Course Name: Pulmonary Function Test -Interpretation and Application in clinical practice

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W1_L3_Mechanics of Respiration

This section on Mechanics of Breathing, compliance, elastance, surface tension and surfactant and work of breathing are discussed.

Elastance

Basically, elastance is nothing but resistance to a stretch. When a rubber band is stretched, it offers some resistance; that is called as elastance. The lungs are elastic in nature. Hence, it has got its own recoiling forces that is acting on the lungs and that is called as elastance.

Compliance

The effort with which the lungs are distensible - that distensibility is called as compliance. In order to distend the lungs, one need to overcome the two important elastic forces. One is the recoiling forces of the thoracic wall; the other is the recoiling forces of the lungs. The thoracic wall usually tends to pop outwards when it tries to expand. This movement is provided by the rib cage muscles and the tendons; whereas the elastic recoiling of the lungs is the tendency to collapse inwards. So, they tend to collapse inwards inside the thoracic cavity. This is provided by the tissue forces which include the smooth muscle tissue, elastic and collagen fibers which are present in the lung parenchyma. The other important force is the surface tension which is present within the alveoli. There occurs a point where this elastic recoil of the chest wall and the elastic recoil of the lungs balance each other. At that balancing point, there occurs no airflow into the lungs and outside the lungs. This is called as relaxation point. The basic definition of compliance is the measure of distensibility of lungs. It is nothing but the change in the lung volume per unit change in the transpulmonary pressure. That is the change in the volume per unit change in the pressure and it is represented in liters per centimeters of water.

It is given by the formula,

$$\text{Compliance, } C = \frac{\Delta V}{\Delta P}$$

ΔV = change in the volume and ΔP = change in the pressure

The compliance of the lungs and the thoracic wall together, comes to around 130 ml per Cms of H_2O. The compliance of the lungs alone, comes to around 220 ml per Cms of H_2O. This is because if the lungs have to be expanded in vivo, one need to expand the lungs, the alveoli, the lung parenchyma, the pleura, and the chest wall. Hence, it requires more effort; whereas if the compliance of the lungs alone is measured experimentally, it comes to around 220 ml, a slightly higher value.

Measurement of compliance

Static compliance: Measurement of compliance of the lung can be done at a given volume when there is no air flow. This is called the static compliance Here, there is no pressure component involved. To measure the static compliance, the different pressures are measured across a range of lung volume. For example, the patient is asked to take incremental breaths from a spirometer which gives a known volume of the air that is breath. The mouthpiece of the spirometer is usually attached with a manometric device. After inhaling a particular known volume, the mouthpiece will be shut down and the pressure can be measured. The pressure for that volume is noted. Same way, the pressure for the next increment of volume is measured. In this way, we can measure the incremental volumes during inspiration and the pressures associated with that. The same can be repeated during deflation. Such a measurement represents a function of the elastic recoil of the lungs alone which is including the surface tension component also.

Dynamic compliance: There is another compliance called as dynamic compliance wherein the compliance is measured during respiration in which it involves continuous pressure changes along with the volume changes. This includes the pressure as well as the volume that is required to generate the required air flow into the lungs and outside the lungs. This is quite specific but it is always less than the static compliance because here it involves not only the lungs but also the chest wall.

Specific compliance: This is the most specific compliance. This is a calculated one which is given by the formula compliance divided by the lung volume. It is more reliable, specific, and constant for a particular individual.

Compliance graph: We measure the intra-pleural pressure by inserting an intra-esophageal balloon that is tipped with a manometric device. This intra-esophageal pressure will reflect the intra-pleural pressure as well. Every time the patient is inspiring from a spirometer with a known volume, we measure the pressure. When we measure a series of inspiratory pressures with the volumes and similarly during the deflation process and plot this pressure and the volume both during inspiration and expiration, we can get a typical pattern of graph. From the series of inspiratory pressures and volumes, we can see during inspiration when the pressure becomes more negative, the volume also increases. Similarly, during deflation when the pressure becomes less negative, the volume also decreases. This forms a slope,

which is called as a compliance. We can observe that the inspiration slope is lagging the expiration which means at any given point of pressure, the volume of air that is present inside the lungs during inspiration is lesser than that present during expiration.

Or, it can be said that compliance of the lung is more during the expiration. This lagging behind is called as hysteresis. Hysteresis means to "lag behind" This is the typical pressure volume curve when we measure the compliance. The graph relationship between the pressure and the volume is not a straight line. It is curved. This is because the lung has to overcome certain resistance.

