CHAPTER

29

Success is the plateau that one rests upon to take a breath and look down from upon the straight and difficult path, but one does not climb upon a plateau.

—Josephine Preston Peabody

Nursing Management

Obstructive Pulmonary Diseases

Jane Steinman Kaufman

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LEARNING OUTCOMES

1. Describe the etiology, pathophysiology, clinical manifestations, and collaborative care of asthma.
2. Describe the nursing management of the patient with asthma.
3. Differentiate among the etiology, pathophysiology, clinical manifestations, and collaborative care of the patient with chronic obstructive pulmonary disease (COPD).
4. Describe the effects of cigarette smoking on the lungs.
5. Identify the indications for O_2 therapy, methods of delivery, and complications of O_2 administration.
6. Explain the nursing management of the patient with COPD.
7. Describe the pathophysiology, clinical manifestations, collaborative care, and nursing management of the patient with cystic fibrosis.
8. Describe the pathophysiology, clinical manifestations, collaborative care, and nursing management of the patient with bronchiectasis.

KEY TERMS

α_1-antitrypsin deficiency, p. 611
asthma, p. 588
bronchiectasis, p. 635
chest physiotherapy, p. 623
chronic bronchitis, p. 610
chronic obstructive pulmonary disease (COPD), p. 610
cor pulmonale, p. 613
cystic fibrosis, p. 631
emphysema, p. 610
O_2 toxicity, p. 621
postural drainage, p. 624
pursed-lip breathing, p. 623

Imagine needing to consciously think about every breath that you take for minutes, hours, or days. Many individuals with obstructive lung disease have this experience. Approximately 30 million adult Americans are living with chronic obstructive pulmonary disease (COPD) or asthma.[1] Obstructive pulmonary disease, the most common chronic lung disease, includes diseases characterized by increased resistance to airflow as a result of airway obstruction or airway narrowing. Types of obstructive lung diseases are asthma, COPD, cystic fibrosis, and bronchiectasis. *Asthma* is a chronic inflammatory lung disease that results in variable episodes of airflow obstruction, but it is usually reversible. *COPD* is an obstructive pulmonary disease with progressive limitation in airflow that is not fully reversible.[2-4]

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The patient with asthma has variations in airflow over time, usually with normal lung function between exacerbations, whereas the limitation in expiratory airflow in the patient with COPD is generally more constant. The pathology of asthma and response to therapy differ from COPD. However, the patient with a diagnosis of obstructive pulmonary disease may have features of both asthma and COPD. Patients with asthma who have less responsive reversible airflow obstruction are difficult to distinguish from COPD patients.[2]

Cystic fibrosis, another form of obstructive pulmonary disease, is a genetic disorder that produces airway obstruction because of changes in exocrine glandular secretions, resulting in increased mucus production. *Bronchiectasis* is an obstructive disease characterized by dilated bronchioles most frequently resulting from untreated or poorly treated pulmonary infections that cause an increase in sputum production.

ASTHMA

Asthma is a chronic inflammatory disorder of the airways. The chronic inflammation leads to recurrent episodes of wheezing, breathlessness, chest tightness, and cough, particularly at night or in the early morning. These episodes are associated with widespread but variable airflow obstruction that is usually reversible, either spontaneously or with treatment. The clinical course of asthma is unpredictable, ranging from periods of adequate control to exacerbations with poor control of symptoms.[4]

Asthma affects an estimated 16 million adult Americans. Among adults, women are 66% more likely to have asthma than men. Asthma is a public health concern with over 10 million lost workdays in adults.[5] The significant morbidity rates related to asthma may be attributed to limited access to health care, an inaccurate assessment of disease severity, a delay in seeking help, inadequate medical treatment, nonadherence to prescribed therapy because of the high cost, and an increase in allergens in the environment, especially in the inner city. Older adults may be underdiagnosed with asthma primarily because their symptoms are similar to those of COPD and pulmonary function testing is not commonly done in this age-group.[5]

After a long period of a steady increase in rates, it appears that mortality and morbidity rates from asthma have reached a plateau and/or decreased. However, in people older than 15 years of age, annually there are over 3700 deaths with more than 50% in people older than 65 years of age. (Gender and cultural/ethnic differences are presented in boxes below.)

Risk Factors for Asthma and Triggers of Asthma Attacks

Risk factors for asthma and triggers of asthma attacks can be related to the patient (e.g., genetic factors) or the environment (Table 29-1). Male gender is a risk factor for asthma in children (but not adults) for unclear reasons. Obesity has also been shown to be a risk factor for asthma.[3] Other factors and triggers are discussed in this section.

Genetics. Asthma has a component that is inherited, but the genetics are complex. Numerous genes may be involved in the development of asthma and different ethnicities may have different genes.[3,4] *Atopy*, the genetic predisposition to develop an allergic (immunoglobulin E [IgE]–mediated) response to common allergens, is a major risk factor for asthma.

Immune Response. The *hygiene hypothesis* suggests that a newborn baby's immune system must be educated so it will function properly during infancy and the rest of life. If a person is exposed to certain infections early in life, uses few antibiotics, is exposed to other children (e.g., siblings, day care), or lives in the country or with pets, he or she will have a lower incidence of asthma. If these factors are not present in one's childhood, the person has a higher rate of asthma.[4]

Allergens. Indoor and outdoor allergens are well known to trigger asthma symptoms, but their role in the development of asthma is unclear. House dust mites are a pervasive problem as

GENDER DIFFERENCES

Asthma

MEN	WOMEN
• Before puberty, boys are more affected than girls.	• After puberty and into adulthood, more women are affected than men. • Women who are admitted to the emergency department are more likely to need hospitalization. • Death rate from asthma is greater in women than men.

CULTURAL AND ETHNIC HEALTH DISPARITIES

Obstructive Pulmonary Diseases

- Asthma prevalence rates are over 38% higher among African Americans than whites.
- Puerto Ricans have higher asthma prevalence rates and age-adjusted death rates than all other racial and ethnic subgroups.
- Female African Americans have the highest mortality rates from asthma among all ethnic/gender groups.
- Whites have the highest incidence of chronic obstructive pulmonary disease despite high rates of smoking among other ethnic groups.
- Whites have the highest incidence of cystic fibrosis.
- Cystic fibrosis is uncommon among African Americans, Hispanics, and Asian Americans.

TABLE 29-1 TRIGGERS OF ACUTE ASTHMA ATTACKS

Allergen inhalation
- Animal dander (e.g., cats, mice, guinea pigs)
- House dust mite
- Cockroaches
- Pollens
- Molds

Air pollutants
- Exhaust fumes
- Perfumes
- Oxidants
- Sulfur dioxides
- Cigarette smoke
- Aerosol sprays

Viral upper respiratory infection
Sinusitis
Exercise and cold, dry air
Stress
Drugs
- Aspirin
- Nonsteroidal antiinflammatory drugs
- β-Adrenergic blockers

Occupational exposure
- Agriculture, farming
- Paints, solvents
- Laundry detergents
- Metal salts
- Wood and vegetable dusts
- Industrial chemicals and plastics
- Pharmaceutical agents

Food additives
- Sulfites (bisulfites and metabisulfites)
- Beer, wine, dried fruit, shrimp, processed potatoes
- Monosodium glutamate
- Tartrazine

Hormones/menses
Gastroesophageal reflux disease (GERD)

they are almost impossible to eliminate. Cockroaches, furry animals, fungi, and molds can trigger asthma attacks, but the role in the actual development of asthma is not as clear.[3,4]

Exercise. Asthma that is induced or exacerbated during physical exertion is called *exercise-induced asthma* (EIA). Typically, EIA occurs after vigorous exercise, not during it (e.g., jogging, aerobics, walking briskly, climbing stairs). Symptoms of EIA are pronounced during activities where there is exposure to cold, dry air. For example, swimming in an indoor heated pool is less likely to produce symptoms than downhill skiing. Airway obstruction may occur due to changes in the airway mucosa caused by the hyperventilation occurring during exercise with either cooling or rewarming of air and capillary leakage in the airway wall.

Air Pollutants. Various air pollutants, cigarette or wood smoke, vehicle exhaust, elevated ozone levels, sulfur dioxide, and nitrogen dioxide can trigger asthma attacks. In heavily industrialized or densely populated areas, climatic conditions often lead to concentrated pollution in the atmosphere, especially with thermal inversions and stagnant air masses. Ozone alert days are regularly noted on the news reports, and patients should minimize outdoor activity during these times. Cigarette smoking is associated with an accelerated decline of lung functioning in a person with asthma, increases the severity of the disease, may cause the patient to be less responsive to treatment with corticosteroids (either systemic or inhaled), and reduces the chance of the asthma being controlled.[3]

Occupational Factors. Occupational asthma is the most common occupational respiratory disorder with up to 15% of new asthma cases arising from job-related exposures.[6] Irritants cause a change in the responsiveness of the airways. Agricultural workers, painters (including spray painting), plastics manufacturing, and cleaning work are occupations with a high risk. Characteristically, patients will give a history of arriving at work feeling well but experience gradual development of symptoms by the end of the day.

Respiratory Infections. Respiratory infections (i.e., viral and not bacterial) are often the major precipitating factor of an acute asthma attack. The respiratory syncytial virus (RSV) in children and the rhinovirus are two major factors in the development and possibly the severity of asthma.[4] Infections cause an increase in the hyperresponsiveness of the bronchial system that may last from 2 to 8 weeks after the infection in both normal and asthmatic persons. It is thought that viruses cause asthma exacerbations by activating the immune system. This ultimately results in production of inflammatory mediators leading to the onset of asthma symptoms.

Nose and Sinus Problems. Allergic rhinitis is a major predictor of adult asthma.[7] Treatment of allergic rhinitis reduces the frequency of asthma exacerbations. Some patients with asthma have chronic sinus problems that cause inflammation of the mucous membranes. Although the cause is usually noninfectious (e.g., allergies), bacterial infections may also be a cause. Sinusitis must be treated and large nasal polyps removed for the asthma patient to have good control. (Sinusitis is discussed in Chapter 27.)

Drugs and Food Additives. Sensitivity to specific drugs may occur in some persons, especially those with nasal polyps and sinusitis. Some people with asthma have what is termed the *asthma triad*—nasal polyps, asthma, and sensitivity to aspirin and nonsteroidal antiinflammatory drugs (NSAIDs). Salicylic acid can be found in many over-the-counter (OTC) drugs and some foods, beverages, and flavorings. In some asthmatics who use aspirin or NSAIDs (e.g., ibuprofen [Motrin]), wheezing will develop within 2 hours. In addition, there is usually profound rhinorrhea, congestion, and tearing. Facial flushing, gastrointestinal symptoms, and angioedema can occur. Although sensitivity to salicylates persists for many years, the nature and severity of the reaction can change over time. Avoidance of aspirin and NSAIDs is required. However, patients with aspirin sensitivity under the care of an allergist can be desensitized by daily administration of the drug.

β-Adrenergic blockers in oral form (e.g., metoprolol [Toprol]) or topical eyedrops (e.g., timolol [Timoptic]) may trigger asthma because of bronchospasm. Angiotensin-converting enzyme (ACE) inhibitors (e.g., lisinopril [Prinivil]) may produce cough in susceptible individuals, thus making asthma symptoms worse. Other agents that may precipitate asthma in the susceptible patient are tartrazine (yellow dye no. 5, found in many foods) and sulfiting agents widely used in the food and pharmaceutical industries as preservatives and sanitizing agents. Sulfiting agents are commonly found in fruits, beer, and wine and used extensively in salad bars to protect vegetables from oxidation. Asthma exacerbations have been reported after the use of sulfite-containing preservatives found in topical ophthalmic solutions, intravenous (IV) corticosteroids, and some inhaled bronchodilator solutions. Food allergies triggering asthma reactions in adults are rare. Avoidance diets are not recommended until an allergy has been demonstrated, usually by oral challenges.[3]

Gastroesophageal Reflux Disease. The exact mechanism by which gastroesophageal reflux disease (GERD) triggers asthma is unknown. It is postulated that reflux of stomach acid into the esophagus can be aspirated into the lungs, causing reflex vagal stimulation and bronchoconstriction. Although GERD is primarily involved in nocturnal asthma, it can trigger daytime asthma as well. (GERD is discussed in Chapter 42.)

Psychologic Factors. Asthma is not a psychosomatic disease. However, emotional stress that is seen with extremes of emotion such as crying, laughing, anger, and fear can lead to hyperventilation and hypocapnia, which can cause airway narrowing.[3] An asthma attack caused by any triggering mechanism can produce panic, stress, and anxiety, which are not unexpected emotions during this experience. Panic is a normal response to not being able to breathe. The extent to which psychologic factors contribute to the induction and continuation of any given acute exacerbation is unknown, but it probably varies from patient to patient and in the same patient from episode to episode.

Pathophysiology

The primary pathophysiologic process in asthma is persistent but variable inflammation of the airways. The airflow is limited because the inflammation results in bronchoconstriction, airway hyperresponsiveness (hyperreactivity), and edema of the airways. Exposure to allergens or irritants initiates the inflammatory cascade (Fig. 29-1). A variety of inflammatory cells are involved including mast cells, macrophages, eosinophils, neutrophils, T and B lymphocytes, and epithelial cells of the airways.[4]

As the inflammatory process begins, mast cells (found beneath the basement membrane of the bronchial wall) degranulate and release multiple inflammatory mediators (Fig. 29-2). IgE antibodies are linked to mast cells and the allergen cross-links the IgE. Then common inflammatory mediators such as

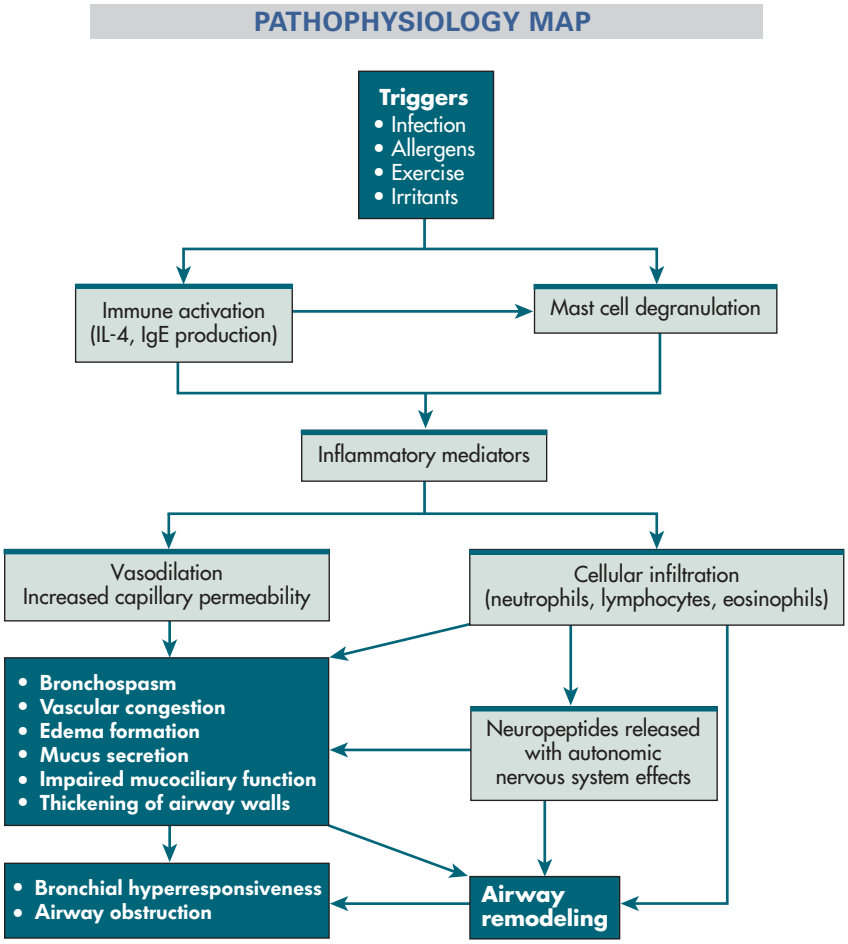


FIG. 29-1 Pathophysiology of asthma. *IL,* Interleukin.

leukotrienes, histamine, cytokines (e.g., interleukins-4 and 5), prostaglandins, and nitric oxide are released. Some inflammatory mediators have effects on the blood vessels, causing vasodilation and increasing capillary permeability. Some mediators result in the airways being infiltrated by eosinophils, lymphocytes, and neutrophils. The resulting inflammatory process results in vascular congestion; edema formation; production of thick, tenacious mucus; bronchial muscle spasm; thickening of airway walls; and increased bronchial hyperresponsiveness[8] (Fig. 29-3). This whole process is sometimes referred to as the *early-phase response* in asthma. Clinically it can occur within 30 to 60 minutes after exposure to the allergen or irritant.

Symptoms can recur 4 to 10 hours after the initial attack because of eosinophil and lymphocyte activation and further release of more inflammatory mediators. The epithelial cells also produce cytokines and other inflammatory mediators. This delayed response is called the *late-phase response* in asthma. Only about 30% to 50% of patients experience this delayed response. It can be more severe than the early-phase response and persist for 24 hours or more. It is characterized by a self-sustaining cycle of inflammation. Airflow may be limited from the swelling of the airways with or without bronchoconstriction. Corticosteroids are effective in treating this inflammation.

Alterations in the neural control of the airways also occur in asthma. The autonomic nervous system, consisting of the parasympathetic and sympathetic systems, innervates the bronchi. Airway smooth muscle tone is regulated by the parasympathetic nervous system. In asthma, there is overactivity of the parasympathetic nervous system. When airway nerve endings are stimulated by mechanical or chemical stimuli (e.g., air pollution, cold air, dust, allergens), increased release of acetylcholine results in increased smooth muscle contraction and mucus secretion, ultimately leading to bronchoconstriction.

Chronic inflammation may result in structural changes in the bronchial wall known as *remodeling.* A progressive loss of lung function occurs that is not prevented or fully reversed by therapy. The changes in structure may include fibrosis of the subepithelium, smooth muscle hypertrophy of the airways, mucus hypersecretion, continued inflammation, and angiogenesis (proliferation of new blood vessels). Remodeling is thought to explain why some individuals have persistent asthma and limited response to therapy.[3,4]

Hyperventilation occurs during an asthma attack as lung receptors respond to increased lung volume from trapped air and airflow limitation. Decreased perfusion and ventilation of the alveoli and increased alveolar gas pressure lead to ventilation-perfusion abnormalities in the lungs. The patient will be hypoxemic early on with decreased $PaCO_2$ and increased pH (respiratory alkalosis) as he or she is hyperventilating. As the airflow limitation worsens with air trapping, the patient works much harder to breathe. The $PaCO_2$ will normalize as the patient tires, and then it will increase to produce respiratory acidosis, which is an ominous sign signifying respiratory failure.[8]

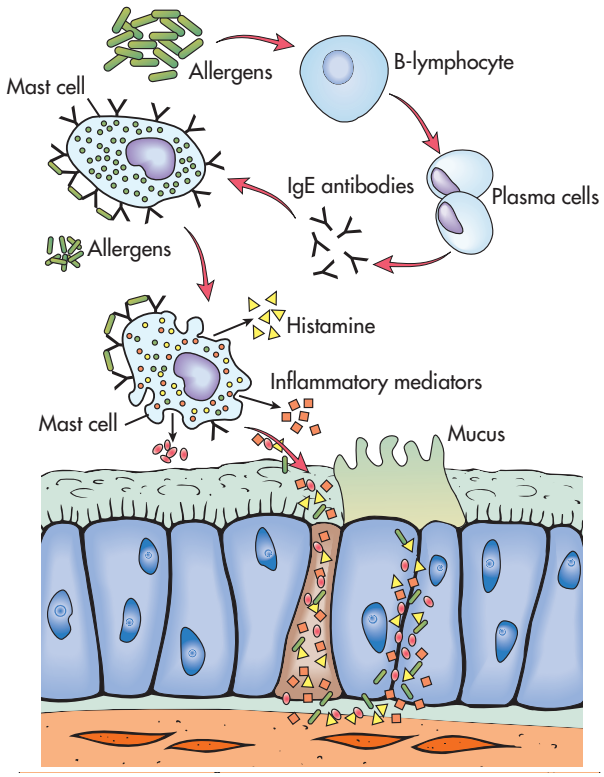


FIG. 29-2 Allergic asthma is triggered when an allergen cross-links IgE receptors on mast cells, which are then activated to release histamine and other inflammatory mediators (early-phase response). A late-phase response may occur due to further inflammation.

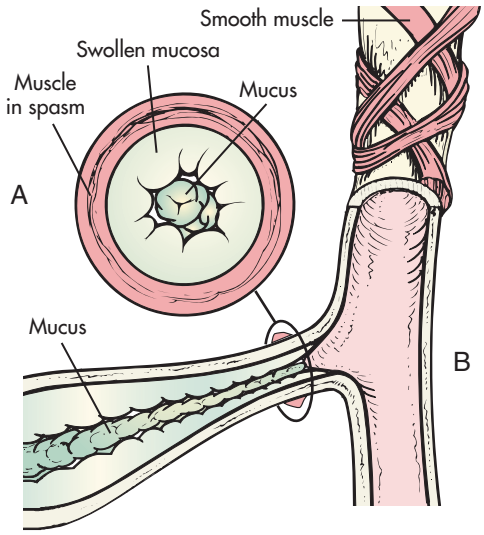


FIG. 29-3 Factors causing obstruction (especially expiratory obstruction) in asthma. **A,** Cross section of a bronchiole occluded by muscle spasm, swollen mucosa, and mucus in the lumen. **B,** Longitudinal section of a bronchiole.

Clinical Manifestations

Asthma is characterized by an unpredictable and variable course from seemingly minor interferences in breathing to life-threatening episodes that can occur in the same person. Depending on an individual's response, asthma can rapidly progress from normal breathing to acute severe asthma. Recurrent episodes of wheezing, breathlessness, chest tightness, and cough, particularly at night and in the early morning, are typical in asthma. An attack of asthma may have an abrupt onset, but usually symptoms occur more gradually. Attacks may last for a few minutes to several hours. Between attacks the patient may be asymptomatic with normal or near-normal pulmonary function, depending on the severity of disease. However, in some persons, compromised pulmonary function may result in a state of continuous symptoms and chronic debilitation characterized by irreversible airway disease.

The characteristic clinical manifestations of asthma are wheezing, cough, dyspnea, and chest tightness after exposure to a precipitating factor or trigger. Expiration may be prolonged. Instead of a normal inspiratory-expiratory ratio of 1:2, it may be prolonged to 1:3 or 1:4. Normally the bronchioles constrict during expiration. However, as a result of bronchospasm, edema, and mucus in the bronchioles, the airways become narrower than usual. Thus it takes longer for the air to move out of the bronchioles. This produces the characteristic wheezing, air trapping, and hyperinflation.

Wheezing is an unreliable sign to gauge the severity of an attack. Many patients with minor attacks wheeze loudly, whereas others with severe attacks do not wheeze. The patient with severe asthmatic attacks may have no audible wheezing because of the marked reduction in airflow. For wheezing to occur, the patient must be able to move enough air to produce the sound. Wheezing usually occurs first on exhalation. As asthma progresses, the patient may wheeze during inspiration and expiration.

In some patients with asthma, cough is the only symptom, and this is termed *cough variant asthma.* The bronchospasm may not be severe enough to cause airflow obstruction, but it can increase bronchial tone and cause irritation with stimulation of the cough receptors. The cough may be nonproductive. Secretions may be thick, tenacious, white, gelatinous mucus, which makes their removal difficult.

The person with asthma has difficulty with air movement in and out of the lungs, which creates a feeling of suffocation. Therefore during an acute attack, the person with asthma usually sits upright or slightly bent forward using the accessory muscles of respiration to try to get enough air. The more difficult the breathing becomes, the more anxious the patient feels.

Examination of the patient during an acute attack usually reveals signs of hypoxemia, which may include restlessness, increased anxiety, inappropriate behavior, increased pulse and blood pressure, and *pulsus paradoxus* (a drop in systolic pressure during the inspiratory cycle greater than 10 mm Hg). (Measurement of pulsus paradoxus is presented in Table 37-8.) As the patient worsens, it becomes difficult to speak in complete sentences. The respiratory rate is significantly increased (greater than 30 breaths/min) with the use of accessory muscles. Percussion of the lungs indicates hyperresonance, and auscultation indicates the presence of inspiratory or expiratory wheezing. As the episode resolves, coughing produces thick, stringy mucus.

Diminished or absent breath sounds may indicate a significant decrease in air movement resulting from exhaustion and an inability to generate enough muscle force to ventilate. Severely diminished breath sounds, often referred to as the "silent chest," are an ominous sign, indicating severe obstruction and impending respiratory failure.

Classification of Asthma

Asthma can be classified as intermittent, mild persistent, moderate persistent, or severe persistent[4] (Table 29-2). The classification system is used at diagnosis to determine the initial treatment. The classification is based on the current impairment of the person (i.e., symptoms, lung function measurements) and the risk for future exacerbations that require oral corticosteroids. Patients may move to different asthma classifications over the course of their disease.

Complications

Severe Acute Asthma and Life-Threatening Asthma. Severe asthma exacerbations occur when the patient is dyspneic at rest and the patient speaks in words, not sentences, because of the difficulty breathing. The patient is usually sitting forward to maximize the diaphragmatic movement with prominent wheezes and a respiratory rate higher than 30 per minute and pulse higher than 120 per minute. Accessory muscles in the neck are straining to try to lift the chest wall and the patient is often agitated. The peak flow (peak expiratory flow rate [PEFR]) is 40% of the personal best or less than 150 mL. Arterial blood gas (ABG) changes are listed in Table 29-3. Neck vein distention and a pulsus paradoxus of 40 mm Hg or greater may result. Usually it is difficult to auscultate pulsus paradoxus secondary to a noisy chest or increased work of breathing. These patients usually are seen in emergency departments (EDs) or hospitalized.[3,4]

A few patients perceive asthma symptoms poorly and may have a significant decrease in lung function without any change in symptoms. Patients with life-threatening asthma are typically too dyspneic to speak and will be perspiring profusely. They may even be drowsy or confused as the ABGs further deteriorate. The breath sounds may be very difficult to hear, and no wheezing is apparent as the airflow is exceptionally limited. Peak flow is less than 25% of the personal best. They become bradycardic and are close to respiratory arrest. These patients require ED or hospital care and are often admitted to an intensive care unit.

SAFETY ALERT

- If the patient has been wheezing and then there is an absence of a wheeze (i.e., silent chest) and the patient is obviously struggling, this is a life-threatening situation that may require mechanical ventilation.

Diagnostic Studies

Underdiagnosis of asthma is common. A detailed history is important in determining if a person has had previous attacks of a similar nature, often precipitated by a known cause or trigger as discussed previously in the chapter. Because wheezing and cough are seen with a variety of disorders, this complicates the diagnosis of asthma. These disorders include COPD, pulmonary embolism, GERD, obesity, vocal cord dysfunction, and heart failure.

Some controversy exists about how to best diagnose asthma. Common diagnostic measures are presented in Table 29-4. In general, the health care provider should consider the diagnosis of asthma if various indicators (i.e., clinical manifestations,

TABLE 29-2 CLASSIFICATION OF ASTHMA SEVERITY

	CLASSIFICATION OF ASTHMA SEVERITY			
		PERSISTENT		
COMPONENTS OF SEVERITY	**INTERMITTENT**	**MILD**	**MODERATE**	**SEVERE**
Impairment				
Symptoms	≤2 days/wk	>2 days/wk, not daily	Daily	Continuous
Nighttime awakenings	≤2×/mo	3-4×/mo	>1×/wk, not nightly	Often, 7×/wk
SABA use for symptoms	≤2 days/wk	>2 days/wk, not daily	Daily	Several times per day
Interference with normal activity	None	Minor limitation	Some limitation	Extremely limited
Lung function*	Normal FEV_1 between exacerbations FEV_1 >80% FEV_1/FVC normal	FEV_1 >80% predicted FEV_1/FVC normal	FEV_1 60%-80% predicted FEV_1/FVC reduced by 5%	FEV_1 <60% predicted FEV_1/FVC reduced by 5%
Risk				
Exacerbations requiring oral corticosteroids	0-1/yr	≥2/yr even in the absence of impairment → Consider severity and interval since last exacerbation. → Frequency and severity may fluctuate over time. → Relative annual risk of exacerbation may be related to FEV_1. →		
Recommended Step for Initiating Treatment	Step 1	Step 2	Step 3†	Step 4 or 5†
	Reevaluate asthma control in 2-6 wk and adjust therapy accordingly			

Guidelines for Using Table

- Patients should be assigned to the most severe step in which any feature occurs. Clinical features for individual patients may overlap across steps. Determine level of severity by assessment of both impairment and risk. Assess impairment by patient's recall of previous 2-4 wk and spirometry results.
- An individual's classification should change over time as treatment is initiated. After treatment, the focus switches to level of control, not the classification of severity.
- Patients at any level of severity of chronic asthma can have mild, moderate, or severe exacerbations of asthma. Some patients with intermittent asthma experience severe and life-threatening exacerbations separated by long periods of normal lung function and no symptoms.

Source: Adapted from *Expert Panel Report 3: Guidelines for the Diagnosis and Management of Asthma.* National Asthma Education and Prevention Program, National Heart, Lung, and Blood Institute, 2007.

FEV_1, Forced expiratory volume in 1 second; *FVC*, forced vital capacity; *SABA*, short-acting bronchodilator.

*Percent predicted values for forced expiratory volume in 1 second (FEV_1) or ratio of FEV_1/forced vital capacity (FVC). Normal FEV_1/FVC: 8-19 yr, 85%; 20-39 yr, 80%; 40-59 yr, 75%; 60-80 yr, 70%.

†Consider short-term corticosteroid therapy.

health history, and peak flow variability or spirometry) are positive. Pulmonary function tests can be used to determine the reversibility of bronchoconstriction and thus establish the diagnosis of asthma.

The PEFR measured by the peak flow meter generally correlates with forced expiratory volume in 1 second (FEV_1) and is a helpful tool to diagnose and manage asthma.[3] However, to confirm the diagnosis of asthma, spirometry is preferred as there are a variety of peak flow meters on the market, but no standardized PEFR reference values. In general, however, peak flow meters are best designed as monitoring, and not diagnostic, devices.[4]

TABLE 29-3 COMPARISON OF ASTHMA AND COPD*

	ASTHMA	COPD
Clinical Features		
Age	Usually <40 yr (onset)	Usually 40-50 yr (onset)
Smoking history	Not causal	Long history (>10-20 pack-years)
Health and family history	Presence of allergy, rhinitis, eczema; family history of asthma	Infrequent allergies; may have exposure to environmental pollutants; with α_1-antitrypsin deficiency, a family history of lung or liver disease without smoking history
Clinical symptoms	Intermittent, vary day to day, at night or early morning	Slowly progressive and persistent
Dyspnea	Absent except in exacerbations or poor control	Dyspnea during exercise
Sputum	Infrequent	Often
Disease course	Stable (with exacerbations)	Progressive worsening (with exacerbations)
Diagnostic Study Results		
ABGs	Normal between exacerbations	Between exacerbations in advanced COPD • Often low-normal pH and PaO_2 • High-normal $PaCO_2$ with high HCO_3^- (compensated respiratory acidosis)
pH	↑ Early in exacerbation, then ↓ if prolonged or severe exacerbation	N→↓
PaO_2	↓	N→↓
$PaCO_2$	↓ Early in exacerbation, then ↑ if prolonged or severe exacerbation	N→↑
Chest x-ray	May reveal hyperinflation	Hyperinflation; may have cardiac enlargement, flattened diaphragm
Lung volumes	Often normalizes	Never normalizes
• Total lung capacity	Increased	Increased
• Residual volume	Increased	Increased
• FEV_1	Decreased	Decreased
• FEV_1/FVC	Normal to decreased	Decreased (<70%)

ABGs, Arterial blood gases; *FEV_1,* forced expiratory volume in 1 second; *FVC,* forced vital capacity.
*Individuals may have features of both asthma and COPD.

Pulmonary function tests (PFTs) are usually normal between asthma attacks if the patient has no other underlying pulmonary disease. However, the patient with asthma may show an obstructive pattern with asthma including a decrease in forced vital capacity (FVC), FEV_1, PEFR, and FEV_1 to FVC ratio (FEV_1/FVC). (The normal values for pulmonary function tests are discussed in Chapter 26.)

When PFTs are done, the patient is asked to withhold taking any bronchodilator medications for 6 to 12 hours before the test. PFTs can be done before and after the administration of a bronchodilator to determine the degree of the response. This may help to document reversibility of airway obstruction, as this is critical information for diagnosing asthma. A positive response to the bronchodilator is an increase of more than 200 mL and an increase of more than 12% between preadministration and postadministration values.

