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. Relate pleurisy, pleural effusion, and empyema to pulmonary infection.
5. Describe smoking and air pollution as causes of pulmonary disease.
6. Relate the therapeutic management techniques of acute respiratory distress syndrome to the underlying pathophysiology of the syndrome.
7. Describe risk factors for and measures appropriate for prevention and management of pulmonary embolism.
8. Describe preventive measures appropriate for controlling and eliminating the problem of occupational lung disease.
9. Discuss the modes of therapy and related nursing management for patients with lung cancer.
10. Describe the complications of chest trauma and their clinical manifestations and nursing management.
11. Describe nursing measures to prevent aspiration.
Conditions affecting the lower respiratory tract range from acute problems to long-term chronic disorders. Many of these disorders are serious and often life-threatening. The patient with a lower respiratory tract disorder requires care from nurses with astute assessment and clinical management skills as well as an understanding of the impact of the 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 frequently in patients who are immobilized and have a shallow, monotonous breathing pattern. Excess secretions or mucus 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 the adult as a result of reduced alveolar ventilation or any type of blockage that impedes the passage of air to and from the alveoli that normally receive air through the bronchi and network of airways. The trapped alveolar air becomes absorbed into the bloodstream, but outside air cannot replace the absorbed air because of the blockage. As a result, the isolated portion of the lung becomes airless and the alveoli collapse. This may occur with 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). Persistent low lung volumes, secretions or a mass obstructing or impeding airflow, and compression of lung tissue may all cause collapse or obstruction of the airways, which leads to atelectasis.

The postoperative patient is at high risk for atelectasis because of the numerous respiratory changes that may occur. A monotonous low tidal breathing pattern may cause airway closure and alveolar collapse. This results from the effects of anesthesia or analgesic agents, supine positioning, splinting of the chest wall because of pain, and abdominal distention. The postoperative patient may also have secretion retention, airway obstruction, and an impaired cough reflex or may be reluctant to cough because of pain. Figure 23-1 shows the pathogenic mechanisms and consequences of acute atelectasis in the postoperative patient.

Atelectasis resulting from bronchial obstruction by secretions may occur in patients with impaired cough mechanisms (eg, postoperative, musculoskeletal or neurologic disorders) or in debilitated, bedridden patients. Atelectasis may also result from excessive pressure on the lung tissue, which restricts normal lung expansion on inspiration. Such pressure may 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

Glossary

- **acute respiratory distress syndrome (ARDS):** 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
- **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 CT 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 cracking 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
- **pulmonary fibrosis resulting from exposure to asbestos fibers:** atelectasis
- **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
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 cough, sputum production, and low-grade fever. Fever is universally cited as a clinical sign of atelectasis, but there are few data to support this. Most likely the fever that accompanies atelectasis is due to infection or inflammation distal to the obstructed airway.

In acute atelectasis involving a large amount of lung tissue (lobar atelectasis), marked respiratory distress may be observed. In addition to the above signs and symptoms, dyspnea, tachycardia, tachypnea, pleural pain, and **central cyanosis** (a bluish skin hue that is a late sign of hypoxemia) may be anticipated. The patient characteristically has difficulty breathing in the supine position and is anxious. Signs and symptoms of chronic atelectasis are similar to those of acute atelectasis. Because the alveolar collapse is chronic, infection may occur distal to the obstruction. Thus, the signs and symptoms of a pulmonary infection also may be present.

**Assessment and Diagnostic Findings**

Decreased breath sounds and crackles are heard over the affected area. In addition, chest x-ray findings may reveal patchy infiltrates or consolidated areas. In the patient who is confined to bed,
atelectasis is usually diagnosed by chest x-ray or identified by physical assessment in the dependent, posterior, basilar areas of the lungs. Depending on the degree of hypoxemia, pulse oximetry (SpO2) may demonstrate a low saturation of hemoglobin with oxygen (less than 90%) or a lower-than-normal partial pressure of arterial oxygen (PaO2).

Prevention

Nursing measures to prevent atelectasis include frequent turning, early mobilization, and strategies to expand the lungs and to manage secretions. Deep-breathing maneuvers (at least every 2 hours) assist in preventing and treating atelectasis. The performance of these maneuvers requires a patient who is 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 may include directed cough, suctioning, aerosol nebulizer treatments followed by chest physical therapy (postural drainage and chest percussion), or bronchoscopy. In some settings, a metered-dose inhaler (MDI) is used to dispense a bronchodilator rather than an aerosol nebulizer treatment. Chart 23-1 summarizes measures to prevent atelectasis.

Management

The goal in treating the patient with atelectasis is to improve ventilation and remove secretions. The 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 expiratory pressure or PEP therapy (a simple mask and one-way valve system that provides varying amounts of expiratory resistance [usually 5 to 15 cm H2O]), continuous or intermittent positive-pressure-breathing (IPPB), or bronchoscopy may be used. Although IPPB may be used in some settings, few data support its use in the postoperative setting (Duffy & Farley, 1993). 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?
- Have other factors been evaluated that may impair ventilation or prohibit a good patient effort (eg, lack of turning, mobilization; excessive pain; excessive sedation)?

If the cause of atelectasis is bronchial obstruction from secretions, the secretions must be removed by coughing or suctioning to permit air to re-enter 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 medication or sodium bicarbonate may be used to assist the patient in the expectoration of secretions. If respiratory care measures fail to remove the obstruction, a bronchoscopy is performed. Severe or massive atelectasis may lead to acute respiratory failure, especially in a patient with underlying lung disease. Endotracheal intubation and mechanical ventilation may be necessary. Prompt treatment reduces the risk for acute respiratory failure or pneumonia.

If atelectasis has resulted from 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 above 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. The goal is to reopen the airways and provide ventilation to the collapsed area. 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. A patient with a viral infection has decreased resistance and can readily develop a secondary bacterial infection. Thus, adequate treatment of upper respiratory tract infection is one of the major factors in the prevention of acute bronchitis. Aside from infection, inhalation of physical and chemical irritants, gases, and other air contaminants can also cause acute bronchial irritation.

Pathophysiology

In acute tracheobronchitis, the inflamed mucosa of the bronchi produces mucopurulent sputum, often in response to Streptococcus pneumoniae, Haemophilus influenzae, and Mycoplasma pneumoniae. In addition, a fungal infection (eg, Aspergillus tracheobronchitis) may also cause tracheobronchitis. A sputum culture is essential to identify the specific causative organism.

Clinical Manifestations

Initially, the patient has a dry, irritating cough and expectorates a scanty amount of mucoid sputum. The patient complains of sternal soreness from coughing and has fever or chills and 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. With 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 are usually not prescribed because they may 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 the patient at risk for increasing airway obstruction and the development of a more severe lower respiratory tract infection, such as pneumonia. Suctioning and bronchoscopy may be needed to remove secretions. Rarely, endotracheal intubation may be required in cases of acute tracheobronchitis leading to acute respiratory failure. This may be necessary for 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. The patient is advised to rest. Increasing the vapor pressure (moisture content) in the air will reduce 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 analgesics or antipyretics may be indicated.

**Nursing Management**

Acute tracheobronchitis is frequently treated in the home setting. A primary nursing function is to encourage bronchial hygiene, such as increasing fluid intake and directed coughing to remove secretions. The nurse should encourage and assist the patient to sit up frequently to cough effectively and to prevent retention of mucopurulent sputum. If the patient is treated with 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 must caution the patient against overexertion, which can induce a relapse or exacerbation of the infection.

**PNEUMONIA**

Pneumonia is an inflammation of the lung parenchyma that is caused by a microbial agent. “Pneumonitis” is a more general term that describes an inflammatory process in the lung tissue that may predispose a patient to or place a patient at risk for microbial invasion. Pneumonia is the most common cause of death from infectious diseases in the United States. It is the seventh leading cause of death in the United States for all ages and both genders, resulting in almost 70,000 deaths per year. In persons 65 years of age and older, it is the fifth leading cause of death (National Center for Health Statistics, 2000; Minino & Smith, 2001). It is treated extensively on both an inpatient and outpatient basis.

Bacteria commonly enter the lower airway but do not cause pneumonia in the presence of an intact host defense mechanism. When pneumonia does occur, it is caused by various microorganisms, including bacteria, mycobacteria, chlamydiae, mycoplasma, fungi, parasites, and viruses. Several systems are used to classify pneumonias. Classically, pneumonia has been categorized into one of four categories: bacterial or typical, atypical, anaerobic/cavitary, and opportunistic. However, there is overlap in the microorganisms thought to be responsible for typical and atypical pneumonias. A more widely used classification scheme categorizes the major pneumonias as community-acquired pneumonia, hospital-acquired pneumonia, 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 of hospitalization or institutionalization. The need for hospitalization for CAP depends on the severity of the pneumonia. The agents that most frequently cause CAP requiring hospitalization are **S. pneumoniae**, *H. influenzae*, *Legionella*, *Mycoplasma pneumoniae*, and other gram-negative rods. The specific etiologic agent of CAP is identified in about 50% of the cases. The absence of a responsible caregiver in the home may be another indication for hospitalization. More than 5.5 million people develop CAP and as many as 1.1 million require hospitalization each year (Centers for Disease Control and Prevention [CDC], 1997; Marston, Plouffe, File et al., 1997).

Pneumonia caused by **S. pneumoniae** (pneumococcus) is the most common CAP in people younger than 60 without comorbidity and in those older than 60 with comorbidity. It is most prevalent during the winter and spring, when upper respiratory tract infections are most frequent. **S. pneumoniae** is a gram-positive, encapsulated, nonmotile coccus that resides naturally in the upper respiratory tract. The organism colonizes the upper respiratory tract and can cause the following types of illnesses: disseminated invasive infections, pneumonia and other lower respiratory tract infections, and upper respiratory tract infections, including otitis media and sinusitis (CDC, 1998). It may occur as a lobar or bronchopneumonic form in patients of any age and may follow a recent respiratory illness.

**H. influenzae** is another cause of CAP. It frequently affects elderly people or those with comorbid illnesses (eg, chronic obstructive pulmonary disease [COPD], alcoholism, diabetes mellitus). The presentation of this pneumonia is indistinguishable from that of other forms of bacterial CAP. The presentation may be subacute, with cough or low-grade fever for weeks before diagnosis. Chest x-rays may reveal multilobar, patchy bronchopneumonia or areas of **consolidation** (tissue that solidifies as a result of collapsed alveoli or pneumonia).

**Viruses** are the most common cause of pneumonia in infants and children but are relatively uncommon causes of CAP in adults. The chief causes of viral pneumonia in the immunocompetent adult are influenza viruses types A and B, adenovirus, parainfluenza virus, coronavirus, and varicella-zoster virus. 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. This is 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.
### Table 23-1 • Commonly Encountered Pneumonias

<table>
<thead>
<tr>
<th>TYPE</th>
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<th>CLINICAL FEATURES</th>
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<tr>
<td><strong>Community-Acquired Pneumonia</strong></td>
<td><strong>Streptococcus pneumonia</strong> (pneumococcal)</td>
<td>Highest occurrence in winter months. Incidence greatest in the elderly and in patients with COPD, heart failure, alcoholism, asplenia, following influenza. Leading infectious cause of illness worldwide among young children, persons with underlying chronic health conditions, and the elderly. Death occurs in 14% of hospitalized adults with invasive disease.</td>
<td>Abrupt onset, toxic appearance, pleuritic chest pain. Usually involves one or more lobes. Lobar infiltrate common on chest x-ray or bronchopneumonia pattern. Bacteremia in 15% to 25% of all patients.</td>
<td>Penicillins Alternate antibiotic therapy, such as cefotaxime or ceftriaxone; antipseudomonal fluoroquinolones (levofloxacin, gatifloxacin, moxifloxacin).</td>
<td>Complications include shock, pleural effusion, superinfections, pericarditis, and otitis media.</td>
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<tr>
<td><strong>Haemophilus influenzae</strong></td>
<td><strong>Haemophilus influenzae</strong></td>
<td>Incidence greatest in alcoholics, the elderly, patients in chronic care facilities and nursing homes, patients with diabetes or COPD, and children &lt;5 years old. Accounts for 5% to 20% of community-acquired pneumonias. Mortality rate: 30%.</td>
<td>Frequently insidious onset associated with upper respiratory tract infection 2 to 6 weeks 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.</td>
<td>Ampicillin, 3rd-generation cephalosporin, macrolides (azithromycin, clarithromycin), fluoroquinolones</td>
<td>Complications include lung abscess, pleural effusion, meningitis, arthritis, pericarditis, epiglottitis.</td>
</tr>
<tr>
<td><strong>Legionnaires’ disease</strong></td>
<td><strong>Legionella pneumophila</strong></td>
<td>Highest occurrence in summer and fall. May cause disease sporadically or as part of an epidemic. Incidence greatest in middle-aged and older men, smokers, and patients with chronic diseases, those receiving immunosuppressive therapy, or those in close proximity to excavation sites. Accounts for 15% of community-acquired pneumonias. Mortality rate: 15% to 50%.</td>
<td>Flulike symptoms. High fevers, mental confusion, headache, pleuritic pain, myalgias, dyspnea, productive cough, hemoptysis, leukocytosis. Bronchopneumonia, unilateral or bilateral disease, lobar consolidation.</td>
<td>Erythromycin +/− rifampin (in severely compromised patient) or clarithromycin, or a macrolide (azithromycin), or a fluoroquinolone (ofloxacin, levofloxacin, sparfloxacin).</td>
<td>Complications include hypertension, shock, and acute renal failure.</td>
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**Table 23-1 **Commonly Encountered Pneumonias (Continued)

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<tr>
<td><strong>Mycoplasma pneumoniae</strong></td>
<td><strong>Mycoplasma pneumoniae</strong></td>
<td>Increase in fall and winter.</td>
<td>Onset is usually insidious. Patients not usually as ill as in other pneumonias.</td>
<td>Erythromycin; macrolide, fluoroquinolone or tetracycline.</td>
<td>Complications include aseptic meningitis, meningoencephalitis, transverse myelitis, cranial nerve palsies, pericarditis, myocarditis.</td>
</tr>
<tr>
<td><strong>Viral pneumonia</strong></td>
<td><strong>Influenza viruses types</strong> A, B adenovirus, parainfluenza, cytomegalovirus, coronavirus</td>
<td>Incidence greatest in winter months. Epidemics occur every 2 to 3 years. Most common causative organisms in adults. Other organisms in children (eg, cytomegalovirus and respiratory syncytial virus). Accounts for 20% of community-acquired pneumonias.</td>
<td>Patchy infiltrate, small pleural effusion on chest x-ray. In majority of patients, influenza begins as an acute upper respiratory infection; others have bronchitis, pleurisy, etc., and still others develop gastrointestinal symptoms.</td>
<td>Amantadine; rimantadine; oseltamivir phosphate, ribavirin aerosol. Treated symptomatically. Does not respond to treatment with currently available antimicrobials.</td>
<td>Complications include a superimposed bacterial infection, bronchopneumonia.</td>
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<tr>
<td><strong>Chlamydial pneumonia</strong> (TWAR agent)</td>
<td><strong>C. pneumoniae</strong></td>
<td>Reported mainly in college students, military recruits, and the elderly. May be a common cause of community-acquired pneumonia or observed in combination with other pathogens. Mortality rate is low as the majority of cases are relatively mild. The elderly with coexistent infections, comorbidities, and re-infections may require hospitalization.</td>
<td>Hoarseness, fever, chills, pharyngitis, rhinitis, nonproductive cough, myalgias, arthralgias. Single infiltrate on chest x-ray; pleural effusion possible.</td>
<td>Tetracycline, erythromycin, macrolide, quinolone.</td>
<td>Complications include reinfection and acute respiratory failure.</td>
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Table 23-1 • Commonly Encountered Pneumonias (Continued)

