LEARNING OUTCOMES

For clinical competence and success on the NCLEX Examination, study this chapter with these Learning Outcomes in mind:

Safe and Effective Care Environment
1. Ensure safe oxygen delivery.
2. Ensure the proper oxygen flow rate for patients with hypercarbia.
3. Use appropriate infection control methods to protect the patient with cystic fibrosis from respiratory infections.
4. Ensure appropriate functioning of the chest tube drainage system after a thoracotomy.

Health Promotion and Maintenance
5. Encourage everyone to not smoke or to quit smoking.
6. Encourage all people who are exposed to inhalation irritants in the workplace or at home to use appropriate protection.
7. Teach patients with chronic airflow limitation how to use a peak flowmeter.
8. Teach patients using aerosol or dry powder inhalers for drug delivery the correct way to use these devices.
9. Teach patients who are using preventive drug therapy for asthma the importance of taking the prescribed drugs daily, even when asthma symptoms are not present.
10. Teach patients with asthma to have their rescue inhalers with them at all times.

Psychosocial Integrity
11. Encourage the patient and family to express their feelings about a change in breathing status.
12. Explain all therapeutic procedures, restrictions, and follow-up care to the patient and his or her family.
13. Teach the patient with activity limitations from respiratory problems how to modify techniques and conserve energy to perform ADLs and desired activities independently.

Physiological Integrity
14. Compare the pathophysiology and clinical manifestations of asthma, bronchitis, and emphysema.
15. Identify risk factors for chronic obstructive pulmonary disease (COPD) and lung cancer.
16. Use laboratory data and clinical manifestations to determine the effectiveness of therapy for impaired gas exchange in a patient with breathing problems.
17. Interpret peak expiratory flow (PEF) readings for the need for intervention.
18. Coordinate care needs for the patient immediately after lung volume reduction surgery.
20. Coordinate nursing interventions for the patient with chest tubes.

Go to your Companion CD or Evolve at http://evolve.elsevier.com/Iggy/ for Self-Assessment Questions for the NCLEX Examination keyed to these Learning Outcomes.
lower airway problems directly affect gas exchange and have serious consequences for the human need for oxygenation and tissue perfusion. Many of these problems are chronic and progressive, requiring major changes in a person’s lifestyle. The older patient with a lower airway problem may need special help even before the disorder becomes severe because of age-related changes in breathing effectiveness. Chart 32-1 lists nursing issues for the older patient with a respiratory problem.

**CHRONIC AIRFLOW LIMITATION**

Chronic airflow limitation (CAL) is a group of chronic lung diseases that include asthma, chronic bronchitis, and pulmonary emphysema. Emphysema and chronic bronchitis, termed chronic obstructive pulmonary disease (COPD), are characterized by bronchospasm and dyspnea (see Figure 32-1). The tissue damage is not reversible and increases in severity, eventually leading to respiratory failure. Asthma, unlike COPD, is an intermittent disease with reversible airflow obstruction and wheezing.

More than 40 million Americans suffer from some form of CAL, and 1 million people between the ages of 40 and 65 years have moderate to severe disability from CAL (Centers for Disease Control and Prevention [CDC], 2007). Although some problems are not reversible, good management strategies can help maintain adequate oxygenation and tissue perfusion, as well as improve overall health.

**ASTHMA**

**Pathophysiology**

Bronchial asthma is an intermittent and reversible airflow obstruction affecting only the airways, not the alveoli (Figure 32-1). Airway obstruction can occur in two ways: (1) inflammation and (2) airway hyperresponsiveness (sometimes called “twitchy airways”). Inflammation obstructs the lumen (i.e., the inside) of airways. Airway hyperresponsiveness obstructs airways by constricting bronchial smooth muscle causing a narrowing of the airway from the outside. Airway inflammation can trigger bronchiolar hyperresponsiveness, and many people with asthma have both problems at the same time. Severe airway obstruction can be fatal. More than 5000 deaths from acute asthma occur in the United States each year (CDC, 2007).

**Etiology and Genetic Risk**

Asthma may be classified into different types based on the events known to trigger the attacks; however, the pathophysiology is similar for all types of asthma regardless of triggering event. Inflammation of the mucous membranes lining the airways is a key event in triggering an asthma attack. Inflammation occurs in response to the presence of specific allergens; general irritants such as cold air, dry air, or fine airborne particles; microorganisms; and aspirin. Airway hyper-responsiveness can occur with exercise, with an upper respiratory illness, and for unknown reasons (Sims, 2006).

**GENETIC CONSIDERATIONS**

Asthma from inflammation or hyperresponsive airways may have a genetic component, although a specific gene or mutation has not yet been identified. In addition, genetic variation in the gene that controls the synthesis and activity of beta adrenergic receptors has an impact on drug therapy for asthma. Patients who have a mutation in this gene do not respond as expected to short-acting or long-acting beta agonists and need to have an altered therapy plan (Conboy-Ellis, 2006). Teaching these patients about why their drug therapies are different from standard recommendations is a nursing responsibility that can assist with therapy adherence.

When asthma is well controlled, the airway changes are temporary and reversible. With poor control, chronic inflammation can lead to damage and hyperplasia of the bronchial epithelial cells and of the bronchial smooth muscle. When asthma attacks are frequent, even exposure to low levels of the triggering agent or event may stimulate an attack.

**Inflammation** triggers asthma for some people when allergens bind to specific antibody molecules (especially immunoglobulin E [IgE]). These molecules are attached to tissue cells called mast cells and white blood cells called basophils. These cells are filled with granules containing chemicals that can start local inflammatory responses (see Chapters 19 and 22). Some of these chemicals, such as histamine, start an immediate inflammatory response, which can be blocked by drugs like diphenhydramine (Benadryl). Others, such as leukotriene and estoxin, are slower and cause later, prolonged inflammatory responses, which can be blocked by drugs like montelukast (Singulair), zafirlukast (Accolate), and zileuton (Zyflo). All these chemicals also attract more white blood cells (eosinophils, macrophages, basophils) to the area, which then release even more inflammatory-inducing chemicals (mediators). Inflammation of airway mucous membranes causes blood vessel dilation and capillary leak, leading to tissue swelling with increased secretions and mucus production (McCance & Huether, 2006; Sims, 2006). Inflammation can also occur through general irritation rather than allergic responses. Although some of the same cells and chemicals cause this response, allergy therapy is not useful for general irritation-induced asthma.

**Chart 32-1  NURSING FOCUS ON THE OLDER ADULT**

**Chronic Respiratory Disorder**

- • Provide rest periods between such activities as bathing, meals, and ambulation.
- • Place the patient in an upright position for meals to prevent aspiration.
- • Encourage nutritional fluid intake after the meal to promote increased calorie intake.
- • Schedule drugs around routine activities to increase adherence to drug therapy.
- • Arrange chairs in strategic locations to allow the patient with dyspnea to walk and rest as needed.
- • Encourage prompt access to a health care facility for any manifestation of infection.
- • Ensure that the patient has received the pneumococcal vaccine.
- • Encourage the patient to have an annual influenza vaccination.
Bronchospasm is a narrowing of the bronchial tubes through constriction of the smooth muscle around and within the bronchial walls. It occurs in some people as a result of airway hyperresponsiveness when small amounts of pollutants or respiratory viruses stimulate nerve fibers, causing constriction of bronchial smooth muscle. If these substances also stimulate an inflammatory response at the same time, the chemicals released during inflammation also trigger constriction. Severe bronchospasm alone, especially in smaller bronchioles, can profoundly limit airflow to the alveoli.

Aspirin and other NSAIDs can trigger asthma in some people although this response is not a true allergy. It results from increased production of leukotriene when aspirin or NSAIDs suppress other inflammatory pathways.

Incidence/Prevalence
Asthma can occur at any age. About half of adults with asthma also had the disease in childhood. Asthma is more common in urban settings than in rural settings, possibly as a result of more air pollution.
CONSIDERATIONS FOR OLDER ADULTS

Asthma occurs as a new disorder in about 3% of people older than 55 years. Another 3% of people older than 60 years have asthma as a continuing chronic disorder (CDC, 2007). Lung and airway changes as a part of aging make any breathing problem more serious in the older adult. One problem related to aging is a change in the sensitivity of beta-adrenergic receptors. When stimulated, these receptors relax smooth muscle and cause bronchodilation. As these receptors become less sensitive, they no longer respond as quickly or as strongly to agonists (epinephrine, dopamine) and beta-adrenergic drugs, which are often used as rescue therapy during an acute asthma attack. Thus teaching older patients how to avoid asthma attacks and to correctly use preventive drug therapy is a nursing priority.

WOMEN’S HEALTH CONSIDERATIONS

The incidence of asthma is about 35% higher among women than men, and the asthma death rates are also higher among women. Obesity and hormonal fluctuations around the menstrual cycle are thought to contribute to the difference in incidence, and undertreatment of the disease is thought to be a factor in the higher death rate. Teaching women with asthma how to be partners in asthma management and the correct use of both preventive and rescue drugs remains a nursing priority in improving the outcomes of the disease (Ostrom & Goergen, 2006).

Patient-Centered Collaborative Care

Assessment

Asthma is diagnosed and classified on the basis of the frequency and severity of the manifestations, as well as on the patient’s response to asthma drugs. These classes are the basis for current asthma therapy (Chart 32-2).

History

The patient with asthma usually has a pattern of episodes of dyspnea (shortness of breath), chest tightness, coughing, wheezing, and increased mucus production. Ask whether the manifestations occur continuously, seasonally, in association with specific activities or exposures, or more frequently at night. Some patients notice these manifestations lasting 4 to 8 weeks after a chest cold or other upper respiratory tract infection. The patient with atopic (allergic) asthma may also have other allergic symptoms such as rhinitis, skin rash, or pruritus. Ask whether any other family members have asthma or respiratory problems. Ask about the patient’s current or previous smoking habits. If the patient smokes, use this opportunity to teach him or her about smoking cessation (Chart 32-3). Wheezing in nonsmokers is an important symptom in the diagnosis of asthma.

Physical Assessment/Clinical Manifestations

The patient with mild to moderate asthma may have no manifestations between asthma attacks. During an acute episode, the most common manifestations are an audible wheeze and increased respiratory rate. The wheeze is louder on expiration. When inflammation occurs with asthma, coughing may increase.

The patient may use accessory muscles to help breathe during an attack. Observe for muscle retraction at the sternum and the suprasternal notch and between the ribs. The patient with long-standing, severe asthma may have a “barrel chest,” caused by air trapping (Figure 32-2). The anteroposterior (AP) diameter (diameter between the front and the back of the chest) increases with air trapping, giving the chest a rounded rather than an oval shape. The normal chest is nearly twice as wide as it is thick. In the patient with severe, chronic asthma, the AP diameter may equal or exceed the lateral diameter. Compare the AP diameter of the chest with the lateral diameter. Air trapping also increases the space between the ribs.

Along with an audible wheeze, the breathing cycle is longer and requires more effort. The patient may be unable to complete a sentence of more than five words between breaths. Examine the oral mucosa and nail beds for cyanosis. Pulse oximetry shows hypoxemia (poor blood oxygen levels) related to the degree of dyspnea. Other indicators of hypoxemia include changes in the level of cognition or consciousness and tachycardia.

Laboratory Assessment

Laboratory tests can help determine the type of asthma and the degree of breathing impairment. Arterial blood gas (ABG) levels show how well the patient is obtaining oxygen (see Chapter 14 for discussion of ABGs). The arterial oxygen level (Pao2) may decrease during an asthma attack. Early in the attack, the arterial carbon dioxide level (Paco2) may be decreased as the patient increases respiratory effort. Later in an asthma episode, Paco2 rises, indicating carbon dioxide retention and poor gas exchange. Allergic asthma often occurs with an elevated serum eosinophil count and immunoglobulin E (IgE) levels. The sputum may contain eosinophils and mucous plugs with shed epithelial cells (Curschmann spirals).

Pulmonary Function Tests

The most accurate tests for asthma are the pulmonary function tests (PFTs) measured using spirometry. Baseline PFTs are obtained for all patients diagnosed with asthma. The most important PFTs for a patient with asthma are:

- Forced vital capacity (FVC) (volume of air exhaled from full inhalation to full exhalation)
- Forced expiratory volume in the first second (FEV1) (volume of air blown out as hard and fast as possible during the first second of the most forceful exhalation after the greatest full inhalation)
- Peak expiratory flow (PEF) (fastest airflow rate reached at any time during exhalation).

A decrease in either the FEV1 or the PEF of 15% to 20% below the expected value for age, gender, and size is common for the patient with asthma. An increase of 12% in these values after treatment with bronchodilators is diagnostic for asthma. Airway responsiveness is tested by measuring the PEF and FEV1 before and after the patient inhales the drug methacholine, which induces bronchospasm in susceptible people.

Other Diagnostic Assessment

Chest x-rays may be used to rule out other causes of dyspnea or to track changes in chest structure over time. For the patient taking theophylline, blood drug levels are used to determine whether a therapeutic level is being maintained.
**Interventions**

The goals of asthma therapy are to improve airflow, relieve symptoms, and prevent episodes. Adult asthma is best managed when the patient is an active partner in the management plan. Priority nursing actions focus on patient education about drug therapy and lifestyle management, including exercise, to assist the patient in understanding his or her disease and its treatment.

### Chart 32-2  KEY FEATURES

**Asthma: The Step System**

<table>
<thead>
<tr>
<th>Clinical Manifestations</th>
<th>Treatment Recommendations</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Step I. Mild Intermittent</strong></td>
<td>No daily drugs needed</td>
</tr>
<tr>
<td>Symptoms or episodes occur less than once a week.</td>
<td>Use of short-acting inhaled beta agonist during episodes (rescue inhaler)</td>
</tr>
<tr>
<td>Episodes/exacerbations are short, lasting only a few hours.</td>
<td>Increased use of rescue inhaler more than 2 days per week (except for aftermath of viral infections or exercise-induced bronchospasms) indicates the need to start the next step in long-term therapy</td>
</tr>
<tr>
<td>Symptoms are present at night no more frequently than twice per month.</td>
<td></td>
</tr>
<tr>
<td>PFTs are normal between episodes.</td>
<td></td>
</tr>
<tr>
<td>During episodes/exacerbations, FEV₁ or PEF is at least 80% of normal.</td>
<td></td>
</tr>
<tr>
<td>PEF or FEV₁ variability is less than 20%.</td>
<td></td>
</tr>
<tr>
<td><strong>Step II. Mild Persistent</strong></td>
<td>Use of a daily anti-inflammatory:</td>
</tr>
<tr>
<td>Symptoms or episodes occur more than once per week but not daily.</td>
<td><em>Inhaled corticosteroid (ICS) low-dose</em></td>
</tr>
<tr>
<td>Symptoms are present at night more than twice per month.</td>
<td>Inhaled cromolyn</td>
</tr>
<tr>
<td>Episodes/exacerbations affect activity and sleep.</td>
<td>Leukotriene antagonist</td>
</tr>
<tr>
<td>During episodes/exacerbations, FEV₁ or PEF is at least 80% of normal.</td>
<td></td>
</tr>
<tr>
<td>PEF or FEV₁ variability is 20% to 30%.</td>
<td>Increased use of rescue inhaler for relief during episodes</td>
</tr>
<tr>
<td><strong>Step III. Moderate Persistent</strong></td>
<td>Increase use of rescue inhaler more than 2 days per week (except for aftermath of viral infections or exercise-induced bronchospasms) indicates the need to start the next step in long-term therapy</td>
</tr>
<tr>
<td>Symptoms occur daily.</td>
<td></td>
</tr>
<tr>
<td>Episodes/exacerbations affect activity and sleep.</td>
<td><em>Add a daily long-acting beta agonist to low-dose ICS or Continue ICS alone but increase to medium-dose range or add one of the following to low-dose ICS:</em></td>
</tr>
<tr>
<td>Symptoms are present at night more than once per week.</td>
<td>Leukotriene receptor antagonist</td>
</tr>
<tr>
<td>During episodes/exacerbations, FEV₁ or PEF is only 60% to 80% of normal.</td>
<td>Theophylline</td>
</tr>
<tr>
<td>PEF or FEV₁ variability is greater than 30%.</td>
<td>Zileuton</td>
</tr>
<tr>
<td><strong>Step IV. Severe Persistent</strong></td>
<td>Increased use of rescue inhaler more than 2 days per week (except for aftermath of viral infections or exercise-induced bronchospasms) indicates the need to start the next step in long-term therapy</td>
</tr>
<tr>
<td>Symptoms occur daily.</td>
<td><em>Medium-dose ICS and long-acting beta agonist or Medium-dose ICS and either leukotriene receptor antagonist or theophylline</em></td>
</tr>
<tr>
<td>Episodes/exacerbations are frequent.</td>
<td>Increased use of rescue inhaler more than 2 days per week (except for aftermath of viral infections or exercise-induced bronchospasms) indicates the need to start the next step in long-term therapy</td>
</tr>
<tr>
<td>Symptoms are present at night frequently.</td>
<td><em>High-dose ICS and long-acting beta agonist</em></td>
</tr>
<tr>
<td>Activities are limited.</td>
<td>Omalizumab considered for patients with constant exposure to non-seasonal allergens</td>
</tr>
<tr>
<td>During episodes/exacerbations, FEV₁ or PEF is at 60% or less of normal.</td>
<td><em>High-dose ICS, oral corticosteroids (at lowest possible dose daily or every other day), and long-acting beta agonist</em></td>
</tr>
<tr>
<td>PEF or FEV₁ variability is greater than 30%.</td>
<td>Omalizumab considered for patients with constant exposure to non-seasonal allergens</td>
</tr>
<tr>
<td><strong>Step V. Severe Persistent not responsive to previous step</strong></td>
<td></td>
</tr>
<tr>
<td><strong>Step VI. Severe Persistent not responsive to previous step</strong></td>
<td></td>
</tr>
</tbody>
</table>

*Preferred drug regimen.

**Patient Education**

Asthma is often an intermittent disease. With guided self-care, patients can co-manage this disease, increasing symptom-free periods and decreasing the number and severity of attacks (Ellis, 2008). Good management decreases the number of hospital admissions and increases participation in patient-chosen pleasure, work, and family activities. Self-care requires...
extensive education for the patient to be able to self-assess respiratory status, self-treat (including adjusting the frequency and dosage of prescribed drugs), and determine when to consult the health care provider.

Teach the patient to assess symptom severity at least twice daily with a peak flowmeter and adjust drugs to manage inflammation and bronchospasms to prevent or relieve symptoms. Chart 32-4 describes the correct method to use the meter. The patient should first establish a baseline or “person best” peak expiratory flow (PEF) by measuring his or her PEF twice daily for 2 to 3 weeks when asthma is well controlled and recording the results (Pruitt, 2005). This way, the patient will know when his or her peak flow is reduced to the point that more drugs are needed or that emergency assistance is needed. When the patient has established a “personal best,” all other readings are compared with this value in terms of percent of personal best. Some meters are color-coded to help the patient interpret the results. Green zone readings are at least...
80% or above the “personal best.” This is the ideal range for asthma control and indicates that no increases in drug therapy are needed. Yellow is a range between 50% and 80% of personal best. When a patient has a reading in this range, he or she needs to use the “rescue drug,” as prescribed. Within a few minutes after using the rescue drug, another PEF reading should be made to determine whether the rescue is working. Frequent or consistent readings in the yellow zone indicate the need for a change in preventive (control) drugs. Red is a range below 50% of the patient’s personal best and indicates serious respiratory obstruction. Teach the patient who has a reading in the red zone to immediately use the rescue drugs and seek emergency help.

Education involves a specified drug therapy plan that is tailored to meet the personal pattern of asthma for the patient (Pruitt & Jacobs, 2005). Teach the patient to keep a symptom and intervention diary to learn his or her triggers of asthma symptoms, early cues for impending attacks, and personal response to drugs. Stress the importance of proper use of the asthma action plan for any severity of asthma. Chart 32-4 lists areas to emphasize when teaching the patient with asthma.

**Drug Therapy**
Pharmacologic management of adult patients with asthma is based on the step category for severity and treatment (see Chart 32-2) (National Institutes of Health, 2007). **Preventive therapy drugs** are those used to change airway responsiveness to prevent asthma attacks from occurring. They are used every day, regardless of symptoms. **Rescue drugs** are those used to actually stop an attack once it has started. Some patients may need drug therapy only during an asthma episode. For others, daily drugs are needed to keep asthma episodic rather than a more frequent problem. This therapy involves the use of bronchodilators and various drug types to reduce inflammation. Some drugs reduce the asthma response, and other drugs actually prevent the response. Combination drugs are two agents from different classes combined together for better response. Chart 32-5 lists the most common preferred drugs in each class for preventive and rescue (symptomatic) therapy of asthma. The actions, interventions, and rationales for most drugs within a single class are similar although drug dosages may differ. Be sure to consult a pharmacology text or drug handbook for more information on a specific drug.

**Bronchodilators.** Bronchodilators increase bronchiolar smooth muscle relaxation. They have no effect on inflammatory processes. Thus when a patient with asthma has airflow obstruction by both bronchospasm and inflammation, at least two types of drug therapy are needed. Bronchodilators work by stimulating the beta, 2-adrenergic receptors on bronchial smooth muscle in much the same way that the sympathetic nervous system transmitters epinephrine and norepinephrine do. These drugs include beta, 2 agonists, cholinergic antagonists, and methylxanthines. Beta, 2 agonists bind to the beta, 2-adrenergic receptors and cause an increase in the intracellular level of a substance called cyclic adenosine monophosphate (cAMP). This substance triggers smooth muscle relaxation.