Lung function parameters fall from their baseline levels during an exacerbation. Some patients with symptoms of asthma have normal lung function. Therefore measures of airway responsiveness to known bronchial irritants, such as methacholine, histamine, or exercise, may help establish the diagnosis of asthma.[4]

An elevated serum eosinophil count and elevated serum IgE levels are highly suggestive of *atopy* (allergic tendency), which may be a risk factor in a person's asthma. Allergy skin testing may be of some value to determine sensitivity to specific allergens. However, a positive skin test does not necessarily mean that the allergen is causing the asthma attack. On the other hand, a negative allergy test does not mean that the asthma is not allergy related. A radioallergosorbent test (RAST), which is a blood test, is sometimes used to identify allergic causes in certain patients who show negative skin tests and in those who

TABLE 29-4 COLLABORATIVE CARE

Asthma

Diagnostic
History and physical examination
Pulmonary function studies including response to bronchodilator therapy
Peak expiratory flow rate (PEFR)
Chest x-ray
Measurement of oximetry
Allergy skin testing (if indicated)
Blood level of eosinophils and IgE (if indicated)

Collaborative Therapy
Intermittent or Persistent Asthma
Identification and avoidance/elimination of triggers
Patient and caregiver teaching
Drug therapy (see Tables 29-6 and 29-7 and Fig. 29-4)
Asthma action plan (see Table 29-13)
Desensitization (immunotherapy) if indicated
Assess for control (e.g., Asthma Control Test [ACT]) (see Fig. 29-5)

Severe or Life-Threatening Asthma Exacerbation
SaO_2 monitoring
ABGs
Inhaled β_2-adrenergic agonists
Inhaled anticholinergic agents (only in the initial treatment)
O_2 by mask or nasal prongs
IV or oral corticosteroids
IV fluids
IV magnesium and/or heliox
Intubation and assisted ventilation

ABGs, Arterial blood gases; *IgE,* immunoglobulin E; *SaO_2,* oxygen saturation.

should not be skin tested (e.g., patients with severe eczema). (Allergy testing is discussed in Chapter 14.)

A chest x-ray in an asymptomatic patient with asthma is usually normal, but needs to be obtained as a baseline on initial diagnosis. A chest x-ray obtained during an acute attack usually shows hyperinflation and may reveal other complications of asthma such as mucoid impaction, pneumothorax, atelectasis, or pneumomediastinum.

If the patient has wheezing and acute distress, it is not feasible to obtain a detailed health history (although a family member may supply some pertinent information). During an acute attack of asthma, bedside spirometry (specifically FEV_1 or FVC, but usually PEFR) may be used to monitor obstruction. Pulmonary function test results, serial spirometric parameters, oximetry, and measurement of ABGs help provide information about the severity of the attack and the response to therapy. A complete blood cell count (CBC) and serum electrolytes are also obtained to help monitor the course of therapy.

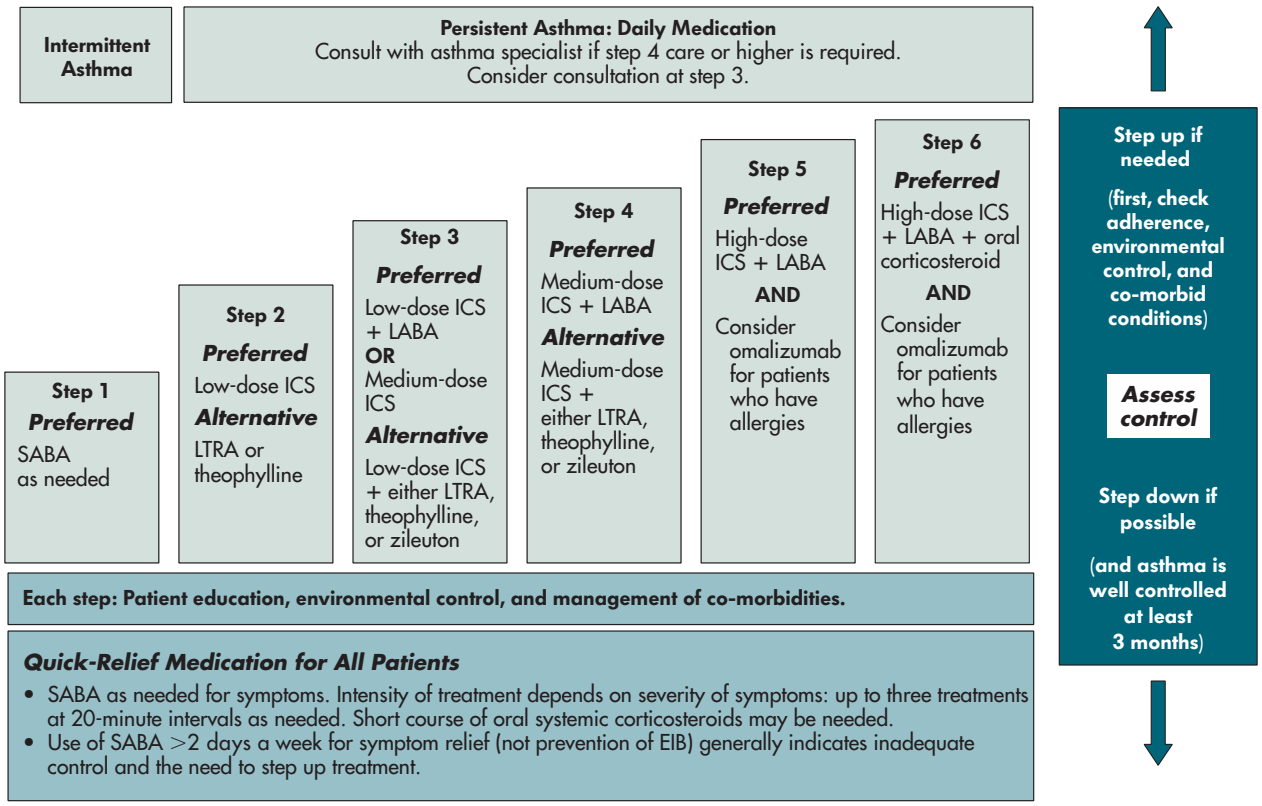
A sputum specimen for culture and sensitivity may be obtained to rule out the presence of bacterial infection, especially if the patient has purulent sputum, a history of upper respiratory tract infection, a fever, or an elevated white blood cell (WBC) count. However, the vast majority of asthma exacerbations are viral in nature and sputum cultures are rarely done on an outpatient basis.

A hand-held, point-of-care device, called Nioxx Mino, measures airway inflammation related to asthma. It measures fractional exhaled nitric oxide (FENO). Nitric oxide levels are increased in the breath of people with asthma, and changes in FENO levels may indicate if inflammation is present and whether or not treatment for asthma is working. However, studies have not been done to determine the effectiveness of FENO to aid in the diagnosis of asthma.

Collaborative Care

The National Asthma Education and Prevention Program (NAEPP) of the National Heart, Lung, and Blood Institute (NHLBI) has convened expert panels to prepare guidelines for the diagnosis and management of asthma.[4] The first NAEPP report served as a basis for the development of reports prepared by asthma experts worldwide, and the Global Initiative for Asthma (GINA) was created. The goals of GINA are to decrease asthma morbidity and mortality rates and improve the management of asthma worldwide.[3] The reports of NAEPP and GINA are quite similar. The NAEPP guidelines are presented in this chapter (e.g., Tables 29-2 and 29-5 and Fig. 29-4).

The goal of asthma treatment is to achieve and maintain control of the disease.[9] Once the patient is diagnosed, the guidelines give direction on classification of severity (see Table 29-2) and which medications (based on steps 1 through 6) the patient requires (Table 29-5 and Fig. 29-4). The current guidelines focus on (1) assessing the severity of the disease at diagnosis and initial treatment and then (2) monitoring periodically to achieve control of the disease. At initial diagnosis, a patient may have severe asthma and require step 4 or 5 of asthma medication. After treatment, the patient is assessed as to the level of control (i.e., well controlled, not well controlled, or very poorly controlled) (see Table 29-5). As the patient achieves control of the symptoms the health care provider steps down the medication or steps it up if the symptoms worsen. Achieving rapid



Key: *EIB*, Exercise-induced bronchospasm; *ICS*, inhaled corticosteroid; *LABA*, long-acting inhaled β_2-agonist; *LTRA*, leukotriene receptor antagonist; *SABA*, inhaled short-acting β_2-agonist.

FIG. 29-4 Drug therapy: stepwise approach for managing asthma.

control of the symptoms is the goal in order to return the patient to her or his daily functioning at the best possible level[4] (see Fig. 29-4). The level of control is based on the patient's responses to symptoms, nighttime awakenings, interferences with normal activity, and use of rescue or reliever medication.

Several validated questionnaires, such as the Asthma Control Test (ACT), provide data on these quality-of-life issues (Fig. 29-5). The level of control is also determined by the patient's current peak flow or FEV_1. In addition, any exacerbations or adverse effects of treatment will determine the level of control. Patients have individual responses to treatment and thus are in a state of flux as they seek to achieve control and minimize the risk for future exacerbations.

Intermittent and Persistent Asthma. The classification of severity of asthma at initial diagnosis helps determine which types of medications are best suited to control the asthma symptoms (see Tables 29-2 and 29-5). Patients in all classifications of asthma will require a short-term (rescue or reliever) medication. The short-acting β_2-adrenergic agonists (SABAs) (e.g., albuterol) are the gold standard and most effective. Patients with persistent asthma must be on a long-term or controller medication (Table 29-6). Inhaled corticosteroids (ICSs) (e.g., fluticasone [Flovent]) are the most effective class of drugs to combat the inflammation. (See later section on drug therapy in this chapter.)

Acute Asthma Exacerbations. Asthma exacerbations may be mild to life threatening. With *mild exacerbations,* the patient has difficulty breathing only with activity and may feel that he or she "can't get enough air." The peak flow is greater than 70% of the patient's personal best, and usually the symptoms are relieved at home promptly with an SABA such as albuterol delivered via a nebulizer or metered-dose inhaler (MDI) with a spacer. For any classification of asthma, in a "rescue plan" patients are instructed to take 2 to 4 puffs of albuterol every 20 minutes three times to gain rapid control of symptoms. Occasionally a short course of oral corticosteroids is needed to decrease airway inflammation.

With a *moderate exacerbation,* dyspnea interferes with usual activities and peak flow is 40% to 60% of personal best. In this situation, the patient usually comes to the ED or a health care provider's office to get help. Relief is provided with the SABA delivered as in the mild exacerbation and oral corticosteroids are needed. Oral routes are usually as effective as IV routes, as well as being less invasive and less expensive. The patient's symptoms may persist for several days even after the corticosteroids are started. Oxygen can be used with both mild and moderate exacerbations to maintain SpO_2 at 90% or greater. The patient's symptoms and peak flow are monitored and lung auscultation is done to ensure the patient is moving air. A good response would be measured by the peak flow (or FEV_1) returning to 70% of personal best, normal airflow on physical examination, alleviation of patient's distress, and findings sustained more than 1 hour after the last treatment.[4]

Severe and Life-Threatening Asthma Exacerbations. Severe and life-threatening asthma was previously discussed on p. 592. Management of the patient with severe and life-threatening asthma focuses on correcting hypoxemia and improving ventilation. The goal is to keep the O_2 saturation at 90% or greater. Continuous monitoring of the patient is critical. Obtaining a PEFR during a severe asthma attack is usually not possible. However, if it can be obtained and it is less than 200 L/min, it indicates severe obstruction in all but very small adults. Many of the therapeutic measures are the same as those for acute asthma. Repetitive or continuous SABA administration is provided in the ED. Initially three treatments of SABA (spaced 20 to 30 minutes apart) are given. Then more SABA is given depending on the patient's airflow, improvement, and side effects from the SABA. The person with a severe asthma exacerbation usually has partial relief from the SABA plus ipratropium (Atrovent). However, the patient with life-threatening asthma will have minimal if any relief from the same medications. After the initial treatment, ipratropium (Atrovent) is not given during the inpatient stay as it has not been found to deliver any added

TABLE 29-5 COMPONENTS OF CONTROL OF ASTHMA

	CLASSIFICATION OF ASTHMA CONTROL		
COMPONENTS OF CONTROL	**WELL CONTROLLED**	**NOT WELL CONTROLLED**	**VERY POORLY CONTROLLED**
Impairment			
Symptoms	≤2 days/wk	>2 days/wk	Throughout the day
Nighttime awakenings	≤2/mo	1-3/wk	≥4/wk
Interference with normal activity	None	Some limitation	Extremely limited
SABA use	≤2 days/wk	>2 days/wk	Several times/day
FEV_1 or peak flow	>80% predicted/personal best	60%-80% predicted/personal best	<60% predicted/personal best
Risk			
Exacerbations requiring oral corticosteroids	0-1/yr	≥2/yr	
	Consider severity and interval since last exacerbation →		
Progressive loss of lung function	Evaluation requires long-term follow-up →		
Treatment-related adverse effects	Can vary in intensity from none to very troublesome and worrisome. Level of intensity does not correlate to specific levels of control but should be considered in the overall assessment of risk. →		
Recommended Action for Treatment			
(Based on assessment of control)	• Maintain current step • Regular follow-up every 1-6 mo to maintain control • Consider step down if well controlled for at least 3 mo	• Step up one step • Reevaluate in 2-6 wk • For side effects, consider alternative treatment options	• Consider oral corticosteroids • Step up one or two steps and reevaluate in 2 wk • For side effects, consider alternative treatment options

Source: *Expert Panel Report 3: Guidelines for the Diagnosis and Management of Asthma.* National Asthma Education and Prevention Program, National Heart, Lung, and Blood Institute, 2007.

FEV_1, Forced expiratory volume in 1 second; *SABA,* short-acting bronchodilator.

1. In the *past 4 weeks,* how much of the time did your *asthma* keep you from getting as much done at work, school, or at home?

☐ 1	☐ 2	☐ 3	☐ 4	☐ 5	☐
All of the time	Most of the time	Some of the time	A little of the time	None of the time	Score

2. During the *past 4 weeks,* how often have you had shortness of breath?

☐ 1	☐ 2	☐ 3	☐ 4	☐ 5	☐
More than once a day	Once a day	3 to 6 times a week	Once or twice a week	Not at all	Score

3. During the *past 4 weeks,* how often did your *asthma* symptoms (wheezing, coughing, shortness of breath, chest tightness, or pain) wake you up at night or earlier than usual in the morning?

☐ 1	☐ 2	☐ 3	☐ 4	☐ 5	☐
4 or more nights a week	2 to 3 nights a week	Once a week	One or twice	Not at all	Score

4. During the *past 4 weeks,* how often have you used your rescue inhaler or nebulizer medication (such as albuterol)?

☐ 1	☐ 2	☐ 3	☐ 4	☐ 5	☐
3 or more times per day	1 or 2 times per day	2 or 3 times per week	Once a week or less	Not at all	Score

5. How would you rate your *asthma* control during the *past 4 weeks?*

☐ 1	☐ 2	☐ 3	☐ 4	☐ 5	☐
Not controlled at all	Poorly controlled	Somewhat controlled	Well controlled	Completely controlled	Score

Total score: ☐

Note: If the total point value ≤19, asthma may not be well controlled. The patient should talk with the health care provider.

FIG. 29-5 Asthma Control Test™ Health Survey 2003, 2004 by QualityMetric Incorporated—All rights reserved. Asthma Control Test™ is a trademark of QualityMetric Incorporated.

TABLE 29-6 DRUG THERAPY

Long-Term Control versus Quick Relief of Asthma

Long-Term Control Medications

Antiinflammatory Drugs

Corticosteroids
- Inhaled (e.g., fluticasone [Flovent])
- Oral (e.g., prednisone)

Leukotriene modifiers (e.g., montelukast [Singulair])
Anti-IgE (omalizumab [Xolair])

Bronchodilators

Long-acting inhaled β_2-adrenergic agonists (e.g., salmeterol [Serevent])
Long-acting oral β_2-adrenergic agonists (e.g., albuterol [VoSpire ER])
Methylxanthines (e.g., theophylline [Uniphyl])
Anticholinergics (inhaled) (e.g., tiotropium [Spiriva])

Quick-Relief Medications

Bronchodilators

Short-acting inhaled β_2-adrenergic agonists (e.g., albuterol [Proventil HFA])
Anticholinergics (inhaled) (e.g., ipratropium [Atrovent])

Antiinflammatory Drugs

Corticosteroids (systemic) (e.g., prednisone)*

*Considered quick-relief drugs when used in a short burst (3 to 10 days) at the start of therapy or during a period of gradual deterioration. Corticosteroids are not used for immediate relief of an ongoing attack.

benefit. Nebulized SABAs are continued for several days even after clinical improvement is noted.[4]

In severe asthma, oral systemic corticosteroids are given to patients who do not respond to the initial SABA. It is no longer recommended that patients double the dose of ICSs in times of an exacerbation as this is not effective. In life-threatening asthma, corticosteroids are administered intravenously and are usually tapered rapidly. IV corticosteroids (e.g., methylprednisolone) are administered every 4 to 6 hours, although their peak effect is not apparent for 4 to 12 hours. Then the patient is started on the oral corticosteroids. The length of oral prednisone treatment for both severe and life-threatening asthma after discharge is usually about 10 days. Inhaled corticosteroids are usually added while the patient is still in the hospital. High-dose ICSs prevent asthma relapse and may be prescribed until the patient can step down to lower doses. In severe and life-threatening asthma, adjunctive medications such as IV magnesium sulfate may be administered in certain adults with a very low FEV_1 or peak flow (less than 40% of predicted or personal best at presentation) or those who fail to respond to initial treatment. In addition, when the patient is in the hospital, heliox (a mixture of helium and oxygen) may be used to deliver the nebulized albuterol as helium has a low density and may improve the bronchodilation of the albuterol.[3,4]

Supplemental O_2 is given by mask or nasal prongs to achieve a PaO_2 of at least 60 mm Hg or an O_2 saturation greater than

90%. An arterial catheter may be inserted to facilitate frequent ABG monitoring. Because the patient's insensible loss of fluids is increased and the metabolic rate is increased, moderate rates of IV fluids are given to provide optimal hydration. Sodium bicarbonate administration is usually limited to treatment of severe metabolic or respiratory acidosis (pH <7.29) in the mechanically ventilated patient because effective bronchodilation by β-adrenergic agonists is not possible if the patient has extreme acidosis. Bronchoscopy, although rarely performed during an acute attack, may be necessary to remove thick mucous plugs.

Occasionally, asthma exacerbations are life threatening and respiratory arrest is pending or actually occurring. Therefore the patient will require intubation and mechanical ventilation if there is no response to treatment. The patient will be provided with 100% oxygen, hourly or continuously nebulized SABAs, IV corticosteroids, and possible other adjunctive therapies as noted previously.

Theophylline, mucolytics, and sedatives are no longer recommended for asthma exacerbations. Avoid sedatives because they can result in depression of the respiratory drive and possible death. Antibiotics are not recommended for asthma treatment unless there are signs of bacterial pneumonia, fever, and purulent sputum, suggesting bacterial infections. Chest physiotherapy has no role and is generally not recommended for asthma because it is too stressful for the breathless patient.[3,4] Although it is no longer listed in the guidelines for usual asthma exacerbation management, epinephrine is occasionally administered for acute treatment of anaphylaxis if selective β_2-adrenergic agonists are not available. If epinephrine is administered, patients need their blood pressure (BP) and electrocardiogram (ECG) monitored closely.

The Alair Bronchial Thermoplasty system is a medical device that uses radiofrequency energy to treat patients with severe and persistent asthma who are not well controlled with drugs. The device is composed of a catheter with an electrode tip that delivers a form of electromagnetic energy (radiofrequency energy) directly to the airways. A controller unit generates and controls the energy. The Alair system treats asthma symptoms that result from inflammation by using radiofrequency energy to heat the lung tissue in a controlled manner, reducing the thickness of smooth muscle in the airways and improving the patient's ability to breathe. To benefit, patients will require multiple sessions targeting different areas in the lungs.

Drug Therapy

A stepwise approach to drug therapy is based initially on the asthma severity and then on level of control. Persistent asthma requires daily long-term therapy in addition to appropriate medications to manage acute symptoms. Medications are divided into two general classifications: (1) quick-relief or rescue medications to treat symptoms and exacerbations, such as SABAs, and (2) long-term control medications to achieve and maintain control of persistent asthma, such as ICSs (see Table 29-6). Some of the controllers are used in combination to gain better asthma control (e.g., fluticasone/salmeterol [Advair]).

Antiinflammatory Drugs

Corticosteroids. Chronic inflammation is a primary component of asthma. Corticosteroids are antiinflammatory medications that reduce bronchial hyperresponsiveness, block the late-phase reaction, and inhibit migration of inflammatory cells. Corticosteroids are more effective in improving asthma control than any other long-term drug. ICSs are first-line therapy for patients with persistent asthma requiring step 2 through 6 therapy (see Fig. 29-4). Usually, ICSs must be administered for 1 to 2 weeks before maximum therapeutic effects can be seen. Some ICSs (e.g., fluticasone [Flovent], budesonide [Pulmicort]) begin to have a therapeutic effect in 24 hours. Administration of these drugs needs to be done on a fixed schedule.

When ICSs are administered, asthma can usually be controlled without significant systemic side effects because little systemic drug absorption occurs from these devices. However, ICSs at the highest dosage levels have been associated with side effects such as easy bruising and accelerated bone loss.[3] Oropharyngeal candidiasis, hoarseness, and dry cough are local side effects caused by inhalation of corticosteroids. These problems can be reduced or prevented by using a spacer (Fig. 29-6) with the MDI and by gargling with water or mouthwash after each use. Using a spacer or holding device for inhalation of inhaled corticosteroids can be helpful in getting more medication into the lungs. However, newer drugs (e.g., ciclesonide [Alvesco]) that are activated in the lungs (not the pharynx) appear to minimize these side effects without the need for a spacer or mouth rinsing.[3]

Short courses of orally administered corticosteroids are indicated for acute exacerbations of asthma to gain prompt control. Maintenance doses of oral corticosteroids may be necessary to control asthma in a minority of patients with severe chronic asthma when long-term therapy is required. A single dose in the morning to coincide with endogenous cortisol production and alternate-day dosing are associated with fewer side effects. (Side effects of long-term corticosteroid therapy are discussed in Chapter 50.) Women, especially postmenopausal women, who have asthma and who use corticosteroids should take adequate amounts of calcium and vitamin D and participate in regular weight-bearing exercise. (Osteoporosis is discussed in Chapter 64.)

Leukotriene Modifiers. Ieukotriene modifiers include leukotriene receptor blockers (antagonists) (zafirlukast [Accolate], montelukast [Singulair]) and leukotriene synthesis inhibitors (zileuton [Zyflo CR]). These drugs interfere with the synthesis or block the action of leukotrienes. Leukotrienes are inflammatory

FIG. 29-6 Example of an AeroChamber spacer used with a metered-dose inhaler.

TABLE 29-7 DRUG THERAPY

Asthma and Chronic Obstructive Pulmonary Disease

DRUG	ROUTE OF ADMINISTRATION	SIDE EFFECTS (SE)	COMMENTS*
Antiinflammatory Agents			
Corticosteroids			
hydrocortisone (Solu-Cortef)	IV	With long-term use: cushingoid appearance, skin changes (acne, striae, bruising), osteoporosis, increased appetite, obesity, peptic ulcer, hypertension, hypokalemia, cataracts, menstrual irregularities, muscle weakness, immunosuppression, catabolism. With short-term use (e.g., <2 wk): sleep disturbances, increased appetite.	Alternate-day therapy minimizes SE. Oral dose should be taken in morning with food or milk. When given in high doses, observe for epigastric distress. Long-term corticosteroid therapy requires supplementation with vitamin D and calcium to prevent osteoporosis. Discontinue gradually over time to prevent adrenal insufficiency. If during tapering symptoms recur, health care provider should be notified.
methylprednisolone (Medrol, Solu-Medrol)	Oral, IV		
prednisone	Oral		
fluticasone (Flovent HFA, Flovent Diskus)	MDI, DPI	Oral candidiasis (thrush), hoarseness, irritated throat, headache, sinus infection, upper respiratory infection.	Not recommended for acute asthma attack. Rinse mouth with water or mouthwash after use to prevent oral fungal infections. Use of spacer device with MDI may decrease incidence of oral candidiasis. With inhaled corticosteroids, may not see effects until after at least 2 wk of regular treatment.
beclomethasone (Qvar)	MDI	Oral candidiasis, hoarseness, irritated throat, dry mouth, cough, few systemic effects except for headache.	Same as fluticasone except less oral candidiasis because of very small particle size which is deposited deeper in the airways.
budesonide (Pulmicort Turbuhaler)	DPI	Same as above.	
mometasone (Asmanex Twisthaler)	DPI	Same as above.	
ciclesonide (Alvesco)	MDI	Headache, nasopharyngitis.	Oral candidiasis and other localized oropharyngeal effects (e.g., hoarseness). Fewer SE than other ICSs because of small particle size with minimal activation in oropharynx.
Anticholinergics			
Short-Acting			
ipratropium (Atrovent HFA)	Nebulizer, MDI	Drying of oral mucosa, cough, flushing of skin, bad taste.	Alternating schedules of β-adrenergic agonists and atropine administration may be helpful in some patients. Temporary blurred vision if sprayed in eyes. Use cautiously in patients with narrow-angle glaucoma or prostatic enlargement. Ongoing review to determine if this class of anticholinergics places patients at risk for cardiovascular events, including strokes.
Long-Acting			
tiotropium (Spiriva HandiHaler)	DPI	Dry mouth, upper respiratory infection.	Blurred vision if powder comes in contact with eyes. Must discontinue use of ipratropium while on tiotropium. Patient must use short-acting β-adrenergic agonists for quick-relief medication. See above related to class risk.
Anti-IgE			
omalizumab (Xolair)	Subcutaneous injection	Injection site reaction (e.g., bruising, redness, warmth, pain).	Only for moderate to severe persistent allergic asthma with symptoms not adequately controlled by ICS. Not for acute bronchospasm. Administer only under direct medical supervision and observe patient for a minimum of 2 hr following administration as anaphylaxis has been reported with use.

BP, Blood pressure; *CNS,* central nervous system; *DPI,* dry powder inhaler; *GI,* gastrointestinal; *HFA,* hydrofluoroalkane (propellant); *ICSs,* inhaled corticosteroids; *IV,* intravenous; *MDI,* metered-dose inhaler.

*For patient instructions in English and Spanish for the devices, see *www.chestnet.org/patients/guides/inhaledDevices.php.*

†FDA is continuing to review clinical trial data to assess mood and behavioral adverse events related to drugs that act through the leukotriene pathway, and further updated information can be found at *www.fda.gov/medwatch.*

TABLE 29-7 DRUG THERAPY—cont'd

Asthma and Chronic Obstructive Pulmonary Disease

DRUG	ROUTE OF ADMINISTRATION	SIDE EFFECTS (SE)	COMMENTS*
Leukotriene Modifiers†			
Leukotriene Receptor Blocker			Not for acute asthma attacks.
zafirlukast (Accolate)	Oral tablets	Headache, dizziness; nausea, vomiting, diarrhea, fatigue, abdominal pain.	Take at least 1 hr before or 2 hr after meals. Affects metabolism of erythromycin and theophylline. Not to be used to treat acute asthma episodes.
montelukast (Singulair)	Oral tablets, chewable tablets, oral granules	Well tolerated.	Not to be used to treat acute asthma episodes.
Leukotriene Inhibitor			
zileuton (Zyflo CR)	Oral tablets	↑ Liver enzymes; dyspepsia, pain, headache.	Monitor liver enzymes. May interfere with metabolism of warfarin (Coumadin) and theophylline. Not to be used to treat acute asthma episodes.
β_2-Adrenergic Agonists			
Inhaled: Short-Acting (SABA)			
albuterol (Proventil HFA, Ventolin HFA, ProAir HFA, AccuNeb, VoSpire ER [oral only])	Nebulizer, MDI, oral tablets including extended release Note: Oral tablets not for acute use, only long acting	Tachycardia, BP changes, nervousness, palpitations, muscle tremors, nausea, vomiting, vertigo, insomnia, dry mouth, headache, hypokalemia.	Use with caution in patients with cardiac disorders as β-agonists may cause ↑ BP and heart rate, CNS stimulation/excitation, and ↑ risk of dysrhythmias. Has rapid onset of action (1-3 min). Duration of action is 4-8 hr.
levalbuterol (Xopenex, Xopenex HFA)	Nebulizer, MDI	Tachycardia, nervousness, tremor (less than albuterol).	Too frequent use can result in loss of effectiveness.
pirbuterol (Maxair Autohaler)	MDI	Same as albuterol but cardiac effects are less.	
Inhaled: Long-Acting (LABA)		*In asthma:* Should never be used as monotherapy. Should be used in combination with inhaled steroids. *In COPD:* Can be used as monotherapy. Not used for rapid relief of dyspnea.	
salmeterol (Serevent)	DPI	Headache, throat dryness, tremor, dizziness, pharyngitis.	Not to exceed 2 puffs 12hr. Not to be used for acute exacerbations. Has a counter.
formoterol (Foradil Aerolizer, Perforomist)	DPI, nebulizer Perforomist is for nebulizer	Angina, tachycardia, nervousness, headache, tremor, dizziness.	Can affect blood glucose levels. Should be used with caution in patients with diabetes.
arformoterol (Brovana)	Nebulizer	See formoterol.	See formoterol. For chronic COPD use.
Methylxanthines			
IV agent: aminophylline (second-line therapy) *Oral:* theophylline (Elixophyllin, TheoCap, Theochron, Theo-24, Uniphyl)	Oral tablets, IV, elixir, sustained-release tablets	Tachycardia, BP changes, dysrhythmias, anorexia, nausea, vomiting, nervousness, irritability, headache, muscle twitching, flushing, epigastric pain, diarrhea, insomnia, palpitations.	Wide variety of response to drug metabolism exists. Half-life is ↓ by smoking and ↑ by heart failure and liver disease. Cimetidine, ciprofloxacin, erythromycin, and other drugs may rapidly ↑ theophylline levels. Taking drug with food or antacids may help GI effects. Patient must be encouraged to take drugs even when feeling well.
Combination Agents		Also see each component of medications for SE.	
ipratropium and albuterol (Combivent, DuoNeb)	MDI, Nebulizer	Chest pain, pharyngitis, diarrhea, nausea.	Patients must be careful not to overuse. Must take as prescribed.
fluticasone/salmeterol (Advair Diskus or HFA)	DPI, MDI	Headache, pharyngitis, oral candidiasis.	See salmeterol and fluticasone. Has a counter. Comes in three different strengths.
budesonide/formoterol (Symbicort)	MDI	Dysrhythmias, hypertension, paradoxic bronchospasm.	See budesonide and formoterol. Has a counter.

mediators produced from arachidonic acid metabolism (see Chapter 13, Fig. 13-2). Leukotrienes are potent bronchoconstrictors, and some also cause airway edema and inflammation, thus contributing to the symptoms of asthma. Because these drugs block the release of some substances from mast cells and eosinophils, they have both bronchodilator and anti-inflammatory effects. These drugs are not indicated for use in the reversal of bronchospasm in acute asthma attacks. They are used for prophylactic and maintenance therapy. One advantage of leukotriene modifiers is that they are administered orally. Leukotriene modifiers can successfully be used as add-on therapy to reduce (not substitute for) the doses of ICS.

Anti-IgE. Omalizumab (Xolair) is a monoclonal antibody to IgE that decreases circulating free IgE levels. Omalizumab prevents IgE from attaching to mast cells, thus preventing the release of chemical mediators. This drug is indicated for patients with moderate to severe persistent, allergic asthma or those requiring step 5 or 6 care with persistent asthma that cannot be controlled with ICSs.[3,4,10] Omalizumab is administered subcutaneously every 2 to 4 weeks and costs about $18,000 per year but most medical insurance will cover it. The drug has a risk of anaphylaxis and patients must receive the medication in a health care provider's office where this emergency can be handled.

Bronchodilators. Three classes of bronchodilator drugs currently used in asthma therapy are β_2-adrenergic agonists (also referred to as β_2-agonists), methylxanthines and derivatives, and anticholinergics (see Table 29-7).

β_2-Adrenergic Agonist Drugs. These drugs may be short-acting β_2-adrenergic agonists (SABAs) or long-acting β_2-adrenergic agonists (LABAs). Because inhaled SABAs are the most effective drugs for relieving acute bronchospasm (as seen in acute exacerbations of asthma), they are known as rescue medications. Examples of these drugs include albuterol (ProAir HFA, Proventil HFA, Ventolin HFA) and pirbuterol (Maxair Autohaler). These drugs have an onset of action within minutes and are effective for 4 to 8 hours. These drugs act by stimulating β-adrenergic receptors in the bronchioles, thus producing bronchodilation. They also increase mucociliary clearance.

