus Formation

Preventing thrombus formation is a major nursing responsibility. The nurse encourages ambulation and active and passive leg exercises to prevent venous stasis in patients prescribed bed rest. The nurse instructs the patient to move the legs in a "pumping" exercise so that the leg muscles can help increase venous flow. The nurse also advises the patient not to sit or lie in bed for prolonged periods, not to cross the legs, and not to wear constrictive clothing. Legs should not be dangled or feet placed in a dependent position while the patient sits on the edge of the bed; instead, feet should rest on the floor or on a chair. In addition, IV catheters (for parenteral therapy or measurements of central venous pressure) should not be left in place for prolonged periods.

### Assessing Potential for Pulmonary Embolism

All patients are evaluated for risk factors for thrombus formation and pulmonary embolus. The nurse conducts a careful assessment of the patient's health history, family history, and medication record. On a daily basis, the patient is asked about pain or discomfort in the extremities. In addition, the extremities are evaluated for warmth, redness, and inflammation.

### Monitoring Thrombolytic Therapy

The nurse is responsible for monitoring thrombolytic and anticoagulant therapy. Thrombolytic therapy (streptokinase, urokinase, tissue plasminogen activator) causes lysis of deep vein thrombi and pulmonary emboli, which helps dissolve the clots. During thrombolytic infusion, while the patient remains on bed rest, vital signs are assessed every 2 hours and invasive procedures are avoided. Tests to determine INR or PTT are performed 3 to 4 hours after the thrombolytic infusion is started to confirm that the fibrinolytic systems have been activated. See Chapter 31 for nursing management for the patient receiving anticoagulant or thrombolytic therapy.

*NURSING ALERT*

**Because of the prolonged clotting time, only essential arterial punctures or venipunctures are performed, and manual pressure is applied to any puncture site for at least 30 minutes. Pulse oximetry is used to monitor changes in oxygenation. The thrombolytic infusion is discontinued immediately if uncontrolled bleeding occurs.**

### Managing Pain

Chest pain, if present, is usually pleuritic rather than cardiac in origin. A semi-Fowler's position provides a more comfortable position for breathing. However, it is important to continue to turn patients frequently and reposition them to improve the **ventilation–perfusion ratio** in the lung. The nurse administers opioid analgesic agents as prescribed for severe pain.

### Managing Oxygen Therapy

Careful attention is given to the proper use of oxygen. It is important to ensure that the patient understands the need for continuous oxygen therapy. The nurse assesses the patient frequently for signs of hypoxemia and monitors the pulse oximetry values to evaluate the effectiveness of the oxygen therapy. Deep breathing and incentive spirometry are indicated for all patients to minimize or prevent atelectasis and improve ventilation. Nebulizer therapy or percussion and postural drainage may be used for management of secretions.

### Relieving Anxiety

The nurse encourages the stabilized patient to talk about any fears or concerns related to this frightening episode, answers the patient's and family's questions concisely and accurately, explains the therapy, and describes how to recognize untoward effects early.

### Monitoring for Complications

When caring for a patient who has had PE, the nurse must be alert for the potential complication of cardiogenic shock or right ventricular failure subsequent to the effect of PE on the cardiovascular system. Nursing activities for managing shock are found in Chapter 15.

### *Providing Postoperative Nursing Care*

If the patient has undergone surgical embolectomy, the nurse measures the patient's pulmonary arterial pressure and urinary output. The nurse also assesses the insertion site of the arterial catheter for hematoma formation and infection. It is important to maintain the blood pressure at a level that supports perfusion of vital organs. To prevent peripheral venous stasis and edema of the lower extremities, the nurse elevates the foot of the bed and encourages isometric exercises, use of anti-embolism stockings, and walking when the patient is permitted out of bed. Sitting is discouraged, because hip flexion compresses the large veins in the legs.

### *Promoting Home and Community-Based Care*

#### Teaching Patients Self-Care

Before hospital discharge and at follow-up visits to the clinic, the nurse instructs the patient about preventing recurrence and reporting signs and symptoms. Patient instructions, presented in Chart 23-9, are intended to help prevent recurrences and side effects of treatment.

#### Continuing Care

During follow-up or home care visits, the nurse monitors the patient's adherence to the prescribed management plan and reinforces previous instructions. The nurse also monitors the patient for residual effects of the PE and recovery. The patient is reminded about the importance of keeping follow-up appointments for coagulation tests and appointments with the primary care provider. The nurse also reminds the patient about the importance of participation in health promotion activities (eg, immunizations) and health screening.

## *Sarcoidosis*

Sarcoidosis is a multisystem, granulomatous disease of unknown etiology. It may involve almost any organ or tissue but most commonly involves the lungs, lymph nodes, liver, spleen, central nervous system, skin, eyes, fingers, and parotid glands. The disease is not gender specific, but some manifestations are more common in women. In the United States, the disease is more common in African Americans (36 cases per 100,000) than in Caucasians (11 cases per 100,000), and the disease affects young and middle-aged adults (Weinberger, 2006).

### Pathophysiology

Sarcoidosis is thought to be a hypersensitivity response to one or more exogenous agents (bacteria, fungi, virus, chemicals) in people with an inherited or acquired predisposition to the disorder. The hypersensitivity response results in noncaseating granuloma formation due to the release of cytokines and other substances that promote replication of fibroblasts. In the lung, granuloma infiltration and fibrosis may occur, resulting in low lung compliance, impaired diffusing capacity, and reduced lung volumes.

### Clinical Manifestations

Hallmarks of sarcoidosis are its insidious onset and lack of prominent clinical signs or symptoms. The clinical picture depends on the systems affected. The lung is most commonly involved; signs and symptoms may include dyspnea, cough, hemoptysis, and congestion. Generalized symptoms include anorexia, fatigue, and weight loss. Other signs include uveitis, joint pain, fever, and granulomatous lesions of the skin, liver, spleen, kidney, and central nervous system. The granulomas may disappear or gradually convert to fibrous tissue. With multisystem involvement, patients may also have fatigue, fever, anorexia, and weight loss.

### Assessment and Diagnostic Findings

Chest x-rays and CT are used to assess pulmonary adenopathy. These may show hilar adenopathy and disseminated miliary and nodular lesions in the lungs. A mediastinoscopy or **transbronchial** biopsy (in which a tissue specimen is obtained through the bronchial wall) may be used to confirm the diagnosis. In rare cases, an **open lung biopsy** is performed. Diagnosis is confirmed by a biopsy that shows noncaseating granulomas. Pulmonary function test results are abnormal if there is restriction of lung function (reduction in total lung capacity). Arterial blood gas measurements may be normal or may show reduced oxygen levels (hypoxemia) and increased carbon dioxide levels (hypercapnia).

### Medical Management

Many patients undergo remission without specific treatment. Corticosteroids may be beneficial because of their anti-inflammatory effects, which relieve symptoms and improve organ function. They have shown to be useful in patients with ocular and myocardial involvement, skin involvement, extensive pulmonary disease that compromises pulmonary function, hepatic involvement, and hypercalcemia. However, it is not known if steroids alter the long-term course of the disease (Weinberger, 2006). Other cytotoxic and immunosuppressive agents have been used, but without the benefit of controlled clinical trials. There is no single test that monitors the progression or recurrence of sarcoidosis. Multiple tests are used to monitor involved systems.

## OCCUPATIONAL LUNG DISEASES: PNEUMOCONIOSES

Pneumoconiosis refers to a nonneoplastic alteration of the lung resulting from inhalation of mineral or inorganic dust (eg, "dusty lung"). Pneumoconioses are caused by inhalation and deposition of mineral dusts in the lungs, resulting in pulmonary fibrosis and parenchymal changes. Usually, extended exposure to irritating or toxic substances accounts for these changes, although severe single exposures may also lead to chronic lung disease. Occupational lung disease is the number one work-related illness in the United States based on the frequency, severity, and preventability. Many people with early pneumoconiosis are asymptomatic, but advanced disease often is accompanied by disability and premature death.

**Table 23-4 OCCUPATIONAL LUNG DISEASES: PNEUMOCONIOSES**

| Disease (Source) | Pathophysiology | Clinical Manifestations |
|---|---|---|
| Silicosis (glass manufacturing, foundry work, stone cutting) | Inhaled silica dust produces nodular lesions in the lungs. Nodules enlarge and coalesce. Dense masses form on upper portion of lungs, resulting in loss of pulmonary volume. Fibrotic destruction of pulmonary tissue can lead to restrictive lung disease, emphysema, pulmonary hypertension, and cor pulmonale. | *Acute silicosis:* Dyspnea, fever, cough, weight loss<br>*Chronic silicosis:* Progressive symptoms indicative of hypoxemia, severe airflow obstruction, and right-sided heart failure |
| Asbestosis (shipbuilding, building demolition) | Inhaled asbestos fibers enter alveoli and are surrounded by fibrous tissue. Fibrous changes can also affect the pleura, which thicken and develop plaque. These changes lead to restrictive lung disease, with a decrease in lung volume, diminished exchange of oxygen and carbon dioxide, hypoxemia, cor pulmonale, and respiratory failure. It also increases risk for lung cancer, mesothelioma, and pleural effusion. | Progressive dyspnea; persistent, dry cough; mild to moderate chest pain; anorexia; weight loss; malaise; clubbing of the fingers |
| Coal worker's pneumoconiosis | Encompasses a variety of lung diseases; is also known as black lung disease. Inhaled dusts that are mixtures of coal, kaolin, mica, and silica are deposited in the alveoli and respiratory bronchioles. When macrophages that engulf the dust can no longer be cleared, they aggregate and fibroblasts appear. The bronchioles and alveoli become clogged with dust, dying macrophages, and fibroblasts, leading to formation of coal macules. Fibrotic lesions develop and subsequently localized emphysema develops, with cor pulmonale and respiratory failure. | Chronic cough, dyspnea, and expectoration of black or gray sputum, especially in miners who are smokers with cavitation in the lungs |

Diseases of the lungs occur in numerous occupations as a result of exposure to several different types of agents, such as mineral dusts, metal dusts, biologic dusts, and toxic fumes. Smoking may compound the problem and may increase the risk of lung cancers in people exposed to the mineral asbestos and other potential carcinogens. The effects of inhaling these materials depend on the composition of the substance, its concentration, its ability to initiate an immune response, its irritating properties, the duration of exposure, and the individual's response or susceptibility to the irritant.

