MECHANICS OF VENTILATION

Ventilation is defined as the movement of air in and out of the lungs. The primary function of the lung is to supply the body with oxygen and remove carbon dioxide, the waste product of metabolism.

The total volume of the lung is divided into smaller units of volume. These units are created based on the total lung capacity, the resting end-expiratory volume of the lung, and several breathing maneuvers. There are four different volumes, and five different capacities. A capacity is a combination of two or more volumes (see fig. 1). The picture below shows a graph of these lung volumes and capacities.



Figure 1 lung volumes and capacities

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Measuring and differentiating the volumes of the lungs this way is helpful, because different respiratory diseases will affect different volumes and capacities, and these changes can be measured through pulmonary function testing to help diagnose and treat different illnesses. Knowing how respiratory conditions affect volumes and capacities of the lung will help you ventilate the patient more effectively and safely when needing to initiate mechanical ventilation.

VOLUMES

Tidal Volume (V_T)—the volume of air that is inhaled and then exhaled with each normal breath

Inspiratory Reserve Volume (IRV)—the maximum volume of air that can be inhaled following and above a normal tidal inspiration

Expiratory Reserve Volume (ERV)—The maximum volume of air that can be exhaled from the resting end-expiratory level

Residual Volume (RV)—The volume of air that remains in the lung following a maximum expiration

CAPACITIES

Inspiratory Capacity (IC)—the maximum volume of air that can be inhaled from the normal resting end-expiratory level

 $IC = V_T + IRV$

Functional Residual Capacity (FRC)—the volume of air remaining in the lungs at the resting end-expiratory level

FRC = ERV + RV

Vital Capacity (VC)—the maximum volume of air that may be exhaled following a maximum inspiration OR inhaled following a maximum expiration

 $VC = IRV + V_T + ERV$

OR

VC = IC + ERV

Total Lung Capacity (TLC)—the volume of air contained within the lungs following a maximum inspiration

 $TLC = IRV + V_T + ERV + RV$

OR

TLC = IC + FRC

OR

TLC = VC + RV

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Specific respiratory conditions will be discussed later in this manual, and the change in lung volumes and capacities will be outlined in both restrictive and obstructive disorders.

PRESSURE DIFFERENCES DURING BREATHING

Changes in lung volume occur in response to pressure gradients created by thoracic expansion and contraction. There are four different thoracic pressures involved in breathing.



Figure 1 pressures across the pulmonary system

Scanlan et al, Egan's Fundamentals of Respiratory Care, 5th Ed., 1990 The C.V. Mosby Company

For these purposes, pulmonary pressures will be referred to in relative terms to atmospheric pressure (0 = 760 mm Hg, or 1 atmosphere). Mouth pressure (P_m) is always 0 unless positive pressure is applied to the airway. Pressure at the body surface (P_{bs}) is also always 0, unless the patient is being ventilated with a negative pressure ventilator. Alveolar pressure (P_{alv}) will vary throughout the respiratory cycle. Pleural pressure (P_{pl}), which is normally negative during quiet breathing, will also vary throughout the breathing cycle.

Differences between these pressures are called *pressure gradients*. There are three key pressure gradients involved in the mechanics of breathing: the *transrespiratory*, the *transpulmonary*, and the *transthoracic* pressure gradients. These are shown in figure 2.

The transrespiratory pressure gradient is the difference between the atmosphere (P_m) and the alveoli, and is responsible for the actual flow of gas into and out of the alveoli during breathing.

The transpulmonary pressure gradient is the difference between the pressure in the alveoli and the pleural space, and is responsible for maintaining alveolar inflation.

The transthoracic pressure gradient is the difference between the pressure in the pleural space and the pressure at the body surface, and represents the total pressure required to expand or contract the lungs and chest wall.

Figure 3 shows the changes in pressures and pressure gradients throughout the lung during a respiratory cycle.





Before inspiration begins, the pressure in the pleural space is $-5 \text{ cm H}_2\text{O}$, and alveolar pressure is at 0 cm H₂0. This translates into a transpulmonary pressure gradient of $-5 \text{ cm H}_2\text{O}$ when the lung is in a resting state; that is, the end of expiration. This negative gradient helps to maintain the FRC.

As inspiration begins, the muscles of inspiration work to expand the thorax. Thoracic expansion increases the transthoracic pressure gradient by causing a drop in pleural pressure. As the pleural pressure drops, the transpulmonary pressure gradient widens, causing the alveoli to expand. As the alveoli expand, alveolar pressures drop below mouth pressures, and this negative transrespiratory pressure gradient causes air to move from the mouth to the alveoli, increasing their volume.

As pleural pressures continue to decrease toward the end of inspiration, alveolar pressures begin to equilibrate with the atmosphere, alveolar filling slows, and inspiratory flow decreases to zero. At end-inspiration, the transpulmonary pressure gradient reaches its maximum value (about -10 cm H_20), corresponding to the tidal inflation volume.

As expiration begins, the thorax contracts, pleural pressure rises, and the alveoli begin to deflate. As alveolar pressure begins to exceed that at the mouth, the positive transrespiratory pressure gradient causes air to move out of the lungs. Expiratory flow drops to zero, and a new respiratory cycle begins.

Scanlan et al, Egan's Fundamentals of Respiratory Care, 5th Ed., 1990 The C.V. Mosby Company

There are some forces that must be overcome in order for ventilation to occur. These include elastance, surface tension, compliance, and resistance.

