Pulmonary Anatomy and Physiology and the Effects of COPD

Chronic obstructive pulmonary disease (COPD) is the fourth leading cause of death in the United States, yet it has received relatively little attention in the literature compared with other leading killers such as cancer and heart disease. To identify, assess, intervene, and care for patients with COPD in the home, a comprehensive understanding of this disease and its effects is imperative. This article discusses the epidemiology of COPD, reviews normal pulmonary anatomy and physiology, and explains the physiological changes to the pulmonary system caused by COPD.

Epidemiology

Incidence and Prevalence

Currently claiming the lives of more than 117,000 Americans annually, COPD is the fourth leading cause of death in the United States (ALA, 2003; USDHHS, 2003). Of the 10 leading causes of death in the United States, COPD is the only disease whose mortality rate is increasing (Honig & Ingram, 2002; O’Brien & Saiers, 2003). As a progressive respiratory disease, COPD is characterized by irreversible airflow obstruction resulting from chronic bronchitis and/or emphysema (Honig & Ingram, 2002; O’Brien & Saiers). These diseases often exist in combination, complicating their recognition, assessment, and management.

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By the year 2020, COPD is projected to be the third leading cause of death in the United States for both men and women (USDHHS, 2003). Although more than 16 million Americans currently have COPD, estimates indicate roughly 24 million adults have impaired lung function, suggesting that COPD is significantly underdiagnosed (USDHHS, 2003).

Overall, COPD affects men more than women, and whites are more frequently affected than are African Americans. Although mortality is increasing in both sexes, the mortality rate in females is increasing far more rapidly than in men, with female mortality rates more than doubling between the years 1980 (20.1 deaths/100,000 women) and 2000 (56.7 deaths/100,000 women) (Mannino et al., 2002; O’Brien & Saiers, 2003; USDHHS, 2003).

The cause for the rapid increase in mortality for females remains suppositional but may be a reflection of the increase in smoking by women in the United States compared with men (Mannino et al., 2002). Overall, the COPD death rate was 46% higher in males than females and 63% higher in whites than African Americans (USDHHS, 2003).

Chronic bronchitis (CB) accounts for as many as 11 million of the 16 million patients with COPD (ALA, 2003). The ALA (2003) defines CB as an “inflammation and eventual scarring of the lining of the bronchial tubes.” Females of both races had higher rates of chronic bronchitis than did their male counterparts across all age groups (USDHHS, 2003). CB is most prevalent in the 65+ age group (Honig & Ingram, 2002; USDHHS). In 2001, 3.7 million males, in contrast with 7.5 million females, had a diagnosis of CB (ALA).

Emphysema affects as many as 3 million Americans (ALA, 2003; USDHHS, 2003). Defined by Honig & Ingram (2002) as a “permanent and destructive enlargement of airspaces distal to the terminal bronchioles without obvious fibrosis and with loss of normal architecture,” emphysema is seen more commonly in men than women (p. 1). Fifty-seven percent of those with the condition are male (ALA). Gender is the only demographic on emphysema that is dissimilar to those seen with CB. As in CB, whites are affected by emphysema more frequently than their African-American counterparts, and those older than 65 years have the highest prevalence of this disease (USDHHS).

COPD's Impact
In addition to the human toll, COPD exacts an enormous economic toll on the healthcare system and society. The USDHHS (2003) and the ALA (2003) report that the annual cost to the nation for COPD in 2002 was a staggering $32.1 billion. Direct healthcare expenditures accounted for $18 billion of this total. The remaining $14.1 billion was in indirect expenses related to morbidity, disability, and premature mortality (USDHHS).

Equally stunning are the reported Medicare expenses for beneficiaries with COPD, which were 2.5 times that of the expenditures for all other patients (USDHHS, 2003). Seventy percent of COPD-related costs are accounted for by the 10% of patients with the most severe symptoms of the disease (O’Brien & Saiers, 2003).

In 2000 alone, COPD was responsible for 8 million physician office and outpatient office visits (Mannino et al., 2002). Emergency departments (ED) and hospitals also feel the effects of this disease because many patients require emergent care.

