RESPIRATORY PATHOPHYSIOLOGY

Chronic Obstructive Pulmonary Disease (COPD)

Read Chapter 30 in your textbook concentrating on asthma, chronic bronchitis, and emphysema before continuing with this lecture.

COPD’s are just as their name implies, chronic obstructive pulmonary diseases. They are chronic because they usually develop over an extended period of time. They are obstructive because they all, in some way, block lung airways. Three major COPD’s are encountered in clinical situations: (1) Asthma; (2) Chronic Bronchitis; (3) and Emphysema. Since the etiology of each is somewhat different, they will be considered separately.

Asthma

Asthma is a reversible bronchiolar airway constriction very often associated with allergies, but not always. In some people, asthma attacks can be triggered by exertion or excitement as well. In asthma, bronchiolar smooth muscle contracts uncontrollably and, quickly producing “wheezing” upon auscultation of breath sounds. In allergic individuals, the bronchiolar constriction is the result of histamine release in the lungs in response to an allergen.

Because bronchioles constrict, asthma causes difficulty with exhalation. Recall that during inspiration, the elastic lung airways are literally “pulled” open as the lungs expand allowing air to enter relatively unobstructed. However, in response to histamine release, the airways swell and secrete thick mucus. These events, when combined with smooth muscle contraction, cause the airways to close completely during exhalation making it difficult to expire the inhaled air. This “trapped air” remains in the lungs raising the FRV and making it more difficult to inhale sufficient air with the next breath. So, as trapped air volume increases, the inspiratory reserve decreases accordingly making it difficult to “turn over” enough of the FRV to maintain blood oxygenation and carbon dioxide removal. In extreme cases, suffocation can result.

Since the bronchiolar constriction is reversible, treatment with a bronchodilator (epinephrine in extreme cases) gives relief or the constriction may subside on its own in time.
**Chronic Bronchitis**

Somewhat like asthma, in chronic bronchitis the conducting airways are inflamed chronically by an irritant and swell with heavy mucus secretion. These inflamed airways close during exhalation causing air trapping. In many cases the air trapping is sufficient to cause a “barrel chested” appearance. Because of the poor lung ventilation, clients are often hypoxic with cyanosis and hypercapnic. The hypercapnea is particularly serious since it develops over a long time and the respiratory centers of the brain adapt to the high carbon dioxide levels to the extent that these clients depend upon their “back up” system to trigger breathing. This back up system relies on low oxygen to stimulate breathing. **Consequently, it is extremely dangerous to administer high amounts of oxygen to these clients even though the cyanosis might indicate it. Since they are using low oxygen to stimulate breathing, too much oxygen can result in respiratory suppression.**

The hyperinflated lungs of these clients increases resistance to blood flow through the pulmonary capillaries on the surfaces of the distended alveoli. This can lead to **pulmonary artery hypertension (PAH)** and eventually to **cor pulmonale (right CHF)**.

**Emphysema**

It is important to note that “pure” emphysema is rarely encountered. It usually occurs in conjunction with bronchitis with irritant particles being the cause of both. Should emphysema occur singularly, it results in a loss of respiratory surface instead of airway obstruction. In fact, in emphysemas lung volume may stay the same or increase but the loss of respiratory membrane surface impairs gas exchange. These individuals may even show diminished breath sounds as airways are lost or fused together. Since capillaries occur on alveolar surfaces, they too may be lost as the disease progresses. This can also increase resistance causing PAH and right CHF.

Since respiratory surface is lost, compensation of gas exchange is possible. Emphysems frequently use “pursed lip breathing” as a compensation. By exhaling against pursed lips, airway pressures are increased above normal and this helps prevent their collapse during exhalation. It also raises the air pressure in remaining alveoli literally driving more oxygen into blood.

**Other Respiratory Disorders**

Look in your textbook at the table of restrictive respiratory disorders (Table 29-1, p - 570). Examine the causes and clinical manifestations of each of the following disorders and be certain that you can explain the physiological perturbations underlying each. Follow the examples used above with the COPD’s.

1. Neuromuscular
2. Traumatic Chest Wall Instability
3. Pleural Disorders
4. Tuberculosis
5. Atelectasis
6. Pulmonary Edema

If you have problems with any of the disorders, post them to the Discussion Group so all may participate.

REVIEW QUESTIONS:

1. During an asthma attack, which lung volumes increase? Decrease? Why?
2. Why might emphysems be described as “pink puffers?”
3. Why might chronic bronchitis clients be described as “blue bloaters?”
4. What cardiovascular problems might occur if COPD is not corrected? Why?
5. What blood chemistry changes might be seen with chronic bronchitis?

DISCUSSION QUESTIONS: (Post answers to the “Patho Discussion Group”)

1. Why might emphysems be described as “pink puffers” and chronic bronchitis clients be described as “blue bloaters”?
2. Describe how TV, FRV, and FEV would change with COPD.