DRUG ALERT—β_2-Adrenergic Agonists

- Use with caution in patients with cardiac disorders as they may cause elevation in BP and heart rate, result in central nervous system stimulation/excitation, and increase risk of dysrhythmias (both short and long acting).
- Overuse may cause rebound bronchospasms (short acting).

β_2-Adrenergic agonists are also useful in preventing bronchospasm precipitated by exercise and other stimuli because they prevent the release of inflammatory mediators from mast cells. They do not inhibit the late-phase response of asthma or have antiinflammatory effects. If used frequently, inhaled β_2-adrenergic agonists may produce tremors, anxiety, tachycardia, palpitations, and nausea. Overuse of β_2-adrenergic agonists may cause rebound bronchospasm, which is especially common with albuterol. Too frequent use of β_2-adrenergic agonists indicates poor asthma control, may mask asthma severity, and may lead to reduced drug effectiveness. The goal in asthma therapy is that the patient with persistent asthma will never need to use SABAs for rescue, but that effective control is achieved with a long-term controller medication.

SABAs are not drugs for long-term control, and they should not be used alone for persistent asthma. They are used in any stage of asthma for quick relief and should be with the patient at all times for that purpose. Oral β_2-agonists are rarely used because of the significant cardiovascular side effects, but are used for long-term control. However, they should not be used alone or as first-line therapy in treating asthma.

DRUG ALERT—Long-Acting β_2-Adrenergic Agonists

- Should not be the first medicine used to treat asthma.
- Should never be used as the only medication to treat asthma but should be added to the treatment plan only if other controller medicines do not control asthma.
- Do not use these drugs to treat wheezing that is getting worse.
- Always use a short-acting β_2-agonist to treat sudden wheezing.

LABAs include salmeterol (Serevent) and formoterol (Foradil) and they are effective for 12 hours. LABAs are added to a daily dose of ICSs for long-term control of moderate to severe persistent asthma (i.e., step 3 or higher for long-term control and prevention of symptoms), particularly those at night. LABAs also decrease the need for SABAs, decrease the number of asthma exacerbations, and allow patients to achieve better asthma control with a lower dose of ICSs.[3,4]

LABAs should never be used as monotherapy for asthma, and should only be used if the patient is on an ICS. Tell patients that these drugs should not be used to treat acute symptoms. Teach the patient that these drugs are used only once every 12 hours and are not used to obtain quick relief from bronchospasm.

Combination therapy using an ICS and LABA is available in several inhalers (e.g., fluticasone/salmeterol [Advair] and budesonide/formoterol [Symbicort]. The combinations are more convenient, improve compliance, and ensure that patients receive the LABA together with an ICS.

Methylxanthines. Sustained-release methylxanthine (theophylline) preparations are not a first-line controller medication. They are used only as an alternative therapy for step 2 care in mild persistent asthma. Methylxanthine is a bronchodilator with mild antiinflammatory effects, but the exact mechanism of action is unknown.

DRUG ALERT—Theophylline

- Instruct patient to report signs of toxicity: nausea, vomiting, seizures, insomnia.
- Avoid caffeine to prevent intensifying adverse effects.

The main problem with theophylline is the relatively high incidence of interaction with other drugs and the occurrence of side effects, which include nausea, headache, insomnia, gastrointestinal distress, tachycardia, dysrhythmias, and seizures. Because theophylline has a narrow margin of safety, monitor serum blood levels regularly to determine if the drug is within therapeutic range.

Anticholinergic Drugs. Anticholinergic agents (e.g., ipratropium [Atrovent]) block the bronchoconstricting influence of the parasympathetic nervous system. These drugs are less effective than β_2-adrenergic agonists. Anticholinergic drugs are used for quick relief in those patients unable to tolerate SABAs. In addition, they are used in a severe asthma exacerbation, often nebulized with an SABA, while the patient is being initially treated. The onset of action of ipratropium is slower than β_2-adrenergic agonists, peaking at 30 minutes to 1 hour and lasting up to 4 to 6 hours. Systemic side effects of inhaled anticholinergics are uncommon because they are poorly absorbed. The most common side effect of anticholinergic drugs is a dry mouth.

Inhalation Devices for Drug Delivery. The multiple devices for asthma drug administration can be confusing. The majority of asthma drugs are administered only or preferably by inhalation because systemic side effects are reduced and the onset of action is faster. Inhalation devices include metered-dose inhalers (MDIs), dry powder inhalers (DPIs), and nebulizers.

Inhalers. MDIs are small, hand-held, pressurized devices that deliver a measured dose of drug with each activation. Dosing is usually accomplished with one or two puffs. Nebulizers are small machines used to convert drug solutions into mists. Inhalation of the mist can be done through a face mask or mouthpiece held between the teeth. They are usually used for severe asthma or for individuals who have difficulty with the MDI inhalation.

Individuals who have poor coordination can solve this problem by using a spacer device (AeroChamber, InspirEase) (see Fig. 29-6). Adding a spacer to an MDI also improves the amount of drug delivered to the lungs.

All MDIs are mandated by international law to have an ozone-friendly propellant, which is a hydrofluoroalkane (HFA). This propellant is nontoxic, evaporates almost instantly once it forces medicine out of the MDI canister, and is not harmful to the patient. Before HFAs, the MDI propellants were chlorofluorocarbons (CFCs), which were known to deplete the Earth's ozone layer. In 2010 the U.S. FDA mandated that all MDIs that use CFC be phased out. By 2013 CFC will not be used for any MDI. Patients who have had to switch to the HFA need reassurance that the HFA MDIs are delivering safe, appropriate amounts of drug, but there are differences in what they feel and taste. You must be vigilant in assessing the patient's use of these drugs as some patients do not believe the medication "works" because it is different.

The MDI should be cleaned by removing the dust cap and rinsing it in warm water at least two times per week (Fig. 29-7). The patient who needs to use several MDIs is often unclear about the order in which to take the medications. Historically it has been recommended that short-term β_2-adrenergic agonists

Using an inhaler seems simple, but most patients do not use it the right way. When you use your inhaler the wrong way, less medicine gets to your lungs. (Your physician may give you other types of inhalers.)

For the next 2 weeks, read these steps aloud as you do them or ask someone to read them to you. Ask your physician or nurse to check how well you are using your inhaler.

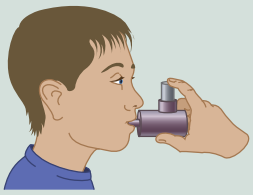
Use your inhaler in one of the three ways pictured below (**A** or **B** is best, but **C** can be used if you have trouble with **A** or **B**).

Steps for Using Your Inhaler

Getting ready	1. Take off the cap and shake the inhaler. 2. Breathe out all the way. 3. Hold your inhaler the way your doctor said (**A, B,** or **C** below).
Breathe in slowly	4. As you start breathing in **slowly** through your mouth, press down on the inhaler **one** time. (If you use a holding chamber, first press down on the inhaler. Within 5 seconds, begin to breathe in slowly.) 5. Keep breathing in **slowly,** as deeply as you can.
Hold your breath	6. Hold your breath as you count to 10 slowly, if you can. 7. For inhaled quick-relief medicine (β_2-agonists), wait about 1 minute between puffs. There is no need to wait between puffs for other medicines.

A. Hold inhaler 1 to 2 inches in front of your mouth (about the width of two fingers).

B. Use a spacer/holding chamber. These come in many shapes and can be useful to any patient.



C. Put the inhaler in your mouth.

Clean Your Inhaler as Needed

Look at the hole where the medicine sprays out from your inhaler. If you see "powder" in or around the hole, clean the inhaler. Remove the metal canister from the L-shaped plastic mouthpiece. Rinse only the mouthpiece and cap in warm water. Let them dry over night. In the morning, put the canister back inside. Put the cap on.

Know When to Replace Your Inhaler

For medicines you take each day (an example):
Say your new canister has 200 puffs (number of puffs is listed on canister) and you are told to take 8 puffs per day.

$$8 \text{ puffs per day} \overline{)\ 200 \text{ puffs in canister}} \quad 25 \text{ days}$$

So this canister will last 25 days. If you started using this inhaler on May 1, replace it on or before May 25.
You can write the date on your canister.

For **quick-relief medicine take as needed** and count each puff.

Do not put your canister in water to see if it is empty as water may enter MDI and impair inhaler.

FIG. 29-7 How to use your metered-dose inhaler correctly.

TABLE 29-8 PROBLEMS ENCOUNTERED WITH METERED-DOSE INHALER (MDI) USE

1. Failing to coordinate activation with inspiration
2. Activating MDI in the mouth while breathing through nose
3. Inspiring too rapidly
4. Not holding the breath for 10 sec (or as close to 10 sec as possible)
5. Holding MDI upside down or sideways
6. Inhaling more than 1 puff with each inspiration
7. Not shaking MDI before use
8. Not waiting a sufficient amount of time between each puff
9. Not opening mouth wide enough, causing medication to bounce off teeth, tongue, or palate
10. Not having adequate strength to activate MDI
11. Unable to understand and incorporate directions

should be used first to open up the airway and improve the delivery of subsequent medications. However, this is no longer recommended because there is no evidence demonstrating that it is beneficial, and it is a potential source of confusion to patients because the short-term β_2-adrenergic agonists are usually used on an as-needed (PRN) basis.[11]

One of the major problems with metered-dose drugs is the potential for overuse, that is, using them much more frequently than prescribed (more than two canisters per month) rather than seeking needed medical care (Table 29-8). As a patient develops additional asthmatic symptoms, she or he may use the β_2-adrenergic agonist MDI repeatedly. β_2-Adrenergic agonists help by relieving bronchospasm; they do not treat the inflammatory response. Therefore teach the patient the correct therapeutic use of these drugs. The patient needs to know the correct way to determine if the MDI is empty (see Fig. 29-7). In the past, floating the MDI in water was an appropriate way to determine if medication remained in the MDI. Now this is not recommended because it is not accurate and water can enter the MDI. Teach patients that shaking the canister is not an accurate way to determine if the MDI is empty because they may be hearing only the propellant when the MDI is nearly empty.

DPIs are simpler to use than MDIs (Fig. 29-8 and Table 29-9). The DPI contains dry, powdered medication and is breath activated. No propellant is used; instead an aerosol is created when the patient inhales through a reservoir containing a dose of powder. The convenient-to-carry diskus has several advantages over MDIs: (1) less manual dexterity is needed; (2) there is no need to coordinate device puffs with inhalation; (3) an easily visible color or number system indicates the number of doses left in the diskus; and (4) no spacer is required. Disadvantages are that commonly prescribed drugs are not yet available in DPIs and the medication may clump if exposed to humidity. Since the medicine is only delivered by the patient's inspiratory effort, patients with a low FEV_1 (less than 1 L) may not be able to inspire the medication adequately.

Differences between MDIs and DPIs are presented in Table 29-10. Aerosolized medication delivery systems, when used with comparable drug doses, provide equivalent efficacy. Therefore patients should use the device best suited to their needs.

Nebulizers. Nebulizers are devices that deliver a suspension of fine particles of liquid in a gas. Medication is *nebulized,* or reduced to a fine spray. Nebulizers are usually powered by a compressed air or O_2 generator. At home the patient may have an air-powered compressor; in the hospital, wall O_2 or compressed air is used to power the nebulizer.

FIG. 29-8 Example of a dry powder inhaler (DPI).

TABLE 29-9 PATIENT AND CAREGIVER TEACHING GUIDE

How to Use a Dry Powder Inhaler (DPI)

You should include the following instructions when teaching a patient to use a dry powder inhaler:

1. Remove mouthpiece cap or open the device according to manufacturer's instructions. Check for dust or dirt. If there is an external counter, note the number of doses remaining.
2. Load the medicine into the inhaler or engage the lever to allow the medicine to become available. Some DPIs should be held upright while loading. Others should be held sideways or in a horizontal position.
3. Do not shake your medicine.
4. Tilt your head back slightly and breathe out, getting as much air out of your lungs as you can. Do not breathe into your inhaler because this could affect the dose.
5. Close your lips tightly around the mouthpiece of the inhaler.
6. Breathe in deeply and quickly. This will ensure that the medicine moves down deeply into your lungs. You may not taste or sense the medicine going into your lungs.
7. Hold your breath for 10 seconds or as long as you can to disperse the medicine into your lungs.
8. If there is an external counter, note the number of doses remaining. It should be one less than the number in step 1 above.
9. Do not keep your DPI in a humid place such as a shower room because the medicine may clump.

Aerosolized medication orders must include the medication, dose, diluent, and whether it is to be nebulized with O_2 or compressed air. The advantage of nebulization therapy is that it is easy to use. Medications that are routinely nebulized include albuterol and ipratropium.

The patient is placed in an upright position that allows for most efficient breathing to ensure adequate penetration and deposition of the aerosolized medication. The patient must breathe slowly and deeply through the mouth and hold inspiration for 2 or 3 seconds. Deep diaphragmatic breathing helps

TABLE 29-10 COMPARISON OF METERED-DOSE AND DRY POWDER INHALERS

	METERED-DOSE INHALER (MDI)	DRY POWDER INHALER (DPI)
Shake before use	Yes, shake well*	No
Inspiration	Slow	Rapid
Spacer	Yes, at least with inhaled corticosteroids*	None permitted
Counting device	Few have external device	Most preloaded forms include counter
Inhalations/dose	Often 2/dose	Often 1/dose
Cleaning	Use water for plastic case	Avoid moisture

*Most MDIs use hydrofluoroalkane (HFA) as a propellant and shaking or a spacer may not be needed. For a large list of devices and downloadable patient teaching instructions, see *www.ginasthma.org*.

TABLE 29-11 NONPRESCRIPTION ASTHMA DRUGS

DRUG PRODUCT	INGREDIENTS	
	SYMPATHOMIMETIC	EXPECTORANT
Broncholate Syrup	Ephedrine	Guaifenesin
Bronkaid Dual Action Caplets	Ephedrine	Guaifenesin
Mini Ephedrine Two-Way Action Tablets	Ephedrine	Guaifenesin
Primatene tablets	Ephedrine	Guaifenesin
Bronkaid Mist	Epinephrine	
Primatene Mist Inhaler	Epinephrine	

ensure deposition of the medication. Instruct the patient to breathe normally in between these large forced breaths to prevent alveolar hypoventilation and dizziness. After the treatment instruct the patient to cough effectively.

A disadvantage of nebulizer equipment use is the potential for bacterial growth. Because home nebulization is used for the patient with COPD, it is important for you to review cleaning procedures for home respiratory equipment with the patient. A frequently used, effective home-cleaning method is to wash the nebulizer daily in soap and water, rinse it with water, and soak it for 20 to 30 minutes in a 1:1 white vinegar–water solution followed by a water rinse and air drying. Commercial respiratory cleaning agents may also be used if directions are followed carefully. Cleaning the nebulizer in the top shelf of an automatic dishwasher saves time, and the hot water destroys most organisms.

Patient Teaching Related to Drug Therapy. Information about medications should include the name, purpose, dosage, method of administration, and schedule, taking into consideration activities of daily living (ADLs) that require energy expenditure and thus oxygen, such as bathing. Teaching should also include side effects, appropriate action if side effects occur, how to properly use and clean devices, and consequences for breathing if not taking medications as prescribed. One of the major factors determining success in asthma management is the correct administration of drugs.

Poor adherence with asthma therapy is a major challenge in the long-term management of chronic asthma. Lack of adherence often occurs because the patient has no symptoms. Thus the patient does not realize that the inflammatory process is ongoing at all times and the patient needs ICSs. In addition, the inhaled medications are expensive and patients may not be able to afford them. The patient will use β_2-adrenergic agonist inhalers because they provide immediate relief of symptoms. However, if the patient is symptom free, he or she often does not use the long-term therapy (e.g., ICS) regularly because no immediate benefit is felt. Explain to the patient the importance and purpose of taking the long-term therapy regularly, emphasizing that maximum improvement may take longer than 1 week. It is important to emphasize that without regular use, the swelling in the airways may progress and the asthma will likely worsen over time. Become familiar with the vast array of compassionate use programs offered by the pharmaceutical companies to help lower-income patients obtain medications (e.g., *www.needymeds.com*).

In addition to the typical MDI and DPI devices, a variety of other devices are used to deliver inhalant pulmonary medications. Be certain that the patient understands exactly how to use the device, and printed instructions should be given. (See *http://asthma.nationaljewish.org/treatments/devices* for instructions.) Most inhalant drugs have very clear patient instructions, but you need to either use a placebo device or the actual drug to assess the patient's ability to deliver the medication. At every visit reassess the patient's understanding of how to deliver the drugs.

New drug delivery devices are becoming available at a rapid pace and you must keep informed of the correct operation of these. If there are package inserts available, you can review them before teaching the patient. The drug company websites have numerous excellent teaching videos available. One promising delivery device soon to be available in the United States is the Respimat, which is an easy-to-use hand-held device that provides a high deposition of drug to the lungs and low mouth and throat deposition. The Respimat simplifies coordination between activation of the medication and inhalation yet there is no propellant and it is independent of inspiratory flow. It also has a dose indicator.

Nonprescription Combination Drugs. Several nonprescription combination drugs are available OTC. They are usually combinations of a bronchodilator and an expectorant (Table 29-11). These agents are advertised as drugs to relieve bronchospasm. In general they should be avoided. Many persons consider these drugs safe because they can be obtained without a prescription. Dangers exist in drugs containing epinephrine as it acts only for a short time and may increase the patient's heart rate and blood pressure. This drug is not recommended for use.

Drugs containing ephedrine (found in many OTC decongestants) cause stimulation of the central nervous and cardiovascular systems. Side effects include nervousness, heart palpitations and dysrhythmias, tremors, insomnia, and increases in blood pressure. Many OTC respiratory products contain ephedrine and as of September 2006 are located behind the counter at pharmacies or a prescription is required. This limited access is to prevent the diversion of ephedrine to the production of methamphetamine. Also many of the OTC products have been

reformulated with phenylephrine, instead of ephedrine, which works well topically but has very modest effects in the oral form.

An important teaching responsibility is to warn the patient about the dangers associated with nonprescription combination drugs. These drugs are especially dangerous to a patient with underlying cardiac problems because elevated blood pressure and tachycardia often occur. Caution the patient who persists in taking one of these medications to read and follow the accompanying directions on the label. Often patients seek OTC drugs as they are less expensive than prescription medication. Patients should be informed that after December, 2011 OTC sprays with epinephrine will no longer be available as they use the propellant CFC that is harmful to the environment. Whether a reformulation using HFA will be developed is yet to be determined. In the meantime patients need to be counseled to seek professional medical help to determine the best drug to use to control the inflammation of asthma.[12]

NURSING MANAGEMENT ASTHMA

NURSING ASSESSMENT

If a patient can speak and is not in acute distress, a detailed health history, including identification of any precipitating factors and what has helped alleviate attacks in the past, can be taken. Subjective and objective data that should be obtained from a patient with asthma are presented in Table 29-12. You may also assess the patient's asthma control using one of the validated self-administered questionnaires (e.g., ACT in Fig. 29-5).

NURSING DIAGNOSES

Nursing diagnoses for the patient with asthma may include, but are not limited to, those presented in NCP 29-1.

PLANNING

The overall goals are that the patient with asthma will have asthma control as evidenced by (1) minimal symptoms during the day and night, (2) acceptable activity levels (including exercise and other physical activity), (3) maintenance of greater than 80% of personal best PEFR or FEV_1, (4) few or no adverse effects of therapy, (5) no recurrent exacerbations of asthma, and (6) adequate knowledge to participate in and carry out management.

NURSING IMPLEMENTATION

HEALTH PROMOTION. Your role in preventing asthma attacks or decreasing the severity focuses primarily on teaching the patient and caregiver. Teach the patient to identify and avoid known personal triggers for asthma (e.g., cigarette smoke, pet dander) and irritants (e.g., cold air, aspirin, foods, cats, indoor air pollution) (see Table 29-1). Use of special dust covers on mattresses and pillows may reduce exposure to dust mites and improve symptoms. Washing bedclothes in hot water or cooler water with detergent and bleach has some effect on allergen levels. Avoidance of furred animals is suggested, but the allergen of pets is nearly impossible to avoid. Pet allergen can be found in many public areas for many months even after removal of the animal. Many people are allergic to cockroach remains and the dried droppings, so measures to avoid or control cockroaches are partly effective in removing allergens.[3]

TABLE 29-12 NURSING ASSESSMENT

Asthma

Subjective Data

Important Health Information

Past health history: Allergic rhinitis, sinusitis, or skin allergies; previous asthma attack and hospitalization or intubation; symptoms worsened by triggers in the environment; gastroesophageal reflux; occupational exposure to chemical irritants (e.g., paints, dust)

Medications: Adherence to medication, inhaler technique; use of antibiotics; pattern and amount of short-acting β-adrenergic agonist used per week; medications that may precipitate an attack in susceptible asthmatics such as aspirin, nonsteroidal antiinflammatory drugs, β-adrenergic blockers

Functional Health Patterns

Health perception–health management: Family history of allergies or asthma; recent upper respiratory infection or sinus infection

Activity-exercise: Fatigue, decreased or absent exercise tolerance; dyspnea, cough (especially at night), productive cough with yellow or green sputum or sticky sputum; chest tightness, feelings of suffocation, air hunger, talk in short sentences or words/phrases, sitting upright to breathe

Sleep-rest: Awakened from sleep because of cough or breathing difficulties, insomnia

Coping–stress tolerance: Emotional distress, stress in work environment or home

Objective Data

General

Restlessness or exhaustion, confusion, upright or forward-leaning body position

Integumentary

Diaphoresis, cyanosis (circumoral, nail bed), eczema

Respiratory

Nasal discharge, nasal polyps, mucosal swelling; wheezing, crackles, diminished or absent breath sounds, and rhonchi on auscultation; hyperresonance on percussion; sputum (thick, white, tenacious), ↑ work of breathing with use of accessory muscles; intercostal and supraclavicular retractions; tachypnea with hyperventilation; prolonged expiration

Cardiovascular

Tachycardia, pulsus paradoxus, jugular venous distention, hypertension or hypotension, premature ventricular contractions

Possible Diagnostic Findings

Abnormal ABGs during attacks, ↓ O_2 saturation, serum and sputum eosinophilia, ↑ serum IgE, positive skin tests for allergens, chest x-ray demonstrating hyperinflation with attacks, abnormal pulmonary function tests showing ↓ flow rates; FVC, FEV_1, PEFR, and FEV_1/FVC ratio that improve between attacks and with bronchodilators

FEV_1, Forced expiratory volume in 1 second; *FVC,* forced vital capacity; *PEFR,* peak expiratory flow rate.

If cold air cannot be avoided, dressing properly with scarves or using a mask helps reduce the risk of an asthma attack. Aspirin and NSAIDs should be avoided if they are known to precipitate an attack. Many OTC drugs contain aspirin. Therefore teach the patient to read the labels carefully. Nonselective β blockers (e.g., propranolol [Inderal]) are contraindicated because they inhibit bronchodilation. Selective β blockers (e.g., atenolol) should be used with caution. Desensitization (immunotherapy) may be partially effective in decreasing the patient's sensitivity to known allergens (see Chapter 14).

NURSING CARE PLAN 29-1

Patient with Asthma

NURSING DIAGNOSIS **Ineffective airway clearance** *related to* bronchospasm, excessive mucus production, tenacious secretions, and fatigue *as evidenced by* ineffective cough, inability to raise secretions, adventitious breath sounds

PATIENT GOALS
1. Maintains clear airway with removal of excessive secretions
2. Experiences normal breath sounds and respiratory rate

OUTCOMES (NOC)	INTERVENTIONS (NIC) AND *RATIONALES*
Respiratory Status: Airway Patency	***Asthma Management***
• Respiratory rate _____ • Respiratory rhythm _____ • Moves sputum out of airway _____ • Ease of breathing _____ **Measurement Scale** 1 = Severely compromised 2 = Substantially compromised 3 = Moderately compromised 4 = Mildly compromised 5 = Not compromised	• Determine baseline respiratory status *to use as a comparison point.* • Monitor rate, rhythm, depth, and effort of respiration *to determine need for intervention and evaluate effectiveness of interventions.* • Observe chest movement, including symmetry, use of accessory muscles, and supraclavicular and intercostal muscle retractions, *to evaluate respiratory status.* • Auscultate breath sounds, noting areas of decreased/absent ventilation and adventitious sounds, *to evaluate respiratory status.* • Administer medication as appropriate and/or per policy and procedural guidelines *to improve respiratory function.* • Coach in breathing/relaxation techniques *to improve respiratory rhythm and rate.* • Offer warm fluids to drink *to liquefy secretions and promote bronchodilation.*

NURSING DIAGNOSIS **Anxiety** *related to* difficulty breathing, perceived or actual loss of control, and fear of suffocation *as evidenced by* restlessness, elevated pulse, respiratory rate, and blood pressure

PATIENT GOALS
1. Reports decreased anxiety with increased control of respirations
2. Experiences vital signs within normal limits

OUTCOMES (NOC)	INTERVENTIONS (NIC) AND *RATIONALES*
Anxiety Level	***Anxiety Reduction***
• Restlessness _____ • Increased blood pressure _____ • Increased pulse rate _____ • Increased respiratory rate _____ • Verbalized anxiety _____ • Facial tension _____ **Measurement Scale** 1 = Severe 2 = Substantial 3 = Moderate 4 = Mild 5 = None	• Identify when level of anxiety changes *to determine possible precipitating factors.* • Use calm, reassuring approach *to provide reassurance.* • Stay with patient *to promote safety and reduce fear.* • Encourage verbalization of feelings, perceptions, and fears *to identify problem areas so appropriate planning can take place.* • Provide factual information concerning diagnosis, treatment, and prognosis *to help patient know what to expect.* • Instruct patient in the use of relaxation techniques *to relieve tension and to promote ease of respirations.*

NURSING DIAGNOSIS **Deficient knowledge** *related to* lack of information and education about asthma and its treatment *as evidenced by* frequent questioning regarding all aspects of long-term management

PATIENT GOALS
1. Describes the disease process and treatment regimen
2. Demonstrates correct administration of aerosol medications
3. Expresses confidence in ability for long-term management of asthma

OUTCOMES (NOC)	INTERVENTIONS (NIC) AND *RATIONALES*
Asthma Self-Management	***Asthma Management***
• Describes causal factors _____ • Initiates action to avoid and manage personal triggers _____ • Seeks early treatment of infections _____ • Monitors peak flow routinely _____ • Monitors peak flow when symptoms occur _____ • Makes appropriate medication choices _____ • Demonstrates appropriate use of inhalers, spacers, and nebulizers _____ • Self-manages exacerbations _____ • Reports uncontrolled symptoms to health care provider _____ **Measurement Scale** 1 = Never demonstrated 2 = Rarely demonstrated 3 = Sometimes demonstrated 4 = Often demonstrated 5 = Consistently demonstrated	• Determine patient/family understanding of disease and management *to assess learning needs.* • Teach patient to identify and avoid triggers as possible *to prevent asthma attacks.* • Encourage verbalization of feelings about diagnosis, treatment, and impact on lifestyle *to offer support and increase compliance with treatment.* • Educate patient about the use of the peak expiratory flow rate (PEFR) meter at home *to promote self-management of symptoms.* • Instruct patient/family on antiinflammatory and bronchodilator medications and their appropriate use *to promote understanding of effects.* • Teach proper techniques for using medication and equipment (e.g., inhaler, nebulizer, peak flow meter)* *to promote self-care.* • Assist in the recognition of signs/symptoms of impending asthmatic reaction and implementation of appropriate response measures *to prevent escalation of attacks.* • Establish a written plan with the patient for managing exacerbations *to plan adequate treatment of future exacerbations.*

*See Tables 29-7, 29-8, 29-10 and Fig. 29-7.

Prompt diagnosis and treatment of upper respiratory tract infections and sinusitis may prevent an exacerbation of asthma. If occupational irritants are involved as etiologic factors, the patient may need to consider changing jobs. Individuals who are obese often find that weight loss will improve control of asthma. Treatment of GERD and preventive measures for it may increase asthma control. Encourage the patient to maintain a fluid intake of 2 to 3 L per day, good nutrition, and adequate rest. If exercise is planned or if the patient only has asthma with exercise, the health care provider can suggest a medication regimen for pretreatment or long-term control of symptoms to prevent bronchospasm.

ACUTE INTERVENTION. A goal in asthma care is to maximize the ability of the patient to safely manage acute asthma exacerbations via an asthma action plan developed in conjunction with the health care provider (Table 29-13). Action plans are particularly important for those individuals with moderate to severe persistent asthma or severe exacerbations. The action plan will dictate what symptoms or peak flow reading necessitates a change in asthma care to gain control. The patient can take two

TABLE 29-13 ASTHMA ACTION PLAN*

General Information

- Name _____
- Emergency contact _____
- Physician/health care provider _____
- Physician signature _____

Phone numbers _____
Phone numbers _____
Date _____

Severity Classification

❍ Mild intermittent ❍ Moderate persistent
❍ Mild persistent ❍ Severe persistent

Triggers

❍ Colds ❍ Smoke ❍ Weather
❍ Exercise ❍ Dust ❍ Air pollution
❍ Animals ❍ Food
❍ Other _____

Exercise

1. Premedication (how much and when) _____
2. Exercise modifications _____

Green Zone: Doing Well

Symptoms

- Breathing is good
- No cough or wheeze
- Can work and play
- Sleeps all night

Peak Flow Meter
More than 80% of personal best or _____

Peak Flow Meter Personal Best =

Control Medications

Medicine	How Much to Take	When to Take It
_____	_____	_____
_____	_____	_____
_____	_____	_____

Yellow Zone: Getting Worse

Symptoms

- Some problems breathing
- Cough, wheeze, or chest tight
- Problems working or playing
- Wake at night

Peak Flow Meter
Between 50% and 80% of personal best or _____ and _____

Contact Physician if Using Quick Relief More than Two Times per Week

Continue Control Medicines and Add:

Medicine	How Much to Take	When to Take It
_____	_____	_____
_____	_____	_____
_____	_____	_____

IF your symptoms (and peak flow, if used) return to Green Zone after 1 hour of the quick-relief treatment, THEN

❍ Take quick-relief medication every 4 hours for 1 or 2 days
❍ Change your long-term control medicines by _____
❍ Contact your physician for follow-up care

IF your symptoms (and peak flow, if used) do NOT return to the Green Zone after 1 hour of the quick-relief treatment, THEN

❍ Take quick-relief treatment again
❍ Change your long-term control medicines by _____
❍ Call your physician/health care provider within _____ hours of modifying your medication routine

Red Zone: Medical Alert

Symptoms

- Lots of problems breathing
- Cannot work or play
- Getting worse instead of better
- Medicine is not helping

Peak Flow Meter
Between 0% and 50% of personal best or _____ and _____

Ambulance/Emergency Phone Number

Continue Control Medicines and Add

Medicine	How Much to Take	When to Take It
_____	_____	_____
_____	_____	_____
_____	_____	_____

Go to the hospital or call for an ambulance if

❍ Still in the red zone after 15 minutes
❍ You have not been able to reach your physician/health care provider for help

Call an ambulance immediately if the following danger signs are present

❍ Trouble walking/talking due to shortness of breath
❍ Lips or fingernails are blue

Source: Reproduced with permission © 2010, American Lung Association, available at *www.lungusa.org.*
*Available in Spanish on the Evolve website for this chapter.

to four puffs of an SABA every 20 minutes three times as a rescue plan. Depending on the response with alleviation of symptoms or improved peak flow, continued SABA use and/or oral corticosteroids may be a part of the home management plan at this point. If symptoms persist or if the patient's peak flow is less than 50% of the personal best, the health care provider or emergency medical services (EMS) needs to be immediately contacted.

When the patient is in the health care facility with an acute exacerbation it is important for you to monitor the patient's respiratory and cardiovascular systems. This includes auscultating lung sounds; taking the heart and respiratory rate and BP; and monitoring ABGs, pulse oximetry, and peak flow.

You should note that louder wheezing may actually occur in the airways that are responding to the therapy as airflow in the airways increases. As improvement continues and airflow increases, breath sounds increase and wheezing decreases. As the patient begins to respond to therapy and symptoms begin to subside, it is important to remember that despite the disappearance of most of the bronchospasm, the edema and cellular infiltration of the airway mucosa and the viscous mucous plugs may take several days to improve. Thus intensive therapy must be continued even after clinical improvement has occurred.

