Factors Affecting Pulmonary Ventilation

Factors Affecting Pulmonary Ventilation: Surface Tension of Alveolar Fluid

The surface tension of alveolar fluid is regulated by pulmonary surfactant, allowing efficient respiration.

The alveoli are highly elastic structures in the parenchyma of the lungs that are the functional site of gas exchange. As the alveoli fill with air during inhalation they expand, and as air leaves the lung with exhalation, the alveoli return to their non-inflated size. The reason for the elasticity of the alveoli is a protein found in the extracellular matrix of the alveoli, called elastin, as well as the surface tension of water molecules on the alveoli themselves.

1- Surface Tension in the Lung

Surface tension is the force exerted by water molecules on the surface of the lung tissue as those water molecules pull together. Water (H_2O) is a highly polar molecule, so it forms strong covalent bonds with other water molecules. The force of these covalent bonds effectively creates an inward force on surfaces, such as lung tissue, with the effect of lowering the surface area of that surface as the tissue is pulled together. As the air inside the lungs is moist, there is considerable surface tension within the tissue of the lungs. Because the alveoli of the lungs are highly elastic, they do not resist surface tension on their own, which allows the force of that surface tension to deflate the alveoli as air is forced out during exhalation by the contraction of the pleural cavity.

2- Pulmonary Surfactant

The force of surface tension in the lungs is so great that without something to reduce the surface tension, the airways would collapse after exhalation, making re-inflation during inhalation much more difficult and less effective. Collapse of the lungs is called atelectasis. Fortunately, the type II epithelial cells of the alveoli continually secrete a molecule called surfactant that solves this problem.

Surfactant is a lipoprotein molecule that reduces the force of surface tension from water molecules on the lung tissue. The main reason that surfactant has this function is due to a lipid called dipalmitoylphosphatidylcholine (DPPC) which contains hydrophilic and hydrophobic ends. The hydrophilic ends are water soluable and attach to the water molecules on the surface of the lungs. The hydrophilic ends are water insoluable and face towards the air and pull away from the water. The net result is that the surface tension of the lungs from water is reduced so that the lungs can still inflate and deflate properly without the possibility of collapse from surface tension alone.

As unborn humans grow and develop in the womb, they receive oxygen from the mother, so their lungs aren't fully functional right away. Of particular importance is the fact that they don't produce surfactant until 24 weeks of development and usually don't have enough built up to prevent lung collapse until 35 weeks of development. Therefore prematurely born infants are at a high risk of respiratory distress syndrome from airway collapse, which can cause death if untreated. It is treated through pulmonary surfactant replacement therapy and mechanical ventilator treatment until the infant's lungs are old enough to secrete enough surfactant to survive on their own. Other diseases may cause atelectasis, such as COPD, or any sort of lung trauma and inflammation that involves extensive damage to the pleural cavity or the lung parenchyma.

3-Compliance of the Lungs

Lung compliance refers to the magnitude of change in lung volume as a result of the change in pulmonary pressure.

Compliance is the ability of lungs and pleural cavity to expand and contract based on changes in pressure. Lung compliance is defined as the volume change per unit of pressure change across the lung, and is an important indicator of lung health and function. Measurements of lung volumes differ at the same pressure between inhalation and exhalation, meaning that lung compliance differs between inhalation and exhalation. Lung compliance can either be measured as static or dynamic based on whether only volume and pressure (static) is measured or if their changes over time are measured as well (dynamic).

Compliance and Elastic Recoil of the Lung

Compliance depends on the elasticity and surface tension of the lungs. Compliance is inversely related to the elastic recoil of the lungs, so thickening of lung tissue will decrease lung compliance. The lungs must also be able to overcome the force of surface tension from water on lung tissue during inflation in order to be compliant, and greater surface tension causes lower lung compliance. Therefore, surfactant secreted by type II epithelial cells increases lung compliance by reducing the force of surface tension.

A low lung compliance means that the lungs are "stiff" and have a higher than normal level of elastic recoil. A stiff lung would need a greater-than-average change in pleural pressure to change the volume of the lungs, and breathing becomes more difficult as a result. Low lung compliance is commonly seen in people with restrictive lung diseases, such as pulmonary fibrosis, in which scar tissue deposits in the lung making it much more difficult for the lungs to expand and deflate, and gas exchange is impaired. Pulmonary fibrosis is caused by many different types of inhalation exposures, such as silica dust.

A high lung compliance means that the lungs are too pliable and have a lower than normal level of elastic recoil. This indicates that little pressure difference in pleural pressure is needed to change the volume of the lungs. Exhalation of air also becomes much more difficult because the loss of elastic recoil reduces the passive ability of the lungs to deflate during exhalation. High lung compliance is commonly seen in those with obstructive diseases, such of emphysema, in which destruction of the elastic tissue of the lungs from cigarette smoke exposure causes a loss of elastic recoil of the lung. Those with emphysema have considerable difficulty with exhaling breaths and tend to take fast shallow breaths and tend to sit in a hunched-over position in order to make exhalation easier.

4- Airway Resistance

Airway resistance refers to resistance in the respiratory tract to airflow.

