

Chronic Obstructive Pulmonary Disease

Background: Chronic obstructive pulmonary disease (COPD) is estimated to affect 32 million persons in the United States and is the fourth leading cause of death in this country. Patients typically have symptoms of both chronic bronchitis and emphysema, but the classic triad also includes asthma. Most of the time COPD is secondary to tobacco abuse, although cystic fibrosis, alpha-1 antitrypsin deficiency, bronchiectasis, and some rare forms of bullous lung diseases may be causes as well.

Patients with COPD are susceptible to many insults that can lead rapidly to an acute deterioration superimposed on chronic disease. Quick and accurate recognition of these patients along with aggressive and prompt intervention may be the only action that prevents frank respiratory failure.

Pathophysiology: COPD is a mixture of 3 separate disease processes that together form the complete clinical and pathophysiological picture. These processes are chronic bronchitis, emphysema and, to a lesser extent, asthma. Each case of COPD is unique in the blend of processes; however, 2 main types of the disease are recognized.

Chronic bronchitis

In this type, chronic bronchitis plays the major role. Chronic bronchitis is defined by excessive mucus production with airway obstruction and notable hyperplasia of mucus-producing glands.

Damage to the endothelium impairs the mucociliary response that clears bacteria and mucus. Inflammation and secretions provide the obstructive component of chronic bronchitis. In contrast to emphysema, chronic bronchitis is associated with a relatively undamaged pulmonary capillary bed. Emphysema is present to a variable degree but usually is centrilobular rather than panlobular. The body responds by decreasing ventilation and increasing cardiac output. This V/Q mismatch results in rapid circulation in a poorly ventilated lung, leading to hypoxemia and polycythemia.

Eventually, hypercapnia and respiratory acidosis develop, leading to pulmonary artery vasoconstriction and cor pulmonale. With the ensuing hypoxemia, polycythemia, and increased CO₂ retention, these patients have signs of right heart failure and are known as "blue bloaters."

Emphysema

The second major type is that in which emphysema is the primary underlying process. Emphysema is defined by destruction of airways distal to the terminal bronchiole.

Physiology of emphysema involves gradual destruction of alveolar septae and of the pulmonary capillary bed, leading to decreased ability to oxygenate blood. The body compensates with lowered cardiac output and hyperventilation. This V/Q mismatch results in relatively limited blood flow through a fairly well oxygenated lung with normal blood gases and pressures in the lung, in contrast to the situation in blue bloaters. Because of low cardiac output, however, the rest of the body suffers from tissue hypoxia and pulmonary cachexia. Eventually, these patients develop muscle wasting and weight loss and are identified as "pink puffers."

Frequency:

- **In the US:** Two thirds of men and one fourth of women have emphysema at death. Approximately 8 million people have chronic bronchitis and 2 million have emphysema.

Mortality/Morbidity: COPD is the fourth leading cause of death in the United States, affecting 32 million adults.

Sex: Men are more likely to have COPD than women.

Age: COPD occurs predominantly in individuals older than 40 years.

History: Patients with COPD present with a combination of signs and symptoms of chronic bronchitis, emphysema, and asthma. Symptoms include worsening dyspnea, progressive exercise intolerance, and alteration in mental status. In addition, some important clinical and historical differences can exist between the types of COPD.

- In the chronic bronchitis group, classic symptoms include the following:
 - Productive cough, with progression over time to intermittent dyspnea

- Frequent and recurrent pulmonary infections
- Progressive cardiac/respiratory failure over time, with edema and weight gain
- In the emphysema group, the history is somewhat different and may include the following set of classic symptoms:
 - A long history of progressive dyspnea with late onset of nonproductive cough
 - Occasional mucopurulent relapses
 - Eventual cachexia and respiratory failure

Physical: Depending on the type of COPD, physical examination may vary.

- Chronic bronchitis (blue bloaters)
 - Patients may be obese.
 - Frequent cough and expectoration are typical.
 - Use of accessory muscles of respiration is common.
 - Coarse rhonchi and wheezing may be heard on auscultation.
 - Patients may have signs of right heart failure (ie, cor pulmonale), such as edema and cyanosis.
 - Because they share many of the same physical signs, COPD may be difficult to distinguish from CHF. One crude bedside test for distinguishing COPD from CHF is peak expiratory flow. If patients blow 150-200 mL or less, they are probably having a COPD exacerbation; higher flows indicate a probable CHF exacerbation.
- Emphysema (pink puffers)
 - Patients may be very thin with a barrel chest.
 - They typically have little or no cough or expectoration.
 - Breathing may be assisted by pursed lips and use of accessory respiratory muscles; they may adopt the tripod sitting position.

- The chest may be hyperresonant, and wheezing may be heard; heart sounds are very distant.
- Overall appearance is more like classic COPD exacerbation.

Causes: In general, the vast majority of COPD cases are the direct result of tobacco abuse. While other causes are known, such as alpha-1 antitrypsin deficiency, cystic fibrosis, air pollution, occupational exposure (eg, firefighters), and bronchiectasis, this is a disease process that is somewhat unique in its direct correlation to a human activity

Prehospital Care: The mainstays of therapy for acute exacerbations of COPD are oxygen, bronchodilators, and definitive airway management.

- Oxygen
 - Adequate oxygen should be given to relieve hypoxia. A belief (ingrained from medical school) is held widely that too much oxygen causes significant respiratory depression. Multiple studies in the literature dispute this view. With administration of oxygen, PO_2 and PCO_2 rise but not in proportion to the very minor changes in respiratory drive.
 - The need for intubation can be established quickly at the bedside by asking the patient hold the nebulizer in his or her hand. If the patient becomes so sleepy that the nebulizer starts to fall away, the patient should be intubated regardless of PCO_2 level. The cause of increased CO_2 production is not decreased respiratory drive but probably reversal of hypoxic arterial vasoconstriction in areas of less-ventilated lung tissue, which increases the extent of ventilation/perfusion defects and thus CO_2 . "Stated another way, there is probably no single value for arterial PCO_2 , pH, or PO_2 that by itself constitutes an indication for [intermittent positive pressure ventilation (IPPV)]" (Pierson, 2002)
 - Occasionally, large increases in CO_2 can lead to deterioration of mental status, causing stupor and obtundation. In such cases, decreasing O_2 delivery is the wrong action. The CO_2 narcosis inhibits respiratory drive to the point that decreasing O_2 delivery leads only to worsening of hypoxia. The correct action is immediate intubation and oxygenation.

- Supply the patient with enough oxygen to maintain a near normal saturation (above 90%) and do not be concerned about oxygen supplementation leading to clinical deterioration. If the patient's condition is that tenuous, intubation most likely is needed anyway.
- Bronchodilator
 - In the prehospital setting, administer only beta-agonist nebulizer therapy, which should be given as needed.
 - If necessary and available, continuous positive airway pressure (CPAP) may be used.
 - Of course, in times of respiratory failure, patients may need intubation in the field.

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