These diseases are not treatable once they develop, but they are preventable. Therefore, a major role for nurses, especially occupational health nurses, is that of advocate for employees. Nurses need to make every effort to promote measures to reduce the exposure of workers to industrial products. Strategies to control exposure should be identified and encouraged; these strategies include the use of protective devices (face masks, hoods, industrial respirators) to minimize exposure and screening/monitoring of individuals at risk.

Key aspects of any assessment of patients with a potential occupational respiratory history include job and job activities, exposure levels, general hygiene, time frame of exposure, effectiveness of respiratory protection used, and direct versus indirect exposures. Specific information that should be obtained includes the following:

- Exposure to an agent known to cause an occupational disorder
- Length of time from exposure of agent to onset of symptoms
- Congruence of symptoms with those of known exposure-related disorder
- Lack of other more likely explanations of the signs and symptoms

The most common pneumoconioses are silicosis, asbestosis, and coal worker's pneumoconiosis (Table 23-4). The nurse teaches preventive measures to patients and their families, assesses patients for a history of exposure to environmental agents, and makes referrals so that pulmonary function can be evaluated and the patient can be treated early in the course of the disease. There is no effective treatment for these diseases because damage is irreversible. Supportive therapy is aimed at preventing infections and managing complications.

## CHEST TUMORS

Tumors of the lung may be benign or malignant. A malignant chest tumor can be primary, arising within the lung, chest wall, or mediastinum, or it can be a metastasis from a primary tumor site elsewhere in the body.

### *Lung Cancer (Bronchogenic Carcinoma)*

Lung cancer is the leading cancer killer among men and women in the United States, with almost 162,000 deaths estimated in 2008. It is estimated that 31% of cancer deaths in men and 26% in women are related to lung or bronchus

cancers. Approximately 213,000 new cases of lung cancer are diagnosed annually; 15% of new cancers for men and women involve the lung or bronchus (American Cancer Society, 2008). In approximately 70% of patients with lung cancer, the disease has spread to regional lymphatics and other sites by the time of diagnosis. As a result, the long-term survival rate is low. Overall, the 5-year survival rate is 16%.

## Pathophysiology

The most common cause of lung cancer is inhaled carcinogens, most often cigarette smoke (90%); other carcinogens include radon gas and occupational and environmental agents (American College of Chest Physicians [ACCP], 2007). Lung cancers arise from a single transformed epithelial cell in the tracheobronchial airways, in which the carcinogen binds to and damages the cell's DNA. This damage results in cellular changes, abnormal cell growth, and eventually a malignant cell. As the damaged DNA is passed on to daughter cells, the DNA undergoes further changes and becomes unstable. With the accumulation of genetic changes, the pulmonary epithelium undergoes malignant transformation from normal epithelium eventually to invasive carcinoma. Carcinoma tends to arise at sites of previous scarring (TB, fibrosis) in the lung.

## Classification and Staging

For purposes of staging and treatment, most lung cancers are classified into one of two major categories: small cell lung cancer and non–small cell lung cancer. Small cell carcinoma represents 15% to 20% of tumors; non–small cell lung carcinoma (NSCLC) represents approximately 80% of tumors (ACCP, 2007). In NSCLC, the cell types include squamous cell carcinoma (20% to 30%), large cell carcinoma (15%), and adenocarcinoma (40%), including bronchoalveolar carcinoma. Most small cell cancers arise in the major bronchi and spread by infiltration along the bronchial wall (Yoder, 2006).

Non–small cell lung cancer is further classified by cell type. Squamous cell cancer is usually more centrally located and arises more commonly in the segmental and subsegmental bronchi. Adenocarcinoma is the most prevalent carcinoma of the lung in both men and women; it occurs peripherally as peripheral masses or nodules and often metastasizes. Large cell carcinoma (also called undifferentiated carcinoma) is a fast-growing tumor that tends to arise peripherally. Bronchoalveolar cell cancer is found in the terminal bronchi and alveoli and is usually slower growing compared with other bronchogenic carcinomas.

In addition to classification according to cell type, lung cancers are staged. The stage of the tumor refers to the size of the tumor, its location, whether lymph nodes are involved, and whether the cancer has spread (Collaborative Staging Task Force of the American Joint Committee on Cancer, 2007). NSCLC is staged as I to IV. Stage I is the earliest stage and has the highest cure rate, whereas stage IV designates metastatic spread. Estimated 5-year survival rates for the stages of NSCLC are as follows: stages IA and IB, 50% to 80%; stage IIA and IIB, 30% to 50%; stage IIIA, 10% to 40%; stage IIIB, 5% to 20%; and stage IV, less than 5% (ACCP, 2007). Small cell lung cancers are classified as limited or extensive. Diagnostic tools and further information on staging are described in Chapter 16.

## Risk Factors

Various factors have been associated with the development of lung cancer, including tobacco smoke, secondhand (passive) smoke, environmental and occupational exposures, gender, genetics, and dietary deficits. Other factors that have been associated with lung cancer include genetic predisposition and underlying respiratory diseases, such as COPD and TB.

### *Tobacco Smoke*

Tobacco use is responsible for more than one of every six deaths in the United States from pulmonary and cardiovascular diseases. Smoking is the most important single preventable cause of death and disease in this country. Lung cancer is 10 times more common in cigarette smokers than nonsmokers. Risk is determined by the pack-year history (number of packs of cigarettes used each day, multiplied by the number of years smoked), the age of initiation of smoking, the depth of inhalation, and the tar and nicotine levels in the cigarettes smoked. The younger a person is when he or she starts smoking, the greater the risk of developing lung cancer.

### *Secondhand Smoke*

Passive smoking has been identified as a possible cause of lung cancer in nonsmokers (U.S. Department of Health and Human Services, 2006). It is estimated that secondhand smoke causes about 3000 deaths per year (ACCP, 2007). People who are involuntarily exposed to tobacco smoke in a closed environment (house, automobile, building) have an increased risk of lung cancer when compared with unexposed nonsmokers.

### *Environmental and Occupational Exposure*

Various carcinogens have been identified in the atmosphere, including motor vehicle emissions and pollutants from refineries and manufacturing plants. Evidence suggests that the incidence of lung cancer is greater in urban areas as a result of the buildup of pollutants and motor vehicle emissions.

Radon is a colorless, odorless gas found in soil and rocks. For many years, it has been associated with uranium mines, but it is now known to seep into homes through ground rock. High levels of radon have been associated with the development of lung cancer, especially when combined with cigarette smoking. Homeowners are advised to have radon levels checked in their houses and to arrange for special venting if the levels are high.

Chronic exposure to industrial carcinogens, such as arsenic, asbestos, mustard gas, chromates, coke oven fumes, nickel, oil, and radiation, has been associated with the development of lung cancer. Laws have been passed to control exposure to these carcinogens in the workplace.

### *Genetics*

Some familial predisposition to lung cancer seems apparent, because the incidence of lung cancer in close relatives of patients with lung cancer appears to be two to three times that in the general population regardless of smoking status.

## Clinical Manifestations

Often, lung cancer develops insidiously and is asymptomatic until late in its course. The signs and symptoms depend on the location and size of the tumor, the degree of obstruction, and the existence of metastases to regional or distant sites.

The most frequent symptom of lung cancer is cough or change in a chronic cough. People frequently ignore this symptom and attribute it to smoking or a respiratory infection. The cough may start as a dry, persistent cough, without sputum production. When obstruction of airways occurs, the cough may become productive due to infection. Cough is present in 65% to 75% of patients with lung cancer, and more than 25% have a productive cough (Yoder, 2006).

*NURSING ALERT*

**A cough that changes in character should arouse suspicion of lung cancer.**

Dyspnea occurs in up to 60% of patients early in their disease (Yoder, 2006). Causes of dyspnea may include tumor occlusion of the airway or lung parenchyma, pleural effusion, pneumonia, or complications of treatment. Hemoptysis or blood-tinged sputum may be expectorated. Chest or shoulder pain may indicate chest wall or pleural involvement by a tumor. Pain also is a late manifestation and may be related to metastasis to the bone.

In some patients, a recurring fever is an early symptom in response to a persistent infection in an area of pneumonitis distal to the tumor. In fact, cancer of the lung should be suspected in people with repeated unresolved upper respiratory tract infections. If the tumor spreads to adjacent structures and regional lymph nodes, the patient may present with chest pain and tightness, hoarseness (involving the recurrent laryngeal nerve), dysphagia, head and neck edema, and symptoms of pleural or pericardial effusion. The most common sites of metastases are lymph nodes, bone, brain, contralateral lung, adrenal glands, and liver (Fig. 23-7). Nonspecific symptoms of weakness, anorexia, and weight loss also may be present.