Of the 10 leading causes of death in the United States, COPD is the only disease for which the mortality rate is increasing. By the year 2020, COPD is projected to move from the fourth leading cause of death in the United States to the third for both men and women.
treatment and hospitalization during exacerbations. COPD was responsible for 1.5 million ED visits and 726,000 hospitalizations in the year 2000 (Mannino et al.).

With COPD on the rise, nurses have a pivotal role in caring for these patients in a variety of practice settings. Proper assessment, early recognition, and treatment of COPD may change the course of this disease for patients. A fundamental understanding of pulmonary anatomy and physiology is the foundation nurses must have to assess and intervene on behalf of patients with COPD.

**Pulmonary Anatomy**

This article focuses on the structure and function of the lower respiratory tract, which consists of the trachea, the left and right mainstem bronchi, the lungs, bronchioles, and alveoli. The pulmonary system's main purpose is to exchange gases between the air and the blood. This is accomplished through three processes: ventilation, diffusion, and perfusion. The pulmonary system is responsible for ventilation and diffusion, whereas the cardiovascular system is responsible for perfusion.

*Ventilation* is defined simply as “the movement of air into and out of the lungs” (McCance & Huether, 2002, p. 1082).

*Diffusion* is “the movement of gases between air spaces in the lungs and bloodstream” (McCance & Huether, 2002, p. 1082).

The trachea, which connects the larynx to the bronchi, is part of the conducting airways of the pulmonary system (Figure 1). The conducting airways are a set of “tubes” that carry air to all the areas of the lungs. The trachea divides into the left and right mainstem bronchi (McCance & Huether, 2002). The right and left mainstem bronchi enter each respective lung. Both lungs are divided into lobes: three in the right lung, and two in the left. Each lobe divides into segments and lobules.

The right and left mainstem bronchi branch off several times into smaller conducting airways, ending at the terminal bronchioles. Air is delivered to each section of the lungs through these airways. This division results in very small air-
ways in the gas exchange portions of the lungs that have decreased airflow compared with their larger counterparts. The end effect is optimal diffusion (McCance & Huether, 2002).

The gas exchange airways, consisting of the respiratory bronchioles, alveolar ducts, and alveoli, start where the conducting airways end. In these airways “oxygen (O2) enters the blood and carbon dioxide (CO2) is removed from it” (McCance & Huether, 2002, p. 1084). The alveoli are the main gas exchange components of the pulmonary system.

Pulmonary Circulation
Blood enters the pulmonary circulation from the right ventricle of the heart. The unoxygenated blood pumped from this chamber enters the pulmonary artery, which splits into the right and left pulmonary arteries (Figure 2). These two arteries transport the unoxygenated blood from the right side of the heart to each respective lung. The pulmonary arteries, much like the conducting airways, divide multiple times so that each bronchus and bronchiole has an accompanying artery or arteriole.

Arterioles divide at the terminal bronchiole to make a network of pulmonary capillaries that surround the gas exchange airways. Capillary walls often fuse with the alveolus membrane, creating the alveolocapillary membrane. Gas exchange in the blood occurs across this membrane. Any disease that thickens the alveolocapillary membrane impairs gas exchange (McCance & Huether, 2002).

Four pulmonary veins, two from each lung, carry oxygenated blood from the lungs to the left side of the heart. Each vein drains several pulmonary capillaries, and unlike the pulmonary arteries, the veins are dispersed randomly throughout the lung. The oxygenated blood is deposited into the left atrium, then the left ventricle (LV). The LV then
pumps the oxygenated blood out into the aorta, which delivers it into systemic circulation.

**Ventilation and Gas Exchange**

“Ventilation is the mechanical movement of gas or air into and out of the lungs” (McCance & Huether, 2002, p. 1088). A patient’s respiratory rate is his or her ventilatory rate. With each breath in, oxygen is inhaled, and upon exhalation CO₂ is eliminated. This process is essential to maintain a normal acid base balance. The only definitive way to assess adequacy of ventilation is through obtaining an arterial blood gas to measure the arterial CO₂ (PaCO₂) level. Normal is 40 mm Hg.