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<tr>
<td>Hospital-Acquired Pneumonia</td>
<td>Pseudomonas aeruginosa</td>
<td>Incidence greatest in those with pre-existing lung disease, cancer (particularly leukemia); those with homograft transplants, burns; debilitated persons; and patients receiving antimicrobial therapy and treatments such as tracheostomy, suctioning, and in postoperative settings. Almost always of nosocomial origin. Accounts for 15% of hospital-acquired pneumonias. Mortality rate: 40% to 60%.</td>
<td>Diffuse consolidation on chest x-ray. Toxic appearance: fever, chills, productive cough, relative bradycardia, leukocytosis.</td>
<td>Aminoglycoside and anti-pseudomonal agents (ticarcillin, piperacillin, mezlocillin, ceftazidine).</td>
<td>Complications include lung cavitation. Has capacity to invade blood vessels, causing hemorrhage and lung infarction. Usually requires hospitalization.</td>
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<td>Staphylococcal pneumonia</td>
<td>Staphylococcus aureus</td>
<td>Incidence greatest in immunocompromised patients, IV drug users, and as a complication of epidemic influenza. Commonly nosocomial in origin. Accounts for 10% to 30% of hospital-acquired pneumonias. Mortality rate: 25% to 60%.</td>
<td>Severe hypoxemia, cyanosis, necrotizing infection. Bacteremia is common.</td>
<td>Nafcillin/oxacillin +/- rifampin or gentamicin; methicillin-resistant: vancomycin +/- rifampin or gentamicin.</td>
<td>Complications include pleural effusion/pneumothorax, lung abscess, empyema, meningitis, endocarditis. Frequently requires hospitalization. Treatment must be vigorous and prolonged because disease tends to destroy lung tissue.</td>
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<td>Klebsiella pneumonia (Friedlander’s bacillus-encapsulated gram-negative aerobic bacillus)</td>
<td>Klebsiella pneumoniae</td>
<td>Incidence greatest in the elderly; alcoholics; patients with chronic disease, such as diabetes, heart failure, COPD; patients in chronic care facilities and nursing homes. Accounts for 2% to 5% of community-acquired and 10% to 30% of hospital-acquired pneumonias. Mortality rate: 40% to 50%.</td>
<td>Tissue necrosis occurs rapidly. Toxic appearance: fever, cough, sputum production, bronchopneumonia, lung abscess. Lobar consolidation, bronchopneumonia pattern on chest x-ray.</td>
<td>Third-generation cephalosporins (cefotaxime, ceftriaxone) plus aminoglycoside, antipseudomonal penicillin, monobactam (aztreonam), or quinolone.</td>
<td>Complications include multiple lung abscesses with cyst formation, empyema, pericarditis, pleural effusion. May be fulminating, progressing to fatal outcome.</td>
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Hospital-acquired pneumonia (HAP), also known as nosocomial pneumonia, is defined as the onset of pneumonia symptoms more than 48 hours after admission to the hospital. HAP accounts for approximately 15% of hospital-acquired infections but is the most lethal nosocomial infection. It is estimated to occur in 0.5% to 1% of all hospitalized patients and in 15% to 20% of intensive care patients. Ventilator-associated pneumonia can be considered a type of nosocomial pneumonia that is associated with endotracheal intubation and mechanical ventilation.

The common organisms responsible for HAP include the pathogens Enterobacter species, Escherichia coli, Klebsiella species, Proteus, Serratia marcescens, P. aeruginosa, and methicillin-sensitive or methicillin-resistant Staphylococcus aureus. These respiratory infections occur when at least one of three conditions exists: host defenses are impaired, an inoculum of organisms reaches the patient’s lower respiratory tract and overwhelms the host’s defenses, or a highly virulent organism is present. Certain illnesses may predispose a patient to HAP because of impaired host defenses. Examples include severe acute or chronic illness, a variety of comorbid conditions, coma, malnutrition, prolonged hospitalization, hypotension, and metabolic disorders. The hospitalized patient is 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.

Dominant pathogens for HAP are gram-negative bacilli (P. aeruginosa and Enterobacteriaceae/Klebsiella species, Enterobacter, Proteus, Serratia) and S. aureus. Pseudomonal pneumonia occurs in patients who are debilitated, those with altered mental status, and those with prolonged intubation or with tracheostomies. 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. Although responsible for less than 10% of cases of CAP,

<table>
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<tr>
<th>Type</th>
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<th>Epidemiology</th>
<th>Clinical Features</th>
<th>Treatment Comments</th>
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<tr>
<td>Pneumocystis carinii Pneumonia (PCP)</td>
<td>Pneumocystis carinii</td>
<td>Incidence greatest in patients with AIDS and patients receiving immunosuppressive therapy for cancer, organ transplants, and other disorders. Frequently seen with cytomegalovirus infection. Mortality rate 15% to 20% hospitalized and fatal if not treated.</td>
<td>Pulmonary infiltrates on chest x-ray. Nonproductive cough, fever, dyspnea.</td>
<td>Trimethoprim/sulfamethoxazole (TMP-SMZ), dapsone-trimethoprim, pentamidine, primequine plus clindamycin.</td>
</tr>
<tr>
<td>Tuberculosis</td>
<td>Mycobacterium tuberculosis</td>
<td>Incidence increased in indigent, immigrant, and prison populations, people with AIDS, and the homeless. Mortality rate &lt;1% (depending on comorbidity)</td>
<td>Weight loss, fever, night sweats, cough, sputum production, hemoptysis, non-specific infiltrate (lower lobe), hilar node enlargement, pleural effusion on chest x-ray.</td>
<td>Rifampin, streptomycin, ethambutol, INH (isoniazid), pyrazinamide.</td>
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+/- = may add depending upon situation

**Table 23-1 • Commonly Encountered Pneumonias (Continued)**
Aspiration is described in more detail at the end of this chapter.

Changes, and lead to bacterial growth and a resulting pneumonia. This type of aspiration or may be aspirated into the lung, such as gastric contents, exogenous chemical contents, or irritating gases. Aspiration pneumonia may normally reside in the upper airways. Aspiration pneumonia may result from the entry of endogenous or exogenous substances resulting from the entry of endogenous or exogenous substances that enter the pulmonary circulation and are trapped in the pulmonary capillary bed, becoming a potential source of pneumonia.

Pneumonia often 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-containing 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 exits to the left side of the heart poorly oxygenated. The mixing of oxygenated and un oxygenated 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).

Pathophysiology

Upper airway characteristics normally prevent potentially infectious particles from reaching the normally sterile lower respiratory tract. Thus, patients with pneumonia caused by infectious agents often have an acute or chronic underlying disease that impairs host defenses. Pneumonia arises from normally present flora in a patient whose resistance has been altered, or it results from aspiration of flora present in the oropharynx. It may also result from bloodborne organisms that enter the pulmonary circulation and are trapped in the pulmonary capillary bed, becoming a potential source of pneumonia.

Risk Factors

Being knowledgeable about the factors and circumstances that commonly predispose a person to pneumonia will aid in identifying patients at high risk for this disorder (Chart 23-2).

**Physiology/Pathophysiology**

![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.](image)
Chart 23-2
Risk Factors for Pneumonia

<table>
<thead>
<tr>
<th>Risk Factor</th>
<th>Preventive Measure</th>
</tr>
</thead>
<tbody>
<tr>
<td>Conditions that produce mucus or bronchial obstruction and interfere with normal lung drainage (eg, cancer, cigarette smoking, COPD)</td>
<td>Promote coughing and expectoration of secretions. Encourage smoking cessation.</td>
</tr>
<tr>
<td>Immunosuppressed patients and those with a low neutrophil count (neutropenic)</td>
<td>Initiate special precautions against infection. Encourage smoking cessation.</td>
</tr>
<tr>
<td>Smoking; cigarette smoke disrupts both mucociliary and macrophage activity</td>
<td>Reposition frequently and promote lung expansion exercises and coughing. Initiate suctioning and chest physical therapy if indicated.</td>
</tr>
<tr>
<td>Prolonged immobility and shallow breathing pattern</td>
<td>Reposition frequently to prevent aspiration and administer medications judiciously, particularly those that increase risk for aspiration. Perform suctioning and chest physical therapy if indicated.</td>
</tr>
<tr>
<td>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</td>
<td>Promote frequent oral hygiene. Minimize risk for aspiration by checking placement of tube and proper positioning of patient.</td>
</tr>
<tr>
<td>Nothing-by-mouth (NPO) status; placement of nasogastric, orogastric, or endotracheal tube</td>
<td>Encourage reduced or moderate alcohol intake (in case of alcohol stupor, position patient to prevent aspiration).</td>
</tr>
<tr>
<td>Antibiotic therapy (in very ill people, the oropharynx is likely to be colonized by gram-negative bacteria)</td>
<td>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.</td>
</tr>
<tr>
<td>Alcohol intoxication (because alcohol suppresses the body’s reflexes, may be associated with aspiration, and decreases white cell mobilization and tracheobronchial ciliary motion)</td>
<td>Promote frequent turning, early ambulation and mobilization, effective coughing, breathing exercises, and nutritious diet. Make sure that respiratory equipment is cleaned properly; participate in continuous quality improvement monitoring with the respiratory care department.</td>
</tr>
<tr>
<td>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</td>
<td></td>
</tr>
<tr>
<td>Advanced age, because of possible depressed cough and glottic reflexes and nutritional depletion</td>
<td></td>
</tr>
<tr>
<td>Respiratory therapy with improperly cleaned equipment</td>
<td></td>
</tr>
</tbody>
</table>

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 and often follow a viral illness.

Pneumonia is common 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 developing 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.

The CDC has identified three specific strategies for preventing HAP: (1) staff education and infection surveillance, (2) interruption of transmission of microorganisms through person-to-person transmission and equipment transmission, and (3) modification of host risk of infection (CDC, 1997). Providing anticipatory and preventive care is an important nursing measure.

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, pulmonary, diabetes mellitus, chronic liver disease)
- 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 (CDC, 1998)

The vaccine provides specific prevention against pneumococcal pneumonia and other infections caused by this organism (otitis media, other upper respiratory tract infections). Vaccines should be avoided in the first trimester of pregnancy.

Clinical Manifestations

Pneumonia varies in its signs and symptoms depending on the organism and the patient’s underlying disease. However, regardless of the type of pneumonia (CAP, HAP, immunocompromised host, aspiration), a specific type of pneumonia cannot be diagnosed by clinical manifestations alone. For example, the patient with streptococcal (pneumococcal) pneumonia usually has a sudden onset of shaking chills, rapidly rising fever (38.5°C to 40.5°C [101°F 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 beats/min for every degree
of temperature (Celsius) elevation. A relative bradycardia for the amount of fever 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 days, mucoid or mucopurulent sputum is expectorated. In severe pneumonia, the cheeks are flushed and the lips and nailbeds demonstrate central cyanosis (a late sign of poor oxygenation [hypoxemia]).

Typically, the patient has orthopnea (shortness of breath when reclining); he or she prefers to be propped up in bed leaning forward (orthopneic position), trying 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; this is not a reliable indicator of the etiologic agent. Rusty, blood-tinted sputum may be expectorated with streptococcal (pneumococcal), staphylococcal, and *Klebsiella* pneumonia.

Signs and symptoms of pneumonia may also depend on underlying conditions. Differing signs occur in patients with other conditions, such as cancer, or in those who are undergoing treatment with immunosuppressants, which lower the resistance to infection. Such patients have fever, crakles, 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 studies, blood culture (bloodstream invasion, called bacteremia, occurs frequently), and sputum examination. The sputum sample is obtained by having the patient: (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 Chap. 21). Bronchoscopy is often used in patients with acute severe infection, patients with chronic or refractory infection, or immunocompromised patients when a diagnosis cannot be made from an expectorated or induced specimen.

### Medical Management

The treatment of pneumonia includes administration of the appropriate antibiotic as determined by the results of the Gram stain. However, an etiologic agent is not identified in 50% of CAP cases and empiric therapy must be initiated. Therapy for CAP is continuing to evolve. Guidelines exist to guide antibiotic choice; however, the resistance patterns, prevalence of etiologic agents, patient risk factors, and costs and availability of newer antibiotic agents must all be taken into consideration.

Several organizations have published guidelines for the medical management of CAP (Bartlett et al., 2000; American Thoracic Society, 2001). Recommendations are classified by existing risk factors, setting (inpatient vs. outpatient treatment), or specific pathogens. Examples of risk factors that may increase the risk of infection with certain types of pathogens appear in Chart 23-3.

Recommendations for treatment of outpatients with CAP who have no cardiopulmonary disease or other modifying factors include a macrolide (erythromycin, azithromycin [Zithromax], or clarithromycin [Biaxin]), doxycycline (Vibramycin), or a fluoroquinolone (eg, gatifloxacin [Tequin], levofloxacin [Levaquin]) with enhanced activity against *S. pneumoniae* (Bartlett et al., 2000; American Thoracic Society, 2001). Erythromycin should be avoided in areas where *H. influenzae* and *S. aureus* are more prevalent (Kenreich & Wagner, 2000; Lynch, 2000). For those outpatient patients who have cardiopulmonary disease or other modifying factors, treatment should include a beta-lactam (oral cepodoxime [Vantin], cefuroxime [Zinacef, Ceftin], high-dose amoxicillin or amoxicillin/clavulanate [Augmentin, Clavulin]) plus a macrolide or doxycycline. Also, a beta-lactam plus an antipseudomoccal fluoroquinolone can be used (American Thoracic Society, 2001). These are guidelines; treatment for individual patients may be modified.