An important nursing goal during an acute attack is to decrease the patient's sense of panic. A calm, quiet, reassuring attitude may help the patient relax. Position the patient comfortably (usually sitting) to maximize chest expansion. You need to stay with the patient and be available to provide additional comfort. A technique called "talking down" can help the patient to remain calm. In talking down you gain eye contact with the patient. In a firm, calm voice coach the patient to use pursed-lip breathing, which keeps the airways open by maintaining positive pressure (Table 29-14) and abdominal breathing, which slows the respiratory rate and encourages deeper breaths. You or the caregiver should stay with the patient until the respiratory rate (with the assistance of the medications) has slowed.

When the acute attack subsides, you should provide rest and a quiet, calm environment for the patient. When the patient has recovered from exhaustion, you should attempt to obtain the health history and pattern of asthma along with a physical assessment. If the caregiver and other family members are present, they may be able to provide information about the patient's health history (see Table 29-12). This information, which is important in planning an individualized nursing care plan, helps the patient with the goal of control.

AMBULATORY AND HOME CARE. Control of symptoms can also be obtained by educating the patient about medications. The drug regimen itself can be confusing and complex. Teach patients the need and importance of monitoring their response to medication. It is easy to undermedicate or overmedicate unless careful monitoring is ongoing. Some patients may benefit from keeping a diary to record medication use, the presence of wheezing or coughing, peak flow, the drug's side effects, and the activity level. This information will be valuable in helping the health care provider adjust the medication.

Good nutrition is important. Physical exercise (e.g., swimming, walking, stationary cycling) within the patient's limit of tolerance is also beneficial and may require pretreatment with an SABA as noted previously. Sleep that is uninterrupted by asthma symptoms is an important goal. If patients with asthma wake up because of asthma symptoms, their asthma is not under good control and their therapeutic plan should be reevaluated.

Together with the patient and caregiver, develop written asthma action plans (see Table 29-13). Base these plans on the patient's asthma symptoms and peak expiratory flow rate (PEFR). To follow the management plan, the patient must measure his or her peak flow at least daily. Some patients may desire to just follow symptoms for self-management. Patients with asthma frequently do not perceive changes in their breathing. Therefore peak flow monitoring, when done correctly, can be a reliable, objective measurement of asthma control (Table 29-15).

If a patient's PEFR is within the green zone (usually 80% to 100% of the person's personal best), the patient should remain on her or his usual medications. If the PEFR is within the yellow zone (usually 50% to 80% of personal best), it indicates caution. Something is triggering the patient's asthma (e.g., viral infection). The patient should have a written asthma action plan that prescribes a step increase in medications during the acute phase of the infection. The dose is stepped down once the viral infection symptoms subside. Different strategies may be used by the patient based on the asthma management plan. For example, the patient could use the SABA more frequently.

If the PEFR is in the red zone (50% or less of personal best), it indicates a serious problem. A rescue plan should be a part of the asthma action plan. Teach the patient and caregiver to take definitive action as noted previously. It is important to emphasize to the patient the need to monitor PEFR daily or several times a day to have an objective measure that can be correlated with symptoms. Although it may occur, it is unusual for a patient's PEFR to drop from the green zone to the red zone quickly. Usually the patient has time to make changes in medications, avoid triggers, and notify the health care provider.

It is very important to involve the patient's caregiver or family. They should know where the patient's inhalers, oral medications, and emergency phone numbers are located. Instruct the caregiver or family on how to decrease the patient's anxiety if an asthma attack occurs. When the patient is stabilized or controlled, the caregiver can gently remind the patient about doing daily PEFR by asking questions such as, "What zone are you in? How's your peak flow today?" A patient and caregiver teaching guide for the patient with asthma is presented in Table 29-16.

TABLE 29-14 PATIENT AND CAREGIVER TEACHING GUIDE

Guidelines for Pursed-Lip Breathing (PLB)

You should teach the patient how to do pursed-lip breathing using the following guidelines.

1. Use PLB before, during, and after any activity causing you to be short of breath.
2. Inhale slowly and deeply through the nose.
3. Exhale slowly through pursed lips, almost as if whistling.
4. Relax your facial muscles without puffing your cheeks—like whistling—while you are exhaling.
5. Make breathing out (exhalation) three times as long as breathing in (inhalation).
6. The following activities can help you get the "feel" of PLB:
   - Blow through a straw in a glass of water with the intent of forming small bubbles.
   - Blow a lit candle enough to bend the flame without blowing it out.
   - Steadily blow a table-tennis ball across a table.
   - Blow a tissue held in the hand until it gently flaps.
7. Practice 8-10 repetitions of PLB three or four times a day.

TABLE 29-15 PATIENT AND CAREGIVER TEACHING GUIDE

How to Use Your Peak Flow Meter

You should include the following instructions when teaching the patient to use a peak flow meter.

Why Use a Peak Flow Meter?

- Peak flow meters are used to check your asthma the way that blood pressure cuffs are used to check blood pressure. A peak flow meter is a device that measures how well air moves out of your lungs.
- During an asthma episode, the airways of the lungs usually begin to narrow slowly. The peak flow meter may tell you if there is narrowing in the airways hours—sometimes even days—before you have any asthma symptoms.
- By taking your medicine(s) early (before symptoms), you may be able to stop the episode quickly and avoid a severe asthma episode.
- The peak flow meter also can be used to help you and your health care provider
  - Learn what makes your asthma worse.
  - Decide if your treatment plan is working well.
  - Decide when to add or stop medicine.
  - Decide when to seek emergency care.

How to Use Your Peak Flow Meter

- Do the following five steps with your peak flow meter:
  1. Move the indicator to the bottom of the numbered scale.
  2. Stand up.
  3. Take a deep breath, filling your lungs completely.
  4. Place the mouthpiece in your mouth and close your lips around it. Do not put your tongue inside the hole.
  5. Blow out as hard and fast as you can in a single blow.
- Write down the number you get. But if you cough or make a mistake, do not write down the number. Do it over again.
- Repeat steps 1 through 5 two more times, and write down the best of the three blows in your asthma diary.

Find Your Personal Best Peak Flow Number

- Your personal best peak flow number is the highest peak flow number you can achieve over a 2-week period when your asthma is under good control. Good control is when you feel good and do not have any asthma symptoms.
- Each patient's asthma is different, and your best peak expiratory flow (PEF) may be higher or lower than the peak flow of someone of your same height, weight, and sex. This means that it is important for you to find your own personal best peak flow number. Your treatment plan needs to be based on your own personal best peak flow number.
- To find out your personal best peak flow number, take peak flow readings:
  - At least twice a day for 2-3 weeks.
  - When you wake up and in late afternoon or early evening.
  - 15-20 minutes after taking inhaled short-acting β_2-agonist for quick relief.
  - As instructed by your health care provider.

The Peak Flow Zone System

Once you know your personal best peak flow number, your health care provider will give you the numbers that tell you what to do. The peak flow numbers are put into zones that are set up like a traffic light. This will help you know what to do when your peak flow number changes. For example:

- Green Zone (more than __L/min [80% of your personal best number]) signals good control. No asthma symptoms are present. Take your medicines as usual.
- Yellow Zone (between __L/min and __L/min [50% to less than 80% of your personal best number]) signals caution. If you remain in the yellow zone after several measures of peak flow, take an inhaled short-acting β_2-agonist. If you continue to register peak flow readings in the yellow zone, your asthma may not be under good control. Ask your doctor if you need to change or increase your daily medicines.
- Red Zone (below __L/min [less than 50% of your personal best number]) signals a medical alert. You must take an inhaled short-acting β_2-agonist (quick-relief medicine) right away. Call your health care provider or the emergency department and ask what to do, or go directly to the hospital emergency department.

Use the Diary to Keep Track of Your Peak Flow

- Record your personal best peak flow number and peak flow zones in your asthma diary.
- Measure your peak flow when you wake up, *before* taking medicine. Write down your peak flow number in the diary every day, or as instructed by your health care provider.

Actions to Take When Peak Flow Numbers Change

- PEF goes between __L/min and __L/min (50% to less than 80% of personal best, yellow zone).
  ACTION: Take an inhaled short-acting β_2-agonist (quick-relief medicine) as prescribed by your doctor.
- PEF increases 20% or more when measured before and after taking an inhaled short-acting β_2-agonist (quick-relief medicine).
  ACTION: Talk to your doctor about adding more medicine to control your asthma better (for example, an antiinflammatory medication).

Source: Adapted from *Expert Panel Report 3: Guidelines for the Diagnosis and Management of Asthma*. National Asthma Education and Prevention Program, National Heart, Lung, and Blood Institute, 2007.

An increased number of older adults are diagnosed with asthma. This is a special concern because they have more complicated health issues than younger patients with asthma. Issues that face older adults (especially urban and minority people) are costly medications, nonadherence to medical regimen, and difficulty in accessing the health system. Keep these factors in mind when implementing a management plan for older adults.

A variety of factors may contribute to African Americans and Hispanics (especially those from Puerto Rico) having higher rates of poorly controlled asthma and deaths compared with whites. Some factors may be disparities in socioeconomic status and access to proper health care, cultural beliefs about the management of asthma, and underutilization of the long-term controller medications because of high costs. You should seek to explore and eliminate any barriers to health care. You should also seek out culturally appropriate resources and education material that is available in languages other than English in order to improve the asthma control of these individuals.

Relaxation therapies (e.g., yoga, meditation, relaxation techniques, breathing techniques) may help a patient relax respiratory muscles and decrease the respiratory rate. (Chapter 8 discusses relaxation breathing and other relaxation strategies.) A healthy emotional outlook can also be important in preventing future asthma attacks. A variety of websites have excellent resources for patient teaching (see Resources at the end of the chapter). Some communities have asthma support groups.

EVALUATION

The expected outcomes for the patient with asthma are presented in NCP 29-1.

TABLE 29-16 PATIENT AND CAREGIVER TEACHING GUIDE

Asthma

You should include the following information in the teaching plan.

Goal

To assist patient in improving quality of life through education, increased understanding, and promotion of lifestyle practices that support successful living with asthma.

TEACHING TOPIC	RESOURCES
What Is Asthma?	
• Basic anatomy and physiology of lung • Pathophysiology of asthma • Relationship of pathophysiology to signs and symptoms • Measurement and correlation of pulmonary function tests and peak expiratory flow rate	*What Is Asthma?* (National Institutes of Health/National Heart, Lung, and Blood Institute) Available at *www.nhlbi.nih.gov/health/dci/Diseases/Asthma/Asthma_WhatIs.html* About Asthma? (Global Initiative for Asthma). Available at *www.ginasthma.com/Patients.asp?l1=3&;l2=0* Understanding Asthma (National Jewish Health). Available at *www.njhealth.org/healthinfo/conditions/asthma/index.aspx*
What Is Good Asthma Control?	Resource for patient on personal ideas of good control *Asthma Control Test* (American Lung Association) (Fig 29-5). Also in Spanish. Available at *www.asthmacontrol.com*
Hindrances to Asthma Treatment and Control	
• Intermittent nature of symptoms • Role of denial • Poor perception of asthma severity by patient	Discussion with patient and caregiver about possible hindrances
Environmental/Trigger Control	
• Identifications of possible triggers and possible preventive measures • Avoidance of allergens and other triggers • Need to maintain good hydration	*Home Control of Asthma and Allergies* (American Lung Association). Available at *www.lungusa.org* (search "Home control") *Pollen count report. Sign up for daily notification about a particular area* (American Academy of Asthma Allergy and Immunology). Available at *www.aaaai.org/nab* Trigger diary kept by patient
Medications	
Types (include mechanism of action) • β_2-agonists • Corticosteroids • Methylxanthines • Leukotriene modifiers Establishing medication schedule Use of preventive/maintenance agents (e.g., anti-inflammatory agents)	*Pocket Guide for Asthma Management and Prevention* (Global Initiative for Asthma). Available under Guidelines and Resources at *www.ginasthma.org* Asthma Action Plan (see Table 29-13) Write out medication list and schedule

TEACHING TOPIC	RESOURCES
Correct Use of Inhalers, Spacer, and Nebulizer	Demonstration–return demonstration with placebo devices (see Figs. 29-6, 29-7, and 29-8; see Tables 29-8, 29-9, and 29-10) *Instructions for Inhaler and Spacer Use* (Global Initiative for Asthma). Available under Guidelines and Resources at *www.ginasthma.org*
Breathing Techniques	
• Pursed-lip breathing	See Table 29-14
Correct Use of Peak Flow Meter	See Table 29-15 *Peak Flow Meters?* (American Lung Association). Available at *www.lungusa.org*—search "peak flow meters"
Asthma Action Plan	
• Peak flow zones • Individualize plan • Early recognition of infection • Keeping a partnership with your health care provider • Questions patients may have about asthma, but patient cannot reach the provider	See Table 29-13 Patient completes asthma action plan and discusses it with health care provider Asthma research (American Lung Association). Available at *www.lungusa.org*—search "asthma research/studies" Wallet card with personalized information: Available at National Heart, Lung, and Blood Institute: *www.nhlbi.nih.gov/health/public/lung/asthma/asthma_walletcard.htm* *Asthma: Nex Profiler Treatment Option Tool.* Online decision support tool assists patients in understanding treatment options and side effects and provides them with personalized questions to ask the provider in addition to research reports (American Lung Association). Available at *www.lungusa.org* *Lung Line:* Ask a specialized nurse questions about early detection, care, and prevention of respiratory, allergic, and immune diseases (National Jewish Health). Telephone: 1-800-222-LUNG (5864) or (outside the United States) 303-355-5864 (7700) or *www.njhealth.org/about/contact/lung-line.aspx*

CHRONIC OBSTRUCTIVE PULMONARY DISEASE

Chronic obstructive pulmonary disease (COPD) is a preventable and treatable disease state characterized by chronic airflow limitation that is not fully reversible. The airflow limitation is usually progressive and associated with an abnormal inflammatory response of the lungs to noxious particles or gases, primarily caused by cigarette smoking. COPD also results in some significant systemic effects that contribute to the severity of disease exhibited in individual patients.[2,13]

Previous definitions of COPD encompassed two types of obstructive airway diseases, chronic bronchitis and emphysema. **Chronic bronchitis** is the presence of chronic productive cough for 3 months in each of 2 consecutive years in a patient in whom other causes of chronic cough have been excluded. **Emphysema** is an abnormal permanent enlargement of the air spaces distal to the terminal bronchioles, accompanied by destruction of their walls and without obvious fibrosis. Only about 10% of patients with COPD have pure emphysema, and there are several other structural changes in patients with COPD. In patients with COPD, cough and sputum may precede airflow limitations and some patients may exhibit significant airflow limitation without having chronic cough and sputum production. Patients may have a predominance of one of these conditions, but in reality it is often difficult to determine because the conditions usually coexist. COPD is discussed in this section as one disease state from the standpoint of pathophysiology and management.[2]

Patients with COPD may have asthma, and some patients with asthma may go on to develop fixed or irreversible airflow obstruction. It may be nearly impossible to differentiate asthma from COPD, especially if the individual has a history of cigarette smoking.[13]

An estimated 12.1 million adults in the United States over age 18 have COPD. Persons with COPD are greatly underestimated because the disease is usually not diagnosed until it is moderately advanced. The number of women with COPD is on the rise, probably because of the large number of women smoking cigarettes and also having exposure to environmental pollutants. COPD is the fourth leading cause of death in the United States. More women die from COPD than men. More white Americans are likely to have and die from COPD compared with any other ethnic group. Death rates related to COPD for Hispanics are significantly lower than for other ethnic groups.[14] Worldwide it is predicted that COPD will become the third leading cause of death by 2020 because of the increase in cigarette smoking and increased life spans.

Etiology

The many factors involved in the etiology of COPD are discussed in this section.

Cigarette Smoking. The major risk factor for developing COPD is cigarette smoking. COPD is more than four times as prevalent among smokers as nonsmokers. It affects about 15% of smokers and just 3% of nonsmokers. An intriguing question is why more smokers do not get COPD.[2,15]

Cigarette smoke has several direct effects on the respiratory tract (Table 29-17). The irritating effect of the smoke causes hyperplasia of cells, including goblet cells, which subsequently results in increased production of mucus. Hyperplasia reduces airway diameter and increases the difficulty in clearing secretions. Smoking reduces the ciliary activity and may cause actual loss of cilia. Smoking also produces abnormal dilation of the distal air space with destruction of alveolar walls. Many cells develop large, atypical nuclei, which are considered a precancerous condition.

After a short time of smoking, changes in small airway function can develop. In the early stages these changes are mostly inflammatory with mucosal edema and an influx of inflammatory cells. In later stages, however, thickening of the airway wall occurs by a remodeling process related to tissue repair and the inability of cilia to clear mucus, thus resulting in accumulation of inflammatory exudates in the airway lumen. Quitting smoking can prevent or delay the development of airflow limitation

ETHICAL DILEMMAS

Durable Power of Attorney for Health Care

Situation

A 50-year-old woman is being treated for complications of COPD. She is currently on a ventilator and not coherent because of the drugs she is receiving. Her life partner has been with her throughout this hospitalization. The patient had executed a valid durable power of attorney for health care decisions before this admission and named her partner as her primary agent. However, the patient's parents and siblings have arrived and demand to be in charge of her treatment decisions. They do not accept the partner or the patient's appointment of this person to make decisions for her.

Important Points for Consideration

- Durable power of attorney for health care is one type of advance directive in which the person, when she or he is competent, identifies someone else to make decisions for the person, should the person lose her or his decision-making ability in the future.
- The surrogate decision maker, who is named as durable power of attorney for health care, is often selected because the person believes that personal values, beliefs, and wishes will be respected when making treatment decisions for her or him.
- According to the Patient Bill of Rights, patients, family, and significant others have a right to be treated with discretion and sensitivity regarding their values and beliefs.
- Advance directives are legal documents. However, it is often difficult for health care providers when family members or designated surrogates do not agree.
- State laws may differ regarding surrogate decision makers, so it is imperative to be familiar with the statutes in the state in which one practices.

Clinical Decision-Making Questions

1. How would you handle a situation in which the family and surrogate decision maker disagree?
2. How can you assess the patient and family's understanding of durable power of attorney and assist them in understanding their role in decision making?

GENDER DIFFERENCES

Chronic Obstructive Pulmonary Disease (COPD)

MEN	WOMEN
• COPD is more common in men than women, but trend for men is not increasing. • Fewer men are dying from COPD than women.	• Number of women with disease is increasing. • Increase is probably due to increased number of women smoking cigarettes and increased susceptibility (e.g., smaller lungs and airways, lower elastic recoil). • Women with disease have lower quality of life, more exacerbations, increased dyspnea, and better response to O_2 therapy.

TABLE 29-17 EFFECTS OF TOBACCO SMOKE ON THE RESPIRATORY SYSTEM

AREA OF DEFECT	ACUTE EFFECTS	LONG-TERM EFFECTS
Respiratory mucosa		
• Nasopharyngeal	↓ Sense of smell	Cancer
• Tongue	↓ Sense of taste	Cancer
• Vocal cords	Hoarseness	Chronic cough, cancer
• Bronchus and bronchioles	Bronchospasm, cough	Chronic bronchitis, asthma, cancer
Cilia	Paralysis, sputum accumulation, cough	Chronic bronchitis, cancer
Mucous glands	↑ Secretions, ↑ cough	Hyperplasia and hypertrophy of glands, chronic bronchitis
Alveolar macrophages	↓ Function	↑ Incidence of infection
Elastin and collagen fibers	↑ Destruction by proteases ↓ Function of antiproteases (α_1-antitrypsin) ↓ Synthesis and repair of elastin	Emphysema

or reduce its progression.[2] (See Chapter 12 for more detail about cigarette smoking.)

Passive smoking is the exposure of nonsmokers to cigarette smoke, also known as *environmental tobacco smoke* (ETS) or secondhand smoke. In adults, involuntary smoke exposure is associated with decreased pulmonary function, increased respiratory symptoms, and severe lower respiratory tract infections such as pneumonia. ETS is also associated with increased risk for lung cancer and nasal sinus cancer.

Occupational Chemicals and Dusts. If a person has intense or prolonged exposure to various dusts, vapors, irritants, or fumes in the workplace, COPD can develop independently of cigarette smoking. If the person smokes, the risk of COPD increases.[2]

Air Pollution. High levels of urban air pollution are harmful to persons with existing lung disease. However, the effect of outdoor air pollution as a risk factor for COPD appears to be small compared with the effect of cigarette smoking. Another risk factor for COPD development is fossil fuels that are used for indoor heating and cooking. Many women, particularly worldwide, who have never smoked are developing COPD because of cooking with these fuels in poorly ventilated areas.[2]

Infection. Infections are a risk factor for developing COPD. Severe recurring respiratory tract infections in childhood have been associated with reduced lung function and increased respiratory symptoms in adulthood. After initiating factors, such as childhood respiratory infections or smoking, the normal lung defense mechanisms are impaired and the microbes colonize, setting up a cycle of chronic inflammation and infection. The same microbes are the cause of acute exacerbations of COPD that further intensifies the destruction of lung tissue and the progression of COPD. The most common microbes associated with exacerbations are *Haemophilus influenzae, Streptococcus pneumoniae, Moraxella catarrhalis,* and rhinovirus.[2,16]

Genetics. The fact that a relatively small percentage of smokers get COPD strongly suggests that there are genetic factors that influence which smokers get the disease. Because of the genetic-environment interaction, two people may have the same smoking history, but only one develops COPD. Another possible explanation is that because of genetic predisposition, certain people live longer and that influences which smoker or person develops COPD.[2,15]

To date, one genetic factor has been identified (see following discussion on α_1-antitrypsin [AAT] deficiency). Research is ongoing to identify genes that predispose a person to developing COPD.[2,15]

α_1-Antitrypsin (AAT) Deficiency. **α_1-Antitrypsin (AAT) deficiency** is a genetic risk factor that leads to COPD (see the Genetics in Clinical Practice box). AAT deficiency is an autosomal recessive disorder that may affect the lungs or liver. Approximately 3% of all people diagnosed with COPD may have undetected AAT deficiency.[17] Also known as α_1-protease inhibitor, AAT is a serum protein produced by the liver and normally found in the lungs. Severe AAT deficiency leads to premature bullous emphysema in the lungs found via x-ray. Normally AAT inhibits the breakdown of lung tissues by proteolytic enzymes from neutrophils and macrophages. Emphysema occurs because of the AAT deficiency. Lower levels of AAT result in insufficient inactivation and subsequent destruction of lung tissue. Smoking greatly exacerbates the disease process in these patients.

The most common abnormal genes are the S and Z alleles, and normal genes are labeled M. The most common genotype associated with AAT disease is ZZ. There are many different variations in the AAT gene alleles, but there are five combinations that seem to be the most relevant clinically. Individuals with ZZ mutations will have only 10% to 15% of the normal level of α_1-antitrypsin, but they have severe COPD and usually have liver involvement. At the other end of the spectrum, the MS mutation has 80% of the normal level of α_1-antitrypsin and affected individuals have no detectable disease as they are carriers.[15]

Clues to AAT deficiencies are the onset of symptoms often occurring by age 40, minimal to no tobacco use, and family

GENETICS IN CLINICAL PRACTICE

α_1-Antitrypsin (AAT) Deficiency

Genetic Basis
- Autosomal recessive disorder
- Mutations in *SERPINA1* gene (located on chromosome 14) cause AAT deficiency
- Gene provides instructions for making the protein AAT

Incidence
- 1 in 1700 to 3500 live births in the United States
- Persons of northern European descent most affected

Genetic Testing
- DNA testing is available.
- Screening of siblings is useful.
- Serum assay is available to measure the amount of α_1-antitrypsin.

Clinical Implications
- α_1-Antitrypsin is produced mainly in liver; deficiency can cause lung and liver disease.
- Onset of disease appears between ages 20 and 40 years.
- Treatment includes α_1-antitrypsin replacement (Prolastin).
- Disease predisposes to early-onset emphysema.

history of emphysema. Chronic liver disease as an infant or adult with increased liver enzyme tests may also be seen. Individuals with this type of emphysema are primarily of northern European origin. A simple blood test can determine low levels of AAT. Those with borderline or low levels can then be genetically tested.

IV-administered AAT (e.g., Prolastin) augmentation therapy is used for persons with AAT deficiency. The infusions are administered weekly. Its effectiveness in slowing the progression of the disease continues to be evaluated.

Aging. Some degree of emphysema is common in the lungs of the older person, even a nonsmoker. Aging results in changes in the lung structure, the thoracic cage, and the respiratory muscles. As people age there is gradual loss of the elastic recoil of the lung. The lungs become more rounded and smaller. The number of functional alveoli decreases as peripheral airways lose supporting tissues. These changes are similar to those seen in the patient with emphysema. Clinically significant emphysema, however, is usually not caused by aging alone.

Thoracic cage changes result from osteoporosis and calcification of the costal cartilages. The thoracic cage becomes stiff and rigid, and the ribs are less mobile. The shape of the rib cage gradually changes because of the increased residual volume (RV), causing it to expand and become rounded. Decreased chest compliance and elastic recoil of the lungs caused by aging affects the mechanical aspects of ventilation and increases the work of breathing. Changes in the elasticity of the lungs reduce the ventilatory reserve, and ability to clear secretions decreases with age.[18]

Pathophysiology

COPD is characterized by chronic inflammation found in the airways, lung parenchyma (respiratory bronchioles and alveoli), and pulmonary blood vessels (Fig. 29-9). The pathogenesis of COPD is complex and involves many mechanisms. The defining features of COPD are irreversible airflow limitation during forced exhalation caused by loss of elastic recoil and airflow obstruction caused by mucus hypersecretion, mucosal edema, and bronchospasm.

In COPD various disease processes occur such as airflow limitation, air trapping, gas exchange abnormalities, mucus hypersecretion, and in severe disease pulmonary hypertension and systemic features (see Fig. 29-9). COPD results in an uneven distribution of pathologic changes leaving severely destroyed lung areas existing with areas of relatively normal lung.[19]

The inflammatory process starts with inhalation of noxious particles and gases (e.g., cigarette smoke) but is magnified in the person with COPD. The abnormal inflammatory process causes tissue destruction and disrupts the normal defense mechanisms and repair process of the lung.

The predominant inflammatory cells in COPD are neutrophils, macrophages, and lymphocytes. This pattern of inflammatory cells is different from asthma. These inflammatory cells attract other inflammatory mediators (e.g., leukotrienes, interleukins). This cascading inflammatory process results in proinflammatory cytokines such as tumor necrosis factor being activated. In addition, growth factors are recruited into the area and activated resulting in structural changes in the lungs.

The inflammatory process may also be magnified by oxidative stress. Oxidants are produced by cigarette smoke and other inhaled particles and released from the inflammatory cells, such as macrophages and neutrophils, during inflammation. The oxidative stress adversely affects the lungs as it inactivates antiproteases (which prevent the natural destruction of

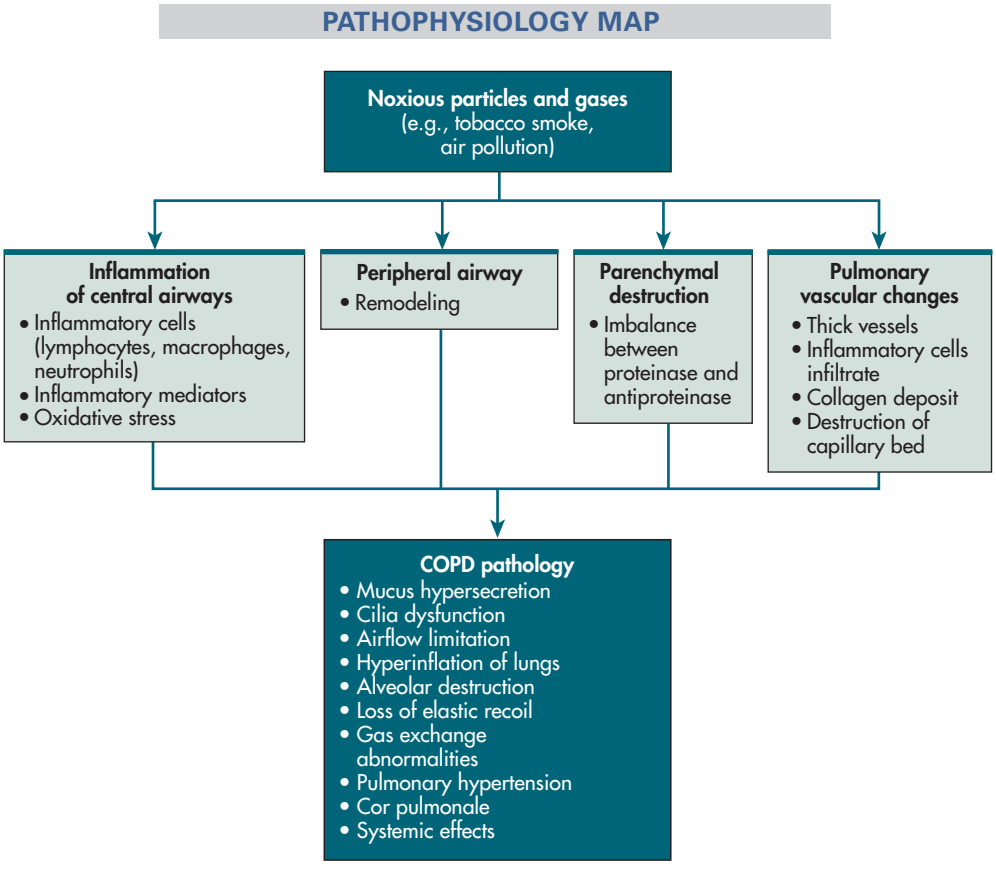


FIG. 29-9 Pathophysiology of COPD.

the lungs), stimulates mucus secretion, and increases fluid in the lungs.[2]

After the inhalation of oxidants in tobacco or air pollution, the activity of proteases (which break down the connective tissue of the lungs) increases and the antiproteases (which protect against the breakdown) are inhibited. Therefore the natural balance of protease/antiprotease is tipped in favor of destruction of the alveoli and loss of the elastic recoil of the lung.[2,8]

Inability to expire air is a main characteristic of COPD. The primary site of the airflow limitation is in the smaller airways and is due to remodeling. As the peripheral airways become obstructed, air is progressively trapped during expiration. The residual air becomes significant in severe disease as alveolar attachments to small airways (similar to rubber bands) are destroyed. The residual air, combined with the loss of elastic recoil, makes passive expiration of air difficult and air is trapped in the lungs. The chest hyperexpands and becomes barrel shaped as the respiratory muscles are not able to function effectively. The functional residual capacity is increased and the patient is now trying to breathe in when the lungs are in an "overinflated" state, thus the patient appears dyspneic and exercise capacity is limited.[2]

Gas exchange abnormalities result in hypoxemia and hypercarbia (increased CO_2) as the disease worsens. As the air trapping worsens and alveoli are destroyed, bullae (large air spaces in the parenchyma) and blebs (air spaces adjacent to pleurae) can form (Fig. 29-10). Bullae and blebs are not effective in gas exchange as the capillary bed that normally surrounds each alveolus does not exist in the bullae or bleb. Therefore there is a significant ventilation-perfusion (V/Q) mismatch and hypoxemia results. Peripheral airway obstruction also results in V/Q imbalance and, combined with the respiratory muscle impairment, can result in CO_2 retention, particularly in severe disease.[2,8]

Excess mucus production, resulting in a chronic productive cough, is a feature of individuals with predominant chronic bronchitis and is not necessarily associated with limitation in airflow. However, not all COPD patients have sputum production. Excess mucus production is a result of an increased number of mucus-secreting goblet cells and enlarged submucosal glands, which respond to the chronic irritation of smoke or other inhalants. In addition, dysfunction of cilia leads to chronic cough and sputum production. Some of the inflammatory mediators also stimulate mucus production.

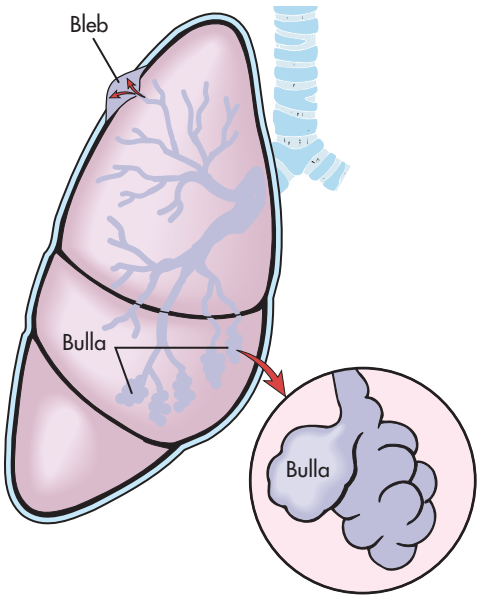


FIG. 29-10 Pulmonary blebs and bullae.