Short-acting beta, 2 agonists (SABAs) provide rapid but short-term relief. These inhaled drugs are most useful when an attack begins (rescue drug) or as premedication when the...
# COMMON EXAMPLES OF DRUG THERAPY

## Asthma Prevention and Treatment

<table>
<thead>
<tr>
<th>Drug/Usual Dosage</th>
<th>Purpose/Action</th>
<th>Nursing Interventions</th>
<th>Rationale</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Bronchodilators</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Short-Acting Beta Agonist (SABA)</strong></td>
<td>Causes bronchodilation by relaxing bronchiolar smooth muscle through binding to and activating pulmonary beta2 receptors. Primary use is a fast-acting “rescue” drug to be used either during an asthma attack or just before engaging in activity that usually triggers an attack.</td>
<td>Teach patient to carry with him or her at all times. Teach patient to monitor heart rate. When taking this drug with other inhaled drugs, teach patient to use this drug at least 5 minutes before the other inhaled drugs. Teach patient the correct technique for using the MDI or DPI, and obtain a return demonstration.</td>
<td>The drug can stop or reduce life-threatening bronchoconstriction, which can occur anytime. Excessive use causes systemic symptoms, especially tachycardia. The bronchodilation effect of the drug allows better penetration of the other inhaled drugs. Correct technique is essential to getting the drug to the site of action. Poor technique allows the drug to escape through the nose and mouth.</td>
</tr>
<tr>
<td>Albuterol (Proventil, Ventolin) 1-2 inhalations every 4-6 hr (90 mcg/inhaled dose)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Long-Acting Beta Agonist (LABA)</strong></td>
<td>Causes bronchodilation by relaxing bronchiolar smooth muscle through binding to and activating pulmonary beta2 receptors. Onset of action is slow with a long duration. Primary use is prevention of an asthma attack.</td>
<td>Teach patient to shake inhaler (MDI) well before using. Teach patient to not use this drug with the onset of asthma symptoms or worsening of wheezing. Teach patient the correct technique for using the MDI or DPI, and obtain a return demonstration.</td>
<td>Drug separates easily. Drug has slow onset of action and does not relieve or reverse symptoms. Correct technique is essential to getting the drug to the site of action. Poor technique allows the drug to escape through the nose and mouth.</td>
</tr>
<tr>
<td>Salmeterol (Serevent) 2 inhalations every 12 hr (25 mcg/inhalation with MDI) (50 mcg/inhalation with DPI)</td>
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</tr>
<tr>
<td><strong>Cholinergic Antagonist</strong></td>
<td>Causes bronchodilation by inhibiting the parasympathetic nervous system, allowing the sympathetic system to dominate, releasing norepinephrine that activates beta2 receptors. Purpose is to both rescue and prevent asthma. Drug does not work as well as SABAs but can be used in place of SABAs by patients who cannot tolerate side effects of beta2 agonists.</td>
<td>If patient is to use this as a “rescue” drug, teach him or her to carry it at all times. Teach patient to shake MDI well before using. Teach patient to drink at least 4 L of fluid daily unless another health problem requires fluid restriction. Teach patient to observe for and report blurred vision, eye pain, headache, nausea, palpitations, tremors, inability to sleep. Teach patient the correct technique for using the MDI or DPI, and obtain a return demonstration.</td>
<td>The drug can stop or reduce life-threatening bronchoconstriction, which can occur anytime. Drug separates easily. Drug causes mouth dryness. These are systemic symptoms of overdose and require intervention. Correct technique is essential to getting the drug to the site of action. Poor technique allows the drug to escape through the nose and mouth.</td>
</tr>
<tr>
<td>Ipratropium (Atrovent, Apo-Ipravent) 2-4 inhalations 4-6 times daily (18 mcg/inhalation)</td>
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</tbody>
</table>

### COMMON EXAMPLES OF DRUG THERAPY

#### Asthma Prevention and Treatment—cont’d

<table>
<thead>
<tr>
<th>Drug/Usual Dosage</th>
<th>Purpose/Action</th>
<th>Nursing Interventions</th>
<th>Rationale</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Methylxanthines</strong></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Theophylline (Elixophyllin, Theo-Dur, Uniphyl, Theolair, many others)</td>
<td>Acts like caffeine to cause bronchodilation by relaxing bronchiolar smooth muscles through inhibiting an enzyme that breaks down the intracellular trigger for relaxation.</td>
<td>Understand that a higher dose is required at the beginning of therapy (loading dose) than is used to maintain the effect. Teach patient to take the daily dose in evenly spaced divided doses. Teach patient to make and keep appointments to monitor blood levels of the drug. Teach patient not to drink coffee or other caffeinated beverages while on this drug.</td>
<td>Drug requires a specific blood level to work. A loading dose is required to achieve this level. Then, lower doses are used for maintenance because the drug is eliminated slowly. Maintains an even blood level and has a better effect. Drug has a narrow margin of safety and many severe side effects. Drug is similar to caffeine, and taking it with caffeine increases the risk for toxicity.</td>
</tr>
<tr>
<td>5 mg/kg IV or 10-12 mg/kg orally as loading dose 200-800 mg orally daily</td>
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<td></td>
<td></td>
</tr>
<tr>
<td><strong>Anti-Inflammatories</strong></td>
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</tr>
<tr>
<td>All of these drugs help improve bronchiolar airflow by decreasing the inflammatory response of the mucous membranes in the airways. They do not cause bronchodilation.</td>
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<td></td>
</tr>
<tr>
<td><strong>Corticosteroid</strong></td>
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</tr>
<tr>
<td>Fluticasone (Flovent) 50 mcg by MDI twice daily 100-250 mcg by DPI daily</td>
<td>Disrupts all known production pathways of inflammatory mediators. The main purpose is to prevent an asthma attack caused by inflammation or allergies.</td>
<td>Teach patient to use the drug daily, even when no symptoms are present. Teach patient to perform good mouth care and to check the mouth daily for lesions or drainage. Teach patient to not use this drug with the onset of asthma symptoms or worsening of wheezing. Teach patient the correct technique for using the MDI or DPI, and obtain a return demonstration.</td>
<td>Maximum effectiveness requires continued use for 48-72 hr and depends on regular use. Drug reduces local immunity and increases the risk for local infections, especially <em>Candida albicans</em> (yeast). Drug has slow onset of action and does not relieve or reverse symptoms. Correct technique is essential to getting the drug to the site of action. Poor technique allows the drug to escape through the nose and mouth.</td>
</tr>
<tr>
<td>Prednisone (Deltasone, Predone) 1-40 mg orally daily</td>
<td>Not recommended unless asthma symptoms cannot be controlled with any other therapy.</td>
<td>Teach patient about the numerous expected side effects (GI ulceration, fat redistribution, weight gain, hyperglycemia). Teach patient to avoid anyone who has an upper respiratory infection. Teach patient to avoid activities that lead to injury. Teach patient to take drug with food. Teach patient not to suddenly stop taking the drug for any reason. If patient cannot take the oral drug because of vomiting, he or she should receive the drug parenterally.</td>
<td>Knowing the side effects to expect reduces anxiety. Drug reduces all protective inflammatory responses, increasing the risk for infection. Blood vessels become more fragile, leading to bruising and petechiae. The drug increases the risk for GI ulceration; food helps reduce the risk. The drug suppresses adrenal production of corticosteroids, which are essential for life.</td>
</tr>
</tbody>
</table>

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Continued
The patient is about to begin an activity that is likely to induce an asthma attack (Fitzgerald, 2006; National Institutes of Health, 2007). Such agents include albuterol (Proventil, Ventolin), bitolterol (Tornalate), levalbuterol (Xopenex), pirbuterol (Maxair), and terbutaline (Brethaire). When inhaled from either a metered dose inhaler (MDI) or a dry powder inhaler (DPI), the drug is delivered directly to the site of action and systemic effects are minimal (unless the agent is overused or abused). Teach the patient the correct technique to use with an inhaled drug to achieve the greatest benefit from the drug. Chart 32-6 describes the proper way to use an MDI. Figure 32-3 shows a patient using a “spacer” with an MDI. Chart 32-7 describes the proper care and use of a DPI. Figure 32-4 shows a patient using a DPI.

**Chart 32-5 COMMON EXAMPLES OF DRUG THERAPY**

**Asthma Prevention and Treatment—cont’d**

<table>
<thead>
<tr>
<th>Drug/Usual Dosage</th>
<th>Purpose/Action</th>
<th>Nursing Interventions</th>
<th>Rationale</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>NSAID</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Nedocromil (Tilade) 4 mg by MDI every 6 hr</td>
<td>Stabilizes the membranes of mast cells and prevents the release of inflammatory mediators. Purpose is to prevent asthma attack triggered by inflammation or allergens.</td>
<td>Teach patient to use the drug daily, even when no symptoms are present. Teach patient to not use this drug with the onset of asthma symptoms or worsening of wheezing. Teach patient the correct technique for using the MDI, and obtain a return demonstration.</td>
<td>Drug has slow onset of action for asthma prevention and is most effective when taken consistently. Drug does not relieve or reverse symptoms. Correct technique is essential to getting the drug to the site of action. Poor technique allows the drug to escape through the nose and mouth.</td>
</tr>
<tr>
<td><strong>Leukotriene Antagonist</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Montelukast (Singular) 10 mg orally daily</td>
<td>Blocks the leukotriene receptor, preventing the inflammatory mediator from stimulating inflammation. Purpose is to prevent asthma attack triggered by inflammation or allergens.</td>
<td>Teach patient to use the drug daily, even when no symptoms are present. Teach patient not to decrease the dose of or stop taking any other asthma drugs unless otherwise instructed by the health care professional.</td>
<td>Drug has slow onset of action for asthma prevention and is most effective when taken consistently. This drug is for long-term asthma control and does not replace other drugs, especially corticosteroids and rescue drugs.</td>
</tr>
<tr>
<td>Omalizumab (Xolair) 150-375 mg subcutaneously every 2-3 wk</td>
<td>Drug is an antibody that binds to the IgE receptors on mast cells and basophils, preventing allergens from triggering the release of inflammatory mediators. Purpose is prevention of allergen-triggered asthma attacks.</td>
<td>Administer drug in a facility equipped to handle anaphylaxis. Do not administer more than 150 mg per injection site. Keep patient at the facility for 30-60 min after injection. Teach patient not to decrease the dose of or stop taking any other asthma drugs unless otherwise instructed by the health care professional.</td>
<td>Drug is associated with anaphylaxis. Larger doses can cause severe injection site reactions with bruising, erythema, warmth, burning, stinging, pruritus, hives, pain, induration, mass, and inflammation lasting up to 7 days. Allergic reactions and anaphylaxis are most likely within the first 30-60 min after injection. This drug may take months before it is effective and, even then, does not stop an attack that has started. It is an additional drug for allergic asthma and does not replace other drugs.</td>
</tr>
</tbody>
</table>
Teach the patient to always carry the rescue drug inhaler with him or her and to ensure that enough drug remains in the inhaler to provide a quick dose when needed. Dry powder inhalers indicate the amount of remaining drug; however, aerosol (MDI) inhalers do not. Demonstrate how to check aerosol inhaler drug levels by placing the inhaler in water (Figure 32-5). Full inhalers sink to the bottom. An empty inhaler floats on its side.

**Chart 32-6  PATIENT AND FAMILY EDUCATION GUIDE**

**How to Use an Inhaler Correctly***

<table>
<thead>
<tr>
<th>With a Spacer</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Before each use, remove the caps from the inhaler and the spacer.</td>
</tr>
<tr>
<td>2. Insert the mouthpiece of the inhaler into the non-mouthpiece end of the spacer.</td>
</tr>
<tr>
<td>3. Shake the whole unit vigorously three or four times.</td>
</tr>
<tr>
<td>4. Place the mouthpiece into your mouth, over your tongue, and seal your lips tightly around it.</td>
</tr>
<tr>
<td>5. Press down firmly on the canister of the inhaler to release one dose of medication into the spacer.</td>
</tr>
<tr>
<td>6. Breathe in slowly and deeply. If the spacer makes a whistling sound, you are breathing in too rapidly.</td>
</tr>
<tr>
<td>7. Remove the mouthpiece from your mouth, and, keeping your lips closed, hold your breath for at least 10 seconds and then breathe out slowly.</td>
</tr>
<tr>
<td>8. Wait at least 1 minute between puffs.</td>
</tr>
<tr>
<td>9. Replace the caps on the inhaler and the spacer.</td>
</tr>
<tr>
<td>10. At least once a day, clean the plastic case and cap of the inhaler by thoroughly rinsing in warm, running tap water; at least once a week, clean the spacer in the same manner.</td>
</tr>
</tbody>
</table>

**Without a Spacer (Preferred Technique)**

1. Before each use, remove the cap and shake the inhaler according to the instructions in the package insert.
2. Tilt your head back slightly, and breathe out fully.
3. Open your mouth, and place the mouthpiece 1 to 2 inches away.
4. As you begin to breathe in deeply through your mouth, press down firmly on the canister of the inhaler to release one dose of medication.
5. Continue to breathe in slowly and deeply (usually over 3-5 sec).
6. Hold your breath for at least 10 seconds to allow the medication to reach deep into the lungs, and then breathe out slowly.
7. Wait at least 1 minute between puffs.
8. Replace the cap on the inhaler.
9. At least once a day, remove the canister and clean the plastic case and cap of the inhaler by thoroughly rinsing in warm, running tap water.

**Without a Spacer (Alternative Method)**

1. Follow steps 1 and 2 of the preferred technique for using an inhaler without a spacer.
2. Place the mouthpiece into your mouth, over your tongue, and seal your lips tightly around it.
3. Follow steps 4 to 9 of the preferred technique for using an inhaler without a spacer.

* Avoid spraying in the direction of the eyes.

**Chart 32-7  PATIENT AND FAMILY EDUCATION GUIDE**

**How to Use a Dry Powder Inhaler (DPI)**

**For Inhalers Requiring Loading**

- First load the drug by:
  - Turning the device to the next dose of drug, or
  - Inserting the capsule into the device, or
  - Inserting the disk or compartment into the device

**After Loading the Drug and for Inhalers That Do Not Require Drug Loading**

- Read your doctor’s instructions for how fast you should breathe for your particular inhaler.
- Place your lips over the mouthpiece, and breathe in forcefully (there is no propellant in the inhaler; only your breath pulls the drug in).
- Remove the inhaler from your mouth as soon as you have breathed in.
- Never exhale (breathe out) into your inhaler. Your breath will moisten the powder, causing it to clump and not be delivered accurately.
- Never wash or place the inhaler in water.
- Never shake your inhaler.
- Keep your inhaler in a dry place at room temperature.
- If the inhaler is preloaded, discard the inhaler after it is empty.
- Because the drug is a dry powder and there is no propellant, you may not feel, smell, or taste it as you inhale.

Teach the patient to always carry the rescue drug inhaler with him or her and to ensure that enough drug remains in the inhaler to provide a quick dose when needed. Dry powder inhalers indicate the amount of remaining drug; however, aerosol (MDI) inhalers do not. Demonstrate how to check aerosol inhaler drug levels by placing the inhaler in water (Figure 32-5). Full inhalers sink to the bottom. An empty inhaler floats on its side.
Long-acting beta₂ agonists (LABAs) are also delivered by inhaler directly to the site of action—the bronchioles. Proper use of the long-acting agonists can decrease the need to rescue as often with short-acting agonists. Unlike short-acting agonists, long-acting drugs need time to build up an effect but the effects are longer lasting. Thus these drugs are useful in preventing an asthma attack but have no value during an acute attack. Therefore teach patients not to use LABAs to rescue them during an attack or when wheezing is getting worse but, instead, to use a SABA. Relying on LABAs during an attack can lead to worsening of symptoms and death. Examples of LABAs include formoterol (Foradil) and salmeterol (Serevent). Teach the patient to use these drugs daily as prescribed, even when no symptoms are present.

Cholinergic antagonists, also called anticholinergic drugs, are similar to atropine and block the parasympathetic nervous system. This blockade allows the sympathetic nervous system to dominate, resulting in increased bronchodilation and decreased pulmonary secretions. The most common drug in this class is ipratropium (Atrovent), which is used as an inhalant. Most cholinergic antagonists are short acting and must be used several times a day, although long-acting agents such as tiotropium (Spiriva) are available for use once a day. These drugs are not as effective as beta₂ agonists and are recommended as first-line asthma therapy only for those patients who cannot tolerate the side effects of beta, agonists.

Methylxanthines are used when other types of management are ineffective. The classic drug in this class is theophylline (Theo-Dur). Other drugs include aminophylline (Truphylline), oxtriphylline (Choledyl (Théo-Dur). Other drugs include aminophylline (Truphylline), oxtriphylline (Choledyl , and dyphylline (Dilor, Lu-fyllin). These drugs are given systemically, have narrow therapeutic ranges, and have many side effects. Blood levels of these drugs need to be monitored closely because the drug level that causes dangerous side effects is not much higher than the level needed to dilate the bronchioles. Teach the patient who takes these drugs daily to keep all appointments for monitoring blood levels of the drug and not to self-increase the dose. The most dangerous side effects result from excessive cardiac and central nervous system stimulation and include dysrhythmias, hypertensions, and seizure activity.

Anti-inflammatory agents. Anti-inflammatory agents decrease the inflammatory responses in the airways. Some are given systemically and have more side effects. Others are used as inhalants and have few systemic side effects. Corticosteroids decrease inflammatory and immune responses in many ways, including by preventing the synthesis of mediators. Inhaled corticosteroids (ICSs) can be helpful in preventing the manifestations of asthma. Newer high-potency steroid inhalers, such as fluticasone (Flovent), budesonide (Pulmicort), and fluticasone (Asmanex), may be used once per day for maintenance. Systemic corticosteroids, because of severe side effects, are avoided for mild to moderate intermittent asthma and are used on a short-term basis for moderate asthma. For some patients with severe asthma, daily oral corticosteroids may be needed. Both inhaled corticosteroids and those taken orally are preventive. They are not effective in reversing symptoms during an asthma attack and should not be used as rescue drugs. Teach patients the difference between ICSs and rescue drug inhalers. Preventive or controller drugs must be used on a scheduled basis, even when asthma symptoms are not present.

Nonsteroidal anti-inflammatory drugs (NSAIDs), both those that are inhaled and those that are taken orally, are useful as preventive asthma therapy and should be taken on a scheduled basis. They include a variety of agents that have different mechanisms of action to reduce airway inflammation. Nedocromil (Tilade) inhibits the release of inflammatory mediators from respiratory cells and white blood cells. Mast cell stabilizers, such as cromolyn sodium (Intal), prevent mast cell membranes from opening when an allergen binds to IgE. Thus these drugs help prevent atopic asthma attacks but are not useful during an acute episode. The inhaled NSAIDs are not effective in reversing symptoms during an asthma attack and should not be used as rescue drugs.

Leukotriene antagonists are oral drugs that work in several ways to prevent an asthma episode. Montelukast (Singulair) and zafirlukast (Accolate) block the leukotriene receptor. Zileuton (Zyflo) prevents leukotriene synthesis. These drugs are not effective in reversing symptoms during an asthma attack and should not be used as rescue drugs.

Immunomodulators are monoclonal antibodies that prevent allergens from binding to receptor sites on mast cells and basophils. This action prevents allergens from triggering the release of mediators from mast cells and basophils. Thus these drugs help prevent atopic asthma attacks but are not useful during an acute episode. Omalizumab (Xolair) is currently the

Fig. 32-4 - Patient using a dry powder inhaler (DPI).

Fig. 32-5 - Checking the drug level in an aerosol inhaler.
only drug in this class. It is injected subcutaneously every 2 to 3 weeks. Because there is a relatively high risk of anaphylaxis from this drug, it should be administered only in a setting capable of handling this type of reaction.

**Exercise/Activity**

Regular exercise, including aerobic exercise, is a recommended part of asthma therapy. Aerobic exercise assists in maintaining cardiac health, enhancing skeletal muscle strength, and promoting ventilation and perfusion. Patients with asthma should examine the conditions that trigger an attack and adjust the exercise routine as needed. Some may need to premedicate with inhaled beta agonists (SABAs) before beginning activity. For others, adjusting the environment may be needed. For example, outdoor ice-skating in cold, dry air can trigger an attack; indoor ice-skating may be less of a problem. Sports that involve more “rest” action, such as baseball, are less likely to trigger symptoms than “nonrest” action sports, such as basketball.

**Oxygen Therapy**

Supplemental oxygen is often used during an acute asthma attack. Oxygen is delivered by mask, nasal cannula, or endotracheal tube. High flow rates or concentrations may be needed when bronchospasms are severe and limit flow of oxygen through the bronchiole tubes. Heliox, a mixture of helium and oxygen (often 50% helium and 50% oxygen), can help improve oxygen delivery to the alveoli. This gas mixture is lower in density than oxygen alone or oxygen with atmospheric air (which contains nitrogen) and flows even when airway resistance is high (Pruitt, 2007). Ensure that no open flames (e.g., cigarette smoking, fireplaces, burning candles) or other combustion hazards are in rooms where oxygen is in use.

**Status Asthmaticus**

Status asthmaticus is a severe, life-threatening acute episode of airway obstruction that intensifies once it begins and often does not respond to common therapy. The patient arrives in the emergency department with extremely labored breathing and wheezing. Use of accessory muscles for breathing and distention of neck veins are observed. If the condition is not reversed, the patient may develop pneumothorax and cardiac or respiratory arrest. The physician immediately prescribes IV fluids, potent systemic bronchodilators, steroids (to decrease inflammation), epinephrine, and oxygen in an attempt to reverse the acute condition. Prepare for emergency intubation. When wheezing decreases, management is similar to that for any patient with asthma.