For patients with CAP who are hospitalized and do not have cardiopulmonary disease or modifying factors, management consists of intravenous azithromycin (Zithromax) or monotherapy with an antipseudomoccal fluoroquinolone. For inpatients with cardiopulmonary disease or modifying factors, the treatment involves an intravenous beta-lactam plus an intravenous or oral macrolide or doxycycline. An intravenous antipseudomoccal fluoroquinolone may also be used alone (American Thoracic Society, 2001). For acutely ill patients admitted to the intensive care unit, management includes an intravenous beta-lactam plus either an intravenous macrolide or fluoroquinolone. For patients
at high risk for *P. aeruginosa*, more select antipseudomonal antibiotics are administered intravenously.

If specific pathogens have been identified for the CAP, more specific agents may be utilized. Mycoplasma pneumonia is treated with doxycycline or a macrolide. PCP responds best to pentamidine and trimethoprim–sulfamethoxazole (TMP-SMZ). Amantadine and rimantadine are effective with influenza A and have been shown to reduce the duration of fever and other systemic complications when administered within 24 to 48 hours of the onset of an uncomplicated influenza infection. These medications also reduce the duration and quantity of virus shedding in the respiratory secretions. They are most effective when used in combination with influenza vaccine. Ganciclovir is used to treat cytomegalovirus in the non-AIDS patient; cytomegalovirus immunoglobulin may also be used.

HAP has a different etiology from CAP. In suspected HAP or nosocomial pneumonia, empirical treatment is usually initiated with a broad-spectrum intravenous antibiotic and may be monotherapy or combination therapy. In patients who are mildly to moderately ill with a low risk of *Pneumononas*, the following antibiotics may be used: second-generation cephalosporins (eg, cefuroxime [Cefxin, Zinacef] or cefamandole [Mandol]), non-pseudomonal third-generation cephalosporins (ceftriaxone [Rocephin], cefotaxime [Clavoral], ampicillin–sulbactam [Unasyn]), or fluoroquinolones (eg, ciprofloxacin [Cipro], levofloxacin [Levaquin]). For combination therapy, any of the above may be used with an aminoglycoside.

For patients at high risk for *Pseudomonas* infection, an antipseudomonal penicillin plus an aminoglycoside (amikacin [Amikin], gentamicin) or beta-lactamase inhibitor (ampicillin/sulbactam [Unasyn], ticarcillin/clavulanate [Timentin]) may be used. Other types of combination therapy may also be used depending upon the individual characteristics of the patient.

Of concern is the rampant rise in respiratory pathogens that are resistant to available antibiotics. Examples include vancomycin-resistant enterococcus (VRE) and drug-resistant *S. pneumoniae* (McGeer & Low, 2000). There is a tendency for clinicians to aggressively use antibiotics inappropriately or to 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 to use evidence-based guidelines in the treatment of respiratory infection is important. Monitoring and surveillance of susceptibility patterns for pathogens are also important.

Therapy with parenteral agents usually is changed to oral antimicrobial agents when there is evidence of a clinical response and the patient is able to tolerate oral medications. The recommended duration of treatment for pneumococcal pneumonia is 72 hours after the patient becomes afebrile. Most other forms of pneumonia caused by bacterial pathogens are treated for 1 to 2 weeks after the patient becomes afebrile. Atypical pneumonia is usually treated for 10 to 21 days (Bartlett, Dowell, Mandell et al., 2000).

Treatment of viral pneumonia is primarily supportive. Antibiotics are ineffective in viral upper respiratory infections and pneumonia and 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 when a secondary bacterial pneumonia, bronchitis, or sinusitis is present. 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 may cause rebound nasal congestion. Treatment of viral pneumonia (with the exception of antimicrobial therapy) is the same as that for bacterial pneumonia. The patient is placed on bed rest 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 performed to determine the need for oxygen and to evaluate the effectiveness of the therapy. A high concentration of oxygen is contraindicated in patients with COPD because it may worsen alveolar ventilation by decreasing the patient's ventilatory drive, leading to further respiratory decompensation. Respiratory support measures include high oxygen concentrations (fraction of inspired oxygen [FiO2]), endotracheal intubation, and mechanical ventilation. Different modes of mechanical ventilation may be required; see Chapter 25.

Figure 23-3 provides an algorithm for patients with suspected CAP.

**Gerontologic Considerations**

Pneumonia in the elderly patient may occur as a primary problem or as a complication of a chronic disease process. Pulmonary infections in the elderly frequently are difficult to treat and have a higher mortality rate than in younger patients. 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 the elderly patient. Also, the presence of some signs may be misleading. Abnormal breath sounds, for example, may be due to microatelectasis that occurs in the aged as a result of decreased mobility, decreased lung volumes, and other respiratory function changes. Because chronic heart failure is often seen in the elderly, chest x-rays may be obtained to assist in differentiating it from pneumonia as the cause of clinical signs and symptoms.

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

**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 and when a comorbid disease complicates the pneumonia.

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 intravenously by continuous 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 in apparent danger of dying of the infection. Patients may require endotracheal intubation and mechanical ventilation. Congestive heart failure, cardiac dysrhythmias, pericarditis, and myocarditis also are complications of pneumonia that may lead to shock.

ATELECTASIS AND PLEURAL EFFUSION
Atelectasis (from obstruction of a bronchus by accumulated secretions) may occur at any stage of acute pneumonia. 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. (Empyema is discussed in greater detail in the section Pleural Conditions, below.) 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. Sometimes surgical management is required.

SUPERINFECTION
Superinfection may occur with the administration of very large doses of antibiotics, such as penicillin, or with combinations of antibiotics. Superinfection may also occur in the patient who has been receiving numerous courses and types of antibiotics. In such cases, bacteria may become resistant to the antibiotic therapy. If the patient improves and the fever diminishes after initial antibiotic therapy, but subsequently there is a rise in temperature with increasing cough and evidence that the pneumonia has spread, a superinfection is likely. Antibiotics are changed appropriately or discontinued entirely in some cases.

NURSING PROCESS: THE PATIENT WITH PNEUMONIA
Assessment
Nursing assessment is critical in detecting pneumonia. A fever, chills, or night sweats in a patient who also has respiratory symptoms should alert the nurse to the possibility of bacterial pneumonia. A respiratory assessment will further identify the clinical manifestations of pneumonia: pleuritic-type pain, fatigue, tachypnea, use of accessory muscles for breathing, bradycardia or relative bradycardia, coughing, and purulent sputum. It is important to identify the severity, location, and cause of the chest pain, along
with any medications or procedures that provide relief. The nurse should monitor 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)
- Changes in the chest x-ray findings

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

**Diagnosis**

**NURSING DIAGNOSES**

Based on the assessment data, the patient’s major nursing diagnoses may include:

- Ineffective airway clearance related to copious tracheobronchial secretions
- Activity intolerance related to impaired respiratory function
- Risk for deficient fluid volume related to fever and dyspnea
- 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:

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

**Planning and Goals**

The major goals for the patient 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 facemask (using either compressed air or oxygen) delivers warm, humidified air to the tracheobronchial tree, helps to 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. A directed cough may be necessary 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 patient’s lower rib cage (anteriorly or posteriorly) to focus the patient on a slow deep breath, and then manually assisting the patient by applying external pressure during the expiratory phase.

Chest physiotherapy (percussion and postural drainage) is important in loosening and mobilizing secretions (see Chap. 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, or 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.

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 Chap. 25). It may take time for secretions to mobilize and move into the central airways for expectoration. Thus, it is important for the nurse to monitor the patient for cough and sputum production after the completion of chest physiotherapy.

The nurse administers and titrates oxygen therapy as prescribed. The effectiveness of oxygen therapy is monitored by improvement in clinical signs and symptoms, and adequate oxygenation values 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) and should change positions frequently to enhance secretion clearance and ventilation/perfusion in the lungs. 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 a patient 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.

**MAINTAINING NUTRITION**

Patients with shortness of breath and fatigue often have a decreased appetite and will take 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, fluids and nutrients may be administered intravenously if necessary.

**PROMOTING THE PATIENT’S KNOWLEDGE**

The patient and family are instructed about the cause of pneumonia, management of symptoms of pneumonia, and the need for follow-up (discussed later). The patient also needs informa-
tion about factors (both patient risk factors and external factors) that may have contributed to developing pneumonia and strategies to promote recovery and to prevent recurrence. If hospitalized for treatment, the patient 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 need to be given simply and in language that the patient can understand. If possible, written instructions and information should be provided. 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**

Patients usually begin to respond to treatment within 24 to 48 hours after antibiotic therapy is initiated. The patient is observed for response to antibiotic therapy. The patient is monitored for changes in physical status (deterioration of condition or resolution of symptoms) and for persistent recurrent fever, which may be due to medication allergy (signaled possibly by a rash); medication resistance or slow response (greater than 48 hours) of the susceptible organism to therapy; superinfection; pleural effusion; or pneumonia caused by an unusual organism, such as *P. carinii* 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 described earlier, lung cancers may invade or compress airways, causing an obstructive atelectasis that may lead to a pneumonia.

In addition to monitoring for continuing symptoms of pneumonia, the nurse also monitors for other complications, such as shock and multisystem failure, atelectasis, pleural effusion, and superinfection, 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 intravenous 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.

**Atelectasis and Pleural Effusion**

The patient is assessed for atelectasis, and preventive measures are initiated to prevent its development. 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 Chap. 25 for more information on care of the patient with a chest tube).

**Superinfection**

The patient is monitored for manifestations of superinfection (ie, minimal improvement in signs and symptoms, rise in temperature with increasing cough, increasing fremitus and adventitious breath sounds on auscultation of the lungs). These signs are reported, and the nurse assists in implementing therapy to treat superinfection.

**Confusion**

The 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 the patient’s 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 intravenous antibiotics as an inpatient and then be discharged to continue the intravenous antibiotics in the home setting. 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 this patient in both settings. In addition, if oral antibiotics are prescribed, it is important to teach the patient about their proper administration and potential side effects.

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. It is important to instruct the patient to return to the clinic or caregiver’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 the patient susceptible to recurring respiratory tract infections.

**Continuing Care**

Patients who are severely debilitated or who cannot care for themselves 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. The nurse encourages the patient to obtain an influenza vaccine 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 encourages the patient to seek medical advice about receiving the vaccine (Pneumovax) against *S. pneumoniae*.
Tuberculosis (TB) is an infectious disease that primarily affects the body, including the meninges, kidneys, bones, and lymph nodes. The primary infectious agent, Mycobacterium 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 spreads from person to person by airborne transmission. An infected person releases droplet nuclei (generally particles 1 to 5 micrometers in diameter) through talking, coughing, sneezing, laughing, or singing. Larger droplets settle; smaller droplets remain suspended in the air and are inhaled by the susceptible person. Risk factors for TB are listed in Chart 23-4. Chart 23-5 summarizes the CDC’s recommendations for prevention of TB transmission in health care settings.

### Evaluation

#### EXPECTED PATIENT OUTCOMES

Expected patient outcomes may include:

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 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. Has normal 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

### Transmission and Risk Factors

TB spreads from person to person by airborne transmission. An infected person releases droplet nuclei through talking, coughing, sneezing, laughing, or singing. Larger droplets settle; smaller droplets remain suspended in the air and are inhaled by the susceptible person. Risk factors for TB are listed in Chart 23-4.
Pathophysiology

A susceptible person inhales mycobacterium bacilli 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 around the granulomas. Granulomas 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 col-lagenous scar. At this point, the bacteria become dormant, and there is no further progression of active disease.

After initial exposure and infection, the person may develop active disease 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 the 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 characterized by long remissions when the disease is arrested, only to be 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 within...
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 pulmonary symptoms are usually chronic and may have been present for weeks to months. The elderly usually present with less pronounced symptoms than do younger patients. Extrapulmonary disease occurs in up to 16% of cases in the United States. In patients with AIDS, extrapulmonary disease is more prevalent and may occur in up to 70% of cases (Niederman & Sarosi, 2000; Small & Fujiwara, 2001).

**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 person is infected with TB, the chest x-ray usually reveals lesions in the upper lobes and the acid-fast bacillus smear contains mycobacterium.

**TUBERCULIN SKIN TEST**

The Mantoux test is used to determine if a person has been infected with the TB bacillus. The Mantoux test is a standardized 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. 23-4). Intermediate-strength (5 TU) 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 wheal or bleb. 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 noted. 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. 23-4), and the size of the induration is documented. Erythema without induration is not considered significant.

**Interpretation of Results.** 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 individuals who are considered at risk. An induration of 10 mm or greater is usually considered significant in individuals who have normal or mildly impaired immunity. A significant reaction indicates that a patient has been exposed to *M. tuberculosis* recently or in the past or has been vaccinated with bacille Calmette-Guerin (BCG) vaccine. The BCG vaccine is given to produce a greater resistance to developing TB. It is effective in up to 76% of those who receive it. The vaccine is used in Europe and Latin America but not routinely in the United States.

A reaction of 5 mm or greater is defined as positive for patients who are HIV-positive or have HIV risk factors and are of unknown HIV status, those who are close contacts with an active case, and those who have chest x-ray results consistent with tuberculosis.
A significant (positive) reaction does not necessarily mean that active disease is present in the body. Most (more than 90%) 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 adequate to produce a positive skin test. This is referred to as anergy.

The accuracy of the skin test depends on the skill of the person interpreting the test result. One study (Kendig, Kirkpatrick, Carter et al., 1998) revealed that health care professionals tend to underestimate the size of induration: only 7% of a sample of 107 health care providers charted the correct size of induration.

**CLASSIFICATION OF TB**

Data from the history, physical examination, skin 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 (American Thoracic Society, 2000).

- 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. Many elderly patients may have no reaction (loss of immunologic memory) or delayed reaction for up to a week (recall phenomenon). A second skin test is performed in 1 to 2 weeks.

**Medical Management**

Pulmonary TB is treated primarily with chemotherapeutic agents (antituberculosis agents) for 6 to 12 months. A prolonged treatment duration is necessary to ensure eradication of the organism and to prevent relapse. A worldwide concern and challenge in TB therapy is the continuing (since the 1950s) and increasing resistance of *M. tuberculosis* to TB medications. 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 a person who has not had previous treatment
- **Secondary or acquired drug resistance**: resistance to one or more antituberculosis agents in a patient undergoing therapy
- **Multidrug resistance**: resistance to two agents, isoniazid (INH) and rifampin. The populations at highest 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, five first-line medications are used (Table 23–2): INH, rifampin, pyrazinamide, and either streptomycin or ethambutol.

Combination medications, such as INH and rifampin (Rifamate) or INH, pyrazinamide and rifampin and medications administered twice a week (eg, rifapentine) are available to help improve patient adherence. Capreomycin, ethionamide, paraaminosalicylate sodium, and cycloserine are second-line medications. Additional potentially effective medications include other aminoglycosides, quinolones, rifabutin, clofazimine, and combinations of medications.