Gas exchange has four basic steps:

- Ventilation,
- Diffusion of O₂ across the alveolocapillary membrane,
- Perfusion of the systemic capillaries with oxygenated blood, and
- Diffusion of O₂ from the capillaries to the cells (McCance & Huether, 2002).

When referring to CO₂ removal, the steps are reversed:

- Diffusion of CO₂ from the cells into the systemic capillaries,
- Pulmonary capillary perfusion by unoxygenated venous blood,
- Diffusion of CO₂ into the alveoli, and
- Removal of CO₂ by the exhalation phase of ventilation (McCance & Huether, 2002).

CO₂ must be eliminated on a continual basis to maintain the body’s acid base balance. Acid base balance is monitored within the body by chemoreceptors. Chemoreceptors located near the body’s respiratory center are sensitive to changes in the pH of cerebrospinal fluid (CSF) (McCance & Huether, 2002). When there is *inadequate* ventilation, the pH drops and PaCO₂ rises. This stimulates the respiratory center to increase the size of each breath and the respiratory rate to remove the CO₂. These receptors are sensitive to small changes in pH.

If hypoventilation is chronic, as may be the case in patients with COPD, chemoreceptors lose their sensitivity and control PaCO₂ inadequately. In addition, prolonged hypoventilation can result in compensation by the renal system, with the kidneys retaining bicarbonate to normalize the body’s pH. This deletes the effect a low pH, as a result of a high PaCO₂, has on the respiratory drive (McCance & Huether, 2002).

When central chemoreceptors fail, peripheral chemoreceptors step in to regulate pulmonary function and acid base balance. Peripheral chemoreceptors are sensitive to the amount of oxygen in arterial blood (PaO₂). Therefore, the patient’s stimulus to breathe is originated from a low PaO₂ sensed by the peripheral receptors.

*If the PaO₂ is increased too significantly by instituting supplemental O₂, the peripheral chemoreceptors will not stimulate breathing. This creates a rise in PaCO₂, ultimately resulting in apnea (McCance & Huether, 2002). A PaO₂ of 60 is optimal. If adequate oxygenation, as reflected by arterial blood gases (ABG) monitoring, cannot be achieved without loss of respiratory drive, mechanical ventilation must be instituted.*

**COPD**

COPD is characterized by airflow limitation that is not fully reversible (Snider, 2003). The mechanisms of airflow limitation are different in both CB and emphysema.

In CB, airflow is limited because of inflammation and thickening of bronchial walls. This inflammation is caused by exposure to inhaled irritants, such as cigarette smoke (McCance & Huether, 2002). The inflammation causes increased sputum production, which often becomes a source of infection (McCance & Huether).

Airflow limitation in emphysema is caused by actual structural remodeling in lung tissue in comparison to the solely inflammatory changes seen in CB. In emphysema, gas exchange airways are permanently enlarged, which results in the destruction of the alveolar walls without fibrosis.
being present (McCance & Huether, 2002). Emphysema has two main causes: an inherited enzyme deficiency (1%–2% of cases) or injury to the lung from inhaled toxins (McCance & Huether).

The diagnosis of COPD may be difficult in the early stages of the illness and may be misdiagnosed as chronic bronchitis, emphysema, or asthma. Accurate diagnosis of COPD is based on patient history, signs and symptoms, physical examination, chest radiography, ABG, and pulmonary function tests (PFTs).

**Signs and Symptoms of COPD**

**Early COPD**

In the early phase of COPD, patients may begin to experience wheezing, chronic productive cough, and minimal shortness of breath. Patients may not be aware of these subtle symptoms (Snider, 2003). However, the patient’s quality of life diminishes as the disease progresses. Symptoms include worsening dyspnea, progressive exercise intolerance, intermittent chest illness (occurring with greater frequency), increased cough, purulent sputum, with subsequent increased intervals and severity of acute exacerbations (Table 1) (Kleinschmidt, 2004; Snider).

**Acute Phase of COPD**

In the acute phases of the illness, exacerbations cause hypoxemia and hypercapnia. Hypoxemia in patients with COPD is most commonly caused by hypoventilation (McCance & Huether, 2002). Hypercapnia, defined as “increased carbon dioxide in the arterial blood” (McCance & Huether, 2002, p. 1108) is caused by hypoventilation of the alveoli.