## Assessment and Diagnostic Findings

If pulmonary symptoms occur in heavy smokers, cancer of the lung should always be considered. A chest x-ray is performed to search for pulmonary density, a solitary pulmonary nodule (coin lesion), atelectasis, and infection. CT scans of the chest are used to identify small nodules not easily visualized on the chest x-ray and also to serially examine areas for lymphadenopathy.

Sputum cytology is rarely used to make a diagnosis of lung cancer. Fiberoptic bronchoscopy is more commonly used; it provides a detailed study of the tracheobronchial tree and allows for brushings, washings, and biopsies of suspicious areas. For peripheral lesions not amenable to bronchoscopic biopsy, a transthoracic **fine-needle aspiration** may be performed under CT guidance to aspirate cells from a suspicious area. In some circumstances, an endoscopy with esophageal ultrasound may be used to obtain a transesophageal biopsy of enlarged subcarinal lymph nodes that are not easily accessible by other means.

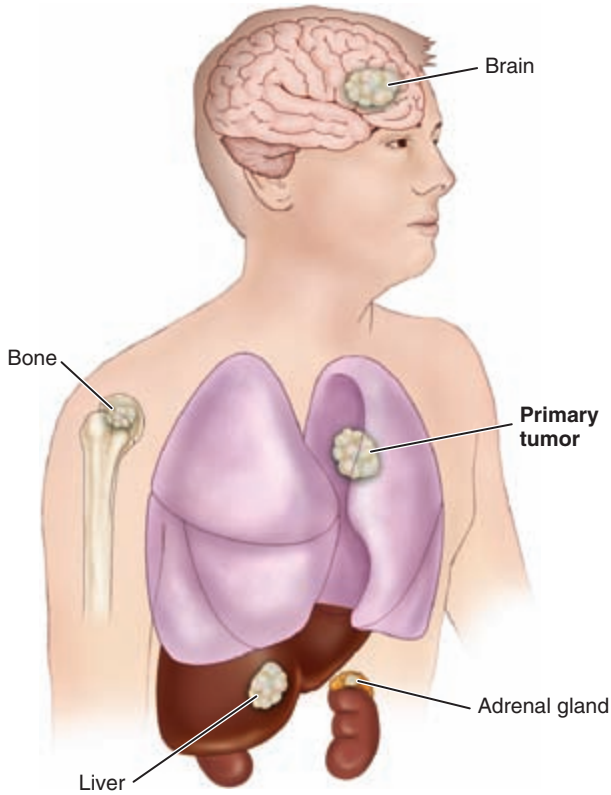


**Figure 23-7** Frequent sites of lung cancer metastasis.

A variety of scans may be used to assess for metastasis of the cancer. These may include bone scans, abdominal scans, positron emission tomography (PET) scans, and liver ultrasound. CT of the brain, magnetic resonance imaging (MRI), and other neurologic diagnostic procedures are used to detect central nervous system metastases. Mediastinoscopy or mediastinotomy may be used to obtain biopsy samples from lymph nodes in the mediastinum.

If surgery is a potential treatment, the patient is evaluated to determine whether the tumor is resectable and whether the patient can tolerate the physiologic impairment resulting from such surgery. Pulmonary function tests, arterial blood gas analysis, $\dot{V}/\dot{Q}$ scans, and exercise testing may all be used as part of the preoperative assessment.

## Medical Management

The objective of management is to provide a cure, if possible. Treatment depends on the cell type, the stage of the disease, and the patient's physiologic status (particularly cardiac and pulmonary status). In general, treatment may involve surgery, radiation therapy, or chemotherapy—or a combination of these. Newer and more specific therapies to modulate the immune system (gene therapy, therapy with defined tumor antigens) are under study and show promise.

### *Surgical Management*

Surgical resection is the preferred method of treating patients with localized non–small cell tumors, no evidence of metastatic spread, and adequate cardiopulmonary function. If the patient's cardiovascular status, pulmonary function,

### Chart 23-10 • *Types of Lung Resection*

- Lobectomy: a single lobe of the lung is removed
- Bilobectomy: two lobes of the lung are removed
- Sleeve resection: cancerous lobe(s) is removed and a segment of the main bronchus is resected
- Pneumonectomy: removal of entire lung
- Segmentectomy: a segment of the lung is removed*
- Wedge resection: removal of a small, pie-shaped area of the segment*
- Chest wall resection with removal of cancerous lung tissue: for cancers that have invaded the chest wall

*Not recommended as curative resection for lung cancer.

and functional status are satisfactory, surgery is generally well tolerated. However, coronary artery disease, pulmonary insufficiency, and other comorbidities may contraindicate surgical intervention. The cure rate of surgical resection depends on the type and stage of the cancer. Surgery is primarily used for NSCLCs, because small cell cancer of the lung grows rapidly and metastasizes early and extensively. Lesions of many patients with bronchogenic cancer are inoperable at the time of diagnosis.

Several different types of lung resection may be performed (Chart 23-10). The most common surgical procedure for a small, apparently curable tumor of the lung is lobectomy (removal of a lobe of the lung). In some cases, an entire lung may be removed (pneumonectomy) (see Chapter 25 for further details).

#### *Radiation Therapy*

Radiation therapy may offer cure in a small percentage of patients. It is useful in controlling neoplasms that cannot be surgically resected but are responsive to radiation. Irradiation also may be used to reduce the size of a tumor, to make an inoperable tumor operable, or to relieve the pressure of the tumor on vital structures. It can reduce symptoms of spinal cord metastasis and superior vena caval compression. Also, prophylactic brain irradiation is used in certain patients to treat microscopic metastases to the brain. Radiation therapy may help relieve cough, chest pain, dyspnea, hemoptysis, and bone and liver pain. Relief of symptoms may last from a few weeks to many months and is important in improving the quality of the remaining period of life.

Radiation therapy usually is toxic to normal tissue within the radiation field, and this may lead to complications such as esophagitis, pneumonitis, and radiation lung fibrosis. These may impair ventilatory and diffusion capacity and significantly reduce pulmonary reserve. The patient's nutritional status, psychological outlook, fatigue level, and signs of anemia and infection are monitored throughout the treatment. See Chapter 16 for management of the patient receiving radiation therapy.

#### *Chemotherapy*

Chemotherapy is used to alter tumor growth patterns, to treat distant metastases or small cell cancer of the lung, and as an adjunct to surgery or radiation therapy. Chemotherapy may provide relief, especially of pain, but it does not usually cure the disease or prolong life to any great degree. Chemotherapy is also accompanied by side effects. It is valuable in reducing pressure symptoms of lung cancer and in treating brain, spinal cord, and pericardial metastasis. See Chapter 16 for a discussion of chemotherapy for the patient with cancer.

The choice of agent depends on the growth of the tumor cell and the specific phase of the cell cycle that the medication affects. In combination with surgery, chemotherapy may be administered before surgery (neoadjuvant therapy) or after surgery (adjuvant therapy). Combinations of two or more medications may be more beneficial than single-dose regimens. A variety of agents are used, including platinum analogues (cisplatin [Platinol] and carboplatin [Paraplatin]) and non–platinum-containing agents—taxanes (paclitaxel [Taxol, Onxol], docetaxel [Taxotere]), vinca alkaloids (vinblastine [Velban] and vindesine [Eldisine]), doxorubicin (Adriamycin, Doxil), gemcitabine (Gemzar), vinorelbine (Navelbine), irinotecan (Camptosar), etoposide (Toposar), and pemetrexed (Alimta). Other approved chemotherapeutic agents in oral form are gefitinib (Iressa) and erlotinib (Tarceva), which are epidermal growth factor tyrosine kinase inhibitors. Numerous new agents with cellular targets, including protein kinase C, vascular endothelial growth factor, cyclo-oxygenase-2, and farnesyl transferase, are being investigated for various types of lung cancer.

#### *Palliative Therapy*

Palliative therapy may include radiation therapy to shrink the tumor to provide pain relief, a variety of bronchoscopic interventions to open a narrowed bronchus or airway, and pain management and other comfort measures. Evaluation and referral for hospice care are important in planning for comfortable and dignified end-of-life care for the patient and family.

## Treatment-Related Complications

A variety of complications may occur as a result of treatment for lung cancer. Surgical resection may result in respiratory failure, particularly if the cardiopulmonary system is compromised before surgery. Surgical complications and prolonged mechanical ventilation are potential outcomes. Radiation therapy may result in diminished cardiopulmonary function and other complications, such as pulmonary fibrosis, pericarditis, myelitis, and cor pulmonale. Chemotherapy, particularly in combination with radiation therapy, can cause pneumonitis. Pulmonary toxicity is a potential side effect of chemotherapy.

## Nursing Management

Nursing care of patients with lung cancer is similar to that for other patients with cancer (see Chapter 16) and addresses the physiologic and psychological needs of the patient. The physiologic problems are primarily due to the respiratory manifestations of the disease. Nursing care includes strategies to ensure relief of pain and discomfort and to prevent complications.

#### *Managing Symptoms*

The nurse instructs the patient and family about the potential side effects of the specific treatment and strategies to manage them. Strategies for managing such symptoms as

CHART 23-11 NURSING RESEARCH PROFILE

## *Quality of Life of Women With Lung Cancer and Their Families*

Sarna, L., Cooley, M. E., Brown, J. K., et al. (2006). Quality of life and health status of dyads of women with lung cancer and family members. *Oncology Nursing Forum, 33*(6), 1109–1116.