Recommended treatment guidelines for newly diagnosed cases of pulmonary TB (CDC, 2000) consist of a multiple-medication regimen of INH, rifampin, pyrazinamide, and either streptomycin or ethambutol. This initial intensive-treatment regimen is usually administered daily for 8 weeks. If cultures demonstrate that the organism is sensitive to the medications before the 8 weeks of therapy have been completed, either ethambutol or streptomycin can be discontinued. After 8 weeks of this medication regimen, pyrazinamide can be discontinued and INH and rifampin are administered for an additional 4 months. The medication regimen, however, may continue for 12 months. A person is considered noninfectious after 2 to 3 weeks of continuous medication therapy. Vitamin B (pyridoxine) is usually administered with INH to prevent INH-associated peripheral neuropathy (see Table 23–2).

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

- Household family members of patients with active disease
- HIV-infected patients with a PPD test reaction of 5 mm of induration or more
- Patients with fibrotic lesions detected on a chest x-ray, suggestive of old TB, and a PPD reaction of 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 (also called skin test converters)
- Drug (intravenous or injectable) users with PPD test results of 10 mm of induration or more
- Patients with high-risk comorbid conditions with a PPD result of 10 mm of induration or more

Other candidates for preventive INH therapy are those age 35 years or younger with PPD test results of 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 enzyme, blood urea nitrogen, and creatinine levels are monitored monthly. Sputum culture results are monitored for acid-fast bacilli to evaluate the effectiveness of treatment and the patient’s compliance with therapy.

In 1998, the federal Advisory Council for the Elimination of Tuberculosis published recommendations for the development of TB vaccines. The recommendations include a focus on a “postinfection vaccine” to prevent people infected with TB from developing active disease (CDC, 1998). To date, this vaccine has
not become clinically available. In 2000, recommendations were released regarding the treatment of latent TB infection (American Thoracic Society and CDC, 2000). Isoniazid (INH) for 6 to 12 months has been the mainstay of treatment for latent TB infection. However, this long duration of treatment has been limited due to poor adherence and concerns of toxicity. The American Thoracic Society and CDC released newer guidelines in the 2000 document, which focused on treating a latent infection over a shorter period of time. The CDC released case reports of liver injury associated with the 2-month rifampin-pyrazinamide (RIF-PZA) dosing regimen in August 2001 (MMWR, 2001). This prompted a review and changes to the 2000 guidelines. In summary, a 2-month RIF-PZA treatment regimen for latent TB infection should be used with caution, especially in patients who are concurrently taking medications for liver disease or those with a history of alcoholism. For patients not infected with HIV, 9 months of daily INH remains the preferred treatment, and 4 months of daily RIF is an acceptable alternative. No more than a 2-week supply of RIF-PZA should be dispensed at any one time to facilitate periodic clinical assessments. Lastly, serum aminotransferase and bilirubin should be measured at baseline and at 2, 4, and 6 weeks of treatment in patients taking RIF-PZA (MMWR, 2001).

**NURSING PROCESS: THE PATIENT WITH TUBERCULOSIS**

**Assessment**

The nurse performs a complete history and physical examination. 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, egophony, and dullness on percussion. Enlarged, painful lymph nodes may be palpated as well. The nurse also assesses the patient’s living

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**Table 23-2 • First-Line Antitubercular Medications**

<table>
<thead>
<tr>
<th>COMMONLY USED AGENTS</th>
<th>ADULT DAILY DOSAGE*</th>
<th>MOST COMMON SIDE EFFECTS</th>
<th>DRUG INTERACTIONS†</th>
<th>REMARKS*</th>
</tr>
</thead>
<tbody>
<tr>
<td>isoniazid (INH)</td>
<td>5 mg/kg (300 mg maximum daily)</td>
<td>Peripheral neuritis, hepatic enzyme elevation, hepatitis, hypersensitivity</td>
<td>Phenytoin—synergistic Antabuse Alcohol</td>
<td>Bactericidal. Pyridoxine as prophylaxis for neuritis. Monitor AST (SGOT) and ALT (SGPT).</td>
</tr>
<tr>
<td>rifampin (Rifadin)</td>
<td>10 mg/kg (600 mg maximum daily)</td>
<td>Hepatitis, febrile reaction, purpura (rare), nausea, vomiting</td>
<td>Rifampin increases metabolism of oral contraceptives, quinidine, corticosteroids, coumarin derivatives and methadone, digoxin, oral hypoglycemics; PAS may interfere with absorption of rifampin.</td>
<td>Bactericidal. Orange urine and other body secretions. Discoloring of contact lenses. Monitor AST (SGOT) and ALT (SGPT).</td>
</tr>
<tr>
<td>rifabutin (Mycobutin)</td>
<td>5 mg/kg (300 mg maximum daily)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>streptomycin</td>
<td>15 mg/kg (1 g maximum daily)*</td>
<td>8th cranial nerve damage (may lead to deafness), nephrotoxicity</td>
<td>Neuromuscular blocking agents; may be potentiated to cause prolonged paralysis</td>
<td>Bactericidal in alkaline pH. Use with caution in elderly or in those with renal disease. Monitor vestibular function, audiograms, BUN and creatinine.</td>
</tr>
<tr>
<td>pyrazinamide</td>
<td>15 to 30 mg/kg (2.0 g maximum daily)*</td>
<td>Hyperuricemia, hepatotoxicity, skin rash, arthralgias, GI distress</td>
<td></td>
<td></td>
</tr>
<tr>
<td>ethambutol (Myambutol)</td>
<td>15 to 25 mg/kg (no maximum daily dose, but base on lean body wt)*</td>
<td>Optic neuritis (may lead to blindness; very rare at 15 mg/kg), skin rash</td>
<td></td>
<td>Bacteriostatic. Use with caution with renal disease or when eye testing is not feasible. Monitor visual acuity, color discrimination.†</td>
</tr>
<tr>
<td>Combinations: INH + rifampin (eg, Rifamate)</td>
<td>150-mg &amp; 300-mg caps (2 caps daily)</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*Check product labeling for detailed information on dose, contraindications, drug interaction, 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.
arrangements, perceptions and understanding of TB and its treatment, and readiness to learn.

Nursing Diagnoses

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

- Ineffective airway clearance related to copious tracheobronchial secretions
- Deficient knowledge about treatment regimen and preventive health measures and related ineffective individual management of the therapeutic regimen (noncompliance)
- Activity intolerance related to fatigue, altered nutritional status, and fever

Collaborative Problems/
Potential Complications

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

- Malnutrition
- Adverse side effects of medication therapy: hepatitis, neurologic changes (deafness or neuritis), skin rash, gastrointestinal upset
- Multidrug resistance
- Spread of TB infection (miliary TB)

Planning and Goals

The major goals for the patient include maintenance of a patent airway, increased knowledge about the disease and treatment regimen and adherence to the medication regimen, increased activity tolerance, and absence of complications.

Nursing Interventions

PROMOTING AIRWAY CLEARANCE

Copious secretions obstruct the airways in many patients with TB and interfere with adequate gas exchange. Increasing 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 a patient must follow can be quite complex. Understanding the medications, schedule, and side effects is important. The patient must understand 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. 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 hand hygiene.

PROMOTING ACTIVITY AND ADEQUATE NUTRITION

Patients with TB are often debilitated from a 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 person has few resources. A nutritional plan that allows for small, frequent meals may be required. Liquid nutritional supplements may assist in meeting basic caloric requirements.

MONITORING AND MANAGING
POTENTIAL COMPLICATIONS

Malnutrition

This may be a consequence of the patient’s lifestyle, lack of knowledge about adequate nutrition and its role in health maintenance, lack of resources, fatigue, or lack of appetite because of coughing and mucus production. To counter the effects of these factors, the nurse collaborates with the dietitian, physician, social worker, family, and patient to identify strategies to ensure an adequate nutritional intake and availability of nutritious food. Identifying facilities (eg, shelters, soup kitchens, Meals on Wheels, and other community resources) 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. High-calorie nutritional supplements may be suggested as a strategy for increasing dietary intake using food products normally found in the home. Purchasing food supplements may be beyond the patient’s budget, but a dietitian can help develop recipes to increase caloric intake despite minimal resources.

Side Effects of Medication Therapy

It is important to assess medication side effects because they are often a reason the patient fails to adhere to the prescribed medication regimen. Efforts are made to reduce the side effects to increase the patient’s willingness to take the medications as prescribed.

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 containing tyramine and histamine (tuna, aged cheese, red wine, soy sauce, yeast extracts). Eating these types of foods while taking INH may result in headache, flushing, hypotension, light-headedness, palpitations, and diaphoresis.

In addition, rifampin can increase the metabolism of 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. 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, so 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 enzyme, blood urea nitrogen, and serum creatinine levels are monitored to detect medication-related changes in liver and kidney function. Sputum culture results are monitored for acid-fast bacillus to evaluate the effectiveness of the treatment regimen and adherence to therapy.

Multidrug Resistance

The nurse carefully monitors vital signs and observes for spikes in temperature or changes in the clinical status. The nurse reports any change in the patient’s respiratory status to the primary
health care provider. The nurse instructs the patient about the risk of drug resistance if the medication regimen is not strictly and continuously followed.

Spread of TB Infection
Spread 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 (Ghon tubercle). 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 an indolent 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. Within a few weeks, however, the chest x-ray reveals small densities scattered diffusely throughout both lung fields; these are the miliary tubercles, which gradually grow.

The possibility of TB in nonpulmonary sites in the body requires careful monitoring for this very serious form of the infection. 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.

PROMOTING HOME AND COMMUNITY-BASED CARE
Teaching Patients Self-Care
The nurse plays a vital role in caring for the patient with TB and the family, which includes assessing the patient’s ability to continue therapy at home. The nurse instructs the patient and family about infection control procedures, such as proper disposal of tissues, covering the mouth during coughing, and hand hygiene. Assessment of the patient’s adherence to the medication regimen is imperative because of the risk of developing resistant strains of TB if the regimen is not followed faithfully. In some cases, when the patient’s ability to comply with the medication regimen is in question, referral to an outpatient clinic for daily medication administration may be required. This is referred to as directly observed therapy (DOT).

Continuing Care
The nurse evaluates the patient’s environment, including home or workplace and social setting, to identify other people who may have been in contact with the patient during the infectious stage. It is important to arrange follow-up screening for any contacts of the infected person. Nurses who have contact with the patient in home, shelter, hospital, clinic, or work settings assess the patient’s physical and psychological status and ability to adhere to the prescribed treatment. The nurse assesses the patient for adverse effects of medications and adherence to the therapeutic regimen (eg, taking medications as prescribed, practicing safe hygiene, consuming a nutritious and adequate diet, and participating in an appropriate level of activity). The nurse reinforces previous teaching and emphasizes the importance of keeping scheduled appointments with the primary health care provider. In addition, the patient is reminded of the importance of other health promotion activities and recommended health screening.

Evaluation
EXPECTED PATIENT OUTCOMES
Expected patient outcomes may include:
1. Maintains a patent airway by managing secretions with hydration, humidification, coughing, and postural drainage
2. Demonstrates an adequate level of knowledge
   a. Lists medications by name and the correct schedule for taking them
   b. Names expected side effects of medications
   c. Identifies how and when to contact health care provider
3. Adheres to treatment regimen by taking medications as prescribed and reporting for follow-up screening
4. Participates in preventive measures
   a. Disposes of used tissues properly
   b. Encourages people who are close contacts to report for testing
   c. Adheres to hand hygiene recommendations
5. Maintains activity schedule
6. Exhibits no complications
   a. Maintains adequate weight or gains weight if indicated
   b. Exhibits normal results of tests of liver and kidney function
7. Takes steps to minimize side effects of medications
   a. Takes supplemental vitamins (vitamin B), as prescribed, to minimize peripheral neuropathy
   b. Avoids use of alcohol
   c. Avoids foods containing tyramine and histamine
   d. Has regular physical examinations and blood tests to evaluate liver and kidney function, neuropathy, hearing and visual acuity

LUNG ABSCESS
A lung abscess is a localized necrotic lesion of the lung parenchyma containing purulent material that collapses and forms a cavity. It is generally caused by aspiration of anaerobic bacteria. By definition, the chest x-ray will demonstrate a cavity of at least 2 cm. Patients who have impaired cough reflexes and cannot close the glottis, or those with swallowing difficulties, are at risk for aspirating foreign material and developing a lung abscess. Other at-risk patients include those with central nervous system disorders (seizure, stroke), drug addiction, alcoholism, esophageal disease, or compromised immune function, those without teeth, as well as patients receiving nasogastric tube feedings and those with an altered state of consciousness from 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, or chest trauma.

Most 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 the patient’s 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 in which lung abscess occurs. 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. Anaerobic organisms, however, may also be present. The organism varies 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, often bloody, sputum. Leukocytosis may be present. Pleurisy or dull chest pain, dyspnea, weakness, anorexia, and weight loss are common. Fever and cough may develop insidiously and may have been present for several weeks before diagnosis.

**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 pictures of different cross-sectional areas of the lung.

**Prevention**

The following measures will 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. The patient should be assessed for an adequate cough. A few patients need a percutaneous chest catheter placed 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 when there is massive hemoptysis (coughing up of blood) or little or no response to medical management.

**PHARMACOLOGIC THERAPY**

Intravenous antimicrobial therapy depends on the results of the sputum culture and sensitivity and is administered for an extended period. Penicillin G or clindamycin (Cleocin) is the medication of choice, followed by penicillin with metronidazole. Large intravenous doses are generally required because the antibiotic must penetrate the necrotic tissue and the fluid in the abscess. The intravenous dose is continued until there is evidence of symptom improvement.

Long-term therapy with oral antibiotics replaces intravenous 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 the 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. If treatment stops too soon, a relapse may occur.

**Nursing Management**

The nurse administers antibiotics and intravenous therapies 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 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.** The patient who has had surgery may return home before the wound closes entirely or with a drain or tube in place. Thus, the patient or a caregiver needs instruction on 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 instructs the patient to perform deep-breathing and coughing exercises every 2 hours during the day and shows a caregiver how to perform chest percussion and postural drainage to facilitate expectoration of lung secretions.