Pulmonary vascular changes resulting in mild to moderate pulmonary hypertension may occur late in the course of COPD. The small pulmonary arteries vasoconstrict due to hypoxia and their structure changes, resulting in thickening of the vascular smooth muscle as the disease advances. Because of the loss of alveolar walls and the capillaries surrounding them, there is increased pressure in the pulmonary circulation. Typically, the patient does not have difficulty with hypoxemia at rest until late in the disease. However, hypoxemia may develop during exercise, and the patient may benefit from supplemental O_2.

Pulmonary hypertension may progress and lead to hypertrophy of the right ventricle of the heart or **cor pulmonale,** with or without right-sided heart failure. COPD has been shown to have systemic effects, especially in severe disease. These extrapulmonary changes contribute greatly to the clinical findings of the patient and affect their survival and management. The mechanisms that cause the changes are unclear and are likely multifaceted, but systemic inflammation and inactivity of the patients are likely key factors.[20] Cachexia is common with a loss of skeletal muscle mass, and weakness is likely due to increased apoptosis (programmed cell death) and/or muscle disuse.[2] Patients may have weakness in all muscles in the upper and lower extremites.[21] They also have exercise intolerance, deconditioning, and osteoporosis. Patients with severe COPD also may develop chronic anemia, anxiety, depression, and an increased incidence of cardiovascular (CV) disease. The latter is likely due to an increase in C-reactive protein (another inflammatory marker linked with CV disease).[2,20]

Clinically it is common to find a combination of emphysema and chronic bronchitis in the same person, often with one condition predominating. Patients with COPD may also have asthma, and if they experience poorly reversible airflow limitation, the symptoms may be indistinguishable from COPD, but clinically are treated as asthma.

Clinical Manifestations

Clinical manifestations of COPD typically develop slowly around 50 years of age after 20 pack-years of cigarette smoking.[22] A diagnosis of COPD should be considered in any patient who has symptoms of cough, sputum production, or dyspnea, and/or a history of exposure to risk factors for the disease. A chronic intermittent cough usually occurs in the morning and may or may not be productive of small amounts of sticky mucus. These symptoms can occur many years before actual airflow limitation.[2]

Typically dyspnea is progressive, usually occurs with exertion, and is present every day. Patients usually ignore the symptoms and rationalize that "I'm getting older" and "I'm out of shape." They change behaviors to avoid dyspnea, such as taking the elevator. Gradually the dyspnea interferes with daily activities, such as carrying grocery bags, and they cannot walk as fast as their spouse or peers. However, as dyspnea worsens the patient usually seeks medical help and may be diagnosed with COPD. Other individuals do not experience the usual dyspnea, cough, or sputum production and may not seek help until they have a respiratory infection.

In late stages of COPD, dyspnea may be present at rest. As more alveoli become overdistended, increasing amounts of air are trapped. This causes a flattened diaphragm and the patient

must breathe from partially inflated lungs. Effective abdominal breathing is decreased because of the flattened diaphragm from the overdistended lungs. The person becomes more of a chest breather, relying on the intercostal and accessory muscles. However, chest breathing is not efficient breathing.

Wheezing and chest tightness may be present, but may vary by time of the day or from day to day, especially in patients with more severe disease. The wheeze may arise from the laryngeal area, or wheezes may not be present on auscultation. Chest tightness, which often follows activity, may feel similar to muscular contraction.

The person with advanced COPD frequently experiences weight loss and anorexia. Even when the patient has adequate caloric intake, weight loss is still experienced. Fatigue is a highly prevalent symptom that affects the patient's activities of daily living. Hemoptysis can occur during respiratory tract infections.

During physical examination a prolonged expiratory phase of respiration, wheezes, or decreased breath sounds are noted in all lung fields. The patient may need to breathe louder than normal for auscultated breath sounds to be heard. The anterior-posterior diameter of the chest is increased ("barrel chest") from the chronic air trapping. The patient may sit upright with arms supported on a fixed surface such as an overbed table *(tripod position)*. The patient may naturally purse lips on expiration (pursed-lip breathing) and use accessory muscles, such as those in the neck, to aid with inspiration. Edema in the ankles may be the only clue to right-sided heart involvement.

Over time, hypoxemia (PaO_2 <60 mm Hg or O_2 saturation <88%) may develop with hypercapnia ($PaCO_2$ >45 mm Hg) later in the disease. The bluish-red color of the skin results from polycythemia and cyanosis. Polycythemia develops as a result of increased production of red blood cells as the body attempts to compensate for chronic hypoxemia. Hemoglobin concentrations may reach 20 g/dL (200 g/L) or more. However, the person may also have lowered hemoglobin and hematocrit due to a chronic anemia.

As noted previously, it is sometimes difficult for the health care provider to distinguish COPD from asthma. However, there are some clinical features that are different (see Table 29-3).

Classification of COPD. COPD should be considered in any person with an exposure to risk factors such as cigarettes, environmental or occupational pollutants, and/or chronic cough and dyspnea. The diagnosis is confirmed by spirometry whether or not the patient has chronic symptoms. COPD can be classified as mild, moderate, severe, and very severe (Fig. 29-11). The FEV_1/FEV less than 70% establishes the diagnosis of COPD, and the severity of obstruction (as indicated by FEV_1) determines the stage of COPD. The management of COPD is primarily based on the patient's symptoms, but the staging provides a general guideline for the type of interventions.

Complications

Cor Pulmonale. Cor pulmonale results from pulmonary hypertension, which is caused by diseases affecting the lungs or pulmonary blood vessels (Fig. 29-12). In North America 50% of the cases of cor pulmonale are due to COPD.[23] Cor pulmonale is a late manifestation of COPD. Once the patient develops cor pulmonale the prognosis worsens. In COPD, pulmonary hypertension is caused primarily by constriction of the pulmonary vessels in response to alveolar hypoxia, with acidosis further potentiating the vasoconstriction. Chronic alveolar hypoxia causes vascular remodeling. Chronic hypoxia also stimulates erythropoiesis, which causes polycythemia. This results in increased viscosity of the blood. In COPD there may be an anatomic reduction of the pulmonary vascular bed as seen in emphysema with bullae. These patients have increased pulmonary vascular resistance and thus develop pulmonary hypertension.

Normally the right ventricle and pulmonary circulatory system are low-pressure systems compared with the left ventricle and systemic circulation. When pulmonary hypertension develops, the pressures on the right side of the heart must increase to push blood into the lungs. Eventually, right-sided heart failure develops.

Dyspnea is the most common symptom of chronic cor pulmonale due to the hyperinflation of the lungs in COPD. Lung sounds are normal or crackles may be heard in the bases of the lungs bilaterally. Heart sound changes occur but are usually masked by the underlying lung disease. Other manifestations of right-sided heart failure may develop, including distended

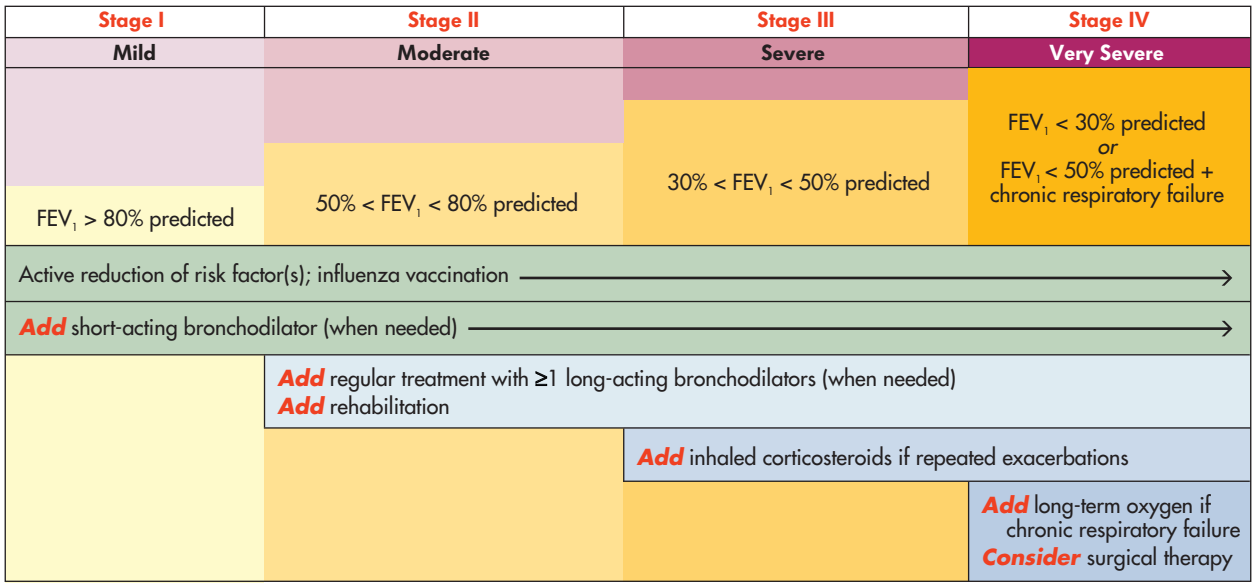


FIG. 29-11 Stages of COPD and therapy by stages.

neck veins (jugular venous distention), hepatomegaly with right upper quadrant tenderness, peripheral edema, and weight gain. ECG changes such as a tendency for right axis deviation may be seen. Typically the patient will have large pulmonary vessels on chest x-ray and increased pressure on a right-sided heart cardiac catheterization. Echocardiogram may reflect right-sided heart enlargement. B-type natriuretic peptide (BNP) levels (which are used to determine cardiac causes of heart failure) will be falsely elevated as the cause of the heart failure is the lung disease, not the heart, unless the left side of the heart is also failing.

Management of cor pulmonale includes continuous low-flow O_2. Long-term O_2 therapy improves survival of hypoxemic patients, especially when used more than 15 hours per day.[24] Digoxin has uncertain benefit in cor pulmonale treatment and may lead to cardiac dysrhythmias as the patient has tissue hypoxia and acidosis. Diuretics are generally used similarly to their use with chronic heart failure.[23] (Cor pulmonale is discussed further in Chapter 28.)

COPD Exacerbations. According to the Global Initiative for Chronic Obstructive Lung Disease (GOLD) guidelines, a *COPD exacerbation* is an event in the natural course of the disease characterized by a change in the patient's baseline dyspnea, cough, and/or sputum that is beyond the normal day-to-day variations, is acute in onset, and may warrant a change in regular medication in a patient with underlying COPD.

Exacerbations of COPD are typical in the course of the disease with increasing frequency (average one to two a year) as the disease progresses. The primary causes of exacerbations are bacterial (50%) or viral infections and air pollution/other environmental sources (15% or 20%).[16] Exacerbations are signaled by an acute change in the patient's usual dyspnea, cough, and/or sputum (i.e., something different from the usual daily patterns). Assess patients for the classic signs of exacerbation such as an increase in dyspnea, sputum volume, and/or sputum purulence. They may also have nonspecific complaints of malaise, insomnia, fatigue, depression, confusion, decreased exercise tolerance, increased wheezing, or fever without other causes.[25]

As the severity of COPD increases, exacerbations of COPD are associated with poorer outcomes. Exacerbations may be treated at home or in the hospital, depending on the severity. The severity is determined by the patient's medical history before the exacerbation, the presence of other diseases, symptoms, ABGs, and other laboratory tests. Typically in the later stages of COPD the patient has a low-normal pH, high-normal or above-normal $PaCO_2$, and high-normal HCO_3^-. This indicates compensated respiratory acidosis as the patient has chronically retained CO_2 and the kidneys have conserved HCO_3^- to increase the pH to within the normal range (see Table 29-3).

Carefully assess the patient's ABGs for any movement toward respiratory acidosis and further hypoxemia indicating respiratory failure. Also assess the patient's medical history for the stage of COPD as noted by the level of FEV_1. Assess for new symptoms or worsening of usual symptoms. Determine the number of previous exacerbations per year and if treatment occurred in the home or hospital. The presence of other co-morbid conditions will complicate the exacerbation. In addition, the current type of treatment

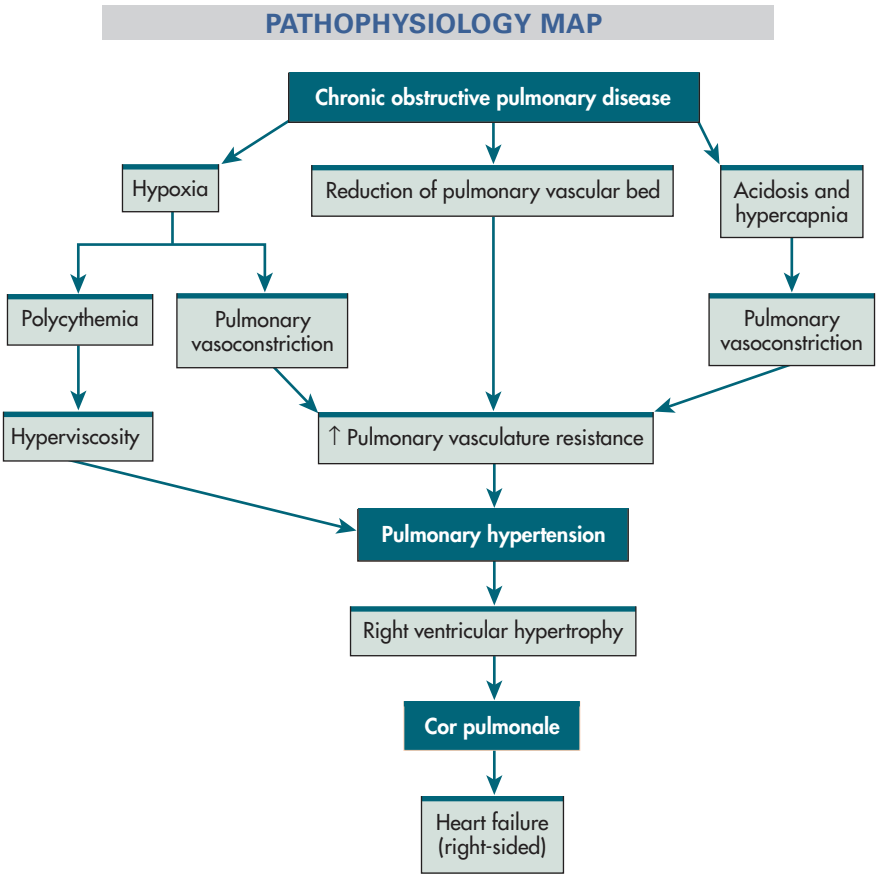


FIG. 29-12 Mechanisms involved in the pathophysiology of cor pulmonale secondary to chronic obstructive pulmonary disease.

will affect the exacerbation management. Be alert for signs of severity such as use of accessory muscles, central cyanosis, development of edema in the lower extremities, unstable blood pressure, signs of right-sided heart failure, and altered alertness.[2] All of these signs affect the management decisions (i.e., to treat as an inpatient or outpatient).

Bronchodilators and oral systemic corticosteroids decrease airway resistance during exacerbations of COPD. If a patient has clinical signs of airway infection (e.g., increased volume and change in color of sputum and/or fever, especially in the severe stages of COPD with more than three to four exacerbations per year), antibiotic treatment is usually used. Therapies used to treat exacerbations of COPD in the hospital are similar to home management, except supplemental oxygen therapy titrated by ABG measurement may be used.[2] Attempts are made to use noninvasive mechanical methods (e.g., continuous positive airway pressure [CPAP] to support ventilation rather than invasive ventilatory support [e.g., intubation]). Teach the patient and caregiver early recognition of signs and symptoms of exacerbations to promote early treatment and thus prevent hospitalization and possible respiratory failure.

Acute Respiratory Failure. Patients with severe COPD who have exacerbations are at risk for the development of respiratory failure. Frequently, COPD patients wait too long to contact their health care provider when they develop fever, increased cough and dyspnea, or other symptoms suggestive of exacerbations of COPD. An exacerbation of cor pulmonale may also lead to acute respiratory failure. Discontinuing bronchodilator or corticosteroid medication may also precipitate respiratory failure. The use of β-adrenergic blockers (e.g., propranolol [Inderal]) may also exacerbate acute respiratory failure in the patient with a reversible component to the COPD. However, cardioselective β-adrenergic blockers (e.g., atenolol, metoprolol) are used with mild to moderate disease as patients are closely monitored. Typically, cardioselective β-adrenergic blockers do not produce clinically significant problems with respiration.

Indiscriminate use of sedatives, benzodiazepines, and opioids, especially in the preoperative or postoperative patient who retains CO_2, may suppress the ventilatory drive and lead to respiratory failure. The person with COPD who retains CO_2 should be treated with low flow rates of O_2 with careful monitoring of ABGs to avoid hypercarbia. It is thought that high flow rates of O_2 depress the respiratory center and that the patient's respirations will diminish or cease. However, it is vital to provide adequate oxygen while assessing the ABGs, rather than not providing O_2 because of the fear of CO_2 narcosis (discussed on p. 620).

Surgery or severe, painful illness involving the chest or abdominal organs may lead to splinting and ineffective ventilation and respiratory failure. To prevent postoperative pulmonary complications, careful preoperative screening, which includes pulmonary function tests and ABG assessment, is important in the patient with a heavy smoking history and/or COPD. (Respiratory failure is discussed in Chapter 68.)

Depression and Anxiety. Patients with COPD experience many losses as the disease progresses over time. Approximately 50% of all patients with COPD experience depression.[26] The patient may also experience anxiety. You should assess for both. You should ask patients if they "feel down or blue" most of the time. Do they appear anxious about being able to control their symptoms of breathlessness or know what to do if they have an exacerbation? Are they exhibiting concern over more difficulty in self-care activities such as bathing? How is the family coping with the patient as emotions can run high in both? You can help the patient with progressive muscle relaxation exercises that can reduce anxiety. In addition, you can provide teaching about the treatment and disease. This can give patients a sense of control that they can manage daily activities along with the often complex medication regimens. It is important to include the patient's caregiver in the teaching so he or she can help the patient cope physically and emotionally.

A consultation with a mental health specialist may be needed for proper screening and diagnosis of depression or other mental health problems. Cognitive and behavioral therapy along with COPD education has been found to improve the quality of life.[27] Medications may be used to treat both the depression and anxiety. Buspirone (BuSpar), which is used to treat anxiety, has few if any respiratory depression effects. Benzodiazepines should be avoided because they may depress the respiratory drive and may be habit forming. When the patient becomes anxious because of dyspnea, the use of pursed-lip breathing (see Table 29-14) and short-acting bronchodilators may be appropriate.

Diagnostic Studies

The diagnosis of COPD is confirmed by pulmonary function tests. Goals of the diagnostic workup are to confirm the diagnosis of COPD via spirometry, evaluate the severity of the disease, and determine the impact of the disease on the patient's quality of life. These factors enable the health care provider to design an individualized treatment plan. In addition to pulmonary function tests, other diagnostic studies are performed (Table 29-18). Chest x-rays taken early in the disease are seldom diagnostic unless bullous disease is present. Patients may

TABLE 29-18 COLLABORATIVE CARE

Chronic Obstructive Pulmonary Disease

Diagnostic

History and physical examination
Pulmonary function tests
Chest x-ray
Serum α_1-antitrypsin levels
ABGs
6-minute walk test

Collaborative Therapy

Cessation of cigarette smoking
Treatment of exacerbations
Bronchodilator therapy (see Table 29-7)
- β_2-adrenergic agonists
- Anticholinergic agents
- Long-acting theophylline preparations

Corticosteroids
- Oral for exacerbations
- Inhaled for maintenance

Airway clearance techniques
Breathing exercises and retraining
Hydration of 3 L/day (if not contraindicated)
Patient and caregiver teaching
Influenza immunization yearly
Pneumovax immunization
Long-term O_2 (if indicated)
Progressive plan of exercise, especially walking and upper body strengthening
Pulmonary rehabilitation program
Nutritional supplementation if low BMI
Surgery
- Lung volume reduction
- Lung transplantation

ABGs, Arterial blood gases; *BMI,* body mass index.

TABLE 29-19 CORRELATION OF FEV_1 WITH CLINICAL MANIFESTATIONS

APPROXIMATE FEV_1	PROBABLE CLINICAL MANIFESTATION
1500 mL	Shortness of breath just beginning to be noticed
1000 mL	Shortness of breath with activity
500 mL	Shortness of breath at rest

have significant airflow limitation as demonstrated by spirometry but not chronic cough. Most patients seek medical help because of dyspnea that starts affecting their daily activities. Later in the disease the findings presented in Table 29-3 may be present.

A history and physical examination are extremely important in a diagnostic workup. Pulmonary function studies are useful in diagnosing and assessing the severity of COPD. Usually spirometry is ordered before and after bronchodilation. The most significant findings are related to increased resistance to expiratory airflow. Typical findings include the following:

- Reduced FEV_1, FEV_1/FVC ratio, diffusing capacity for carbon monoxide
- Increased residual volume, functional residual capacity (FRC)

When the FEV_1/FVC ratio is less than 70% along with the appropriate symptoms, a diagnosis of COPD is made. The value of FEV_1 as a percentage compared with normal provides a guideline for the degree of severity of the patient's lung disease and the stage of the disease (Table 29-19 and Fig. 29-11).

The body mass index (BMI) and degree of dyspnea are useful in predicting outcomes, such as survival. Current practice guidelines recommend that the BMI and dyspnea be evaluated in all patients. BMI is obtained by dividing weight (in kilograms [kg]) by height (in square meters [m^2]). A BMI less than 21 kg/m^2 is associated with an increase in mortality rate. ABGs are usually assessed in the severe stages and monitored in patients hospitalized with acute exacerbations. In the later stages of COPD, typical findings are low PaO_2, elevated $PaCO_2$, decreased or low-normal pH, and increased bicarbonate (HCO_3^-) levels. In early stages there may be a normal or only slightly decreased PaO_2 and a normal $PaCO_2$. A 6-minute walk test to determine O_2 saturation using a pulse oximetry may be done to evaluate the degree of O_2 desaturation that occurs with exercise. An ECG may be normal or show signs indicative of right ventricular failure. An echocardiogram or gated pool nuclear blood study (see Table 32-6) can be used to evaluate right-sided ventricular and left ventricular function. Sputum for culture and sensitivity may be obtained if the patient is hospitalized for an acute exacerbation and has not responded to empiric therapy with antibiotics.

Collaborative Care

The Global Initiative for Chronic Obstructive Lung Disease (GOLD) report is the major evidence-based reference used in the United States for care of the patient with COPD. It lists a summary of recommended treatment by each stage (see Fig. 29-11). The primary goals of care for the COPD patient are to (1) prevent disease progression, (2) relieve symptoms and improve exercise tolerance, (3) prevent and treat complications, (4) promote patient participation in care, (5) prevent and treat exacerbations, and (6) improve quality of life and reduce mortality risk. Most patients are treated as outpatients. They are hospitalized for exacerbations and potential complications when respiratory failure, pneumonia, and cor pulmonale are present.

Evaluate the patient's exposure to environmental or occupational irritants, and determine ways to control or avoid them. For example, teach the patient to avoid aerosol hair sprays and smoke-filled rooms. The patient with COPD is extremely susceptible to pulmonary infections. The patient with COPD and/or smokers should have a vaccination with influenza virus vaccine yearly and with pneumococcal vaccine[28,29] (see Table 28-5).

Exacerbations of COPD should be treated as soon as possible, especially if the patient is in the severe stages of COPD. Some patients are given a prescription for antibiotics and are instructed to begin taking them at the first symptoms/signs of an exacerbation. The most common antibiotics given for outpatients are macrolides (e.g., azithromycin [Zithromax]), doxycycline, and cephalosporins (e.g., cefpodoxime [Vantin]). If the patient has failed prior antibiotic therapy or is hospitalized, common antibiotics are amoxicillin/clavulanate (Augmentin) or respiratory fluoroquinolones (e.g., levofloxacin [Levaquin]).[2,16]

Smoking Cessation. Cessation of cigarette smoking in all stages of COPD is the single most effective and cost-effective intervention to reduce the risk of developing COPD and stops the progression of the disease.[2] After discontinuing smoking, the accelerated decline in pulmonary function slows and pulmonary function usually improves. Normally individuals over 35 years old lose approximately 20 to 25 mL (as measured by FEV_1) of lung function per year as measured by spirometry. Persons with COPD who continue to smoke lose approximately 50 mL per year. With the cessation of smoking, the loss can fall to almost nonsmoking levels at 35 mL per year.[30] Thus the sooner the smoker stops, the less pulmonary function is lost and the sooner the symptoms decrease, especially cough and sputum production. One interesting stop-smoking technique to improve the likelihood of a person stopping smoking is to provide patients with their "lung age" (i.e., the age of the average healthy person who would perform similarly to them on spirometry). For example, if a 32-year-old person who smokes had a very low FEV_1, he or she would have the lungs of a 74-year-old.[31] (Smoking cessation techniques are discussed in Chapter 12 and in Tables 12-4 through 12-7.)

Drug Therapy. Medications for COPD can reduce or abolish symptoms, increase the capacity to exercise, improve overall health, and reduce the number and severity of exacerbations.[32] Bronchodilator drug therapy relaxes smooth muscles in the airway and improves the ventilation of the lungs, thus reducing the degree of breathlessness. Although patients with COPD do not respond as dramatically as those with asthma to bronchodilator therapy, a reduction in dyspnea and an increase in FEV_1 are usually achieved. The inhaled route of medication is preferred and given on a PRN or regular basis. Medications are given in a stepwise fashion, stepping up but usually not stepping down as in asthma, because in COPD there are likely continual symptoms (see Fig. 29-11).

Bronchodilator medications commonly used are β_2-adrenergic agonists, anticholinergic agents, and methylxanthines (see Table 29-7). The choice of bronchodilator depends on the availability and the patient's response. However, when the patient has mild COPD or intermittent symptoms, a short-acting bronchodilator is used as needed. Short-acting bronchodilators increase exercise tolerance. Albuterol or ipratropium (Atrovent) may be used as single agents, but combining bronchodilators improves their effect and decreases the risk of adverse effects, compared with the use of a single agent. As a single agent, ipratropium (Atrovent) is

superior to albuterol because the only side effect is usually dry mouth. These two agents can be nebulized together (DuoNeb) or delivered by one MDI (Combivent).

As symptoms persist or moderate stages of COPD develop, a long-acting bronchodilator is used in addition to a short-acting bronchodilator (see Table 29-7). Salmeterol (Serevent) and formoterol (Foradil) are widely used long-acting β_2-adrenergic agonists, and they can be used in COPD as monotherapy (unlike drug therapy for asthma).

Tiotropium (Spiriva), a long-acting anticholinergic, dosed once a day can be used for dyspnea management in COPD. Tiotropium also improves lung function, improves quality of life, and decreases the number of COPD exacerbations and hospitalizations.[33,34]

The use of long-acting theophylline in the treatment of COPD is controversial because it interacts with many drugs. Although it has some action as a mild bronchodilator in the patient with partial reversibility of airflow obstruction, its main value may be to improve contractility of the diaphragm and decrease diaphragmatic fatigue.

ICS therapy is beneficial for patients with stage 3 (severe) or stage 4 (very severe) COPD as it reduces the frequency of COPD exacerbations. ICSs combined with long-acting β_2-adrenergic agonists (e.g., fluticasone/salmeterol [Advair]) are more effective than the single-drug therapy in reducing exacerbations and improving lung function.[2] Some patients are on triple therapy with salmeterol/fluticasone (Advair) and tiotropium (Spiriva).[35] Oral corticosteroids should not be used for long-term therapy in COPD, but are effective in the short term for exacerbations.

O_2 Therapy. O_2 therapy is frequently used in the treatment of COPD and other problems associated with hypoxemia. Long-term O_2 therapy (LTOT) improves survival, exercise capacity, cognitive performance, and sleep in hypoxemic patients.[13] O_2 is a colorless, odorless, tasteless gas that constitutes 21% of the atmosphere. Administering supplemental O_2 increases the partial pressure of O_2 (PO_2) in inspired air. Used clinically it is considered a drug. For reimbursement purposes Medicare will pay for oxygen when certain clinical criteria are met (i.e., the patient's O_2 saturation needs to be ≤88%, PaO_2 ≤55 mm Hg).

Indications for Use. Goals for O_2 therapy are to reduce the work of breathing, maintain the PaO_2, and/or reduce the workload on the heart, keeping the SaO_2 greater than 90% during rest, sleep, and exertion, or PaO_2 greater than 60 mm Hg. O_2 is usually administered to treat hypoxemia caused by (1) respiratory disorders such as COPD, pulmonary hypertension, cor pulmonale, pneumonia, atelectasis, lung cancer, and pulmonary emboli; (2) cardiovascular disorders such as myocardial infarction, dysrhythmias, angina pectoris, and cardiogenic shock; and (3) central nervous system disorders such as overdose of opioids, head injury, and sleep disorders (sleep apnea).

Methods of Administration. The goal of O_2 administration is to supply the patient with adequate O_2 to maximize the O_2-carrying ability of the blood. There are various methods of O_2 administration (Table 29-20 and Fig. 29-13 The)method selected depends on factors such as the fraction of inspired O_2 (FIO_2)

TABLE 29-20 METHODS OF OXYGEN ADMINISTRATION

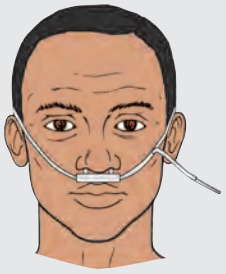
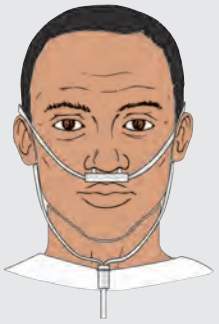
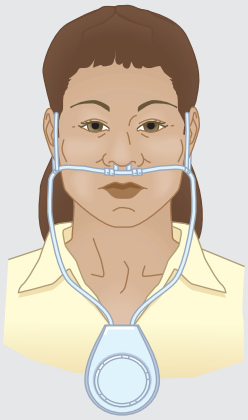
DESCRIPTION	NURSING INTERVENTIONS	
Low-Flow Delivery Devices		
Nasal Cannula		
• Most commonly used device. • Oxygen delivered via plastic nasal prongs. • Safe and simple method that allows some freedom of movement and patient can eat, talk, or cough while wearing device. • Useful for a patient requiring low O_2 concentrations. • O_2 concentrations of 24% (at 1 L/min) to 44% (at 6 L/min) can be obtained.	• Nasal cannula should be stabilized when caring for a restless patient. • Amount of O_2 inhaled depends on room air and patient's breathing pattern. • Most patients with COPD can tolerate 2 L/min via cannula. • Assess patient's nares and ears for skin breakdown and may need to pad cannula where it sits on ears. • If flow rates are >5 L/min, nasal membranes may dry and may cause pain in frontal sinuses.	 
Simple Face Mask		
• Covers patient's nose and mouth. • Used only for short periods, especially when transporting patients. • Longer use is typically not tolerated as it is uncomfortable with tight seal and heat generated around nose/mouth from face. • O_2 concentrations of 35%-50% can be achieved with flow rates of 6-12 L/min. • Mask provides adequate humidification of inspired air.	• Wash and dry under mask q2hr. • Mask must fit snugly. • Nasal cannula may be provided while patient is eating. • Watch for pressure necrosis at top of ears from elastic straps if patient wears for a longer period of time. (Gauze or other padding may be used to alleviate this problem.)	