### Purpose

The purpose of this study was to describe and compare the quality of life and health status of dyads of women with lung cancer and their family members. Primary outcomes evaluated included quality of life and health status for both patients and family members.

### Design

The investigators used a descriptive, cross-sectional research approach in which a convenience sample of 51 dyads of subjects participated. The patient sample consisted of women, and the family sample consisted primarily of men. A one-time assessment was conducted to evaluate quality of life (QOL), with a generic questionnaire, Short Form-36 (SF-36); comorbidities, with the Charlson Comorbidity Index; and depression, with the Center for Epidemiologic Studies Depression (CES-D). The participants also provided a self-report of smoking and drinking history as well as demographic data.

### Findings

Analysis of data from 50 dyads revealed that the QOL of the patients and families was not significantly correlated. Family members of patients with lung cancer reported a significantly higher quality of life than patients with the disease. Poorer physical QOL of family members was related to increased age, comorbid conditions, less education, and alcohol use. Significantly more family members continued to smoke and use alcohol following the patient's diagnosis.

### Nursing Implications

Nurses should consider not only the health status of patients with lung cancer but also the health status and QOL of family members. The health and QOL of these caregivers can affect their ability to care for the patient with cancer and to cope with the family member's illness.

dyspnea, fatigue, nausea and vomiting, and anorexia help the patient and family cope with therapeutic measures.

#### *Relieving Breathing Problems*

Airway clearance techniques are key to maintaining airway patency through the removal of excess secretions. This may be accomplished through deep-breathing exercises, chest physiotherapy, directed cough, suctioning, and in some instances bronchoscopy. Bronchodilator medications may be prescribed to promote bronchial dilation. As the tumor enlarges or spreads, it may compress a bronchus or involve a large area of lung tissue, resulting in an impaired breathing pattern and poor gas exchange. At some stage of the disease, supplemental oxygen will probably be necessary.

Nursing measures focus on decreasing dyspnea by encouraging the patient to assume positions that promote lung expansion and to perform breathing exercises for lung expansion and relaxation. Patient education about energy conservation and airway clearance techniques is also necessary. Many of the techniques used in pulmonary rehabilitation can be applied to patients with lung cancer. Depending on the severity of disease and the patient's wishes, a referral to a pulmonary rehabilitation program may be helpful in managing respiratory symptoms.

#### *Reducing Fatigue*

Fatigue is a devastating symptom that affects quality of life in patients with cancer. It is commonly experienced by patients with lung cancer and may be related to the disease itself, the cancer treatment and complications (eg, anemia), sleep disturbances, pain and discomfort, hypoxemia, poor nutrition, or the psychological ramifications of the disease (eg, anxiety, depression). Nursing strategies to promote energy conservation and reduce fatigue are presented in Chapter 16.

#### *Providing Psychological Support*

Another important part of the nursing care of patients with lung cancer is provision of psychological support and identification of potential resources for the patient and family. Often, the nurse must help the patient and family deal with the following:

- The poor prognosis and relatively rapid progression of this disease
- Informed decision making regarding the possible treatment options
- Methods to maintain the patient's quality of life during the course of this disease (Chart 23-11)
- End-of-life treatment options

### Gerontologic Considerations

At the time of diagnosis of lung cancer, most patients are older than 65 years of age and have stage III or IV disease. Although age is not a significant prognostic factor for overall survival and response to treatment for either NSCLC or small cell lung cancer, older patients have specific needs. Depending on the comorbidities and functional status of elderly patients, chemotherapy agents, doses, and cycles may need to be adjusted to maintain quality of life. Issues that must be considered in care of elderly patients with lung cancer include functional status, comorbid conditions, nutritional status, cognition, concomitant medications, and psychological and social support (ACCP, 2007; Yoder, 2006).

## *Tumors of the Mediastinum*

Tumors of the mediastinum include neurogenic tumors, tumors of the thymus, lymphomas, germ cell tumors, cysts, and mesenchymal tumors. These tumors may be malignant

or benign. They are usually described in relation to location: anterior, middle, or posterior masses or tumors.

### Clinical Manifestations

Nearly all symptoms of mediastinal tumors result from the pressure of the mass against important intrathoracic organs. Symptoms may include cough, wheezing, dyspnea, anterior chest or neck pain, bulging of the chest wall, heart palpitations, angina, other circulatory disturbances, central cyanosis, superior vena cava syndrome (ie, swelling of the face, neck, and upper extremities), marked distention of the veins of the neck and the chest wall (evidence of the obstruction of large veins of the mediastinum by extravascular compression or intravascular invasion), and dysphagia and weight loss from pressure or invasion into the esophagus.

### Assessment and Diagnostic Findings

Chest x-rays are the major method used initially to diagnose mediastinal tumors and cysts. CT is the standard diagnostic test for assessment of the mediastinum and surrounding structures. MRI, as well as PET, may be used in some circumstances.

### Medical Management

If the tumor is malignant and has infiltrated the surrounding tissue and complete surgical removal is not feasible, radiation therapy, chemotherapy, or both are used.

#### *Surgical Management*

Many mediastinal tumors are benign and operable. The location of the tumor (anterior, middle, or posterior compartment) in the mediastinum dictates the type of incision. The common incision used is a median sternotomy; however, a thoracotomy may be used, depending on the location of the tumor. Additional approaches include a bilateral anterior thoracotomy (clamshell incision) and video-assisted thoracoscopic surgery (see Chapter 25). The care is the same as for any patient undergoing thoracic surgery. Major complications include hemorrhage, injury to the phrenic or recurrent laryngeal nerve, and infection.

## CHEST TRAUMA

Major chest trauma may occur alone or in combination with multiple other injuries. Chest trauma is classified as either blunt or penetrating. Blunt chest trauma results from sudden compression or positive pressure inflicted to the chest wall. Penetrating trauma occurs when a foreign object penetrates the chest wall.

## *Blunt Trauma*

Although blunt chest trauma is more common than penetrating trauma, it is often difficult to identify the extent of the damage because the symptoms may be generalized and vague. In addition, patients may not seek immediate medical attention, which may complicate the problem.

### Pathophysiology

The most common causes of blunt chest trauma are motor vehicle crashes (trauma from steering wheel, seat belt), falls, and bicycle crashes (trauma from handlebars). Mechanisms of blunt chest trauma include acceleration (moving object hitting the chest or patient being thrown into an object), deceleration (sudden decrease in rate of speed or velocity, such as a motor vehicle crash), shearing (stretching forces to areas of the chest causing tears, ruptures, or dissections), and compression (direct blow to the chest, such as a crush injury). Injuries to the chest are often life-threatening and result in one or more of the following pathologic states:

- Hypoxemia from disruption of the airway; injury to the lung parenchyma, rib cage, and respiratory musculature; massive hemorrhage; collapsed lung; and pneumothorax
- Hypovolemia from massive fluid loss from the great vessels, cardiac rupture, or hemothorax
- Cardiac failure from cardiac tamponade, cardiac contusion, or increased intrathoracic pressure

These pathologic states frequently result in impaired ventilation and perfusion leading to acute renal failure, hypovolemic shock, and death.

### Assessment and Diagnostic Findings

Time is critical in treating chest trauma. Therefore, it is essential to assess the patient immediately to determine the following: time elapsed since injury occurred, mechanism of injury, level of responsiveness, specific injuries, estimated blood loss, recent drug or alcohol use, and prehospital treatment. Initial assessment of thoracic injuries includes assessment for airway obstruction, tension pneumothorax, open pneumothorax, massive hemothorax, flail chest, and cardiac tamponade. These injuries are life-threatening and require immediate treatment. Secondary assessment includes assessment for simple pneumothorax, hemothorax, pulmonary contusion, traumatic aortic rupture, tracheobronchial disruption, esophageal perforation, traumatic diaphragmatic injury, and penetrating wounds to the mediastinum. Although listed as secondary, these injuries may be life-threatening as well.

The physical examination includes inspection of the airway, thorax, neck veins, and breathing difficulty. Specifics include assessing the rate and depth of breathing for abnormalities such as stridor, cyanosis, nasal flaring, use of accessory muscles, drooling, and overt trauma to the face, mouth, or neck. The chest is assessed for symmetric movement, symmetry of breath sounds, open chest wounds, entrance or exit wounds, impaled objects, tracheal shift, distended neck veins, subcutaneous emphysema, and paradoxical chest wall motion. In addition, the chest wall is assessed for bruising, petechiae, lacerations, and burns. The vital signs and skin color are assessed for signs of shock. The thorax is palpated for tenderness and crepitus, and the position of the trachea is also assessed.

The initial diagnostic workup includes a chest x-ray, CT scan, complete blood count, clotting studies, type and crossmatch, electrolytes, oxygen saturation, arterial blood gas analysis, and ECG. The patient is completely undressed to avoid missing additional injuries that may complicate care.

Many patients with injuries involving the chest have associated head and abdominal injuries that require attention. Ongoing assessment is essential to monitor the patient's response to treatment and to detect early signs of clinical deterioration.

### Medical Management

The goals of treatment are to evaluate the patient's condition and to initiate aggressive resuscitation. An airway is immediately established with oxygen support and, in some cases, intubation and ventilatory support. Reestablishing fluid volume and negative intrapleural pressure and draining intrapleural fluid and blood are essential.