**Continuing Care.** Referral for home care may be required by some patients whose condition requires therapy at home. During visits to the patient at home, the nurse assesses the patient’s physical condition, nutritional status, and home environment as well as the patient’s and family’s ability to carry out the therapeutic regimen. Patient teaching is reinforced during home visits, and nutrition counseling is provided with the goal of attaining and maintaining an optimal state of nutrition. To prevent a relapse, the nurse emphasizes the importance of completing the antibiotic regimen and of following the suggestions for rest and appropriate activity. If intravenous antibiotic therapy is to continue at home, the services of a home care nurse may be arranged to initiate intravenous therapy and to evaluate its administration by the patient or family. Although most outpatient intravenous therapy
is administered in the home setting, a patient may visit a nearby clinic or physician’s office for this treatment. In some cases the patient with lung abscess may have ignored his or her 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 pulmonary embolism; in patients with primary and metastatic cancer; and after thoracotomy. The parietal pleura has nerve endings; 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 restricted in distribution rather than diffuse; it usually occurs only on one side. The pain may become minimal or absent when the breath is held, or 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 examinations, 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 analgesics and topical applications of heat or cold provide symptomatic relief. Indomethacin (Indocin), a nonsteroidal anti-inflammatory drug (NSAID), 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 can offer 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 can teach 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 but 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. 23-5). Pleural effusion may be a complication of heart failure, TB, pneumonia, pulmonary infections (particularly viral infections), nephrotic syndrome, connective tissue disease, pulmonary embolism, and neoplastic tumors. Bronchogenic carcinoma is the most common malignancy associated with a pleural effusion.

**Pathophysiology**

In certain disorders, fluid may accumulate in the pleural space to a point where it becomes clinically evident. This almost always has pathologic significance. The effusion can be composed of a relatively clear fluid, or it can be bloody or purulent. An effusion...
Clinical Manifestations

Usually the clinical manifestations are those caused by the underlying disease. Pneumonia causes fever, chills, and pleuritic chest pain, whereas a malignant effusion may result in dyspnea and coughing. The size of the effusion and the patient’s underlying lung disease determine the severity of symptoms. A large pleural effusion causes shortness of breath. When a small to moderate pleural effusion is present, dyspnea may be absent or only minimal. The severity of the symptoms assessed depends on the time course of the development of the pleural effusion and the patient’s underlying disease.

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 when percussed. In an extremely large pleural effusion, the assessment reveals a patient in acute respiratory distress. Tracheal deviation away from the affected side may also be noted.

Physical examination, chest x-ray, chest CT scan, 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, lactic dehydrogenase, protein), cytologic analysis for malignant cells, and pH. A pleural biopsy also may be performed.

Medical Management

The objectives of treatment are to discover the underlying cause, 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, lung cancer, 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 Chap. 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.

If the underlying cause is a malignancy, however, 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 a thoroscopic approach or via a chest tube. Chemically irritating agents (eg, bleomycin or t alc) are instilled in the pleural space. With the chest tube insertion 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 malignant pleural effusions include surgical pleurectomy, insertion of a small catheter attached to a drainage bottle for outpatient management, 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 the reservoir daily to move fluid from the pleural space to the peritoneal space (Taubert & Wright, 2000).

Nursing Management

The nurse’s role in the care of the patient with a pleural effusion includes implementing the medical regimen. The nurse prepares and positions the patient for thoracentesis and offers support throughout the procedure. Pain management is a priority, and the nurse assists the patient to assume positions that are the least painful. However, frequent turning and ambulation are important to facilitate drainage. The nurse administers analgesics as prescribed and as needed.

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 the patient is to be managed as an outpatient with a pleural catheter for drainage, the nurse is responsible for educating the patient and family regarding 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. Most empyemas occur as complications of bacterial pneumonia or lung abscess. Other causes include penetrating chest trauma, hematogenous infection of the pleural space, nonbacterial infections, or iatrogenic causes (after thoracic surgery or thoracentesis).

Pathophysiology

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

With an empyema, 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 more 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 a chest x-ray or chest CT scan. 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 full 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 not too purulent or thick
- Tube thoracostomy (chest drainage using a large-diameter intercostal tube attached to water-seal drainage [see Chap. 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, or rib resection and drainage). When a patient is discharged to 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 and/or alveolar space. 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 of this is in the patient who has undergone pneumonectomy. When one lung has been removed, all the cardiac output then 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. This type of pulmonary edema is sometimes termed “flash” pulmonary edema. A second example is called re-expansion pulmonary edema. This may be due to a rapid re-inflation of the lung after removal of air from a pneumothorax or evacuation of fluid from a large pleural effusion.

Clinical Manifestations

The patient has increasing respiratory distress, characterized by dyspnea, air hunger, and central cyanosis. The patient is 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 suction out these foamy, frothy, and often blood-tinged secretions. The patient has acute respiratory distress and may become confused or stuporous.

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 due to the movement of air through the alveolar fluid. The chest x-ray reveals increased interstitial markings. The patient may be tachycardic, the pulse oximetry values begin to fall, and arterial blood gas analysis demonstrates increasing 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 given. Additional cardiac measures (eg, intra-aortic balloon pump) may be indicated if the patient does not respond. If the problem is fluid overload, diuretics are given and the patient is placed on fluid restrictions. Oxygen is administered to correct the hypoxemia; in some circumstances, intubation and mechanical ventilation are necessary. The patient is extremely anxious, and morphine is administered to reduce anxiety and control pain.
**Chapter 23  Management of Patients With Chest and Lower Respiratory Tract Disorders**

**Nursing Management**

Nursing management of the patient with pulmonary edema includes assisting with administration of oxygen and intubation and mechanical ventilation if respiratory failure occurs. The nurse also administers medications (ie, morphine, vasodilators, inotropic medications, preload and afterload agents) as prescribed and monitors the patient’s response. 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. It exists when the exchange of oxygen for carbon dioxide in the lungs cannot keep up with the rate of oxygen consumption and carbon dioxide production by the cells of the body.

Acute respiratory failure (ARF) is defined as a fall in arterial oxygen tension (PaO₂) to less than 50 mm Hg (hypoxemia) and a rise in arterial carbon dioxide tension (PaCO₂) to greater than 50 mm Hg (hypercapnia), with an arterial pH of less than 7.35. In ARF, the ventilation or perfusion mechanisms in the lung are impaired. Respiratory system mechanisms leading to ARF include:

- Alveolar hypoventilation
- Diffusion abnormalities
- Ventilation–perfusion mismatching
- Shunting

It is important to distinguish between ARF and chronic respiratory failure. Chronic respiratory failure is defined as a deterioration in the gas exchange function of the lung that has developed insidiously or has persisted for a long period after an episode of ARF. 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 Chap. 24) and neuromuscular diseases (discussed in Chap. 65). Patients with these disorders develop a tolerance to the gradually worsening hypoxemia and hypercapnia. However, a patient with chronic respiratory failure may develop ARF. This is seen in the COPD patient who develops an exacerbation or infection that causes additional deterioration of the gas exchange mechanism. The principles of management of acute versus chronic respiratory failure are different; the following discussion will be limited to ARF.

**Pathophysiology**

Common causes of ARF can be classified into four categories: decreased respiratory drive, dysfunction of the chest wall, dysfunction of the lung parenchyma, and other causes.

**DECREASED RESPIRATORY DRIVE**

Decreased respiratory drive may occur with severe brain injury, large lesions of the brain stem (multiple sclerosis), use of sedative medications, and metabolic disorders such as hypothyroidism. These disorders impair the normal response of chemoreceptors in the brain to normal respiratory stimulation.

**DYSFUNCTION OF THE CHEST WALL**

The impulses arising in the respiratory center travel through nerves that extend from the brain stem down the spinal cord to receptors in the muscles of respiration. Thus, any disease or disorder of the nerves, spinal cord, muscles, or neuromuscular junction involved in respiration seriously affects ventilation and may ultimately lead to ARF. These include musculoskeletal disorders (muscular dystrophy, polymyositis), neuromuscular junction disorders (myasthenia gravis, poliomyelitis), some peripheral nerve disorders, and spinal cord disorders (amyotrophic lateral sclerosis, Guillain-Barré syndrome, and cervical spinal cord injuries).

**DISFUNCTION OF LUNG PARENCHYMA**

Pleural effusion, hemothorax, pneumothorax, and upper airway obstruction are conditions that interfere with ventilation by preventing expansion of the lung. These conditions, which may cause respiratory failure, usually are produced by an underlying lung disease, pleural disease, or trauma and injury. Other diseases and conditions of the lung that lead to ARF include pneumonia, status asthmaticus, lobar atelectasis, pulmonary embolism, and pulmonary edema.

**OTHER CAUSES**

In the postoperative period, especially after major thoracic or abdominal surgery, inadequate ventilation and respiratory failure may occur because of several factors. During this period, for example, ARF may be caused by the effects of anesthetic agents, analgesics, and sedatives, 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 mismatch of ventilation to perfusion 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 ARF.

**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 the patient with ARF includes assisting with intubation and maintaining mechanical ventilation (described in Chap. 25). The nurse assesses the patient’s respiratory status by monitoring the patient’s level of response, arterial blood gases, pulse oximetry, and vital signs and assessing the respiratory system. The nurse 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 his or her needs to the health care team. Nursing care also addresses the problems...
Acute Respiratory Distress Syndrome

Acute respiratory distress syndrome (ARDS; previously called adult respiratory distress syndrome) is a clinical syndrome characterized by a sudden and progressive pulmonary edema, increasing bilateral infiltrates on chest x-ray, hypoxemia refractory to oxygen supplementation, and reduced lung compliance. These signs occur in the absence of left-sided heart failure. Patients with ARDS usually require mechanical ventilation with a higher-than-normal airway pressure. 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 as high as 50% to 60%. The major cause of death in ARDS is nonpulmonary multiple-system organ failure, often with sepsis.

Pathophysiology

ARDS occurs as a result of an inflammatory trigger that initiates the release of cellular and chemical mediators, causing injury to the alveolar capillary membrane. This results in leakage of fluid into the alveolar interstitial spaces and alterations in the capillary bed. Severe ventilation–perfusion mismatching occurs in ARDS. Alveoli collapse because of the inflammatory infiltrate, blood, fluid, and surfactant dysfunction. Small airways are narrowed because of interstitial fluid and bronchial obstruction. The lung compliance becomes markedly decreased (stiff lungs), and the result is a characteristic decrease in 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 a shunt to develop. This means that blood is interfacing with nonfunctioning alveoli and gas exchange is markedly impaired, resulting in severe, refractory hypoxemia. Figure 23-6 shows the sequence of pathophysiologic events leading to ARDS.

Clinical Manifestations

Clinically, 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. A characteristic feature is arterial hypoxemia that does not respond to supplemental oxygen. On chest x-ray, the findings are similar to those seen with cardiogenic pulmonary edema and present 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). Clinically, a 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 (Ware & Matthay, 2000).

Assessment and Diagnostic Findings

Intercostal retractions and crackles, as the fluid begins to leak into the alveolar interstitial space, are evident on physical examination. A diagnosis of ARDS may be made based on the following crite-
ria: a history of systemic or pulmonary risk factors, acute onset of respiratory distress, bilateral pulmonary infiltrates, clinical absence of left-sided heart failure, and a ratio of partial pressure of oxygen of arterial blood to fraction of inspired oxygen (PaO₂/FiO₂) less than 200 mm Hg (severe refractory hypoxemia).

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.

Positive end-expiratory pressure (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 to 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 FiO₂ may be required. The goal is a PaO₂ greater than 60 mm Hg or an oxygen saturation level of greater than 90% at the lowest possible FiO₂. 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. Intravenous crystalloid solutions are administered, with careful monitoring of pulmonary status. 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

Numerous pharmacologic treatments are under investigation to stop the cascade of events leading to ARDS. These include human recombinant interleukin-1 receptor antagonist, neutrophil inhibitors, pulmonary-specific vasodilators, surfactant replacement therapy, antisepsis agents, antioxidant therapy, and corticosteroids late in the course of ARDS (Ware & Matthay, 2000).

Nutritional Therapy

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

Nursing Management

General Measures

The patient with ARDS is critically ill and requires close monitoring because the condition could quickly change to a life-threatening situation. 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 should turn 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 the ARDS patient is sometimes improved in the prone position and may be used in special circumstances; studies to assess the benefits and problems of such positioning are ongoing (Curley, 2000; Marion, 2001).

The patient is extremely anxious and agitated because of the increasing hypoxemia and dyspnea. The nurse should explain all procedures and provide care in a calm, reassuring manner. It is important to reduce the patient’s anxiety because anxiety prevents rest and increases oxygen expenditure. Rest is essential to reduce oxygen consumption, thereby reducing 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 the patient. The patient may be anxious and “fight” the ventilator. Nursing assessment is important to assess for 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 drop in the oxygen level; the patient’s 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. Possible sedatives are lorazepam (Ativan), midazolam (Versed), haloperidol (Haldol), propofol (Diprivan), and short-acting barbiturates.

If the PEEP level cannot be maintained despite the use of sedatives, neuromuscular blocking agents, such as pancuronium (Pavulon), vecuronium (Norcuron), atracurium (Tracrium), and rocuronium (Zemuron), may be given to paralyze the patient. This allows the patient to be ventilated more easily. With paralysis, the patient appears 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.

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 at all times. All ventilator and patient alarms should 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 patients to the development of 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.
Analgiesia 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 family. This experience can be very frightening to family members if they are unaware that these agents have been administered.

**Pulmonary Hypertension**

Pulmonary hypertension is a condition that is not clinically evident until late in its progression. Pulmonary hypertension exists when the systolic pulmonary artery pressure exceeds 30 mm Hg or the mean pulmonary artery pressure exceeds 25 mm Hg. These pressures cannot be measured indirectly as can systemic blood pressure; instead, they must be measured during right-sided heart catheterization. In the absence of these measurements, clinical recognition becomes the only indicator for the presence of pulmonary hypertension.

There are two forms of pulmonary hypertension: primary (or idiopathic) and secondary. Primary pulmonary hypertension is an uncommon disease in which the diagnosis is made by excluding all other possible causes. The exact cause is unknown, but there are several possible causes (Chart 23-7). The clinical presentation of primary pulmonary hypertension exists with no evidence of pulmonary and cardiac disease or pulmonary embolism. It occurs most often in women 20 to 40 years of age and is usually fatal within 5 years of diagnosis.

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

**Primary or Idiopathic**
- Altered immune mechanisms
- Silent pulmonary emboli
- Raynaud’s phenomenon
- Oral contraceptive use
- Sickle cell disease
- Collagen diseases

**Secondary**
- Pulmonary vasoconstriction due to hypoxemia
  - Chronic obstructive pulmonary disease
  - Kyphoscoliosis
  - Obesity
  - Smoke inhalation
  - High altitude
  - Neuromuscular disorders
  - Diffuse interstitial pneumonia
- Reduction of the pulmonary vascular bed (must impair 50% to 75% of the vascular bed)
  - Pulmonary emboli
  - Vasculitis
  - Widespread interstitial lung disease (sarcoidosis, systemic sclerosis)
  - Tumor emboli
- Primary cardiac disease
  - Congenital (patent ductus arteriosus, atrial septal defect, ventricular septal defect)
  - Acquired (rheumatic valvular disease, mitral stenosis, myxoma, left ventricular failure)

Secondary pulmonary hypertension 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 secondary pulmonary hypertension is pulmonary artery constriction due to hypoxemia from COPD.