TABLE 29-20 METHODS OF OXYGEN ADMINISTRATION—cont'd

DESCRIPTION	NURSING INTERVENTIONS
Low-Flow Delivery Devices—cont'd	
Partial and Non-Rebreathing Masks	
• Useful for short-term (24 hr) therapy for patients needing higher O_2 concentrations (60%-90% at 10-15 L/min). • O_2 flows into reservoir bag and mask during inhalation. • This bag allows patient to rebreathe about first third of exhaled air (rich in O_2) in conjunction with flowing O_2. • Vents remain open on partial mask only; some facilities prefer this over non-rebreather as a safety issue.	• Oxygen flow rate must be sufficient to keep bag from collapsing during inspiration to avoid CO_2 buildup. • If deflation occurs, liter flow needs to be increased to keep bag inflated. • Mask should fit snugly. • With non-rebreather masks, make sure valves are open during expiration and closed during inhalation to prevent drastic decrease in FIO_2. • Monitor patient closely because intubation may be next required intervention.
Oxygen-Conserving Cannula	
• Generally indicated for long-term O_2 therapy at home versus during hospitalization (e.g., pulmonary fibrosis, pulmonary hypertension). • May be "moustache" (Oxymizer) or "pendant" type. • Cannula has a built-in reservoir that ↑ O_2 concentration and allows patient to use lower flow, usually 30%-50%, which increases comfort, lowers cost, and can be increased with activities. • Can deliver up to 8 L/min O_2.	• May cause necrosis over tops of ears; can be padded. • Cannula cannot be cleaned; manufacturer recommends changing cannula every week. • It is more expensive than standard cannulas and requires evaluation with ABGs and oximetry to determine correct flow for patient. • Cannula is highly visible.
High-Flow Delivery Devices	
Tracheostomy Collar	
• Collar attaches to neck with elastic strap and can deliver high humidity and O_2 via tracheostomy. • O_2 concentration is lost into atmosphere because collar does not fit tightly. • Venturi device can be attached to flow meter and thus can deliver exact amounts of oxygen via collar.	• Secretions collect inside collar and around tracheostomy and collar should be removed and cleaned at least q4hr to prevent aspiration of fluid and infection. • Condensation occurs in tubing and needs to be periodically drained distally to tracheostomy.
Tracheostomy T Bar	
• Almost identical to tracheostomy collar, but it has a vent and a T connector that allow an inline catheter (e.g., Ballard catheter) to be connected for suctioning. • Tight fit allows better O_2 and humidity delivery than tracheostomy collar.	• It should be emptied as necessary. • See tracheostomy collar above.



Pendant-type oxygen-conserving cannula.

Continued

TABLE 29-20 METHODS OF OXYGEN ADMINISTRATION—cont'd

DESCRIPTION	NURSING INTERVENTIONS
High-Flow Delivery Devices—cont'd	
Venturi Mask	
• Mask can deliver precise, high flow rates of O_2. • Lightweight plastic, cone-shaped device is fitted to face. • Masks are available for delivery of 24%, 28%, 31%, 35%, 40%, and 50% O_2. • Method is especially helpful for administering low, constant O_2 concentrations to patients with COPD. • Adaptors can be applied to increase humidification.	• Entrainment device on mask must be changed to deliver higher concentrations of O_2. • Air entrainment ports must not be occluded. • Mask is uncomfortable and must be removed when patient eats. • Patient can talk but voice may be muffled. • See other applicable nursing interventions under simple face mask above.

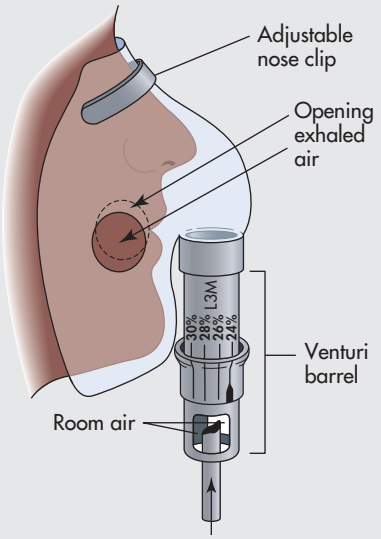


FIG. 29-13 Golfer uses Helios liquid portable oxygen system.

required by the patient and delivered by the device, the mobility of the patient, humidification required, patient cooperation, comfort, cost, and available financial resources.

O_2 delivery systems are classified as low- or high-flow systems. Most methods of O_2 administration are low-flow devices that deliver O_2 in concentrations that vary with the person's respiratory pattern. In contrast, the Venturi mask is a high-flow device that delivers fixed concentrations of O_2 (e.g., 24%, 28% independent of the patient's respiratory pattern). Mechanical ventilators are another example of a high-flow O_2 delivery system. Because room air is mixed with O_2, in low-flow systems, the percentage of O_2 delivered to the patient is not as precise as with high-flow systems.

Humidification and Nebulization. O_2 obtained from cylinders or wall systems is dry. Dry O_2 has an irritating effect on mucous membranes and dries secretions. Therefore it is important that a high liter flow of O_2 delivering more than 35% to 50% oxygen be humidified when administered, either by humidification or nebulization. A common device used for humidification when the patient has a cannula or a mask is a bubble-through humidifier. It is a small plastic jar filled with sterile distilled water that is attached to the O_2 source by means of a flow meter. O_2 passes into the jar, bubbles through the water, and then goes through tubing to the patient's cannula or mask. The purpose of the bubble-through humidifier is to restore the humidity conditions of room air. However, the need for bubble-through humidifiers at flow rates between 1 and 4 L per minute is based on the patient's comfort.

Another means of administering humidified O_2 is via a nebulizer. It delivers particulate water mist (aerosols) with nearly 100% humidity. The humidity can be increased by heating the water, which increases the ability of the gas to hold moisture. Humidified (100%) gas is required when the upper airway is bypassed in acute care. However, patients with established tracheostomies do not always require 100% humidity. When nebulizers are used, large-size tubing should be employed to connect the device to a face mask or T bar. If small-size tubing is used, condensation can occlude the flow of O_2. Vapotherm Precision Flow can deliver high flows (up to 40 L/min) of warm humidified gas (air/oxygen) with precise percentages to the patient through a nasal cannula and it is noninvasive.[36]

Complications

Combustion. O_2 supports combustion and increases the rate of burning, so it is important that smoking be prohibited in the area in which O_2 is being used. A "No Smoking" sign should be prominently displayed on the patient's door. Also caution the patient against smoking cigarettes with an O_2 cannula in place.

CO_2 Narcosis. The chemoreceptors in the respiratory center that control the drive to breathe respond to CO_2 and O_2. Normally, CO_2 accumulation is the major stimulant of the respiratory center. Over time some COPD patients develop a tolerance for high CO_2 levels (the respiratory center loses its sensitivity to the elevated CO_2 levels). Theoretically, for these individuals the "drive" to breathe is hypoxemia. Thus there has been concern regarding the dangers of administering O_2 to COPD patients and reducing their drive to breathe. This

DELEGATION DECISIONS

Oxygen Administration

All members of the health care team should be alert to possible problems with gas exchange in patients who are receiving oxygenation. Patients who are hypoxemic should be cared for by the registered nurse (RN) until they consistently have oxygen saturations ≥90%.

Role of Registered Nurse (RN)
- Assess need for adjustments in oxygen flow rate.
- Evaluate response to oxygen therapy.
- Monitor patient for signs of adverse effects of oxygen therapy.
- In many cases, the RN chooses the optimal oxygen delivery device (e.g., a nasal cannula or simple face mask).
- Educate patient and caregivers about home oxygen use.

Role of Licensed Practical/Vocational Nurse (LPN/LVN)
- For stable patients, adjust oxygen flow rate depending on desired oxygen saturation level.

Role of Nursing Assistive Personnel (NAP)
- Use pulse oximetry to obtain oxygen saturation.
- Report oxygen saturation level to RN.
- Assist patient with adjustment of oxygen delivery devices (e.g., nasal cannula, face mask).
- Report to RN any change in patient level of consciousness or complaints of shortness of breath.

has been a pervasive myth but is not a serious threat. In fact, not providing adequate O_2 to these patients is much more detrimental and it is much easier for the health care team to reverse high CO_2 than low O_2. Although O_2 administration should be titrated to the lowest effective dose, many patients who have end-stage COPD require high flow rates and higher concentrations for survival. They may, in fact, exhibit higher than normal levels of CO_2 in their blood, but this is of little concern. What is important is careful, ongoing assessment when providing O_2 to these patients, monitoring both the physical and cognitive effects of O_2.

It is critical to start O_2 at low flow rates until ABGs can be obtained. ABGs are used as a guide to determine what FIO_2 level is sufficient and can be tolerated. The patient's mental status and vital signs should be assessed before starting O_2 therapy and frequently thereafter.

O_2 Toxicity. Pulmonary **O_2 toxicity** may result from prolonged exposure to a high level of O_2 (PaO_2). The development of O_2 toxicity is relatively rare, but is determined by patient tolerance, exposure time, and effective dose. High concentrations of O_2 can result in a severe inflammatory response because of oxygen radicals and damage to alveolar-capillary membranes resulting in severe pulmonary edema, shunting of blood, and hypoxemia. These individuals develop acute respiratory distress syndrome (ARDS) (see Chapter 68). Prevention of O_2 toxicity is important for the patient who is receiving O_2. The amount of O_2 administered should be just enough to maintain the PaO_2 within a normal or acceptable range for the patient. ABGs should be monitored frequently to evaluate the effectiveness of therapy and to guide the tapering of supplemental O_2. A safe limit of O_2 concentrations has not yet been established. All levels above 50% and used for longer than 24 hours should be considered potentially toxic. Levels of 40% and below may be regarded as relatively nontoxic and may not result in development of significant O_2 toxicity if the exposure period is short.

Absorption Atelectasis. Normally, nitrogen (which constitutes 79% of the air that is breathed) is not absorbed into the bloodstream. This prevents alveolar collapse. When high concentrations of O_2 are given, nitrogen is washed out of the alveoli and replaced with O_2. If airway obstruction occurs, the O_2 is absorbed into the bloodstream and the alveoli collapse. This process is called **absorption atelectasis.**

Infection. Infection can be a major hazard of O_2 administration. Heated nebulizers present the highest risk. The constant use of humidity supports bacterial growth, with the most common infecting organism being *Pseudomonas aeruginosa.* Disposable equipment that operates as a closed system, such as the Ballard closed suctioning system, should be used. Each hospital has a policy stating the required frequency of equipment changes based on the type of equipment used at that particular institution.

Chronic O_2 Therapy at Home. Improved survival occurs in patients with COPD who receive long-term O_2 therapy (LTOT) (more than 15 hours/day) to treat hypoxemia.[24] The improved prognosis results from preventing progression of the disease and subsequent cor pulmonale. The benefits of LTOT include improved mental acuity, lung mechanics, sleep, and exercise tolerance; decreased hematocrit; and reduced pulmonary hypertension. Some patients believe they will become "addicted" to O_2 and are reluctant to use it. Tell them that it is not "addicting" and that it needs to be used because of the positive effects on the heart, lungs, and brain. Evaluate the need for LTOT when the patient's condition has stabilized. The goal of O_2 therapy is to maintain SaO_2 greater than 90% during rest, sleep, and exertion.

Short-term home O_2 therapy (1 to 30 days) may be indicated for the patient in whom hypoxemia persists after discharge from the hospital. For example, the patient with underlying COPD who develops a serious respiratory infection may continue to have clearing of the infection after completion of antibiotic therapy and discharge from the hospital. This patient may demonstrate continued hypoxemia for 4 to 6 weeks after discharge. It is important to measure the patient's oxygenation status by SaO_2 30 to 90 days after an acute episode to determine if the O_2 is still warranted.

Desaturation only during exercise or sleep suggests consideration of O_2 therapy specifically under those conditions. Patients may receive O_2 only during exercise and/or sleep. The need for O_2 during these periods should be evaluated with a 6-minute walk test or overnight oximetry. (Pulse oximetry is discussed in Chapter 26.)

Periodic reevaluations are necessary for the patient who is using chronic supplemental O_2. Generally the recommendation is that the patient should be reevaluated every 30 to 90 days during the first year of therapy and annually after that, as long as the patient remains stable.

Nasal cannulas, either regular or the O_2-conserving type (see Table 29-20), are usually used to deliver O_2 from a central source in the home. The source may be a liquid O_2 storage system, compressed O_2 in tanks, or an O_2 concentrator or extractor, depending on the patient's home environment, insurance coverage, activity level, and proximity to an O_2 supply company (Table 29-21). The patient can use extension tubing (up to 50 feet) without adversely affecting the O_2 flow delivery to increase mobility in the home. Small, portable systems, such as liquid O_2, may be provided for the patient who remains active outside the home (see Fig. 29-13).

Home O_2 systems are usually rented from a company that sends a respiratory therapist to the patient's home (Fig. 29-14). The therapist teaches the patient and caregiver how to use the O_2 system, how to care for it, and how to recognize when the

TABLE 29-21 HOME OXYGEN DELIVERY SYSTEMS

Liquid Oxygen

Portable* unit can be refilled by patient from reservoir (see Table 29-20). Portable unit holds 6-8 hr supply at 2 L/min; reservoir will last approximately 7-10 days at 2 L/min continuously. Patients do not use the reservoir continuously due to the high expense. Instead patients use the O_2 concentrator as the cost is much lower. Liquid system is strictly for portable and emergency use. Not available everywhere; generally limited to urban areas.

Compressed O_2 Cylinders

Cylinders or tanks of varying sizes, e.g., D, M, E, H, J duration varies with tank size and liter flow, e.g., J tank at 2 L/min flow lasts about 50 hr. Portability possible with cart and some of the smaller tanks may be refilled from large cylinders. Smaller tanks weigh about 10 lb and can be carried on shoulder strap, backpack, or fanny pack carried or placed on a portable cart.

Concentrator or Extractor

Because the O_2 supply is produced from room air, they never need to be "filled." On wheels, movable from room to room, but usually kept centrally located in the dwelling with extension tubing reaching to the furthest area. Patients need to be very cautious to prevent falling over the tubing. Compact, excellent system for rural or homebound patient. Convenient, safe, and reliable. Patient will need backup O_2 tank in case electricity fails. Concentrator noise may be bothersome and should be kept in a room other than bedroom.

Portable Oxygen Concentrator

These are lighter-weight devices (8.5-17 lb) that are portable via carts or shoulder straps that may provide pulsed or continuous O_2. 5-6 L/min flow depending on the particular device. Batteries provide up to 8 hr of operation with recharging in AC or DC (e.g., car) and are approved by many airlines. This combined with another stationary system in the patient's dwelling provides the patient with exceptional freedom and mobility as the system continuously provides a renewable source of O_2 outside the home. Systems are EverGo, Inogen One, and Eclipse Oxygen Generator.

O_2 Conserving or Pulsed Devices

Delivers a pulse of O_2 only during inhalation to conserve O_2. Allows patient increased mobility. Devices are relatively lightweight; varying from approximately 3-6 lb with a supply of oxygen up to 20 hr. System may clip on belt or be contained in a backpack or shoulder bag. Audible pulses may be annoying. Some devices may require batteries. Becomes less efficient at higher O_2 flow rates. Usually best for low activity levels. Monitor patient's O_2 saturation during rest and exercise to determine if oxygenation is acceptable.

*Portable usually refers to units weighing more than 10 lb (4.5 kg) and ambulatory units weigh less than 10 lb.

supply is running low and needs to be reordered. A patient and caregiver teaching guide for the use of O_2 at home is presented in Table 29-22 .

Encourage the patient who uses home O_2 to remain active and travel normally. If travel is by automobile, arrangements can be made for O_2 to be available at the destination point. O_2 supply companies can often assist in these arrangements. If a patient wishes to travel by bus, train, or airplane, the patient should inform the appropriate people when reservations are made that O_2 will be needed for travel. If there is a potential for the patient to become hypoxic, oxygen needs for flying can be determined via a hypoxia inhalation test or through a mathematical formula. Portable oxygen concentrators are a ready source of renewable O_2 and can be available by recharging at

FIG. 29-14 A portable liquid O_2 unit can be refilled from a liquid O_2 reservoir unit.

home or in a DC (e.g., auto) power supply. These systems are widely approved by airlines for in-flight use. The patient should contact the specific airline to determine the particular accommodations and policies for in-flight O_2.

Surgical Therapy for COPD. Three different surgical procedures have been used in severe COPD. One type of surgery is *lung volume reduction surgery* (LVRS). The goal of this therapy is to reduce the size of the lungs by removing the most diseased lung tissue so the remaining healthy lung tissue can perform better. The rationale for this type of surgery is that by reducing the size of the hyperinflated emphysematous lungs, there is decreased airway obstruction and increased room for the remaining normal alveoli to expand and function. The procedure reduces lung volume and improves lung and chest wall mechanics. There is some survival advantage for those having the LVRS compared with medical therapy and improvement in work capacity, quality of life, and reduced COPD exacerbations. However, the selection criteria for these patients are very strict and it is an expensive palliative procedure.

The second surgical procedure is *bullectomy.* This procedure is used for carefully selected patients with emphysematous COPD who have large bullae (larger than 1 cm). The bullae are usually resected via thoracoscope. This procedure has resulted in improved lung function and reduction in dyspnea.[2]

The third surgical procedure is *lung transplantation* that will benefit carefully selected patients with advanced COPD. Although single-lung transplant is the most commonly used technique because of a shortage of donors, bilateral transplantation can be performed. However, organ rejection, effects of immunosuppressive therapy, and the high cost of the surgery remain obstacles to any widespread use of transplantation for COPD. (Lung transplantation is discussed in Chapters 14 and 28.)

Breathing Retraining. The patient with COPD develops an increased respiratory rate with a prolonged expiration to compensate for the obstruction to air flow resulting in dyspnea. In addition, the accessory muscles of breathing in the neck and upper part of the chest are used excessively to promote chest wall movement. These muscles are not designed for long-term use, and as a result the patient experiences increased fatigue. Breathing exercises may assist the patient during rest and activity (e.g., lifting, walking, stair climbing) by decreasing

TABLE 29-22 PATIENT AND CAREGIVER TEACHING GUIDE

Home Oxygen Use

The company that provides the prescribed oxygen therapy equipment will instruct the patient on equipment care. The following are some general instructions that you may include when teaching the patient and/or caregiver about the use of home oxygen.

Decreasing Risk for Infection

- Brush teeth or use mouthwash several times a day.
- Wash nasal cannula (prongs) with a liquid soap and thoroughly rinse once or twice a week.
- Replace cannula every 2-4 weeks.
- If you have a cold, replace the cannula after your symptoms pass.
- Always remove secretions that are coughed out.
- If you use an O_2 concentrator, every day unplug the unit and wipe down the cabinet with a damp cloth and dry.
- Ask the company providing the equipment how often the filter should be changed.

Safety Issues

- Post "No Smoking" warning signs outside the home.
- Oxygen will not "blow up," but it will increase the rate of burning; it is a fuel for the flame/fire.
- Do not allow smoking in the home and do not smoke yourself while wearing O_2. Nasal cannulas and masks can catch fire and cause serious burns to face and airways.
- Do not use flammable liquids such as paint thinners, cleaning fluids, gasoline, kerosene, oil-based paints, aerosol sprays, etc, while using O_2. Do not use blankets or fabrics that carry a static charge, such as wool or synthetics.
- Inform your electric company if you are using a concentrator. In case of a power failure, it will know the medical urgency of restoring your power.

Adapted from *www.YourLungHealth.org.*

dyspnea, improving oxygenation, and slowing the respiratory rate. The main types of breathing exercises commonly taught are (1) pursed-lip breathing and (2) diaphragmatic breathing.

The purpose of **pursed-lip breathing** (PLB) is to prolong exhalation and thereby prevent bronchiolar collapse and air trapping. PLB is simple and easy to teach and learn and gives the patient more control over breathing, especially during exercise and periods of dyspnea (see Table 29-14). Patients should be taught to use "just enough" positive pressure with the pursed lips because excessive resistance may increase the work of breathing. PLB can significantly reduce dyspnea.[37]

Diaphragmatic (abdominal) breathing focuses on using the diaphragm instead of the accessory muscles of the chest to (1) achieve maximum inhalation and (2) slow the respiratory rate. To date, evidence from controlled studies does not support the use of diaphragmatic breathing in patients with COPD, and the actual effect of diaphragmatic breathing on dyspnea is unknown.[37,38] For patients with severe COPD, diaphragmatic breathing may result in hyperinflation because of increased fatigue and dyspnea and abdominal paradoxic breathing (the inward movement of the abdomen and the outward movement of the upper chest during inspiration) rather than with normal chest wall motion (the outward movement of the abdomen and upper chest simultaneously during inspiration).

PLB slows the respiratory rate and is much easier to learn than diaphragmatic breathing. In the setting of extreme acute dyspnea, it is most important to focus on helping the patient slow the respiratory rate by using the principles of PLB.

Airway Clearance Techniques. Many patients with COPD or other conditions (e.g., cystic fibrosis, bronchiectasis) who retain secretions require help to adequately clear their airways. Airway clearance techniques (ACTs) loosen mucus and secretions so they can be cleared by coughing. A variety of techniques can be used to achieve airway clearance. When ACTs are compared, no one is really better than another, and it is largely patient preference.[39,40] Respiratory therapists (RTs), physical therapists (PTs), and nurses are involved in performing these techniques.

ACTs are often used with other treatments. Typically the patient will receive bronchodilator therapy via an inhaled device (e.g., nebulization) before ACT. Then the ACT is used followed by effective coughing (e.g., huff coughing).

Effective Coughing. Many patients with COPD have developed ineffective coughing patterns that do not adequately clear their airways of sputum. They also fear they may develop spastic coughing, resulting in increased dyspnea. Although other techniques (e.g., chest physiotherapy, aerosol devices) will be used to loosen secretions and mucus, the patient must cough effectively to bring the secretions to the central airways in order to expectorate.

A forced expiratory technique, *huff coughing,* is an effective technique that the patient can be easily taught. This technique clears secretions with less change in pleural pressure and less likelihood of bronchial collapse. Before coughing you should ensure the patient is breathing deeply from the diaphragm. You should place the patient's hands on the lower, lateral chest wall and then ask the patient to breathe deeply through the nose; you should feel the patient's hands move outward, which represents a breath from the diaphragm. Guidelines for effective huff coughing are presented in Table 29-23 .

Chest Physiotherapy. **Chest physiotherapy** (CPT) is primarily used for patients with excessive bronchial secretions who have difficulty clearing them (e.g., cystic fibrosis, bronchiectasis). CPT consists of postural drainage, percussion, and vibration.

Percussion and *vibration* are manual or mechanical techniques used to augment postural drainage. These techniques are used after the patient has assumed a postural drainage position to assist in loosening the mobilized secretions (see eFig. 29-1 on the Evolve website for this chapter). Percussion, vibration, and postural drainage assist in bringing secretions into larger,

TABLE 29-23 PATIENT AND CAREGIVER TEACHING GUIDE

Guidelines for Effective Huff Coughing

When teaching effective coughing, you should:

1. Help the patient assume a sitting position with head slightly flexed, shoulders relaxed, knees flexed, and forearms supported by pillow, and if possible, with feet on the floor.

Then you should instruct the patient to:

2. Inhale slowly through the mouth while breathing deeply from the diaphragm.
3. Hold the breath for 2-3 seconds.
4. Forcefully exhale quickly as if one is fogging up a mirror with one's breath (thus creating a "huff"). (This moves the secretions to larger airways.)
5. Repeat the "huff" one or two more times while refraining from a "regular" cough.
6. Cough when mucus is felt in the breathing tubes.
7. Rest for five to ten regular breaths.
8. Repeat the huffs (three to five cycles) until you feel you have cleared mucus or you become tired.

more central airways. Effective coughing (huff coughing) is then necessary to help raise these secretions.

Postural Drainage. **Postural drainage** is the use of positioning techniques that drain secretions from specific segments of the lungs and bronchi into the trachea. The postural drainage positions used depend on the areas of lung that are involved. This is determined by patient assessment (including the patient's preference), chest x-rays, and chest auscultation. For example, some patients with left lower lobe involvement will require postural drainage of only the affected region, whereas a person with cystic fibrosis may require postural drainage of all segments.

The purpose of various positions in postural drainage is to drain each segment toward the larger airways. A side-lying position can be used for the patient who cannot tolerate a head-down position. Aerosolized bronchodilators and hydration therapy are usually administered before postural drainage. The chosen postural drainage position is maintained for about 5 minutes during percussion/vibration. A common order is two to four times a day. In acute situations, postural drainage may be performed as frequently as every 4 hours. The procedure should be planned to occur and be completed at least 1 hour before meals or 3 hours after meals.

There are also beds available that can rotate and percuss in various postural drainage positions, and these are quite effective. Some positions for postural drainage (e.g., Trendelenburg) should not be performed on the patient with chest trauma, hemoptysis, heart disease, pulmonary embolus, or head injury, and in other situations where the patient's condition is not stable.

Percussion. *Percussion* is performed in the appropriate postural drainage position with the hands in a cuplike position (Fig. 29-15). The hands are cupped, and the fingers and thumbs are closed. The cupped hand should create an air pocket between the patient's chest and the hand. Both hands are cupped and used in an alternating rhythmic fashion. Percussion is accomplished with flexion and extension of the wrists. If it is performed correctly, a hollow sound should be heard. The air-cushion impact facilitates the movement of thick mucus. A thin towel should be placed over the area to be percussed, or the patient may choose to wear a T-shirt or hospital gown.

Vibration. *Vibration* is accomplished by tensing the hand and arm muscles repeatedly and pressing mildly with the flat of the hand on the affected area while the patient slowly exhales a deep breath. The vibrations facilitate movement of secretions to larger airways. Mild vibration is tolerated better than percussion and can be used in situations where percussion may be contraindicated. Commercial vibrators are available for hospital and home use.

CPT should be performed by an individual who has been properly trained. Contraindications for CPT include situations in which there is head, neck, chest, or back instability and/or injuries; anatomic deformities; severe spasticity; mental limitations; or the patient cannot tolerate the position for other reasons. Complications associated with improperly performed CPT include fractured ribs, bruising, hypoxemia, and discomfort to the patient. CPT may not be beneficial and may be stressful for some patients. Some patients may develop hypoxemia and bronchospasms with CPT. (Consult a nursing procedures book for more detail on CPT.)

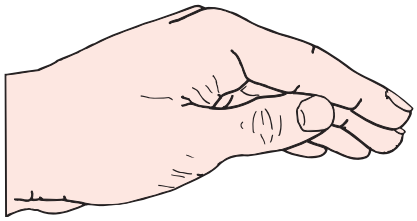


FIG. 29-15 Cupped-hand position for percussion. The hand should be cupped as though scooping up water.

Airway Clearance Devices. Various airway clearance devices are available to mobilize secretions, are easier to tolerate than CPT, and take less than half the time of conventional CPT sessions.[40,41] These devices include the Flutter, Acapella, and TheraPEP Therapy System.

The Flutter mucus clearance device is a hand-held device that is shaped like a small, fat pipe (Fig. 29-16). It provides positive expiratory pressure (PEP) treatment for patients with mucus-producing conditions. The Flutter has a mouthpiece, a high-density stainless steel ball, and a cone that holds the ball. When the patient exhales through the Flutter, the steel ball moves, which causes oscillations (vibrations) in the airways and loosens mucus. It helps move mucus up through the airways to the mouth where the mucus can be expectorated. The patient must be upright, and the angle at which the Flutter is held is critical.

The Acapella is another small hand-held device (Fig. 29-17) that combines the benefits of both PEP therapy and airway vibrations to mobilize pulmonary secretions. It can be used in virtually any setting as the patients are free to sit, stand, or recline. The patient may also inhale through it and nebulizers can be attached to the Acapella. This saves time as the treatment does not have to be preceded by the nebulizer.

TheraPEP Therapy System can also provide sustained PEP and can simultaneously deliver aerosols so the patient can inhale and exhale through it. TheraPEP has a mouthpiece attached to tubing connected to a small cylindric resistor and a pressure indicator. The pressure indicator provides visual reinforcement about the pressure the patient needs to hold in an exhalation to receive the PEP. The therapist will initially determine the pressure and you can reinforce the treatment. (A description and photo of this system are available at *www.smiths-medical.com/catalog/bronchial-hygiene/therapep/therapep-system.html*.)

High-Frequency Chest Wall Oscillation. High-frequency chest wall oscillation technology uses an inflatable vest (e.g., the VestAirway system or the SmartVest) with hoses connected to a high-frequency pulse generator. (A description and photo are available at *www.hevest.com/products*.) The pulse generator delivers air to the vest, which vibrates the chest. The high-frequency airwaves dislodge mucus from the airways, mobilize the mucus, and move it toward larger airways. The vest can be used without the aid of another person. The units weigh 23 to 30 lb and are quiet. They come in a suitcase and are portable.

Nutritional Therapy. Approximately one third of COPD patients are underweight with loss of muscle mass and cachexia, especially in the severe stages.[42] Weight loss is a predictor of poor prognosis and increased frequency of COPD exacerbations. Weight gain after nutritional support can decrease mortality risk. The cause of weight loss is not entirely known. Eating becomes an effort because of dyspnea that occurs as a result of the energy expended to chew, the reduction of airflow while swallowing, and O_2 desaturation. Therefore weight loss and muscle wasting are likely. Weight loss is also thought to occur because of the systemic inflammation that causes the metabolism to increase. This hypermetabolism could be the explanation why some individuals with COPD lose weight despite adequate nutritional intake.

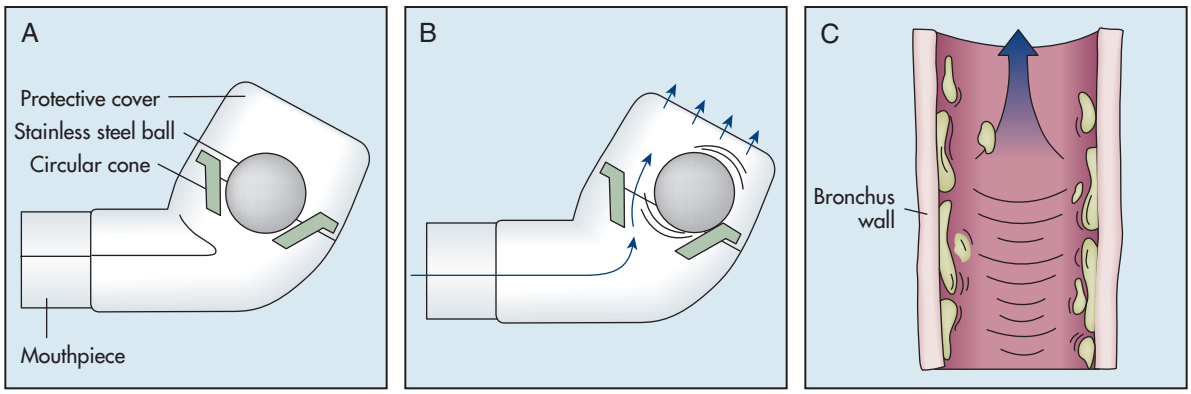


FIG. 29-16 Flutter mucus clearance device is a small hand-held device that provides positive expiratory pressure (PEP) therapy. It is used to facilitate removal of mucus from the lungs. **A,** It consists of a hard plastic mouthpiece, a plastic perforated cover, and a high-density stainless steel ball resting in a circular cone. **B,** The Flutter effects occur during expiration. Before exhalation, the ball blocks the conical canal of the Flutter. During exhalation, the position of the ball is the result of an equilibrium between the pressure of the exhaled air, the force of gravity on the ball, and the angle of the cone where contact with the ball occurs. As the steel ball rolls and moves up and down, it creates an opening and closing cycle that repeats itself many times throughout each exhalation. The net result is that vibrations occur in the airways resulting in the "fluttering" sensation. **C,** These vibrations loosen mucus from the airway walls and facilitate their movement up the airways.

FIG. 29-17 Acapella.

Nutritional supplements are needed for patients with a BMI less than 21 kg/m^2. Patients should aim to keep the BMI around 25 kg/m^2. If the patient is at BMI of 25 kg/m^2, but losing weight, you need to determine possible causes to prevent further loss. The dietitian can serve as a good resource to determine what supplement is the best for the patient.[42]

To decrease dyspnea and conserve energy, the patient should rest for at least 30 minutes before eating, use a bronchodilator before meals, and select foods that can be prepared in advance. The patient should eat five or six small, frequent meals a day to avoid feelings of bloating and early satiety when eating. Liquid, blenderized, or commercial diets may be helpful. Foods that require a great deal of chewing should be avoided or served in another manner (e.g., grated, pureed). Cold foods may give less of a sense of fullness than hot foods. Teach the patient to avoid exercise and treatments for at least 1 hour before and after eating. The exertion involved in preparing and eating food is often fatiguing. Use of frozen foods and a microwave oven may help conserve the patient's energy in food preparation. Assess the patient's dentition because broken or missing teeth or loose dentures make eating more difficult. Activity such as walking or getting out of bed during the day can stimulate the appetite and promote weight gain. Sensations of bloating and early satiety when eating can be attributed to swallowing air while eating, side effects of medication (especially corticosteroids and theophylline), and the abnormal position of the diaphragm relative to the stomach in association with hyperinflation of the lungs. Intestinal gas–forming foods should be avoided, such as cabbage, Brussels sprouts, and beans.

Underweight patients with emphysematous COPD have an increased need for protein and calories. They may need 25 to 45 kcal/kg and 1.2 to 1.9 g of protein per kilogram to maintain their weight. A diet high in calories and protein, moderate in carbohydrate, and moderate to high in fat is recommended and can be divided into five or six small meals a day. High-protein, high-calorie nutritional supplements can be offered between meals. Ice cream added to these supplements increases calories. Drinking skim or 1% milk rather than whole milk may cause less mucus production. (Nutritional supplements are discussed in Chapter 40.) Nonprotein calories should be divided evenly between fat and carbohydrate while not overfeeding the patient.[43] Getting the patient to eat adequate amounts of any foods may be difficult. If a patient has O_2 prescribed, use of supplemental O_2 by nasal cannula while eating may also be beneficial, because eating expends energy.