The potential for massive blood loss and exsanguination with blunt or penetrating chest injuries is high because of injury to the great blood vessels. Many patients die at the scene of the injury or are in shock by the time help arrives. Agitation and irrational and combative behavior are signs of decreased oxygen delivery to the cerebral cortex. Strategies to restore and maintain cardiopulmonary function include ensuring an adequate airway and ventilation; stabilizing and reestablishing chest wall integrity; occluding any opening into the chest (open pneumothorax); and draining or removing any air or fluid from the thorax to relieve pneumothorax, hemothorax, or cardiac tamponade. Hypovolemia and low cardiac output must be corrected. Many of these treatment efforts, along with the control of hemorrhage, are carried out simultaneously at the scene of the injury or in the emergency department. Depending on the success of efforts to control the hemorrhage in the emergency department, the patient may be taken immediately to the operating room. Principles of management are essentially those pertaining to care of the postoperative thoracic patient (see Chapter 25).

## STERNAL AND RIB FRACTURES

Sternal fractures are most common in motor vehicle crashes with a direct blow to the sternum via the steering wheel. Rib fractures are the most common type of chest trauma, occurring in more than 60% of patients admitted with blunt chest injury (Wilkins, Dexter & Gold, 2007). Most rib fractures are benign and are treated conservatively. Fractures of the first three ribs are rare but can result in a high mortality rate because they are associated with laceration of the subclavian artery or vein. The fifth through ninth ribs are the most common sites of fractures. Fractures of the lower ribs are associated with injury to the spleen and liver, which may be lacerated by fragmented sections of the rib.

### Clinical Manifestations

Patients with sternal fractures have anterior chest pain, overlying tenderness, ecchymosis, crepitus, swelling, and possible chest wall deformity. For patients with rib fractures, clinical manifestations are similar: severe pain, point tenderness, and muscle spasm over the area of the fracture that are aggravated by coughing, deep breathing, and movement. The area around the fracture may be bruised. To reduce the pain, the patient splints the chest by breathing in a shallow manner and avoids sighs, deep breaths, coughing, and movement. This reluctance to move or breathe deeply results in diminished ventilation, atelectasis (collapse of unaerated alveoli), pneumonitis, and hypoxemia. Respiratory insufficiency and failure can be the outcomes of such a cycle.

### Assessment and Diagnostic Findings

The patient must be closely evaluated for underlying cardiac injuries. A crackling, grating sound in the thorax (subcutaneous crepitus) may be detected with auscultation. The diagnostic workup may include a chest x-ray, rib films of a specific area, ECG, continuous pulse oximetry, and arterial blood gas analysis.

### Medical Management

Medical management is directed toward relieving pain, avoiding excessive activity, and treating any associated injuries. Surgical fixation is rarely necessary unless fragments are grossly displaced and pose a potential for further injury.

The goals of treatment for rib fractures are to control pain and to detect and treat the injury. Sedation is used to relieve pain and to allow deep breathing and coughing. Care must be taken to avoid oversedation and suppression of respiratory drive. Alternative strategies to relieve pain include an intercostal nerve block and ice over the fracture site. A chest binder may be used as supportive treatment to provide stability to the chest wall and may decrease pain. The patient is instructed to apply the binder snugly enough to provide support, but not to impair respiratory excursion. Usually the pain abates in 5 to 7 days, and discomfort can be relieved with epidural analgesia, patient-controlled analgesia, or nonopioid analgesia. Most rib fractures heal in 3 to 6 weeks. The patient is monitored closely for signs and symptoms of associated injuries.

## FLAIL CHEST

Flail chest is frequently a complication of blunt chest trauma from a steering wheel injury. It usually occurs when three or more adjacent ribs (multiple contiguous ribs) are fractured at two or more sites, resulting in free-floating rib segments. It may also result as a combination fracture of ribs and costal cartilages or sternum. As a result, the chest wall loses stability, causing respiratory impairment and usually severe respiratory distress.

### Pathophysiology

During inspiration, as the chest expands, the detached part of the rib segment (flail segment) moves in a paradoxical manner (pendelluft movement) in that it is pulled inward during inspiration, reducing the amount of air that can be drawn into the lungs. On expiration, because the intrathoracic pressure exceeds atmospheric pressure, the flail segment bulges outward, impairing the patient's ability to exhale. The mediastinum then shifts back to the affected side (Fig. 23-8). This paradoxical action results in increased dead space, a reduction in alveolar ventilation, and decreased compliance. Retained airway secretions and atelectasis frequently accompany flail chest. The patient has hypoxemia, and if gas exchange is greatly compromised, respiratory acidosis develops as a result of carbon dioxide retention. Hypotension, inadequate tissue perfusion, and metabolic acidosis often follow

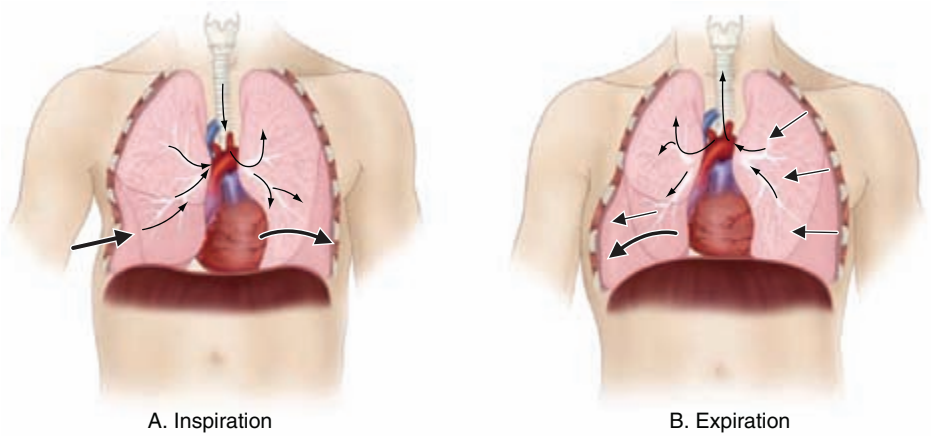


**Figure 23-8** Flail chest is caused by a free-floating segment of rib cage resulting from multiple rib fractures. **A,** Paradoxical movement on inspiration occurs when the flail rib segment is sucked inward and the mediastinal structures shift to the unaffected side. The amount of air drawn into the affected lung is reduced. **B,** On expiration, the flail segment bulges outward and the mediastinal structures shift back to the affected side.

as the paradoxical motion of the mediastinum decreases cardiac output.

## Medical Management

As with rib fracture, treatment of flail chest is usually supportive. Management includes providing ventilatory support, clearing secretions from the lungs, and controlling pain. Specific management depends on the degree of respiratory dysfunction. If only a small segment of the chest is involved, the objectives are to clear the airway through positioning, coughing, deep breathing, and suctioning to aid in the expansion of the lung, and to relieve pain by intercostal nerve blocks, high thoracic epidural blocks, or cautious use of IV opioids.

For mild to moderate flail chest injuries, the underlying pulmonary contusion is treated by monitoring fluid intake and appropriate fluid replacement while relieving chest pain. Pulmonary physiotherapy focusing on lung volume expansion, and secretion management techniques are performed. The patient is closely monitored for further respiratory compromise.

For severe flail chest injuries, endotracheal intubation and mechanical ventilation are required to provide internal pneumatic stabilization of the flail chest and to correct abnormalities in gas exchange. This helps treat the underlying pulmonary contusion, serves to stabilize the thoracic cage to allow the fractures to heal, and improves alveolar ventilation and intrathoracic volume by decreasing the work of breathing. This treatment modality requires endotracheal intubation and ventilator support. Differing modes of ventilation are used depending on the patient's underlying disease and specific needs (see Chapter 25).

In rare circumstances, surgery may be required to more quickly stabilize the flail segment. This may be used for patients who are difficult to ventilate or for high-risk patients with underlying lung disease who may be difficult to wean from mechanical ventilation.

Regardless of the type of treatment, the patient is carefully monitored by serial chest x-rays, arterial blood gas analysis, pulse oximetry, and bedside pulmonary function monitoring. Pain management is key to successful treatment. Patient-controlled analgesia, intercostal nerve blocks, epidural analgesia, and intrapleural administration of opioids may be used to relieve or manage thoracic pain.

# PULMONARY CONTUSION

Pulmonary contusion is a common thoracic injury and is frequently associated with flail chest. It is defined as damage to the lung tissues resulting in hemorrhage and localized edema. It is associated with chest trauma when there is rapid compression and decompression to the chest wall (ie, blunt trauma). Pulmonary contusion represents a spectrum of lung injury characterized by the development of infiltrates and various degrees of respiratory dysfunction and sometimes respiratory failure. It is often cited as the most common potentially life-threatening chest injury; however, mortality is often attributed to other associated injuries. A contusion is sustained in 30% to 70% of patients who experience blunt force trauma. Pulmonary contusion may not be evident initially on examination but develops in the posttraumatic period; it may involve a small portion of one lung, a massive section of a lung, one entire lung, or both lungs.

## Pathophysiology

The primary pathologic defect is an abnormal accumulation of fluid in the interstitial and intra-alveolar spaces. It is thought that injury to the lung parenchyma and its capillary network results in a leakage of serum protein and plasma. The leaking serum protein exerts an osmotic pressure that enhances loss of fluid from the capillaries. Blood, edema, and cellular debris (from cellular response to injury) enter the lung and accumulate in the bronchioles and alveoli, where they interfere with gas exchange. An increase in pulmonary vascular resistance and pulmonary artery pressure occurs. The patient has hypoxemia and carbon dioxide retention. Occasionally, a contused lung occurs on the other side of the point of body impact; this is called a contrecoup contusion.