**Pathophysiology**

The underlying process of pulmonary hypertension varies, and multiple factors are often responsible. 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 an increase in 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.

**Clinical Manifestations**

Dyspnea is the main symptom of pulmonary hypertension, occurring at first with exertion and eventually at rest. Substernal chest pain also is common, affecting 25% to 50% of patients. 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).

**Assessment and Diagnostic Findings**

A complete diagnostic evaluation includes a history, physical examination, chest x-ray, pulmonary function studies, electrocardiogram (ECG), echocardiogram, ventilation–perfusion scan, and cardiac catheterization. In some cases, a lung biopsy, performed by thoracotomy or thoracoscopy, may be needed to make a definite diagnosis. Cardiac catheterization of the right side of the heart reveals elevated pulmonary arterial pressure. An echocardiogram can assess the progression of the disease and rule out other conditions with similar signs and symptoms. 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 and/or T-wave inversion anteriorly. The PaO2 also is decreased (hypoxemia). A ventilation–perfusion scan or pulmonary angiography detects defects in pulmonary vasculature, such as pulmonary emboli. Pulmonary function studies may be normal or show a slight decrease in vital capacity (VC) and lung compliance, with a mild decrease in the diffusing capacity.

**Medical Management**

The goal of treatment is to manage the underlying cardiac or pulmonary condition. Most patients with primary pulmonary hypertension do not have hypoxemia at rest but require supplemental
oxygen with exercise. However, patients with severe right ventricular failure, decreased cardiac output, and progressive disease may have resting hypoxemia and require continuous oxygen supplementation. Appropriate oxygen therapy (see Chap. 25) reverses the vasoconstriction and reduces the pulmonary hypertension in a relatively short time.

In the presence of cor pulmonale, which is discussed in the section that follows, treatment should include fluid restriction, diuretics to decrease fluid accumulation, cardiac glycosides (eg, digitalis) in an attempt to improve cardiac function, calcium channel blockers for vasodilation, and rest. In primary pulmonary hypertension, vasodilators have been administered with variable success (eg, calcium channel blockers, intravenous prostacyclin). Prostacyclin (PGX [Flolan]) is one of the prostaglandins produced by the pulmonary endothelium. Intravenous prostacyclin (epoprostenol) helps to decrease pulmonary hypertension by reducing pulmonary vascular resistance and pressures and increasing cardiac output. Anticoagulants such as warfarin (Coumadin) have been given to patients because of chronic pulmonary emboli. Heart–lungen transplantation has been successful in select patients with primary hypertension who have not been responsive to other therapies.

**Nursing Management**

The major nursing goal is to identify patients at high risk for pulmonary hypertension, such as those with COPD, pulmonary emboli, congenital heart disease, and mitral valve disease. The nurse also must be alert for signs and symptoms, administer oxygen therapy appropriately, and instruct patients and their families about the use of home oxygen supplementation.

**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. Any disease affecting the lungs and accompanied by hypoxemia may result in cor pulmonale. The most frequent cause is severe COPD (see Chap. 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 (deformities of the thoracic cage, massive obesity), or conditions that reduce the pulmonary vascular bed (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 a heart murmur. 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 the patient’s 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.

Better survival rates and greater reduction in pulmonary vascular resistance have been reported with continuous, 24-hour oxygen therapy for patients with severe hypoxemia. 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.

Ventilation is further improved with chest physical therapy and bronchial hygiene maneuvers as indicated to remove accumulated secretions, and the administration of bronchodilators. Further 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 (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 pulmonary 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. (Management of acute respiratory failure was presented earlier in this chapter.)

**Nursing Management**

Nursing care of the patient with cor pulmonale addresses the underlying disorder leading to cor pulmonale as well as the problems related to pulmonary hypertventilation and right-sided cardiac failure. If intubation and mechanical ventilation are required to manage ARF, 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 monitoring (fluid retention, weight gain, edema) and adherence to the therapeutic regimen, especially the 24-hour use of oxygen. Factors that affect the patient’s adherence to the treatment regimen are explored and addressed.

PROMOTING HOME AND COMMUNITY-BASED CARE

Teaching Patients Self-Care. Most of the care and monitoring of the patient with cor pulmonale is performed by the patient and family in the home because it is a chronic disorder. If supplemental oxygen is administered, the nurse instructs the patient and family in its 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 urges the patient to stop smoking.

Continuing Care. A referral for home care may be warranted for the patient who cannot manage self-care or for the patient whose physical condition warrants close assessment. During the home visit, 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 if 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 patient is reminded 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. 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). It is estimated that more than half a million people develop PE yearly, resulting in more than 50,000 deaths. 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 an apparently healthy person. Risk factors for developing PE are identified in Chart 23-8.

Although most thrombi originate in the deep veins of the legs, other sites include the pelvic veins and the right atrium of the heart. A venous thrombosis can result from slowing of blood flow (stasis), secondary to damage to the blood vessel wall (particularly the endothelial lining) or changes in the blood coagulation mechanism. Atrial fibrillation is also a cause of pulmonary embolism. 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.

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, GI, GU, 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 |
| Other Predisposing Conditions |
| Advanced age |
| Obesity |
| Pregnancy |
| Oral contraceptive use |
| History of previous thrombophlebitis, pulmonary embolism |
| Constrictive clothing |

Pathophysiology

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. Thus, gas exchange is impaired or absent in this area. In addition, various substances are released from the clot and surrounding area, causing regional blood vessels and bronchioles to constrict. This causes an increase in pulmonary vascular resistance. This reaction compounds the ventilation–perfusion imbalance.

The hemodynamic consequences are increased pulmonary vascular resistance from 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.

Clinical Manifestations

The 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; tachypnea (very rapid respiratory rate) is the most frequent sign (Goldhaber, 1998). The duration and intensity of the dyspnea depend on the extent of embolization. Chest pain is common and is usually sud-
den and pleuritic. It may be substernal and mimic angina pectoris or a myocardial infarction. Other symptoms include anxiety, fever, tachycardia, apprehension, cough, diaphoresis, hemoptyasis, and syncope.

A massive embolism 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 the bifurcation of the pulmonary arteries that produces pronounced dyspnea, sudden substernal pain, rapid and weak pulse, shock, syncope, and sudden death. Multiple small emboli can lodge in the terminal pulmonary arterioles, producing multiple small infarctions of the lungs. A pulmonary infarction causes ischemic necrosis of an area of the lung and occurs in less than 10% of cases of PE (Arroliga, Matthy & Matrhy, 2000). The clinical picture may mimic that of bronchopneumonia or heart failure. In atypical instances, the disease causes few signs and symptoms, whereas in other instances it mimics various other cardiopulmonary disorders.

Assessment and Diagnostic Findings

Death from PE commonly occurs within 1 hour of symptoms; thus, 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. Deep venous thrombosis is closely associated with the development of PE. Typically, patients report sudden onset of pain and/or swelling and warmth of the proximal or distal extremity, skin discoloration, and superficial vein distention. The pain is usually relieved with elevation. The diagnostic workup includes a ventilation–perfusion scan, pulmonary angiography, chest x-ray, ECG, peripheral vascular studies, impedance plethysmography, and arterial blood gas analysis.

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. Peripheral vascular studies may include impedance plethysmography, Doppler ultrasonography, or venography (see Chap. 31). Test results confirm or exclude the diagnosis of PE. Arterial blood gas analysis may show hypoxemia and hypocapnia (from tachypnea); however, arterial blood gas measurements are normal in up to 20% of patients with PE.

A ventilation–perfusion scan is the test of choice in patients with suspected PE. The perfusion portion of the scan may indicate areas of diminished or absent blood flow and is the most useful test to rule out clinically important PE. A ventilation scan may show whether there is also a ventilation abnormality present. A normal perfusion scan rules out the diagnosis of PE. If there is a ventilation–perfusion mismatch, the probability of PE is high. Spiral CT of the chest may also assist in the diagnosis.

If lung scan results are not definitive, pulmonary angiography, considered the gold standard for the diagnosis of PE, can be used. This test is invasive and is performed in the interventional radiology department. A contrast agent is injected into the pulmonary arterial system, allowing visualization of obstructions to blood flow and abnormalities.

Prevention

For those at risk, the most effective approach to preventing PE is to prevent deep venous thrombosis. Active leg exercises to avoid venous stasis, early ambulation, and use of elastic compression stockings are general preventive measures. Additional strategies for prevention are listed in the checklist in Chart 23-9.
Patients who are older than 40, whose hemostasis is adequate, and who are undergoing major elective abdominal or thoracic surgery may receive anticoagulant therapy. Low doses of heparin may be given before surgery to reduce the risk of postoperative deep venous thrombus and PE. Heparin should be administered subcutaneously 2 hours before surgery and continued every 8 to 12 hours until the patient is discharged. Low-dose heparin is thought to enhance the activity of antithrombin III, a major plasma inhibitor of clotting factor X. This regimen is not recommended for patients with an active thrombotic process or for those undergoing major orthopedic surgery, open prostatectomy, or surgery on the eye or brain. Low-molecular-weight heparin (eg, enoxaparin [Lovenox]) is an alternative therapy. It has a longer half-life, enhanced subcutaneous absorption, a reduced incidence of thrombocytopenia, and reduced interaction with platelets as compared to unfractionated heparin (Ansell, Hickey, Kleinschmidt et al., 2000).

The intermittent pneumatic leg compression device is useful in preventing thromboembolism. The device inflates a bag that intermittently compresses the leg from the calf to the thigh, thereby improving venous return. It may be applied before surgery and continued until the patient is ambulatory. The device is particularly useful for patients who are not candidates for anticoagulant therapy (Clagett, Anderson, Geerts et al., 1998).

Medical Management

Because PE is often a medical emergency, emergency management is of primary concern. After emergency measures have been taken and the patient’s condition stabilizes, the treatment goal is to dissolve (lyse) the existing emboli and prevent new ones from forming. The treatment of PE may include a variety of modalities:

- General measures to improve respiratory and vascular status
- Anticoagulation therapy
- Thrombolytic therapy
- Surgical intervention

EMERGENCY MANAGEMENT

Massive PE is a life-threatening emergency. The immediate objective is to stabilize the cardiopulmonary system. A sudden rise in pulmonary resistance increases the work of the right ventricle, which can cause acute right-sided heart failure with cardiogenic shock. Most patients who die of massive PE do so in the first 1 to 2 hours after the embolic event. Emergency management consists of the following:

- Nasal oxygen is administered immediately to relieve hypoxemia, respiratory distress, and central cyanosis.
- Intravenous infusion lines are started 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. Spiral CT is more advanced and quicker than routine tomography. With spiral CT, the patient continuously moves as the x-ray tube rotates. With this type of CT, images can be reconstructed at select levels and locations for diagnostic purposes.
- 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 dysrythmias and right ventricular failure, which may occur suddenly.
- Digitalis glycosides, intravenous 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 intravenous morphine or sedatives are administered to relieve the patient’s 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 the patient’s respiratory and vascular status. Oxygen therapy is administered to correct the hypoxemia, relieve the pulmonary vascular vasoconstriction, and reduce the pulmonary hypertension. Using elastic compression 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) has traditionally been the primary method for managing acute deep vein thrombosis and PE (Goldhaber, 1998). Heparin is used to prevent recurrence of emboli but has no effect on emboli that are already present. It is administered as an intravenous bolus of 5,000 to 10,000 units, followed by a continuous infusion initiated at a dose of 18 U/kg per hour, not to exceed 1,600 U/hour in otherwise healthy patients (Goldhaber, 1998). The rate is reduced in patients with a high risk of bleeding. The goal is to keep the partial thromboplastin time 1.5 to 2.5 times normal (or an INR [international normalized ratio] of 2.0 to 3.0). Heparin is usually administered for 5 to 7 days. Low-molecular-weight heparin (eg, enoxaparin [Lovenox]) may also be used.

Warfarin sodium (Coumadin) administration is begun within 24 hours after the start of heparin therapy because its onset of action is 4 to 5 days. Warfarin is usually continued for 3 to 6 months. The prothrombin time is maintained at 1.5 to 2.5 times normal (or an INR [international normalized ratio] of 2.0 to 3.0). Anticoagulation therapy is contraindicated in patients who are at risk for bleeding (eg, those with gastrointestinal conditions or with postoperative or postpartum bleeding).

Thrombolytic Therapy. Thrombolytic therapy (urokinase, streptokinase, alteplase, anistreplase, reteplase) also may be used in treating PE, particularly in patients who are severely compromised (eg, those who are hypotensive and have significant hypoxemia despite oxygen supplementation). Thrombolytic therapy resolves the thrombi or emboli more quickly and restores more normal hemodynamic functioning of the pulmonary circulation, thereby reducing pulmonary hypertension.
and improving perfusion, oxygenation, and cardiac output. Bleeding, however, is a significant side effect. Contraindications to thrombolytic therapy include a cerebrovascular accident within the past 2 months, other active intracranial processes, active bleeding, surgery within the past 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, prothrombin time, partial thromboplastin time, hematocrit values, and platelet counts are obtained. Heparin 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 and the condition being treated), the patient is given anticoagulants.

**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. Pulmonary embolectomy requires a thoracotomy with cardiopulmonary bypass technique. 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 (Goldhaber, 1998). An inferior caval 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 is intolerant of 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 inserting a filter (eg, Greenfield filter) through the internal jugular vein or common femoral vein (Fig. 23-7). This filter is advanced into the inferior vena 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 caval 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 patients 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 any patient, but particularly in those with conditions predisposing to a slowing of venous return (see Chart 23-8).
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 10 times more common in African Americans (40 cases per 100,000) than in Caucasians (5 cases per 100,000), and the disease usually begins in the third or fourth decade of life (American Thoracic Society, 1999).

Pathophysiology
Sarcoidosis is thought to be a hypersensitivity response to one or more agents (bacteria, fungi, virus, chemicals) in people with an inherited or acquired predisposition to the disorder. The hypersensitivity response results in 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 (American Thoracic Society, 1999).

Clinical Manifestations
A hallmark of this disease is its insidious onset and lack of prominent clinical signs or symptoms. The clinical picture depends on the systems involved. With pulmonary involvement, 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, the patient has fatigue, fever, anorexia, weight loss, and joint pain.

Assessment and Diagnostic Findings
Chest x-rays and CT scans are used to assess pulmonary adenopathy. The chest x-ray 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. Corticosteroid therapy may benefit some patients because of its anti-inflammatory effect, which relieves symptoms and improves organ function. It is useful for patients with ocular and myocardial involvement, skin involvement, extensive pulmonary disease that compromises pulmonary function, hepatic involvement, and hypercalcemia. 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 the involved systems.

