



Management of Patients With Chronic Pulmonary Disease

LEARNING OBJECTIVES

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

- 1 Describe the pathophysiology of chronic obstructive pulmonary disease (COPD).
- 2 Discuss the major risk factors for developing COPD and nursing interventions to minimize or prevent these risk factors.
- 3 Describe nursing management of patients with COPD.
- 4 Develop a teaching plan for patients with COPD.
- 5 Describe the pathophysiology of bronchiectasis and relate it to signs and symptoms of bronchiectasis.
- 6 Identify medical and nursing management of bronchiectasis.
- 7 Describe the pathophysiology of asthma.
- 8 Discuss the medications used in asthma management.
- 9 Describe asthma self-management strategies.
- 10 Describe the pathophysiology of cystic fibrosis.

GLOSSARY

air trapping: incomplete emptying of alveoli during expiration due to loss of lung tissue elasticity (emphysema), bronchospasm (asthma), or airway obstruction

alpha₁-antitrypsin deficiency: genetic disorder resulting from deficiency of alpha₁ antitrypsin, a protective agent for the lung; increases patient's risk for developing panacinar emphysema even in the absence of smoking

asthma: a disease with multiple precipitating mechanisms resulting in a common clinical outcome of reversible airflow obstruction; no longer considered a category of COPD

bronchiectasis: chronic dilation of a bronchus or bronchi; the dilated airways become saccular and are a medium for chronic infection; no longer considered a category of COPD

bronchitis: a disease of the airways defined as the presence of cough and sputum production for at least a combined total of 3 months in each of 2 consecutive years; a category of COPD

chronic obstructive pulmonary disease: disease state characterized by airflow limitation that is not fully reversible; sometimes referred to as chronic airway obstruction or chronic obstructive lung disease

emphysema: a disease of the airways characterized by destruction of the walls of overdistended alveoli; a category of COPD

metered-dose inhaler: patient-activated medication canister that provides aerosolized medication that the patient inhales into the lungs

polycythemia: increase in the red blood cell concentration in the blood; in COPD, the body attempts to improve oxygen carrying capacity by producing increasing amounts of red blood cells

spirometry: pulmonary function tests that measure specific lung volumes (eg, FEV₁, FVC) and rates (FEF_{25-75%}); may be measured before and after bronchodilator administration

Chronic pulmonary disorders are a leading cause of morbidity and mortality in the United States. Nurses care for patients with chronic pulmonary disease across the spectrum of care, from outpatient and home care to emergency department, critical care, and hospice settings. To care for these patients, nurses need not only have astute assessment and clinical management skills but also knowledge of how these disorders can affect quality of life. In addition, the nurse's knowledge of palliative and end-of-life care is important for affected patients. Patient and family teaching is an important nursing intervention to enhance self-management in patients with any chronic pulmonary disorder.

Chronic Obstructive Pulmonary Disease

The Global Initiative for Chronic Obstructive Lung Disease (GOLD) has defined **chronic obstructive pulmonary disease** (COPD) as “a preventable and treatable disease with some significant extrapulmonary effects that may contribute to the severity in individual patients. Its pulmonary component is characterized by airflow limitation that is not fully reversible. The airflow limitation is usually progressive and associated with an abnormal inflammatory response of the lung to noxious particles or gases” (GOLD, 2008, p. 2). This updated definition is a broad description that explains COPD and its signs and symptoms. Although previous definitions have categorized emphysema and chronic bronchitis as types of COPD, this was often confusing because most patients with COPD present with overlapping signs and symptoms of these two distinct disease processes.

COPD may include diseases that cause airflow obstruction (eg, emphysema, chronic bronchitis) or any combination of these disorders. Other diseases such as cystic fibrosis, bronchiectasis, and asthma that were previously classified as types of COPD are now classified as chronic pulmonary disorders. Asthma is now considered a distinct, separate disorder and is classified as an abnormal airway condition characterized primarily by reversible inflammation. COPD can coexist with asthma. Both of these diseases have the same major symptoms; however, symptoms are generally more variable in asthma than in COPD. This chapter discusses COPD as a disease and describes chronic bronchitis and emphysema as distinct disease states, providing a foundation for understanding the pathophysiology of COPD. Bronchiectasis, asthma, and cystic fibrosis are discussed separately.

While mortality from other major causes of death has been decreasing, deaths from COPD have continued to rise. Currently, COPD and associated conditions (chronic lower respiratory diseases) are the fourth leading cause of death in the United States and account for the death of almost 125,000 Americans per year (National Heart, Lung, and Blood Institute [NHLBI], 2007). Mortality from COPD among women has dramatically increased since World War II, and in 2005, more women than men died of COPD. Approximately 12 million Americans live with a diagnosis of COPD; however, many patients do not receive optimal treatment. An additional 12 million Americans may have COPD but remain undiagnosed. The annual cost of COPD

(annual expenditures for health and low productivity) is approximately \$42.6 billion with overall health care expenditures (hospital care, physician services, medications, and home health and nursing home care) of \$26.7 billion (NHLBI, 2007).

People with COPD commonly become symptomatic during the middle adult years, and the incidence of the disease increases with age. Although certain aspects of lung function normally decrease with age—for example, vital capacity and forced expiratory volume in 1 second (FEV₁), COPD accentuates and accelerates these physiologic changes.

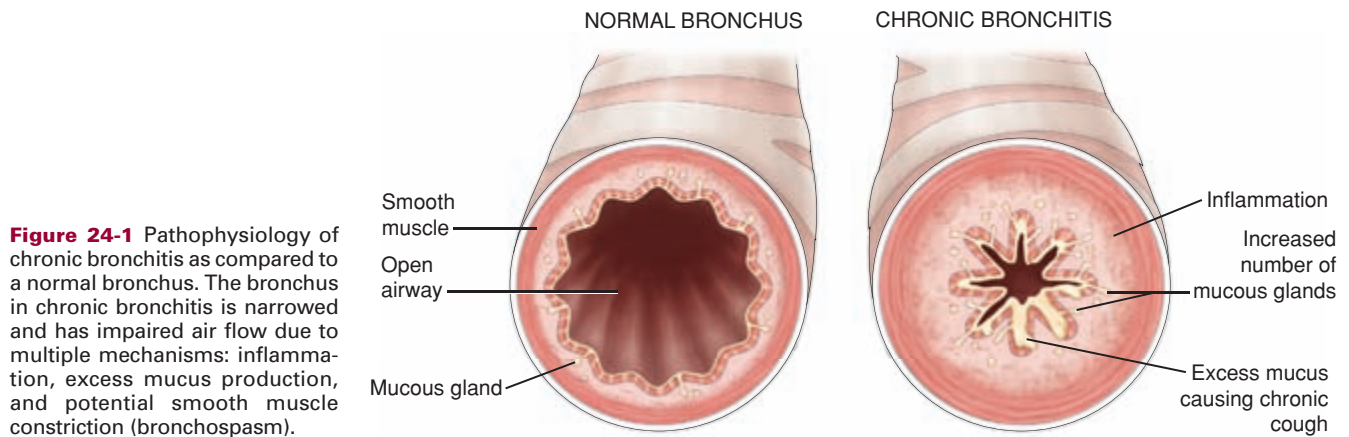
Pathophysiology

In COPD, the airflow limitation is both progressive and associated with an abnormal inflammatory response of the lungs to noxious particles or gases. The inflammatory response occurs throughout the proximal and peripheral airways, lung parenchyma, and pulmonary vasculature (GOLD, 2008). Because of the chronic inflammation and the body's attempts to repair it, changes and narrowing occur in the airways. In the proximal airways (trachea and bronchi greater than 2 mm in diameter), changes include increased numbers of goblet cells and enlarged submucosal glands, both of which lead to hypersecretion of mucus. In the peripheral airways (bronchioles less than 2 mm diameter), inflammation causes thickening of the airway wall, peribronchial fibrosis, exudate in the airway, and overall airway narrowing (obstructive bronchiolitis). Over time, this ongoing injury-and-repair process causes scar tissue formation and narrowing of the airway lumen (GOLD, 2008). Inflammatory and structural changes also occur in the lung parenchyma (respiratory bronchioles and alveoli). Alveolar wall destruction leads to loss of alveolar attachments and a decrease in elastic recoil. Finally, the chronic inflammatory process affects the pulmonary vasculature and causes thickening of the lining of the vessel and hypertrophy of smooth muscle, which may lead to pulmonary hypertension (GOLD, 2008).

Processes related to imbalances of substances (proteinases and antiproteinases) in the lung may also contribute to airflow limitation. When activated by chronic inflammation, proteinases and other substances may be released, damaging the parenchyma of the lung. These parenchymal changes may also occur as a consequence of inflammation or environmental or genetic factors (eg, alpha₁-antitrypsin deficiency).

Chronic Bronchitis

Chronic **bronchitis**, a disease of the airways, is defined as the presence of cough and sputum production for at least 3 months in each of 2 consecutive years. Although, chronic bronchitis is a clinically and epidemiologically useful term, it does not reflect the major impact of airflow limitation on morbidity and mortality in COPD (GOLD, 2008). In many cases, smoke or other environmental pollutants irritate the airways, resulting in inflammation and hypersecretion of mucus. Constant irritation causes the mucus-secreting glands and goblet cells to increase in number, leading to increased mucus production. Mucus plugging of the airway reduces ciliary function. Bronchial walls also become



thickened, further narrowing the bronchial lumen (Fig. 24-1). Alveoli adjacent to the bronchioles may become damaged and fibrosed, resulting in altered function of the alveolar macrophages. This is significant because the macrophages play an important role in destroying foreign particles, including bacteria. As a result, the patient becomes more susceptible to respiratory infection. A wide range of viral, bacterial, and mycoplasmal infections can produce acute episodes of bronchitis. Exacerbations of chronic bronchitis are most likely to occur during the winter when viral and bacterial infections are more prevalent.

Emphysema

In **emphysema**, impaired oxygen and carbon dioxide exchange results from destruction of the walls of overdistended alveoli. *Emphysema* is a pathologic term that describes an abnormal distention of the airspaces beyond the terminal bronchioles and destruction of the walls of the alveoli (GOLD, 2008). This is the end stage of a process that progresses slowly for many years. As the walls of the alveoli are destroyed (a process accelerated by recurrent infections), the alveolar surface area in direct contact with the pulmonary capillaries continually decreases. This causes an increase in dead space (lung area where no gas exchange can occur) and impaired oxygen diffusion, which leads to hypoxemia. In the later stages of disease, carbon dioxide elimination is impaired, resulting in increased carbon dioxide tension in arterial blood (hypercapnia) leading to respiratory acidosis. As the alveolar walls continue to break down, the pulmonary capillary bed is reduced in size. Consequently, resistance to pulmonary blood flow is increased, forcing the right ventricle to maintain a higher blood pressure in the pulmonary artery. Hypoxemia may further increase pulmonary artery pressures. For this reason, right-sided heart failure (*cor pulmonale*) is one of the complications of emphysema. Congestion, dependent edema, distended neck veins, or pain in the region of the liver suggests the development of cardiac failure.

There are two main types of emphysema, based on the changes taking place in the lung (Fig. 24-2). Both types may occur in the same patient. In the panlobular (panacinar) type of emphysema, there is destruction of the respiratory bronchiole, alveolar duct, and alveolus. All airspaces within the lobule are essentially enlarged, but there

is little inflammatory disease. A hyperinflated (hyperexpanded) chest, marked dyspnea on exertion, and weight loss typically occur. To move air into and out of the lungs, negative pressure is required during inspiration, and an adequate level of positive pressure must be attained and maintained during expiration. Instead of being an involuntary passive act, expiration becomes active and requires muscular effort.

In the centrilobular (centroacinar) form, pathologic changes take place mainly in the center of the secondary lobule, preserving the peripheral portions of the acinus. Frequently, there is a derangement of ventilation-perfusion ratios, producing chronic hypoxemia, hypercapnia, **polycythemia**, and episodes of right-sided heart failure. This leads to central cyanosis and respiratory failure. The patient also develops peripheral edema, which is treated with diuretic therapy.

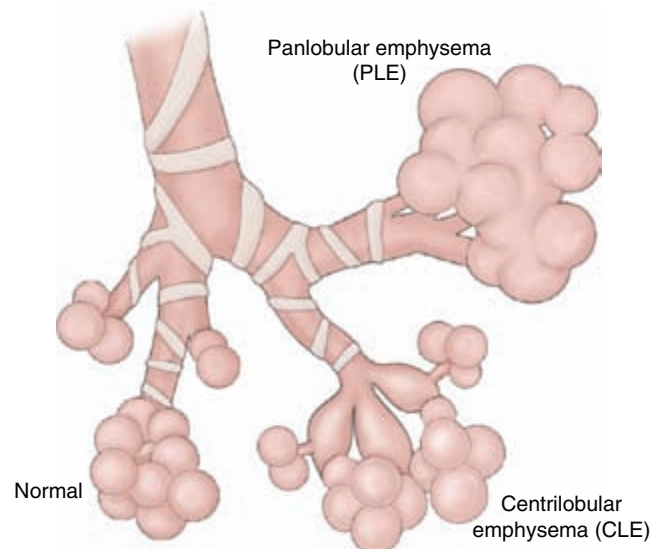


Figure 24-2 Changes in alveolar structure in centrilobular and panlobular emphysema. In panlobular emphysema, the bronchioles, alveolar ducts, and alveoli are destroyed, and the airspaces within the lobule are enlarged. In centrilobular emphysema, the pathologic changes occur in the lobule, whereas the peripheral portions of the acinus are preserved.

CHART
24-1

Risk Factors for Chronic Obstructive Pulmonary Disease (COPD)

- Exposure to tobacco smoke accounts for an estimated 80% to 90% of COPD cases
- Passive smoking
- Occupational exposure—dust, chemicals
- Ambient air pollution
- Genetic abnormalities, including a deficiency of alpha₁-antitrypsin, an enzyme inhibitor that normally counteracts the destruction of lung tissue by certain other enzymes

Risk Factors

Risk factors for COPD include environmental exposures and host factors (Chart 24-1). The most important environmental risk factor for COPD is cigarette smoking. Other environmental risk factors include smoking pipes, cigars, and other types of tobacco. Passive smoking (ie, second-hand smoke) also contributes to respiratory symptoms and COPD (GOLD, 2008). Smoking depresses the activity of scavenger cells and affects the respiratory tract's ciliary cleansing mechanism, which keeps breathing passages free of inhaled irritants, bacteria, and other foreign matter. When smoking damages this cleansing mechanism, airflow is obstructed and air becomes trapped behind the obstruction. The alveoli greatly distend, diminishing lung capacity. Smoking also irritates the goblet cells and mucous glands, causing an increased accumulation of mucus, which in turn produces more irritation, infection, and damage to the lung. In addition, carbon monoxide (a byproduct of smoking) combines with hemoglobin to form carboxyhemoglobin. Hemoglobin that is bound by carboxyhemoglobin cannot carry oxygen efficiently.

Other environmental risk factors for COPD include prolonged and intense exposure to occupational dusts and chemicals, indoor air pollution, and outdoor air pollution (GOLD, 2008). In the United States, it has been estimated that COPD in 19% of smokers and in as many as 31% of nonsmokers may be attributable to such exposure (GOLD, 2008).

One of six Americans with COPD has never smoked (NHLBI, 2007), and COPD involves a gene–environment interaction (GOLD, 2008). The well-documented genetic risk factor is a deficiency of alpha₁-antitrypsin, an enzyme inhibitor that protects the lung parenchyma from injury. This deficiency of alpha₁-antitrypsin predisposes young people to rapid development of lobular emphysema, even if they do not smoke. **α₁-antitrypsin deficiency** is one of the most common genetically linked lethal diseases among Caucasians. There are approximately 25 million carriers of this genetic defect in the United States, and the disease affects approximately 100,000 Americans (American Lung Association, 2007a). Genetically susceptible people are sensitive to environmental factors (eg, smoking, air pollution, infectious agents, allergens) and eventually develop chronic obstructive symptoms. Carriers must be identified so that they can modify environmental risk factors to delay or prevent overt symptoms of disease. Genetics counseling should be offered. Alpha-protease inhibitor replacement therapy, which slows the progression of the disease, is available for patients with this genetic defect and for those with severe disease. However, this intermittent infusion therapy is costly and is required on an ongoing basis.

Clinical Manifestations

Although the natural history of COPD is variable, it is generally a progressive disease characterized by three primary symptoms: chronic cough, sputum production, and dyspnea

on exertion (GOLD, 2008). These symptoms often worsen over time. Chronic cough and sputum production often precede the development of airflow limitation by many years. However, not all people with cough and sputum production develop COPD. The cough may be intermittent and may be unproductive in some patients (GOLD, 2008). Dyspnea may be severe and often interferes with the patient's activities. It is usually progressive, is worse with exercise, and is persistent. As COPD progresses, dyspnea may occur at rest. Weight loss is common, because dyspnea interferes with eating and the work of breathing is energy depleting. As the work of breathing increases over time, the accessory muscles are recruited in an effort to breathe. Patients with COPD are at risk for respiratory insufficiency and respiratory infections, which in turn increase the risk of acute and chronic respiratory failure.

In patients with COPD that has a primary emphysematous component, chronic hyperinflation leads to the “barrel chest” thorax configuration. This configuration results from a more fixed position of the ribs in the inspiratory position (due to hyperinflation) and from loss of lung elasticity (Fig. 24-3).