Airway Resistance

Airway resistance is the resistance to flow of air caused by friction with the airways, which includes the conducting zone for air, such as the trachea, bronchi

and bronchioles. The main determinants of airway resistance are the size of the airway and the properties of the flow of air itself.

Size of the Airway

Resistance in an airway is inversely proportional to the radius of the airway. However the ratio for this relationship is not 1:1. Below is the equation for calculating airway resistance (R).

$R=8(\text{Length}\times\text{Gas Viscosity})(\pi r4)$

The radius of the airways of the conducting zone become smaller as air goes deeper into the lungs. Therefore the resistance to air in the bronchi is greater than the resistance to air in the trachea. The number of airways also plays a large role in the resistance to air, with more airways reducing resistance because there are more paths for the air to flow into. Therefore, despite the fact that the terminal bronchioles are the smallest airway in terms of radius, their high number compared to the larger airways means that the bronchi actually have greater resistance because there are less of them compared to the terminal bronchioles. Another important fact is that airway resistance is inversely related to lung volumes because the airways expand a bit as they inflate, so the airways in a fully inflated lung will have lower resistance than a lung after exhalation.

Airway resistance can be indirectly measured with body plethysmography, which is an instrument used for measuring changes in volume within a structure, such as the airways. The resistance of the airways is an important indicator of lung health and function and can be used to diagnose lung diseases.

The size of the airways, and thus the resistance can change based on the health and conditions of the lungs. Most lung diseases increase airway resistance in many different ways. For example, in asthma attacks the bronchioles spasm and constrict, which increases resistance. Emphysema also increases airway resistance because the lung tissue becomes too pliable and it the airways become more difficult to hold open by the flow of air.

Flow of Air

The air that flows through the lungs varies considerably in the properties of the flow of air. The air flow can either be turbulent, transitional or laminar based on the airway. Laminar flow involves an orderly and concentric distribution of layers of air particles and tends to occur in smaller airways, and has lower resistance. Turbulent flow is disorganized distribution of the layers of air and tends to occur in larger airways and places where the airways branch, and has a higher resistance. Transitional flow occurs in places that branch within smaller airways, in which the air flow becomes in between laminar and turbulent flow and has moderate resistance. The relationship between resistance and type of airflow is difficult to measure and apply, but some mathematical models (such as the Reynold's number) can provide a rough estimate.

Breathing Patterns

Breathing is an autonomic process that moves air in and out of the lungs.

Breathing patterns refer to the respiratory rate, which is defined as the frequency of breaths over a period of time, as well as the amount of air cycled during breathing (tidal volume). Breathing patterns are an important diagnostic criteria for many diseases, including some which involve more than the respiratory system itself.

Characteristics of the Breathing Patterns

The respiratory rate is frequency of breaths over time. The time period is variable, but usually expressed in breaths per minute because it that time period allows for estimation of minute ventilation. During normal breathing, the volume of air cycled through inhalation and exhalation is called tidal volume (VT), and is the amount of air exchanged in a single breath. Tidal volume multiplied by the respiratory rate is minute ventilation, which is one of the most important indicators of lung function. In an average human adult, the average respiratory rate is 12 breaths per minute, with a tidal volume of .5 liters and a minute ventilation of 6 liters per minute, though these numbers vary from person to person. Infants and children have considerably higher respiratory rates than adults.

The respiratory rate is controlled by involuntary processes of the autonomic nervous system. In particular, the respiratory centers of the medulla and the pons

control the overall respiratory rate based on a variety of chemical stimuli from within the body. The hypothalamus can also influence the respiratory rate during emotional and stress responses.

Normal and Altered Breathing Patterns

Eupnea: is the term for the normal respiratory rate for an individual at rest. Several other terms describe abnormal breathing patterns that are indicative of symptoms of many diseases, many of which aren't mainly respiratory diseases. Some of the more common terms for altered breathing patterns include:

Dyspnea: commonly called shortness of breath. It describes dramatically decreased tidal volume and sometimes increased respiratory rate, leading to a sensation of breathlessness. It is a common symptom of anxiety attacks, pulmonary embolisms, heart attacks, and emphysema, among other things.