## Clinical Manifestations

Pulmonary contusion may be mild, moderate, or severe. The clinical manifestations vary from decreased breath sounds, tachypnea, tachycardia, chest pain, hypoxemia, and blood-tinged secretions to more severe tachypnea, tachycardia, crackles, frank bleeding, severe hypoxemia (cyanosis), and respiratory acidosis. Changes in sensorium,

including increased agitation or combative irrational behavior, may be signs of hypoxemia.

In addition, patients with moderate pulmonary contusion have a large amount of mucus, serum, and frank blood in the tracheobronchial tree; patients often have a constant cough but cannot clear the secretions. Patients with severe pulmonary contusion have the signs and symptoms of ARDS; these may include central cyanosis, agitation, combativeness, and productive cough with frothy, bloody secretions.

### Assessment and Diagnostic Findings

The efficiency of gas exchange is determined by pulse oximetry and arterial blood gas measurements. Pulse oximetry is also used to measure oxygen saturation continuously. The initial chest x-ray may show no changes; changes may not appear for 1 or 2 days after the injury and appear as pulmonary infiltrates on chest x-ray.

### Medical Management

Treatment priorities include maintaining the airway, providing adequate oxygenation, and controlling pain. In mild pulmonary contusion, adequate hydration via IV fluids and oral intake is important to mobilize secretions. However, fluid intake must be closely monitored to avoid hypervolemia. Volume expansion techniques, postural drainage, physiotherapy including coughing, and endotracheal suctioning are used to remove the secretions. Pain is managed by intercostal nerve blocks or by opioids via patient-controlled analgesia or other methods. Usually, antimicrobial therapy is administered because the damaged lung is susceptible to infection. Supplemental oxygen is usually given by mask or cannula for 24 to 36 hours.

In patients with moderate pulmonary contusions, bronchoscopy may be required to remove secretions. Intubation and mechanical ventilation with PEEP (see Chapter 25) may also be necessary to maintain the pressure and keep the lungs inflated. Diuretics may be administered to reduce edema. A nasogastric tube is inserted to relieve gastrointestinal distention.

In patients with severe contusion, who may develop respiratory failure, aggressive treatment with endotracheal intubation and ventilatory support, diuretics, and fluid restriction may be necessary. Colloids and crystalloid solutions may be used to treat hypovolemia.

Antimicrobial medications may be prescribed for the treatment of pulmonary infection. This is a common complication of pulmonary contusion (especially pneumonia in the contused segment), because the fluid and blood that extravasates into the alveolar and interstitial spaces serve as an excellent culture medium.

## Penetrating Trauma: Gunshot and Stab Wounds

Gunshot and stab wounds are the most common causes of penetrating chest trauma. These wounds are classified according to their velocity. Stab wounds are generally considered low-velocity trauma because the weapon destroys a small area around the wound. Knives and switchblades cause most stab wounds. The appearance of the external wound may be very deceptive, because pneumothorax, hemothorax, lung contusion, and cardiac tamponade, along with severe and continuing hemorrhage, can occur from any small wound, even one caused by a small-diameter instrument such as an ice pick.

Gunshot wounds may be classified as low, medium, or high velocity. The factors that determine the velocity and resulting extent of damage include the distance from which the gun was fired, the caliber of the gun, and the construction and size of the bullet. A bullet can cause damage at the site of penetration and along its pathway, and a gunshot wound to the chest can produce a variety of pathophysiologic changes. The bullet may ricochet off bony structures and damage the chest organs and great vessels. If the diaphragm is involved in a gunshot wound or a stab wound, injury to the chest cavity must be considered.

### Medical Management

The objective of immediate management is to restore and maintain cardiopulmonary function. After an adequate airway is ensured and ventilation is established, examination for shock and intrathoracic and intra-abdominal injuries is necessary. The patient is undressed completely so that additional injuries are not missed. There is a high risk for associated intra-abdominal injuries with stab wounds below the level of the fifth anterior intercostal space. Death can result from exsanguinating hemorrhage or intra-abdominal sepsis.

The diagnostic workup includes a chest x-ray, chemistry profile, arterial blood gas analysis, pulse oximetry, and ECG. The patient's blood is typed and cross-matched in case blood transfusion is required. After the status of the peripheral pulses is assessed, a large-bore IV line is inserted. An indwelling catheter is inserted to monitor urinary output. A nasogastric tube is inserted and connected to low suction to prevent aspiration, minimize leakage of abdominal contents, and decompress the gastrointestinal tract.

Shock is treated simultaneously with colloid solutions, crystalloids, or blood, as indicated by the patient's condition. Diagnostic procedures are carried out as dictated by the needs of the patient (eg, CT scans of chest or abdomen, flat plate x-ray of the abdomen, abdominal tap to check for bleeding).

A chest tube is inserted into the pleural space in most patients with penetrating wounds of the chest to achieve rapid and continuing re-expansion of the lungs. The insertion of the chest tube frequently results in a complete evacuation of the blood and air. The chest tube also allows early recognition of continuing intrathoracic bleeding, which would make surgical exploration necessary. If the patient has a penetrating wound of the heart or great vessels, the esophagus, or the tracheobronchial tree, surgical intervention is required.

## Pneumothorax

Pneumothorax occurs when the parietal or visceral pleura is breached and the pleural space is exposed to positive atmospheric pressure. Normally the pressure in the pleural

space is negative or subatmospheric; this negative pressure is required to maintain lung inflation. When either pleura is breached, air enters the pleural space, and the lung or a portion of it collapses.

## Types of Pneumothorax

Types of pneumothorax include simple, traumatic, and tension pneumothorax.

### *Simple Pneumothorax*

A simple, or spontaneous, pneumothorax occurs when air enters the pleural space through a breach of either the parietal or visceral pleura. Most commonly, this occurs as air enters the pleural space through the rupture of a bleb or a bronchopleural fistula. A spontaneous pneumothorax may occur in an apparently healthy person in the absence of trauma due to rupture of an air-filled bleb, or blister, on the surface of the lung, allowing air from the airways to enter the pleural cavity. It may be associated with diffuse interstitial lung disease and severe emphysema.

### *Traumatic Pneumothorax*

A traumatic pneumothorax occurs when air escapes from a laceration in the lung itself and enters the pleural space or from a wound in the chest wall. It may result from blunt trauma (eg, rib fractures), penetrating chest or abdominal trauma (eg, stab wounds or gunshot wounds), or diaphragmatic tears. Traumatic pneumothorax may occur during invasive thoracic procedures (ie, thoracentesis, transbronchial lung biopsy, insertion of a subclavian line) in which the pleura is inadvertently punctured, or with barotrauma from mechanical ventilation.

A traumatic pneumothorax resulting from major injury to the chest is often accompanied by hemothorax (collection of blood in the pleural space resulting from torn intercostal vessels, lacerations of the great vessels, or lacerations of the lungs). Often both blood and air are found in the chest cavity (hemopneumothorax) after major trauma. Chest surgery can be classified as a traumatic pneumothorax as a result of the entry into the pleural space and the accumulation of air and fluid in the pleural space.

Open pneumothorax is one form of traumatic pneumothorax. It occurs when a wound in the chest wall is large enough to allow air to pass freely in and out of the thoracic cavity with each attempted respiration. Because the rush of air through the wound in the chest wall produces a sucking sound, such injuries are termed sucking chest wounds. In such patients, not only does the lung collapse, but the structures of the mediastinum (heart and great vessels) also shift toward the uninjured side with each inspiration and in the opposite direction with expiration. This is termed mediastinal flutter or swing, and it produces serious circulatory problems.

*NURSING ALERT*

**Traumatic open pneumothorax calls for emergency interventions. Stopping the flow of air through the opening in the chest wall is a lifesaving measure.**

### *Tension Pneumothorax*

A **tension pneumothorax** occurs when air is drawn into the pleural space from a lacerated lung or through a small opening or wound in the chest wall. It may be a complication of other types of pneumothorax. In contrast to open pneumothorax, the air that enters the chest cavity with each inspiration is trapped; it cannot be expelled during expiration through the air passages or the opening in the chest wall. In effect, a one-way valve or ball valve mechanism occurs where air enters the pleural space but cannot escape. With each breath, tension (positive pressure) is increased within the affected pleural space. This causes the lung to collapse and the heart, the great vessels, and the trachea to shift toward the unaffected side of the chest (mediastinal shift). Both respiration and circulatory function are compromised because of the increased intrathoracic pressure, which decreases venous return to the heart, causing decreased cardiac output and impairment of peripheral circulation. In extreme cases, the pulse may be undetectable—this is known as pulseless electrical activity.

*NURSING ALERT*

**Relief of tension pneumothorax is considered an emergency measure.**

## Clinical Manifestations

The signs and symptoms associated with pneumothorax depend on its size and cause. Pain is usually sudden and may be pleuritic. The patient may have only minimal respiratory distress with slight chest discomfort and tachypnea with a small simple or uncomplicated pneumothorax. If the pneumothorax is large and the lung collapses totally, acute respiratory distress occurs. The patient is anxious, has dyspnea and air hunger, has increased use of the accessory muscles, and may develop central cyanosis from severe hypoxemia.