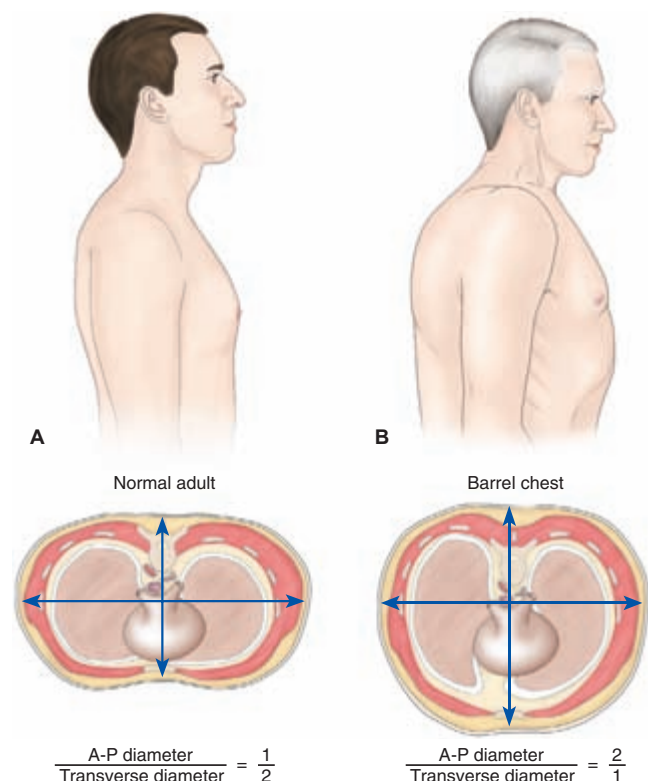


Figure 24-3 Characteristics of normal chest wall and chest wall in emphysema. **A**, The normal chest wall and its cross-section. **B**, The barrel-shaped chest of emphysema and its cross-section



Figure 24-4 Typical posture of a person with chronic obstructive pulmonary disease (COPD)—primarily emphysema. The person tends to lean forward and uses the accessory muscles of respiration to breathe, forcing the shoulder girdle upward and causing the supraclavicular fossae to retract on inspiration.

Retraction of the supraclavicular fossae occurs on inspiration, causing the shoulders to heave upward (Fig. 24-4). In advanced emphysema, the abdominal muscles may also contract on inspiration.

Assessment and Diagnostic Findings

The nurse should obtain a thorough health history from patients with known or potential COPD. Chart 24-2 lists the key factors to assess. Pulmonary function studies are used to help confirm the diagnosis of COPD, determine disease severity, and monitor disease progression. **Spirometry** is used to evaluate airflow obstruction, which is determined by the ratio of FEV₁ to forced vital capacity (FVC). Spirometric results are expressed as an absolute volume and as a percentage of the predicted value using appropriate normal values for gender, age, and height. With obstruction, the patient either has difficulty exhaling or cannot forcibly exhale air from the lungs, reducing the FEV₁. Spirometry is also used to determine reversibility of obstruction after use of bronchodilators (GOLD, 2008). Spirometry is initially performed, the patient is given an inhaled bronchodilator treatment according to a standard protocol, and then spirometry is repeated. The patient demonstrates a degree of reversibility if the pulmonary function values improve after administration of the bronchodilator. Even patients who do not show a significant response to a short-acting bronchodilator test may benefit symptomatically from long-term bronchodilator treatment.

Arterial blood gas measurements may also be obtained to assess baseline oxygenation and gas exchange and are especially important in advanced COPD. A chest x-ray may be obtained to exclude alternative diagnoses. A computed tomography (CT) chest scan is not routinely obtained in the diagnosis of COPD, but a high-resolution CT scan may help in the differential diagnosis. Lastly, screening for alpha₁-antitrypsin deficiency may be performed for patients younger than 45 years of age and for those with a strong family history of COPD.

COPD is classified into four stages depending upon the severity (measured by pulmonary function tests) and symptoms (GOLD, 2008). Stage I (mild) is defined by an FEV₁/FVC less than 70% and an FEV₁ greater than or equal to 80% predicted, and the patient may be with or without symptoms of cough and sputum production. Stage II (moderate) is defined by an FEV₁/FVC less than 70%, an FEV₁ 50% to 80% predicted, and shortness of breath typically developing upon exertion. Stage III (severe) is defined as an FEV₁/FVC less than 70% and an FEV₁ less than 30% to 50% predicted. Severe COPD symptoms include increased shortness of breath, reduced exercise capacity, and repeated exacerbations. Lastly, stage IV (very severe) is defined as an FEV₁/FVC less than 70%, an FEV₁ less than 30% to 50% predicted, and symptoms/signs of chronic respiratory failure.

Factors that determine the clinical course and survival of patients with COPD include history of cigarette smoking, passive smoking exposure, age, rate of decline of FEV₁, hypoxemia, pulmonary artery pressure, resting heart rate, weight loss, and reversibility of airflow obstruction. See Chart 24-3 for additional information about the assessment of symptoms in COPD.

In diagnosing COPD, several differential diagnoses must be ruled out. The primary differential diagnosis is asthma. It may be difficult to differentiate between a patient with COPD and one with chronic asthma. Other diseases that must be considered in the differential diagnosis include heart failure, bronchiectasis, tuberculosis, obliterative bronchiolitis, and diffuse panbronchiolitis (GOLD, 2008). Key factors in determining the diagnosis are the patient's history and the patient's responsiveness to bronchodilators.

Complications

Respiratory insufficiency and failure are major life-threatening complications of COPD. The acuity of the onset and the severity of respiratory failure depend on baseline pulmonary function, pulse oximetry or arterial blood gas values, comorbid conditions, and the severity of other complications of COPD. Respiratory insufficiency and failure may be chronic (with severe COPD) or acute (with severe bronchospasm or pneumonia in a patient with severe COPD). Acute respiratory insufficiency and failure may necessitate ventilatory support until other acute complications, such as infection, can be treated. Management of the patient requiring ventilatory support is discussed in Chapter 25. Other complications of COPD include pneumonia, chronic atelectasis, pneumothorax, and pulmonary arterial hypertension (cor pulmonale).

CHART
24.2

Assessing Patients With Chronic Obstructive Pulmonary Disease (COPD)

Health History

- Has the patient been exposed to risk factors (types, intensity, duration)?
- Does the patient have a past medical history of respiratory diseases/problems, including asthma, allergy, sinusitis, nasal polyps, or respiratory infections?
- Does the patient have a family history of COPD or other chronic respiratory diseases?
- How long has the patient had respiratory difficulty?
- What is the pattern of symptom development?
- Does exertion increase the dyspnea? What type of exertion?
- What are the limits of the patient's tolerance for exercise?
- At what times during the day does the patient complain most of fatigue and shortness of breath?
- Which eating and sleeping habits have been affected?
- What is the impact of respiratory disease on quality of life?
- What does the patient know about the disease and his or her condition?
- What is the patient's smoking history (primary and secondary)?
- Is there occupational exposure to smoke or other pollutants?
- What are the triggering events (eg, exertion, strong odors, dust, exposure to animals)?
- Does the patient have a history of exacerbations or previous hospitalizations for respiratory problems?
- Are comorbidities present?
- How appropriate are current medical treatments?

- Does the patient have available social and family support?
- What is the potential for reducing risk factors (eg, smoking cessation)?

Physical Assessment

- What position does the patient assume during the interview?
- What are the pulse and the respiratory rates?
- What is the character of respirations? Even and without effort? Other?
- Can the patient complete a sentence without having to take a breath?
- Does the patient contract the abdominal muscles during inspiration?
- Does the patient use accessory muscles of the shoulders and neck when breathing?
- Does the patient take a long time to exhale (prolonged expiration)?
- Is central cyanosis evident?
- Are the patient's neck veins engorged?
- Does the patient have peripheral edema?
- Is the patient coughing?
- What are the color, amount, and consistency of the sputum?
- Is clubbing of the fingers present?
- What types of breath sounds (ie, clear, diminished or distant, crackles, wheezes) are heard? Describe and document findings and locations.
- What is the status of the patient's sensorium?
- Is there short-term or long-term memory impairment?
- Is there increasing stupor?
- Is the patient apprehensive?

CHART
24.3

NURSING RESEARCH PROFILE

Symptom Assessment of Patients With Chronic Obstructive Pulmonary Disease

Jablonski, A., Gift, A. & Cook, K. E. (2007). Symptom assessment of patients with chronic obstructive pulmonary disease. *Western Journal of Nursing Research*, 29(7), 845–863.

Purpose

The purpose of this secondary analysis of data was to evaluate the Memorial Symptom Assessment Scale (MSAS) for use in patients with severe chronic obstructive pulmonary disease (COPD). Although the multidimensional MSAS was developed for patients with cancer, it has potential for assessment of symptoms of people with other diseases.

Design

This descriptive study was a secondary analysis of data that examined the relationship between symptoms and functional status in patients with COPD. The investigators recruited the convenience sample of 72 subjects who met the criteria for severe COPD from an outpatient pulmonary clinic. Ages ranged from 36 to 79 years. The MSAS has 32 items but for the purposes of the study, the researchers shortened it to the 19 items, or symptoms, that patients with COPD most frequently identify. The included symptoms related to prevalence, frequency, severity, and distress.

Findings

The top 10 symptoms identified by this COPD population were shortness of breath, lack of energy, dry mouth, cough, feeling nervous, feeling sad, feeling irritable, worrying, feeling drowsy, and difficulty sleeping. Shortness of breath and lack of energy were the most severe, frequent, and distressful symptoms. When researchers evaluated the prevalence and characteristics of symptoms, they found that both clinical and emotional symptoms (feeling nervous, sad, or irritable; worrying) were equally represented. Reliability of the revised MSAS tool, with 19 items, remained high (Cronbach's alpha = 0.86), and this study also confirmed content and convergent validity.

Nursing Implications

Although many tools are available to measure the frequency of symptoms, few tools are available to evaluate symptoms on a multidimensional perspective; the MSAS enables assessment of symptom prevalence, severity, frequency, and distress. Nursing implications of this study include not only a potential new tool to evaluate patients with severe COPD but also the need to recognize that emotional symptoms are as important as clinical symptoms in people with this disease. Nursing interventions need to focus not only on patients' clinical symptoms but also on their emotional needs.

Medical Management

Risk Reduction

Smoking cessation is the single most cost-effective intervention to reduce the risk of developing COPD and to stop its progression (GOLD, 2008). However, smoking cessation is difficult to achieve and even more difficult to sustain in the long term. Nurses are key in promoting smoking cessation and educating patients about its importance. Patients diagnosed with COPD who continue to smoke must be encouraged and assisted to quit. Factors associated with continued smoking vary among patients and may include the strength of the nicotine addiction, continued exposure to smoking-associated stimuli (at work or in social settings), stress, depression, and habit. Continued smoking is also more prevalent among those with low incomes, low levels of education, or psychosocial problems (CDC, 2007b).

Because multiple factors are associated with continued smoking, successful cessation often requires multiple strategies. Health care providers should promote cessation by explaining the risks of smoking and personalizing the “at-risk” message to the patient. After giving a strong warning about smoking, health care providers should work with the patient to set a definite “quit date.” Referral to a smoking cessation program may be helpful. Follow-up within 3 to 5 days after the “quit date” to review progress and to address any problems is associated with an increased rate of success; this should be repeated as needed. Continued reinforcement with telephone calls or clinic visits is extremely beneficial. Relapses should be analyzed, and the patient and health care provider should jointly identify possible solutions to prevent future backsliding. It is important to emphasize successes rather than failures. Nicotine replacement, first-line pharmacotherapy that reliably increases long-term smoking abstinence rates, comes in a variety of forms (gum, inhaler, nasal spray, transdermal patch, sublingual tablet, or lozenges). Bupropion SR (Wellbutrin, Zyban) and nortriptyline (Aventyl), both antidepressants, may also increase long-term quit rates. Other pharmacologic agents include the anti-hypertensive agent clonidine (Catapres); however, its side effects limit its use. Varenicline (Chantix), a nicotinic acetylcholine receptor partial agonist, may assist in smoking cessation (GOLD, 2008). Patients who are not appropriate candidates for the use of pharmacotherapy include those with medical contraindications, light smokers (less than 10 cigarettes per day), and pregnant and adolescent smokers.

Smoking cessation can begin in a variety of health care settings—outpatient clinic, nursing center, pulmonary rehabilitation, community, hospital, and in the home. Regardless of the setting, nurses have the opportunity to teach patients about the risks of smoking and the benefits of smoking cessation. Various materials, resources, and programs developed by several organizations (eg, Agency for Healthcare Research and Quality, U.S. Public Health Service, CDC, National Cancer Institute, American Lung Association, American Cancer Society) are available to assist with this effort.

Pharmacologic Therapy

Bronchodilators

Bronchodilators relieve bronchospasm by altering smooth muscle tone and reduce airway obstruction by allowing increased oxygen distribution throughout the lungs and

improving alveolar ventilation. Although regular use of bronchodilators that act primarily on the airway smooth muscle does not modify the decline of function or the prognosis of COPD, their use is central in the management of COPD (GOLD, 2008). These agents can be delivered through a metered-dose inhaler or other type of inhaler, by nebulization, or via the oral route in pill or liquid form. Bronchodilators are often administered regularly throughout the day as well as on an as-needed basis. They may also be used prophylactically to prevent breathlessness by having the patient use them before participating in or completing an activity, such as eating or walking.

Several devices are available to deliver medication via an aerosolized method. These include **metered-dose inhalers** (MDIs), breath-actuated MDIs, dry powder inhalers, spacer or valved-holding chambers, and nebulizers. Key aspects of each are described in Table 24-1. An MDI is a pressurized device that contains an aerosolized powder of medication. A precise amount of medication is released with each activation of the canister. Patients must be instructed on the correct use of the device. A spacer or valved-holding chamber may also be used to enhance deposition of the medication in the lung and help the patient coordinate activation of the MDI with inspiration. Spacers come in several designs, but all are attached to the MDI and have a mouthpiece on the opposite end (Fig. 24-5). Specific package insert information is available for different types of aerosol delivery devices.

Several classes of bronchodilators are used, including beta-adrenergic agonists (short- and long-acting), anticholinergic agents (short- and long-acting), methylxanthines, and combination agents. These medications may be used in combination to optimize bronchodilation. Long-acting bronchodilators are more convenient for patient use. Examples of these medications are described in Table 24-2. Nebulized medications also known as wet nebulizers (nebulization of medication via an air compressor) may also be effective in patients who cannot use an MDI properly or who prefer this method of administration. However, wet nebulizers are more expensive than other devices and require appropriate maintenance (GOLD, 2008).

Bronchodilators are key to symptom management in stable COPD. Before these agents are used, the following information should be considered: Inhaled therapy is preferred, choice of bronchodilator is dependent on availability and individual response in terms of symptom relief and side effects, they may be prescribed on an as-needed or regular basis to reduce symptoms, long-acting bronchodilators are more convenient for patient use, and combining bronchodilators with different durations of action and different mechanisms may optimize symptom management (GOLD, 2008).

Corticosteroids

Although inhaled and systemic corticosteroids may improve the symptoms of COPD, they do not slow the decline in lung function. Their effects are less dramatic than in asthma. A short trial course of oral corticosteroids may be prescribed for patients to determine whether pulmonary function improves and symptoms decrease. Long-term

Table 24-1 AEROSOL DELIVERY DEVICES

| Devices/Drugs | Optimal Technique | Therapeutic Issues |
|---|--|--|
| Metered-dose inhaler (MDI) Beta ₂ -agonists Corticosteroids Cromolyn sodium Anticholinergics | Actuation during a slow (30 L/min or 3–5 sec) deep inhalation, followed by 10-sec breathhold | Slow inhalation and coordination of actuation may be difficult for some patients. Patients may incorrectly stop inhalation at actuation. Deposition of 50–80% of actuated dose in the oropharynx. Mouth washing and spitting is effective in reducing the amount of drug swallowed and absorbed systemically. |
| Breath-actuated MDI Beta ₂ -agonists | Tight seal around mouthpiece and slightly more rapid inhalation than standard MDI (see above) followed by 10-sec breathhold | May be particularly useful for patients unable to coordinate inhalation and actuation. May also be useful for elderly patients. Patients may incorrectly stop inhalation at actuation. Cannot be used with currently available spacer/valved-holding chamber devices. |
| Dry powder inhaler (DPI) Beta ₂ -agonists Corticosteroids Anticholinergics | Rapid (1–2 sec) deep inhalation. Minimally effective inspiratory flow is device dependent. | Dose is lost if patient exhales through device after actuating. Delivery may be greater or lesser than MDIs, depending on device and technique. Delivery is more flow dependent in devices with highest internal resistance. Rapid inhalation promotes greater deposition in larger central airways. Mouth washing and spitting are effective in reducing amount of drug swallowed and absorbed systemically. |
| Spacer or valved-holding chamber (VHC) | Slow (30 L/min or 3–5 sec) deep inhalation, followed by 10-sec breathhold immediately following actuation. Actuate only once into spacer/VHC per inhalation. Rinse plastic VHCs once a month with low concentration of liquid household dishwashing detergent (1:5000 or 1–2 drops per cup of water) and let drip dry. | Indicated for patients who have difficult performing adequate MDI technique. May be bulky. Simple tubes do not obviate coordinating actuation and inhalation. VHCs are preferred. Spacers or VHCs may increase delivery of inhalational corticosteroids to the lungs. |
| Nebulizer Beta ₂ -agonists Corticosteroids Cromolyn sodium Anticholinergics | Slow tidal breathing with occasional deep breaths. Tightly fitting face mask for those unable to use mouthpiece. | Less dependent on patient's coordination and cooperation. May be expensive, time consuming, and bulky; output is dependent on device and operating parameters (fill volume, driving gas flow); internebulizer and intranebulizer output variances are significant. Use of a face mask reduces delivery to lungs by 50%. Choice of delivery system is dependent on resources, availability, and clinical judgment of clinician caring for patient. There is potential for infections if device is not cleaned properly. |

From Expert Panel Report 3. (2007). *Guidelines for the diagnosis and management of asthma* (pp. 31–32). NIH Publication Number 08-5846. National Asthma Education and Prevention Program. Summary Report. Bethesda, MD: U.S. Department of Health and Human Services, National Heart, Lung and Blood Institute.

treatment with oral corticosteroids is not recommended in COPD and can cause steroid myopathy, leading to muscle weakness, decreased ability to function, and, in advanced disease, respiratory failure (GOLD, 2008).

Medication regimens used to manage COPD are based on disease severity. For stage I (mild) COPD, a short-acting bronchodilator may be prescribed. For stage II or III COPD, a short-acting bronchodilator along with regular treatment of one or more long-acting bronchodilators may be used. For stage III or IV (severe or very severe) COPD, medication therapy includes regular treatment with one or more bronchodilators and inhaled corticosteroids for repeated exacerbations. Combination long-term beta₂-agonists plus corticosteroids in one inhaler may be appropriate; examples

include formoterol/budesonide (Symbicort) and salmeterol/fluticasone (Seretide).