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**DECISION-MAKING CHALLENGE**

**Critical Rescue**

The patient is a 42-year-old woman who has had asthma since she was a child. She has always treated her asthma only when symptoms appeared but now is on an asthma management plan for both prevention and treatment. She comes to the emergency department with audible wheezes on inhalation and exhalation. Her PEF and FEV1 are 40% below her personal best. Her respiratory rate is 34 breaths/min, pulse 122 bpm. She has suprasternal and intercostal retractions. Her asthma management drugs include:

- salmeterol (Serevent) 2 puffs every 12 hr
- terbutaline (Brethaire) 2 puffs PRN
- fluticasone (Flovent) 2 puffs daily
- cromolyn sodium (Intal) 1-2 puffs 4 times per day

Her partner tells you that she has not used any of her inhalers for the past week because the drugs are expensive and she has felt well.

1. Should you start oxygen on this patient? Why or why not?
2. What additional assessment data should you obtain?
3. Which of the patient’s current drugs should be administered immediately? Why?
4. What is your interpretation of this patient’s immediate condition based on PEF?
5. What teaching priorities are needed for this patient?

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**CHRONIC OBSTRUCTIVE PULMONARY DISEASE**

**Pathophysiology**

Most patients with emphysema have chronic bronchitis at the same time, but each condition has its own pathophysiologic process (Figure 32-6).

**Emphysema**

The two major changes that occur with pulmonary emphysema are loss of lung elasticity and hyperinflation of the lung (see Figure 32-1). These changes result in dyspnea and the need for an increased respiratory rate.

In the healthy lung, protein degrading enzymes called proteases are present to destroy and eliminate protein-based particulate matter and organisms inhaled during breathing. If these proteases are present in higher-than-normal levels, they damage the alveoli and the small airways by breaking down elastin. High protease levels cause the alveolar sacs to lose their elasticity and the small airways to collapse or narrow. Some alveoli are destroyed, and others become large and flabby, with decreased area for effective gas exchange.

An increased amount of air becomes trapped in the lungs. Causes of air trapping are loss of elastic recoil in the alveolar walls, overstretching and enlargement of the alveoli into air-filled spaces called bullae, and collapse of small airways (bronchioles). These changes greatly increase the work of breathing. The hyperinflated lung flattens the diaphragm (Figure 32-7), weakening the effect of this muscle. As a result, the patient...
with emphysema needs to use additional muscles (accessory muscles) in the neck, chest wall, and abdomen to inhale and exhale. This increased effort increases the need for oxygen, making the patient work harder and have an “air hunger” sensation. Often, inhalation starts before exhalation is completed, resulting in an uncoordinated pattern of breathing.

Gas exchange is affected by the increased work of breathing and the loss of alveolar tissue. Although some alveoli enlarge, the curves of alveolar walls decrease and less surface area is available for gas exchange. Often, inhalation starts before exhalation is completed, resulting in an uncoordinated pattern of breathing.

Gas exchange is affected by the increased work of breathing and the loss of alveolar tissue. Although some alveoli enlarge, the curves of alveolar walls decrease and less surface area is available for gas exchange. Often, inhalation starts before exhalation is completed, resulting in an uncoordinated pattern of breathing.

Emphysema is classified as panlobular, centrilobular, or paraseptal depending on the pattern of destruction and dilation of the gas-exchanging units (acini) (see Figure 32-1). Each type can occur alone or in combination in the same lung. Most are associated with smoking or chronic exposure to other inhalation irritants.

Chronic Bronchitis
Bronchitis is an inflammation of the bronchi and bronchioles caused by chronic exposure to irritants, especially tobacco smoke. The irritant triggers inflammation, with vasodilation, congestion, mucosal edema, and bronchospasm. Unlike emphysema, bronchitis affects only the airways rather than the alveoli.

Chronic inflammation causes an increase in the number and size of mucous glands, which produce large amounts of thick mucus. The bronchial walls thicken (often to twice the normal thickness) and impair airflow. This thickening, along with excessive mucus, blocks some of the smaller airways and narrows larger ones. Small airways are affected before large airways become involved.

Chronic bronchitis hinders airflow and gas exchange because of mucous plugs and infection narrowing the airways. As a result, the PaO₂ decreases (hypoxemia) and the arterial blood carbon dioxide (PaCO₂) level increases (respiratory acidosis).

Etiology and Genetic Risk
Cigarette smoking is the most important risk factor for COPD. The patient with an 8–pack-year history usually has obstructive lung changes but no manifestations of disease. The patient with a 20–pack-year history or longer often has early-stage COPD found as changes in pulmonary function tests (PFTs).

The harmful effects of tobacco result in part because inhaled smoke triggers the release of excessive amounts of the proteases from cells in the lungs. These enzymes break down elastin, the major component of alveoli. By impairing the action of cilia, smoking also inhibits the cilia from clearing the bronchi of mucus, cellular debris, and fluid.

In addition to the increased risk for COPD from active smoking, passive smoking (or secondhand smoke) contributes to upper and lower respiratory problems. The risk is greater when exposure occurs in small, confined spaces.

**CULTURAL AWARENESS**

The prevalence of smoking remains higher among African Americans, blue-collar workers, and less educated people than in the overall population of the United States. Smoking prevalence is highest among Northern Plains American Indians and Alaskan Natives. The overall prevalence of smoking for both men and women has decreased over the past two decades, but the decrease for women has been less than it has for men (American Cancer Society, 2008). Development of culturally appropriate smoking cessation programs as well as research examining barriers to cessation in these populations may help reduce this disparity.
**Alpha,-antitrypsin deficiency** is a less common but important risk factor for COPD. A special enzyme, alpha,-antitrypsin (AAT), is made by the liver and is normally present in the lungs. One purpose of AAT is to regulate the proteases that are present to break down inhaled pollutants and organisms. AAT, a protease inhibitor, prevents the proteases from working on lung structures.

The production of normal amounts of AAT depends on the inheritance of a pair of normal gene alleles for this protein. The AAT gene is recessive. Thus if one of the pair of alleles is faulty and the other allele is normal, the person makes enough AAT to prevent COPD unless there is significant exposure to cigarette smoke and other precipitating factors. This person, however, is a carrier for AAT deficiency. If both alleles are faulty, COPD develops at a fairly young age even when the person is not exposed to cigarette smoke or other irritants.

About 100,000 Americans have severe AAT deficiency, and many more have mild to moderate deficiencies (Nussbaum et al., 2007). Although an AAT deficiency causes problems in other organs, such as the skin and liver, lung diseases are the most common problem caused by the deficiency.

### GENETIC CONSIDERATIONS

The gene for AAT has many known mutations, some of which increase the risk for emphysema. Variation of mutations (polymorphisms) results in different levels of AAT deficiency. This variation is one reason why the disease is more severe for some people than for others. The most serious mutation for an increased risk for emphysema is the Z mutation, although others also increase the risk but to a lesser degree. Table 32-1 shows the most common AAT mutations increasing the risk for emphysema.

Air pollution alone plays a relatively small role in the patient with emphysema and chronic bronchitis. The effect of air pollution is additive to tobacco exposure.

### Incidence/Prevalence

The prevalence of chronic bronchitis and emphysema in the United States has been estimated at about 13.5 million (for chronic bronchitis) and 2 million (for emphysema). Chronic obstructive pulmonary disease/chronic airflow limitation (COPD/CAL) is the fourth leading cause of morbidity and mortality in the United States (Global Initiative for Chronic Obstructive Lung Disease [GOLD], 2007).

### Complications

COPD affects the oxygenation and tissue perfusion to all tissues. Complications of the disorder can result in organ anoxia and tissue death. Major problems occur, such as hypoxemia, acidosis, respiratory infection, cardiac failure, and dysrhythmias.

**Hypoxemia and acidosis** occur because the patient with COPD is less able to exchange gas, oxygenation decreases and carbon dioxide levels increase. These problems reduce general cellular function.

**Respiratory infection** risk increases because of the increased mucus and poor oxygenation. The organisms most often causing bacterial infections include Streptococcus pneumoniae, Haemophilus influenzae, and Moraxella catarrhalis. Acute respiratory infections make COPD manifestations worse by increasing inflammation and mucus production and inducing more bronchospasm. Airflow becomes even more limited, the work of breathing increases, and dyspnea results.

**Cardiac failure,** especially cor pulmonale (right-sided heart failure caused by pulmonary disease), occurs with bronchitis or emphysema. Air trapping, airway collapse, and stiff alveolar walls increase the lung tissue pressure, making blood flow through lung vessels more difficult. The increased pressure makes the workload heavy on the right side of the heart, which pumps blood into the lungs. As the disease progresses, the amount of oxygen in the blood decreases, causing major blood vessels in the lung to constrict. To pump blood through these narrowed vessels, the right side of the heart must generate high pressures. In response to this heavy workload, the right chambers of the heart enlarge and thicken, causing right-sided heart failure with backup of blood into the general venous system. Chart 32-8 lists key features of cor pulmonale.

**Cardiac dysrhythmias** are common in patients with COPD. They may be a result of hypoxemia (from decreased oxygen to the heart muscle), other cardiac disease, drug effects, or acidosis.

### Table 32-1

**Characteristics Associated with the Most Common Alpha-1-Antitrypsin Gene Mutations**

<table>
<thead>
<tr>
<th>Mutation Genotype</th>
<th>Level of Serum Alpha-1 Antitrypsin (% of normal)</th>
<th>Disease Severity</th>
</tr>
</thead>
<tbody>
<tr>
<td>M/S</td>
<td>80%</td>
<td>No detectable disease</td>
</tr>
<tr>
<td>S/S</td>
<td>50%-60%</td>
<td>Minimal to no disease expression</td>
</tr>
<tr>
<td>M/Z</td>
<td>50%-55%</td>
<td>Minimal to no disease expression</td>
</tr>
<tr>
<td>S/Z</td>
<td>30%-35%</td>
<td>Pulmonary disease, early age</td>
</tr>
<tr>
<td>Z/Z</td>
<td>10%-15%</td>
<td>Severe COPD, extra-pulmonary involvement</td>
</tr>
</tbody>
</table>


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**Chart 32-8**  **KEY FEATURES**

**Cor Pulmonale**

- Hypoxia and hypoxemia
- Increasing dyspnea
- Fatigue
- Enlarged and tender liver
- Warm, cyanotic hands and feet, with bounding pulses
- Cyanotic lips
- Distended neck veins
- Right ventricular enlargement (hypertrophy)
- Visible pulsations below the sternum
- GI disturbances, such as nausea or anorexia
- Dependent edema
- Metabolic and respiratory acidosis
- Pulmonary hypertension
Health Promotion and Maintenance
Health experts agree that the incidence and severity of COPD would be drastically reduced by smoking cessation (Crawford & Harris, 2008). COPD is rare among people who have never smoked cigarettes. Disease progression can be slowed by smoking cessation. Encourage all people who smoke to quit smoking. Chart 32-3 provides tips to teach people about smoking cessation.

Other measures to reduce the incidence of COPD are to avoid inhalation irritants in all environments. Teaching all people to use masks when working in areas with high levels of particulate matter can reduce individual exposure. Proper venting of workplaces and recreation areas that have airborne or particulate matter also reduces exposure.

Patient-Centered Collaborative Care
The Concept Map on p. *** addresses assessment and nursing care issues related to COPD.

Assessment

History

Ask about risk factors such as age, gender, occupational history, and ethnic-cultural background when taking a history from a patient who may have chronic obstructive pulmonary disease (COPD). COPD is seen more often in older men. Some types of emphysema occur in families, especially those with alpha1-antitrypsin (AAT) deficiency.

Obtain a thorough smoking history, because tobacco use is a major risk factor. Ask about the length of time the patient has smoked and the number of packs smoked daily. Use these data to determine the pack-year smoking history. If the patient smokes, use this opportunity as a teachable moment to discuss smoking cessation strategies (see Chart 32-3).

Ask the patient to describe his or her breathing problems. Assess whether the patient has any difficulty breathing while talking. Can he or she speak in complete sentences, or is it necessary to take a breath between every one or two words? Ask about the presence, duration, or worsening of wheezing, coughing, and shortness of breath. Determine what activities trigger these problems. Assess the patient’s cough pattern. If the cough is productive, ask whether sputum is clear or colored and how much is produced each day. Ask the patient to recall the time of day when the sputum production is greatest. Smokers often have a productive cough when they get up in the morning; nonsmokers generally do not. Ask whether sputum production has increased or changed during the past year.

Check the relationship between activity tolerance and dyspnea by asking the patient to compare his or her activity level and shortness of breath now with those of a month ago and a year ago. Likewise, ask about any difficulty with eating and sleeping. Many patients sleep in a semi-sitting position because breathlessness is worse when lying down (orthopnea). Ask about usual daily activities and any difficulty with sleeping, bathing, dressing, or sexual activity. Document this initial assessment to serve as a starting point for determining the intervention plan and its effectiveness.

Weigh the patient, and compare this weight with previous weights. Unplanned weight loss occurs with an increase in COPD severity. COPD increases metabolic needs as a result of the increased work of breathing. Dyspnea and mucus production often result in poor food intake and inadequate nutrition. Ask the patient to recall a typical day’s meals and fluid intake.

When heart failure is present with COPD, general edema with weight gain may occur.

Physical Assessment/Clinical Manifestations

General appearance can provide clues about the patient’s respiratory status and energy level. Observe his or her weight in proportion to height, posture, mobility, muscle mass, and overall hygiene. The patient with increasingly severe COPD is thin, with loss of muscle mass in the extremities, although the neck muscles may be enlarged. He or she tends to be slow moving and slightly stooped. Usually the person sits with a forward-bending posture, sometimes with the arms held forward (Figure 32-8). When dyspnea becomes severe, activity intolerance may be so great that bathing and general grooming are neglected.
Concept Map: Respiratory Acidosis (COPD Related)

**Risk Factors**
- Age (+)
- Chronic illness(es) (+)

**Martin Sternberg**
63-year-old farmer with chronic bronchitis reports fatigue, shortness of breath, difficulty thinking, and anorexia
- ABG pH = 7.30, PaCO₂ = 68 mm Hg, HCO₃⁻ = 29 mEq/L, PaO₂ = 65 mm Hg
- VS: P 62 and irregular, R 35, T 99° F, BP 106/72
- Physical exam: shallow respiration, muscle tone + 1, extremities cool and pale
- Laboratory: RBC 11.7, WBC 12.5, serum potassium 5.8
- Radiology: Chest x-ray shows consolidation in lower lobes

**Central Nervous System**
- Difficulty thinking (+)
- Neuromuscular
  - Diminished muscle strength (+)

**Cardiovascular**
- Decreased heart rate (+)
- Hypotension (+)

**Skin**
- Pale and cool (+)

**Respiratory**
- Tachypnea (+)
- Shortness of breath (+)
- Shallow respiration (+)

**Gastrointestinal**
- Anorexia (+)

**Diagnostic Tests**
- Acidosis (+)
- Hypercapnia (+)
- Hyperkalemia (+)
- Chest x-ray shows consolidation (+)
- Sputum production (?)

**Ineffective Breathing Pattern**
- Patient is able to maintain a PaO₂ above 65 and a respiratory rate below 25
- Patient is able to maintain a heart rate above 70 per minute
- Patient is free from falls
- Patient is able to carry out desired ADLs

**Desired Outcomes**
- Patient is able to maintain a PaO₂ above 65 and a respiratory rate below 25
- Patient is able to maintain a heart rate above 70 per minute
- Patient is free from falls
- Patient is able to carry out desired ADLs
Respiratory changes occur as a result of obstruction, changes in chest size, and fatigue. Inspect the chest to assess the breathing rate and pattern. The patient with respiratory muscle fatigue breathes with rapid, shallow respirations and may have paradoxical respirations or use accessory muscles in the abdomen or neck. The respiratory rate could be as high as 40 to 50 breaths/min. The breathing patterns often seen with respiratory muscle fatigue use abdominal muscles and the intercostal muscles more than the diaphragm. Respiratory movement is jerky and appears uncoordinated.

Check the patient’s chest for abnormal retractions and for symmetric chest expansion. The patient with emphysema has limited diaphragmatic movement (excursion) because the diaphragm is flattened and below its usual resting state. Chest vibration (fremitus) is often decreased and the chest sounds hyperresonant on percussion because of trapped air.

Auscultate the chest to assess the depth of inspiration and any abnormal breath sounds. Wheezes and other abnormal sounds occur with emphysema and chronic bronchitis, often on inspiration and expiration, although crackles are usually not present. Note the pitch and location of the sound and the point in the respiratory cycle at which the sound is heard. A silent chest may indicate obstruction or pneumothorax.

Assess the degree of dyspnea using an assessment tool called a Visual Analog Dyspnea Scale (VADS). The VADS is a straight line with verbal anchors at the beginning and end of a 0- to 100-mm line. Ask the patient to place a mark on the line to indicate his or her perceived breathing difficulty (Figure 32-9). Document the response, and use this scale to assess dyspnea, determine the therapy effectiveness, and pace the patient’s activities.

Examine the patient’s chest for the presence of a “barrel chest” (see Figure 32-2). With a barrel chest, the ratio between the anteroposterior (AP) diameter of the chest and its lateral diameter is 2:2 rather than the normal ratio of 1:2. This shape change results from lung overinflation and diaphragm flattening.

The patient with chronic bronchitis often has a cyanotic, or blue-tinged, dusky appearance and has excessive sputum production. Assess the patient for cyanosis, delayed capillary refill, and clubbing of the fingers (Figure 32-10), which indicate chronically decreased arterial oxygen levels.

Cardiac changes occur as a result of the anatomic changes associated with COPD. Assess the patient’s heart rate and rhythm. Check for swelling of the feet and ankles (dependent edema) or other manifestations of right-sided heart failure.

Examine nail beds and oral mucous membranes. The patient with later-stage emphysema may have pallor or frank cyanosis.

Psychosocial Assessment
COPD affects all aspects of a person’s life. Socialization may be reduced when friends and family avoid the patient with COPD because of annoying coughs, excessive sputum, or dyspnea. The patient may choose to be isolated because dyspnea causes fatigue or because of embarrassment from coughing and excessive sputum production. In addition, because of the association with cigarette smoking and disease development, the patient may feel a social stigma.

Ask the patient about interests and hobbies to assess whether socialization has decreased or whether hobbies cause exposure to inhalation irritants. Ask about home conditions for exposure to smoke or crowded living conditions that promote transmission of respiratory infections.

Economic status may be affected by the disease through changes in income and health insurance coverage. If the patient is the head of the household, severe COPD may require role changes that have a negative impact on self-image. Drugs, especially the metered dose inhalers (MDIs) and dry powder inhalers ( DPI), are expensive, and many patients with limited incomes may use them only during exacerbations and not as prescribed on a scheduled basis.

Anxiety and fear related to dyspnea and feelings of breathlessness may reduce the patient’s ability to participate in a full life. Work, family, social, and sexual roles can be affected. Encourage the patient and family to express their feelings about the limitations on lifestyle and disease progression. Assess their awareness and use of support groups and services.

Laboratory Assessment
Arterial blood gas ( ABG) values identify abnormal oxygenation, ventilation, and acid-base status. Compare serial or repeated ABG values to assess changes in the patient’s respiratory status. Once baseline ABG values are obtained, pulse oximetry can gauge the response to treatment. As COPD worsens, the amount of oxygen in the blood decreases ( hypoxemia) and the amount of carbon dioxide in the blood increases ( hypercarbia). Chronic respiratory acidosis (increased arterial carbon dioxide [PaCO₂]) then results; metabolic alkalosis (increased arterial bicarbonate) occurs as
compensation by the kidney retention of bicarbonate. This change is seen on ABGs as an elevation of HCO$_3^-$.

Not all patients with COPD are carbon dioxide retainers, even when hypoxemia is present. Carbon dioxide diffuses more easily across lung membranes than does oxygen. Hypercarbia is a problem in advanced emphysema (because the alveoli are affected) rather than in bronchitis (wherein the airways are affected). For more detailed information about acidosis, see Chapter 14.

Sputum samples are obtained for culture from hospitalized patients with an acute respiratory infection. In the community, sputum cultures are rarely obtained. The infection is treated on the basis of manifestations and the common bacterial organisms. A white blood cell count helps confirm the presence of infection.

Other blood tests include hemoglobin and hematocrit to determine polycythemia (a compensatory increase in red blood cells in the chronically hypoxic patient). Serum electrolyte levels are examined because hypophosphatemia, hyperkalemia, hypocalcemia, and hypomagnesemia reduce muscle strength. In patients with a family history of COPD, serum AAT levels may be drawn.

**Imaging Assessment**

Chest x-rays are obtained to rule out other chest diseases and to check the progress of patients with respiratory infections or chronic disease. With advanced emphysema, chest x-rays show hyperinflation and a flattened diaphragm. They may not be helpful in the diagnosis of early or moderate disease.

**Other Diagnostic Assessment**

COPD is classified from mild to severe on the basis of manifestations and pulmonary function test (PFT) changes (Table 32-2; see also Table 29-6 in Chapter 29). Airflow rates and lung volume measurements help distinguish airway disease (obstructive disease) from interstitial lung disease (restrictive disease). PFTs determine lung volumes, flow volume curves, and diffusion capacity. Each test is performed before and after the patient inhales a bronchodilator agent. The person being tested for COPD usually has some manifestations and may be anxious about the potential diagnosis. Encourage the patient to express his or her feelings about testing and the potential impact of the results. Explain the preparations for the procedures (if any), whether pain or discomfort will be involved, and any needed follow-up care.