In assessing the chest for any type of pneumothorax, the nurse assesses tracheal alignment, expansion of the chest, breath sounds, and percussion of the chest. In a simple pneumothorax, the trachea is midline, expansion of the chest is decreased, breath sounds may be diminished, and percussion of the chest may reveal normal sounds or hyperresonance depending on the size of the pneumothorax. In a tension pneumothorax, the trachea is shifted away from the affected side, chest expansion may be decreased or fixed in a hyperexpansion state, breath sounds are diminished or absent, and percussion to the affected side is hyperresonant. The clinical picture is one of air hunger, agitation, increasing hypoxemia, central cyanosis, hypotension, tachycardia, and profuse diaphoresis. Figure 23-9 compares open and tension pneumothorax.

## Medical Management

Medical management of pneumothorax depends on its cause and severity. The goal of treatment is to evacuate the air or blood from the pleural space. A small chest tube (28 Fr) is inserted near the second intercostal space; this space is used because it is the thinnest part of the chest wall, minimizes the danger of contacting the thoracic nerve, and

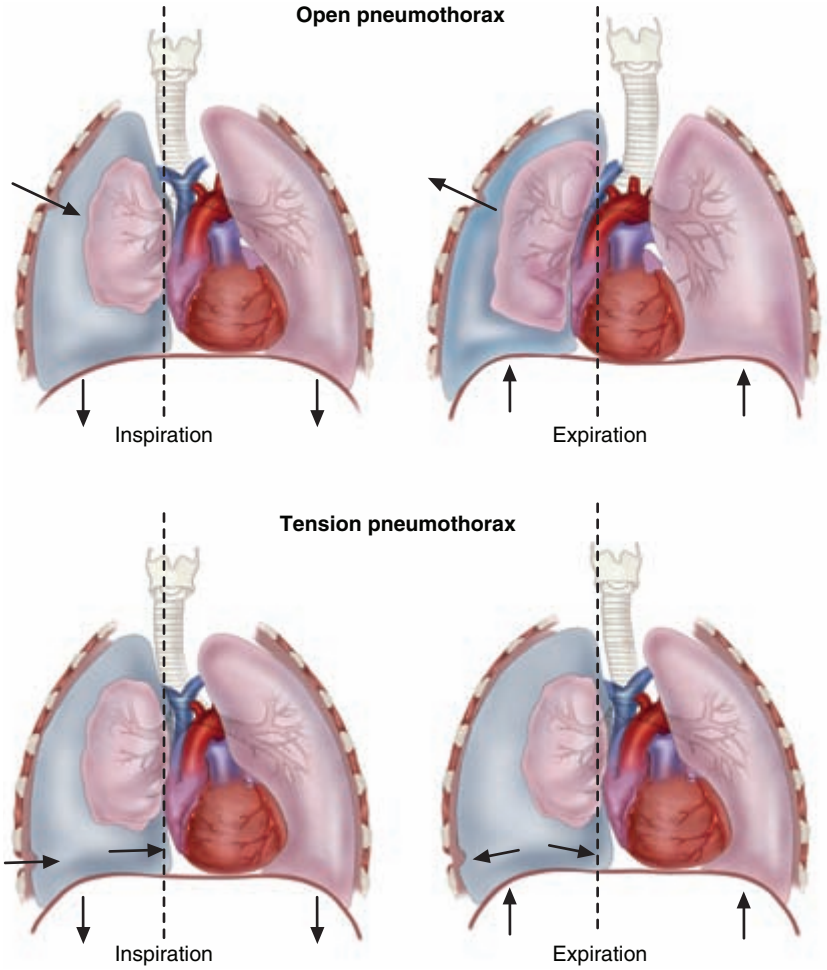


**Figure 23-9** Open pneumothorax (*top*) and tension pneumothorax (*bottom*). In open pneumothorax, air enters the chest during inspiration and exits during expiration. A slight shift of the affected lung may occur because of a decrease in pressure as air moves out of the chest. In tension pneumothorax, air enters but cannot leave the chest. As the pressure increases, the heart and great vessels are compressed and the mediastinal structures are shifted toward the opposite side of the chest. The trachea is pushed from its normal midline position toward the opposite side of the chest, and the unaffected lung is compressed.

leaves a less visible scar. If a patient also has a hemothorax, a large-diameter chest tube (32 Fr or greater) is inserted, usually in the fourth or fifth intercostal space at the midaxillary line. The tube is directed posteriorly to drain the fluid and air. Once the chest tube or tubes are inserted and suction is applied (usually to 20 mm Hg suction), effective decompression of the pleural cavity (drainage of blood or air) occurs.

If an excessive amount of blood enters the chest tube in a relatively short period, an autotransfusion may be needed. This technique involves taking the patient's own blood that has been drained from the chest, filtering it, and then transfusing it back into the vascular system.

In such an emergency, anything may be used that is large enough to fill the chest wound—a towel, a handkerchief, or the heel of the hand. If conscious, the patient is instructed to inhale and strain against a closed glottis. This action assists in re-expanding the lung and ejecting the air from the thorax. In the hospital, the opening is plugged by sealing it with gauze impregnated with petrolatum. A pressure dressing is applied. Usually, a chest tube connected to water-seal drainage is inserted to remove air and fluid. Antibiotics usually are prescribed to combat infection from contamination.

The severity of open pneumothorax depends on the amount and rate of thoracic bleeding and the amount of air in the pleural space. The pleural cavity can be decompressed by needle aspiration (thoracentesis) or by chest tube drainage of the blood or air. The lung is then able to re-expand and resume the function of gas exchange. As a rule of thumb, the chest wall is opened surgically (thoracotomy) if more than 1500 mL of blood is aspirated initially by thoracentesis (or is the initial chest tube output) or if chest tube output continues at greater than 200 mL/h. The urgency with which the blood must be removed is determined by the degree of respiratory compromise. An emergency thoracotomy may also be performed in the emergency department if a cardiovascular injury secondary to chest or penetrating trauma is suspected. The patient with a possible tension pneumothorax should immediately be given a high concentration of supplemental oxygen to treat the hypoxemia, and pulse oximetry should be used to monitor oxygen saturation. In an emergency situation, a tension pneumothorax can be decompressed or quickly converted to a simple pneumothorax by inserting a large-bore needle (14-gauge) at the second intercostal space, midclavicular line on the affected side. This relieves the pressure and vents the positive pressure to the external environment. A chest tube is then inserted and connected to suction to remove the remaining air and fluid, reestablish the negative pressure, and re-expand the lung. If the lung re-expands and air leakage from

the lung parenchyma stops, further drainage may be unnecessary. If a prolonged air leak continues despite chest tube drainage to underwater seal, surgery may be necessary to close the leak.

## Cardiac Tamponade

Cardiac tamponade is compression of the heart resulting from fluid or blood within the pericardial sac. It usually is caused by blunt or penetrating trauma to the chest. A penetrating wound of the heart is associated with a high mortality rate. Cardiac tamponade also may follow diagnostic cardiac catheterization, angiographic procedures, and pacemaker insertion, which can produce perforations of the heart and great vessels. Pericardial effusion with fluid compressing the heart also may develop from metastases to the pericardium from malignant tumors of the breast, lung, or mediastinum and may occur with lymphomas and leukemias, renal failure, TB, and high-dose radiation to the chest. Cardiac tamponade is discussed in detail in Chapter 30.

## Subcutaneous Emphysema

No matter what kind of chest trauma a patient has, when the lung or the air passages are injured, air may enter the tissue planes and pass for some distance under the skin (eg, neck, chest). The tissues give a crackling sensation when palpated, and the subcutaneous air produces an alarming appearance as the face, neck, body, and scrotum become misshapen by subcutaneous air. Fortunately, subcutaneous emphysema is of itself usually not a serious complication. The subcutaneous air is spontaneously absorbed if the underlying air leak is treated or stops spontaneously. In severe cases in which there is widespread subcutaneous emphysema, a tracheostomy is indicated if airway patency is threatened by pressure of the trapped air on the trachea.

## CRITICAL THINKING EXERCISES

**1** You are caring for a 42-year-old woman who underwent gastric bypass surgery. After being in the intensive care unit for 3 days, she was transferred to a general surgery unit. She is a former smoker and is taking multiple medications for high blood pressure and diabetes. When you started your shift, she was stable, but she has become increasingly anxious with some shortness of breath in the past hour. What are potential risk factors you might observe or identify in this patient? What assessment strategies would you use to evaluate changes in her respiratory status? What are the potential causes of the shortness of breath? What decision process would you use to determine when the physician should be contacted?

EBP **2** An 89-year-old woman is admitted to your unit from a long-term care facility. She is extremely frail and has multiple comorbidities. She has been diagnosed with community-acquired pneumonia. What evidence-based nursing interventions are important in her care and recovery? How would you determine the strength of the evidence for the interventions you choose?

EBP **3** A 48-year-old patient is admitted to the hospital with a diagnosis of malnutrition, possible aspiration pneumonia, and stage 4 pressure ulcer. The patient experienced a traumatic brain injury 4 years ago; he is not fully aware of his surroundings and has difficulty swallowing. Tube feedings have been prescribed in an effort to improve his nutritional status prior to débridement and skin grafting to the pressure ulcer. Identify evidence-based nursing interventions appropriate for this patient related to possible aspiration pneumonia. How would you assess the strength of that evidence?

EBP **4** You are on a surgical unit caring for a 68-year-old man who has undergone a left pneumonectomy for lung cancer. The patient has COPD and continues to smoke despite the diagnoses of COPD and lung cancer. What strategies would you use to prevent or minimize pulmonary complications in this patient? What parameters would you use to monitor the patient's postoperative respiratory status? What strategies would you consider to encourage the patient to stop smoking? What is the evidence base for the strategies that you consider? How would you evaluate the strength of the evidence?