Other Medications

Other pharmacologic treatments that may be used in COPD include alpha₁-antitrypsin augmentation therapy, antibiotic agents, mucolytic agents, antitussive agents, vasodilators, and narcotics. Vaccines may also be effective. Influenza vaccines can reduce serious morbidity and death in patients with COPD by approximately 50% (GOLD, 2008). It is recommended that people limit their risk through influenza vaccination and smoking cessation. Pneumococcal vaccination also reduces the incidence of pneumonia, hospitalizations for cardiac conditions, and deaths in the

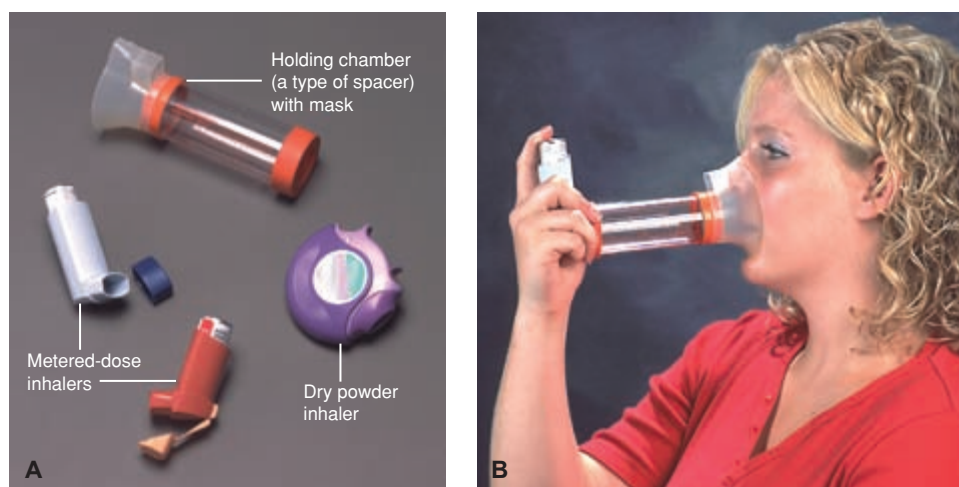


Figure 24-5 A, Examples of metered-dose inhalers and spacers. B, A metered-dose inhaler and spacer in use.

general elderly population. Pneumococcal pneumonia is responsible for approximately 175,000 hospitalized cases per year (National Pneumonia Medicare Quality Improvement Project, 2007). Vaccination is recommended in patients 65 years or older who have COPD (GOLD, 2008).

Management of Exacerbations

An exacerbation of COPD is defined as an event in the natural course of the disease characterized by an acute change in the patient's baseline dyspnea, cough, or sputum production beyond the normal day-to-day variations. It may warrant a change in regular medications (GOLD, 2008). Primary causes of an acute exacerbation include tracheobronchial infection and air pollution. However, the cause of approximately one third of severe exacerbations cannot be identified (GOLD, 2008). If possible, the primary cause of the exacerbation is identified, and specific treatment is adminis-

tered. Optimization of bronchodilator medications is first-line therapy and involves identifying the best medication or combinations of medications taken on a regular schedule for a specific patient. Depending on the signs and symptoms, corticosteroids, antibiotic agents, oxygen therapy, and intensive respiratory interventions may also be used. Indications for hospitalization for acute exacerbation of COPD include severe dyspnea that does not respond adequately to initial therapy, confusion or lethargy, respiratory muscle fatigue, paradoxical chest wall movement, peripheral edema, worsening or new onset of central cyanosis, persistent or worsening hypoxemia, and need for noninvasive or invasive assisted mechanical ventilation (GOLD, 2008). The outcome from an exacerbation of COPD is closely related to the development of respiratory acidosis, the presence of significant comorbidities, and the need for noninvasive or invasive positive pressure ventilatory support.

Table 24-2 COMMON TYPES OF BRONCHODILATOR MEDICATIONS FOR COPD

| Class/Drug (Trade Name) | Method of Administration | | | Duration of Action [†] |
|--|--------------------------|-----------|------|---------------------------------|
| | Inhaler* | Nebulizer | Oral | |
| Beta₂-Adrenergic Agonist Agents | | | | |
| salbutamol, albuterol (Proventil, Ventolin) | X | X | X | Short |
| fenoterol (Alupent, Isuprel) | X | X | X | Short |
| terbutaline (Brethine) | X | | | Short |
| formoterol (Foradil) | X | | | Long |
| salmeterol (Serevent Diskus) | X | | | Long |
| Anticholinergic Agents | | | | |
| Ipratropium bromide (Atrovent) | X | X | | Short |
| Combination Short-Acting Beta-2 Adrenergic Agonist and Anticholinergic Agents | | | | |
| fenoterol/ipratropium (Duovent) | X | X | | |
| salbutamol/ipratropium (Combivent) | X | X | | |
| Methylxanthines | | | | |
| aminophylline (Phyllocontin, Truphylline) | | | X | Variable |
| theophylline (Theo-Dur, Slo-Bid) | | | X | Variable |

*Inhaler may include metered-dose inhaler, powdered inhalation with inhaler, or discus.

[†]Short-acting, 4–6 h; long-acting, 12+ h.

The GOLD guidelines (2008) provide indications for assessment, hospital admission, and intensive care admission for patients with exacerbations of COPD. These include signs of increasing severity on physical examination (use of accessory muscles, paradoxical chest wall movement, worsening or new onset of central cyanosis, peripheral edema, signs of right heart failure, reduced alertness).

On the patient's arrival at the emergency department, the first line of treatment is supplemental oxygen therapy and rapid assessment to determine if the exacerbation is life-threatening (GOLD, 2008). A short-acting inhaled bronchodilator may be used to assess response to treatment. Oral or intravenous corticosteroids, in addition to bronchodilators, are recommended in the hospital management of a COPD exacerbation. Antibiotics have been shown to be of some benefit in patients with increased dyspnea, increased sputum volume, and increased sputum purulence, and in those needing mechanical ventilation (GOLD, 2008).

Oxygen Therapy

Oxygen therapy can be administered as long-term continuous therapy, during exercise, or to prevent acute dyspnea during an exacerbation. The goal of supplemental oxygen therapy is to increase the baseline resting partial arterial pressure of oxygen (PaO_2) to at least 60 mm Hg at sea level and an arterial oxygen saturation (SaO_2) at least 90% (GOLD, 2008). Long-term oxygen therapy (more than 15 hours per day) has also been shown to improve quality of life, reduce pulmonary arterial pressure and dyspnea, and improve survival (GOLD, 2008). Long-term oxygen therapy is usually introduced in very severe COPD, and indications generally include a PaO_2 of 55 mm Hg or less or evidence of tissue hypoxia and organ damage such as cor pulmonale, secondary polycythemia, edema from right-sided heart failure, or impaired mental status (GOLD, 2008). For patients with exercise-induced hypoxemia, oxygen supplementation during exercise may improve performance. There is no evidence to support the idea that short bursts of oxygen before or after exercise provide any symptomatic relief (GOLD, 2008). Patients who are hypoxemic while awake are likely to be so during sleep. Therefore, nighttime oxygen therapy is recommended as well, and the prescription for oxygen therapy is for continuous, 24-hour use. Intermittent oxygen therapy is indicated for patients who desaturate only during exercise or sleep.

The main objective in treating patients with hypoxemia and hypercapnia is to give sufficient oxygen to improve oxygenation. Patients with COPD who require oxygen may have respiratory failure that is caused primarily by a ventilation–perfusion mismatch. These patients respond to oxygen therapy and should be treated to keep the resting oxygen saturation above 90%. However, a small subset of patients with COPD and chronic hypercapnia (elevated partial pressure of arterial carbon dioxide [PaCO_2] levels) may be more oxygen sensitive; their respiratory failure is caused more by alveolar hypoventilation. Administering too much oxygen can result in the retention of carbon dioxide. Patients with alveolar hypoventilation cannot increase ventilation to adjust for this increased load, and increasing

hypercapnia occurs. Monitoring and assessment are key in the care of patients with COPD on supplemental oxygen. Pulse oximetry is helpful in assessing response to therapy but does not assess PaCO_2 levels. Optimal oxygenation of patients is important while monitoring for any possible complications of oxygen supplementation.

NURSING ALERT

Oxygen therapy is variable in COPD patients; its aim in COPD is to achieve an acceptable oxygen level without a fall in the pH (increasing hypercapnia).

Surgical Management

Bullectomy

A bullectomy is a surgical option for select patients with bullous emphysema. Bullae are enlarged airspaces that do not contribute to ventilation but occupy space in the thorax; these areas may be surgically excised. These bullae compress areas of the lung and may impair gas exchange. Bullectomy may help reduce dyspnea and improve lung function. It can be performed via a video-assisted thoracoscope or a limited thoracotomy incision (see Chapter 25).

Lung Volume Reduction Surgery

Treatment options for patients with end-stage COPD (stage IV) with a primary emphysematous component are limited, although lung volume reduction surgery is a palliative surgical option in a selected subset of patients. This subset includes patients with homogenous disease or disease that is focused in one area and not widespread throughout the lungs. Lung volume reduction surgery involves the removal of a portion of the diseased lung parenchyma. This reduces hyperinflation and allows the functional tissue to expand, resulting in improved elastic recoil of the lung and improved chest wall and diaphragmatic mechanics. This type of surgery does not cure the disease or improve life expectancy, but it may decrease dyspnea, improve lung function, and improve the patient's overall quality of life (GOLD, 2008).

Careful selection of patients for lung volume reduction surgery is essential to decrease morbidity and mortality. A large multisite trial, the National Emphysema Treatment Trial, found that the addition of lung volume reduction surgery to optimal medical management and rehabilitation led to overall improvement in exercise tolerance and survival in a very select subgroup of patients with predominantly upper lobe disease (American Lung Association, 2007b; GOLD, 2008).

Lung Transplantation

Lung transplantation is a viable option for definitive surgical treatment of end-stage emphysema. It has been shown to improve quality of life and functional capacity in a selected group of patients with COPD. Limited not only by the shortage of donor organs, it is also a costly procedure with financial implications for months to years because of complications and the need for costly immunosuppressive medication regimens (GOLD, 2008).

Pulmonary Rehabilitation

Pulmonary rehabilitation for patients with COPD is well established and widely accepted as a means to alleviate symptoms and optimize functional status (Ries, Bauldoff, Carlin, et al., 2007). The primary goals of rehabilitation are to reduce symptoms, improve quality of life, and increase physical and emotional participation in everyday activities (GOLD, 2008). The benefits of this therapy include improvement of exercise capacity, reduction of the perceived intensity of breathlessness, improvement in health-related quality of life, reduction in the number of hospitalizations and days in the hospital, and reduction of the anxiety and depression associated with COPD (GOLD, 2008). Pulmonary rehabilitation services are multidisciplinary and include assessment, education, smoking cessation, physical reconditioning, nutritional counseling, skills training, and psychological support. Patients are taught methods to alleviate symptoms. Breathing exercises as well as retraining and exercise programs are used to improve functional status.

Pulmonary rehabilitation is appropriate for stages II through IV (GOLD, 2008). The minimum length of an effective program is 6 weeks; the longer the program, the more effective the results (GOLD, 2008; Ries, et al., 2007). It may be conducted in inpatient, outpatient, or home settings, and the lengths of programs vary. Program selection depends on the patient's physical, functional, and psychosocial status; insurance coverage; availability of programs; and preference. Pulmonary rehabilitation may also be used therapeutically in other disorders besides COPD, including asthma, cystic fibrosis, lung cancer, interstitial lung disease, thoracic surgery, and lung transplantation.

Patient Education

Nurses play a key role in identifying potential candidates for pulmonary rehabilitation and in facilitating and reinforcing the material learned in the rehabilitation program. Not all patients have access to a formal rehabilitation program. However, nurses can be instrumental in teaching patients and families as well as facilitating specific services, such as respiratory therapy education, physical therapy for exercise and breathing retraining, occupational therapy for conserving energy during activities of daily living, and nutritional counseling. Patient education is a major component of pulmonary rehabilitation and includes a broad variety of topics.

Depending on the length and setting of the educational program, topics may include normal anatomy and physiology of the lung, pathophysiology and changes with COPD, medications and home oxygen therapy, nutrition, respiratory therapy treatments, symptom alleviation, smoking cessation, sexuality and COPD, coping with chronic disease, communicating with the health care team, and planning for the future (advance directives, living wills, informed decision making about health care alternatives). Education, including that relating to smoking cessation, should be incorporated into all aspects of care for COPD and in many settings (physicians' offices, clinics, hospitals, home and community health care settings, and comprehensive rehabilitation programs).

Breathing Exercises

The breathing pattern of most people with COPD is shallow, rapid, and inefficient; the more severe the disease, the more inefficient the breathing pattern. With practice, this type of upper chest breathing can be changed to diaphragmatic breathing, which reduces the respiratory rate, increases alveolar ventilation, and sometimes helps expel as much air as possible during expiration (see Chapter 25 for technique). Pursed-lip breathing helps slow expiration, prevents collapse of small airways, and helps the patient control the rate and depth of respiration. It also promotes relaxation, enabling the patient to gain control of dyspnea and reduce feelings of panic.

Activity Pacing

People with COPD have decreased exercise tolerance during specific periods of the day, especially in the morning on arising, because bronchial secretions have collected in the lungs during the night while the person was lying down. The patient may have difficulty bathing or dressing and may become fatigued. Activities that require the arms to be supported above the level of the thorax may produce fatigue or respiratory distress but may be tolerated better after the patient has been up and moving around for an hour or more. The nurse can help the patient reduce these limitations by planning self-care activities and determining the best times for bathing, dressing, and other daily activities.

Self-Care Activities

As gas exchange, airway clearance, and the breathing pattern improve, the patient is encouraged to assume increasing participation in self-care activities. The patient is taught to coordinate diaphragmatic breathing with activities such as walking, bathing, bending, or climbing stairs. The patient should bathe, dress, and take short walks, resting as needed to avoid fatigue and excessive dyspnea. Fluids should always be readily available, and the patient should begin to drink fluids without having to be reminded. If management of secretions is a problem and some type of postural drainage or airway clearance maneuver is to be performed at home, the nurse or respiratory therapist instructs and supervises the patient before discharge or in an outpatient setting.

Physical Conditioning

People with COPD of all stages benefit from exercise training programs, which result in increased exercise tolerance and decreased dyspnea and fatigue (GOLD, 2008). Physical conditioning techniques include breathing exercises and general exercises intended to conserve energy and increase pulmonary ventilation. Graded exercises and physical conditioning programs using treadmills, stationary bicycles, and measured level walks can improve symptoms and increase work capacity and exercise tolerance. Any physical activity that can be performed regularly is helpful. Walking aids may be beneficial (GOLD, 2008). Lightweight portable oxygen systems are available for ambulatory patients who require oxygen therapy during physical activity.

Oxygen Therapy

Oxygen supplied to the home comes in compressed gas, liquid, or concentrator systems. Portable oxygen systems allow the patient to exercise, work, and travel. To help the patient

adhere to the oxygen prescription, the nurse explains the proper flow rate and required number of hours for oxygen use as well as the dangers of arbitrary changes in flow rate or duration of therapy. The nurse should caution the patient that smoking with or near oxygen is extremely dangerous. The nurse also reassures the patient that oxygen is not “addictive” and explains the need for regular evaluations of blood oxygenation by pulse oximetry or arterial blood gas analysis.

Nutritional Therapy

Nutritional assessment and counseling are important aspects in the rehabilitation process for patients with COPD. Nutritional status is important in COPD and is reflected in severity of symptoms, degree of disability, and prognosis. Significant weight loss is often a major problem, but excessive weight can also be problematic although it occurs less often. Most people have difficulty gaining and maintaining weight. A thorough assessment of caloric needs and counseling about meal planning and supplementation is part of the rehabilitation process. Continual monitoring of weight and interventions as necessary are important parts of the care of patients with COPD.

Coping Measures

Any factor that interferes with normal breathing quite naturally induces anxiety, depression, and changes in behavior. Constant shortness of breath and fatigue may make the patient irritable and apprehensive to the point of panic. Restricted activity (and reversal of family roles due to loss of employment), the frustration of having to work to breathe, and the realization that the disease is prolonged and unrelenting may cause the patient to become angry, depressed, and demanding. Sexual function may be compromised, which also diminishes self-esteem. The nurse should provide education and support to spouses or significant others and families, because the caregiver role in end-stage COPD can be difficult.

Nursing Management

Assessing the Patient

Assessment involves obtaining information about current symptoms as well as previous disease manifestations. See Chart 24-2 for sample questions that may be used to obtain a clear history of the disease process. In addition to the history, nurses review the results of available diagnostic tests.

Achieving Airway Clearance

Bronchospasm, which occurs in many pulmonary diseases, reduces the caliber of the small bronchi and may cause dyspnea, static secretions, and infection. Bronchospasm can sometimes be detected on auscultation with a stethoscope when wheezing or diminished breath sounds are heard. Increased mucus production, along with decreased mucociliary action, contributes to further reduction in the caliber of the bronchi and results in decreased airflow and decreased gas exchange. This is further aggravated by the loss of lung elasticity that occurs with COPD (GOLD, 2008). These changes in the airway require that the nurse monitor the patient for dyspnea and hypoxemia. If bronchodilators or

corticosteroids are prescribed, the nurse must administer the medications properly and be alert for potential side effects. The relief of bronchospasm is confirmed by measuring improvement in expiratory flow rates and volumes (the force of expiration, how long it takes to exhale, and the amount of air exhaled) as well as by assessing the dyspnea and making sure that it has lessened.

Diminishing the quantity and viscosity of sputum can clear the airway and improve pulmonary ventilation and gas exchange. All pulmonary irritants should be eliminated or reduced, particularly cigarette smoking, which is the most persistent source of pulmonary irritation. The nurse instructs the patient in directed or controlled coughing, which is more effective and reduces the fatigue associated with undirected forceful coughing. Directed coughing consists of a slow, maximal inspiration followed by breath-holding for several seconds and then two or three coughs. “Huff” coughing may also be effective. The technique consists of one or two forced exhalations (“huffs”) from low to medium lung volumes with the glottis open.

Chest physiotherapy with postural drainage, intermittent positive pressure breathing, increased fluid intake, and bland aerosol mists (with normal saline solution or water) may be useful for some patients with COPD. The use of these measures must be based on the response and tolerance of each patient.