Fluid intake should be at least 3 L per day unless contraindicated for other medical conditions, such as heart or renal failure. Fluids should be taken between meals (rather than with them) to prevent excess stomach distention and to decrease pressure on the diaphragm. Sodium restriction may be indicated if there is accompanying heart failure.

In contrast, some patients with COPD are obese, which also causes dyspnea. The patient may have increased appetite if on oral corticosteroids and may have little mobility. You can counsel patients about food portion control and increasing their exercise.

NURSING MANAGEMENT CHRONIC OBSTRUCTIVE PULMONARY DISEASE

NURSING ASSESSMENT

Subjective and objective data that should be obtained from a person with COPD are presented in Table 29-24.

TABLE 29-24 NURSING ASSESSMENT

Chronic Obstructive Pulmonary Disease

Subjective Data

Important Health Information

Past health history: Long-term exposure to chemical pollution, respiratory irritants, occupational fumes, dust; recurrent respiratory infections; previous hospitalizations

Medications: Use of O_2 and duration of O_2 use, bronchodilators, corticosteroids, antibiotics, anticholinergics, OTC drugs, herbs, medications purchased from outside United States or Canada

Functional Health Patterns

Health perception–health management: Smoking (pack-years, including passive smoking, willingness to stop smoking, and previous attempts); family history of respiratory disease (especially α_1-antitrypsin deficiency)

Nutritional-metabolic: Anorexia, weight loss or gain

Activity-exercise: Increasing dyspnea and/or increase in sputum volume or purulence (to detect exacerbation); fatigue, ability to perform ADLs; swelling of feet; progressive dyspnea, especially on exertion; ability to walk up one flight of stairs without stopping; wheezing; recurrent cough; sputum production (especially in the morning); orthopnea

Elimination: Constipation, gas, bloating

Sleep-rest: Insomnia; sitting up position for sleeping, paroxysmal nocturnal dyspnea

Cognitive-perceptual: Headache, chest or abdominal soreness

Coping–stress tolerance: Anxiety, depression

Objective Data

General

Debilitation, restlessness, assumption of upright position

Integumentary

Cyanosis (bronchitis), pallor or ruddy color, poor skin turgor, thin skin, digital clubbing, easy bruising; peripheral edema (cor pulmonale)

Respiratory

Rapid, shallow breathing; inability to speak; prolonged expiratory phase; pursed-lip breathing; wheezing; rhonchi, crackles, diminished or bronchial breath sounds; ↓ chest excursion and diaphragm movement; use of accessory muscles; hyperresonant or dull chest sounds on percussion

Cardiovascular

Tachycardia; dysrhythmias, jugular vein distention, distant heart tones, right-sided S_3 (cor pulmonale), edema (especially in feet)

Gastrointestinal

Ascites, hepatomegaly (cor pulmonale)

Musculoskeletal

Muscle atrophy, ↑ anterior-posterior diameter (barrel chest)

Possible Diagnostic Findings

Abnormal ABGs (compensated respiratory acidosis, ↓ PaO_2 or SaO_2, ↑ $PaCO_2$), polycythemia, pulmonary function tests showing expiratory airflow obstruction (e.g., low FEV_1, low FEV_1/FVC, large RV), chest x-ray showing flattened diaphragm and hyperinflation or infiltrates

ABGs, Arterial blood gases; *ADLs,* activities of daily living; *FEV_1,* forced expiratory volume in 1 second; *FVC,* forced vital capacity; *OTC,* over-the-counter; *RV,* residual volume.

NURSING CARE PLAN 29-2

Patient with Chronic Obstructive Pulmonary Disease

NURSING DIAGNOSIS **Ineffective breathing pattern** *related to* alveolar hypoventilation, anxiety, chest wall alterations, and hyperventilation *as evidenced by* assumption of three-point position, dyspnea, increased anterior-posterior diameter, nasal flaring, orthopnea, prolonged expiration, pursed-lip breathing, use of accessory muscles to breathe

PATIENT GOALS

1. Returns to baseline respiratory function
2. Demonstrates an effective rate, rhythm, and depth of respirations

OUTCOMES (NOC)

Respiratory Status: Ventilation

- Ease of breathing _____
- Respiratory rate _____
- Respiratory rhythm _____
- Depth of inspiration _____
- Auscultated breath sounds _____
- Pulmonary function tests _____

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

- Accessory muscle use _____
- Pursed-lip breathing _____
- Dyspnea at rest _____
- Shortness of breath _____

Measurement Scale
1 = Severe
2 = Substantial
3 = Moderate
4 = Mild
5 = None

INTERVENTIONS (NIC) AND *RATIONALES*

Ventilation Assistance

- Monitor respiratory and oxygenation status *to assess need for intervention.*
- Auscultate breath sounds, noting areas of decreased or absent ventilation, and presence of adventitious sounds *to obtain ongoing data on patient's response to therapy.*
- Encourage slow deep breathing, turning, and coughing *to promote effective breathing techniques and secretion mobilization.*
- Administer medications (e.g., bronchodilators and inhalers) that promote airway patency and gas exchange.
- Position to minimize respiratory efforts (e.g., elevate the head of the bed and provide overbed table for patient to lean on) *to save energy for breathing.*
- Monitor for respiratory muscle fatigue *to determine a need for ventilatory assistance.*
- Initiate a program of respiratory muscle strength and/or endurance training *to establish effective breathing patterns and techniques.*

NURSING CARE PLAN 29-2—cont'd

Patient with Chronic Obstructive Pulmonary Disease

NURSING DIAGNOSIS **Ineffective airway clearance** *related to* expiratory airflow obstruction, ineffective cough, decreased airway humidity, and tenacious secretions *as evidenced by* ineffective or absent cough, presence of abnormal breath sounds, or absence of breath sounds

PATIENT GOALS
1. Maintains clear airway by effectively coughing
2. Experiences clear breath sounds

OUTCOMES (NOC)	INTERVENTIONS (NIC) AND *RATIONALES*
Respiratory Status: Airway Patency	***Cough Enhancement***
• Ease of breathing _____ • Moves sputum out of airway _____ **Measurement Scale** 1 = Severely compromised 2 = Substantially compromised 3 = Moderately compromised 4 = Mildly compromised 5 = Not compromised	• Assist patient to sitting position with head slightly flexed, shoulders relaxed, and knees flexed *to allow for adequate chest expansion.* • Instruct patient to inhale deeply, bend forward slightly, and perform three or four huffs (against an open glottis) *to prevent airway collapse during exhalation.** • Instruct patient to inhale deeply several times, exhale slowly, and cough at the end of exhalation *to loosen secretions before coughing.* • Instruct the patient to follow coughing with several maximal inhalation breaths *to reoxygenate the lungs.*
• Adventitious breath sounds _____ • Anxiety _____ • Choking _____ **Measurement Scale** 1 = Severe 2 = Substantial 3 = Moderate 4 = Mild 5 = None	***Airway Management*** • Encourage slow, deep breathing; turning; and coughing *to mobilize pulmonary secretions.* • Position patient *to maximize ventilation potential.* • Regulate fluid intake to optimize fluid balance *to liquefy secretions for easier expectoration.* • Perform endotracheal or nasotracheal suctioning as appropriate *to clear the airway.* • Administer bronchodilators and use airway clearance devices *to facilitate clearance of retained secretions and increase ease of breathing.*

NURSING DIAGNOSIS **Impaired gas exchange** *related to* alveolar hypoventilation *as evidenced by* headache on awakening, $PaCO_2$ ≥45 mm Hg, PaO_2 <60 mm Hg, or SaO_2 <90% at rest

PATIENT GOALS
1. Returns to baseline respiratory function
2. $PaCO_2$ and PaO_2 return to levels normal for patient

OUTCOMES (NOC)	INTERVENTIONS (NIC) AND *RATIONALES*
Respiratory Status: Gas Exchange	***Oxygen Therapy***
• Cognitive status _____ • Ease of breathing _____ • PaO_2 _____ • $PaCO_2$ _____ • Oxygen saturation _____ **Measurement Scale** 1 = Severely compromised 2 = Substantially compromised 3 = Moderately compromised 4 = Mildly compromised 5 = Not compromised	• Administer supplemental oxygen as ordered. • Set up oxygen equipment and administer through a heated, humidified system. • Periodically check oxygen delivery device *to ensure that the prescribed concentration is being delivered.* • Monitor the effectiveness of oxygen therapy (e.g., pulse oximetry, ABGs) *to evaluate patient response to therapy.* • Observe for signs of oxygen-induced hypoventilation *because this occurs with carbon dioxide narcosis.* • Instruct patient and family about use of oxygen at home *to promote safe long-term oxygen therapy.*

NURSING DIAGNOSIS **Imbalanced nutrition: less than body requirements** *related to* poor appetite, lowered energy level, shortness of breath, gastric distention, sputum production, and depression *as evidenced by* weight loss >10% of ideal body weight, serum albumin level below normal values, lack of interest in food

PATIENT GOALS
1. Maintains body weight within normal range for height and age
2. Consumes adequate nutrients for metabolic needs

OUTCOMES (NOC)	INTERVENTIONS (NIC) AND *RATIONALES*
Appetite	***Nutrition Therapy***
• Desire to eat _____ • Enjoyment of food _____ **Measurement Scale** 1 = Severely compromised 2 = Substantially compromised 3 = Moderately compromised 4 = Mildly compromised 5 = Not compromised	• Monitor food/fluid ingested and calculate daily caloric intake *to determine adequacy of intake.* • Monitor laboratory values *for evidence of malnutrition.* • Provide oral care before meals *to moisten and clean the mouth of sputum taste.* • Provide patient with high-protein, high-calorie, nutritious finger foods and drinks that can be readily consumed *to provide adequate calories and protein that do not require much energy to consume.* • Select nutritional supplements *to provide nutritional between-meal snacks.*

*Guidelines for effective huff coughing are presented in Table 29-23.

Continued

NURSING CARE PLAN 29-2—cont'd

Patient with Chronic Obstructive Pulmonary Disease

NURSING DIAGNOSIS **Imbalanced nutrition: less than body requirements**—cont'd

OUTCOMES (NOC)	INTERVENTIONS (NIC) AND *RATIONALES*
Nutritional Status: Nutrient Intake	***Nutrition Management***
• Caloric intake _____ • Protein intake _____ • Vitamin intake _____ • Mineral intake _____	• Weigh patient at appropriate intervals *to assess nutritional status.* Provide food selection *to stimulate the appetite.* • Adjust diet to patient's lifestyle *to reduce bloating.* • Provide appropriate information about nutritional needs and how to meet them *to ensure nutritional adequacy after discharge.*
Measurement Scale 1 = Not adequate 2 = Slightly adequate 3 = Moderately adequate 4 = Substantially adequate 5 = Totally adequate	

NURSING DIAGNOSES

The nursing diagnoses for the patient with COPD may include, but are not limited to, those presented in NCP 29-2.

PLANNING

The overall goals are that the patient with COPD will have (1) prevention of disease progression, (2) ability to perform ADLs and improved exercise tolerance, (3) relief from symptoms, (4) no complications related to COPD, (5) knowledge and ability to implement a long-term treatment regimen, and (6) overall improved quality of life.

NURSING IMPLEMENTATION

HEALTH PROMOTION. The incidence of COPD would decrease dramatically if people would not begin smoking or would stop smoking. (Techniques to help patients stop smoking are discussed in Chapter 12 and Tables 12-4 through 12-7.) Avoiding or controlling exposure to occupational and environmental pollutants and irritants is another preventive measure to maintain healthy lungs. (These factors are discussed in the section on nursing management of lung cancer in Chapter 28.)

Early detection of COPD is important. However, using spirometry to screen the general population who do not report respiratory symptoms to detect COPD is not recommended as having a favorable benefit/harm ratio. Counseling the patient in smoking cessation is vital as it is the only way to stop the progression of COPD. One would think that confronting a person who smokes with abnormal spirometry findings would motivate them to stop smoking. However, this approach does not always work.[44] As health care professionals, nurses who smoke should reevaluate their own smoking behavior and its relationship to their health. Nurses and other health care providers who smoke should be aware that the odor of smoke is obvious on their clothes, and it can be offensive or tempting to patients.

Early diagnosis and treatment of respiratory tract infections and exacerbations of COPD are other ways to decrease the incidence of COPD. Avoiding exposure to large crowds in the peak periods for influenza may be necessary, especially for the older adult and the person with a history of respiratory problems. Influenza and pneumococcal pneumonia vaccines are recommended for the patient with COPD.

Families with a history of COPD, as well as AAT deficiency, should be aware of the genetic nature of the disease and these individuals should have spirometry screening regularly during their adult life even though they do not have symptoms.[44] Genetic counseling is appropriate for the patient with AAT deficiency who is planning to have children.

ACUTE INTERVENTION. The patient with COPD will require acute intervention for complications such as exacerbations of COPD, pneumonia, cor pulmonale, and acute respiratory failure. (The nursing care for these conditions is discussed in Chapters 28 and 68.) Once the crisis in these situations has been resolved, you can assess the degree and severity of the underlying respiratory problem. The information obtained will help plan the nursing care.

AMBULATORY AND HOME CARE. By far the most important aspect in the long-term care of the patient with COPD is teaching. (A patient and caregiver teaching guide is presented in Table 29-25 .)

Pulmonary Rehabilitation. The widely accepted definition of pulmonary rehabilitation is an evidence-based intervention that includes many disciplines working together to individualize treatment of the patient with chronic respiratory disease who has symptoms and decreased quality of life. Pulmonary rehabilitation is designed to reduce symptoms and improve the functional abilities of the patient while reducing health care costs as the patient is stabilized or the systemic aspects of the disease are reversed.[45] Pulmonary rehabilitation should be considered in all patients who continue to be disabled by pulmonary symptoms even though they are receiving appropriate and standard medical care. Rehabilitation benefits patients with COPD by improving their exercise capacity and their health-related quality of life. There are also reductions in perceived intensity of breathlessness, number of hospitalizations and days in the hospital, anxiety, and depression.[2] Pulmonary rehabilitation should no longer be viewed as a "last ditch" effort for patients with severe COPD.

Pulmonary rehabilitation can be done in an inpatient or outpatient setting, or in home settings. A mandatory component of any pulmonary rehabilitation program is exercise that focuses on the muscles used in ambulation.[46] Ideally, pulmonary rehabilitation includes exercise training, nutrition counseling, and education. Other important topics include health promotion,

TABLE 29-25 PATIENT AND CAREGIVER TEACHING GUIDE

Chronic Obstructive Pulmonary Disease

You should include the following information in the teaching plan.

Goal

To assist a patient and caregiver in improving quality of life through education and promotion of lifestyle practices that support successful living with chronic obstructive pulmonary disease (COPD).

TEACHING TOPIC	RESOURCES
Overall Guide	*Global Initiative for Lung Disease (GOLD) Patient Guide: What You Can Do about a Lung Disease Called COPD.* Available at *www.goldcopd.org.* Also available in foreign languages.
What Is COPD?	
• Basic anatomy and physiology of lung • Basic pathophysiology of COPD • Signs and symptoms of COPD, exacerbation, cold, flu, pneumonia • Tests to assess breathing	*COPD Statement: Patient Education Section* (American Thoracic Society [ATS]). Available at *www.thoracic.org.* (Also in Spanish.) *Human Respiratory System* and *Learn About Your Respiratory System* (American Lung Association [ALA]). Available under Your Lungs at *www.lungsusa.org.*
Breathing and Airway Clearance Exercises	
• Pursed-lip breathing • Airway clearance technique—huff cough	See Table 29-14. See Table 29-23.
Energy Conservation Techniques	
• Daily activities (e.g., waking up, bathing, grooming, shopping, traveling)	Consult with physical therapist and occupational therapist. *Around the Clock with COPD: Helpful Hints for Respiratory Patients* (ALA). Available in COPD Center at *www.lungusa.org.*
Medications	
Types (include mechanism of action and types of devices) • Methylxanthines • β_2-adrenergic agonists • Corticosteroids • Anticholinergics • Antibiotics • Other medications	*COPD Statement: Patient Education Section: Medications and Other Treatments* (ATS). Available at *www.thoracic.org.* **OR** *COPD Medicines Chart* (ALA). Available in COPD Center at *www.lungusa.org.* **OR** *COPD Medications (National Jewish Medicine and Research Center).* Available at *www.nationaljewish.org/medfacts.html.*
Establishing medication schedule	Write out medication list and schedule. Form available at National Jewish link above "Manage Your Medications."
Correct Use of Inhalers, Spacer, and Nebulizer	See Figs. 29-6, 29-7, and 29-8. See Tables 29-8, 29-9, and 29-10.
Home Oxygen	
• Explanation of rationale for use • Guide for home O_2 use and equipment	*Around the Clock with COPD: Helpful Hints for Respiratory Patients and Traveling with Oxygen* (ALA). Available in COPD Center at *www.lungusa.org.*
Psychosocial/Emotional Issues	
Concerns about interpersonal relationships • Dependency • Intimacy	Open discussion (sharing with patient, significant other, and family). COPD Lung NexProfiler (interactive decision support tool via ALA). Available at *www.lungusa.org.*
Problems with emotions • Depression, anxiety, panic	*Questions about Pulmonary Rehabilitation* (English and Spanish) (ATS). Available at *www.thoracic.org.*
Treatment decisions • Support and rehabilitation groups	*Better Breathers Clubs* and *Living with Lung Disease* (online support group) (ALA). Available at *www.lungusa.org.*
End-of-life issues	
COPD Management Plan	
• Focus on self-management • Need to report changes • Cause of flare-ups or exacerbation • Recognition of signs and symptoms of respiration infection, heart failure • Reduce risk factors, especially smoking cessation • Exercise program of walking and arm strengthening • Yearly follow-up	Nurse and patient develop and write up COPD management plan that meets individual needs.
Healthy Nutrition	
• Strategies to lose weight (if overweight) • Strategies to gain weight (if underweight)	Consultation with dietitian.

psychologic counseling, and vocational rehabilitation. Smoking cessation is critical to the success of the patients. Some rehabilitation programs will not accept patients who are current smokers and not committed to quitting. Physical therapists or nurses who have experience in pulmonary care are often responsible for the management of pulmonary rehabilitation centers. A large part of your role is to teach patients self-management of their disease. The minimum length of an effective program is 6 weeks, but the longer the program, the more effective the results.

Activity Considerations. Exercise training leads to energy conservation, which is an important component in COPD rehabilitation. The COPD patient is typically an upper thoracic and neck breather who uses accessory muscles rather than the diaphragm. Thus the patient has difficulty performing upper-extremity activities, particularly those activities that require arm elevation above the head. Exercise training of the upper extremities may improve muscle function and help to reduce dyspnea. Frequently the patient has already adapted alternative energy-saving practices for ADLs. Alternative methods of hair care, shaving, showering, and reaching may need to be explored. An occupational therapist may help with ideas in these areas. Assuming a tripod posture (elbows supported on a table, chest in fixed position) and a mirror placed on the table

during use of an electric razor or hair dryer conserves much more energy than when the patient stands in front of a mirror to shave or blow-dry hair. If the patient uses home O_2 therapy, it is essential that the patient use the O_2 during activities of hygiene, because these are energy consuming. Encourage the patient to make a schedule and plan daily and weekly activities to leave plenty of time for rest periods. The patient should also try to sit as much as possible when performing activities. Another energy-saving tip is to exhale when pushing, pulling, or exerting effort during an activity and inhale during rest.

Walking or other endurance exercises (e.g., cycling) combined with strength training is likely the best intervention to strengthen muscles and improve the patient's endurance. Coordinated walking with slow, pursed-lip breathing without breath holding is a difficult task that requires conscious effort and frequent reinforcement. During coordinated walking and breathing, teach the patient to breathe in through the nose while taking one step, then to breathe out through pursed lips while taking two to four steps (the number depends on the patient's tolerance). Walking should occur at a slow pace with rest periods when necessary so the patient can sit or lean against an object such as a tree or post. The patient may need to ambulate using O_2. The patient may be able to successfully perform coordinated walking with pursed-lip breathing. You should walk with the patient, giving verbal reminders when necessary regarding breathing (inhalation and exhalation) and steps. Walking with the patient helps decrease anxiety and helps maintain a slow pace. It also enables you to observe the patient's actions and physiologic responses to the activity. Many patients with moderate or severe COPD are anxious and fearful of walking or performing exercise. These patients and their caregivers require much support while they build the confidence they need to walk or to perform daily exercises.

In many situations pulmonary rehabilitation programs are not an option, and patients are advised to exercise on their own. Encourage the patient to walk 15 to 20 minutes a day at least three times a week with gradual increases. Severely disabled patients can begin at a slow pace by walking for 2 to 5 minutes three times a day and slowly building up to 20 minutes a day, if possible. Allow adequate rest periods. Some patients benefit from using their β_2-adrenergic agonist approximately 10 minutes before exercise. Parameters that may be monitored in the patient with mild COPD are resting pulse and pulse rate after walking. Pulse rate after walking should not exceed 75% to 80% of the maximum heart rate (maximum heart rate is age in years subtracted from 220). In some patients it is usually dyspnea and the limitation in breathing rather than increased heart rate that limits the exercise. Thus it is better to use the patient's perceived sense of dyspnea as an indication of exercise tolerance.

Tell the patient that shortness of breath will probably increase during exercise (as it does for a healthy individual) but that the activity is not being overdone if this increased shortness of breath returns to baseline within 5 minutes after the cessation of exercise. Instruct the patient to wait 5 minutes after completion of exercise before using the β_2-adrenergic agonist to allow a chance to recover. During this time, slow, pursed-lip breathing should be used. If it takes longer than 5 minutes to return to baseline, the patient most likely has overdone it and should proceed at a slower pace during the next exercise period. The patient may benefit from keeping a diary or log of the exercise program. The diary can help provide a realistic evaluation of the patient's progress. In addition, the diary can help motivate the patient and add to the patient's sense of accomplishment. Stationary cycling can also be used either alone or with walking. Cycles and treadmills are particularly good when weather prevents walking outside.

Fatigue, sleep disturbances, and dyspnea are common complaints of patients with COPD. Of these symptoms, dyspnea appears to be the only symptom that affects the patient's ability to carry out daily activities. Therefore you and other health team members should focus your interventions on improving dyspnea, which would then improve the patient's functional performance. Nutritional counseling is integral to a pulmonary rehabilitation program and this has been discussed previously in the chapter. Education is an important component of pulmonary rehabilitation and it should include information on self-management and prevention and treatment of exacerbations (see Table 29-25).

Sexuality and Sexual Activity. Modifying but not abstaining from sexual activity can also contribute to a healthy psychologic well-being. Most of the patients with COPD are older. You need to assess and reflect on your own attitudes and feelings about sexuality, sexual functioning, and aging before exploring sexual issues with the older COPD patient. You need to first assess the patient related to sexuality and concerns of functioning. Ask open-ended questions to determine if the patient wants to discuss any of these concerns. You could ask, "How has your breathing problem affected how you see yourself as a woman or man?" Another question could be, "How does your shortness of breath affect your desire for intimacy with your partner?" Moving through these types of questions will give the person an opening to discuss concerns. Much of the sexual performance issues experienced by the patient with COPD are changes related to aging and if you are aware of these changes, the patient can be taught the "normalcy" of the changes.

Dyspnea is the predominant symptom in COPD but it should not be a major problem with success in sexual functioning, except for those patient in stage 3 or 4. Erectile dysfunction can occur, and is related to the severity of the underlying disease. Using an inhaled bronchodilator before sexual activity can help ventilation. The patient with COPD may also find these suggestions helpful: (1) plan sexual activity during the part of the day when breathing is best, which is usually late morning or early afternoon (plus older men often achieve an erection easier in the morning); (2) use slow pursed-lip breathing; (3) refrain from sexual activity after eating or alcohol consumption; (4) choose less stressful positions during intercourse; (5) use O_2 if prescribed; and (6) understand that cigarettes can increase male impotence. Often the patient has coexisting cardiac disease, and obtaining advice from a health care provider related to appropriate levels of activity would be advisable.[47] These aspects of sexual activity require open communication between partners regarding their needs and expectations, and the changes that may be necessary as the result of a chronic disease (e.g., changes in body image, role reversal).

Sleep. Adequate sleep is extremely important. Getting adequate amounts of quality sleep can be difficult for the COPD patient. The hyperinflation of the lungs and the reduction in ventilation can result in severe drops in O_2 saturation (down to 60% or less) during rapid eye movement (REM) sleep. This leads to a strain on the heart. In addition, impaired sleep hypercapnia develops and the patient may awaken more often. The net result is poor quality of sleep and awakening unrefreshed and

fatigued. B_2-Agonists often cause restlessness and insomnia. Theophylline can impair sleep by increasing the time it takes for the patient to go to sleep.[48] Many patients with COPD have postnasal drip or nasal congestion that may cause coughing and wheezing at night. Nasal saline sprays or rinses before sleep and in the morning may help. If the patient is a restless sleeper, snores, stops breathing while asleep, and has a tendency to fall asleep during the day, the patient may need to be tested for sleep apnea (see Chapter 9).

Psychosocial Considerations. Healthy coping is often the most difficult task for a patient with COPD to accomplish. People with COPD frequently have to deal with many lifestyle changes that may involve decreased ability to care for themselves, decreased energy for social activities, and loss of a job.

When a patient with COPD is first diagnosed or when a patient has complications that require hospitalization, you should expect a variety of emotional responses. Emotions frequently encountered include guilt, depression, anxiety, social isolation, denial, and dependence. Guilt may result from the knowledge that the disease was caused largely by cigarette smoking. Many patients struggle with depression.[49] You should convey a sense of understanding and caring to the patient. The patient with COPD may benefit from stress management techniques (e.g., massage, progressive muscle relaxation) (see Chapter 8). Support groups at local American Lung Associations, such as the Better Breathers Club, hospitals, and clinics can also be helpful.

ETHICAL DILEMMAS

Advance Directives

Situation

A 79-year-old man with COPD is in respiratory failure when he is admitted to the hospital. He is placed on a ventilator and responds occasionally by opening his eyes. His advance directives (ADs) were drawn up 5 years ago and copies were given to his wife and health care provider at that time. The patients's wife brings the documents to the intensive care unit and tells you that the hospital must stop treating her husband and allow him to die as he requested. However, the patient's oldest son is threatening the hospital with a lawsuit if the staff does not provide full care to his father.

Important Points for Consideration

- ADs are prepared by the person indicating his or her treatment wishes should the person become terminally ill or in a situation where there is no hope of recovery.*
- On admission, determine whether the patient has an AD and if the patient does, place a copy in the medical record.
- A determination needs to be made whether this is a respiratory crisis that is reversible or whether the patient is terminally ill.
- Durable power of attorney for health care is a form of an AD in which a person names another to make health care decisions in the event the person is no longer able to do so.
- ADs are legally binding in most states.
- Health care providers are obligated to follow the patient's ADs when a patient is no longer able to speak for himself or herself.

Clinical Decision-Making Questions

1. What should you do next with the information provided by the patient's wife?
2. How should you address the needs of each member of this family in the patient's plan of care?
3. What resources can you use to facilitate decision making in this situation?

*Table 11-6 explains common documents used in end-of-life care.

Patients frequently ask whether moving to a warmer or drier climate will help. In general, such a move is not significantly beneficial. Moving to places with an elevation of 4000 feet or more should be discouraged because of the lower partial pressure of O_2 found in the air at higher elevations. A disadvantage of moving may be that a person leaves an occupation, friends, and familiar environment, which could be psychologically stressful. Any advantage gained from a different climate may be outweighed by the psychologic effects of the move.

Patients need to know that symptoms can be controlled for the most part, but COPD cannot be cured. End-of-life issues and advance directives are important topics for discussion in the terminal stages of COPD. However, this may be difficult for the patient and family to consider because of the uncertainty of the disease. (Palliative care and end-of-life care are discussed in Chapter 11.)

EVALUATION

The expected outcomes for the patient with COPD are presented in NCP 29-2.

CYSTIC FIBROSIS

Cystic fibrosis (CF) is an autosomal recessive, multisystem disease characterized by altered function of the exocrine glands primarily involving the lungs, pancreas, and sweat glands.[50] (Autosomal recessive disorders are discussed in Chapter 14.) Abnormally thick, abundant secretions from mucous glands can lead to a chronic, diffuse, obstructive pulmonary disorder in almost all patients. Exocrine pancreatic insufficiency is associated with most cases of CF. Sweat glands excrete increased amounts of sodium and chloride. End-stage lung disease is the principal cause of death.

Approximately 30,000 children and adults in the United States have CF. About 10 million (about 1 in every 31) Americans are carriers of the CF gene, but do not have the disease. Of the CF patients in the Cystic Fibrosis Registry, over 45% are 18 years of age or older. CF is most common in whites, but it can affect all races. In adults, the ethnic breakdown is 94% white, 7% Hispanic, and 4% African American. Nearly 52% of the patients with CF are males. The first signs and symptoms typically occur in children, but some patients are not diagnosed until they are adults and some live to age 80. The severity and progression of the disease vary from person to person. With early diagnosis and improvements in therapy, the prognosis of patients with CF has significantly improved. The median predicted survival in 1970 was 16 years, but has increased to more than 37 years.[51] Nurses who work in adult care settings will increasingly manage patients with CF.[50]

Etiology and Pathophysiology

The CF gene is located on chromosome 7 and produces a protein called CF transmembrane regulator (CFTR). The CFTR protein localizes to the lining of the exocrine portion of particular organs such as the airways, pancreatic duct, sweat gland duct, and reproductive tract. CFTR regulates sodium and chloride channels. Mutations in the CFTR gene alter this protein in such a way that the channels are blocked. As a result, cells that line the passageways of the lungs, pancreas, and other organs produce abnormally thick, sticky mucus. This mucus fills (plugs up) the glands in these organs and causes the glands to atrophy, ultimately resulting in organ failure. The high concentrations of

GENETICS IN CLINICAL PRACTICE

Cystic Fibrosis (CF)

Genetic Basis
- Autosomal recessive disorder
- Mutations in CFTR gene cause CF
- Gene location on chromosome 7
- Many different mutations of the gene have been identified

Incidence
- In the United States 1 in 3000 white births
- Uncommon in other ethnic populations
- 1 in 20-25 whites are carriers of the gene
- If both parents carry the affected gene, there is a 25% chance each offspring will have the disease (see Figs. 14-1 and 14-2 and eFig. 14-2 on the Evolve website for that chapter)

Genetic Testing
- Blood-based DNA testing is available for disease and carrier states.
- All 50 states have passed legislation requiring that all newborns be screened for cystic fibrosis (CF).
- Testing is usually done in children if CF is suspected or if parents are possible carriers.
- In parents who are known carriers, amniocentesis or chorionic villus sampling in pregnant women may be useful for prenatal testing.

Clinical Implications
- CF is the most common autosomal recessive disease in whites.
- CF has a wide range of clinical expression of disease.
- CF requires long-term medical management.
- Advances in medical care have improved life expectancy.
- Most people who have a child with CF are not aware of a family history of disease.
- CF screening should be offered to all individuals of reproductive age regardless of family history.

sodium and chloride in the sweat of the patient with CF result from decreased chloride reabsorption in the sweat duct.[15,52]

In the respiratory system, both the upper and lower respiratory tracts can be affected. Upper respiratory tract manifestations may be present and include chronic sinusitis and nasal polyposis. The hallmark of respiratory involvement in CF is its effect on the airways. The disease progresses from being a disease of the small airways *(chronic bronchiolitis)* to involvement of the larger airways, and finally causes destruction of lung tissue. The mucus becomes dehydrated and tenacious due to the defect in the chloride secretion and excess sodium absorption. Cilia mobility is decreased, thus allowing mucus to adhere to the airways. The bronchioles become obstructed with thick mucus, leading to air trapping and hyperinflation of the lungs.