Patients notice a significant decline in their quality of life during exacerbation. Supplemental oxygen or mechanical ventilatory support may be required during these phases (ALA, 2003). Careful assessment of the patient is essential when supplemental O2 is used because of the risk of respiratory depression discussed earlier.

**Physical Examination**

The physical examination of a patient with COPD also changes with disease progression. In the early phase of the disease, the patient may demonstrate:

- Prolonged expiration,
- Wheezing on forced exhalation (Snider, 2003),
- Chronic cough, which may be present with varying degrees of sputum production, and
- “Barrel chest.”

As airflow obstruction progresses because of inflammation (in CB) or structural damage to airway walls (emphysema), air gets trapped in the lungs, making them hyperinflated (McCance & Huether, 2002; Snider, 2003). The prolonged, forced expiration seen in patients with COPD is a result of the body trying to fully exhale the trapped gases. Hyperinflation eventually causes chest wall deformity, described as “barrel chest.”

| Table 1. Signs and Symptoms of Chronic Bronchitis, Emphysema, and COPD |
|--------------------------|-----------------|-----------------|
| **Chronic Bronchitis**    | **Emphysema**   | **COPD**        |
| Airflow obstruction       | Yes             | Yes             | Yes (as a result of inflammation and destruction) |
| Wheezing                  | Occasional      | Minimal         | Occasional               |
| Dyspnea                   | Late in disease course | Yes            | Yes (worsening later in course) |
| Productive cough          | Yes (classic sign) | Occasional (later in disease course) | Yes (chronic) |
| Exercise intolerance       | Yes             | Yes             | Yes                       |
| Prolonged expiration      | Yes             | Yes             | Yes                       |
| Barrel chest              | Occasional      | Yes (classic sign) | Occasional          |

*Note. COPD = chronic obstructive pulmonary disease.*
Late in the disease:

- Accessory muscles will be used to breathe,
- Purse-lip breathing may be evident,
- Breath sounds become decreased,
- Patient will sit in tripod position to relieve dyspnea,
- Coarse crackles and wheezing often can be auscultated,
- Heart sounds are distant,
- Evidence of right-sided heart failure develops, including:
  - Edema
  - An enlarged, tender liver (Kleinschmidt, 2004; Snider, 2003).

Right-sided heart failure develops because of increased resistance to flow in the lungs caused by the narrowing and/or obstruction of the pulmonary arteries caused by COPD (Kleinschmidt, 2004; Snider, 2003). Pulmonary hypertension may also occur in the late phase of COPD secondary to severe hypoxemia and hypercapnia. It is associated with right-sided heart failure and indicative of a poor prognosis (National Heart, Lung and Blood Institute, National Institutes of Health & the World Health Organization, 1998).

Diagnosis

Chest radiography is a helpful tool in diagnosing COPD. Flattening of the diaphragm and overdistention of the lungs can be seen (Snider, 2003). ABG analysis provides information regarding the severity and progression of the disease. ABG results show mild to moderate hypoxemia in the early stages of the disease. Hypoxemia gradually becomes more severe. Eventually, hypoxemia and hypercapnia are seen together (Snider, 2003). Blood gas abnormalities worsen with disease progression and acute exacerbation and may worsen during exercise and sleep (Snider). A pH of 7.3 or less is indicative of acute respiratory compromise (Kleinschmidt, 2004).

Lastly, PFTs provide important information that is necessary in diagnosing and monitoring the progression of COPD (Snider, 2003). PFTs provide information about the functional quality of the lung. The values most important in diagnosing COPD are the forced expiratory volume in 1 second (FEV\(_1\)), the forced vital capacity (FVC), the total lung capacity (TLC), and the residual volume (RV). Patients with COPD have an increased or normal TLC and FVC, with an increase in RV (Kleinschmidt, 2004). This is a result of the progressive airflow obstruction.

The FEV\(_1\) and the FEV\(_1\)/FVC ratio decrease progressively as the disease worsens, further demonstrating lung damage (Snider, 2003). The FEV\(_1\) is considered to be the gold standard in diagnosing airflow obstruction because of its accuracy in measuring airway dynamics (Snider). Patients with COPD demonstrate poor or absent reversibility with bronchodilator therapy during PFT (Kleinschmidt, 2004).