Improving Breathing Patterns

Ineffective breathing patterns and shortness of breath are due to the ineffective respiratory mechanics of the chest wall and lung resulting from **air trapping**, ineffective diaphragmatic movement, airway obstruction, the metabolic cost of breathing, and stress. Inspiratory muscle training and breathing retraining may help improve breathing patterns. Training in diaphragmatic breathing reduces the respiratory rate, increases alveolar ventilation, and sometimes helps expel as much air as possible during expiration. Pursed-lip breathing helps slow expiration, prevent collapse of small airways, and control the rate and depth of respiration. It also promotes relaxation, which allows patients to gain control of dyspnea and reduce feelings of panic.

Improving Activity Tolerance

Patients with COPD experience progressive activity and exercise intolerance and disability. Education is focused on rehabilitative therapies to promote independence in executing activities of daily living. These may include pacing activities throughout the day or using supportive devices to decrease energy expenditure. The nurse evaluates the patient’s activity tolerance and limitations and uses teaching strategies to promote independent activities of daily living. The patient may be a candidate for exercise training to strengthen the muscles of the upper and lower extremities and to improve exercise tolerance and endurance. Use of walking aids may be recommended to improve activity levels and ambulation (GOLD, 2008). Other health care professionals (rehabilitation therapist, occupational therapist, physical therapist) may be consulted as additional resources.

Monitoring and Managing Potential Complications

The nurse must assess for various complications of COPD, such as life-threatening respiratory insufficiency and failure, as well as respiratory infection and chronic atelectasis, which may increase the risk of respiratory failure. The nurse monitors for cognitive changes (personality and behavioral changes, memory impairment), increasing dyspnea, tachypnea, and tachycardia, which may indicate increasing hypoxemia and impending respiratory failure.

The nurse monitors pulse oximetry values to assess the patient's need for oxygen and administers supplemental oxygen as prescribed. The nurse also instructs the patient about signs and symptoms of respiratory infection that may worsen hypoxemia and reports changes in the patient's physical and cognitive status to the physician.

Bronchopulmonary infections must be controlled to diminish inflammatory edema and to permit recovery of normal ciliary action. Minor respiratory infections that are of no consequence to people with normal lungs can be life-threatening to people with COPD. Infection compromises lung function and is a common cause of respiratory failure in people with COPD. In COPD, infection may be accompanied by subtle changes. The nurse instructs the patient to report any signs of infection, such as a fever or change in sputum color, character, consistency, or amount. Any worsening of symptoms (increased tightness of the chest, increased dyspnea, and fatigue) also suggests infection and must be reported. Viral infections are hazardous to the patient because they are often followed by infections caused by bacterial organisms, such as *Streptococcus pneumoniae* and *Haemophilus influenzae*.

To prevent infection, the nurse should encourage the patient with COPD to be immunized against influenza and *S. pneumoniae*, because the patient is prone to respiratory infection. It is important to caution the patient to avoid going outdoors if the pollen count is high or if there is significant air pollution, because of the risk of bronchospasm. The patient also should avoid exposure to high outdoor temperatures with high humidity.

Pneumothorax is a potential complication of COPD and can be life-threatening in patients with COPD who have minimal pulmonary reserve. Patients with severe emphysematous changes can develop large bullae, which may rupture and cause a pneumothorax. Development of a pneumothorax may be spontaneous or related to an activity such as severe coughing or large intrathoracic pressure changes. If a rapid onset of shortness of breath occurs, the nurse should quickly evaluate the patient for potential pneumothorax by assessing the symmetry of chest movement, differences in breath sounds, and pulse oximetry.

Over time, pulmonary hypertension may occur as a result of chronic hypoxemia. The pulmonary arteries respond to hypoxemia by constricting, which results in pulmonary hypertension. The complication may be prevented by maintaining adequate oxygenation through an adequate hemoglobin level, improved ventilation-perfusion of the lungs, or continuous administration of supplemental oxygen (if needed).

Promoting Home and Community-Based Care

Teaching Patients Self-Care

When providing instructions about self-management, it is important for the nurse to assess the knowledge of patients and family members about self-care and the therapeutic regimen. The nurse should also consider whether they are comfortable with this knowledge. Familiarity with prescribed medications' potential side effects is essential. In addition, patients and family members need to learn the early signs and symptoms of infection and other complications so that they seek appropriate health care promptly.

NURSING ALERT

Teaching is essential and should be tailored to the stage of COPD.

A major area of teaching involves setting and accepting realistic short-term and long-range goals. If the COPD is mild, the objectives of treatment are to increase exercise tolerance and prevent further loss of pulmonary function. If the COPD is severe, these objectives are to preserve current pulmonary function and relieve symptoms as much as possible. It is important to plan and share the goals and expectations of treatment with the patient. Both the patient and the care provider need patience to achieve these goals.

The nurse instructs the patient to avoid extremes of heat and cold. Heat increases the body temperature, thereby raising oxygen requirements, and cold tends to promote bronchospasm. Air pollutants such as fumes, smoke, dust, and even talcum, lint, and aerosol sprays may initiate bronchospasm. High altitudes aggravate hypoxemia.

A patient with COPD should adopt a lifestyle of moderate activity, ideally in a climate with minimal shifts in temperature and humidity. As much as possible, the patient should avoid emotional disturbances and stressful situations that might trigger a coughing episode. The medication regimen can be quite complex; patients receiving aerosol medications by an MDI or other type of inhaler may be particularly challenged. It is crucial to review educational information and to have the patient demonstrate correct MDI use before discharge, during follow-up visits to a caregiver's office or clinic, and during home visits (Chart 24-4).

Smoking cessation goes hand in hand with lifestyle changes, and reinforcement of the patient's efforts is a key nursing activity. Smoking cessation is the single most important therapeutic intervention for patients with COPD. There are many strategies, including prevention, cessation with or without oral or topical patch medications, and behavior modification techniques.

Numerous educational materials are available to assist nurses in teaching patients with COPD. Potential resources include those of organizations such as the American Lung Association, the American Association of Cardiovascular and Pulmonary Rehabilitation, the American Thoracic Society, the American College of Chest Physicians, and the American Association for Respiratory Therapy.

CHART
24.4

PATIENT EDUCATION

Use of Metered-Dose Inhaler (MDI)

To administer medication:

- Remove the cap and hold the inhaler upright.
- Shake the inhaler.
- Tilt your head back slightly and breathe out slowly and all the way.
- Position the inhaler approximately 1–2 inches away from the open mouth, or use a spacer/holding chamber. When using a medicine chamber, place the lips around the mouthpiece.
- Start breathing in slowly through your mouth, and press down on the inhaler one time. If using a chamber, first

press down on the inhaler and within 5 seconds, begin to breathe in slowly.

- Breathe in slowly and deeply for as long as possible.
- Hold your breath as you count to 10 slowly to allow the medication to reach down into your airways.
- Repeat puffs as directed, allowing 15–30 seconds between puffs for quick-acting medications. There is no need to wait for other medications.
- Apply the cap to the MDI for storage.
- After inhalation, rinse mouth with water when using a corticosteroid-containing MDI.

Expert Panel Report 3. (2007). *Guidelines for the diagnosis and management of asthma*. Bethesda, MD: National Asthma Education and Prevention Program, National Institutes of Health.

Continuing Care

Referral for home care is important to enable the nurse to assess the patient's home environment and physical and psychological status, to evaluate the patient's adherence to a prescribed regimen, and to assess the patient's ability to cope with changes in lifestyle and physical status. Home care visits provide an opportunity to reinforce the information and activities learned in the inpatient or outpatient pulmonary rehabilitation program and to have the patient and family demonstrate correct administration of medications and oxygen, if indicated, and performance of exercises. If the patient does not have access to a formal pulmonary rehabilitation program, it is important to provide the education and breathing retraining necessary to optimize the patient's functional status.

The nurse may direct the patient to community resources such as pulmonary rehabilitation programs and smoking cessation programs to help improve the patient's ability to cope with his or her chronic condition and the therapeutic regimen and to give the patient a sense of worth, hope, and well-being. In addition, the nurse reminds the patient and family about the importance of participating in general health promotion activities and health screening.

It is important to address quality of life and issues surrounding the end of life in patients with end-stage COPD. Patients with COPD have indicated that information about their end-of-life needs is limited. Based on a structured literature review, the following end-of-life care issues are important: symptom management, quality of life, satisfaction with care, information/communication, use of care professionals, use of care facilities, hospital admission, and place of death (Habracken, Willems, de Kort, et al., 2007). It is crucial that patients know what to expect as the disease progresses. In addition, they should have information about their role in decisions regarding aggressiveness of care near the end of life and access to specialists who may help them and their families. As the disease course progresses, a holistic assessment of physical and psychological needs should be undertaken at each hospitalization, clinic visit, or home visit. This helps gauge the patient's assessment of the progression of the disease and its impact on quality of life and guides planning for future interventions and management.

Chart 24-5 provides further information on providing nursing care for the patient with COPD.

Bronchiectasis

Bronchiectasis is a chronic, irreversible dilation of the bronchi and bronchioles. Under the new definition of COPD, it is considered a disease process separate from COPD (GOLD, 2008). Bronchiectasis may be caused by a variety of conditions, including

- Airway obstruction
- Diffuse airway injury
- Pulmonary infections and obstruction of the bronchus or complications of long-term pulmonary infections
- Genetic disorders such as cystic fibrosis
- Abnormal host defense (eg, ciliary dyskinesia or humoral immunodeficiency)
- Idiopathic causes

People may be predisposed to bronchiectasis as a result of recurrent respiratory infections in early childhood, measles, influenza, tuberculosis, or immunodeficiency disorders.

Pathophysiology

The inflammatory process associated with pulmonary infections damages the bronchial wall, causing a loss of its supporting structure and resulting in thick sputum that ultimately obstructs the bronchi. The walls become permanently distended and distorted, impairing mucociliary clearance. In saccular bronchiectasis, each dilated peribronchial tube virtually amounts to a lung abscess, the exudate of which drains freely through the bronchus. Bronchiectasis is usually localized, affecting a segment or lobe of a lung, most frequently the lower lobes.

The retention of secretions and subsequent obstruction ultimately cause the alveoli distal to the obstruction to collapse (atelectasis). Inflammatory scarring or fibrosis replaces functioning lung tissue. In time, the patient develops respiratory insufficiency with reduced vital capacity, decreased ventilation, and an increased ratio of residual volume to total lung capacity. There is impairment in the match of ventilation to perfusion (ventilation–perfusion imbalance) and hypoxemia.


**CHART
24-5**
PLAN OF NURSING CARE
Care of the Patient With COPD
NURSING DIAGNOSIS: Impaired gas exchange and airway clearance due to chronic inhalation of toxins

GOAL: Improvement in gas exchange

Nursing Interventions

1. Evaluate current smoking status, educate regarding smoking cessation, and facilitate efforts to quit.
 - a. Evaluate current smoking habits of patient and family.
 - b. Educate regarding hazards of smoking and relationship to COPD.
 - c. Evaluate previous smoking cessation attempts.
 - d. Provide educational materials.
 - e. Refer to a smoking cessation program or resource.
2. Evaluate current exposure to occupational toxins or pollutants and indoor/outdoor pollution.
 - a. Evaluate current exposures to occupational toxins, indoor and outdoor air pollution (eg, smog, toxic fumes, chemicals).
 - b. Emphasize primary prevention to occupational exposures. This is best achieved by elimination or reduction of exposures in the workplace.
 - c. Educate regarding types of indoor and outdoor air pollution (eg, biomass fuel burned for cooking and heating in poorly ventilated buildings, outdoor air pollution).
 - d. Advise patient to monitor public announcements regarding air quality.

Rationale

1. Smoking causes permanent damage to the lung and diminishes the lungs' protective mechanisms. Airflow is obstructed, secretions are increased, and lung capacity is reduced. Continued smoking increases morbidity and mortality in COPD and is also a risk factor for lung cancer.
2. Chronic inhalation of both indoor and outdoor toxins causes damage to the airways and impairs gas exchange.

Expected Outcomes

- Identifies the hazards of cigarette smoking
- Identifies resources for smoking cessation
- Enrolls in smoking cessation program
- Reports success in stopping smoking
- Verbalizes types of inhaled toxins
- Minimizes or eliminates exposures
- Monitors public announcements regarding air quality and minimizes or eliminates exposures during episodes of severe pollution

NURSING DIAGNOSIS: Impaired gas exchange related to ventilation–perfusion inequality

GOAL: Improvement in gas exchange

Nursing Interventions

1. Administer bronchodilators as prescribed:
 - a. Inhalation is the preferred route.
 - b. Observe for side effects: tachycardia, dysrhythmias, central nervous system excitation, nausea, and vomiting.
 - c. Assess for correct technique of metered-dose inhaler (MDI) or other type of administration.
2. Evaluate effectiveness of nebulizer or MDI treatments.
 - a. Assess for decreased shortness of breath, decreased wheezing or crackles, loosened secretions, and decreased anxiety.
 - b. Ensure that treatment is given before meals to avoid nausea and to reduce fatigue that accompanies eating.

Rationale

1. Bronchodilators dilate the airways. The medication dosage is carefully adjusted for each patient, in accordance with clinical response.
2. Combining medication with aerosolized bronchodilators is typically used to control bronchoconstriction in an acute exacerbation. Generally, however, the MDI with spacer is the preferred route (less cost and time to treatment).

Expected Outcomes

- Verbalizes need for bronchodilators and for taking them as prescribed
- Evidences minimal side effects; heart rate near normal, absence of dysrhythmias, normal mentation
- Reports a decrease in dyspnea
- Shows an improved expiratory flow rate
- Uses and cleans respiratory therapy equipment as applicable
- Demonstrates diaphragmatic breathing and coughing
- Uses oxygen equipment appropriately when indicated
- Evidences improved arterial blood gases or pulse oximetry
- Demonstrates correct technique for use of MDI

Continued on following page


 CHART
24-5

PLAN OF NURSING CARE
Care of the Patient With COPD (Continued)
Nursing Interventions

3. Instruct and encourage patient in diaphragmatic breathing and effective coughing.
4. Administer oxygen by the method prescribed.
 - a. Explain rationale and importance to patient.
 - b. Evaluate effectiveness; observe for signs of hypoxemia. Notify physician if restlessness, anxiety, somnolence, cyanosis, or tachycardia is present.
 - c. Analyze arterial blood gases and compare with baseline values. When arterial puncture is performed and a blood sample is obtained, hold puncture site for 5 minutes to prevent arterial bleeding and development of ecchymoses.
 - d. Initiate pulse oximetry to monitor oxygen saturation.
 - e. Explain that no smoking is permitted by patient or visitors while oxygen is in use.

Rationale

3. These techniques improve ventilation by opening airways to facilitate clearing the airways of sputum. Gas exchange is improved and fatigue is minimized.
4. Oxygen will correct the hypoxemia. Careful observation of the liter flow or the percentage administered and its effect on the patient is important. These patients generally require low-flow oxygen rates of 1 to 2 L/min. Monitor and titrate to achieve desired PaO₂. Periodic arterial blood gases and pulse oximetry help evaluate adequacy of oxygenation. Smoking may render pulse oximetry inaccurate because the carbon monoxide from cigarette smoke also saturates hemoglobin.

Expected Outcomes

NURSING DIAGNOSIS: Ineffective airway clearance related to bronchoconstriction, increased mucus production, ineffective cough, bronchopulmonary infection, and other complications

GOAL: Achievement of airway clearance

Nursing Interventions

1. Adequately hydrate the patient.
2. Teach and encourage the use of diaphragmatic breathing and coughing techniques.
3. Assist in administering nebulizer or MDI.
4. If indicated, perform postural drainage with percussion and vibration in the morning and at night as prescribed.
5. Instruct patient to avoid bronchial irritants such as cigarette smoke, aerosols, extremes of temperature, and fumes.
6. Teach early signs of infection that are to be reported to the clinician immediately:
 - a. Increased sputum production
 - b. Change in color of sputum
 - c. Increased thickness of sputum
 - d. Increased shortness of breath, tightness in chest, or fatigue
 - e. Increased coughing
 - f. Fever or chills

Rationale

1. Systemic hydration keeps secretions moist and easier to expectorate. Fluids must be given with caution if right- or left-sided heart failure is present.
2. These techniques help to improve ventilation and mobilize secretions without causing breathlessness and fatigue.
3. This ensures adequate delivery of medication to the airways.
4. This uses gravity to help raise secretions so they can be more easily expectorated or suctioned.
5. Bronchial irritants cause bronchoconstriction and increased mucus production, which then interfere with airway clearance.
6. Minor respiratory infections that are of no consequence to the person with normal lungs can produce fatal disturbances in the lungs of the person with emphysema. Early recognition is crucial.

Expected Outcomes

- Verbalizes need to drink fluids
- Demonstrates diaphragmatic breathing and coughing
- Performs postural drainage correctly
- Coughing is minimized
- Does not smoke
- Verbalizes that pollens, fumes, gases, dusts, and extremes of temperature and humidity are irritants to be avoided
- Identifies signs of early infection
- Is free of infection (no fever, no change in sputum, lessening of dyspnea)
- Verbalizes need to notify health care provider at the earliest sign of infection
- Verbalizes need to stay away from crowds or people with colds in flu season
- Discusses flu and pneumonia vaccines with clinician to help prevent infection

Continued


CHART
24-5
PLAN OF NURSING CARE
Care of the Patient With COPD (Continued)
Nursing Interventions

7. Administer antibiotics as prescribed.
8. Encourage patient to be immunized against influenza and *Streptococcus pneumoniae*.

Rationale

7. Antibiotics may be prescribed to prevent or treat infection.
8. People with respiratory conditions are prone to respiratory infections and are encouraged to be immunized.

Expected Outcomes

NURSING DIAGNOSIS: Ineffective breathing pattern related to shortness of breath, mucus, bronchoconstriction, and airway irritants

GOAL: Improvement in breathing pattern

Nursing Interventions

1. Teach patient diaphragmatic and pursed-lip breathing.
2. Encourage alternating activity with rest periods. Allow patient to make some decisions (bath, shaving) about care based on tolerance level.
3. Encourage use of an inspiratory muscle trainer if prescribed.

Rationale

1. This helps patient prolong expiration time and decreases air trapping. With these techniques, patient will breathe more efficiently and effectively.
2. Pacing activities permit patient to perform activities without excessive distress.
3. This strengthens and conditions the respiratory muscles.

