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Chronic Obstructive Pulmonary Disease
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Introduction
Chronic Obstructive Pulmonary Disease (COPD) is a gradually progressive disease that affects the airways or pulmonary parenchyma, or both, and results in airflow obstruction (Gaseem et al. 2011). COPD is a set of diseases that limit airflow, cause dyspnea, decrease exercise capacity, and include both emphysema and chronic bronchitis (CDC, 2017). “In the United States, COPD affects more than 5% of the adult population; it is the third leading cause of death and the 12th leading cause of morbidity. The total economic cost of COPD in the United States was estimated to be $49.9 billion in 2010, and the cost of COPD in the United States were estimated to be $195 billion in 2017” (Mitchell, 2015).

Risk Factors, Signs & Symptoms
According to the World Health Organization (WHO, 2017) risk factors associated with COPD include:

- Smoking (the primary cause of COPD).
- Recurrent lower respiratory tract infections.
- Indoor air pollution:
  - Occupational dusts and chemicals
- Age
- Genetic susceptibility
- Air pollution

Underlying Pathophysiology
A characteristic of COPD is the on-going inflammation affecting central airways, peripheral airways, lung parenchyma and the alveoli, as well as the pulmonary vasculature (Mitchell, 2015). These pathophysiological characteristics combined result in narrowing and remodeling of the airways, an augmentation of goblet cells, amplification of the mucus-secreting glands of the central airways and subsequent vascular bed changes leading to pulmonary hypertension (Mitchell, 2015).

The primary feature of COPD is airflow limitation that is not fully reversible (figure 1). For those with emphysema, the smooth muscle in the airways becomes thickened and constricted which results in a narrowing of the airway. These narrowed airways are prone to collapse, particularly during expiration (Mitchell, 2015).

In emphysema, the surface area for gaseous exchange is severely reduced, resulting in the damage of pulmonary capillaries which restricts the diffusion of gases, resulting in a mismatch between ventilation, and perfusion (Mitchell, 2015).

According to Mitchell (2015), gas exchange (known as external respirations) takes place at the extremely thin alveolar capillary membranes by diffusion. Oxygen moves from the alveoli into the pulmonary capillaries as shown in figure 2; therefore blood leaving the lungs to go to the pulmonary veins and the left side of the heart should be saturated with oxygen and have a high CO2 to be exhaled and eliminated by the body (p.445); Alveolar dead space (dead space ventilation) takes place when air remains in the conducting airways and is unable to reach the alveoli, due to alveolar damage. This then, is air that does not take part in alveolar ventilation (Mitchell, 2015).

Dirldication of smooth bronchial muscle (bronchoconstriction), in addition to limitation in airflow from inflammation and mucus production, attribute to noisy musical sounds known as wheeze. This occurs mainly with expiration (Mitchell, 2015). Over secretion of mucus along with ciliary dysfunction leads to the chronic cough and sputum production most COPD patients experience (Mitchell, 2015).

Coughing is initiated by irritant receptors in response to chemical, inflammatory, or mechanical stimuli and is a defense mechanism to protect our airways. Coughing results in bronchoconstriction and hypersecretion of mucus. Chronic cough due to COPD manifests as prolonged bouts of productive coughing to mucous-secreting goblet cells in the mucous glands of the bronchial wall. Fatigue is a factor in ineffective coughing with COPD patients as well (Mitchell, 2015).

Dyspnea is the most prominent symptom in COPD patients and is typically the reason the patient seeks medical help (Mitchell, 2015). Changes in respiratory rate, pattern, depth, and degree of shortness of breath are challenging for the APRN to assess because of the chronic nature of the disease as well as the subjective experience of dyspnea.

Significance of Pathophysiology
Gas exchange at the alveoli changes over time in some COPD patients; this is due to the destruction of the alveolar-capillary barrier (Mitchell, 2015). Mitchell (2015) further discusses that this change has the body’s protective mechanism in an attempt to maintain the gas exchange and this is called hypoxic pulmonary vasoconstriction. This causes the pulmonary arteries to narrow because of low oxygen levels, diverting blood away from the poorly ventilated alveoli (p. 446).

When alveoli are unventilated (airless), full of fluid or infection (debris), or low in oxygen, blood is diverted within the pulmonary capillaries to more ventilated alveoli with a higher level of oxygen to limit what is termed ‘shunting’ (low blood oxygen levels) resulting in blood circulating alveolus and returning to the left heart vessels un oxygenated (hypoxemia) (Mitchell, 2015). Hypoxemia results when the oxygen levels within the alveoli are low, whereas hypoxia results when there is low oxygen levels at the cellular tissue level (Peate & Dutton, 2014, Ch. 5).

Implications for Nursing Care
COPD is a disabling disease. Repetitive exacerbations result in a higher use of our health resources, especially among those patients with advanced COPD and co-morbidities (Bourbeau & Saad, 2012). Bourbeau & Saad (2012) point out that the sooner the patient with COPD can learn and understand the disease process; the better it will be for them. They discuss ways to achieve this mastery is by implementing self-management programs while following fundamental principles with respect to the patients’ needs and abilities (p. 99). To achieve this, Bourbeau & Saad (2012) propose: identify, teach, support, follow-up, and assess. This model provides a plan for fragmentation of information and adds to the patient’s knowledge and understanding of the disease. It is an important tool for enhancing self-management and patient empowerment. It allows the patient to become more informed about COPD and teach them how to maintain the disease at home. To achieve this, the patient must assume healthy behaviors and many of the unfortunate outcomes related to chronic disease can be avoided (Bourbeau & Saad, 2012).

Conclusion
Current guidelines recommend screening spirometry in smokers and those patients at risk for COPD as COPD is a common disease and morbidity and reduced life expectancy associated with COPD (Ebeheyb, Webb, Neder & O’Donnell, 2013). Smoking cessation is a pivotal intervention and is known to favorably change the natural course of COPD (Ebeheyb, Webb, Neder & O’Donnell, 2013). The care giver must use best clinical judgement regarding treatment options on a case-by-case basis for these patients. Primary care nurses (APRNs) are ideally placed to provide the ongoing education and close monitoring to recognize symptoms and act on them early and accordingly.

References
Qaseem, A., Wilt T.J., Weinberger S. E., Hanania N. A., Criqui, D. Van Der Molen, T., & Shekelle, P. (2011). Diagnosis: an action plan in the event of an exacerbation and an integrated health-care system coordinated by a case manager for educational sessions and regular communication reinforces the premise that if the patient’s confidence, that is, self confidence and self-esteem is an important tool in the patient’s self-care plan, they can be empowered to assume healthy behaviors and many of the unfortunate outcomes related to chronic disease can be avoided (Bourbeau & Saad, 2012).