The lung volumes measured for COPD are vital capacity (VC), residual volume (RV), and total lung capacity (TLC) (see Chart 29-6 in Chapter 29). RV is most profoundly affected, although all volumes and capacities change to some degree in COPD. The RV increase reflects the trapped, stale air remaining in the lungs.

Flow volume curves measure the patient’s ability to move air into and out from the lungs. The rate of airflow out of the lungs during a rapid, forceful, and complete exhalation from TLC to RV (forced expiratory volume [FEV]) indirectly measures the flow resistance of the lung. A diagnosis of COPD is based mostly on the FEV$_1$ (the FEV in the first second of exhalation). FEV$_1$ can also be expressed as a percentage of the forced vital capacity (FVC). As the disease progresses, the ratio of FEV$_1$ to FVC becomes smaller.

The diffusion test measures how well a test gas (carbon monoxide) diffuses across the alveolar-capillary membrane and combines with the hemoglobin of red blood cells. In emphysema, alveolar wall destruction causes a large decrease in surface area for diffusion of gas into the blood, leading to a decreased diffusion capacity. In bronchitis, even though lung volumes are increased, the diffusion capacity is usually normal.

The patient with COPD has decreased oxygen saturation, often much lower than 90%. Changes in pulse oximetry results below the patient’s usual saturation require medical attention.

Peak expiratory flowmeters are used to monitor the effectiveness of the prescribed drugs to relieve obstruction. Peak flow rates increase as obstruction resolves. Teach the patient to self-monitor the peak expiratory flow rates at home and adjust drugs as needed.

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**TABLE 32-2** Classification of COPD Severity

<table>
<thead>
<tr>
<th>Stage</th>
<th>Manifestations</th>
<th>Pulmonary Function Test Results</th>
</tr>
</thead>
<tbody>
<tr>
<td>0 (At risk)</td>
<td>≤Chronic cough ≤Chronic sputum production Exposure to environmental risk factors</td>
<td>Normal</td>
</tr>
<tr>
<td>I (Mild)</td>
<td>+Chronic cough ≤Sputum production</td>
<td>FEV$_1$/FVC &lt;70% FEV$_1$ &lt;80% of predicted</td>
</tr>
<tr>
<td>II (Moderate)</td>
<td>≤Dyspnea ≤Chronic cough ≤Sputum production</td>
<td>FEV$_1$/FVC &lt;70% FEV$_1$ &lt;80% but at least ≤50% of predicted</td>
</tr>
<tr>
<td>III (Severe)</td>
<td>+Dyspnea +Chronic cough +Sputum production</td>
<td>FEV$_1$/FVC &lt;70% FEV$_1$ &lt;50% but at least ≤30% of predicted</td>
</tr>
<tr>
<td>IV (Very severe)</td>
<td>++Dyspnea ++Chronic cough ++Sputum production</td>
<td>FEV$_1$/FVC &lt;70% FEV$_1$ &lt;30% of predicted OR FEV$_1$ &lt;50% of predicted with respiratory failure</td>
</tr>
</tbody>
</table>

The patient has all of the following ABG results. Which one alerts the nurse to the fact that the patient has a long-term respiratory problem with CO2 retention?

A. pH = 7.12
B. HCO3− = 31 mEq/L
C. PaCO2 = 68 mm Hg
D. PaO2 = 78 mm Hg

For the correct answer, go to http://evolve.elsevier.com/Iggy/.

Analysis

Common Nursing Diagnoses and Collaborative Problems

These are priority nursing diagnoses for patients with chronic obstructive pulmonary disease (COPD):

1. Impaired Gas Exchange related to alveolar-capillary membrane changes, reduced airway size, ventilatory muscle fatigue, and excessive mucus production
2. Ineffective Breathing Pattern related to airway obstruction, diaphragm flattening, fatigue, and decreased energy
3. Ineffective Airway Clearance related to excessive secretions, fatigue, decreased energy, and ineffective cough
4. Imbalanced Nutrition: Less Than Body Requirements related to dyspnea, excessive secretions, anorexia, and fatigue
5. Anxiety related to dyspnea, a change in health status, and situational crisis
6. Activity Intolerance related to fatigue, dyspnea, and an imbalance between oxygen supply and demand

A primary collaborative problem for patients with COPD is Potential for Pneumonia or Other Respiratory Infections.

Additional Nursing Diagnoses and Collaborative Problems

In addition to the common nursing diagnoses and collaborative problems, patients with COPD may have one or more of these:

- Fatigue related to a change in metabolic energy or hypoxemia
- Deficient Knowledge (disease process, prescribed treatments, activity limitations) related to unfamiliarity with information resources
- Sexual Dysfunction related to extreme fatigue
- Impaired Spontaneous Ventilation related to ventilatory muscle fatigue
- Sleep Deprivation related to dyspnea or an unfamiliar environment (hospitalization)
- Anxiety related to dyspnea, a change in health status, and situational crisis
- Ineffective Coping related to high degree of threat, inadequate level of perception of control, changes in lifestyle, situational crisis, or knowledge deficit

Other collaborative problems for patients with COPD include Potential for Respiratory Failure and Potential for Right-Sided Heart Failure.

Planning and Implementation

Impaired Gas Exchange

**NCLEX EXAMINATION CHALLENGE**

Planning: expected outcomes. The patient with COPD is expected to attain and maintain gas exchange at a level within his or her chronic baseline values. Indicators include:

- Maintenance of SpO2 of at least 88%
- Absence of cyanosis
- Maintenance of cognitive orientation

Interventions. Most patients with COPD use nonsurgical management to improve or maintain gas exchange. Surgical management requires that the patient meet strict criteria.

Nonsurgical management. The mainstays of nursing management for patients with COPD include airway maintenance, monitoring, drug therapy, cough enhancement, oxygen therapy, and pulmonary rehabilitation. Nursing priorities are teaching the patient to be a partner in COPD management by participating in therapies to improve ventilation and by adhering to prescribed drug therapy.

Airway maintenance is the most important intervention to improve gas exchange. Keep the patient’s head, neck, and chest in alignment. Assist him or her to liquefy secretions and clear the airway of secretions.

**DECISION-MAKING CHALLENGE**

Delegation/Supervision

The patient is a 72-year-old African-American man who is a resident of the nursing home because of severe dyspnea related to long-standing COPD. He is on continuous oxygen by nasal cannula at 2 L/min. The UAP assigned to him reports that he has gained 7 lbs since the last time he was weighed (1 week ago) and that he seemed “grouchy.” Results of his last set of vital signs (taken 12 hours ago) are close to his usual results.

1. Who should take his next vital signs—the UAP, the LPN/LVN, or the RN? Provide a rationale for your choice.
2. What should you ask the UAP about this patient?

For suggested answer guidelines, go to http://evolve.elsevier.com/Iggy/.

O2 Therapy for COPD

Oxygen therapy for COPD is prescribed for relief of hypoxemia (decreased blood oxygen levels) and hypoxia (decreased tissue oxygenation). The need for oxygen therapy and its effectiveness can be determined by arterial blood gas values and oxygen saturation by pulse oximetry. The patient with COPD may need an oxygen flow of 2 to 4 L/min via nasal cannula or up to 40% via Venturi mask. The patient who is hypoxic and also has chronic hypercarbia requires lower levels of oxygen delivery, usually 1 to 2 L/min via nasal cannula. A low arterial oxygen level is this patient’s primary drive for breathing. Do not increase the oxygen flow rate in patients with hypercarbia because this may lower their respiratory rate or even make them stop breathing spontaneously. Ensure that there are no open flames or other combustion hazards in rooms in which oxygen is in use. More information on oxygen therapy is found in Chapter 30.

Drug therapy for COPD involves the same inhaled and systemic drugs as for asthma. These drugs include beta-adrenergic
agents, cholinergic antagonists, methylxanthines, corticosteroids, and NSAIDS (see Chart 32-5). The focus is on long-term control therapy with longer duration drugs, such as arformoterol (Brovana) and tiotropium (Spiriva). The patient with COPD is more likely to be taking systemic agents (in addition to inhaled drugs) than is the patient with asthma. An additional drug class for COPD is the mucolytics, which thin secretions, making them easier to expectorate.

Mucolytic agents are prescribed for the patient with thick, tenacious (sticky) mucous secretions. Nebulizer treatments with normal saline or with a mucolytic agent such as acetylcysteine (Mucosil, Mucomyst) or dornase alfa (Pulmozyme) and normal saline help thin secretions and facilitate expectoration. Guaiifenesin (Organidin, Naldecon Senior EX) is a systemic mucolytic that is taken orally.

Stepped therapy, which adds drugs as COPD progresses, is recommended for patients with chronic bronchitis or emphysema, although the patient’s responses to drug therapy is the best indicator of when drugs or their dosages need changing. The expected outcomes are for the patient to have more awareness of the disease and to participate in symptom management. Newly diagnosed patients and their family members have concerns about being able to use inhalers correctly (Carlson et al., 2006). Teach patients and family members the correct techniques for using inhalers and to care for them properly.

Pulmonary rehabilitation can be used to improve function and endurance in patients with COPD. Patients often respond to the dyspnea of COPD by limiting their activity, even basic ADLs. Over time, the muscles of ventilation and other large muscle groups weaken and are less efficient in the use of oxygen. The result is increased dyspnea with lower activity levels.

Pulmonary rehabilitation involves education and exercise training to prevent general and pulmonary muscle deconditioning. Formal programs are usually at least 6 weeks long; however, many patients can benefit from ongoing exercise. Each patient’s exercise program is personalized to reflect his or her current limitations and outcome goals. All exercises should be performed at least 2 or 3 times each week (Bauldoff & Diaz, 2006). The simplest plan involves having the patient walk (indoors or outdoors) daily at a self-paced rate until symptoms limit further walking, followed by a rest period, and then continue walking until 20 minutes of actual walking has been accomplished. As the time during rest periods decreases, the patient can add 5 more minutes of walking time. The benefits of this type of exercise have been shown even for people with severe COPD (GOLD, 2007). Teach patients whose symptoms are severe to modify the exercise by using a walker with wheels or, if needed, to use oxygen therapy during the exercise period.

Exercise conditioning of the large muscle groups or retraining of the ventilatory muscles also may be part of a pulmonary rehabilitation program. Two techniques are isocapnic hyperventilation and resistive breathing. Isocapnic hyperventilation, in which the patient hyperventilates into a
machine that controls the levels of oxygen and carbon dioxide, increases endurance. In resistive breathing, the patient breathes against a set resistance. Resistive breathing increases ventilatory muscle strength and endurance.

**Surgical management.** Lung transplantation is performed for select patients with end-stage COPD. (See the lung transplantation section under Surgical Management [Cystic Fibrosis], p. ***) The more common surgical procedure for patients with COPD is lung reduction surgery.

Lung transplantation and lung reduction surgery can improve gas exchange in the patient with COPD. Transplantation is a relatively rare procedure because of cost and the scarce availability of donor lungs. For this reason, the few transplants performed for COPD are usually single lung (GOLD, 2007).

The goal of lung reduction surgery is improvement of gas exchange through removal of hyperinflated lung tissue. These areas of the lungs are filled with stagnant air that is not renewed with some atmospheric air (containing oxygen) during each respiratory cycle. Instead, this stagnant air continues to receive carbon dioxide until the level of carbon dioxide in the hyperinflated alveolus is the same as that in the capillary. Hyperinflated lung areas are useless for gas exchange. After successful lung reduction, most patients have at least a 75% improvement in FEV₁, decreased TLC and RV; and increased activity tolerance. Oxygen therapy may no longer be needed.

**Preoperative care.** Patients are selected for this procedure on the basis of having end-stage emphysema, minimal chronic bronchitis, and stable cardiac function; being ambulatory and not dependent on a ventilator; not having pulmonary fibrosis, asthma, or late-stage cancer; and having been a nonsmoker for at least 6 months. The patient must complete pulmonary rehabilitation before surgery to maximize lung and muscle function. The patient must reach a state in which he or she is able to walk, without stopping, for 30 minutes at 1 mile/hr and maintain a 90% or better oxygen saturation level.

In addition to standard preoperative testing, the patient having lung reduction surgery has tests to determine the location of greatest lung hyperinflation and poorest lung blood flow. These tests include pulmonary plethysmography, gas dilution, and perfusion scans.

**Operative Procedures.** Usually, lung reduction is performed on both lungs through either a large midline incision or a transverse anterior thoracotomy. Each lung is deflated separately and examined for color and texture differences. Normal lung tissue darkens to purple or gray when deflated and becomes more dense or rubbery in texture. Hyperinflated areas do not deflate, and they remain pink with a spongy texture. After areas to remove have been identified, the surgeon removes as much of this tissue as possible, sealing off and reinforcing the remaining normal lung tissue.

**Postoperative Care.** After lung reduction surgery, the patient needs close monitoring for continuing respiratory problems as well as for usual postoperative complications. In addition to the usual care required after thoracotomy (see Surgical Management [Lung Cancer], p. ***) bronchodilator and mucolytic therapies are maintained. Pulmonary hygiene includes incentive spirometry 10 times per hour while awake, chest physiotherapy starting on the first day after surgery, and hourly pulmonary assessment.

Pain is usually managed by epidural delivery of opioids during the early period after surgery. This type of analgesic delivery reduces pain, limits sedation and cognitive dysfunction, and allows the patient to more fully participate in pulmonary hygiene measures.

**DECISION-MAKING CHALLENGE**

**Critical Rescue**

The 72-year-old nursing home resident described earlier is disoriented on an oxygen flow rate of 4 L/min. His vital signs are: P = 112, tachyarrhythmic and irregular, R = 12 through pursed lips, BP = 140/110. His fingers are clubbed, he has a “barrel” chest, he has pitting edema of his lower extremities, and his neck veins are flat in the upright position.

1. Which of these assessment findings is important? Why?
2. What additional assessment data should you obtain?
3. Should you increase his oxygen flow rate? Why or why not?

**Ineffective Breathing Pattern**

**NOC Planning: expected outcomes.** The patient with COPD is expected to achieve an effective breathing pattern that decreases the work of breathing. Indicators include:

- Respiratory rhythm within normal limits for the patient’s age
- Presence of synchronous thoracoabdominal movement
- Use of accessory muscles appropriate to the patient’s activity level
- Increased activity tolerance

**Interventions.** Before any intervention, assess the patient to determine the breathing pattern, especially the rate, rhythm, depth, and use of accessory muscles. The patient with COPD relies more on accessory muscles than on the diaphragm for breathing. These muscles, however, are less efficient than the diaphragm, and the work of breathing increases. Determine whether there are any contributing factors to the increased work of breathing, such as respiratory infection. Interventions aim to improve the patient’s breathing efforts and decrease the work of breathing through the use of specific breathing techniques, positioning, exercise conditioning, and energy conservation.

**Breathing techniques,** such as diaphragmatic or abdominal and pursed-lip breathing, may be helpful for managing dyspneic episodes (Warren & Livesay, 2006). The patient uses these techniques, shown in Chart 32-10, during all activities. The amount of stale air in the lungs is reduced, and the patient gains confidence and control in managing dyspnea. Teach these techniques when the patient is free of dyspnea.

In diaphragmatic breathing, the patient consciously increases movement of the diaphragm. Lying on the back allows the abdomen to relax. Breathing through pursed lips uses the mild resistance of partially closed lips to prolong exhalation and to increase airway pressure. This technique delays airway compression and reduces air trapping. Pursed-lip breathing can be used during diaphragmatic or abdominal breathing.

**Positioning** the patient in an upright position with the head of the bed elevated can help alleviate dyspnea by increasing chest expansion, relaxing the chest muscles, and placing the diaphragm in the proper position to contract. This position also conserves energy by supporting the patient’s arms and upper body.
Energy conservation is the planning and pacing of activities for maximum tolerance and minimum discomfort. Once the FEV₁ falls below 50% predicted, the patient's ability to perform ADLs is limited. Ask the patient to describe a typical daily schedule. Each activity is divided into its smaller parts to determine whether that task can be performed in a different way or at a different time of the day. Assist him or her to plan and pace daily activities. Rest periods are paced between activities. Help the patient develop a personal chart outlining the day's activities and planned rest periods.

Encourage the patient to avoid working with the arms raised. Activities involving the arms decrease exercise tolerance because the accessory muscles of ventilation are then used to stabilize the arms and shoulders. Many activities involving the arms can be done sitting at a table leaning on the elbows. Teach the patient to adjust work heights to reduce back strain and fatigue. Remind him or her to keep arm motions smooth and flowing to prevent jerky motions that waste energy. Teach about the use of adaptive tools for housework, such as long-handled dustpans, sponges, and dusters, to reduce bending and reaching.

Suggest how the patient can organize work spaces so that items used most often are within easy reach. Measures such as dividing laundry or groceries into small parcels that can be handled easily, using disposable plates to save washing time, and letting dishes dry in the rack also conserve energy. Talking requires energy and use of the lungs; therefore teach the patient not to talk when engaged in other activities that require energy, such as walking. In addition, teach him or her to avoid breath-holding while performing any activity.

**Ineffective Airway Clearance**

**NOC Planning: expected outcomes.** The patient with COPD is expected to maintain a patent airway. Indicators include:

- Coughs effectively
- No occurrence of aspiration
- Maintenance of SPO₂ of at least 88%

**Interventions.** The patient with COPD often has difficulty with removal of secretions, which results in compromised breathing and poor oxygenation and tissue perfusion. Excessive mucus also increases the risk for respiratory infections. Assess breath sounds routinely as part of physical assessment and before and after interventions. Careful use of drugs combined with controlled coughing, hydration, and postural drainage may help in airway clearance. If these measures fail, a tracheostomy may be needed on a temporary or permanent basis.

**Coughing** at specific times of the day is helpful because the patient with COPD has excessive mucus. Teach him or her to cough on arising in the morning to eliminate mucus that collected during the night. Coughing clear mucus before mealtimes may facilitate a more pleasant meal. Coughing before bedtime may ensure clear lungs for a less interrupted night's sleep.

To cough effectively, teach the patient to sit in a chair or on the side of a bed with feet placed firmly on the floor. Instruct him or her to turn the shoulders inward and to bend the head slightly downward, hugging a pillow against the stomach. The patient then takes a few deep breaths. After the third to fifth deep breath (in through the nose, out through pursed lips), instruct him or her to bend forward slowly while coughing two or three times from the same breath. Observe the color, consistency, odor, and amount of secretions. On return to a sitting position, the patient takes a comfortable deep breath. The entire coughing procedure is repeated at least twice. After coughing exercises, allow him or her to rest and provide mouth care.

**Chest physiotherapy (PT) with postural drainage** (Figure 32-11) helps some patients move secretions into central airways, re-expand lung tissue, and have more efficient use of the ventilatory muscles. It combines chest percussion with vibration to loosen secretions. Postural drainage uses specific positions and gravity to help remove secretions. Because it does not have a proven benefit for all patients with COPD, postural drainage with chest PT is not used routinely in this population.

**Suctioning** is performed only when abnormal breath sounds are present—not on a routine schedule. For the patient with a
weak cough, weak pulmonary muscles, and inability to expectorate effectively, the nurse or respiratory therapist performs nasotracheal suctioning. Assess the patient for dyspnea, tachycardia, and dysrhythmias during the procedure. Assess for improved breath sounds after suctioning. Suctioning is discussed in detail in Chapter 31.

Positioning may improve airway clearance. Assist the patient who can tolerate sitting in a chair out of bed for 1-hour periods two to three times a day. This position helps move secretions and keeps the diaphragm in a better position for ventilation. Hydration helps airway clearance by thinning secretions, making them easier to remove by coughing. Unless hydration needs to be avoided for other health problems, teach the patient with COPD to drink at least 2 to 3 L/day. Humidifiers may be useful for those living in a dry climate or those who use dry heat during the winter. Instruct the patient to clean the humidifier daily to prevent the growth of mold spores.

Flutter valve mucus clearance devices can be helpful to assist patients to remove airway secretions (Warren & Livesay, 2006). The device is a small, handheld plastic pipe with a short, fat stem and a perforated lid over the bowl (Figure 32-12). Inside the bowl is a free-moving steel ball. The patient inhales deeply and exhales forcefully through the device, causing the ball to move and set up vibrations that are transmitted to the patient’s chest and airways. The vibrations loosen secretions and allow them to be coughed out more easily.

**Imbalanced Nutrition: Less Than Body Requirements**

**Planning: expected outcomes.** The patient with COPD is expected to achieve and maintain a body weight within 10% of ideal. Indicators include:

- Maintains an appropriate weight/height ratio
- Maintains serum albumin or prealbumin within the normal range
The patient with COPD often has food intolerance, nausea, early satiety, loss of appetite, and meal-related dyspnea. The increased work of breathing raises calorie and protein needs. These conditions lead to protein-calorie malnutrition for many patients. Malnourished patients lose total body mass, ventilatory muscle mass and strength, lung elasticity, and alveolar-capillary surface area. All of these problems reduce effective breathing.

Identify patients at risk for or who have this complication and request nutritional consultation. Monitor his or her weight and other indicators of nutrition, such as skin condition and serum prealbumin levels.

Dyspnea management is needed because shortness of breath (dyspnea) is the most common problem related to eating. Dyspnea during mealtimes can be reduced by resting before meals. Teach the patient to plan the biggest meal of the day for the time when he or she is most hungry and well rested. Four to six small meals a day may be preferred to three larger ones. Teach the patient to use pursed-lip and abdominal breathing to alleviate dyspnea. Suggest that using a bronchodilator 30 minutes before the meal may be helpful to reduce dyspnea due to bronchospasm.