Expected Outcomes

- Practices pursed-lip and diaphragmatic breathing and uses them when short of breath and with activity
- Shows signs of decreased respiratory effort and paces activities
- Uses inspiratory muscle trainer as prescribed

NURSING DIAGNOSIS: Self-care deficits related to fatigue secondary to increased work of breathing and insufficient ventilation and oxygenation

GOAL: Independence in self-care activities

Nursing Interventions

1. Teach patient to coordinate diaphragmatic breathing with activity (eg, walking, bending).
2. Encourage patient to begin to bathe self, dress self, walk, and drink fluids. Discuss energy conservation measures.
3. Teach postural drainage if appropriate.

Rationale

1. This will allow the patient to be more active and to avoid excessive fatigue or dyspnea during activity.
2. As condition resolves, patient will be able to do more but needs to be encouraged to avoid increasing dependence.
3. This encourages patient to become involved in own care and prepares patient to manage at home.

Expected Outcomes

- Uses controlled breathing while bathing, bending, and walking
- Paces activities of daily living to alternate with rest periods to reduce fatigue and dyspnea
- Describes energy conservation strategies
- Performs same self-care activities as before
- Performs postural drainage correctly

NURSING DIAGNOSIS: Activity intolerance due to fatigue, hypoxemia, and ineffective breathing patterns

GOAL: Improvement in activity tolerance

Nursing Interventions

1. Support patient in establishing a regular regimen of exercise using treadmill and exercise bicycle, walking, or other appropriate exercises, such as mall walking.
 - a. Assess the patient's current level of functioning and develop exercise plan based on baseline functional status.
 - b. Suggest consultation with a physical therapist or pulmonary rehabilitation program to determine an exercise program specific to the patient's capability. Have portable oxygen unit available if oxygen is prescribed for exercise.

Rationale

1. Muscles that are deconditioned consume more oxygen and place an additional burden on the lungs. Through regular, graded exercise, these muscle groups become more conditioned, and the patient can do more without getting as short of breath. Graded exercise breaks the cycle of debilitation.

Expected Outcomes

- Performs activities with less shortness of breath
- Verbalizes need to exercise daily and demonstrates an exercise plan to be carried out at home
- Walks and gradually increases walking time and distance to improve physical condition
- Exercises both upper and lower body muscle groups

Continued on following page


CHART
24-5
PLAN OF NURSING CARE
Care of the Patient With COPD (Continued)

NURSING DIAGNOSIS: Ineffective coping related to reduced socialization, anxiety, depression, lower activity level, and the inability to work

GOAL: Attainment of an optimal level of coping

Nursing Interventions

1. Help the patient develop realistic goals.
2. Encourage activity to level of symptom tolerance.
3. Teach relaxation technique or provide a relaxation tape for patient.
4. Enroll patient in pulmonary rehabilitation program where available.

Rationale

1. Developing realistic goals will promote a sense of hope and accomplishment rather than defeat and hopelessness.
2. Activity reduces tension and decreases degree of dyspnea as patient becomes conditioned.
3. Relaxation reduces stress, anxiety, and dyspnea and helps patient to cope with disability.
4. Pulmonary rehabilitation programs have been shown to promote a subjective improvement in a patient's status and self-esteem as well as increased exercise tolerance and decreased hospitalizations.

Expected Outcomes

- Expresses interest in the future
- Participates in the discharge plan
- Discusses activities or methods that can be performed to ease shortness of breath
- Uses relaxation techniques appropriately
- Expresses interest in a pulmonary rehabilitation program

NURSING DIAGNOSIS: Deficient knowledge about self-management to be performed at home

GOAL: Adherence to therapeutic program and home care

Nursing Interventions

1. Help patient identify/develop short- and long-term goals.
 - a. Teach the patient about disease, medications, procedures, and how and when to seek help.
 - b. Refer patient to pulmonary rehabilitation.
2. Give strong message to stop smoking. Discuss smoking cessation strategies. Provide information about resource groups (eg, SmokEnders, American Cancer Society, American Lung Association).

Rationale

1. Patient needs to be a partner in developing the plan of care and needs to know what to expect. Teaching about the condition is one of the most important aspects of care; it will prepare the patient to live and cope with the condition and improve quality of life.
2. Smoking causes permanent damage to the lung and diminishes the lungs' protective mechanisms. Air flow is obstructed and lung capacity is reduced. Smoking increases morbidity and mortality and is also a risk factor for lung cancer.

Expected Outcomes

- Understands disease and what affects it
- Verbalizes the need to preserve existing lung function by adhering to the prescribed program
- Understands purposes and proper administration of medications
- Stops smoking or enrolls in a smoking cessation program
- Identifies when and whom to call for assistance

COLLABORATIVE PROBLEM: Atelectasis

GOAL: Absence of atelectasis on x-ray and physical examination

Nursing Interventions

1. Monitor respiratory status, including rate and pattern of respirations, breath sounds, signs and symptoms of respiratory distress, and pulse oximetry.
2. Instruct in and encourage diaphragmatic breathing and effective coughing techniques.
3. Promote use of lung expansion techniques (eg, deep-breathing exercises, incentive spirometry) as prescribed.

Rationale

1. A change in respiratory status, including tachypnea, dyspnea, and diminished or absent breath sounds, may indicate atelectasis.
2. These techniques improve ventilation and lung expansion and ideally improve gas exchange.
3. Deep-breathing exercises and incentive spirometry promote maximal lung expansion.

Expected Outcomes

- Normal (baseline for patient) respiratory rate and pattern
- Normal breath sounds for patient
- Demonstrates diaphragmatic breathing and effective coughing
- Performs deep-breathing exercises, incentive spirometry as prescribed
- Pulse oximetry is $\geq 90\%$

Continued

CHART
24-5

PLAN OF NURSING CARE

Care of the Patient With COPD (Continued)

COLLABORATIVE PROBLEM: Pneumothorax

GOAL: Absence of signs and symptoms of pneumothorax

Nursing Interventions

1. Monitor respiratory status, including rate and pattern of respirations, symmetry of chest wall movement, breath sounds, signs and symptoms of respiratory distress, and pulse oximetry.
2. Assess pulse.
3. Assess for chest pain and precipitating factors.
4. Palpate for tracheal deviation/ shift away from the affected side.
5. Monitor pulse oximetry and, if indicated, arterial blood gases.
6. Administer supplemental oxygen therapy, as indicated.
7. Administer analgesics agents, as indicated, for chest pain.
8. Assist with chest tube insertion and use pleural drainage system, as prescribed.

Rationale

1. Dyspnea, tachypnea, tachycardia, acute pleuritic chest pain, tracheal deviation away from the affected side, absence of breath sounds on the affected side, and decreased tactile fremitus may indicate pneumothorax.
2. Tachycardia is associated with pneumothorax and anxiety.
3. Pain may accompany pneumothorax.
4. Early detection of pneumothorax and prompt intervention will prevent other serious complications.
5. Recognition of a deterioration in respiratory function will prevent serious complications.
6. Oxygen will correct hypoxemia; administer it with caution.
7. Pain interferes with deep breathing, resulting in a decrease in lung expansion.
8. Removal of air from the pleural space will re-expand the lung.

Expected Outcomes

- Normal respiratory rate and pattern for patient
- Normal breath sounds bilaterally
- Normal pulse for patient
- Normal tactile fremitus
- Absence of pain
- Tracheal position is midline
- Pulse oximetry $\geq 90\%$
- Maintains normal oxygen saturation and arterial blood gas measurements
- Exhibits no hypoxemia and hypercapnia (or returns to baseline values)
- Absence of pain
- Symmetric chest wall movement
- Lung is re-expanded on chest x-ray
- Breath sounds are heard on the affected side

COLLABORATIVE PROBLEM: Respiratory failure

GOAL: Absence of signs and symptoms of respiratory failure; no evidence of respiratory failure on laboratory tests

Nursing Interventions

1. Monitor respiratory status, including rate and pattern of respirations, breath sounds, and signs and symptoms of acute respiratory distress.
2. Monitor pulse oximetry and arterial blood gases.
3. Administer supplemental oxygen and initiate mechanisms for mechanical ventilation, as prescribed.

Rationale

1. Early recognition of a deterioration in respiratory function will avert further complications, such as respiratory failure, severe hypoxemia, and hypercapnia.
2. Recognition of changes in oxygenation and acid-base balance will guide in correcting and preventing complications.
3. Acute respiratory failure is a medical emergency. Hypoxemia is a hallmark sign. Administration of oxygen therapy and mechanical ventilation (if indicated) are critical to survival.

Expected Outcomes

- Normal respiratory rate and pattern for patient with no acute distress
- Recognizes symptoms of hypoxemia and hypercapnia
- Maintains normal arterial blood gases/pulse oximetry or returns to baseline values

COLLABORATIVE PROBLEM: Pulmonary arterial hypertension

GOAL: Absence of evidence of pulmonary arterial hypertension on physical examination or laboratory tests

Nursing Interventions

1. Monitor respiratory status, including rate and pattern of respirations, breath sounds, pulse oximetry, and signs and symptoms of acute respiratory distress.
2. Assess for signs and symptoms of right-sided heart failure, including peripheral edema, ascites, distended neck veins, crackles, and heart murmur.
3. Administer oxygen therapy, as prescribed.

Rationale

1. Dyspnea is the primary symptom of pulmonary arterial hypertension. Other symptoms include fatigue, angina, near syncope, edema, and palpitations.
2. Right-sided heart failure is a common clinical manifestation of pulmonary arterial hypertension due to increased right ventricular workload.
3. Continuous oxygen therapy is a major component of management of pulmonary arterial hypertension by preventing hypoxemia and thereby reducing pulmonary vascular constriction (resistance) secondary to hypoxemia.

Expected Outcomes

- Normal respiratory rate and pattern for patient
- Exhibits no signs and symptoms of right-sided failure
- Maintains baseline pulse oximetry values and arterial blood gases

Clinical Manifestations

Characteristic symptoms of bronchiectasis include chronic cough and the production of purulent sputum in copious amounts. Many patients with this disease have hemoptysis. Clubbing of the fingers also is common because of respiratory insufficiency. Patients usually have repeated episodes of pulmonary infection. Even with modern treatment approaches, the average age at death is approximately 55 years.

Assessment and Diagnostic Findings

Bronchiectasis is not readily diagnosed because the symptoms can be mistaken for those of simple chronic bronchitis. A definite sign is a prolonged history of productive cough, with sputum consistently negative for tubercle bacilli. The diagnosis is established by a CT scan, which reveals bronchial dilation.

Medical Management

Treatment objectives are to promote bronchial drainage to clear excessive secretions from the affected portion of the lungs and to prevent or control infection. Postural drainage is part of all treatment plans, because draining of the bronchiectatic areas by gravity reduces the amount of secretions and the degree of infection. Sometimes mucopurulent sputum must be removed by bronchoscopy. Chest physiotherapy, including percussion and postural drainage, is important in the management of secretions. Smoking cessation is important, because smoking impairs bronchial drainage by paralyzing ciliary action, increasing bronchial secretions, and causing inflammation of the mucous membranes, resulting in hyperplasia of the mucous glands.

Antimicrobial therapy based on the results of sensitivity studies on organisms cultured from sputum is used to control infection. A year-round regimen of antibiotic agents may be prescribed, with different types of antibiotics at intervals. Some clinicians prescribe antibiotic agents throughout the winter or when acute upper respiratory tract infections occur. Patients should be vaccinated against influenza and pneumococcal pneumonia. Bronchodilators, which may be prescribed for patients who also have reactive airway disease, may also assist with secretion management.

Surgical intervention, although used infrequently, may be indicated for patients who continue to expectorate large amounts of sputum and have repeated bouts of pneumonia and hemoptysis despite adherence to treatment regimens. The disease must involve only one or two areas of the lung that can be removed without producing respiratory insufficiency. The goals of surgical treatment are to conserve normal pulmonary tissue and to avoid infectious complications. Diseased tissue is removed, provided that postoperative lung function will be adequate. It may be necessary to remove a segment of a lobe (segmental resection), a lobe (lobectomy), or rarely an entire lung (pneumonectomy). (See Chart 25-18 in Chapter 25 for further information.) Segmental resection is the removal of an anatomic subdivision of a pulmonary lobe. The chief advantage is that only diseased tissue is removed, and healthy lung tissue is conserved.

The surgery is preceded by a period of careful preparation. The objective is to obtain a dry (free of infection) tracheobronchial tree to prevent complications (atelectasis,

pneumonia, bronchopleural fistula, and empyema). This is accomplished by postural drainage or, depending on the location, by direct suction through a bronchoscope. A course of antibacterial therapy may be prescribed. After surgery, care is the same as for any patient who has undergone chest surgery (see Chapter 25).

Nursing Management

Nursing management focuses on alleviating symptoms and helping patients clear pulmonary secretions. Patient teaching targets smoking and other factors that increase the production of mucus and hamper its removal. Patients and families are taught to perform postural drainage and to avoid exposure to people with upper respiratory or other infections. If the patient experiences fatigue and dyspnea, he or she is informed about strategies to conserve energy while maintaining as active a lifestyle as possible. The patient is taught about the early signs of respiratory infection and the progression of the disorder, so that appropriate treatment can be implemented promptly. The presence of a large amount of mucus may decrease the patient's appetite and result in an inadequate dietary intake; therefore, the patient's nutritional status is assessed and strategies are implemented to ensure an adequate diet.

Asthma

Asthma is a chronic inflammatory disease of the airways that causes airway hyperresponsiveness, mucosal edema, and mucus production. This inflammation ultimately leads to recurrent episodes of asthma symptoms: cough, chest tightness, wheezing, and dyspnea (Fig. 24-6). In the United

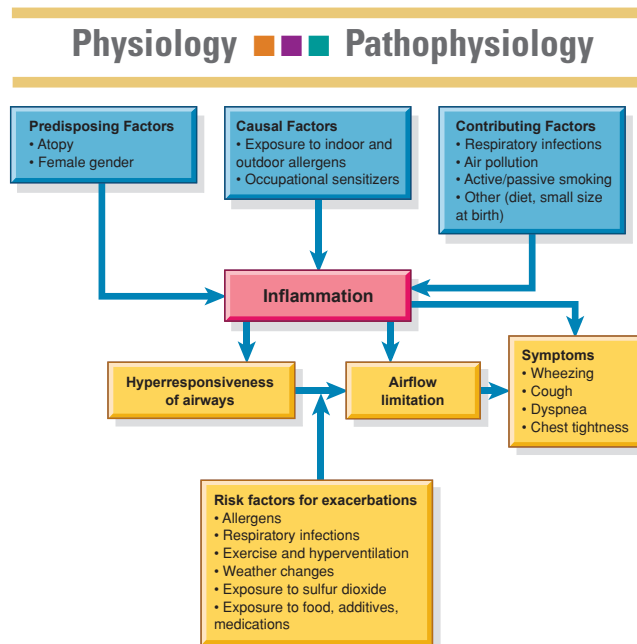


Figure 24-6 Pathophysiology of asthma. Adapted from materials developed for the Global Initiative for Asthma (GINA). *Global Strategy for Asthma Management and Prevention*, Global Initiative for Asthma (GINA) 2008. Available from: <http://www.ginasthma.org>.

States, asthma affects more than 22 million people (Expert Panel Report 3, 2007). Asthma accounts for more than 497,000 hospitalizations annually (Agency for Healthcare Research and Quality [AHRQ, 2007]). The total economic cost of asthma exceeds \$27.6 billion (AHRQ, 2007).

The most common chronic disease of childhood, asthma can occur at any age. For most patients, asthma is a disruptive disease, affecting school and work attendance, occupational choices, physical activity, and general quality of life.

Despite increased knowledge regarding the pathology of asthma and the development of improved medications and management plans, the death rate from the disease continues to rise. Ethnic and racial disparities affect morbidity and mortality in asthma, which are higher in inner-city African Americans and Latinos (Wright & Subramanian, 2007). Contributing to these disparities are epidemiology and risk factors; genetics and molecular aspects; inner-city environments; limited community assets; health care access, delivery, and quality; and lack of insurance coverage.

Unlike other obstructive lung diseases, asthma is largely reversible, either spontaneously or with treatment. Patients with asthma may experience symptom-free periods alternating with acute exacerbations that last from minutes to hours or days.

Allergy is the strongest predisposing factor for asthma. Chronic exposure to airway irritants or allergens also increases the risk of asthma. Common allergens can be seasonal (grass, tree, and weed pollens) or perennial (eg, mold, dust, roaches, animal dander). Common triggers for asthma symptoms and exacerbations include airway irritants (eg, air pollutants, cold, heat, weather changes, strong odors or perfumes, smoke), exercise, stress or emotional upset, rhinosinusitis with postnasal drip, medications, viral respiratory tract infections, and gastroesophageal reflux. Most people who have asthma are sensitive to a variety of triggers.

Pathophysiology



The underlying pathology in asthma is reversible and diffuse airway inflammation that leads to airway narrowing. This narrowing, which is exacerbated by a variety of changes in the airway, includes bronchoconstriction, airway edema, airway hyperresponsiveness, and airway remodeling. The interaction of these factors determines the clinical manifestations and severity of asthma (Expert Panel Report 3, 2007).

Mast cells, neutrophils, eosinophils, and lymphocytes play a key role in the inflammation of asthma. When activated, mast cells release several chemicals called mediators. These chemicals, which include histamine, bradykinin, prostaglandins, and leukotrienes, perpetuate the inflammatory response, causing increased blood flow, vasoconstriction, fluid leak from the vasculature, attraction of white blood cells to the area, and bronchoconstriction (Expert Panel Report 3, 2007).

In acute exacerbations of asthma, bronchial smooth muscle contraction or bronchoconstriction occurs quickly to narrow the airway in response to an exposure. Acute bronchoconstriction due to allergens results from an immunoglobulin E (IgE)-dependent release of mediators from mast cells; these mediators include histamine, tryptase, leukotrienes, and prostaglandins that directly contract the

airway. There are also non-IgE-mediated responses and proinflammatory cytokines (Expert Panel Report 3, 2007). In addition, alpha- and beta₂-adrenergic receptors of the sympathetic nervous system located in the bronchi play a role. When the alpha-adrenergic receptors are stimulated, bronchoconstriction occurs. When the beta₂-adrenergic receptors are stimulated, bronchodilation occurs. The balance between alpha- and beta₂-adrenergic receptors is controlled primarily by cyclic 3',5'-adenosine monophosphate (cAMP). Beta₂-adrenergic stimulation results in increased levels of cAMP, which inhibits the release of chemical mediators and causes bronchodilation.