Occupational Lung Diseases: Pneumoconioses
Diseases of the lungs occur in numerous occupations as a result of exposure to organic and inorganic (mineral) dusts and noxious gases (fumes and aerosols). The effects of inhaling these materi-
Silicosis is a chronic fibrotic pulmonary disease caused by inhalation of silica dust (crystalline silicon dioxide particles). Exposure to silica and silicates occurs in almost all mining, quarrying, and tunneling operations. Glass manufacturing, stone-cutting, the manufacture of abrasives and pottery, and foundry work are other occupations with exposure hazards. Finely ground silica, such as that found in soaps, polishes and filters, is extremely dangerous.

Pathophysiology
When the silica particles, which have fibrogenic properties, are inhaled, nodular lesions are produced throughout the lungs. With the passage of time and further exposure, the nodules enlarge and coalesce. Dense masses form in the upper portion of the lungs, resulting in the loss of pulmonary volume. Restrictive lung disease (inability of the lungs to expand fully) and obstructive lung disease from secondary emphysema result. Cavities can form as a result of superimposed TB. Exposure of 15 to 20 years is usually required before the onset of the disease and shortness of breath are manifested. Fibrotic destruction of pulmonary tissue can lead to emphysema, pulmonary hypertension, and cor pulmonale.

Clinical Manifestations
Patients with acute silicosis present with dyspnea, fever, cough, and weight loss and have a rapid progression of the disease. Symptoms are more severe in patients whose disease is complicated by progressive massive fibrosis. More commonly, this disease is a chronic problem with a long latency period. The patient may have slowly progressive symptoms indicative of hypoxemia, severe air-flow obstruction, and right-sided heart failure. Edema may occur because of the cardiac failure.

Medical Management
There is no specific treatment for silicosis, because the fibrotic process in the lung is irreversible. Supportive therapy is directed at managing complications and preventing infection. Testing is performed to rule out other lung diseases, such as TB, lung cancer, and sarcoidosis. If TB is present, it is aggressively treated. Additional therapy might include oxygen, diuretics, inhaled beta-adrenergic agonists, anticholinergics, and bronchodilator therapy.

ASBESTOSIS
Asbestosis is a disease characterized by diffuse pulmonary fibrosis from the inhalation of asbestos dust. Current laws restrict the use of asbestos, but many industries used it in the past. Therefore, exposure occurred, and may still occur, in numerous occupations, including asbestos mining and manufacturing, shipbuilding, demolition of structures containing asbestos, and roofing. Materials such as shingles, cement, vinyl asbestos tile, fireproof paint and clothing, brake linings, and filters all contained asbestos at one time, and many of these materials are still in existence. Additional diseases related to asbestos exposure include lung cancer, mesothelioma, and asbestos pleural effusion.

Pathophysiology
Inhaled asbestos fibers enter the alveoli, where they are surrounded by fibrous tissue. The fibrous tissue eventually obliterates the alveoli. Fibrous changes also affect the pleura, which thickens and develops plaque. The result of these physiologic changes is a restrictive lung disease, with a decrease in lung volume, diminished exchange of oxygen and carbon dioxide, and hypoxemia.

Clinical Manifestations
The onset of the disease is insidious, and the patient has progressive dyspnea, persistent, dry cough, mild to moderate chest pain, anorexia, weight loss, and malaise. Early physical findings include bibasilar fine, end-inspiratory crackles and in more advanced cases clubbing of the fingers. Cor pulmonale and respiratory failure occur as the disease progresses. A high proportion of workers who have been exposed to asbestos dust die of lung cancer, especially those who smoke or have a history of smoking. Malignant mesotheliomas may also occur. These are rare cancers of the pleura or peritoneum that are strongly associated with asbestos exposure.

Medical Management
There is no effective treatment for asbestosis as the lung damage is permanent and often progressive. Management is directed at controlling infection and treating the lung disease. When oxygen—carbon dioxide exchange becomes severely impaired, continuous oxygen therapy may help improve activity tolerance. The patient must be instructed to avoid additional exposure to asbestos and to stop smoking. A significant contributing cause to mortality in this population is the high incidence of lung carcinoma.

COAL WORKERS’ PNEUMOCONIOSIS
Coal workers’ pneumoconiosis (‘black lung disease”) includes a variety of respiratory diseases found in coal workers who have inhaled coal dust over the years. Coal miners are exposed to dusts that are mixtures of coal, kaolin, mica, and silica.

Pathophysiology
When coal dust is deposited in the alveoli and respiratory bronchioles, macrophages engulf the particles (by phagocytosis) and transport them to the terminal bronchioles, where they are removed by mucociliary action. In time, the clearance mechanisms cannot handle the excessive dust load, and the macrophages aggregate in the respiratory bronchioles and alveoli. Fibroblasts appear and a network of reticulin is laid down surrounding the
dust-laden macrophages. The bronchioles and the alveoli become clogged with coal dust, dying macrophages, and fibroblasts. This leads to the formation of the coal macule, the primary lesion of the disorder. Macules appear as blackish dots on the lungs. Fibrotic lesions develop and, as the macules enlarge, the weakening bronchioles dilate, with subsequent development of a localized emphysema. The disease begins in the upper lobes of the lungs but may progress to the lower lobes.

Clinical Manifestations

The first signs are a chronic cough and sputum production, similar to the signs encountered in chronic bronchitis. As the disease progresses, the patient develops dyspnea and coughs up large amounts of sputum with varying amounts of black fluid (melanoptysis), particularly if the individual is a smoker. Eventually, cor pulmonale and respiratory failure result. The diagnosis may first be made based on chest x-ray findings and a history of exposure.

Medica l Management

Preventing this disease is key because there is no effective treatment. Instead, treatment focuses on early diagnosis and management of complications. (See Chap. 24 for discussion of emphysema.)

Nursing Management

TEACHING ABOUT PREVENTION

The occupational health nurse serves as an employee advocate, making every effort to promote measures to reduce the exposure of workers to industrial products. Laws require that the work environment be ventilated properly to remove any noxious agent. Dust control can prevent many of the pneumoconioses. Dust control includes ventilation, spraying an area with water to control dust, and effective and frequent floor cleaning. Air samples need to be monitored. Toxic substances should be enclosed and placed in restricted areas. Workers must wear or use protective devices (facemasks, hoods, industrial respirators) to provide a safe air supply when a toxic element is present. Employees who are at risk should be carefully screened and followed. There is a risk of developing serious smoking-related illness (cancer) in industries in which there are unsafe levels of certain gases, dusts, fumes, fluids, and other toxic substances. Additionally, there is the potential for second-hand exposure. Asbestos and toxic dusts and substances may be transferred to others through the handling of clothing or shoes that have been exposed. Ongoing educational programs should be designed to teach workers to take responsibility for their own health and to stop smoking and receive an influenza vaccination.

The Right to Know law stipulates that employees must be informed about all hazardous and toxic substances in the workplace. Specifically, they must be educated about any hazardous or toxic substances they work with, what effects these substances can have on their health, and the measures they can take to protect themselves. The responsibility for implementing these controls inevitably falls on the federal or state government.

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. Metastatic lung tumors occur frequently because the bloodstream transports cancer cells from primary cancers elsewhere in the body to the lungs.

LUNG CANCER
(BRONCHOGENIC CARCINOMA)

Lung cancer is the number-one cancer killer among men and women in the United States, accounting for 31% of cancer deaths in men and 25% in women (American Cancer Society, 2002; Greenlee et al., 2001). For men, the incidence of lung cancer has remained relatively constant, but in women it continues to rise. Lung cancer affects primarily those in the sixth or seventh decade of life; less than 5% of patients are under the age of 40. In approximately 70% of lung cancer patients, the disease has spread to regional lymphatics and other sites by the time of diagnosis. As a result, the long-term survival rate for lung cancer patients is low. Evidence indicates that carcinoma tends to arise at sites of previous scarring (TB, fibrosis) in the lung. More than 85% of lung cancers are caused by the inhalation of carcinogenic chemicals, most commonly cigarette smoke (Schottenfeld, 2000).

Pathophysiology

Lung cancers arise from a single transformed epithelial cell in the tracheobronchial airways. A carcinogen (cigarette smoke, radon gas, other occupational and environmental agents) binds to a cell’s DNA and damages it. 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 to eventual invasive carcinoma.

Squamous cell carcinoma is more centrally located and arises more commonly in the segmental and subsegmental bronchi in response to repetitive carcinogenic exposures. Adenocarcinoma is the most prevalent carcinoma of the lung for both men and women; it presents more 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. Bronchioalveolar cell cancer arises from the terminal bronchus and alveoli and is usually slower growing as compared to other bronchogenic carcinomas. Lastly, small cell carcinomas arise primarily as a proximal lesion or lesions but may arise in any part of the tracheobronchial tree.

Classification and Staging

Non-small cell carcinoma represents 70% to 75% of tumors; small cell carcinoma represents 15% to 20% of tumors. For non-small cell carcinoma, the cell types include squamous cell carcinoma (30%), large cell carcinoma (10% to 16%), and adenocarcinoma (31% to 34%), including bronchioalveolar carcinoma (3% to 4%). Most small cell carcinomas arise in the major bronchi and spread by infiltration along the bronchial wall. Small cell cancers account for 20% to 25% of all bronchogenic cancers (Matthy, Tanoue & Carter, 2000).

In addition to cell type, lung cancers also 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 (American Joint Committee on Cancer, 2002). Non-small cell lung cancer is staged as I to IV. Stage I is the earliest stage with the highest cure rates, while stage IV designates metastatic spread.
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, second-hand (passive) smoke, environmental and occupational exposures, gender, genetics, and dietary deficits. Other factors that have been associated with lung cancer include genetic predisposition and other 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. More than 85% of lung cancers are attributable to inhalation of carcinogenic chemicals, such as cigarette smoke (American Cancer Society, 2002). 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. The risk of lung cancer decreases as the duration of smoking cessation increases.

**SECOND-HAND SMOKE**

Passive smoking has been identified as a possible cause of lung cancer in nonsmokers. In other words, people who are involuntarily exposed to tobacco smoke in a closed environment (home, car, building) are at increased risk for developing lung cancer as compared to unexposed nonsmokers. An average lifetime passive smoke exposure to a smoking spouse or partner increases a nonsmoker’s risk of lung cancer by about 35% compared to the risk of 100% for a lifetime of active smoking (Matthay, Tanoue & Carter, 2000).

**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.

**DIETARY FACTORS**

Prior research has demonstrated that smokers who eat a diet low in fruits and vegetables have an increased risk of developing lung cancer (Bast, Kufe, Pollock et al., 2000). The actual active agents in a diet rich in fruits and vegetables have yet to be determined. It has been hypothesized that carotenoids, particularly carotene or vitamin A, may be important. Several ongoing trials may help to determine if carotene supplementation has anticancer properties. Other nutrients, including vitamin E, selenium, vitamin C, fat, and retinoids, are also being evaluated regarding their protective role against lung cancer (Bast, Kufe, Pollock et al., 2000).

**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 starts as a dry, persistent cough, without sputum production. When obstruction of airways occurs, the cough may become productive due to infection.

**WHEEZING**

Wheezing is noted (occurs when a bronchus becomes partially obstructed by the tumor) in about 20% of patients with lung cancer. Patients also may report dyspnea. Hemoptyis or blood-tinted sputum may be expectorated. In some patients, a recurring fever occurs as 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. 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.

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. Non-specific symptoms of weakness, anorexia, and weight loss also may be diagnostic.

**Assessment and Diagnostic Findings**

If pulmonary symptoms occur in a heavy smoker, cancer of the lung is suspected. A chest x-ray is performed to search for pulmonary density, a solitary peripheral nodule (coin lesion), atelectasis, and infection. CT scans of the chest are used to identify small nodules not visualized on the chest x-ray and also to examine serially areas of the thoracic cage not clearly visible on the chest x-ray.

Sputum cytology is rarely used to make a diagnosis of lung cancer; however, fiberoptic bronchoscopy is more commonly
used and 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 or fluoroscopic guidance to aspirate cells from a suspicious area. In some circumstances, an endoscopy with esophageal ultrasound (EUS) may be used to obtain a transesophageal biopsy of enlarged subcarinal lymph nodes that are not easily accessible by other means.

A variety of scans may be used to assess for metastasis of the cancer. These include bone scans, abdominal scans, positron emission tomography (PET) scans, or liver ultrasound or scans. 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 physiologic impairment resulting from such surgery can be tolerated. Pulmonary function tests, arterial blood gas analysis, ventilation–perfusion scans, and exercise testing may all be used as part of the preoperative assessment (Knippel, 2001).

**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 physiologic status (particularly cardiac and pulmonary status) of the patient. 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 in treating lung cancer.

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

Several different types of lung resections 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 Chap. 25 for further details).

**RADIATION THERAPY**

Radiation therapy may cure a small percentage of patients. It is useful in controlling neoplasms that cannot be surgically resected but are responsive to radiation. Radiation 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 control 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 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 patients with distant metastases or small cell cancer of the lung, and as an adjunct to surgery or radiation therapy. Combinations of two or more medications may be more beneficial than single-dose regimens. A large number of medications are active against lung cancer. A variety of chemotherapeutic agents are used, including alkylating agents (ifosfamide), platinum analogues (cisplatin and carboplatin), taxanes (paclitaxel, docetaxel), vinca alkaloids (vinblastine and vindesine), doxorubicin, gemcitabine, vinorelbine, irinotecan (CPT-11), and etoposide (VP-16). The choice of agent depends on the growth of the tumor cell and the specific phase of the cell cycle that the medication affects. Numerous combinations of chemotherapy are undergoing investigation to identify the optimal regimen to treat differing types of lung cancer.

Chemotherapy may provide relief, especially of pain, but it does not usually cure the disease, nor does it often 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.

**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 lung cancer treatments. 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. Surgical resection may result in respiratory failure, particularly when the cardiopulmonary system is compromised before surgery. Surgical complications and prolonged mechanical ventilation are potential outcomes.

**Nursing Management**

Nursing care of the patient with lung cancer is similar to that of other patients with cancer (see Chap. 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 dyspnea, fatigue, nausea and vomiting, and anorexia will assist the patient and family to cope with the therapeutic measures.

**RELEIVING 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, breathing exercises for lung expansion and relaxation, and educating the patient on energy conservation and airway clearance techniques (Connolly & O’Neill, 1999). Many of the techniques used in pulmonary rehabilitation can be applied to the lung cancer patient. 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 the cancer patient. It is commonly experienced by the lung cancer patient 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). The nurse is pivotal in thoroughly assessing the patient’s level of fatigue, identifying potentially treatable causes, and validating with the patient that fatigue is indeed an important symptom. Educating the patient in energy conservation techniques or referring the patient to a physical therapy, occupational therapy, or pulmonary rehabilitation program may be helpful. In addition, guided exercise has been recently identified as a potential intervention for treating fatigue in cancer patients. This is an important area for research because few studies have been conducted, and only in select populations of cancer patients.