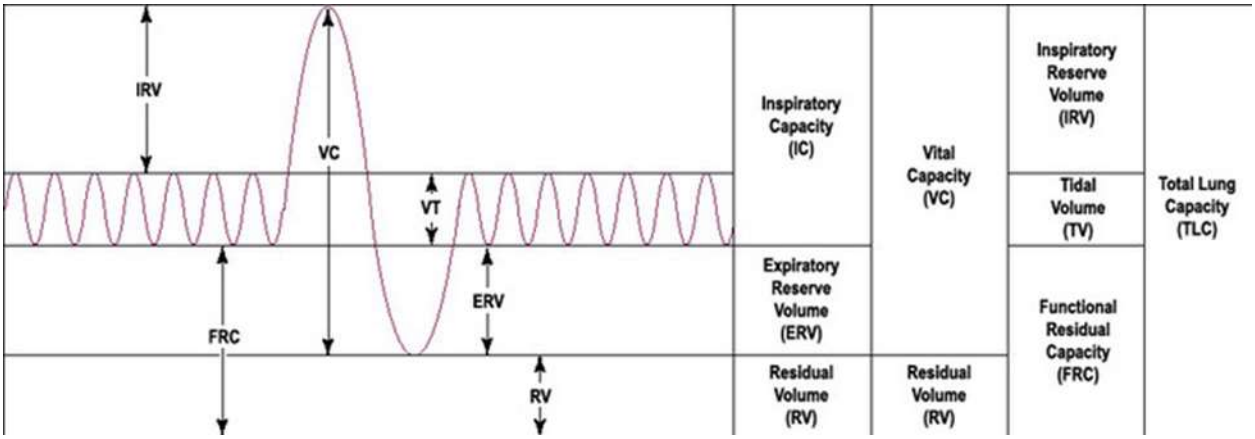
Hypernea: refers to increased volume of air cycled to meet the body's metabolic needs, which may or may not involve a change in frequency of breathing. It is a symptom of exercise and adjustment to high altitude, which are generally not problematic, but can also be seen in those with anemia or septic shock, which is problematic.

Tachypnea: describes increased respiratory rate. Often a symptom of carbon monoxide poisoning or pnuemonia.

Bradypnea: describes decreased respiratory rate. Often a symptom of hypertension, heart arryhmias, or slow metabolic rate from hypothyroidism.

Apnea: Transient stopped breathing that begins again soon afterwards. It is the main symptom of sleep apnea, in which breathing temporarily stops during sleep.

These terms all describe an altered breathing pattern through increased or decreased (or stopped) tidal volume or respiratory rate. It is important to distinguish these terms from hyperventilation and hypoventilation, which refer to abnormalities in alveolar gas exchange (and thus blood pH) instead of an altered breathing pattern, but they may be associated with an altered breathing pattern. For example dyspnea or tachypnea often occur together with hyperventilation during anxiety attacks, though not always.



Spirometry curve: The normal respiratory rate refers to the cyclical inhalation and exhalation of tidal volume (VT).

The Ventilation Rate

In respiratory physiology, ventilation rate is the rate at which gas enters or leaves the lung. There are several different terms used to describe the nuances of the ventilation rate.

Minute Ventilation (VE): The amount of air entering the lungs per minute. It can be defined as tidal volume (the volume of air inhaled in a single breath) times the amount of breaths in a minute.

Alveolar Ventilation (VA): The amount of gas per unit of time that reaches the alveoli (the functional part of the lungs where gas exchange occurs). It is defined as tidal volume minus dead space (the space in the lungs where gas exchange does not occur) times the respiratory rate.

Dead Space Ventilation (VD): The amount of air per unit of time that doesn't reach the alveoli. It is defined as volume of dead space times the respiratory rate.

Dead space is any space that isn't involved in alveolar gas exchange itself, and it typically refers to parts of the lungs that are conducting zones for air, such as the trachea and bronchioles. If someone breathes through a snorkeling mask, the length

of their conducting zones increases, which increases dead space and reduces on alveolar ventilation. Feedback mechanisms increase the ventilation rate in such a case, but if dead space becomes too great, they won't be able to counteract the effect. The ventilation rate is controlled by several centers of the autonomic nervous system in the brain, primarily the medulla and the pons.

The respiratory system through the ages

Breathing for the premature baby

When a baby is born, it must convert from getting all of its oxygen through the placenta to absorbing oxygen through its lungs. This is a complicated process, involving many changes in both air and blood pressures in the baby's lungs. For a baby born preterm (before 37 weeks gestation), the change is even harder. This is because the baby's lungs may not yet be mature enough to cope with the transition. The major problem with a preterm baby's lungs is a lack of something called 'surfactant'. This is a substance produced by cells in the lungs which helps keep the air sacs, or alveoli, open. Without surfactant, the pressures in the lungs change and the smaller alveoli collapse.

This reduces the area across which oxygen and carbon dioxide can be exchanged, and not enough oxygen will be taken in. Normally, a foetus will begin producing surfactant from around 28-32 weeks gestation. When a baby is born before or around this age, it may not have enough surfactant to keep its lungs open. The baby may develop something called 'Neonatal Respiratory Distress Syndrome', or NRDS. Signs of NRDS include tachypnoea (very fast breathing), grunting, and cyanosis (blueness of the lips and tongue). Sometimes NRDS can be treated by giving the baby artifically made surfactant by a tube down into the baby's lungs.

The respiratory system and ageing

The normal process of ageing is associated with a number of changes in both the structure and function of the respiratory system. These include:

1-Enlargement of the alveoli. The air spaces get bigger and lose their elasticity, meaning that there is less area for gases to be exchanged across. This change is sometimes referred to as ‘senile emphysema’.

2-The compliance (or springiness) of the chest wall decreases, so that it takes more effort to breathe in and out.

3- The strength of the respiratory muscles (the diaphragm and intercostal muscles) decreases. This change is closely connected to the general health of the person.

All of these changes mean that an older person might have more difficulty coping with increased stress on their respiratory system, such as with an infection like pneumonia, than a younger person would.