Food selection can help prevent weight loss and improve appetite. Abdominal bloating and a feeling of fullness often prevent the patient from eating a complete meal. Teach about foods that are easy to chew and not gas-forming. Dry foods stimulate coughing, and foods such as milk and chocolate may increase the thickness of saliva and secretions. Advise the patient to avoid these foods when symptomatic. Inform him or her that caffeinated beverages should be avoided because they increase urine output and may lead to dehydration.

Urge the patient to eat high-calorie, high-protein foods. Dietary supplements, such as Pulmocare, provide nutrition with reduced carbon dioxide production. If early satiety (feeling too “full” to eat) is a problem, advise the patient to avoid drinking fluids before and during the meal.

Psychological interventions are useful when symptoms are worsened because of anxiety. Help the patient understand this effect and have a plan for dealing with anxiety. Together with the patient, develop a written plan that states exactly what he or she should do if symptoms flare. Having a plan provides confidence and control in knowing what to do, which often helps reduce anxiety. Stress the use of pursed-lip and diaphragmatic breathing techniques during periods of anxiety or panic.

Family, friends, and support groups can be helpful. Recommend professional counseling, if needed, as a positive suggestion, and in no way suggest that this need represents a failure of the patient to cope. Stress that talking with a counselor can help identify techniques to maintain control over the dyspnea and feelings of panic.

Explore other approaches to help the patient control dyspneic episodes and panic attacks. Examples include progressive relaxation, hypnosis therapy, and biofeedback. Biofeedback helps the patient determine the impact of various stimuli on symptoms. Ultimately he or she learns to relax and control these stimuli to avoid the aggravating symptoms. At times, anti-anxiety drug therapy may be needed for severe anxiety.

Activity Intolerance

- Verbalizing anxiety is reduced or absent
- Identifying factors that contribute to anxiety
- Describes respiratory infection–monitoring procedures
- Involves participation in family, work, or social activities
- Performance of selected activities with minimal dyspnea or tachycardia
- Performance of ADLs with no or minimal assistance
- Participation in family, work, or social activities as desired
- Verbalizes clinical manifestations of respiratory infection
- Uses prevention activities such as pneumonia and influenza vaccination and crowd avoidance
- Seeks medical assistance when manifestations of respiratory infection first appear
- Verbalizes clinical manifestations of respiratory infection
- Uses respiratory infection–monitoring procedures
- Seeks medical assistance when manifestations of respiratory infection first appear

Interventions. The patient with COPD often has chronic fatigue. While in the acute phases of the illness, he or she may need extensive help with the ADLs of eating, bathing, and grooming. As the acute problem resolves, encourage the patient to pace activities and provide as much self-care as possible. Teach him or her not to rush through morning activities, because rushing increases dyspnea, fatigue, and hypoxemia. As activity gradually increases, assess the patient’s response by noting skin color changes, pulse rate and regularity, blood pressure, and work of breathing. Suggest the use of supplemental oxygen during periods of high energy use, such as bathing or walking.

Potential for Pneumonia or Other Respiratory Infections

- Verbalizing anxiety is reduced or absent
- Identifying factors that contribute to anxiety
- Involves participation in family, work, or social activities
- Performance of selected activities with minimal dyspnea or tachycardia
- Performance of ADLs with no or minimal assistance
- Participation in family, work, or social activities as desired
- Verbalizes clinical manifestations of respiratory infection
- Uses prevention activities such as pneumonia and influenza vaccination and crowd avoidance
- Seeks medical assistance when manifestations of respiratory infection first appear
- Verbalizes clinical manifestations of respiratory infection
- Uses respiratory infection–monitoring procedures
- Seeks medical assistance when manifestations of respiratory infection first appear

Interventions. Pneumonia is one of the most common complications of COPD. Patients who have excessive secretions or who have artificial airways are at increased risk for respiratory tract infections. The risk is greatly increased for older adults. Teach patients to avoid large crowds, and stress the importance of receiving a pneumonia vaccination and a yearly influenza vaccine (“flu shot”) (Bruce & McEvoy, 2007).
Community-Based Care

Home Care Management

Most patients with chronic obstructive pulmonary disease (COPD) are treated in the ambulatory care setting and cared for at home. When pneumonia or a severe exacerbation of the disease develops, the patient usually returns home after treatment. For those with advanced disease, however, 24-hour care may be needed for ADLs and for monitoring for acute episodes or progression of the illness. Patients may not be able to enjoy work or recreational activities because of severe dyspnea and fatigue. If home care is not possible, placement in a long-term care setting may be needed.

Hypoxemic patients can benefit from long-term use of oxygen at home. Home oxygen may be needed only during periods of exercise or sleep if hypoxemia occurs only during these times. Continuous, long-term oxygen therapy can reverse tissue hypoxia and decrease pulmonary vascular resistance. It can also improve cognitive ability and well-being. For more information on oxygen therapy, see Chapter 30.

Most patients can benefit from a structured pulmonary rehabilitation program. The overall goal of these collaborative programs is to increase a person’s ability to compensate for and live with COPD. The patient with COPD is referred to a pulmonary rehabilitation program before illness becomes severe. Those with the least severe functional loss benefit the most.

Coordinate with the case manager to obtain the equipment needed for care at home. Patient needs may include oxygen therapy, a hospital-type bed, a nebulizer, a tub transfer bench, and visits from a home care nurse to continue monitoring the health status, review the drug regimen, and evaluate home care needs.

The patient with COPD faces a lifelong disease with remissions and exacerbations. Explain to the patient and family that he or she may have periods of anxiety, depression, and ineffec-
tive coping. The person who was a smoker may also have self-directed anger.

Financial concerns often increase anxiety and interfere with disease management. The condition may worsen to the point that the patient cannot work. Disability benefits through Social Security or private disability insurance plans can help ease the financial burden. Medicare or other health insurers may assist with payment for home oxygen therapy and nebulizer treatments. Coordinate with the social worker or case manager to help the patient make the needed arrangements.

Health Teaching

Patients with COPD need to know as much about the disease as possible so that they can better manage it and themselves. Patients and families should be able to discuss drug therapy, manifestations of infection, avoidance of respiratory irritants, the nutrition therapy regimen, and activity progression. Instruct them to identify and avoid stressors that can worsen the disease.

Teach the patient techniques of pursed-lip breathing, diaphragmatic breathing, positioning, relaxation therapy, energy conservation, and coughing and deep breathing. Two factors that interfere with teaching hospitalized patients are the shortened length of stay and the presence of dyspnea. It may be unrealistic to cover all of the topics in the education checklist during a single hospitalization. The primary nurse or case manager should coordinate teaching with the home care or clinic staff.

Health Care Resources

Provide appropriate referrals as needed. Home care visits may be warranted, particularly if the patient must use home oxygen therapy for the first time. Chart 32-11 lists assessment areas for the patient with COPD at home. Referral to assistance programs, such as Meals on Wheels, can be helpful. Provide a list of support groups, as well as Better Breather clubs sponsored by the American Lung Association. If the patient is having difficulty with smoking cessation and indicates the desire for assistance, make the referrals.

Chart 32-11  HOME CARE ASSESSMENT

The Patient with Chronic Obstructive Pulmonary Disease

- Assess Respiratory Status and Adequacy of Ventilation
  - Measure rate, depth, and rhythm of respirations.
  - Examine mucous membranes and nail beds for evidence of hypoxia.
  - Determine use of accessory muscles.
  - Examine chest and abdomen for paradoxical breathing.
  - Count number of words patient can speak between breaths.
  - Determine need and use of supplemental oxygen. (How many liters per minute is the patient using?)
  - Determine level of consciousness and presence/absence of confusion.
  - Auscultate lungs for abnormal breath sounds.
  - Measure oxygen saturation by pulse oximetry.
  - Determine sputum production, color, and amount.
  - Ask about activity level.
  - Observe general hygiene.
  - Measure body temperature.

- Assess Cardiac Status
  - Measure rate, quality, and rhythm of pulse.
  - Check dependent areas for edema.
  - Check neck veins for distention with the patient in a sitting position.
  - Measure capillary refill.

- Assess Nutritional Status
  - Weight maintenance, loss, or gain
  - Food and fluid intake
  - Use of nutritional supplements
  - General condition of the skin
  - Assess patient’s and caregiver’s adherence and understanding of illness and treatment, including:
    - Correct use of supplemental oxygen
    - Correct use of inhalers
    - Drug schedule and side effects
    - Manifestations to report to the health care provider indicating the need for acute care
    - Increasing severity of resting dyspnea
    - Increasing severity of usual symptoms
    - Development of new symptoms associated with poor oxygenation
    - Respiratory infection
    - Failure to obtain the usual degree of relief with prescribed therapies
    - Unusual change in condition
    - Use of pursed-lip and diaphragmatic breathing techniques
    - Scheduling of rest periods and priority activities
    - Participation in rehabilitation activities

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The patient is a 67-year-old retired teacher with COPD who lives in a first-floor condo with her husband, who is also a retired teacher. The home care nurse is making a follow-up visit 1 week after the patient returned home after a 4-day hospital stay for exacerbation of symptoms. On arriving at the house, the home care nurse finds the patient upset and crying. When asked what has upset her, she replies that her husband “won’t let me do anything around the house because I am so short of breath. I might as well be dead.”

1. How should the nurse respond to this statement?
2. What psychosocial assessment of this patient and her situation should be made?
3. Should the visiting nurse include the husband in any part of this discussion? Why or why not?
4. What adjustments in household tasks could this patient make to conserve her energy?

**DECISION-MAKING CHALLENGE**

**Coordination of Care**

The patient is a 67-year-old retired teacher with COPD. She lives in a first-floor condo with her husband, who is also a retired teacher. The home care nurse is making a follow-up visit 1 week after the patient returned home after a 4-day hospital stay for exacerbation of symptoms. On arriving at the home, the home care nurse finds the patient upset and crying. When asked what has upset her, she replies that her husband “won’t let me do anything around the house because I am so short of breath. I might as well be dead.”

1. How should the nurse respond to this statement?
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4. What adjustments in household tasks could this patient make to conserve her energy?

**Evaluation: Outcomes**

Evaluate the care of the patient with COPD on the basis of the identified nursing diagnoses and collaborative problems. The expected outcomes are that the patient should:

- Achieve effective breathing pattern that decreases the work of breathing
- Maintain a patent airway
- Achieve and maintain a body weight within 10% of his or her ideal weight
- Avoid serious respiratory infections

Specific indicators for these outcomes are listed for each nursing diagnosis and collaborative problem under the Planning and Implementation section on p. 27.

**CYSTIC FIBROSIS**

**Pathophysiology**

Cystic fibrosis (CF) is a genetic disease that affects many organs and lethally impairs pulmonary function. Although this disorder is present from birth and usually is first seen in early childhood, almost half of all people with cystic fibrosis in the United States are adults (Cystic Fibrosis Foundation, 2007).

The underlying problem of CF is blocked chloride transport in the cell membranes. The error in chloride transport causes the formation of mucus that has little water content and is thick. The thick, sticky mucus causes problems in the lungs, pancreas, liver, salivary glands, and testes. The mucus plugs up glands in these organs, causing atrophy and organ dysfunction. Nonpulmonary problems include pancreatic insufficiency with malnutrition and intestinal obstruction, poor growth, male sterility, and cirrhosis of the liver. These primary problems cause many additional health problems in young adulthood, especially osteoporosis and diabetes mellitus (Cystic Fibrosis Foundation, 2007). The primary cause of death in the patient with CF is respiratory failure.

The pulmonary problems of CF result from the constant presence of thick, sticky mucus and are the most serious complications of the disease. The mucus narrows airways, reducing airflow and interfering with oxygenation and tissue perfusion. The constant presence of mucus results in chronic respiratory tract infections, chronic bronchitis, and chronic dilation of the bronchioles (bronchiectasis). Lung abscesses are common. Over time, the bronchioles distend and have increased numbers (hyperplasia) of mucus-producing cells and increased mucus-producing cell size (hypertrophy). Complications include pneumothorax, arterial erosion and hemorrhage, and respiratory failure.

The disorder is most common among white people, and about 4% are carriers. CF is very rare among African Americans and Asians. Males and females are affected equally.

**GENETIC CONSIDERATIONS**

CF is an autosomal recessive disorder in which both gene alleles must be mutated for the disease to be expressed. The CF gene is located on chromosome 7 and produces a protein that controls chloride movement across cell membranes (Nussbaum et al., 2007). The severity of CF varies greatly; however, life expectancy is always considerably reduced, with an average of 32 years. People with one mutated allele are carriers and have few or no symptoms of CF but can pass the abnormal allele on to their children. Currently, more than 1200 different mutations have been identified. The inheritance of different mutations is thought to be responsible for the wide variation in disease severity.

With improvement in specific testing, people with health problems who were not known to have CF previously may be identified so that therapies can be tailored for better outcomes. In acute care settings, patients with what appear to be acute pulmonary problems who do not respond as expected to proven therapy may, in actuality, have some undiagnosed form of CF that unfavorably influences the clinical course of a superimposed pulmonary problem (Workman & Winkelman, 2008). In this type of case, the nurse should obtain a good history of the patient’s previous respiratory problems and his or her response to therapy. The possibility of CF should always be kept in mind when a patient does not respond as expected to standard therapies.

**Patient-Centered Collaborative Care**

**Assessment**

Usually, cystic fibrosis (CF) is diagnosed in childhood. The major diagnostic test is sweat chloride analysis (Gardner, 2007). Additional genetic testing can be performed to determine which specific mutation a person may have. This distinction can be important because different mutations result in different degrees of disease severity. The defect in chloride movement prevents absorption of sodium chloride in the sweat glands; thus more chloride than normal is present in the sweat. The sweat chloride test is positive for CF when the chloride level in the sweat ranges between 60 and 200 mEq/L (mmol/L), compared with the normal value of 5 to 35 mEq/L.

Nonpulmonary manifestations include abdominal distention, gastroesophageal reflux, rectal prolapse, foul-smelling stools, and steatorrhea (excessive fat in stools). The patient may be malnourished and have many vitamin deficiencies, especially of the fat-soluble vitamins like vitamins A, D, E, and K. As pancreatic function decreases, he or she has symptoms...
of diabetes mellitus from loss of insulin production. In addition, the adult with CF is usually smaller and thinner than average.

Pulmonary manifestations caused by CF are progressive. The respiratory infections are frequent or chronic with periods of exacerbations. Patients usually have chest congestion, limited exercise tolerance, cough, sputum production, use of accessory muscles, and decreased pulmonary function (especially FVC and FEV₁) (Grossman & Grossman, 2005). Chest x-rays show persistent infiltrate and an increased anteroposterior (AP) diameter.

During an acute exacerbation or when the disease progresses to end stage, the patient has increased chest congestion, reduced activity tolerance, increased crackles, increased cough, increased sputum production (often with hemoptysis), and severe dyspnea. Fatigue increases in proportion with the dyspnea. Arterial blood gas (ABG) studies show acidosis with greatly reduced partial pressure of arterial oxygen (PaO₂), increased partial pressure of arterial carbon dioxide (PaCO₂), increased bicarbonate levels, and low pH.

When infection is present, the patient has fever, an elevated white blood cell count, and decreased oxygen saturation. Other manifestations of infection include tachypnea, tachycardia, intercostal retractions, weight loss, and increased fatigue.

Interventions

The patient with CF needs daily therapy to slow disease progression and enhance gas exchange. There is no cure for CF.

Nonsurgical Management

The management of the patient with CF is complex and lifelong. Nutritional management focuses on weight maintenance, vitamin supplementation, diabetes management, and pancreatic enzyme replacement. Pulmonary management is focused on preventive maintenance and management of pulmonary exacerbation. Priority nursing interventions focus on teaching about drug therapy, infection prevention, pulmonary hygiene, nutrition, and vitamin supplementation.

Preventive/maintenance therapy involves the use of a regimen of chest physiotherapy, positive expiratory pressure, active cycle breathing technique, and an individualized regular exercise program. Pulmonary function tests (PFTs), especially FEV₁, are monitored regularly. Maintenance drugs include bronchodilators, anti-inflammatory agents, mucolytics, and antibiotics.

Exacerbation therapy is needed when the patient with CF has a change in manifestations from baseline. Such changes include increased chest congestion, decreased activity tolerance, increased or new-onset crackles, and at least a 10% decrease in FEV₁. Other manifestations occurring with exacerbation include increased sputum production with bloody or purulent sputum, increased frequency and duration of coughing, decreased appetite, weight loss, fatigue, decreased SaO₂, and ventilatory muscle retractions. Often infection is present and the patient also has fever, increased lung infiltrate on chest x-ray, and an elevated white blood cell count.

Every attempt is made to avoid having the patient with CF mechanically ventilated. Treatment focuses on airway clearance, increased oxygenation, and antibiotic therapy. Supplemental oxygen is prescribed on the basis of SaO₂ levels. Heliox delivery of 50% oxygen and 50% helium may improve gas exchange and oxygen saturation. The respiratory therapist initiates airway clearance techniques (ACTs) four times a day. Bronchodilator and mucolytic therapies are intensified (higher doses given more frequently than for maintenance). Steroidal anti-inflammatory agents are started or increased.

Depending on the severity of the exacerbation, a 10- to 14-day course of oral antibiotics may be prescribed. If the exacerbation is more severe, aerosolized tobramycin is prescribed. If oral/inhaled antibiotics are not effective or if the exacerbation is very severe, IV antibiotics are used, usually an aminoglycoside, such as tobramycin and colistin or meropenem (Merrem) (Elpern et al., 2007).

A serious bacterial infection for patients with CF is Burkholderia cepacia. The organism lives well in the respiratory systems of patients with CF and becomes resistant to antibiotic therapy relatively quickly. It is spread by casual contact from one CF patient to another. For this reason, the Cystic Fibrosis Foundation bans infected patients from participating in any foundation-sponsored events. It is also possible for B. cepacia to be transmitted to a CF patient during clinic and hospital visits; thus special infection control measures that limit close contact between persons with CF are needed. These measures include separating CF patients on hospital units and seeing them in the clinic on different days. Strict CF Foundation–approved procedures should be used when cleaning clinic rooms, pulmonary function laboratories, and respiratory therapy equipment to reduce the risk of contamination.

Teach patients about protecting themselves by avoiding direct contact of bodily fluids such as saliva and sputum. Teach them not to routinely shake hands or kiss people in social settings. Handwashing is critical because the organism also can be acquired indirectly from contaminated surfaces, such as sinks and tissues.

As specialized treatment for CF improves and life span increases, other problems may occur. Patients may have bronchial bleeding from lung arteries. Interventional radiology may be needed to embolize the bleeding arterial branches. Patients with CF may undergo this procedure repeatedly to control hemoptysis. See Chapter 38 for information on interventional radiology vascular procedures.

Surgical Management

The surgical management of the patient with CF involves lung and/or pancreatic transplantation. The patient has manifestations but is at continuing risk for lethal pulmonary infections, especially with anti-rejection drug therapy. Transplantation extends life by 10 to 20 years, depending on other factors.

Fewer lung transplants are performed compared with transplantation of other solid organs. The problem is related to the scarcity of available lungs. In addition, many of the people who could benefit from lung transplantation have serious problems in other organs that make extended surgical procedures dangerous.

Lung transplant procedures include two lobes or a single lung transplantation, as well as double-lung transplantation. The type of procedure is determined by the patient’s age and overall condition, the cause of the lung problem, and the life expectancy after transplantation. Usually, the patient with CF has a bilateral lobe transplant from either a cadaver donor or living-related donor.

Preoperative care. Many factors are considered before lung transplantation surgery. Recipient and donor criteria vary from one program to another, but some criteria are universal.
Recipient criteria for the person who will receive the transplant include that the recipient must have severe, irreversible lung damage. It is important, however, that the patient be well enough to survive the surgery. Usually only those younger than 55 years receive transplants, although transplantation is considered on an individual basis. Exclusion criteria are:

- Severe psychiatric disorders or self-destructive tendency
- Proven history of noncompliance or poor compliance with medical regimens
- Current cancer or cancer within the past 5 years
- Systemic infection
- Irreversible heart, kidney, or liver damage/disease
- Presence of any problem that would be made worse by immunosuppression

Donor criteria, regardless of whether the lung tissue is obtained from a cadaver or from a living-related donor, include:

- Infection free
- Cancer free
- Healthy lung tissue
- Close tissue match with the recipient
- Same blood type as the recipient

When the donor is living-related, additional criteria are:

- Age is younger than 55 years.
- Donor has normal cardiac function.
- Pulmonary function will remain adequate after tissue removal.
- The donor has had no previous chest surgery.
- Donor is psychologically stable.
- Donor has not been coerced into this situation.

The two nursing priorities before surgery are teaching the patient the expected regimen of pulmonary hygiene to be used in the period immediately after surgery and assisting the patient in a pulmonary muscle strengthening/conditioning regimen.

Operative procedures. The patient may or may not need to be placed on cardiopulmonary bypass, depending on the exact procedure. Those having single-lung or lobe transplantation usually do not need bypass; those having double-lung transplantation usually do.

The most common incision used for lung transplantation is a transverse thoracotomy (“clamshell”) for best access. The diseased lung or lungs are removed. The new lobes, lung, or lungs are placed in the chest cavity with anastomoses (connections) made to the proper airways (trachea, mainstem bronchus, or secondary bronchus) and blood vessels. Usually, lung transplantation surgery is completed within 4 to 6 hours.

Postoperative care. The patient is intubated for at least 48 hours. In addition, chest tubes and arterial lines are in place. Much of the care needed is the same as that for any thoracic surgery (see p. XXX).