CF is characterized by chronic airway infection that is difficult to eradicate. Organisms commonly cultured from the sputum of a patient with CF are *Staphylococcus aureus, Haemophilus influenzae, Burkholderia cepacia,* and *Pseudomonas aeruginosa,* with the latter being by far the most common. Antibiotic resistance often develops. Pulmonary inflammation may precede the chronic infection and can cause a decrease in respiratory function. Inflammatory mediators (e.g., interleukins, oxidants and proteases released by neutrophils) are increased and contribute to the progression of lung disease.[52]

Lung disorders that initially occur are chronic bronchiolitis and bronchitis, but after months or years changes in the bronchial walls lead to bronchiectasis (see Fig. 29-20). Over a long period of time, pulmonary vascular remodeling occurs because of local hypoxia and arteriolar vasoconstriction with pulmonary hypertension and cor pulmonale resulting in the later phases of the disease. Blebs and large cysts in the lung are also severe manifestations of lung destruction, and pneumothorax may develop. Other pulmonary complications include hemoptysis occurring because of erosion of enlarged pulmonary arteries. Hemoptysis may range from scant streaking to major bleeding; it can sometimes be fatal.

The sweat glands of CF patients secrete normal volumes of sweat, but sodium chloride cannot be absorbed from sweat as it moves through the sweat duct. Therefore they excrete four times the normal amount of sodium and chloride in sweat. This abnormality usually does not affect the general health of the person, but it is useful in diagnosis (explained later in the diagnostic studies section).

Pancreatic insufficiency is caused primarily by mucous plugging of the pancreatic exocrine ducts, which results in atrophy of the gland and progressive fibrotic cyst formation. The exocrine function of the pancreas may be completely lost.[15] Because of the exocrine dysfunction, pancreatic enzymes such as lipase, amylase, and the proteases (trypsin, chemotrypsin) do not reach the intestine to digest ingested nutrients. There is malabsorption of fat, protein, and fat-soluble vitamins (vitamins A, D, E, and K). Fat malabsorption results in steatorrhea, and protein malabsorption results in failure to grow and gain weight.

CF-related diabetes mellitus (CFRD) results from fibrotic scarring of the pancreas. CFRD is found in 35% of adults ages 20 to 29 and 43% of those over 30 years old.[51] CFRD is a unique type of diabetes with characteristics of both type 1 and type 2 diabetes. The pancreas in persons with CF produces small amounts of insulin, but not enough to fully respond to carbohydrate intake. Insulin is used to manage CFRD.

Other common disorders that develop in CF include osteopenia and osteoporosis. The etiology relates to malnutrition, insufficient testosterone levels, chronically elevated inflammatory cytokines, and the direct effect of the CFTR mutation on the development of bone.[50]

Individuals with CF often have other gastrointestinal problems, including abdominal pain, which may be caused by conditions such as GERD. The liver and gallbladder can be damaged by mucus deposits. The liver enzymes may become chronically elevated with cirrhosis developing over time. Gallstones, pancreatitis, and portal hypertension can also occur.[15] Distal intestinal obstruction syndrome (DIOS) is a syndrome that results from intermittent obstruction commonly occurring in the terminal ileum. It is usually caused by thickened stool and mucus. The patient may appear to have a small bowel obstruction. DIOS develops because of chronic malabsorption related to exocrine dysfunction, nonadherence to enzyme supplementation, dehydration, swallowing of mucus, and use of opioids.

Nearly all men with CF have reproductive issues because they have congenital absence of the vas deferens, which transports the sperm from the storage in the testes to the penile urethra. However, they make sperm normally and thus with assisted reproductive technology have the capability of fathering a child. Conversely, only 20% of women are infertile, which is related to the thickened cervical mucus or malnutrition. In addition, the higher expression of CFTR in the reproductive tissues may result in fallopian tube/uterine wall abnormalities.[53,54]

Clinical Manifestations

The clinical manifestations of CF vary depending on the severity of the disease. The manifestations of the disorder are caused by the production of abnormally thick, sticky mucus in the body's organs. Carriers are not affected by the mutation. Usually within a family the clinical manifestations are consistent among family members. However, there may be a great variation between different families.[15]

The median age of diagnosis of CF is 6 months of age with the most common symptoms respiratory or gastrointestinal.[51] An initial finding of meconium ileus in the newborn infant prompts the diagnosis in 20% of persons with CF.[52] Early manifestations in childhood are failure to grow, clubbing, persistent cough with mucus production, tachypnea, and large, frequent bowel movements. A large, protuberant abdomen may develop with an emaciated appearance of the extremities. A diagnosis may also be prompted by wheezing, coughing, or frequent pneumonia.

The first symptom of CF in the adult is frequent cough. With time the cough becomes persistent and produces viscous, purulent, often greenish-colored sputum as the prevalence of *Pseudomonas* occurs in over 50% of persons with CF.[51] Other respiratory problems that may be indicative of CF are recurring lung infections such as bronchiolitis, bronchitis, and pneumonia. As the disease progresses, periods of clinical stability are interrupted by exacerbations characterized by increased cough, weight loss, increased sputum, and decreased pulmonary function. Over time the exacerbations become more frequent, bronchiectasis develops, and the recovery of lost lung function is less complete, which may ultimately lead to respiratory failure.

If the patient with CF develops DIOS, he or she may have right lower quadrant pain, loss of appetite, emesis, and often a palpable mass. Insufficient pancreatic enzyme release causes the typical pattern of protein and fat malabsorption with a person being thin with a low BMI and frequent, bulky, foul-smelling stools.

The function of the reproductive system is altered. This finding is important because more persons with CF are living to adulthood. The male adult is usually sterile (although not impotent). The female usually has delayed menarche, likely the result of systemic illness of chronic lung disease and inadequate nutrition.[53] During exacerbations, menstrual irregularities and secondary amenorrhea are common. A majority of women with CF are able to become pregnant, and with adequate pulmonary health and nutrition they do fairly well.[50] The baby is heterozygous (and hence a carrier) for CF if the father is not a carrier. If the father is a carrier, there is a 50% chance that the baby will have CF. (For an explanation of the genetic transmission of CF, see Chapter 14, Figs. 14-1 and 14-2 and eFig 14-2 on the Evolve website for that chapter.)

Complications

Pneumothorax is common because of the formation of bullae and blebs. The presence of small amounts of blood in sputum is common in the CF patient with lung infection. Massive hemoptysis is life threatening. With advanced lung disease, digital clubbing becomes evident in almost all patients with CF. CFRD, bone disease, and liver disease are also common complications.[51] Respiratory failure and cor pulmonale are late complications of CF.

Diagnostic Studies

The diagnostic criteria for CF involve a combination of clinical presentation, laboratory testing, and genetic testing to confirm the diagnosis. Even though the sweat chloride test is the gold standard for CF diagnosis, the diagnosis is not always clear-cut in all individuals, especially in adults. The sweat chloride test is performed with the pilocarpine iontophoresis method. Pilocarpine is placed on the skin and carried by a small electric current to stimulate sweat production. This part of the process takes about 5 minutes and the patient will feel a slight tingling or warmth. The sweat is collected on filter paper or gauze and then analyzed for sweat chloride concentrations. The test takes approximately 1 hour. Values above 60 mmol/L for sweat chloride are consistent with the diagnosis of CF. However, a second sweat chloride test is recommended to confirm the diagnosis unless genetic testing identifies two CF mutations.[55]

In a genetic test, a blood sample or cells taken from the inside of the cheek are sent to a laboratory that specializes in genetic testing. Most laboratories only test for the most common mutations of the CF gene. Because there are more than 1400 mutations that cause CF, screening for all mutations is not possible. A genetic test is often used if the results from a sweat chloride test are unclear.

Collaborative Care

A multidisciplinary team should be involved in the care of a patient with CF. The Cystic Fibrosis Foundation provides funding for and accredits more than 100 CF care centers nationwide. The high quality of specialized care available throughout the care center network has led to the improved length and quality of life for people with CF. Located at teaching and community hospitals across the country, these care centers offer the best care, treatments, and support for those with CF (see *www.cff.org/LivingWithCF/CareCenterNetwork/CFFoundation-accreditedCareCenters*). Team members should include a nurse, physician, respiratory and physical therapist, dietitian, pharmacist, and social worker. The major objectives of therapy in CF are to (1) promote clearance of secretions, (2) control infection in the lungs, and (3) provide adequate nutrition.

Management of pulmonary problems in CF aims at relieving airway obstruction and controlling infection. Drainage of thick bronchial mucus is assisted by aerosol and nebulization treatments of medications used to liquefy mucus and to facilitate coughing. The abnormal viscosity of CF secretions is caused by concentrated DNA from neutrophils involved in chronic infection. Agents that degrade the DNA in CF sputum (e.g., DNase [Pulmozyme]) increase airflow and reduce the number of acute pulmonary exacerbations. Inhaled hypertonic saline (7%) is effective in clearing mucus and also decreases the frequency of exacerbations. Hypertonic saline is safe, but some patients require concomitant bronchodilators to avoid bronchoconstriction. Bronchodilators (e.g., β_2-adrenergic agonists) may be used to control bronchoconstriction, but the long-term benefit is not proven.[50,53]

Airway clearance techniques are critical. These techniques include CPT, positive expiratory pressure (PEP) devices (e.g., Flutter device [see Fig. 29-16], Acapella [see Fig. 29-17]), breathing exercises, and high-frequency chest wall oscillation systems. Individuals with CF may have a preference for a certain technique or device that works well for them in a daily routine. No clear evidence exists that any of the airway clearance

techniques are superior to the others.[40,41] The patient then follows with the huff coughing technique to expel the secretions (see Table 29-23). (Airway clearance techniques are discussed on p. 623.)

More than 95% of CF patients die of complications resulting from lung infection.[53] Standard treatment includes antibiotics for exacerbations and chronic suppressive therapy. The use of antibiotics should be carefully guided by sputum culture results. Early intervention with antibiotics is useful, and long courses of antibiotics are the usual treatment. Prolonged high-dose therapy may be necessary because many drugs are abnormally metabolized and rapidly excreted in the patient with CF. There is no evidence to support the chronic use of oral antibiotics in adults with CF.

Most patients will have *Pseudomonas,* which is difficult to treat. The standard of care to treat patients who are chronically infected is aerosolized tobramycin (TOBI) given every other month, every day, twice a day, and this increases lung function and decreases frequency of exacerbations. In addition, daily doses of azithromycin are often administered. Oral agents commonly used for mild exacerbations (i.e., increased cough and sputum) are oral antibiotics such as a semisynthetic penicillin or trimethoprim/sulfamethoxazole. Oral quinolones, especially ciprofloxacin (Cipro), are rarely used because of the rapid emergence of resistant organisms. More severe exacerbations require a 2- to 4-week course of IV antimicrobial therapy.

If home support and resources are adequate, the CF patient and caregiver may choose to continue IV antibiotic therapy at home. The usual treatment is two antibiotics with different mechanisms of action (e.g., cephalosporin and an aminoglycoside).[50,53] The patient with cor pulmonale or hypoxemia may require home O_2 therapy. (O_2 therapy is discussed on p. 618.) Patients with a large pneumothorax will require chest tube drainage, perhaps repeatedly. Sclerosing of the pleural space or partial pleural stripping and pleural abrasion performed surgically may be indicated for recurrent episodes of pneumothorax. With massive hemoptysis, bronchial artery embolization is performed. CF has become a leading indication for lung transplantation.[56] (Lung transplants are discussed in Chapter 28.)

The management of pancreatic insufficiency includes pancreatic enzyme replacement of lipase, protease, and amylase (e.g., Pancrease, Creon, Ultrase) administered before each meal and snack. Adequate intake of fat, calories, protein, and vitamins is important. Fat-soluble vitamins (vitamins A, D, E, and K) must be supplemented. Use of caloric supplements improves nutritional status. Added dietary salt is indicated whenever sweating is excessive, such as during hot weather, when fever is present, or from intense physical activity. Hyperglycemia may require insulin treatment.

If the patient develops DIOS with complete bowel obstruction, gastric decompression and surgery may be needed. Partial and uncomplicated episodes of DIOS are treated with ingestion of a balanced polyethylene glycol (PEG) electrolyte solution (MiraLax, GoLYTELY) used to thin bowel contents. In addition, water-soluble contrast enemas may be used.[50] Constipation develops in the sigmoid colon and progresses proximally, whereas DIOS develops in the terminal ileum and progresses distally. Careful monitoring of bowel habits and patterns is essential for CF patients.

Aerobic exercise seems to be effective in clearing the airways. Important needs to consider when planning an aerobic exercise program for the patient with CF are (1) frequent rest periods interspersed throughout the exercise regimen, (2) meeting increased nutritional demands of exercise, (3) observing for manifestations of hyperthermia, and (4) drinking large amounts of fluid and replacing salt losses.

More than 20% of adults with CF have depression as CF imposes a significant burden on the individual and family. Issues such as fertility, decreased life expectancy, costs of health care, and career choice are but a few of the issues faced.[51]

EVIDENCE-BASED PRACTICE

How Can Nurses Support Breathless Patients with Advanced Disease?

Clinical Question

For patients with breathlessness due to advanced disease (P), what nondrug and noninvasive interventions (I) decrease the subjective distress of breathlessness (O)?

Best Available Evidence

Systematic review of randomized controlled trials (RCTs) or controlled trials without randomization

Critical Appraisal and Synthesis of Evidence

- Forty-seven RCTs (n = 2532) of patients with breathlessness due to chronic obstructive pulmonary disease, advanced cancer, interstitial lung disease, chronic heart failure, or motor neuron disease
- Major parameter assessed was subjective breathlessness
- Strong evidence for neuroelectrical muscular stimulation and chest wall vibration and moderate evidence for use of walking aids and breathing training to relieve breathlessness

Conclusion

Breathing training, walking aids, neuroelectrical muscular stimulation, and chest wall vibration appear to relieve breathlessness in advanced disease stages.

Implications for Nursing Practice

- Breathless patients may experience severe discomfort and family members may be distressed.
- Counsel patients about nondrug options for managing breathlessness.
- Consult health team members regarding specialized treatments, equipment, and nurse training.

Reference for Evidence

Bausewein C, Booth S, Gysels M, et al: Non-pharmacological interventions for breathlessness in advanced stages of malignant and non-malignant diseases, *Cochrane Database Syst Rev* 2:CD005623, 2008.

P, Patient population of interest; *I,* intervention or area of interest; *O,* outcome(s) of interest (see p. 6).

NURSING MANAGEMENT
CYSTIC FIBROSIS

NURSING ASSESSMENT

Subjective and objective data that should be obtained from the patient with CF are presented in Table 29-26.

NURSING DIAGNOSES

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

- Ineffective airway clearance *related to* abundant, thick bronchial mucus, weakness, and fatigue
- Ineffective breathing pattern *related to* bronchoconstriction, anxiety, and airway obstruction

TABLE 29-26 NURSING ASSESSMENT

Cystic Fibrosis

Subjective Data

Important Health Information

Past health history: Recurrent respiratory and sinus infections, persistent cough with excessive sputum production

Medications: Use of and compliance with bronchodilators, antibiotics, herbs

Functional Health Patterns

Health perception–health maintenance: Family history of cystic fibrosis; diagnosis of cystic fibrosis in childhood, genetic testing in offspring

Nutritional-metabolic: Dietary intolerances, voracious appetite, weight loss, heartburn

Elimination: Intestinal gas; large, frequent bowel movements, constipation

Activity-exercise: Fatigue, ↓ exercise tolerance, amount/type of exercise; dyspnea, cough, excessive mucus or sputum production, coughing up blood, airway clearance techniques

Cognitive-perceptual: Abdominal pain

Sexuality-reproductive: Delayed menarche, menstrual irregularities, problems conceiving or fathering a child

Coping–stress tolerance: Anxiety, depression, problems adapting to diagnosis

Objective Data

General

Restlessness; failure to thrive

Integumentary

Cyanosis (circumoral, nail bed), digital clubbing; salty skin

Eyes

Scleral icterus

Respiratory

Sinus difficulties; persistent runny nose; diminished breath sounds, sputum (thick, white or green, tenacious), hemoptysis, ↑ work of breathing, use of accessory muscles of respiration, barrel chest

Cardiovascular

Tachycardia

Gastrointestinal

Protuberant abdomen; abdominal distention; foul, fatty stools

Possible Diagnostic Findings

Abnormal ABGs and pulmonary function tests; abnormal sweat chloride test, chest x-ray, fecal fat analysis

- Impaired gas exchange *related to* recurring lung infections
- Imbalanced nutrition: less than body requirements *related to* dietary intolerances, intestinal gas, and altered pancreatic enzyme production
- Ineffective coping *related to* multiple life stressors such as decreased life expectancy, cost of treatment, and limitation on career choices

PLANNING

The overall goals are that the patient with CF will have (1) adequate airway clearance, (2) reduced risk factors associated with respiratory infections, (3) adequate nutritional support to maintain appropriate BMI, (4) ability to perform ADLs, (5) recognition and expedient treatment of complications related to CF, and (6) active participation in planning and implementing a therapeutic regimen.

NURSING IMPLEMENTATION

You and other health professionals can assist young adults to gain independence by helping them assume responsibility for their care and for their vocational or school goals. An important issue that should be discussed is sexuality. Delayed or irregular menstruation is not uncommon. There may be delayed development of secondary sex characteristics such as breasts in girls. The person may use the illness to avoid certain events or relationships. The healthy person may hesitate to make friends with someone who is sick. Other crises and life transitions that must be dealt with in the young adult include building confidence and self-respect on the basis of achievements, persevering with employment goals, developing motivation to achieve, learning to cope with the treatment program, and adjusting to the need for dependence if health fails. Disclosing the CF diagnosis to friends, potential spouses, or employers may pose challenges emotionally and financially.

The issue of marrying and having children is difficult. Genetic counseling may be an appropriate suggestion for the couple considering having children. Another concern is the shortened life span of the parent with CF, and the parent's ability to care for the child must be taken into consideration.

Acute intervention for the patient with CF includes relief of bronchoconstriction, airway obstruction, and airflow limitation. Interventions include aggressive CPT, antibiotics, and O_2 therapy in severe disease. Good nutrition is important. Advances in long-term vascular access (e.g., implanted ports) and inhaled antibiotics have made administration of medication much easier. This has also eased the transition for treatment at home.

Home management of CF includes an aggressive plan of airway clearance that may include postural drainage with percussion and vibration, Acapella, Flutter, high-frequency chest wall oscillation (vest), aerosol-nebulization therapy, and breathing retraining. (These were all discussed earlier in the chapter.) Teach the patient huff coughing (see Table 29-23), pursed-lip breathing (see Table 29-14), and progressive exercise conditioning such as a bicycling program.

The family and the person with CF have a great financial and emotional burden. The cost of drugs, special equipment, and health care is often a financial hardship. Because most CF patients live to childbearing age, family planning and genetic counseling are important. The burden of living with a chronic disease at a young age can be emotionally overwhelming. Community resources are often available to help the family. In addition, the Cystic Fibrosis Foundation can be of assistance. As the person continues toward and into adulthood, you and other skilled health professionals should be available to help the patient and family cope with complications resulting from the disease.

BRONCHIECTASIS

Etiology and Pathophysiology

Bronchiectasis is characterized by permanent, abnormal dilation of medium-sized bronchi in either a localized or diffuse pattern. The pathophysiologic change is a result of inflammatory changes that destroy elastic and muscular structures supporting the bronchial wall resulting in dilated bronchi. Stasis of thickened mucus occurs along with impaired clearance by the cilia. This results in a reduced ability to clear mucus from the lungs and decreased expiratory airflow. Thus bronchiectasis is

classified as an obstructive lung disease. In addition, with the chronic inflammation of the bronchi, the blood vessels of the bronchial wall increase and with continued disease progression, hemoptysis can occur.[57,58]

A variety of pathophysiologic processes can result in bronchiectasis. The main cause of diffuse bronchiectasis is bacterial infections of the lungs that are either not treated or receive delayed treatment. Other causes of bronchiectasis are localized endobronchial obstruction or extrinsic compression of the bronchi (tumor), generalized impairment of pulmonary defenses (CF, immunoglobulin deficiencies, disorders of the cilia), systemic effects of inflammatory diseases (ulcerative colitis), or noninfectious causes (heavy metal poisoning).

Infection is the primary reason for the continuing cycle of inflammation, airway damage, and remodeling. Bronchiectasis can follow a severe pneumonia, with a wide variety of infectious agents initiating bronchiectasis, including adenovirus, influenza virus, *S. aureus, Klebsiella*, and anaerobes. Infections cause the bronchial walls to weaken, and pockets of infection begin to form (Fig. 29-18). When the walls of the bronchial system are injured, the mucociliary mechanism is damaged, allowing bacteria and mucus to accumulate within the pockets. The infection becomes worse and results in bronchiectasis.[57,58]

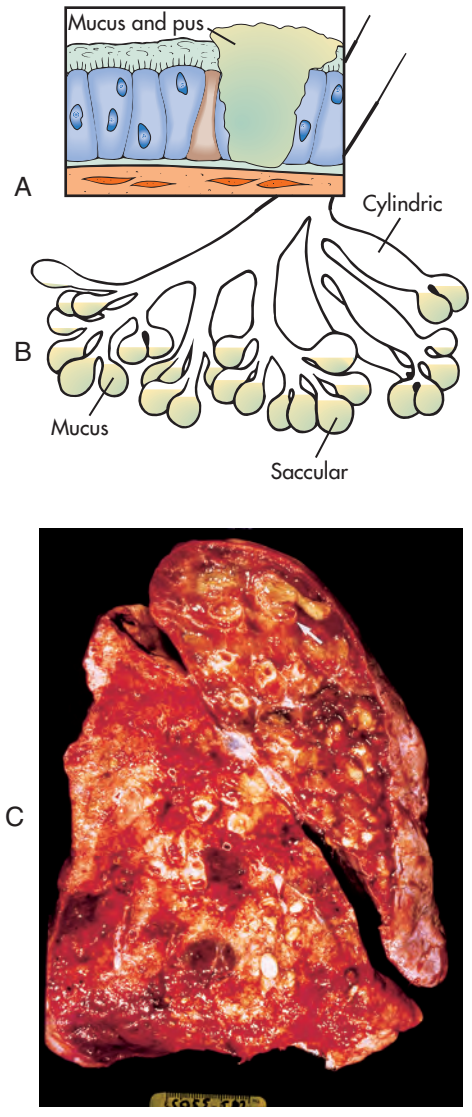


FIG. 29-18 Pathologic changes in bronchiectasis. **A,** Longitudinal section of bronchial wall where chronic infection has caused damage. **B,** Collection of purulent material in dilated bronchioles, leading to persistent infection. **C,** Bronchiectasis in a patient with cystic fibrosis who underwent lung transplantation. Cut surfaces of lung show markedly distended peripheral bronchi filled with mucopurulent secretions.

Clinical Manifestations

The hallmark of bronchiectasis is persistent or recurrent cough with production of purulent sputum. However, some patients with severe disease and upper lobe involvement may have no sputum production and little cough. Hemoptysis occurs in 50% to 70% of the patients and may be massive, necessitating emergency care.[57] The other manifestations of bronchiectasis are dyspnea, fatigue, weight loss, myalgias, and fever. On auscultation of the lungs, a variety of adventitious sounds can be heard (e.g., crackles, wheezes, rhonchi).

Diagnostic Studies

An individual with a chronic productive cough with copious purulent sputum (which may be blood streaked) should be suspected of having bronchiectasis. Chest x-rays may show some nonspecific abnormalities. A high-resolution computed tomography (HRCT) scan of the chest is the gold standard for diagnosing bronchiectasis. Bronchoscopy may be used to diagnose obstruction with patients who have localized bronchiectasis. Sputum may provide additional information regarding the severity of impairment and the presence of active infection. Patients are frequently colonized with *H. influenzae* or *P. aeruginosa*. Pulmonary function studies usually show an obstructive pattern including a decrease in FEV_1 and FEV_1/FVC.[57,58]

Collaborative Care

Bronchiectasis is difficult to treat. Therapy is aimed at treating acute flare-ups and preventing a decline in lung function. Antibiotics are the mainstay of treatment and are often given empirically but attempts are made to culture the sputum. Long-term suppressive therapy with antibiotics is reserved for those patients who have symptoms that recur a few days after stopping antibiotics. Antibiotics may be given orally, given intravenously, or inhaled. Inhaled tobramycin (TOBI) is quite effective in patients with *P. aeruginosa*.

Concurrent bronchodilator therapy with LABAs, SABAs, or anticholinergics is given to prevent bronchospasm and stimulate mucociliary clearance. In addition, ICSs may be used. Mucolytic agents to thin secretions are controversial. Recombinant DNase may be deleterious and should not be used in bronchiectasis not associated with CF.[57] Maintaining good hydration is important to liquefy secretions. Chest physiotherapy and other airway clearance techniques are important to facilitate expectoration of sputum. Teach the patient to reduce exposure to excessive air pollutants and irritants, avoid cigarette smoking, and obtain pneumococcal and influenza vaccinations.

Surgical resection of parts of the lungs, common in the past, has largely been replaced by more effective supportive and antibiotic therapy. For selected patients who are disabled in spite of maximal therapy, lung transplantation is an option. Massive hemoptysis may require surgical resection or embolization of the bronchial artery.[57,58]

NURSING MANAGEMENT
BRONCHIECTASIS

Early detection and treatment of lower respiratory tract infections helps prevent complications such as bronchiectasis. Any obstructing lesion or foreign body should be removed promptly.

An important nursing goal is to promote drainage and removal of bronchial mucus. Various airway clearance techniques can be effectively used to facilitate secretion removal. Chest physiotherapy with postural drainage is widely used with the bronchiectasis patient. Administration of the prescribed medications is important. The patient needs to understand the importance of taking the prescribed regimen of drugs to obtain maximum effectiveness. Rest is important to prevent overexertion. Bed rest may be indicated during the acute phase of the illness, especially with hemoptysis. If hemoptysis occurs, patients should know when they should contact the health care provider. Some patients may periodically expectorate a "spot" of blood that is usual for them. The health care provider will give explicit instructions regarding hemoptysis when emergency contact is needed. In the acute care setting, if the patient has hemoptysis, you should contact the provider immediately, elevate the head of the bed, and place the patient in a side-lying position with the suspected bleeding side down.[57]

Good nutrition is important and may be difficult to maintain because the patient is often anorexic. Oral hygiene to cleanse the mouth and remove dried sputum crusts may improve the patient's appetite. Offering foods that are appealing may also increase the desire to eat. Adequate hydration to help liquefy secretions and thus make it easier to remove them is extremely important. Unless there are contraindications, such as renal disease, instruct the patient to drink at least 3 L of fluid daily. To accomplish this, advise the patient to increase fluid consumption from the baseline by increasing intake by one glass per day until the goal is reached. Generally the patient should be counseled to use low-sodium fluids to avoid systemic fluid retention.

Direct hydration of the respiratory system may also prove beneficial in the expectoration of secretions. Usually an aerosol with normal saline solution delivered by a jet-type nebulizer is used. Alternatively, hypertonic saline may be ordered for a more aggressive effect. At home, a steamy shower can prove effective; expensive equipment that requires frequent cleaning is usually unnecessary. Teach the patient and caregiver to recognize significant clinical manifestations to be reported to the health care provider. These manifestations include increased sputum production, bloody sputum, increasing dyspnea, fever, chills, and chest pain.

CLINICAL DECISION-MAKING EXERCISE

CASE STUDY
CHRONIC OBSTRUCTIVE PULMONARY DISEASE

PATIENT PROFILE. H.M. is a 68-year-old white, married, retired traffic police officer. She has been in the hospital for 3 days with a COPD exacerbation and will be discharged tomorrow.

Subjective Data

- Before admission 7 days of exceptional shortness of breath and increased volume of sputum, which turned a greenish color
- Had increased albuterol MDI use at home to five or six times a day for dyspnea
- Had jitters and racing heart
- Had three or four bouts of bronchitis in the past year that she treated at home
- 30-pack-year history of smoking; smokes half a pack per day now to "clear out lungs" in the morning
- Eats a regular diet but "gets full fast"
- Cannot climb one flight of stairs without stopping; walks down the flat driveway 10 yards without difficulty
- Awakens two or three times per night coughing and short of breath

Objective Data

- Weight 129 lb, height 5 ft 8 in, BMI 20 kg/m^2
- BP 136/76 mm Hg, pulse 86, respiratory rate 28
- Increased anterior-posterior diameter of chest (barrel shaped)
- Slight use of accessory (neck) muscles with breathing
- Distant breath sounds with occasional rhonchi
- No peripheral edema

Diagnostic Studies

- Last PFT: decreased FEV_1 (48%) and FEV_1/FVC (62%)
- ABGs on admission: pH 7.34, $PaCO_2$ 49 mm Hg, HCO_3^- 27 mEq/L, PaO_2 70 mm Hg
- WBC: 14,000/µL on admission
- Chest x-ray: hyperinflation, flat diaphragm, no sign of pneumonia

Collaborative Care

- Stage 3 (severe) COPD with acute exacerbation
- Oxygen 2 L via nasal catheter while in hospital
- Prednisone 30 mg daily oral for 3 days, 20 mg for 3 days, 10 mg for 10 days
- Azithromycin 250 mg orally: take two tablets day one, then take one tablet days 2 through 4
- Proventil HFA MDI 2 puffs 4 times a day
- At discharge: Advair Diskus 250/50 one inhalation every 12 hours

DISCUSSION QUESTIONS

1. What classic manifestations indicate the patient had a COPD exacerbation?
2. What are some likely causes of her COPD?
3. What symptoms indicate the overuse of inhalers, and which drug would cause the symptoms described?
4. What is the only way H.M. can halt the progression of her lung disease?
5. Why would H.M. "feel full fast" when eating? What could you do to minimize this issue?
6. Interpret the ABGs. What pattern do you see?
7. ***Priority Decision:*** What are nursing priorities for discharge planning and teaching?
8. ***Priority Decision:*** Based on the assessment data presented, what are the priority nursing diagnoses? Are there any collaborative problems?

Answers and a corresponding concept map are available at *http://evdve.elsevier.com/Lewis/medsurg.*

BRIDGE TO NCLEX® EXAMINATION

The number of the question corresponds to the same-numbered outcome at the beginning of the chapter.

1. A patient is concerned that he may have asthma. Of the symptoms that he relates to the nurse, which ones suggest asthma or risk factors for asthma *(select all that apply)?*
   a. Allergic rhinitis
   b. Prolonged inhalation
   c. History of skin allergies
   d. Cough, especially at night
   e. Gastric reflux or heartburn
2. In evaluating an asthmatic patient's knowledge of self-care, the nurse recognizes that additional instruction is needed when the patient says,
   a. "I use my corticosteroid inhaler when I feel short of breath."
   b. "I get a flu shot every year and see my health care provider if I have an upper respiratory infection."
   c. "I use my inhaler before I visit my aunt who has a cat, but I only visit for a few minutes because of my allergies."
   d. "I walk 30 minutes every day but sometimes I have to use my bronchodilator inhaler before walking to prevent me from getting short of breath."
3. A plan of care for the patient with COPD could include *(select all that apply)*
   a. exercise such as walking.
   b. high flow rate of O_2 administration.
   c. low-dose chronic oral corticosteroid therapy.
   d. use of peak flow meter to monitor the progression of COPD.
   e. breathing exercises such as pursed-lip breathing that focus on exhalation.
4. The effects of cigarette smoking on the respiratory system include
   a. hypertrophy of capillaries causing hemoptysis.
   b. hyperplasia of goblet cells and increased production of mucus.
   c. proliferation of alveolar macrophages thus increasing chance of infection.
   d. increased proliferation of cilia and decreased clearance of mucus.
5. The major advantage of a Venturi mask is that it can
   a. deliver up to 80% O_2.
   b. provide continuous 100% humidity.
   c. deliver a precise concentration of O_2.
   d. be used while a patient eats and sleeps.
6. Which of the following guidelines would be a part of teaching patients how to use a metered-dose inhaler (MDI)?
   a. After activating the MDI, breathe in as quickly as you can.
   b. Estimate the amount of remaining medicine in the MDI by floating the canister in water.
   c. Disassemble the plastic canister from the inhaler and rinse both pieces under running water every week.
   d. To determine how long the canister will last, divide the total number of puffs in the canister by the puffs needed per day.
7. Which of the following treatments in CF would the nurse expect to implement as collaborative management of patients with CF *(select all that apply)?*
   a. Sperm banking
   b. IV corticosteroids on a chronic basis
   c. Airway clearance techniques (e.g., Acapella)
   d. GoLYTELY given PRN for severe constipation
   e. Inhaled tobramycin to combat *Pseudomonas* infection
8. A patient who has bronchiectasis asks the nurse, "What conditions would warrant a call to the clinic?"
   a. Blood clots in the sputum
   b. Sticky sputum on a hot day
   c. Increased shortness of breath after eating a large meal
   d. Production of large amounts of sputum on a daily basis

1. a, c, d, e, 2. a, 3. a, e, 4. b, 5. c, 6. d, 7. a, c, d, e, 8. a

For rationales to these questions and even more NCLEX review questions, visit *http://evolve.elsevier.com/Lewis/medsurg.*

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RESOURCES

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