As asthma becomes more persistent, the inflammation progresses and other factors may be involved in airflow limitation. These include airway edema, mucus hypersecretion, and the formation of mucous plugs. Also, airway "remodeling" may occur in response to chronic inflammation, causing further airway narrowing.

Clinical Manifestations

The three most common symptoms of asthma are cough, dyspnea, and wheezing. In some instances, cough may be the only symptom. An asthma attack often occurs at night or early in the morning, possibly because of circadian variations that influence airway receptor thresholds.

An asthma exacerbation may begin abruptly but most frequently is preceded by increasing symptoms over the previous few days. There is cough, with or without mucus production. At times, the mucus is so tightly wedged in the narrowed airway that the patient cannot cough it up. There may be generalized wheezing (the sound of airflow through narrowed airways), first on expiration and then possibly during inspiration as well. Generalized chest tightness and dyspnea occur. Expiration requires effort and becomes prolonged. As the exacerbation progresses, diaphoresis, tachycardia, and a widened pulse pressure may occur along with hypoxemia and central cyanosis (a late sign of poor oxygenation). Although severe, life-threatening hypoxemia can occur in asthma, it is relatively uncommon. The hypoxemia is secondary to a ventilation-perfusion mismatch and readily responds to supplemental oxygenation.

Symptoms of exercise-induced asthma include maximal symptoms during exercise, absence of nocturnal symptoms, and sometimes only a description of a "choking" sensation during exercise.

Assessment and Diagnostic Findings

To establish the diagnosis, the clinician must determine that episodic symptoms of airflow obstruction are present, airflow is at least partially reversible, and other causes have been excluded. A positive family history and environmental factors, including seasonal changes, high pollen counts, mold, pet dander, climate changes (particularly cold air), and air pollution, are primarily associated with asthma. In addition, asthma is associated with a variety of occupation-related chemicals and compounds. Comorbid conditions that may accompany asthma include gastroesophageal reflux disease (GERD), drug-induced asthma, and allergic bronchopulmonary aspergillosis. Other possible allergic reactions that may accompany asthma include eczema, rashes, and temporary edema.

During acute episodes, sputum and blood tests may disclose eosinophilia (elevated levels of eosinophils). Serum levels of IgE may be elevated if allergy is present. Arterial blood gas analysis and pulse oximetry reveal hypoxemia during acute attacks. Initially, hypocapnia and respiratory alkalosis are present. As the patient's condition worsens and he or she becomes more fatigued, the PaCO₂ may increase. Because carbon dioxide is 20 times more diffusible than oxygen, it is rare for PaCO₂ to be normal or elevated in a person who is breathing very rapidly.

NURSING ALERT

Normal PaCO₂ during an asthma attack may be a signal of impending respiratory failure.

During an exacerbation, the FEV₁ and FVC are markedly decreased but improve with bronchodilator administration (demonstrating reversibility). Pulmonary function is usually normal between exacerbations. The occurrence of a severe, continuous reaction is referred to as status asthmaticus and is considered life-threatening (see later discussion).

Asthma severity is considered in the selection of the initial type, amount, and schedule of treatment (Expert Panel Report 3, 2007). The severity of disease is classified by current impairment and future risk of adverse events. Impairment is defined by the following factors: nighttime awakenings, need for short-acting bronchodilators for relief of symptoms, work/school days missed, ability to engage in normal activities, and quality of life. Lung function is evaluated by spirometry. Assessment of risk of future adverse events is evaluated by numbers of exacerbations, need for emergency department care or hospitalizations in the past year, demographic data (gender, ethnicity, nonuse of prescribed inhaled corticosteroid therapy, existing smoking), psychosocial factors and attitudes, and beliefs about taking medication (Expert Panel Report 3, 2007).

Prevention

Patients with recurrent asthma should undergo tests to identify the substances that precipitate the symptoms. Possible causes are dust, dust mites, roaches, certain types of cloth, pets, horses, detergents, soaps, certain foods, molds, and pollens. If the attacks are seasonal, pollens can be strongly suspected. Patients are instructed to avoid the causative agents whenever possible. Knowledge is the key to quality asthma care. Evaluation of impairment and risk are key in the control.

Complications

Complications of asthma may include status asthmaticus, respiratory failure, pneumonia, and atelectasis. Airway obstruction, particularly during acute asthmatic episodes, often results in hypoxemia, requiring the administration of oxygen and the monitoring of pulse oximetry and arterial blood gases. Fluids are administered, because people with asthma are frequently dehydrated from diaphoresis and insensible fluid loss with hyperventilation.

Medical Management

Immediate intervention may be necessary, because continuing and progressive dyspnea leads to increased anxiety, aggravating the situation. The Expert Panel 3 Guidelines for

the Diagnosis and Management of Asthma (2007) are based on the concept of severity and control of asthma along with the domains of impairment and risk as keys to improving care. Primary concerns in the treatment of patients are impairment of lung function and normal life and risk of exacerbations, decline in lung function, and adverse effects from medications (Expert Panel Report 3, 2007).

Pharmacologic Therapy

Figure 24-7 shows the pharmacologic treatment of asthma using a stepwise approach. There are two general classes of asthma medications: quick-relief medications for immediate treatment of asthma symptoms and exacerbations and long-acting medications to achieve and maintain control of persistent asthma (Tables 24-3 and 24-4). Because the underlying pathology of asthma is inflammation, control of persistent asthma is accomplished primarily with regular use of anti-inflammatory medications. These medications have systemic side effects when used over the long term. The route of choice for administration of these medications is an MDI or other type of inhaler, because it allows for topical administration (see Chart 24-4 and Table 24-1).

Quick-Relief Medications

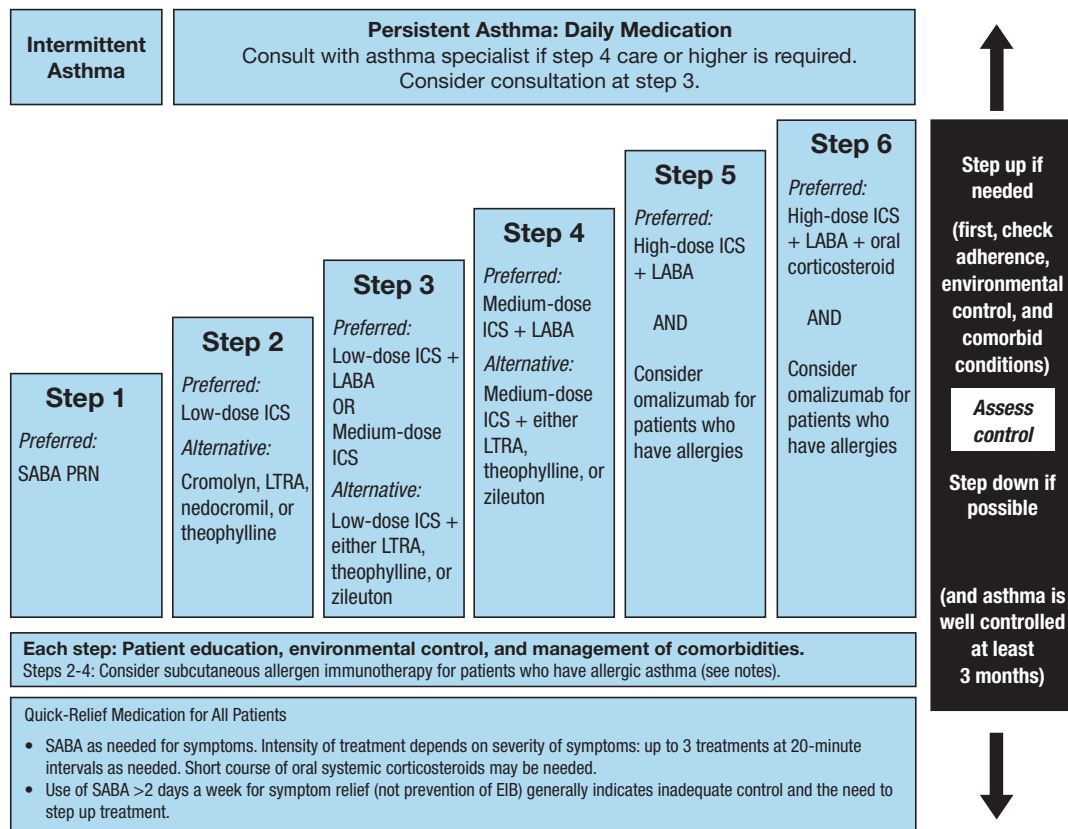
Short-acting beta₂-adrenergic agonists (albuterol [Proventil, Ventolin], levalbuterol [Xopenex], and pirbuterol [Maxair]) are the medications of choice for relief of acute symptoms and prevention of exercise-induced asthma. They are used to relax smooth muscle.

Anticholinergics (eg, ipratropium bromide [Atrovent]) inhibit muscarinic cholinergic receptors and reduce intrinsic vagal tone of the airway. These may be used in patients who do not tolerate short-acting beta₂-adrenergic agonists.

Long-Acting Control Medications

Corticosteroids are the most potent and effective anti-inflammatory medications currently available. They are broadly effective in alleviating symptoms, improving airway function, and decreasing peak flow variability. Initially, an inhaled form is used. A spacer should be used with inhaled corticosteroids, and patients should rinse their mouth after administration to prevent thrush, a common complication associated with use of inhaled corticosteroids. A systemic preparation may be used to gain rapid control of the disease; to manage severe, persistent asthma; to treat moderate to severe exacerbations; to accelerate recovery; and to prevent recurrence. Cromolyn sodium (Crolom, NasalCrom) and nedocromil (Alocril, Tilade) are mild to moderate anti-inflammatory agents and are considered alternative medications for treatment. These medications stabilize mast cells. They also are effective on a prophylactic basis to prevent exercise-induced asthma or in unavoidable exposure to known triggers. These medications are contraindicated in acute asthma exacerbations.

Long-acting beta₂-adrenergic agonists are used with anti-inflammatory medications to control asthma symptoms, particularly those that occur during the night. These agents are also effective in the prevention of exercise-induced asthma. Long-acting beta₂-adrenergic agonists are not indicated for immediate relief of symptoms. Theophylline (Slo-Bid, Theo-Dur) is a mild to moderate bronchodilator that is



Key: **Alphabetical order is used when more than one treatment option is listed within either preferred or alternative therapy.** EIB, exercise-induced bronchospasm; ICS, inhaled corticosteroid; LABA, inhaled long-acting beta₂-agonist; LTRA, leukotriene receptor antagonist; SABA, inhaled short-acting beta₂-agonist

Notes:

- The stepwise approach is meant to assist, not replace, the clinical decision making required to meet individual patient needs.
- If alternative treatment is used and response is inadequate, discontinue it and use the preferred treatment before stepping up.
- Zileuton is a less desirable alternative due to limited studies as adjunctive therapy and the need to monitor liver function. Theophylline requires monitoring of serum concentration levels.
- In step 6, before oral systemic corticosteroids are introduced, a trial of high-dose ICS + LABA + either LTRA, theophylline, or zileuton may be considered, although this approach has not been studied in clinical trials.
- Steps 1, 2, and 3 preferred therapies are based on evidence A; step 3 alternative therapy is based on evidence A for LTRA, evidence B for theophylline, and evidence D for zileuton. Step 4 preferred therapy is based on evidence B, and alternative therapy is based on evidence B for LTRA and theophylline and evidence D for zileuton. Step 5 preferred therapy is based on evidence B. Step 6 preferred therapy is based on (EPR—2 1997) and evidence B for omalizumab.
- Immunotherapy for steps 2-4 is based on evidence B for house-dust mites, animal danders, and pollens; evidence is weak or lacking for molds and cockroaches. Evidence is strongest for immunotherapy with single allergens. The role of allergy in asthma is greater in children than in adults.
- Clinicians who administer immunotherapy or omalizumab should be prepared and equipped to identify and treat anaphylaxis that may occur.

Figure 24-7 Stepwise approach for managing asthma in youths 12 years of age and older and adults. Redrawn from Expert Panel Report 3. (2007). *Guidelines for the diagnosis and management of asthma* (p. 343). NIH Publication Number 08-5846. National Asthma Education and Prevention Program. Summary Report. Bethesda, MD: U.S. Department of Health and Human Services, National Heart, Lung and Blood Institute.

Table 24-3



LONG-TERM MEDICATIONS FOR TREATMENT OF ASTHMA (CONTROLLER MEDICATIONS)

| Medication | Indications/Mechanisms | Potential Adverse Effects | Nursing Considerations |
|---|--|--|--|
| Inhaled Corticosteroids (ICs) beclomethasone dipropionate (QVAR) beclomethasone (Beconase-AQ) budesonide (Pulmicort) ciclesonide (Alvesco) flunisolide (AeroBid) fluticasone (Flovent) mometasone furoate (Asmanex) triamcinolone acetonide (Azmacort) | <p><i>Indications</i></p> Long-term prevention of symptoms; suppression, control, and reversal of inflammation Reduce need for oral corticosteroid. | Cough, dysphonia, oral thrush (candidiasis), headache. In high doses, systemic effects may occur (eg, adrenal suppression, osteoporosis, skin thinning, and easy bruising). | Instruct patient in correct use of metered-dose inhaler (MDI) and use of spacer/holding chamber devices. Instruct patient to rinse mouth after inhalation to reduce local side effects. |
| Systemic Corticosteroids methylprednisolone (Medrol) prednisolone (Prelone) prednisone (Deltasone, Orasone) | <p><i>Indications</i></p> For short-term (3–10 days) “burst”: to gain prompt control of inadequately controlled persistent asthma. For long-term prevention of symptoms in severe persistent asthma: suppression, control, and reversal of inflammation. | Short-term use: reversible abnormalities in glucose metabolism, increased appetite, fluid retention, weight gain, mood alteration, hypertension, peptic ulcer, and rarely aseptic necrosis. Long-term use: adrenal axis suppression, growth suppression, dermal thinning, hypertension, diabetes, Cushing’s syndrome, cataracts, muscle weakness, and—in rare instances—impaired immune function. Consideration should be given to comorbidities that could be worsened by systemic corticosteroids. | Instruct patient about possible side effects and the importance of taking the medication as prescribed (usually a single dose in the morning daily or on an alternate-day schedule, which may produce less adrenal suppression). |
| Long-Acting Beta₂-Agonists (LABAs) <i>Inhaled:</i> salmeterol (Serevent Diskus) formoterol (Foradil Aerolizer) | <p><i>Indications</i></p> Long-term prevention of symptoms, added to ICS. Prevention of exercise-induced bronchospasm. | Should <i>not</i> be used to treat acute symptoms or exacerbations. Decreased protection against exercise-induced bronchospasm may occur with regular use. Tachycardia, muscle tremor, hypokalemia, ECG changes with overdose. A diminished bronchoprotective effect may occur within 1 wk of chronic therapy. Potential risk of uncommon, severe, life-threatening or fatal exacerbation. | Reinforce to patient that these medications should <i>not</i> be used to treat acute asthma symptoms or exacerbations. Instruct patient about correct use of MDI or aerolizer inhaler. |
| <i>Oral:</i> albuterol (Proventil) sustained-release | <p><i>Mechanisms</i></p> Bronchodilation. Smooth muscle relaxation following adenylate cyclase activation and increase in cyclic AMP, producing functional antagonism of bronchoconstriction. Compared to SABA, salmeterol (but not formoterol) has slower onset of action (15–30 min). Both salmeterol and formoterol have longer duration (>12 h) compared to SABA. | Inhaled route is preferred to oral route because LABAs are longer acting and have fewer side effects than oral sustained-release agents. | |
| Methylxanthines theophylline (Theo-Dur) sustained-release tablets and capsules | <p><i>Indications</i></p> Long-term control and prevention of symptoms in mild persistent asthma or as adjunctive with ICS, in moderate or persistent asthma. | Dose-related acute toxicities include tachycardia, nausea and vomiting, tachyarrhythmias (SVT), central nervous system stimulation, headache, seizures, hematemesis, hyperglycemia, and hypokalemia. | Maintain steady-state serum concentrations between 5 and 15 µg/mL. |