Major problem areas after lung transplantation are bleeding, infection, and transplant rejection. Bleeding is most common in patients who had cardiopulmonary bypass with anti-coagulation. Usually the patient remains in the ICU for several days after transplantation.

Anti-rejection drug regimens must be started immediately after surgery, which increases the risk for infection. The drugs generally used for routine long-term rejection suppression after organ transplantation are combinations of very specific immunosuppressants (cyclosporine [Sandimmune]), less specific immunosuppressants (azathioprine [Imuran] or mycophenolate mofetil [CellCept]), and one of the corticosteroids, such as prednisone (Apo-Prednisone, Deltasone) or prednisolone (Delta-Cortef). Corticosteroids are avoided in the first 10 to 14 days after surgery because of their negative impact on the healing process. (See Chapter 19 for more information on anti-rejection therapy.)

PRIMARY PULMONARY HYPERTENSION

Pathophysiology

Pulmonary hypertension can occur as a complication of other lung disorders. Primary pulmonary hypertension (PPH) occurs in the absence of other lung disorders, and its cause is unknown although exposure to some drugs increases the risk. This disorder is rare and occurs mostly in women between the ages of 20 and 40 years (Widlitz et al., 2007).

GENETIC CONSIDERATIONS

About 50% of patients with the disorder have a genetic mutation in the BMPR2 gene, which codes for a growth factor receptor (McCance & Huether, 2006). Excessive activation of this receptor allows increased growth of arterial smooth muscle in the lungs, making these arteries thicker. Many more people have mutations in this gene than have PPH. It is thought that these mutations increase the susceptibility to PPH when other, often unknown, environmental factors also are present. Often PPH is not diagnosed until late in the disease process when the lungs and heart have already been significantly damaged. Teach people, especially women, who have a first-degree relative (parent or sibling) with PPH to have regular health checks and to consult a health care provider whenever pulmonary problems are present (Ross, 2007).

The pathologic problem in PPH is blood vessel constriction with increasing vascular resistance in the lung. Pulmonary blood pressure rises and blood flow decreases, leading to poor perfusion and hypoxemia. Eventually, the right side of the heart fails (cor pulmonale) from the continuous workload of pumping against the high pulmonary pressures. Without treatment, death usually occurs within 2 years after diagnosis.

Patient-Centered Collaborative Care

Assessment

The most common early manifestations are dyspnea and fatigue in an otherwise healthy adult. Some patients also have angina-like chest pain. Table 32-3 lists the classification of PPH.

Diagnosis is made from the results of right-sided heart catheterization showing elevated pulmonary pressures. Other test results suggesting pulmonary hypertension include abnormal ventilation-perfusion scans and pulmonary function tests (PFTs) showing reduced functional pulmonary volumes with reduced diffusion capacity, and spiral computed tomography (CT).

Interventions

Nonsurgical interventions that reduce pulmonary pressures and slow the development of cor pulmonale involve drugs that dilate pulmonary vessels and prevent clot formation. Warfarin (Coumadin) therapy is taken daily to achieve an international normalized ratio (INR) of 1.5 to 2.0. Calcium channel blockers, such as nifedipine (Procardia) and diltiazem (Cardizem), have been used to dilate blood vessels. Endothelin-receptor
antagonists, such as bosentan (Tracleer), induce blood vessel relaxation and decrease pulmonary arterial pressure. These agents, however, cause general vessel dilation and some degree of hypotension. Natural and synthetic prostacyclin agents provide the best specific dilation of pulmonary blood vessels. Continuous infusion of epoprostenol (Flolan) or treprostinil (Remodulin) through a small IV pump reduces pulmonary pressures and increases lung blood flow. An alternate therapy that is also effective is the delivery of treprostinil (Remodulin) by subcutaneous infusion through a microinfusion pump.

A critical nursing priority for a patient undergoing this therapy is that, although it is very effective, deaths have been reported if the drug delivery is interrupted even for a matter of minutes. Teach the patient to always have backup drug cassettes and battery packs. If these are not available or if the line is disrupted, the patient should go to the emergency department immediately. The second nursing priority is working with the patient to prevent sepsis. The continuous central line IV setup provides an access for organisms to directly enter the bloodstream. Teach the patient and at least one family member to use strict aseptic technique in all aspects of manipulating the drug delivery system. Also teach him or her to notify the pulmonologist at the first sign of any respiratory or systemic infection.

When the heart has undergone some hypertrophy and cardiac output has fallen, the patient may be started on a regimen of digoxin (Lanoxin) and diuretics. Oxygen therapy is used when dyspnea is continuous or uncomfortable. These therapies do not cure the disorder; they just improve function and reduce symptoms. Surgical management of primary pulmonary hypertension involves single-lung or whole-lung transplantation. When cor pulmonale also is present, the patient may need a combined heart-lung transplantation. It is not known whether the process of pulmonary vasoconstriction can begin again in the transplanted lungs or if the transplant is a “cure.”

**NCLEX EXAMINATION CHALLENGE**

The client is a 21-year-old nursing student with PPH who is admitted for an emergency appendectomy and is now in the PACU. She has a peripheral venous line in her left arm and a central venous catheter connected to a continuous infusion pump with Flolan. Her postoperative orders indicate that she is to receive 2 g of cephalothin (Keflin) by IVPB immediately. When the drug arrives, her peripheral line is infiltrated. What is your best action?

A. Infuse the Keflin into the central line along with the Flolan.
B. Disconnect the Flolan for 15 minutes and infuse the Keflin.
C. Restart a peripheral IV and use it to administer the Keflin.
D. Ask whether the Keflin can be given orally since the client is awake.

**INTERSTITIAL PULMONARY DISEASES**

The category of interstitial pulmonary diseases contains a variety of lung disorders, also called fibrotic lung diseases, that have some features in common. All affect the alveoli, blood vessels, and surrounding support tissue of the lungs rather than the airways. Thus these disorders are restrictive (preventing good expansion and recoil of the gas exchange unit) rather than obstructive. With restrictive disease, the lung tissues thicken, causing reduced gas exchange and “stiff” lungs that do not expand well. Unlike obstructive problems, air trapping does not occur and the patient does not develop a “barrel chest.” Often the onset of these disorders is slow and dyspnea is the most common manifestation.

**SARCOIDOSIS**

**Pathophysiology**

Sarcoidosis is a granulomatous disorder of unknown cause that can affect any organ, but the lung is involved most often. It develops over time with growths called granulomas forming in the lungs. Granulomas contain lymphocytes, macrophages, epithelioid cells, and giant cells.

Pulmonary sarcoidosis involves autoimmune responses in which the normally protective T-lymphocytes increase and cause damaging actions in lung tissue. Alveolar cells are the targets of the damaging actions. No single cause for T-lymphocyte activation has been identified, although infection and genetic predisposition appear to play a role. Alveolar inflammation (alveolitis) occurs from the presence of immune cells in the alveoli. Chronic inflammation causes fibrosis (scar tissue formation in the lungs). The fibrosis reduces lung compliance (elasticity) and the ability to exchange gases. Cor pulmonale (right-sided cardiac failure) is often present, because the heart can no longer pump effectively against the stiff, fibrotic lung.

The disease usually affects young adults. Manifestations include enlarged lymph nodes in the hilar area of the lungs, lung...
infiltrate on chest x-ray, skin lesions, and eye lesions. The first indication of disease may be an abnormal chest x-ray in an otherwise healthy patient. The most common symptoms include cough, dyspnea, hemoptysis, and chest discomfort. In many patients, the illness resolves permanently. Others may have progressive pulmonary fibrosis and severe systemic disease.

**Patient-Centered Collaborative Care**

Sarcoidosis is suspected in the patient who has a cough, dyspnea, and abnormal chest x-ray but is otherwise asymptomatic. Other conditions to rule out before diagnosing sarcoidosis are lung infections and cancer. Fiberoptic bronchoscopy may also be used in the diagnosis of this disorder (see Chapter 29).

Sarcoidosis is staged on the basis of x-ray findings. Higher stages have greater damage and more widespread disease. Pulmonary function studies often show a restrictive pattern of decreased lung volumes and impaired diffusing capacity. Irreversible lung changes develop in 10% to 15% of patients. Patients who develop severe restrictive disease may also develop secondary pulmonary hypertension.

The goal of therapy is to lessen symptoms and prevent fibrosis. Management varies. If the patient is asymptomatic and has normal pulmonary function, no treatment is given. Decreased total lung capacity (TLC), diffusing capacity, or forced vital capacity (FVC); involvement of other organs; and hypercalcemia are indicators for treatment.

Corticosteroids are the main type of therapy. Dosages vary from 40 to 60 mg daily with tapering doses over 6 to 8 weeks, to a maintenance dose of 10 to 15 mg daily for 6 months. Further therapy may continue over 12 months. Follow-up and monitoring include assessment of symptom severity, pulmonary function studies, chest x-rays, a complete blood count, serum creatinine, serum calcium, and urinalysis. Teach the patient about side effects of steroid therapy and other aspects of physical care as indicated.

**IDIOPATHIC PULMONARY FIBROSIS**

*Pathophysiology*

Idiopathic pulmonary fibrosis is a common restrictive lung disease. The typical patient is an older adult with a history of cigarette smoking or chronic exposure to inhalation irritants such as metal particles, dust, organic chemicals, and wood fires. Unlike sarcoidosis, pulmonary fibrosis is highly lethal. Most patients have progressive disease with few remission periods. Even with proper treatment, patients usually survive less than 5 years after diagnosis.

Pulmonary fibrosis is an example of excessive wound healing. Once lung injury occurs, an inflammatory process begins tissue repair. The inflammation continues beyond normal healing time, causing extensive fibrosis and scarring. These changes thicken alveolar tissues, making gas exchange difficult.

**Patient-Centered Collaborative Care**

The onset of disease is slow, with early symptoms of mild dyspnea on exertion. Pulmonary function tests show decreased FVC. As the fibrosis progresses, the patient becomes more dyspneic and hypoxemia becomes severe. Eventually, the patient needs high levels of oxygen and often is still hypoxic.

Respirations are rapid and shallow.

The goal of therapy is to slow the fibrotic process and manage dyspnea. Corticosteroids and other immunosuppressants are the mainstays of therapy. Immunosuppressant drugs include cytotoxic drugs such as cyclophosphamide (Cytoxan), Neosar, Procytox (Leukeran), or methotrexate (Folex). These drugs have many side effects, including immunosuppression, nausea, and lung and liver damage and have shown limited benefit. New studies using the combination therapy of corticosteroids, azathioprine, interferon gamma 1b, and N-acetylcysteine show promise of slowing disease progression (Burns, 2006). Starting drug therapy early is critical, even though not all patients respond to therapy. Even among those who have a response to therapy, the disease eventually continues to progress and leads to death by respiratory failure.

Lung transplantation is a curative therapy; however, the selection criteria, cost, and availability of organs make this option unlikely for most patients.

The patient and family need support and help with community resources after diagnosis. Nursing care focuses on assisting the patient and family in understanding the disease process and maintaining hope for control of the fibrosis. It is important to prevent respiratory infections. Teach the patient and family about the manifestations of infection, and encourage them to avoid respiratory irritants, crowds, and other people with known infections.

Home oxygen is needed by the time the patient becomes symptomatic because significant fibrosis has already occurred. Teach him or her about oxygen use as a continuous therapy. Fatigue is a major problem. Teach the patient and family about energy conservation measures (see discussion of energy conservation, p. *** in the Chronic Obstructive Pulmonary Disease section). Activity limitations and rest help reduce the work of breathing and oxygen consumption.

Support the patient’s need to be as independent as possible, and encourage him or her to pace activities and accept assistance as needed. The disease is costly because the patient is often unable to work and may need home care.

In the later stages of the disease, the focus is to reduce the sensation of dyspnea. This is often accomplished with the use of oral, parenteral, or nebulized morphine. Provide information about hospice, which supports and coordinates resources to meet the needs of the patient and family when the prognosis for survival is less than 6 months.

**OCCUPATIONAL PULMONARY DISEASE**

*Pathophysiology*

Exposure to occupational or environmental fumes, dust, vapors, gases, bacterial or fungal antigens, and allergens can result in a variety of respiratory disorders. Depending on the degree, frequency, and intensity of exposure and on the specific disease, patients may have acute reversible effects or chronic lung disease. All occupational pulmonary diseases are made worse by cigarette smoking; thus smoking cessation efforts are very important.

Many occupational diseases have an onset of symptoms long after the initial exposure to the offending agent. The patient’s personal history can provide clues about the presence and cause of occupational pulmonary diseases. Common occupational pulmonary diseases include occupational asthma, pneumoconiosis, diffuse interstitial fibrosis, and extrinsic allergic alveolitis. Chart 32-12 lists the key features of these disorders.
**Chart 32-12  KEY FEATURES**

**Common Occupational Pulmonary Diseases**

<table>
<thead>
<tr>
<th>Disease and Category</th>
<th>Causes and Manifestations</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Occupational Asthmas</strong></td>
<td></td>
</tr>
</tbody>
</table>
| Latency (allergic) asthma | Airway narrowing related only to workplace exposures  
Atopic allergic response to industrial irritants  
Develops after a period of exposure (from several weeks to several years)  
Characterized by airflow limitation  
Usually resolves when exposure ceases  
Obstructive disease |
| Irritant-induced asthma | Manifestations appear only in the workplace  
First onset usually occurs within 24 hours of exposure  
Common irritants are chlorine, ammonia, and phosgene  
Characterized by sloughing of epithelium, thickening of the basement membranes, and mucosal inflammation  
Early manifestations include cough, wheeze, and dyspnea  
High exposures can lead to pulmonary edema, ARDS, and death  
Most tissue changes are permanent  
Obstructive and restrictive disease |
| **Pneumoconiosis** |                           |
| Silicosis | Chronic fibrosis from long-term inhalation of silica dust  
Found among people working in mines, stone quarries, and foundries. Also found in people working in these industries: glass-making, pottery, sandblasting, tile and brick making, soap and polishes, and manufacture of filters  
Characterized by nodule formation between alveoli leading to fibrosis  
Manifestations include dyspnea on exertion, fatigue, weight loss, reduced lung volume, and upper lobe fibrosis  
Restrictive disease |
| Coal Miner’s Disease (Black Lung Disease) | Massive deposits of coal dust in the lungs leading to diffuse fibrosis  
Develops earlier among miners who smoke  
Early manifestations are similar to bronchitis  
Emphysema is a late development  
Restrictive disease |
| **Diffuse Interstitial Fibrosis** |                           |
| Asbestosis | Occurs among people who work in asbestos mines, building construction/remodeling, and shipyards  
Characterized by diffuse pleural thickening and diaphragmatic calcification  
Restrictive disease |
| Talcosis | Occurs among people who work in industries that manufacture paint, ceramics, roofing materials, cosmetics, and rubber goods  
Restrictive disease |
| Berylliosis | Occurs among people who work in industries in which metal is heated (steel mills, welding) or metal is machined, creating dust  
Has a genetic component for increased susceptibility to disease after beryllium exposure  
Restrictive disease |
| **Extrinsic Allergic Alveolitis** |                           |
| “Farmer’s Lung”  “Bird Fancier’s Lung”  “Machine Operator’s Lung” | Hypersensitivity pneumonitis as an immunologic response to inhaling dust or chemical that contains bacterial or fungal antigens  
Characterized by formation of granulomas with central necrosis in the alveoli and surrounding blood vessels  
Restrictive disease |

ARDS, Acute respiratory distress syndrome.

**Patient-Centered Collaborative Care**

Consider an occupational cause for all patients with new-onset asthma or dyspnea. Obtain a thorough history of occupational exposure and onset of symptoms because there may or may not be a latency period between exposure and onset of symptoms. Determine whether the symptoms are acute or chronic. Ask the patient about the use of inhalation protection and about cigarette smoking. Use this opportunity to teach the patient about ways to quit smoking.

Prevention is important to avoiding disability from occupation-related disease. Teach about the importance of
using special respirators and ensuring adequate ventilation when working in potentially harmful environments.

The patient with occupational asthma with a latency period should be removed from the site of exposure, transferred to a job without exposure, and treated with asthma drugs. Nursing care is similar to the care for asthma not caused by the workplace environment. Refer the patient to a social worker, who provides information regarding compensation and pensions.

Nursing interventions for patients with occupational lung restrictive disease are the same as for those with emphysema. Hypoxemic patients require supplemental oxygen. In addition, respiratory therapies to promote sputum clearance are essential.

**Bronchiolitis Obliterans Organizing Pneumonia (BOOP)**

**Pathophysiology**

Bronchiolitis obliterans organizing pneumonia (BOOP) is an inflammatory process that allows connective tissue plugs to form in the lower airways and in the tissue between the alveoli. The luminal inflammation triggers white blood cell clumping with fibroblast (connective cell) growth that occludes and eventually obliterates these airways and leads to restricted lung volumes with decreased vital capacity. Fibrosis is not part of the pathology of BOOP. It is not a true pneumonia, but the manifestations resemble respiratory infection.

No true cause of BOOP has been established although many personal and environmental conditions are associated with it. Suggested triggers include infectious organisms, drugs (chemotherapy agents, certain antibiotics [sulf-based drugs, cephalosporins, amphotericin B], antiseizure drugs, cocaine, and amiodarone), or the presence of another connective tissue disorder such as rheumatoid arthritis or systemic lupus erythematosus. BOOP is also associated with chest radiation therapy for cancers in the breast or lung.

BOOP is most common in people between 30 and 60 years and affects all races and both genders equally. It is not associated with cigarette smoking or other tobacco use. It is more common among patients who have received solid organ transplants and may be part of an acute rejection episode. Depending on how fast the problem progresses and the degree to which it interferes with gas exchange, BOOP can lead to death.

**Patient-Centered Collaborative Care**

An event or condition triggers the inflammatory cascade within lower airway lumens, causing manifestations of dyspnea, fever, mild cough, flu-like symptoms, and crackles on auscultation. In some patients, the problem resolves spontaneously. In others, the problem can rapidly progress and be fatal within 3 days of the appearance of manifestations. Usually, manifestations are present for weeks or months and do not improve with standard antibiotic therapy (White & Ruth-Sahd, 2007).

Diagnosis of BOOP is difficult because the manifestations are nonspecific and are similar to many other respiratory problems. Chest x-rays do not differentiate BOOP from any other respiratory problem. Although computed tomography (CT) scans can show more widespread changes in pulmonary tissue, it can only suggest BOOP, not confirm it. Biopsy with histologic findings are needed to confirm a BOOP diagnosis.

The most effective treatment for BOOP is corticosteroid therapy. A short course of the drug for acute disease can reduce manifestations, and the patient may never have a relapse. For patients with more severe disease and those with any type of additional health problem, a year of corticosteroid therapy may be needed (White & Ruth-Sahd, 2007). In this population, BOOP is more of a chronic disease with some degree of permanent restrictive disease. Exacerbations can occur.

**LUNG CANCER**

**Pathophysiology**

Lung cancer is a leading cause of cancer-related deaths worldwide. In the United States, more deaths from lung cancer occur each year than from prostate cancer, breast cancer, and colon cancer combined. The American Cancer Society estimates that more than 186,000 new cases of lung cancer are diagnosed each year and that more than 165,000 deaths occur each year from it (American Cancer Society, 2008). The overall 5-year survival for all patients with lung cancer is only 14%. This poor long-term survival is due to the fact that most lung cancers are diagnosed at a late stage, when metastasis is present. Only 15% of patients have small tumors and localized disease at the time of diagnosis (American Cancer Society, 2008).

Despite many advances in cancer treatment, the overall prognosis for lung cancer remains poor unless the tumor can be removed completely by surgery. Treatment of lung cancer is often aimed toward relieving symptoms (palliation) rather than cure because of the presence of metastasis.

Most primary lung cancers arise from the bronchial epithelium. These cancers are collectively called bronchogenic carcinomas. Lung cancers can be classified according to their histologic cell type as small cell lung cancer (SCLC), epidermoid (squamous cell) cancer, adenocarcinoma, and large cell cancer. The last three types are now referred to as non–small cell lung cancer (NSCLC) because of their similar responses to treatment. Chapter 23 discusses the general mechanisms and processes of cancer development.

Metastasis (spread) of lung cancer occurs by direct extension through the blood and invading lymph glands and vessels. Tumors in the bronchial tubes can grow and obstruct the bronchus partially or completely. Tumors in other areas of lung tissue can grow so large that they can obstruct the airway by compressing it. Tumors in the edges of the lungs spread and can compress the alveoli, nerves, blood vessels, and lymph vessels. All of these problems interfere with oxygenation and tissue perfusion.

The patterns of metastasis depend on the type of tumor cell and the location of the tumor. Lung lymph nodes, as well as more distant lymph nodes, can be invaded.

Hematogenous (bloodborne) metastasis of lung cancer is due to invasion of blood vessels in the lungs. Emboli (tumor pieces) spread to distant body areas. These sites include the bone, liver, brain, and adrenal glands.

Additional manifestations, known as paraneoplastic syndromes, complicate certain lung cancers. The paraneoplastic syndromes are caused by hormones secreted by tumor cells. Paraneoplastic syndrome commonly occurs with SCLC. Table 32-4 lists the endocrine paraneoplastic syndromes that may occur, most commonly with SCLC.
Hydrocarbons also increases the risk for lung cancer. Air pollution that contains benzopyrenes and oxide, mustard gas, petroleum distillates, radiation, tar, nickel, to asbestos, beryllium, chromium, coal distillates, cobalt, iron oxide, mustard gas, petroleum distillates, radiation, tar, nickel, and uranium. Air pollution that contains benzopyrenes and hydrocarbons also increases the risk for lung cancer.