ELASTANCE

Because of the presence of elastic and collagen fibers in its parenchyma, the lung has principles of elasticity. Elasticity is the tendency of a material to try to maintain its shape and offer resistance to stretching forces. When interpreted according to Hooke's Law, elastance is defined in the following equation:

$$\mathsf{E} = \underline{\Delta \mathsf{P}}_{\Delta \mathsf{V}}$$

where ΔP = the change in pressure applied to the lung ΔV = the change in volume in the lung

The concept of lung stretch can be compared to a spring. With increasing force applied to a spring, the spring lengthens in a linear manner. However, the ability of the spring to stretch is limited. When the point of maximum stretch has been reached, additional force on the spring produces little additional increase in length. Further tension may actually break the spring.

When plotting a single breath on a pressure-volume curve based on the properties of elastance, you would see the same points on inspiration as on expiration. However, a normal pressure-volume curve, as shown in figure 4, shows that during deflation, the lung volume at any given pressure is greater than during inflation. This curve is called *hysteresis*, and is due to the surface tension of the alveoli.

Scanlan et al, Egan's Fundamentals of Respiratory Care, 5th Ed., 1990 The C.V. Mosby Company



Figure 3 normal pressure-volume curve

SURFACE TENSION

The properties of surface tension in the lung were first reported by von Neergaard in 1929. He observed that when you compare a lung filled with saline and a lung filled with air, less pressure was needed to inflate the lung filled with saline to a given volume, and that lung did not exhibit the properties of hysteresis.

The inside of the pulmonary alveoli are lined with a thin film of fluid, creating an air-fluid interphase. Laplace's Law shows us that the pressure required to keep a sphere open is directly proportional to the tension in the wall and inversely proportional to the radius of the sphere. This is demonstrated by the following equation:

where P = pressure required to inflate the lung T = tension in the wall of the alveoli and r = radius of the alveoli

Looking at this equation, you can see that the smaller the radius of an alveoli, the higher the surface tension becomes, and the more difficult it becomes to inflate. If this were true, all small alveoli would have a tendency to collapse into themselves, and would require extraordinary pressures to inflate. Luckily, we are born with our alveoli lined with pulmonary surfactant.

Pulmonary surfactant is produced in the lung by alveolar cells, and consists of the phospholipids *dipalmitoyl lecithin* and *sphingomyelin*. Surfactant will decrease the surface tension of the lung, making it easier to inflate. When the alveoli become smaller at the end of expiration, the concentration of surfactant increases, and the surface tension is reduced.

COMPLIANCE

Compliance is the opposite and reciprocal of elastance. Lungs with low compliance, for example, require more pressure to inflate. In the form of an equation, compliance is defined as:

$$\begin{array}{l} C \equiv \Delta V \\ \Delta P \end{array}$$
where ΔV = the change in volume in liters
 ΔP = the change in pressure in cm H₂O
C = compliance in L/cm H₂O

Lung compliance will change with age, body position, and various pathological entities. Normal adult lung compliance ranges from 0.1 to 0.4 L/cm H_20 . Compliance is measured under static conditions; that is, under conditions of no flow, in order to eliminate the factors of resistance from the equation.

The chest wall has elastic properties just as the lung does, based on the configuration of its bones and musculature. Normal compliance of the chest wall is approximately the same as that of the lungs, 0.2 L/cm H_20 . When measured, however, because the lung and the chest wall work together in tandem, they are measured in a parallel circuit shown by the following equation:

$$\frac{1}{C_{LT}} = \frac{1}{C_{L}} + \frac{1}{C_{T}}$$

where C_{LT} = Compliance of the lung and thorax C_L = Compliance of the lung C_T = Compliance of the thorax

Using this equation, total compliance of the lung and the chest wall becomes approximately 0.2 L/cm H_20 .

In a ventilated patient, compliance can be measured by dividing the delivered tidal volume by the [plateau pressure minus the total peep].

RESISTANCE

Resistance of the lung is divided into two parts: *tissue resistance* and *airway resistance*. Tissue resistance accounts for only about 20% of total resistance, and consists of the impedance to motion (friction) caused by moving the organs and chest wall during the respiratory cycle.

Airway resistance is the friction caused by the movement of air throughout the respiratory system and conducting airways.

The formula for resistance is derived from the physics of looking at the pressure drop from one end of a tube to the other with a given flowrate. This depends on the type of flow (laminar or turbulent), the geometry of the tube, the viscosity of the gas, and the flowrate of the gas. The formula for resistance is given to us by Poiseuilles Law:

$$\Delta P = \frac{n8IV}{_r^4}$$

where ΔP = the driving pressure gradient n = the viscosity of the gas I = the length of the tube V = the flowrate of the gas and r = the radius of the tube

In the airways of the lung, resistance is measured using the following equation:

$$R_{AW} = \underbrace{P_{M} - P_{A}}_{\bullet}$$
where
$$P_{M} = \text{pressure at the mouth in cm } H_{2}O$$

$$P_{A} = \text{pressure at the alveoli in cm } H_{2}O$$

$$\bullet$$

$$V = \text{flowrate in } L/\text{sec}$$

$$R_{AW} = \text{airway resistance in cm } H_{2}O/L/\text{sec}$$

In a spontaneously breathing adult, normal airway resistance is estimated at 2 to 3 cm $H_2O/L/sec$.

In the ventilated patient, resistance can be measured by dividing the [peak pressure minus the plateau pressure] by the flowrate in litres per second.