**PROVIDING PSYCHOLOGICAL SUPPORT**

Another important part of the nursing care of the patient with lung cancer is psychological support and identification of potential resources for the patient and family. Often, the nurse must help the patient and family deal with the poor prognosis and relatively rapid progression of this disease. The nurse must help the patient and family with informed decision making regarding the possible treatment options, methods to maintain the patient’s quality of life during the course of this disease, and end-of-life treatment options.

**TUMORS OF THE MEDIASTINUM**

Tumors of the mediastinum include neurogenic tumors, tumors of the thymus, lymphomas, germ cell, cysts, and mesenchymal tumors. These tumors may be malignant or benign. These tumors are usually described in relation to location: anterior, middle, or posterior masses or tumors.

**Clinical Manifestations**

Nearly all the 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 caval syndrome (ie, swelling of the face, neck, and upper extremities), marked dissection 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 scans are the gold standard for assessment of the mediastinum and surrounding structures. MRI may be used in some circumstances, as well as PET scans.

**Medical Management**

If the tumor is malignant and has infiltrated surrounding tissue, radiation therapy and/or chemotherapy are the therapeutic modalities used when complete surgical removal (discussed below) is not feasible.

**SURGICAL MANAGEMENT**

Many mediastinal tumors are benign and operable. The location of the tumor (anterior, middle, or posterior compartments) 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 may include a bilateral anterior thoracotomy (clamshell incision) or video-assisted thoracoscopic surgery (see Chap. 25). The care is the same as for any patient undergoing thoracic surgery. The major complications include hemorrhage, injury to the phrenic or recurrent laryngeal nerve, and infection.

**Chest Trauma**

Approximately 60% of all multisystem trauma victims have some type of chest or thoracic trauma (Owens, Chaudry, Eggerstedt & Smith, 2000). Chest trauma is classified as either blunt or
penetrating. Blunt chest trauma results from sudden compression or positive pressure inflicted to the chest wall. Motor vehicle crashes (trauma due to steering wheel, seat belt), falls, and bicycle crashes (trauma due to handlebars) are the most common causes of blunt chest trauma. Penetrating trauma occurs when a foreign object penetrates the chest wall. The most common causes of penetrating chest trauma include gunshot wounds and stabings.

BLUNT TRAUMA

Although blunt chest trauma is more common, 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

Injuries to the chest are often life-threatening and result in one or more of the following pathologic mechanisms:

- 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 mechanisms frequently result in impaired ventilation and perfusion leading to ARF, 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:

- When the injury occurred
- Mechanism of injury
- Level of responsiveness
- Specific injuries
- Estimated blood loss
- Recent drug or alcohol use
- Prehospital treatment

The initial assessment of thoracic injuries includes assessment of the patient for airway obstruction, tension pneumothorax, open pneumothorax, massive hemothorax, flail chest, and cardiac tamponade. These injuries are life-threatening and need immediate treatment. Secondary assessment would include simple pneumothorax, hemothorax, pulmonary contusion, traumatic aortic rupture, tracheobronchial disruption, esophageal perforation, traumatic diaphragmatic injury, and penetrating wounds to the mediastinum (Owens, Chaudry, Eggerstedt & Smith, 2000). Although listed as secondary, these injuries may be life-threatening as well depending upon the circumstances.

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 should be 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 should be 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; 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 cross-match, electrolytes, oxygen saturation, arterial blood gas analysis, and ECG. The patient is completely undressed to avoid missing additional injuries that can 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. Re-establishing 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 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 re-establishing 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 usually 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 Chap. 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 and are most common in women, patients over age 50, and those using shoulder restraints (Owens, Chaudry, Eggerstedt & Smith, 2000). Rib fractures are the most common type of chest trauma, occurring in more than 60% of patients admitted with blunt chest injury. 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

The patient with sternal fractures has anterior chest pain, overlying tenderness, ecchymosis, crepitus, swelling, and the potential of a chest wall deformity. For the patient with rib fractures, clinical manifestations are similar: severe pain, point tenderness,
and muscle spasm over the area of the fracture, which is 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, collapse of unaltered alveoli (atelectasis), pneumonitis, and hypoxemia. Respiratory insufficiency and failure can be the outcomes of such a cycle.

**ASSESSMENT AND DIAGNOSTIC FINDINGS**

The patient with a sternal fracture 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 of the patient with a sternal fracture is directed toward controlling 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 the respiratory drive. Alternative strategies to relieve pain include an intercostal nerve block and ice over the fracture site; a chest binder may decrease pain on movement. Usually the pain abates in 5 to 7 days, and discomfort can be controlled 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 (Owens, Chaudry, Eggerstedt & Smith, 2000). As a result, the chest wall loses stability and there is subsequent 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 CO₂ retention. Hypotension, inadequate tissue perfusion, and metabolic acidosis often follow 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. The 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 intravenous opioids.

For mild to moderate flail chest injuries, the underlying pulmonary contusion is treated by monitoring fluid intake and appropriate fluid replacement, while at the same time 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.

When a severe flail chest injury is encountered, 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 to treat the underlying conditions.
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.

In rare circumstances, surgery may be required to more quickly stabilize the flail segment. This may be used in the patient who is difficult to ventilate or the high-risk patient 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 control thoracic pain.

**Pulmonary Contusion**

Pulmonary contusion is observed in about 20% of adult patients with multiple traumatic injuries and in a higher percentage of children due to increased compliance of the chest wall. 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). It may not be evident initially on examination but will develop in the posttraumatic period.

**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 alveolar surface, 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 tachypnea, tachycardia, pleuritic chest pain, hypoxemia, and blood-tinged secretions to severe tachypnea, tachycardia, crackles, frank bleeding, severe hypoxemia, and respiratory acidosis. Changes in sensorium, including increased agitation or combative irrational behavior, may be signs of hypoxemia.

In addition, the patient with moderate pulmonary contusion has a large amount of mucus, serum, and frank blood in the tracheobronchial tree; the patient often has a constant cough but cannot clear the secretions. A patient with severe pulmonary contusion has the signs and symptoms of ARDS; these may include central cyanosis, agitation, combative, 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 chest x-ray may show pulmonary infiltration. The initial chest x-ray may show no changes; in fact, changes may not appear for 1 or 2 days after the injury.

**MEDICAL MANAGEMENT**

Treatment priorities include maintaining the airway, providing adequate oxygenation, and controlling pain. In mild pulmonary contusion, adequate hydration via intravenous 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.

The patient with moderate pulmonary contusion may require bronchoscopy to remove secretions; intubation and mechanical ventilation with PEEP may also be necessary to maintain the pressure and keep the lungs inflated. Diuretics may be given to reduce edema. A nasogastric tube is inserted to relieve gastrointestinal distention.

The patient with severe contusion may develop respiratory failure and may require aggressive treatment with endotracheal intubation and ventilatory support, diuretics, and fluid restriction. 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 types of penetrating chest trauma. They are classified according to their velocity. Stab wounds are generally considered of low velocity 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 to the chest may be classified as of 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 construction and size of the bullet. A gunshot wound can produce a variety of pathophysiologic changes. A bullet can cause damage at the site of penetration and along its pathway. It also may ricochet off bony structures and damage the chest organs and great vessels. If the diaphragm is involved in either 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, the patient is examined for...
shock and intra-thoracic and intra-abdominal injuries. The patient is undressed completely so that additional injuries will not be 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.

After the status of the peripheral pulses is assessed, a large-bore intravenous line is inserted. The diagnostic workup includes a chest x-ray, chemistry profile, arterial blood gas analysis, pulse oximetry, and ECG. Blood typing and cross-matching are done in case blood transfusion is required. An indwelling catheter is inserted to monitor urinary output. A nasogastric tube is inserted 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. Chest x-rays are obtained, and other 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 continued 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 intra-thoracic bleeding, which would make surgical exploration necessary. If the patient has a penetrating wound of the heart and 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 compared to atmospheric pressure; 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 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

Traumatic pneumothorax occurs when air escapes from a laceration in the lung itself and enters the pleural space or enters the pleural space through a wound in the chest wall. It can occur with blunt trauma (eg, rib fractures) or penetrating chest trauma. It may also occur from abdominal trauma (eg, stab wounds or gunshot wounds to the abdomen) and from diaphragmatic tears. Traumatic pneumothorax may occur with 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.

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, and lacerations of the lungs). Often both blood and air are found in the chest cavity (hemopneumothorax) after major trauma. Chest surgery can cause what is 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 hole 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.

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. Severe chest pain may occur, accompanied by tachypnea, decreased movement of the affected side of the thorax, a tympanic sound on percussion of the chest wall, and decreased or absent breath sounds and tactile fremitus on the affected side.

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 French) 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 leaves a less visible scar. If the patient also has a hemothorax, a large-diameter chest tube (32 French 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 patient’s vascular system.

NURSING ALERT Traumatic open pneumothorax calls for emergency interventions. Stopping the flow of air through the opening in the chest wall is a life-saving measure.
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 permit air and fluid to drain. 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 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) when more than 1,500 mL of blood is aspirated initially by thoracentesis (or is the initial chest tube output) or when chest tube output continues at greater than 200 mL/hour. The urgency with which the blood must be removed is determined by the respiratory compromise. An emergency thoracotomy may also be performed in the emergency department if there is suggested cardiovascular injury secondary to chest or penetrating trauma.

**Tension Pneumothorax**

A tension pneumothorax occurs when air is drawn into the pleural space from a lacerated lung or through a small hole 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 hole 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. The increased intrathoracic pressure 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.

**CLINICAL MANIFESTATIONS**

The clinical picture is one of air hunger, agitation, increasing hypoxemia, central cyanosis, hypotension, tachycardia, and profuse diaphoresis. A comparison of open and tension pneumothorax is shown in Figure 23-9.

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

**MEDICAL MANAGEMENT**

If a tension pneumothorax is suspected, the patient 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, re-establish 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 TAMПONADE**

Cardiac tamponade is the compression of the heart as a result of fluid 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, and 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 the 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.

**Aspiration**

Aspiration of stomach contents into the lungs is a serious complication that may 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 from a variety of factors (Chart 23-11).

**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, may 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 proteaceous 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 pH of less than 2.5 and a volume of gastric aspirate greater than 0.3 mL per kilogram of body weight (20 to 25 mL in adults) (Marik, 2001). Aspiration of gastric contents causes a chemical burn of the tracheobronchial tree and pulmonary parenchyma (Marik, 2001). An inflammatory response occurs. This results in 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 following 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 (Marik, 2001).

**Prevention**

Prevention is the primary goal when caring for patients at risk for aspiration.

**COMPENSATING FOR ABSENT REFLEXES**

Aspiration is likely to 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 in a supine position, has the upper extremities immobilized by intravenous infusions or hand restraints, receives local anesthetics to the oropharyngeal or laryngeal area for diagnostic procedures, has been sedated, or has had long-term intubation.

When vomiting, a person can normally protect the 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**

Even when the patient is intubated, aspiration may occur even with a nasogastric tube in place. This aspiration may result in nosocomial pneumonia. Assessment of tube placement is key to prevent aspiration. The best method for determining tube placement is via an x-ray. There are other nonradiologic methods that have been studied. Observation of the aspirate and testing of its pH are the most reliable. Gastric fluid may be grassy green, brown, clear, or colorless. An aspirate from the lungs may be off-white or tan mucus. Pleural fluid is watery and usually straw-colored (Metheny & Titler, 2001). Gastric pH values are typically lower or more acidic that that of the intestinal or respiratory tract. Gastric pH is usually between 1 and 5, while intestinal or respiratory pH is 7 or higher (Metheny & Titler, 2001). There are differences in assessing tube placement with continuous versus intermittent feedings. For intermittent feedings with small-bore tubes, observation of aspirated contents and pH evaluation should be performed. For continuous feedings, the pH method
feeding should be delayed or the continuous feeding stopped for a period of time.

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 rehabilitation therapist experienced in speech and swallowing disorders work with the patient to assess the swallowing reflex.

CRITICAL THINKING EXERCISES

1. Your patient, a 44-year-old unemployed man who lives with his 80-year-old mother, has recently been diagnosed with active TB. He has been started on treatment and given specific instructions about his medications. What strategies would you initiate to be sure that he takes his medications correctly? What strategies would you use to ensure that his mother is not infected? How would your care differ if the patient lived alone or were homeless?

2. You are working on a surgical unit. Your patient is a 67-year-old woman who has had surgery to repair a fractured hip that occurred following a fall associated with heavy alcohol use. She has been a heavy smoker for over 35 years and is reluctant to move in bed because of pain. What are the potential postoperative pulmonary complications? What assessment criteria would you use to assess her respiratory status? What interventions would you implement to prevent pulmonary complications in this patient? What changes, if any, would you implement if she had a history of deep vein thrombosis?

3. Your patient has experienced blunt chest trauma following a motor vehicle crash. A chest tube has been inserted to treat a pneumothorax. The chest drainage system has drained 400 mL of light-red fluid during the first 6 hours following the tube’s insertion. The patient is unable to recall how he was injured or what has happened to him over the last 24 hours. The patient is experiencing pain requiring opioids and is asking that the chest tube be removed to enable him to walk to the bathroom. What additional information would you obtain through assessment and what actions would you take? How would you explain to the patient and his family the purpose of the chest tubes? How would you modify your explanation and teaching if he has little understanding of English?

REFERENCES AND SELECTED READINGS

Books


### Pulmonary Infections


### Trauma


### Tuberculosis


**RESOURCES AND WEBSITES**
American Association for Respiratory Care, 1720 Regal Row, Dallas, TX 75235; 1-214-630-3540; [http://www.aarc.org](http://www.aarc.org).
American Cancer Society, 1599 Clifton Road NE, Atlanta, GA 30329; 1-888-ACS-5552; [http://www.cancer.org](http://www.cancer.org).
American College of Chest Physicians, 3300 Dundee Road, Northbrook, IL 60062; 1-847-498-1400; [http://www.chest.org](http://www.chest.org).
Centers for Disease Control and Prevention, 1600 Clifton Road, NE, Atlanta, GA 30333; [http://www.cdc.gov](http://www.cdc.gov).
National Cancer Institute, National Institutes of Health, 31 Center Drive MSC 2580, Bldg. 31, Room 10A16, Bethesda, MD 20892; 1-800-4-CANCER (Cancer Information Services); [http://www.cancer.gov](http://www.cancer.gov).