**Etiology and Genetic Risk**

Nonsmokers exposed to “passive,” or “secondhand,” smoke also have a greater risk for lung cancer than do nonsmokers who are minimally exposed to cigarette smoke. Passive smoke has many of the carcinogens found in inhaled, or “mainstream,” tobacco smoke.

Other risk factors for lung cancer include chronic exposure to asbestos, beryllium, chromium, coal distillates, cobalt, iron oxide, mustard gas, petroleum distillates, radiation, tar, nickel, and uranium. Air pollution that contains benzopyrenes and hydrocarbons also increases the risk for lung cancer.

### Table 32-4

<table>
<thead>
<tr>
<th>Ectopic Hormone</th>
<th>Manifestation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Adrenocorticotropic hormone (ACTH)</td>
<td>Cushing’s syndrome</td>
</tr>
<tr>
<td>Antidiuretic hormone</td>
<td>Syndrome of inappropriate antidiuretic hormone (SIADH)</td>
</tr>
<tr>
<td></td>
<td>Weight gain</td>
</tr>
<tr>
<td></td>
<td>General edema</td>
</tr>
<tr>
<td></td>
<td>Dilution of serum electrolytes</td>
</tr>
<tr>
<td>Follicle-stimulating hormone (FSH)</td>
<td>Gynecomastia</td>
</tr>
<tr>
<td>Parathyroid hormone</td>
<td>Hypercalcemia</td>
</tr>
<tr>
<td>Ectopic insulin hormone</td>
<td>Hypoglycemia</td>
</tr>
</tbody>
</table>

Staging of lung cancer is performed at diagnosis to assess the size and extent of the disease. These factors are correlated to survival rate. The staging of lung cancer is based on the TNM system (T, primary tumor; N, regional lymph nodes; M, distant metastasis). See Table 23-7 in Chapter 23 for a typical cancer staging system. Higher numbers represent later stages and less chance for cure or long-term survival.

**Incidence/Prevalence**

Lung cancers occur as a result of repeated exposure to inhaled substances that cause chronic tissue irritation or inflammation. Cigarette smoking is the major risk factor and is responsible for 85% of all lung cancer deaths (American Cancer Society, 2007). The risk for lung cancer is directly related to the total exposure to cigarette smoke as determined by the number of years of smoking and number of packs of cigarettes smoked per day (pack-years). Pipe and cigar smoking also increase risk. The incidence of lung cancer decreases when smoking stops, and after 15 years of smoking cessation, it approaches that of those who have never smoked. About 50,000 ex-smokers, however, develop lung cancer in the United States each year.

**Health Promotion and Maintenance**

**Primary prevention for lung cancer is directed at reducing tobacco smoking.** Educational strategies start with elementary school children to discourage them from beginning to smoke. Nurses are actively involved in encouraging nonsmokers not to begin to smoke, in promoting smoking cessation programs, and in establishing a smoke-free environment. Encourage nonsmokers to avoid passive, or secondhand, smoke by avoiding environmental exposure.

Teach workers in industrial settings about safety precautions, such as wearing specialized masks and protective clothing, to reduce occupational hazards. Encourage people who are at high risk for lung cancer development to seek frequent health examinations. Urge patients being treated for lung cancer to quit smoking. The actual diagnosis of the disease and its treatment time represent “teachable moments.”

Secondary prevention by early detection has not been considered feasible in the past with earlier detection not making a difference in long-term survival rates. New data from recent studies indicate that screening people at risk for lung cancer using annual spiral CT scans can detect cancers very early, at stage I, when cure is probable and long-term survival (longer than 5 years) is very likely (Henschke et al, 2006). See the Evidence-Based Practice box on p. **.

**NCLEX Examination Challenge**

The client is a 56-year-old woman who has smoked 3 packs of cigarettes per day from the time she was 14 years old to when she was 42, and then smoked 2 packs of cigarettes per day from age 42 to the present. How should the nurse calculate this client’s pack-year smoking history?

- A. 146 pack-years
- B. 112 pack-years
- C. 86 pack-years
- D. 42 pack-years

**Patient-Centered Collaborative Care**

**Assessment**

*History*

Ask the patient about risk factors, including smoking, hazards in the workplace, and warning signals (Table 32-5). Have the patient describe how many packs of cigarettes per day he or she has smoked and for how many years to determine the pack-year smoking history.

Ask about the presence of lung cancer manifestations, such as hoarseness, cough, sputum production, hemoptysis, shortness of breath, or change in endurance. Assessing for and documenting these manifestations provide information about the extent of nursing care and teaching the patient needs now.
The study was a well-designed controlled study without randomization. 

Commentary: Implications for Practice and Research. Although smoking cessation is the best prevention for most types of lung cancer, cigarette smoking is addictive and the habit is very difficult to stop. For those people who are at risk because of a smoking history or occupational exposure, annual spiral CT screening can be a life-saving method of secondary prevention. However, the procedure is expensive and few insurance companies support its use as a screening tool. Even when covered by insurance, participation in screening is low. Nurses should promote this screening method to people who are at risk for lung cancer. In addition, getting involved in processes, such as lobbying and petitioning insurance groups, to promote acceptance and coverage of this screening method also may lead to improved lung cancer survival rates.

### TABLE 32-5 Warning Signals Associated with Lung Cancer

- Hoarseness
- Change in respiratory pattern
- Persistent cough or change in cough
- Blood-streaked sputum
- Rust-colored or purulent sputum
- Frank hemoptysis
- Chest pain or chest tightness
- Shoulder, arm, or chest wall pain
- Recurring episodes of pleural effusion, pneumonia, or bronchitis
- Dyspnea
- Fever associated with one or two other signs
- Wheezing
- Weight loss
- Clubbing of the fingers

and can be used later to determine therapy effectiveness. Many manifestations are common and may have been present for years. Ask the patient to describe any recent changes in symptoms or if position affects symptoms.

Assess for chest pain or discomfort, which can occur at any stage of tumor development. Chest pain may be localized or on just one side and can range from mild to severe. Ask about any sensation of fullness, tightness, or pressure in the chest, which may suggest obstruction. A piercing chest pain or pleuritic pain may occur on inspiration. Pain radiating to the arm results from tumor invasion of nerve plexuses in advanced disease.

**Physical Assessment/Clinical Manifestations—Pulmonary**

Manifestations of lung cancer are often nonspecific and appear late in the disease process. Specific manifestations depend on the type and location of the tumor. Chills, fever, and cough may be related to pneumonitis or bronchitis that occur with obstruction. Assess sputum quantity and quality. Blood-tinged sputum may occur with bleeding from a tumor. Hemoptysis is a later finding in the course of the disease. If infection or necrosis is present, sputum may be purulent and copious.

Breathing patterns may be labored or painful. An obstructive breathing pattern may occur as prolonged exhalation alternating with periods of shallow breathing. Rapid, shallow breathing occurs with pleuritic chest pain and an elevated diaphragm. Inspiratory efforts are reduced in advanced disease. Look for and document the presence of abnormal retractions, the use of accessory muscles, flared nares, stridor, and asymmetric diaphragmatic movement on inspiration. Dyspnea and wheezing may be present with airway obstruction. Ask about the patient’s level of dyspnea at rest, with activity, and in the supine position (orthopnea). Determine how much the dyspnea interferes with his or her participation in ADLs, work, recreational activities, and family responsibilities. Ask the patient to compare his or her participation in activities during the past week with that of a month ago and a year ago.

Areas of tenderness or masses may be felt when palpating the chest wall. Increased vibrations felt on the chest wall (fremitus) indicate areas of the lung where airspaces are replaced with tumor or fluid. Fremitus is decreased or absent when the bronchus is obstructed. The trachea may be displaced from midline if a mass is present in the area.

Lung areas with masses sound dull or flat rather than hollow or resonant on chest percussion. Breath sounds may change with the presence of a tumor. Wheezes indicate partial obstruction of airflow in passages narrowed by tumors. Decreased or
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absent breath sounds indicate complete obstruction of an airway by a tumor or fluid. Increased loudness or sound intensity of the voice while listening to breath sounds indicates increased density of lung tissue from tumor compression. A pleural friction rub is heard when inflammation also is present.

Physical Assessment/Clinical Manifestations—Nonpulmonary

Many other systems can be affected by lung cancer and have changes at the time of diagnosis. Heart sounds may be muffled by a tumor or fluid around the heart (cardiac tamponade). Dysrhythmias may occur as a result of hypoxemia or direct pressure of the tumor on the heart. Cyanosis of the lips and fingertips or clubbing of the fingers may be present (see Figure 32-10).

Bones become thin with tumor invasion and break easily. The patient may have bone pain or pathologic fractures. Handle the patient carefully. Thin bones can fracture with little pressure and without trauma. Even heavy coughing can break a rib.

Late manifestations of lung cancer usually include fatigue, weight loss, anorexia, dysphagia, and nausea and vomiting. Superior vena cava syndrome may result from tumor pressure in or around the vena cava. This syndrome is an emergency (see Chapter 24) and requires immediate medical attention. Lethargy and somnolence may develop, and the patient may have confusion or personality changes as a result of brain metastasis. Bowel and bladder tone or function may be affected by tumor spread to the spine and spinal cord.

Psychosocial Assessment

The poor prognosis for lung cancer has made it a much-feared disease. Lung cancer manifestations, especially dyspnea, add to the patient’s fear and anxiety. The patient with a history of cigarette smoking may feel guilt and shame. Convey acceptance, and interact with the patient in a nonjudgmental way. Encourage the patient and family to express their feelings about the possible diagnosis of lung cancer.

Few patients with stage III or higher lung cancer are cured or live longer than 5 years after diagnosis. Many are given limited palliative treatment for symptom relief. Fear of pain and death is common.

Other Diagnostic Assessment

The diagnosis of lung cancer is made by direct examination of cancer cells. Cytologic examination of early morning sputum specimens may identify tumor cells; however, cancer cells may not be present in the sputum. When pleural effusion is present, fluid can be obtained by thoracentesis for cytology.

Most commonly, lung lesions are first identified on chest x-rays. Computed tomography (CT) examinations are then used to identify the lesions more clearly. Usually, the entire chest is scanned at 5- to 10-mm slices and the suspicious areas are then scanned at 1- to 2-mm slices for the highest resolution.

Fiberoptic bronchoscopy provides direct visibility of the tracheobronchial tree. Specimens and bronchial brushings can be obtained with this technique, especially when lesions are located within or close to an airway. Needle biopsy during bronchoscopy may be used to obtain cancer cells.

A thoracoscopy may be performed through a video-assisted thoracoscopic entering the chest cavity via small incisions through the chest wall. This procedure allows direct visualization of the lung tissue. To identify metastasis in mediastinal lymph nodes, a mediastinoscopy may be performed through a small chest incision.

Other diagnostic studies may be needed to determine how widely the cancer has spread. Such tests include needle biopsy of lymph nodes, direct surgical biopsy, and thoracentesis with pleural biopsy. Magnetic resonance imaging (MRI) and radiouclide scans of the liver, spleen, brain, and bone help determine the location of metastatic tumors. Pulmonary function tests and arterial blood gas (ABG) analysis help determine the overall respiratory status. Positron emission tomography (PET) scanning is becoming the most thorough way to locate metastases. Together, these tests help determine the extent of the cancer and the best methods to treat it.

DECISION-MAKING CHALLENGE

Coordination of Care

The patient is a 51-year-old man who works as a welder and has just been diagnosed with stage II lung cancer. He smoked heavily as a younger man and was able to stop smoking for 5 years when he was diagnosed with Hodgkin’s lymphoma 15 years ago. He was successfully treated with chemotherapy and radiation at that time and has not had a recurrence of the lymphoma. Ten years ago he started smoking again and now smokes two packs per day. He and his wife are distraught at the diagnosis, and he is verbally blaming himself for the lung cancer, saying he knows he is going to die.

1. What are this man’s risk factors for lung cancer?
2. What should you tell him about his chances of dying?
3. Should you approach him at this distressing time about quitting smoking? Why or why not?

Interventions for Cure

Interventions for the patient with lung cancer can be aimed at curing the disease, increasing survival time, and enhancing quality of life through palliation. Both nonsurgical and surgical interventions are used to achieve these aims. Some patients with lung cancer may undergo interventions for all three aims at different stages in the disease process. Currently, cures are most likely for patients who undergo treatment for stage I or II disease. Cure is rare for patients who undergo treatment for stage III or IV disease, although survival time is increasing, especially for non–small cell lung cancer (Tyson, 2007).

Nonsurgical Management

Chemotherapy is often the treatment of choice for lung cancers, especially small cell lung cancer (SCLC). It may be used alone or as adjuvant therapy in combination with surgery for non–small cell lung cancer (NSCLC). The exact combination of drugs used varies depending on the response of the tumor and the overall health of the patient; however, most include platinum-based agents (Tyson, 2007).

Side effects that occur with chemotherapy for lung cancer include chemotherapy-induced nausea and vomiting (CINV), alopecia (hair loss), open sores on mucous membranes (mucositis), immunosuppression, anemia, thrombocytopenia (decreased numbers of platelets), and peripheral neuropathy (PN). Some of these side effects are presented briefly below. Consult Chapter 24 for a thorough discussion of the nursing care needs for patients who have these side effects.

Reassure patients that hair loss is temporary. Hair regrowth begins about 1 month after chemotherapy is com-
pleted. Hair loss can be disguised by the use of wigs, scarves, turbans, and caps.

The chemotherapy agents used for lung cancer treatment are *emetogenic* (inducing nausea and vomiting). Many effective antiemetic drugs are available. Usually one or more antiemetics are given before and after chemotherapy. Drugs used to control chemotherapy-induced nausea and vomiting are listed in Chart 24-5 in Chapter 24. Patient response to antiemetic therapy varies, and the drug combinations are individualized for best effect.

Frequent mouth assessment and oral hygiene are key in managing mucositis. Stress the importance of good, frequent oral hygiene, including tooth cleaning and mouth rinsing. Teach patients to use a soft-bristled toothbrush or disposable mouth sponges and to avoid using dental floss and water-pressure gum cleaners.

Immunosuppression, which greatly increases the risk for infection, is the major dose-limiting side effect of chemotherapy for lung cancer. Immunosuppression can be managed by the use of biological response modifiers (BRMs) to stimulate bone marrow production of immune system cells. Teach the patient and family about precautions to take to reduce the patient’s chances of developing an infection (see Chart 24-5 in Chapter 24). (See Chapter 24 for more information about chemotherapy and associated nursing care.)

Targeted therapy is now becoming more common in the treatment of later stage lung cancer. As discussed in Chapter 24, these agents take advantage of one or more differences in cancer cell growth or metabolism that is either not present or only slightly present in normal cells. These differences are a result of specific gene expression in cancer cells. Agents used as targeted therapies often are antibodies that work to disrupt cancer cell division in one of several ways. Some of these drugs “target” and block growth factor receptors, especially the epithelial growth factor receptors (EGFR) or the vascular endothelial growth factor receptors (VEGFR). When a lung cancer cell’s growth depends on having the growth factors bind to their specific receptors, blocking the receptors at least slows the cancer cell’s growth. Two agents most often used for targeted therapy of certain types of non–small cell lung cancer are erlotinib (Tarceva), an oral drug, and bevacizumab (Avastin), which is given IV. Neither drug is used alone as therapy for lung cancer, and bevacizumab tends to intensify common chemotherapy side effects.

Radiation therapy can be an effective treatment for locally advanced lung cancers confined to the chest. Best results are seen when radiation is used in addition to surgery or chemotherapy. Radiation may be performed before surgery to shrink the tumor and make resection easier.

Usually radiation therapy for lung cancer is performed daily for a 5- to 6-week period. Only the areas thought to have cancer are positioned in the radiation path. The immediate side effects of this treatment are skin irritation and peeling, fatigue, nausea, and taste changes. Some patients have esophagitis during therapy, making adequate nutrition more difficult. Teach patients to eat foods that are soft, bland, and high in calories. Consult with a nutritionist to provide a list of foods that are easier to swallow and nutritious. Suggest that the patient drink liquid nutrition supplements, such as Ensure or Boost, between meals to maintain weight and energy levels. Narrowing of the esophagus can occur as a late response to radiation therapy for lung cancer and may require dilation or reconstructive surgery.

Skin care in the radiation-treated area can be difficult. If the area has been marked with a dye to outline the areas for radiation, instruct the patient not to wash off the markings. The use of ink or dye markings is rare, with most cancer centers using small permanent tattoos to mark the area. Instruct patients not to use lotions or ointments on the skin of the chest unless the radiologist prescribes them. Because skin in the radiation path is more sensitive to sun damage, advise patients to avoid direct skin exposure to the sun during treatment and for at least 1 year after radiation is completed. See Chapter 24 for other nursing care issues associated with radiation therapy.

**Photodynamic therapy** (PDT) may be used to remove small bronchial tumors when they are accessible by bronchoscopy. Once used only for palliation, this therapy is now used also for cure of select lung cancers. The patient is first injected with an agent that sensitizes cells to light. This drug enters all cells but leaves normal cells more rapidly than cancer cells. Usually, within 48 to 72 hours, most of the drug has collected in high concentrations in cancer cells. At this time, the patient goes to the operating room where, under anesthesia and intubation, a laser light is focused on the tumor. The light activates a chemical reaction within those cells retaining the sensitizing drug that induces irreversible cell damage. Some cells die and slough immediately; others continue to slough for several days. The photosensitizing drug has many effects that require special patient teaching and care both before and after the laser treatment (Collins & Garner, 2007). Chapter 24 describes these issues in detail. In addition to these general care issues, when PDT is used in the airways, the patient usually requires a stay in the ICU for airway management. The sloughing tissue can block the airway as can airway edema from the inflammatory response of the tissues. In addition, the patient is at risk for bronchial hemorrhage, fistula formation, and hemoptysis. A complicating factor in caring for patients who have undergone bronchial PDT is the fact that the patient is now supersensitive to light and will remain so for 30 to 90 days. Thus special precautions are needed along with environmental manipulation to keep the patient safe during his or her hospital stay and during the next 3 months. Chapter 24 discusses environmental safety in detail, and Chart 24-11 in Chapter 24 presents points to teach the patient and family.

**Surgical Management**

Surgery is the main treatment for stage I and stage II NSCLC. Total removal of a non–small cell primary lung cancer is undertaken in hope of achieving a cure. If complete resection is not possible, the surgeon removes the bulk of the tumor. The specific surgery depends on the stage of the cancer and the patient’s overall health and functional status. Lung cancer surgery may involve removal of the tumor only, removal of a lung segment, removal of a lobe (lobectomy), or removal of the entire lung (pneumonectomy). These procedures can be performed by open thoracotomy or by thoracoscopy with minimally invasive surgery in select patients.

**Preoperative care.** The goals of care before surgery are to relieve anxiety and promote the patient’s participation (see Chapter 16 for routine preoperative care). Encourage the patient to express fears and concerns, reinforce the surgeon’s explanation of the surgical procedure, and provide education related to what is expected after surgery. Teach about the probable location of the surgical incision or thoracoscopy...
openings, shoulder exercises, and the chest tube and drainage system (except after pneumonectomy).

Operative procedures. Three types of incisions can be made depending on the location of the cancer: posterolateral, anterolateral, and median sternotomy (Figure 32-13). The incisions are large and are held open with retractors during surgery, contributing to pain after surgery.

A segmental resection (segmentectomy) is a lung resection that includes the bronchus, pulmonary artery and vein, and tissue of the involved lung segment or segments, which are divisions of lobes. A wedge resection is removal of the peripheral portion of small, localized areas of disease. A lobectomy is the removal of an entire lung lobe. A pneumonectomy is the removal of an entire lung, including all blood vessels. The bronchus to that lung is severed and sutured.

Removal of a lobe or even an entire lung can be accomplished through video-assisted thorascopic surgery (VATS) for select patients. The procedure involves making three small incisions in the chest for placement of the thoroscope and other instruments. These same openings are used later for placement of drains and chest tubes. The lung section, lobe, or lung is isolated from the airway, which is surgically closed. The lobe or the lung is closed off from the rest of the lung using a double-stapling technique. Then the tissue is encapsulated in an impermeable bag to prevent leakage of tumor tissue and possible seeding of the cancer and is then removed whole through one of the small incisions.

Postoperative care. Care after surgery for patients who have undergone thoracotomy (except for pneumonectomy) requires closed-chest drainage to drain air and blood that accumulate in the pleural space. A chest tube, a drain placed in the pleural space to restore intrapleural pressure, allows re-expansion of the lung (Figure 32-14). The chest tube also prevents air and fluid from returning to the chest. The drainage system consists of one or more chest tubes or drains, a collection container placed below the chest level, and a water seal to keep air from entering the chest. The drainage system may be a stationary, disposable, self-contained system (Figure 32-15) or a smaller, portable, disposable, self-contained system (Figure 32-16). The basic principles of gravity and pressure are the same with both systems. The nursing care priorities for the patient with a chest tube are to ensure the integrity of the system, promote comfort, ensure chest tube patency, and prevent complications.

Chest tube placement and care. The tip of the tube used to drain air is placed near the front lung apex (see Figure 32-14). The tube that drains liquid is placed on the side near the base of the lung. After lung surgery, two tubes, anterior and posterior, are used. The puncture wounds are covered with airtight dressings.

The chest tube is connected to about 6 feet of tubing that leads to a collection device placed several feet below the chest. The tubing allows the patient to turn and move without pulling on the chest tube. Keeping the collection device below the chest allows gravity to drain the pleural space. When two chest tubes are inserted, they are joined by a Y-connector near the patient’s body; the 6 feet of tubing is attached to the Y-connector.