The focus of treatment and management of this disease must be placed on stopping its progression through eliminating exposure to causative agents and limiting acute “exacerbations” of the disease by careful monitoring for, and treatment of, infection.

It is imperative to remember that COPD itself is absolutely irreversible. The focus of treatment and management of this disease must be placed on stopping its progression through eliminating exposure to causative agents and limiting acute “exacerbations” of the disease by careful monitoring for, and treatment of, infection. Pharmacologic maintenance with bronchodilators will help maintain the patient’s functional status and ultimately reduce the number of acute episodes they experience.

Conclusion

COPD is a formidable health threat for many Americans, with the numbers affected anticipated to rise. A clear understanding of the pulmonary system, COPD, and its progression and treatment are essential for home health nurses, who will be providing care for more of these patients in their homes. This is a disease that greatly affects a patient’s quality of life. Helping to maintain the patient’s health and limit acute exacerbations will help preserve the patient’s functional abilities and subsequently quality of life.
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REFERENCES
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LEARNING OBJECTIVES
After reading this article and taking this test, you will be able to:

1. Of the ten leading causes of death in the United States, COPD is the only disease
   a. that is completely preventable.
   b. whose overall incidence has decreased.
   c. that is totally irreversible.
   d. whose mortality rate is increasing.

2. The single most common risk factor for COPD is
   a. occupational exposure.
   b. cigarette smoking.
   c. genetic predisposition.
   d. impaired immunity.

3. Chronic bronchitis is especially prevalent among
   a. older adults.
   b. males.
   c. African Americans.
   d. children.

4. A classic manifestation of emphysema is
   a. wheezing.
   b. productive cough.
   c. crackles.
   d. barrel chest.

5. A classic manifestation of chronic bronchitis is
   a. wheezing.
   b. productive cough.
   c. dyspnea.
   d. barrel chest.

6. The movement of gases between air spaces in the lungs and bloodstream is called
   a. ventilation.
   b. aeration.
   c. diffusion.
   d. perfusion.

7. The main gas exchange components of the pulmonary system are the
   a. alveoli.
   b. respiratory bronchioles.
   c. bronchi.
   d. terminal bronchioles.

8. Unoxygenated blood is transported from the right side of the heart to each lung by the
   a. pulmonary veins.
   b. right ventricle.
   c. pulmonary arteries.
   d. aorta.

9. The first step of the removal of carbon dioxide (CO₂) from the blood is
   a. pulmonary capillary perfusion by unoxygenated venous blood.
   b. diffusion of CO₂ into each lung’s alveoli.
   c. perfusion of the systemic capillaries with oxygenated blood.
   d. diffusion of CO₂ from the cells into the systemic capillaries.

10. With inadequate ventilation
    a. pH drops and PaCO₂ rises.
    b. pH and PaCO₂ both rise.
    c. pH rises and PaCO₂ drops.
    d. pH and PaCO₂ both drop.

11. With emphysema, airflow is limited primarily by
    a. extensive fibrosis.
    b. persistent inflammation.
    c. structurally remodeled lung tissue.
    d. thickened bronchial walls.
12. An early sign/symptom of COPD is
   a. exercise intolerance.
   b. prolonged expiration.
   c. intermittent chest illness.
   d. copious purulent sputum.

13. Hypoxemia in COPD patients is most often caused by
   a. persistent cough.
   b. infection.
   c. hyperinflation.
   d. hypoventilation.

14. Patients who have advanced COPD tend to assume which position to facilitate breathing?
   a. tripod
   b. semi-Fowler’s
   c. side-lying
   d. supine

15. An especially poor prognostic sign in patients who have COPD is
   a. right-sided heart failure.
   b. pulmonary hypertension.
   c. wheezing on forced exhalation.
   d. moderate dyspnea.

16. As COPD worsens, which pulmonary function value decreases progressively?
   a. forced vital capacity
   b. total lung capacity
   c. forced expiratory volume in one second
   d. residual volume

Test Answers:
[Blank spaces for answers to each question are provided.]

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