Continued

Table 24-3


**LONG-TERM MEDICATIONS FOR TREATMENT OF ASTHMA
(CONTROLLER MEDICATIONS) (Continued)**

| Medication | Indications/Mechanisms | Potential Adverse Effects | Nursing Considerations |
|--|--|--|--|
| | <p><i>Mechanisms</i> Bronchodilation. Smooth muscle relaxation from phosphodiesterase inhibition and possibly adenosine antagonism. May affect eosinophilic infiltration into bronchial mucosa as well as decrease T-lymphocyte numbers in epithelium. Increases diaphragm contractility and mucociliary clearance.</p> | <p>Adverse effects at usual therapeutic doses include insomnia, gastric upset, aggravation of ulcer or reflux, difficulty in urination in elderly males who have prostatism. Not generally recommended for exacerbations. There is minimal evidence for added benefit to optimal doses of SABA. Serum concentration monitoring is mandatory. Available as sustained-release tablets and capsules.</p> | <p>Absorption and metabolism may be affected by numerous factors that can produce significant changes in steady-state serum theophylline concentrations. Instruct patients to discontinue if they experience toxicity. Inform patients about the importance of blood tests to monitor serum concentration. Instruct patient to check with primary health care provider before taking any new medication.</p> |
| <p>Combined Medication fluticasone/salmeterol (Advair)</p> | <p>DPI 100 µg/50 µg 250 µg/50 µg 500 µg/50 µg</p> | <p>Lowest dose of DPI or HFA used for patients whose asthma is not controlled on low- to medium-dose ICS.</p> | |
| <p>budesonide/formoterol (Symbicort)</p> | <p>HFA 45 µg/21 µg 115 µg/21 µg 230 µg/21 µg HFA MDI 80 µg/4.5 µg 160 µg/4.5 µg</p> | <p>Higher doses of DPI or HFA used for patients whose asthma is not controlled on medium- to high-dose ICS. Lower dose used for patients who have asthma not controlled on low- to medium-dose ICS. Higher dose used for patients who have asthma not controlled on medium- to high-dose ICS.</p> | |
| <p>Cromolyn and Nedocromil Cromolyn sodium (Intal) nedocromil (Tilade)</p> | <p><i>Indications</i> Long-term prevention of symptoms in mild persistent asthma; may modify inflammation. Preventive treatment prior to exposure to exercise or known allergen.</p> <p><i>Mechanisms</i> Anti-inflammatory. Blocks early and late reaction to allergen. Stabilizes mast cell membranes and inhibits activation and release of mediators from eosinophils and epithelial cells. Inhibits acute response to exercise, cold dry air, and SO₂.</p> | <p>Cough and irritation. 15–20% of patients complain of an unpleasant taste from nedocromil. Dose of cromolyn by MDI may be inadequate to affect airway hyperresponsiveness. Nebulizer delivery may be preferred for some patients. Safety is the primary advantage of these agents. One dose before exercise or exposure to allergen provides effective prophylaxis for 1–2 h. Not as effective for exercise-induced bronchospasm as short-acting beta₂-agonist.</p> | <p>Inform patient that 4–6-wk trial may be needed to determine maximum benefit. Instruct patient about correct use of inhaler.</p> |
| <p>Leukotriene Modifiers Leukotriene Receptor Antagonists montelukast (Singulair)</p> | <p><i>Mechanism</i> Selective competitive inhibitor of CysLT₁ receptor.</p> <p><i>Indications</i> Long-term control and prevention of symptoms in mild persistent asthma for patients ≥1 yr of age. May also be used with ICS as combination therapy in moderate persistent asthma.</p> | <p>May attenuate EIB in some patients, but less effective than ICS therapy.</p> <p>LTRA + LABA should not be used as a substitute for ICS + LABA. No specific adverse effects have been identified. Available in tablets and granules.</p> | <p>Instruct patient to take at least 1 h before meals or 2 h after meals.</p> <p>Inform patient that zafirlukast can inhibit the metabolism of warfarin. INRs should be monitored if patient is taking both medications.</p> |
| <p>zafirlukast (Accolate)</p> | <p>Long-term control and prevention of symptoms in mild persistent asthma. May also be used with ICS as combination therapy in moderate persistent asthma.</p> | <p>Cases of reversible hepatitis have been reported along with rare cases of irreversible hepatic failure resulting in death and liver transplantation. Available in tablets.</p> | <p>Instruct patients to discontinue use if they experience signs and symptoms of liver dysfunction (right upper quadrant pain, pruritus, lethargy, jaundice, nausea), and to notify their primary health care provider.</p> |

Continued on following page

Table 24-3


**LONG-TERM MEDICATIONS FOR TREATMENT OF ASTHMA (CONTROLLER MEDICATIONS)
(Continued)**

| Medication | Indications/Mechanisms | Potential Adverse Effects | Nursing Considerations |
|--|---|---|---|
| 5-Lipoxygenase Inhibitor zileuton (Zyflo) | <p><i>Mechanism</i> Inhibits the production of leukotrienes from arachidonic acid, both LTB and the cysteinyl leukotrienes.</p> <p><i>Indications</i> Long-term control and prevention of symptoms in mild persistent asthma for patients. May be used with ICS as combination therapy in moderate persistent asthma in patients.</p> | Elevation of liver enzymes has been reported. Limited case reports of reversible hepatitis and hyperbilirubinemia. | Inform patient that zileuton can inhibit the metabolism of warfarin and theophylline. Therefore, the doses of these medications should be monitored accordingly. Teach patient the importance of monitoring medication levels and tests of liver function. |
| Immunomodulators omalizumab (Xolair) | <p><i>Indications</i> Long-term control and prevention of symptoms in adults with moderate or severe persistent allergic asthma inadequately controlled with ICS.</p> <p><i>Mechanisms</i> Monoclonal antibody (anti-IgE) that binds to circulating IgE, preventing it from binding to the high-affinity receptors on basophils and mast cells. Decreases mast cell mediator release from allergen exposure.</p> | Administered by subcutaneous injection. Anaphylaxis has been reported in 0.2% of treated patients. Pain, bruising and skin reactions (itching, redness, stinging) at injection sites. The dose is administered either every 2 or 4 wk and is dependent on the patient's body weight and IgE level before therapy. A maximum of 150 mg can be administered in one injection. Medication needs to be stored under refrigeration at 2–8°C. It is unknown if patients will develop significant antibody titers to the drug with long-term administration. | Monitor patients for allergic reactions or anaphylaxis following administration. Be prepared to initiate emergency treatment if anaphylaxis occurs. Instruct patient about signs and symptoms that indicate allergic reaction and immediate action to take. Remind patient to continue to take other medications prescribed for treatment of asthma. |

AMP, 3',5'-adenosine monophosphate; DPI, dry powder inhaler; ECG, electrocardiogram; EIB, exercise-induced bronchospasm; HFA, hydrofluoroalkane; ICS, inhaled corticosteroid; INR, international normalized ratio; LTB, leukotriene B; LTRA, leukotriene receptor antagonist; SABA, inhaled short-acting beta₂-agonist; SVT, supraventricular tachycardia.

usually used in addition to inhaled corticosteroids, mainly for relief of nighttime asthma symptoms. Salmeterol (Serevent) and formoterol (Foradil) have a duration of bronchodilation for at least 12 hours. They are used with other medications in long-term control of asthma.

Leukotriene modifiers (inhibitors), or *antileukotrienes*, are a class of medications that include montelukast (Singulair), zafirlukast (Accolate), and zileuton (Zyflo). Leukotrienes, which are synthesized from membrane phospholipids through a cascade of enzymes, are potent bronchoconstrictors that also dilate blood vessels and alter permeability. Leukotriene inhibitors act either by interfering with leukotriene synthesis or by blocking the receptors where leukotrienes exert their action. They may provide an alternative to inhaled corticosteroids for mild persistent asthma, or they may be added to a regimen of inhaled corticosteroids in more severe asthma to attain further control.

Immunomodulators prevent binding of IgE to the high-affinity receptors of basophils and mast cells. Omalizumab

(Xolair) is a monoclonal antibody and may be used for patients with allergies and severe persistent asthma.

Management of Exacerbations

Asthma exacerbations are best managed by early treatment and education, including the use of written action plans as part of any overall effort to educate patients about self-management techniques, especially those with moderate or severe persistent asthma or with a history of severe exacerbations (Expert Panel Report 3, 2007). Quick-acting beta₂-adrenergic agonist medications are first used for prompt relief of airflow obstruction. Systemic corticosteroids may be necessary to decrease airway inflammation in patients who fail to respond to inhaled beta-adrenergic medications. In some patients, oxygen supplementation may be required to relieve hypoxemia associated with moderate to severe exacerbations. In addition, response to treatment may be monitored by serial measurements of lung function.

Evidence from clinical trials suggests that antibiotic therapy, whether administered routinely or when suspicion

Table 24-4



QUICK-RELIEF MEDICATIONS FOR TREATMENT OF ASTHMA

| Medication | Indications/Mechanisms | Potential Adverse Effects | Nursing Considerations |
|---|--|--|---|
| Inhaled Short-Acting Beta₂-Agonists (SABAs) albuterol (Proventil HFA, Ventolin HFA) levalbuterol HFA (Xopenex) pirbuterol CFC (Maxair) metaproterenol sulfate (Alupent) | Indications: Relief of acute symptoms; quick-relief medication. Preventive treatment for exercise-induced bronchospasm. Mechanisms: Bronchodilation. Binds to the beta-2 adrenergic receptor, producing smooth muscle relaxation and decreased bronchoconstriction. | Tachycardia, muscle tremor, hypokalemia, increased lactic acid, headache, and hyperglycemia. Inhaled route causes few systematic adverse effects. Patients with preexisting cardiovascular disease, especially the elderly, may have adverse cardiovascular reactions with inhaled therapy. Lack of effect or need for regular use indicates inadequate asthma control. | Patients need to be instructed in correct use of inhaled agents and understanding of how to evaluate amount of remaining medication in MDI. Periodic cleaning of device recommended. Patient is informed about possible side effects and need to inform health care provider about increased use of medication to control symptoms. |
| Anticholinergics ipratropium (Atrovent) | Indications: Relief of acute bronchospasm. Mechanisms: Bronchodilation. Inhibition of muscarinic cholinergic receptors. Reduction of vagal tone of airways. May decrease mucous gland secretion. | Dryness of mouth and respiratory secretions. May cause increased wheezing in some patients. Does not block exercise-induced bronchospasm. Is not effective in long-term control of asthma. | Instruct patient in correct use of inhaled agents. Ensure adequate fluid intake. Assess patient for hypersensitivity to atropine, soybeans, peanuts; glaucoma, prostatic hypertrophy. |
| Corticosteroids Systemic: methylprednisolone (Medrol) prednisolone (Prelone) prednisone (Deltasone, Orasone) | Indications: For moderate or severe exacerbations to prevent progression of exacerbation, reverse inflammation, speed recovery, and reduce rate of relapse. Mechanisms: Anti-inflammatory. Block reaction to allergen and reduce hyperresponsiveness. Inhibit cytokine production, adhesion protein activation, and inflammatory cell migration and activation. Revers beta-2 receptor down-regulation. | Short-term use: reversible blood glucose abnormalities, increased appetite, fluid retention, weight gain, mood alteration, hypertension, peptic ulcer. Consideration must be given to comorbidities that may be worsened by systemic corticosteroids. | Explain to patient that action is often rapid in onset, although resolution of symptoms may take 3–10 days. Instruct patient about possible side effects and the importance of taking the medication as prescribed. |

CFC, chlorofluracarbons; HFA, hydrofluoroalkane; MDI, metered-dose inhaler.

of bacterial infection is low, is not beneficial for asthma exacerbations (Expert Panel Report 3, 2007). Antibiotics may be appropriate in the treatment of acute asthma exacerbations in patients with comorbid conditions (eg, fever and purulent sputum, evidence of pneumonia, suspected bacterial sinusitis).

Despite insufficient data supporting or refuting the benefits of using a written asthma action plan as compared to medical management alone, the 2007 Expert Panel Report 3 recommends the use of a written asthma action plan to educate patients about self-management (Fig. 24-8). Plans can be based on either symptoms or peak flow measurements. They should focus on daily management as well as the recognition and handling of worsening symptoms. Patient self-management and early recognition of problems lead to more efficient communication with health care providers about asthma exacerbations (Expert Panel Report 3, 2007).

Peak Flow Monitoring

Peak flow meters measure the highest airflow during a forced expiration (Fig. 24-9). Daily peak flow monitoring is recommended for patients who meet one or more of the following criteria: have moderate or severe persistent asthma, have poor perception of changes in airflow or worsening symptoms, have unexplained response to environmental or occupational exposures, or at the discretion of the clinician and patient (Expert Panel Report 3, 2007). If peak flow monitoring is used, it helps measure asthma severity and, when added to symptom monitoring, indicates the current degree of asthma control.

The patient is instructed in the proper technique (Chart 24-6), particularly about using maximal effort; peak flows are monitored for 2 or 3 weeks after receipt of optimal asthma therapy. Then the patient's "personal best" value is measured. The green (80% to 100% of personal best), yellow (60% to 80%), and red (less than 60%) zones are

Asthma Action Plan

For: _____ Doctor: _____ Date: _____
 Doctor's Phone Number _____ Hospital/Emergency Department Phone Number _____

GREEN ZONE

Doing Well

- No cough, wheeze, chest tightness, or shortness of breath during the day or night
- Can do usual activities

And, if a peak flow meter is used,
Peak flow: more than _____
 (80% or more of my best peak flow)
 My best peak flow is: _____

Take these long-term-control medicines each day (include an anti-inflammatory).

| Medicine | How much to take | When to take it |
|----------|------------------|-----------------|
| _____ | _____ | _____ |
| _____ | _____ | _____ |

Identify and avoid and control the things that make your asthma worse, like (list here):

Before exercise, if prescribed, take: 2 or 4 puffs _____ 5 to 60 minutes before exercise

YELLOW ZONE

Asthma Is Getting Worse

- Cough, wheeze, chest tightness, or shortness of breath, or
- Waking at night due to asthma, or
- Can do some, but not all, usual activities

-Or-
Peak flow: _____ to _____
 (50% to 79% of my best peak flow)

First Add: quick-relief medicine—and keep taking your GREEN ZONE medicine.

_____ 2 or 4 puffs, every 20 minutes for up to 1 hour
 (short-acting beta₂-agonist) Nebulizer, once

Second If applicable, remove yourself from the thing that made your asthma worse.
 If your symptoms (and peak flow, if used) return to GREEN ZONE after 1 hour of above treatment: Continue monitoring to be sure you stay in the green zone.

-Or-
If your symptoms (and peak flow, if used) do not return to GREEN ZONE after 1 hour of above treatment:

Take: _____ 2 or 4 puffs or Nebulizer
 (short-acting beta₂-agonist)

Add: _____ mg per day For _____ (3-10) days
 (oral corticosteroid)

Call the doctor: _____, before/ within _____ hours after taking the oral corticosteroid.
 (phone)

RED ZONE

Medical Alert!

- Very short of breath, or
- Quick-relief medicines have not helped, or
- Cannot do usual activities, or
- Symptoms are same or get worse after 24 hours in Yellow Zone

-Or-
Peak flow: less than _____
 (50% of my best peak flow)

Take this medicine:

_____ 4 or 6 puffs or Nebulizer
 (short-acting beta₂-agonist)

_____ mg
 (oral corticosteroid)

Then call your doctor NOW. Go to the hospital or call an ambulance if:

- You are still in the red zone after 15 minutes AND
- You have not reached your doctor.

DANGER SIGNS

- Trouble walking and talking due to shortness of breath
- Lips or fingernails are blue

Take 4 or 6 puffs of your quick-relief medicine AND

Go to the hospital or call for an ambulance _____ NOW!
 (phone)

Figure 24-8 Asthma action plan. Redrawn from Expert Panel Report 3. (2007). *Guidelines for the diagnosis and management of asthma* (p. 119). NIH Publication Number 08-5846. National Asthma Education and Prevention Program. Bethesda, MD: U.S. Department of Health and Human Services, National Heart, Lung and Blood Institute.

determined, and specific actions are delineated for each zone, enabling the patient to monitor and manipulate his or her own therapy after careful instruction (Expert Panel Report 3, 2007).

The Expert Panel Report 3 (2007) recommends that peak flow monitoring be considered an adjunct to asthma management for patients with moderate to severe persistent asthma. Peak flow monitoring plans may enhance communication between the patient and health care providers and may increase the patient's awareness of disease status and control.

Nursing Management

The immediate nursing care of patients with asthma depends on the severity of symptoms. The patient may be treated successfully as an outpatient if asthma symptoms are relatively mild or may require hospitalization and intensive care if symptoms are acute and severe. The patient and family are often frightened and anxious because of the patient's dyspnea. Therefore, a calm approach is an important aspect

of care. The nurse assesses the patient's respiratory status by monitoring the severity of symptoms, breath sounds, peak flow, pulse oximetry, and vital signs.

The nurse generally performs the following interventions:

- Obtains a history of allergic reactions to medications before administering medications
- Identifies medications the patient is currently taking
- Administers medications as prescribed and monitors the patient's responses to those medications. These medications may include an antibiotic if the patient has an underlying respiratory infection.
- Administers fluids if the patient is dehydrated

If the patient requires intubation because of acute respiratory failure, the nurse assists with the intubation procedure, continues close monitoring of the patient, and keeps the patient and family informed about procedures. Intubation and mechanical ventilation are discussed in Chapter 25.

Figure 24-9 Peak flow meters measure the highest volume of air flow during a forced expiration (*left*). Volume is measured in color-coded zones (*right*): the green zone signifies 80% to 100% of personal best; yellow, 60% to 80%; and red, less than 60%. If peak flow falls below the red zone, the patient should take the appropriate actions prescribed by his or her health care provider.



Promoting Home and Community-Based Care

Teaching Patients Self-Care

A major challenge is to implement basic asthma management principles at the community level. Strategies include education of health care providers, establishment of programs for asthma education (for patients and providers), use of outpatient follow-up care for patients, and a focus on chronic management versus acute episodic care. Nurses are pivotal to achievement of these objectives.

Patient teaching is a critical component of care for patients with asthma. Multiple inhalers, different types of inhalers, antiallergy therapy, antireflux medications, and avoidance measures are essential for long-term control.

This complex therapy requires a partnership between the patient and the health care providers to determine the desired outcomes and to formulate a plan to achieve those outcomes. The patient then carries out daily therapy as part of self-care management, with input and guidance by his or her health care providers. Before a partnership can be established, the patient must understand the following:

- The nature of asthma as a chronic inflammatory disease
- The definitions of inflammation and bronchoconstriction
- The purpose and action of each medication
- Triggers to avoid, and how to do so
- Proper inhalation technique

CHART
24-6



HOME CARE CHECKLIST Use of Peak Flow Meter

| At the completion of the home care instruction, the patient or caregiver will be able to: | PATIENT | CAREGIVER |
|---|---------|-----------|
| • Describe the rationale for using a peak flow meter in asthma management. | ✓ | ✓ |
| • Explain how peak flow monitoring is used along with symptoms to determine severity of asthma. | ✓ | ✓ |
| • Demonstrate steps for using the peak flow meter correctly. | ✓ | |
| • Move the indicator to the bottom of the numbered scale. | ✓ | |
| • Stand up. | ✓ | |
| • Take a deep breath and fill the lungs completely. | ✓ | |
| • Place mouthpiece in mouth and close lips around mouthpiece. (do not put tongue inside opening). | ✓ | |
| • Blow out hard and fast with a single blow. | ✓ | |
| • Record the number achieved on the indicator. If patient coughs or a mistake is made in the process, do it over. | ✓ | |
| • Repeat steps 1–5 two more times and write the highest number in the asthma diary. | ✓ | |
| • Explain how to determine the “personal best” peak flow reading. | ✓ | ✓ |
| • Describe the significance of the color zones for peak flow monitoring. | ✓ | ✓ |
| • Demonstrate how to clean the peak flow meter. | ✓ | ✓ |
| • Discuss how and when to contact the health care provider about changes or decreases in peak flow values. | ✓ | ✓ |

- How to perform peak flow monitoring (see Chart 24-6)
- How to implement an action plan
- When to seek assistance, and how to do so

An assortment of excellent educational materials is available from the NHLBI and other sources. The nurse should obtain current educational materials for the patient based on the patient's diagnosis, causative factors, educational level, and cultural background. If a patient has a coexisting sensory impairment (ie, vision loss or hearing impairment), materials should be provided in an alternative format.

