

LECTURE 9

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RESPIRATORY PATHOPHYSIOLOGY / REVIEW

Read Chapter 28 in your textbook before proceeding.

Respiratory Gas Exchange via Diffusion

Basic to an understanding of respiratory physiology and pathophysiology is realizing the importance of diffusion in the process of gas exchange between alveolar air and pulmonary capillary blood. The following equation summarizes the factors that affect the rate of diffusion and can affect the rate of gas exchange in the lungs:

$$\text{Diffusion Rate} = \frac{[\text{Conc. Gradient}] \times \text{Temp.} \times \text{Surface Area}}{\text{Molecular Size} \times \text{Distance}}$$

Notice that increases or decreases in any variable in the numerator directly cause corresponding increases or decreases in diffusion rate. Similar changes in denominator values have the opposite, or inverse, effect; increases decrease diffusion rate and vice versa. Molecular size can not be changed to affect diffusion rate and temperature changes dramatic enough to have an impact on diffusion would not be compatible with life. So, these variables can be eliminated from consideration. Normally distances across which gases diffuse are kept small to insure maximal rate of diffusion. The walls of capillaries and alveoli are comprised of thin, simple squamous epithelium. But, certain circumstances can change distance. For example, filling the alveoli with fluid adds a fluid layer to the thickness of the respiratory membrane and slows diffusion accordingly. Destroying alveolar surface area, as in emphysema, would also slow diffusion. Under normal circumstances in a normal human, changes in concentration are the only ways to change diffusion rates. Since concentration gradients are extremely important for diffusion in the lungs, lung ventilation is necessary to insure that oxygen and carbon dioxide concentration gradients are kept sufficiently steep for adequate diffusion.

REVIEW QUESTIONS:

1. How do oxygen tents or nasal prongs (cannulas) work for hypoxic clients?
2. What is Kusmal's respiration? What does it accomplish?

Diffusion and the Respiratory Membrane

The lung surface across which gases are exchanged is termed the **Respiratory Membrane** and consists of the alveolar wall, the pulmonary capillary wall and the interstitial space that separates them. The membrane walls of the alveoli and capillaries are extremely thin and the interstitial fluid filtration in pulmonary capillaries is reduced compared to systemic capillaries to minimize fluid accumulation. These factors keep optimal physical conditions for gas diffusion across the respiratory membrane. Thus, changes in any of these factors can lead to a pathophysiological disruption of gas diffusion leading to **hypoxia** (low blood oxygen with **cyanosis**) and **hypercapnea** (elevated blood carbon dioxide).

For instructional purposes, the respiratory membrane can be considered to have an “air side” within the alveoli and a “blood side” within the pulmonary capillaries. For gases to be exchanged adequately between these two compartments, concentration differences must be maintained by **ventilation** of lung airways and blood flow (**perfusion**) in pulmonary capillaries. Freshening the alveolar air through breathing in atmospheric air keeps oxygen concentrations high and carbon dioxide low on the “air side.” Pumping blood returning from systemic circulation through the lungs keeps oxygen low and carbon dioxide high on the “blood side.” When these two compartments are brought close together across the thin respiratory membrane, gases are exchanged readily by diffusion along these concentration gradients.

Ventilation of the “Air Side”

Lung airways can be divided into “**Conducting**” airways and “**Respiratory**” airways depending upon the presence of gas exchange. Gas exchange occurs only in respiratory airways and not in conducting airways. **Recall the anatomy of the bronchial trees that comprise each lung.** These conducting airways branch into progressively smaller and smaller diameter passageways offering more and more resistance to air flow (air is a fluid and has the same flow properties as blood). Over 20 levels of branching occur in the bronchial tree and airflow becomes slower as the airway diameter gets smaller. At the level of the **terminal bronchiole**, airflow virtually slows to a halt and alveolar ventilation (respiratory airways) further downstream occurs in part by diffusion. As the airflow slows, small particulates in air settle out in the small diameter airways. These particles cannot be removed by coughing and have to be ingested by phagocytic white blood cells. Over ingestion by these blood cells results in their deaths and release of lysosomal proteases, especially **trypsin**. Trypsin is nonspecific and can attack the lung airway structural proteins resulting in loss of surface and large cavities appearing in the lungs, a condition termed **emphysema**. Notice that the risk of emphysema increases with increased particulates in inspired air as might occur with smoking or atmospheric pollution. Protection from trypsin damage is inherited in the form of Alpha 1, Antitrypsin (AAT). People with high AAT levels can endure higher levels of inspired particulates.