**5** You are caring for a 34-year-old patient who experienced her third spontaneous pneumothorax. A chest tube was inserted, relieving the patient's acute symptoms. She has, however, become increasingly short of breath during the past hour. What physical assessment skills and strategies would you use to determine potential changes in the patient's respiratory condition? What are potential causes of this increasing shortness of breath? What would you do to prepare for an emergency situation in this patient?

The Smeltzer suite offers these additional resources to enhance learning and facilitate understanding of this chapter:

- thePoint online resource, thepoint.lww.com/Smeltzer12E
- Student CD-ROM included with the book
- *Study Guide to Accompany Brunner & Suddarth's Textbook of Medical-Surgical Nursing*
- *Handbook for Brunner & Suddarth's Textbook of Medical-Surgical Nursing*

## REFERENCES AND SELECTED READINGS

**Asterisk indicates nursing research*

### Books

Collaborative Staging Task Force of the American Joint Committee on Cancer. (2007). *Collaborative staging manual and coding instructions*. NIH Publication Number 04-5496. U.S. Department of Health and Human Services, National Institutes of Health, National Cancer Institute.

U.S. Department of Health and Human Services. (2006). *The health consequences of involuntary exposure to tobacco smoke. A report of the Surgeon General*. Atlanta, GA: Centers for Disease Control and Prevention, Coordinating Center for Health Promotion, National Center for Chronic Disease Prevention and Health Promotion, Office on Smoking and Health.

Wilkins, R. L., Dexter, J. R. & Gold, P. M. (2007). *Respiratory disease: A case study approach to patient care* (3rd ed.). Philadelphia: F. A. Davis Company.

### Journals and Electronic Documents

#### *General*

American Lung Association. (2005). *Occupational lung disease fast fact sheet*. Available at: www.lungusa.org
Badesch, D. B., Abman, S. H., Simonneau, G., et al. (2007). Medical therapy for pulmonary arterial hypertension: Updated ACCP evidence-based clinical practice guidelines. *Chest, 131*(6), 1917–1928.
Bourgault, A. M, Ipe, L., Weaver, J., et al. (2007). Development of evidence-based guidelines and critical care nurses' knowledge of enteral feeding. *Critical Care Nurse, 27*(4), 17–29.
Casey, K. R., Cantillo, K. O. & Brown, L. K. (2007). Sleep-related hypoventilations/hypoxemic syndromes. *Chest, 131*(6), 1936–1948.
Metheny, N. A., Meert, K. L. & Clouse, R. E. (2007). Complications related to feeding tube placement. *Current Opinions in Gastroenterology, 23*(2), 178–182.
Weinberger, S. E. (2006). A 47-year-old woman with sarcoidosis. *Journal of the American Medical Association, 296*(17), 2133–2140.
Wilson, K. C. & Hollingsworth, H. (2007). What's new in pulmonary, critical care and sleep medicine. *Up to Date*. Available at: www.uptodate.com

#### *Acute Respiratory Failure and ARDS*

Hansen-Flaschen, J. & Siegel, M. D. (2007). Acute respiratory distress syndrome: Definition, epidemiology, diagnosis and etiology. *Up to Date*. Available at: www.uptodate.com

#### *Lung Cancer*

American Cancer Society. (2008). *Cancer statistics, 2008*. Atlanta, GA: American Cancer Society. Available at: www.acs.org
American College of Chest Physicians (ACCP). (2007). Diagnosis and management of lung cancer: ACCP evidence-based clinical practice guidelines (2nd ed). *Chest, 132*(3), supplement, 1–404.
Jemal, A., Siegal, R., Ward, E., et al. (2008). Cancer statistics, 2008. *CA: A Cancer Journal for Clinicians, 58*(2), 71–96.
*Sarna, L., Cooley, M. E., Brown, J. K., et al. (2006). Quality of life and health status of dyads of women with lung cancer and family members. *Oncology Nursing Forum 33*(6), 1109–1116.
U.S. Cancer Statistics Working Group. (2007). *United States cancer statistics: 2004 incidence and morality*. Atlanta, GA: U.S. Department of Health and Human Services Centers for Disease Control and Prevention and National Cancer institute.
Yoder, L. H. (2006). An overview of lung cancer symptoms, pathophysiology and treatment. *MedSurg Nursing, 15*(4), 231–234.

#### *Pulmonary Embolism*

AlMahameed, A. & Carman, T. (2007). Outpatient management of stable acute pulmonary embolism: Proposed accelerated pathway for risk stratification. *American Journal of Medicine, 120*(10B), S18–S25.
American College of Chest Physicians (ACCP). (2009). The seventh ACCP Conference on antithrombotic and thrombolytic therapy: Evidence-based guidelines. *Chest, 135*(4), 1050–1060.
Carlbom, D. J. & Davidson, B. L. (2007). Pulmonary embolism in the critically ill. *Chest, 132*(1), 313–324.
Limpus, A., Chaboyer, W., McDonald, E., et al. (2006). Mechanical thromboprophylaxis in critically-ill patients: A systematic review and meta-analysis. *American Journal of Critical Care, 15*(4), 402–412.
Tapson, V. F., Decousus, H., Chong, B. H., et al. (2007). Venous thromboembolism prophylaxis in acutely ill hospitalized medical patients. *Chest, 132*(3), 936–945.
Thompson, T. & Hales, C. A. (2007). Overview of acute pulmonary embolism. *Up to Date*. Available at: www.uptodate.com

#### *Pulmonary Infections*

Bartlett, J. G. (2006). Aspiration pneumonia in adults. *Up to Date*. Available at: www.uptodate.com
Bartlett, J. G. (2007). Lung abscess. *Up to Date*. Available at: www.uptodate.com
Centers for Disease Control and Prevention. (2004). Guidelines for preventing health-care associated pneumonia, 2004. *MMWR Morbidity and Mortality Weekly Report, 53*(RR03), 1–36.
Centers for Disease Control and Prevention. (2007a). *Pneumonia fact sheet*. National Center for Health Statistics. Available at: www.cdc.gov/nchs/fastats/pneumonia.htm
Centers for Disease Control and Prevention. (2007b). Recommended adult immunization schedule October 2007-September 2008. *MMWR Morbidity and Mortality Weekly Report Quick Guide, 56*(41), 1–4.
File, T. M. (2007, September). Hospital-acquired, ventilator-associated, and healthcare-associated pneumonia in adults. *Up to Date*. Available at: www.uptodate.com
Fiore, A. E., Shay, D. K., Haber, P., et al. (2007). Prevention and control of influenza. Recommendations of the Advisory Committee on Immunization Practices (ACIP). *MMWR Morbidity and Mortality Weekly Report, 56*(RR-6), 1–54.
Hirsch, M. S. (2007). Severe acute respiratory syndrome (SARS). *Up to Date*. Available at: www.uptodate.com
Mandell, L. A., Wunderink, R. G., Anzueto, A., et al. (2007). Infectious Diseases Society of American Thoracic Society consensus guidelines on the management of community-acquired pneumonia in adults. *Clinical Infectious Diseases, 44*(Suppl 2), s27–72.
Minino, A. M., Heron, M. P., Murphy, S. L., et al. (2007). Deaths: Final data for 2004. *National Vital Statistics Reports, 55*(19).
Siegel, J. D., Rhinehart, E., Jackson, M., et al. (2006). *Management of multidrug-resistant organisms in healthcare settings* (pp. 1–74). Atlanta, GA: Centers for Disease Control and Prevention.

#### *Tuberculosis*

Basgoz, N. (2007). Pathogenesis and epidemiology of military tuberculosis. *Up to Date*. Available at: www.uptodate.com
Centers for Disease Control and Prevention. (2003). Treatment of tuberculosis. *MMWR Morbidity and Mortality Weekly Report, 52*(RR-11), 1–77.
Centers for Disease Control and Prevention. (2005a). Controlling tuberculosis in the United States: Recommendations from the American Thoracic Society, CDC and the Infectious Diseases Society of American. *MMWR Morbidity and Mortality Weekly Report, 54*(No RR-12), 1–82.
Centers for Disease Control and Prevention. (2005b). Guidelines for preventing the transmission of mycobacterium tuberculosis in health-care settings, 2005. *MMWR Morbidity and Mortality Weekly Report, 54*(RR17), 1–141.
Centers for Disease Control and Prevention. (2005c). Guidelines for using the QuantiFERON®-TB Gold Test for detecting *Mycobacterium tuberculosis* infection, United States. *MMWR Morbidity and Mortality Weekly Report, 54*(RR-15), 49–55.
World Health Organization. (2007). *Tuberculosis fact sheet*. Revised March 2007. Available at: www.who.int/mediacentre/factsheets

## RESOURCES

Agency for Healthcare Quality and Research, www.ahrq.gov
American Association for Respiratory Care, www.aarc.org
American Cancer Society, www.cancer.org
American College of Chest Physicians, www.chestnet.org
American Lung Association, www.lungusa.org
American Thoracic Society, www.thoracic.org
Centers for Disease Control and Prevention, www.cdc.gov
National Cancer Institute, www.cancer.gov
National Heart, Lung and Blood Institute, www.nhlbi.nih.gov
Occupational Safety and Health Administration (OSHA), www.osha.gov
Pulmonary Hypertension Association (PHA), www.phassociation.org
Respiratory Nursing Society, www.respiratorynursingsociety.org