Continuing Care

Nurses who have contact with patients in the hospital, clinic, school, or office use the opportunity to assess the patient's respiratory status and ability to manage self-care to prevent serious exacerbations. Nurses emphasize adherence to the prescribed therapy, preventive measures, and the need to keep follow-up appointments with health care providers. Home visits to assess the home environment for allergens may be indicated for patients with recurrent exacerbations. Nurses refer patients to community support groups. In addition, nurses remind patients and families about the importance of health promotion strategies and recommended health screening.

Status Asthmaticus

Status asthmaticus is severe and persistent asthma that does not respond to conventional therapy. The attacks can occur with little or no warning and can progress rapidly to asphyxiation. Infection, anxiety, nebulizer abuse, dehydration, increased adrenergic blockage, and nonspecific irritants may contribute to these episodes. An acute episode may be precipitated by hypersensitivity to aspirin.

Pathophysiology

The basic characteristics of asthma (inflammation of bronchial mucosa, constriction of the bronchiolar smooth muscle, and thickened secretions) decrease the diameter of the bronchi and occur in status asthmaticus. The most common scenario is severe bronchospasm, with mucus plugging leading to asphyxia. A ventilation-perfusion abnormality results in hypoxemia. There is a reduced PaO₂ and initial respiratory alkalosis, with a decreased PaCO₂ and an increased pH. As status asthmaticus worsens, the PaCO₂ increases and the pH decreases, reflecting respiratory acidosis.

Clinical Manifestations

The clinical manifestations are the same as those seen in severe asthma; signs and symptoms include labored breathing, prolonged exhalation, engorged neck veins, and wheezing. However, the extent of wheezing does not indicate the severity of the attack. As the obstruction worsens, the wheezing may disappear; this is frequently a sign of impending respiratory failure.

Assessment and Diagnostic Findings

Pulmonary function studies are the most accurate means of assessing acute airway obstruction. Arterial blood gas measurements are obtained if the patient cannot perform pul-

monary function maneuvers because of severe obstruction or fatigue, or if the patient does not respond to treatment. Respiratory alkalosis (low PaCO₂) is the most common finding in patients with asthma.

NURSING ALERT

Frequently, increasing PaCO₂ (to normal levels or levels indicating respiratory acidosis) is a danger sign signifying impending respiratory failure.

Medical Management

Close monitoring of the patient and objective reevaluation for response to therapy are key in status asthmaticus. In the emergency setting, the patient is treated initially with a short-acting beta₂-adrenergic agonist and subsequently a short course of systemic corticosteroids, especially if the patient does not respond to the short-acting beta₂-adrenergic agonist. Corticosteroids are critical in the therapy of status asthmaticus and are used to decrease the intense airway inflammation and swelling. Short-acting inhaled beta₂-adrenergic agonists provide the most rapid relief from bronchospasm. An MDI with or without a spacer may be used for nebulization of the medications. The patient usually requires supplemental oxygen and intravenous (IV) fluids for hydration. Oxygen therapy is initiated to treat dyspnea, central cyanosis, and hypoxemia. High-flow supplemental oxygen is best delivered using a partial or complete non-rebreather mask with the objective of maintaining the PaO₂ at a minimum of 92 mm Hg or O₂ saturation greater than 95%. Sedatives are contraindicated. Magnesium sulfate, a calcium antagonist, may be administered to induce smooth muscle relaxation; the magnesium can relax smooth muscle and hence cause bronchodilation by competing with calcium at calcium-mediated smooth muscle binding sites. Adverse effects of magnesium sulfate may include facial warmth, flushing, tingling, nausea, central nervous system depression, respiratory depression, and hypotension.

If there is no response to repeated treatments, hospitalization is required. Other criteria for hospitalization include poor pulmonary function test results and deteriorating blood gas levels (respiratory acidosis), which may indicate that the patient is tiring and will require mechanical ventilation. Most patients do not need mechanical ventilation, but it is used for patients in respiratory failure, for those who tire and are too fatigued by the attempt to breathe, and for those whose condition does not respond to initial treatment.

Death from asthma is associated with several risk factors, including the following (Expert Panel Report 3, 2007):

- Past history of severe exacerbation (eg, intubation or intensive care unit admission)
- Two or more hospitalizations for asthma within the past year
- Three or more emergency care visits for asthma in the past year
- Hospitalization or emergency care visit for asthma in the past month
- Use of more than two canisters of short-acting beta-agonist inhalers per month

- Difficulty in perceiving asthma symptoms or severity of exacerbations
- Lack of written asthma action plan
- Concurrent cardiovascular disease, COPD, or chronic psychiatric disease
- Low socioeconomic status or inner-city residence
- Illicit drug use

Nursing Management

The main focus of nursing management is to actively assess the airway and the patient's response to treatment. The nurse should be prepared for the next intervention if the patient does not respond to treatment.

The nurse constantly monitors the patient for the first 12 to 24 hours, or until status asthmaticus is under control. The nurse also assesses the patient's skin turgor for signs of dehydration. Fluid intake is essential to combat dehydration, to loosen secretions, and to facilitate expectoration. Nurses administer IV fluids as prescribed, up to 3 to 4 L/day, unless contraindicated. Blood pressure and cardiac rhythm should be monitored continuously during the acute phase and until the patient stabilizes and responds to therapy. The patient's energy needs to be conserved, and his or her room should be quiet and free of respiratory irritants, including flowers, tobacco smoke, perfumes, or odors of cleaning agents. Nonallergenic pillows should be used.

Cystic Fibrosis

Cystic fibrosis (CF) is the most common fatal autosomal recessive disease among the Caucasian population. A person must inherit a defective copy of the CF gene (one from each parent) to have CF. One in 31 Americans is an unknowing carrier of this gene, and approximately 30,000 children and adults in the United States have CF (Cystic Fibrosis Foundation, 2007). CF is less frequently found among Hispanic, Asian, and African Americans. CF was once considered a fatal childhood disease, but the median expected survival age is now 37 years (Flume, O'Sullivan, Robinson, et al., 2007). Patients' quality of life has also improved.

This multisystem genetic disease is usually diagnosed in infancy or early childhood but may be diagnosed later in life. Respiratory symptoms are frequently the major manifestation of CF when it is diagnosed later in life. However, a good number of patients will not demonstrate the classic symptoms of CF, which may potentially cause a diagnostic dilemma (Boyle, 2007).

Pathophysiology

CF is caused by mutations or dysfunction in the protein cystic fibrosis transmembrane conductance regulator (CFTR), which normally transports chloride ions across epithelial cell membranes. Gene mutations affect transport of these ions, leading to CF, which is characterized by thick, viscous secretions in the lungs, pancreas, liver, intestine, and reproductive tract as well as increased salt content in sweat gland secretions. More than 1500 mutations of the CFTR gene have been identified, thus creating multiple variations in presentation and progression of the disease (Boyle, 2007).

The ability to detect the common mutations of this gene allows for routine screening for CF and the detection

of carriers of the disease. Genetics counseling is an important part of health care for couples at risk. People who are heterozygous for CF (ie, have one defective gene and one normal gene) do not have the disease but can be carriers and pass the defective gene on to their children. If both parents are carriers, their risk of having a child with CF is one in four (25%) with each pregnancy. Genetic testing should be offered to adults with a positive family history of CF and to partners of people with CF who are planning a pregnancy or seeking prenatal counseling. Currently, genetic testing for CF is not recommended for the general population.

The hallmark pathology of CF is bronchial mucus plugging, inflammation, and eventual bronchiectasis. Commonly, the bronchiectasis begins in the upper lobes and progresses to involve all lobes. Progressive loss of lung function averaging from 1% to 4% per year begins in the teenage years (Boyle, 2007).

Clinical Manifestations

The pulmonary manifestations of CF include a productive cough, wheezing, hyperinflation of the lung fields on chest x-ray, and pulmonary function test results consistent with obstructive disease of the airways. Chronic respiratory inflammation and infection are caused by impaired mucus clearance. Colonization of the airways with pathogenic bacteria usually occurs early in life. *Staphylococcus aureus* and *H. influenzae* are common organisms during early childhood. As the disease progresses, *Pseudomonas aeruginosa* is ultimately isolated from the sputum of most patients. Upper respiratory manifestations of the disease include sinusitis and nasal polyps.

Nonpulmonary manifestations include gastrointestinal problems (eg, pancreatic insufficiency, recurrent abdominal pain, biliary cirrhosis, vitamin deficiencies, recurrent pancreatitis, weight loss), CF-related diabetes, genitourinary problems (male and female infertility), and clubbing of the digits (fingers and toes). (See Chapter 40 for a discussion of pancreatitis.)

Assessment and Diagnostic Findings

The diagnosis of CF requires a clinical picture consistent with the CF phenotype and laboratory evidence of CFTR dysfunction. The consensus statement of the Cystic Fibrosis Foundation on the diagnosis of CF identifies key aspects of the phenotype (Boyle, 2007):

- Chronic sinopulmonary disease as manifested by chronic cough and sputum production, persistent infection consistent with typical CF pathogens, and x-ray evidence of bronchiectasis and chronic sinusitis, often with nasal polyps
- Gastrointestinal tract and nutritional abnormalities (pancreatic insufficiency, meconium ileus or distal intestinal obstruction syndrome, failure to thrive or chronic malnutrition)
- Male urogenital problems as manifested by congenital bilateral absence of the vas deferens and obstructive azoospermia

Medical Management

CF requires both acute and chronic therapy. Because chronic bacterial infection of the airways occurs in CF, control of infections is essential to treatment. For acute airway

exacerbations, aggressive therapy involves airway clearance and antibiotics based on results of sputum cultures. The majority of patients are colonized with *P. aeruginosa*, and antibiotics are usually given for 14 to 21 days. Because patients with CF may be infected with bacteria that are resistant to multiple antibiotics, they may require several courses of antibiotic agents over long periods. In an acute exacerbation, airway clearance treatments may be used three to four times per day.

For chronic disease, the therapeutic options for CF have undergone tremendous changes. Cornerstones of treatment include airway clearance measures, mucolytic dornase alpha, nebulized antibiotics, oral antibiotics, inhaled hypertonic saline, nutritional support, and exercise (Boyle, 2007).

Airway clearance is a key intervention in the care of patients with CF, and various pulmonary techniques are used to enhance secretion clearance. Examples include manual postural drainage and chest physical therapy, high-frequency chest wall oscillation, autogenic drainage (a combination of breathing techniques at different lung volume levels to move the secretions to where they can be “huff”-coughed out), and other devices that assist in airway clearance, such as masks that generate positive expiratory pressure (PEP masks) and “flutter devices” (devices that provide an oscillatory expiratory pressure pattern with positive expiratory pressure and assist with expectoration of secretions).

Dornase alpha (Pulmozyme) is a nebulized medication administered to degrade the large amount of deoxyribonucleic acid (DNA) that accumulates within CF mucus. This agent helps decrease the viscosity of the sputum and promotes expectoration of secretions. It is recommended for patients with moderate to severe disease (severity of lung disease classification is based on predicted FEV₁ percentage) (Flume, et al., 2007).

Nebulized antibiotics include tobramycin (TOBI), colistin (Colimycin), gentamicin (Garamycin), and ceftazidime (Fortaz, Ceptaz) (Flume, et al., 2007). Inhaled antibiotics provide the advantage of high intrapulmonary drug concentrations and minimal systemic absorption.

Acute infections are treated with a variety of antibiotics. Such infections remain a major cause of mortality related to pulmonary exacerbations in adults with CF (Elpern, Patel & Balk, 2007). Antibiotics such as the macrolide azithromycin (Zithromax) may also be used. Inhaled hypertonic saline may also be used in the chronic treatment of CF. Inhalations increase hydration of the airway surface liquid in patients with CF and improve airway clearance. Regular inhalations have been shown to improve FEV₁ and decreased frequency of pulmonary exacerbations (Boyle, 2007).

Approximately 90% of patients with CF have pancreatic exocrine insufficiency and require oral pancreatic enzyme supplementation with meals (Boyle, 2007). Given the fat malabsorption in CF and increased caloric needs due to the work of breathing, nutritional counseling and monitoring of weight are extremely important. Supplements of fat-soluble vitamins A, D, E, and K are necessary (Boyle, 2007).

Other therapeutic measures may also be necessary. Anti-inflammatory agents may also be used to treat inflammatory response in the airways. The Cystic Fibrosis Foundation states that there is insufficient evidence for the use of routine inhaled or oral corticosteroids. However, chronic use of

nonsteroidal anti-inflammatory agents is recommended to slow the loss of lung function (Flume, et al., 2007). Inhaled bronchodilators may be used in patients who have a significant bronchoconstrictive component as indicated by spirometry pre- and post-bronchodilator therapy.

Supplemental oxygen is used to treat the progressive hypoxemia that occurs with CF. It helps correct the hypoxemia and may minimize the complications seen with chronic hypoxemia (pulmonary hypertension).

Lung transplantation is an option for a small, selected population of patients with CF. A double-lung transplantation technique is used because of chronic infection of both lungs in end-stage CF. Because there is a long waiting list for lung transplants, many patients die while waiting for suitable lungs for transplantation.

Nursing Management

Nursing management is crucial to the interdisciplinary approach required for care of adults with CF. Nursing care includes helping patients manage pulmonary symptoms and prevent complications of CF. Specific measures include strategies that promote removal of pulmonary secretions; chest physiotherapy (including postural drainage, chest percussion, and vibration); and breathing exercises, which are implemented and are taught to the patient and family when the patient is very young. The patient is reminded of the need to reduce risk factors associated with respiratory infections (eg, exposure to crowds or to people with known infections). In addition, the patient is taught the early signs and symptoms of respiratory infection and disease progression that indicate the need to notify a primary health care provider.

Nurses emphasize the importance of an adequate fluid and dietary intake to promote removal of secretions and to ensure an adequate nutritional status. Because CF is a lifelong disorder, patients often have learned to modify their daily activities to accommodate their symptoms and treatment modalities. However, as the disease progresses, assessment of the home environment may be warranted to identify modifications required to address changes in the patient’s needs, increasing dyspnea and fatigue, and nonpulmonary symptoms.

As with any chronic disease, palliative care and end-of-life issues and concerns need to be addressed with the patient when warranted. For the patient whose disease is progressing and who is developing increasing hypoxemia, preferences for end-of-life care should be discussed, documented, and honored (see Chapter 17). Patients and family members require support as they face a shortened lifespan and an uncertain future.

CRITICAL THINKING EXERCISES

- 1 A 71-year-old white man presents to the clinic for follow-up of end-stage COPD and weight loss. He has been seen regularly for 5 years in the clinic, and his wife usually accompanies him. Today he is anxious and short of breath. He is receiving supplemental oxygen at 2 L/min, and his wife states he is unable to lie flat to sleep. What tests or examinations might be appropriate to assess the severity of his shortness of breath, oxygenation status, and

inability to lie flat? What information or tests would be helpful in assessing his weight loss? What are potential causes and interventions for his weight loss?

EBP 2 A 68-year-old retired businessman with COPD reports that he has been using continuous oxygen for the past 5 years. He reports increasing shortness of breath and asks you to increase his oxygen flow. How do you respond to his request? What evidence base would you use to determine your intervention? How would you determine the strength of the evidence?

EBP 3 A 35-year-old Mexican American single mother presents to the emergency department extremely short of breath. She is only able to speak in short phrases in broken English and is extremely anxious. It is noted in her chart that she has been in the hospital three times in the past year for asthma exacerbations. What are the key aspects of your physical examination, and what laboratory tests might you anticipate? What treatments may be used? What other aspects of care of this patient are important to assess and what resources may be necessary? Identify evidence-based interventions for this patient and the evidence used to select those interventions.

4 A 46-year-old woman newly diagnosed with asthma has come to your clinic for the first time. She is a smoker and has had asthmatic symptoms for the last 36 hours. Previous diagnoses have included acute bronchitis, sinusitis, and GERD. She has been prescribed a short-acting bronchodilator via inhaler and a long-acting medication with a return appointment in 1 month. What key educational topics need to be covered during this initial clinic visit? Describe an educational plan you would develop to implement at her next clinic visit.

5 A 21-year-old male college student with CF is admitted to your unit from the emergency department. He obtains his routine CF care closer to home. He has become progressively short of breath over the past week, and his roommate accompanied him to the hospital. In short and choppy phrases, the young man tells you that he is extremely short of breath; he is having paroxysms of coughing. You note the cough is productive of thick, yellow sputum. His roommate says that he has not been following his usual routine of CF care (airway clearance and nebulized medications) for the past 2 weeks. What pathophysiology is associated with these signs and symptoms? What medical and nursing interventions might be used to decrease or alleviate these signs and symptoms? What members of the health care team would you consult and why?



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

- thePoint online resource, thepoint.lww.com/Smeltzer12E
- Student CD-ROM included with the book

- *Study Guide to Accompany Brunner & Suddarth's Textbook of Medical-Surgical Nursing*
- *Handbook for Brunner & Suddarth's Textbook of Medical-Surgical Nursing*

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*Asterisk indicates nursing research.

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RESOURCES

- Agency for Healthcare Research and Quality, www.ahrq.gov or www.ahcpr.gov
- Alpha-1 Association, www.alpha1.org
- American Academy of Allergy, Asthma, and Immunology, www.aaaai.org
- American Association for Respiratory Care, www.aarc.org
- American Association of Cardiovascular and Pulmonary Rehabilitation, www.aacvpr.org
- American College of Chest Physicians, www.chestnet.org
- American Lung Association, www.lungusa.org
- American Thoracic Society, www.thoracic.org
- Centers for Disease Control and Prevention, www.cdc.gov
- Cystic Fibrosis Foundation, www.cff.org
- National Heart, Lung and Blood Institute, www.nhlbi.nih.gov
- Respiratory Nursing Society, www.respiratorynursingsociety.org
- U.S. Department of Health and Human Services, Department of Health and Human Services, www.hhs.gov or www.healthfinder.gov