Ventilation of the conducting airways is accomplished by air flow along **pressure gradients**. Air is fluid and will flow from a high pressure area into a low pressure area. So, ventilating the conducting airways requires lowering their pressure relative to outside air during inspiration and reversing the pressure differences during expiration. Since lungs are elastic and occur in a thoracic cavity that is isolated from outside air, expanding the thorax pulls the lungs open (lowering the airway pressure) and compressing it compresses the lungs (raising the airway pressure). When airway pressure drops below atmospheric, air rushes in (**inspiration**) and when it increases above atmospheric, air rushes out (**expiration**). The space between the lung surface and the inner chest wall (**intrapleural space**) has a negative air pressure (partial vacuum) to keep the lungs expanded and airways open between breaths. Loss of this negative pressure in the thoracic cavity can result in lung collapse. This is common in chest wounds that perforate the chest wall or punctured lungs that allow air to escape from the lung airways into the thoracic cavity.

Ventilation of the respiratory airways, including the alveoli, is directly related to ventilation of the conducting airways. If conducting airway air is freshened by breathing, diffusion gradients are maintained that help move gases into and out of respiratory airways. Also, the lung movements during breathing help to draw some air into and out of the alveoli. So, the breathing activities help maintain diffusion gradients to insure gas exchange at all levels.

Review in your textbook (p.566) the various lung volumes and be sure you understand the specifics of each volume. Don't memorize the average quantities of each volume, but do examine their interrelationships. Pay particular attention to the "Functional Residual Volume" and the "Residual Volume" and speculate how these might change with respiratory disorders.

If lung volumes are reexamined, Tidal Volume deserves more attention. By definition, tidal volume is the amount of lung air turned over with each normal, quiet breath. It averages about 500 ml. However, conducting airways will still contain some unexpired air at the end of each respiratory cycle ("**dead space air**"). This air is the first to reenter the lungs with the next breath. Since this "unexpired air", about 200 ml, is not fresh air, it must be subtracted from the tidal volume leaving only about 300 ml of fresh air entering the lungs with each breath. From this value, "**Alveolar Ventilation Volume**" (volume of fresh air entering the lungs with each breath) can be calculated:

Alveolar Ventilation = 300 ml x 17 (average breaths per minute)

Alveolar Ventilation = 5,100 ml or about 5 L (fresh air entering lungs each minute)

Since the "Functional Residual Volume" (FRV) is not exhaled and remains in the lungs to oxygenate blood between breaths, an interesting relationship can be seen. The lungs don't empty between breaths. Instead, a fraction of the FRV is turned over with each breath. Actually, about 1/7 of the FRV is replaced with each breath meaning that

about 15 or so breaths are required to completely replace the FRV. Comparing that with minute volume shows the FRV to be completely freshened about once each minute in normal quiet breathing. Note that increasing the rate and/or depth of breathing would replace the FRV more quickly. So some compensatory flexibility is possible to correct for increased gas exchange demand due to increased activity or lung damage, but there are limits beyond which the act of breathing becomes too strenuous to be sustained.

REVIEW QUESTIONS:

1. What is atelectasis and what effect would it have on Residual Volume?
2. What is the Forced Expiratory Volume and why does it not appear in a listing of normal lung volumes?

Perfusion of the “Blood Side”

The term “**perfusion**” refers to the amount of blood flowing through an organ in a given period of time, usually one minute. . If you’re alert, you’ll see that this value for the lungs is the same as previously learned for Cardiac Output (about 5 L per minute). This is because the right and left sides of the heart have to output the same volume of blood. So, the same volume passing through systemic circulation also has to pass through the lungs.

Therefore, you have a numerical value for perfusion, 5 L / min.

Ventilation / Perfusion (V/Q) Ratio and Hyper- vs. Hypoventilation

By definition, **ventilation (V)** is the alveolar ventilation (the amount of *fresh air* entering the lungs each minute). Since the definition is the same as that for alveolar ventilation, the previous calculation still holds and normal ventilation is about 5 L per minute.

Perfusion (Q), on the other hand, refers to the volume of blood delivered to an organ and is usually expressed in liters per minute

Now that we have numerical values for both ventilation and perfusion, it is possible to calculate a ratio of the two:

$$\frac{V}{Q} = \frac{5 \text{ L (same as alveolar ventilation volume)}}{5 \text{ L (same as cardiac output)}}$$

Interestingly the V/Q ratio, when solved for a normal person, equals approximately 1 and V/Q’s above 1 are termed **hyperventilation disorders**. Those below 1 are termed **hypoventilation disorders**.

Clearly heavy breathing is a type of hyperventilation, but not the only type. Actually, anything that shifts the V/Q ratio above 1 qualifies as hyperventilation and

anything that drops the V/Q below 1 is hypoventilation. Try the review questions below and apply the V/Q ratio to determine the type of disorder.

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

1. Would a pulmonary embolus be a hyper- or hypoventilation disorder? Why?
2. Blocking conducting airways, as would occur in COPD clients, would be which type of disorder? Why?