Stationary chest tube drainage systems usually use a water seal mechanism that acts as a one-way valve to prevent air or
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liquid from moving back into the chest cavity. The Pleur-Evac system is a common device using a one-piece disposable plastic unit with three chambers. The three chamber are connected to one another. The tube(s) from the patient is(are) connected to the first chamber in the series of three. This chamber is the

Fig. 32-15  - Left, The Pleur-Evac drainage system, a commercial three-bottle chest drainage device. Right, Schematic of the drainage device.

Fig. 32-16  - A portable chest drainage system.

drainage collection container. The second chamber in the series is the water seal to prevent air from moving back up the tubing system and into the chest. The third chamber, when suction is applied, is the suction regulator.

In setting up the system, chamber one (nearest to the patient) does not at first have fluid in it. The tubing from the patient penetrates shallowly into this chamber, as does the tube connecting chamber one with chamber two.

Chamber one collects the fluid draining from the patient. This fluid is measured hourly during the first 24 hours. The fluid in chamber one must never fill to the point that it comes into direct contact with either the tube draining from the patient or the tube connecting this chamber to chamber two. If the tubing from the patient enters the fluid, drainage stops and can lead to a tension pneumothorax.

Chamber two is the water seal that prevents air from entering the patient’s pleural space. Air from the pleural space also enters chamber one but moves immediately to chamber two through the connecting tube. This tube must always be under the water level in chamber two to prevent air from returning to the patient. The tube acts as a one-way valve, allowing air to move into the water and preventing air in this chamber from re-entering the tube. This action is similar to blowing air into a straw that is placed in a glass of liquid. A person can easily blow air through the straw into the liquid, as seen by bubbles leaving the straw and going up to the top of the liquid. Because air is lighter than liquids, when the person sucks on the straw, he or she can pull air back up into the straw only after the water has first been pulled up the straw. Thus, as long as the tip of the tube from the first chamber is under water in the water seal chamber, air that has escaped from the patient’s chest tube cannot re-enter the patient.
The bubbling of the water in chamber two indicates air drainage from the patient. Bubbling is usually seen when intrathoracic pressure is greater than atmospheric pressure, such as when the patient exhales, coughs, or sneezes. When the air in the pleural space has been removed, bubbling stops. A blocked or kinked chest tube also can cause bubbling to stop. Excessive bubbling in the water seal chamber (chamber two) may indicate an air leak. The water in the long tube of the second chamber rises and falls slightly with the patient’s respiratory cycle, a process called *tidaling*. A rise of 2 to 4 inches during inhalation and a fall during exhalation are normal. An absence of fluctuation may mean that the chest tube is obstructed, the expanded lung has blocked the eyelets of the chest tube, or no more air is leaking into the pleural space.

Chamber three is the suction control of the system and has three connections: one from the second chamber; a long, open tube dipped into the water to serve as an air vent; and a short tube connecting to the suction unit. Suction enhances the pressure difference between the pleural space and the drainage system, causing the pressure to drop inside the system by 15 to 20 cm. Although the amount of suction generated by the suction unit can be increased, the amount of suction in the system is determined not by the suction unit but by the depth of the open tube in the water. The health care provider prescribes the amount of water to be placed and maintained in this chamber. While suction is applied, gentle bubbling is seen in this chamber.

Chart 32-13 summarizes best safety practices when caring for a patient with a water seal chest tube drainage system. Check hourly to ensure the sterility and patency of any chest drainage system. Tape tubing junctions to prevent accidental disconnections, and keep an occlusive dressing at the chest tube insertion site. Keep sterile gauze at the bedside to cover the insertion site immediately if the chest tube becomes dislodged. Also keep padded clamps at the bedside for use if the management of the chest tube drainage system is needed.

**Management of Chest Tube Drainage Systems**

**Patient**
- Ensure that the dressing on the chest around the tube is tight and intact. Depending on agency policy and the surgeon’s preference, reinforce or change loose dressings.
- Assess for difficulty breathing.
- Assess breath sounds for each lung.
- Check alignment of trachea.
- Check tube insertion site for condition of the skin. Palpate area for puffiness or crackling that may indicate subcutaneous emphysema.
- Observe site for signs of infection (redness, purulent drainage) or excessive bleeding.
- Check to see if tube “eyelets” are visible.
- Assess for pain and its location and intensity, and administer drugs for pain as prescribed.
- Assist patient to deep breathe, cough, perform maximal sustained inhalations, and use incentive spirometry.
- Reposition the patient who reports a “burning” pain in the chest.

**Drainage System**
- Do not “strip” the chest tube.
- Keep drainage system lower than the level of the patient’s chest.
- Keep the chest tube as straight as possible, avoiding kinks and dependent loops.
- Ensure the chest tube is securely taped to the connector and that the connector is taped to the tubing going into the collection chamber.
- Assess bubbling in the water seal chamber; should be gentle bubbling on patient’s exhalation, forceful cough, position changes.
- Assess for “tidaling.”
- Check water level in the water seal chamber, and keep the level at that recommended by the manufacturer.
- Check water level in suction control chamber, and keep at the level prescribed by the surgeon.
- Clamp the chest tube only for brief periods to change the drainage system or when checking for air leaks.
- Check and document amount, color, and characteristics of fluid in the collection chamber, as often as needed according to the patient’s condition and agency policy.
- Empty collection chamber or change the system before the drainage makes contact with the bottom of the tube.
- When sample of drainage is needed for culture or other laboratory test, obtain it from the chest tube; after cleansing chest tube, use a 20-gauge (or smaller) needle and draw up specimen into a syringe.

**Immediately Notify Physician or Rapid Response Team for:**
- Tracheal deviation.
- Sudden onset or increased intensity of dyspnea.
- Oxygen saturation less than 90%.
- Drainage greater than 70 mL/hr.
- Visible eyelets on chest tube.
- Chest tube falls out of the patient’s chest (first, cover the area with dry, sterile gauze).
- Chest tube disconnects from the drainage system (first, put end of tube in a container of sterile water and keep below the level of the patient’s chest).
- Drainage in tube stops (in the first 24 hours).
drainage system is interrupted. Position the drainage tubing to prevent kinks and large loops of tubing, which can block drainage and prevent lung re-expansion.

Manipulation of the chest tube should be kept to a minimum. Do not vigorously “strip” the chest tube because this can create up to −400 cm of water negative pressure and damage lung tissue. If any tube manipulation is needed, gentle hand-over-hand “milking” of the tube, with stopping between each hand hold, is used to move blood clots and prevent obstruction (Halm, 2007). Follow agency policies and guidelines on this action.

Assess the patient’s respiratory status and document the amount and type of drainage hourly. Usually the drainage in chamber one is not emptied unless the container is so full that the fluid is in danger of coming into contact with the chest drainage tube. The self-contained systems have calibrations on the collection chamber. Record the amount of hourly drainage. Notify the physician of drainage if more than 100 mL/hr occurs. After the first 24 hours, assess drainage at least every 8 hours.

Check the water seal chamber for unexpected bubbling created by an air leak in the system. Bubbling is normal during forceful expiration or coughing because air in the chest is being expelled. Continuous bubbling indicates an air leak that must be identified. Notify the physician if bubbling occurs continuously in the water seal chamber. On the physician’s prescription, gently apply a padded clamp briefly on the drainage tubing close to the occlusive dressing. If the bubbling stops, the air leak may be at the chest tube insertion site or within the chest, requiring physician intervention. Air bubbling that does not cease when a padded clamp is applied indicates that the air leak is between the clamp and the drainage system. Release the clamp as soon as this assessment is made.

Mobile or portable chest tube drainage systems are “dry” chest drainage systems that do not use water to form a seal to prevent air from re-entering the patient’s lung through the chest tube. Instead, these lightweight devices use a dynamic control “flutter” valve that prevents backflow of air. The flutter valve is a soft rubber tube surrounded by a harder plastic tube. When the patient exhales, air is forced from the chest cavity into the chest tube, under pressure. This pressure forces the soft flutter valve open and air moves into the harder surrounding tube shell (which has a vent for air). When the patient inhales, creating negative pressure in the chest tube, the soft sides of the flutter valve collapse on themselves (like the sides of a deflated balloon when a person sucks on the mouthpiece instead of blowing into the mouthpiece), closing the one-way valve.

Although previously recommended only for use in patients who had a simple, uncomplicated pneumothorax, some mobile chest tube drainage units have larger collection chambers that increase their use. These portable units allow the patient to ambulate more freely and even go home with chest tubes still in place (Carroll, 2005).

**NCLEX EXAMINATION CHALLENGE**
The client is 1 day post-op after a right lower lobectomy for stage II lung cancer and has two chest tubes in place. He is grimacing and tells you he has intense burning pain in his lower chest. You note that there is no bubbling on exhalation in the water seal chamber. What is your best first action?

A. Immediately notify either the Rapid Response Team or the thoracic surgical resident.
B. Administer the prescribed opioid analgesic immediately, and then assess the chest tube system.
C. No action is needed because these responses are normal for the first post-op day after lobectomy.
D. Assist the client to a side-lying position and re-assess the water seal chamber for bubbling.

**Pain management.** Most patients experience intense pain after an open thoracotomy for at least the first 24 hours. It is considerably less for the patient after lung cancer surgery using minimally invasive techniques. However, pain control is needed in either case for patient comfort and to assist him or her to participate in techniques to reduce the risk of postoperative complications (see Chapter 18). Administer the prescribed drugs for pain, and assess the patient’s responses to them. Teach patients using patient-controlled analgesia (PCA) devices to self-administer the drug before pain intensity becomes too severe. Monitor vital signs before and after giving opioid analgesics, especially for the patient who is not being mechanically ventilated. Plan care activities around the timing of analgesia to reduce the stimulation of additional pain.

**Respiratory management.** Immediately after surgery the patient is mechanically ventilated. See Chapter 34 for nursing care of the patient receiving mechanical ventilation.

Once the patient is breathing on his or her own, the priorities are to maintain a patent airway, ensure adequate ventilation, and prevent complications. Assess the patient at least every 2 hours for adequacy of ventilation and gas exchange. Check the alignment of the trachea. Assess oxygen saturation and the rate and depth of respiration. Listen to breath sounds in all lobes on the nonoperative side, particularly noting the presence of crackles. Assess the oral mucous membranes for cyanosis and the nail beds for rate of capillary refill. Perform oral suctioning as necessary.

Usually the patient receives oxygen by mask or nasal cannula for the first couple of days after surgery. Warm and humidify the oxygen. Assist the patient to a semi-Fowler’s position or up in a chair as soon as possible. Encourage him or her to use the incentive spirometer every hour while awake. If coughing is permitted, help him or her cough by splinting any incision and ensuring that the chest tube does not pull with movement. Ensuring that pain is handled properly increases the patient’s ability to cough and deep breathe effectively.

**Pneumonectomy care.** After pneumonectomy, the pleural cavity on the affected side is an empty space. The surgeon sometimes inserts a clamped chest tube for only a day because serous fluid may then accumulate in the empty space and create adhesions, which reduce mediastinal shift toward the affected side. Closed-chest drainage is not usually used.

Complications of a pneumonectomy can include empyema and the development of a bronchopleural fistula. Positioning of the patient after pneumonectomy varies according to surgeon preference and the patient’s comfort. Some surgeons want the patient placed on the nonoperative side immediately after a pneumonectomy to reduce stress on the bronchial stump incision. Others prefer that the patient be placed on the operative side to allow fluids to fill in the space formerly taken up by the lungs.
the fluid can rapidly re-form in the pleural space. When fluid space to collect the fluid.

Th e goal of treatment is to remove pleural fluid and prevent its formation. Thoracentesis and pleurodesis are used when pleural effusion is a problem for the patient with lung cancer. The excess fluid increases dyspnea, discomfort, and the risk for infection. The goal of treatment is to remove pleural fluid and prevent its formation. Thoracentesis is fluid removal by suction after the placement of a large needle or catheter into the intrapleural space. Fluid removal temporarily relieves hypoxia; however, the fluid can rapidly re-form in the pleural space. When fluid development is continuous and uncomfortable, a continuously draining catheter may be placed into the intrapleural space to collect the fluid.

Another technique to relieve pleural effusion is to insert a chest tube to drain the fluid and to instill a sclerosing agent, an agent that is an irritant and causes inflammation. The aim of this technique is to cause a pleurodesis (an inflammatory response that causes the pleura to stick to the chest wall). If pleurodesis occurs, it prevents formation of effusion fluid. Liquid sclerosing agents for pleurodesis are instilled after some of the effusion fluid has been removed. The patient is asked to assume a variety of positions to ensure the widest spread of the fluid within the pleural space. Talc pleurodesis involves using a thoroscope to deliver talc (in the form of a powder) to the area where the fluid forms. This procedure also causes inflammation and thickening that reduce the formation of effusion fluid.

These procedures can be performed under local anesthesia at the bedside or in an operating room. Usually the patient is also given an analgesic or sedative. Once the sclerosing agent is instilled, the chest tube is clamped to prevent drainage of the agent. Chart 32-14 reviews best practices for care of the patient undergoing pleurodesis.

Dyspnea management is needed because the patient with lung cancer tires easily and is often most comfortable resting in a semi-Fowler’s position. Dyspnea is reduced with oxygen, use of a morphine drip, and positioning for comfort. The severely dyspneic patient may be most comfortable sitting in a lounge chair or reclining chair.

Pain management may be needed for chest pain and pain radiating to the arm. With bone metastasis, the patient may also have bone pain. Perform a complete pain assessment with attention to onset, intensity, quality, duration, and the patient’s description of the pain. The goal of therapy is to help the patient to be as pain-free and as comfortable as possible.

Pharmacologic management with opioid drugs as oral, parenteral, or transdermal preparations is needed. Nonpharmacologic measures, such as positioning, hot or cold compresses, distractions, and guided imagery, may also be helpful. Prescribed analgesics are most effective when given around the clock. Additional PRN analgesics are used for breakthrough pain. Ongoing assessment and evaluation of the effectiveness of the pain control regimen are primary nursing responsibilities.

Hospice care can be beneficial for the patient in the terminal phase of lung cancer. Hospice programs provide support to the terminally ill patient and the family by meeting physical and psychosocial needs, adjusting the palliative care regimen as needed, making home visits, and providing volunteers for errands and respite care. (See Chapter 9 for a more complete discussion of end-of-life issues.) The American Cancer Society may also be able to provide assistance through support groups for patients and families or through the use of equipment, such as a hospital bed or bedside commode.

Chart 32-14  BEST PRACTICE FOR PATIENT SAFETY & QUALITY CARE

Care of the Patient Undergoing Pleurodesis

- Reinforce explanation of the pleurodesis, and inform the patient that drugs will be used to promote comfort before the procedure. (The physician may administer IV analgesia/sedation immediately before the procedure.)
- Ensure that the chest tube is clamped after instillation of the sclerosing agent.
- Monitor vital signs and respiratory status at the completion of the procedure and then at least every 30 minutes until the effects of the IV drugs have dissipated.
- Thereafter, monitor vital signs every 4 hours for 24 hours. (The patient may have a low-grade fever. Pleurodesis creates pleuritis between the visceral and parietal layers, thus preventing further fluid collection.)
- If a rotation schedule is ordered, assist the patient to the correct position for appropriate time frames and provide reassurance.
- Unclamp the chest tube after completion of the rotation schedule or at the specified time.
- Assess chest tube drainage, and document the amount and character of the drainage.
- Perform a complete respiratory assessment every 2 hours, and observe for manifestations of distress, including those of pneumothorax (rapid respiration, reduced breath sounds on the affected side, dyspnea, decreased oxygen saturation, tracheal deviation, prominence of one side of the chest).
- Analgesics may be administered as needed to promote comfort.
- When drainage has decreased (<150 mL in 12-24 hr), the physician may remove the chest tube. Maintain an occlusive dressing at the insertion site for a minimum of 48 hours.
**Health Promotion and Maintenance**

- Teach all patients who smoke the warning signs of lung cancer.
- Encourage all patients older than 50 years and anyone with a chronic obstructive respiratory problem to receive a yearly influenza vaccination.
- Assist patients interested in smoking cessation to find an appropriate smoking cessation program.

**Safe and Effective Care Environment**

- Ensure proper function of chest tube drainage equipment.
- Protect the patient with cystic fibrosis from hospital-acquired pulmonary infections.
- Ensure proper function of chest tube drainage equipment.

**Physiological Integrity**

- Monitor the rate and depth of respiration at least every hour for any patient with hypercarbia and CO₂ narcosis who is receiving oxygen by mask or nasal cannula.
- Document any known specific allergies that have respiratory manifestations.
- Assess the airway and breathing effectiveness for any patient who experiences shortness of breath or any change in mental status.
- Apply oxygen to anyone who is hypoxicemic.
- Ensure that oxygen therapy delivered to the patient is humidified (and warmed, when possible).

**Psychosocial Integrity**

- Assess the degree to which breathing problems interfere with the patient's ability to perform ADLs, work, and leisure time activities.
- Encourage the patient and family to express their feelings regarding the diagnosis of cancer or the treatment regimen.
- Allow patients to verbalize feelings about changes in appearance resulting from cancer therapy.
- Explain all diagnostic procedures, restrictions, and follow-up care to the patient scheduled for tests.
- Help patients use strategies to improve their appearance when alopecia occurs.
- Refer patients and family members to local cancer resources and support groups.

**HUMAN NEEDS NURSING CARE REVIEW**

**What might you NOTICE if the patient is experiencing inadequate oxygenation and tissue perfusion as a result of chronic obstructive respiratory problems?**

- Respiration rapid and shallow
- Decreased oxygen saturation by pulse oximetry
- Skin cyanosis or pallor (lighter-skinned patients)
- Cyanosis or pallor of the lips and oral mucous membranes (in patients of any skin color)
- Tachycardia
- Patient appears to work hard to inhale and exhale
- Patient is restless or anxious
- Patient's general appearance is thin compared with height
- Muscles of the neck appear thick
- Arm and leg muscles appear thin
- Fingers are clubbed
- Chest is barrel-shaped (has a round rather than an oval shape with the front to back depth increased)
- Ribs are spaced more than a finger-breadth apart.

**What should you INTERPRET and how should you RESPOND to a patient experiencing inadequate oxygenation and tissue perfusion as a result of an acute critical respiratory problem?**

**Perform and interpret physical assessment, including:**

- Taking vital signs
- Auscultating all lung fields
- Monitoring oxygen saturation by pulse oximetry
- Checking the accuracy of pulse oximetry readings
- Assessing cognition (Mini-Mental State Examination)

**GET READY FOR THE NCLEX EXAMINATION!**

**Key Points**

Review these Key Points for each NCLEX Examination Client Needs Category.

**Safe and Effective Care Environment**

- Avoid high liter flow rates of oxygen for patients with COPD.
- Ensure that no open flames or combustion hazards are in rooms where oxygen is in use.
- Protect the patient with cystic fibrosis from hospital-acquired pulmonary infections.
- Ensure proper function of chest tube drainage equipment.

**Health Promotion and Maintenance**

- Teach patients who come into contact with inhalation irritants in their workplaces or leisure time activities to use a mask to avoid respiratory contact with these substances.
- Teach anyone who smokes that smoking increases the risk for development of many pulmonary problems.
- Assist patients interested in smoking cessation to find an appropriate smoking cessation program.
- Encourage older adults who are confined to bed for any reason or who are recovering from surgery to turn, cough, and deep breathe at least every 2 hours.
- Encourage all patients older than 50 years and anyone with a respiratory problem to receive a yearly influenza vaccination.
- Teach all patients who smoke the warning signs of lung cancer.

**Physiological Integrity**

- Assessing for the presence and characteristic of sputum production
- Assessing the patient’s ability to cough and clear the airway

**Interpret laboratory values, including:**

- Elevated red blood cell count, hematocrit, and hemoglobin
- Elevated white blood cell count
- Arterial blood gas values: pH less than 7.35, HCO₃⁻ more than 24 mm Hg, Paco₂ more than 45 mm Hg; Pao₂ less than 80 mm Hg

**Respond by:**

- Assisting the patient to an upright position, with arms resting on a table or armrests
- Performing or assisting the patient to perform chest physiotherapy/pulmonary hygiene
- Ensuring that oxygen delivery is kept low enough to maintain respirations of no fewer than 16 breaths per minute
- Prioritizing and pacing activities to prevent fatigue
- Administering prescribed inhaled drugs
- Administering respiratory therapy treatments or collaborating with the respiratory therapist to administer these treatments
- Re-assessing respiratory status after respiratory therapy treatment
- Ensuring a fluid intake of at least 3 liters per day

**On what should you REFLECT?**

- Observe patient for evidence of improved oxygenation (see Chapter 29)
- Think about what may have made the patient’s dyspnea worse and what steps could be taken to prevent a similar episode
- Think about what patient teaching focus could help reduce the intensity of dyspnea in the future

**Psychosocial Integrity**

- Assess the degree to which breathing problems interfere with the patient’s ability to perform ADLs, work, and leisure time activities.
- Encourage the patient and family to express their feelings regarding the diagnosis of cancer or the treatment regimen.
- Allow patients to verbalize feelings about changes in appearance resulting from cancer therapy.
- Explain all diagnostic procedures, restrictions, and follow-up care to the patient scheduled for tests.
- Help patients use strategies to improve their appearance when alopecia occurs.
- Refer patients and family members to local cancer resources and support groups.
SELECTIONS BIBLIOGRAPHY

Asterisk indicates a classic or definitive work on this subject.


Halm, M. (2007). To strip or not to strip: Physiological effects of chest tube skin in the radiation path (see Chart 24-2 in Chapter 24).