Lung Resistance: This includes a viscous resistance as well as the airway resistance that the lungs has to overcome in order to get inflated and also during deflation. That is the reason for this curved pattern of compliance graph. In the relaxation pressure curve, the x axis represents the intra-pulmonary pressure and the intra-pulmonary pressure is negative on the left side and positive on the right side. In the y axis, the volume is plotted. In the left hand side, we can see that volume is represented as change from the resting volume. And on the right side, the absolute lung volumes are represented. It can be observed that at '0' intra-pulmonary pressure, the volume is also '0'. That means this is a volume which does not change and it is represents the end expiratory volume which is nothing but the functional residual capacity.

At this point, the pressure is zero and the volume is also not changing and this point is said to be the ***relaxation volume***. That is the functional residual capacity which is present at the end expiratory point is said to be the ***relaxation volume***. If you can see that the intra-pulmonary pressure becomes positive at greater lung volumes, whereas it becomes negative at lesser lung volumes. The slope of this relaxation pressure curve gives the compliance of the lung or the respiratory system in total. On the right side, one can see the maximal expiratory curve brought about the compliance curve at a more positive pressure. Similarly, the maximal inspiratory curve which is present on the left side is brought about by the maximal inspiratory effort is seen during the most negative intra-pulmonary pressure. In the graph, instead of intra-pulmonary pressure, it is given as trans-pulmonary pressure. As per definition of compliance, it is the change in the volume per unit change in the trans-pulmonary pressure. So, when the trans-pulmonary pressure increases, the volume also increases until it reaches its peak value of 6 liters. Then it becomes a plateau. This is a normal resting compliance curve. But if you can see in the curve below, that is compliance curve for fibrosis, even though the trans-pulmonary pressure increases, the volume is not increasing. That means the lungs are resistant, it is not getting stretched because of the presence of this restriction because of fibrosis. Similarly, when you see at the top curve for Emphysema, wherein the lungs have lost its elasticity, it is already distended and even with a smaller increase in the trans-pulmonary pressure, the volume of the lungs is greater because there is no force that is resisting the inflation or resisting the stretchability. The elastance is lost. The same thing happens in cigarette smoking also.

Factors affecting compliance

Basically these are the elastic forces of the lung tissue offered by the fibres of collagen and elastin which are present in the lung parenchyma. The other one is the surface tension. This surface tension is given by the fluid that is present in the inner walls of the alveoli. The surface tension is always seen wherever there is an air-fluid interface. In the lungs also there is an air-fluid interface that is better explained in the graph which shows the compliance graph of an air-filled and a saline-filled lung. In case of an air-filled lung, the inspiration is different from the expiration and there occurs a lag behind that is hysteresis is seen. In a saline-filled lung, both inspiratory and expiratory graph are the same. The same effort is put for inspiration and expiration. There is no lagging behind. This demonstrate that the effect of surface tension is greater in an air-filled lung.

1. ***Lung volume:*** The factors affecting compliance includes the lung volume. If the volume is more, the compliance is also greater. In this picture we can see in the situation 1, where there are both right and the left lung, For ex. with 5 centimeters of increase in the trans-pulmonary pressure, there occurred increment of 1 liter inside the lungs. Then the compliance is said to be 1 divided by 5, it becomes 0.2. In the situation 2, wherein only one lung is present, the compliance is exactly the half that is 0.1. In the third situation where the lung is just one-tenth of the normal, we can see that the compliance has drastically reduced to 0.02. Hence, if the lung volume is greater, the compliance is also great. But the specific compliance, which is calculated specific to the given lung volume (Specific compliance = compliance divided by lung volume), is almost the same. In the above example, in the situation 1, it is the 0.2 that is the lung compliance divided by 1 liter, it gives 0.2. So, in the situation 2 and 3, also the specific compliance becomes 0.2. So, specific compliance is more stable.
2. ***Respiratory phase:*** During deflation, the volume of air that is present in the lungs is greater when compared during inspiration. So, the compliance is more during deflation.
3. ***Gravity:*** In standing posture at the apex, the compliance is low because the alveoli is already distended. Hence, it is much difficult to distend the alveoli further.
4. ***Surfactant levels:*** If the surfactant is more, the compliance will also be more and vice versa.

Altered Compliance: Decreased compliance is seen in restrictive lung diseases including fibrosis because this offers more resistance to the stretchability. In Pleural effusion, it needs more pressure to distend the fluid. Conditions like Tension pneumothorax, consolidation, atelectasis are associated with decreased compliance. Increased compliance is seen in emphysema and cigarette smoking. Both the condition results in loss of elastic fibers. Hence, it is very easy to distend the lung tissues and alveoli.

Surface Tension

Have you ever wondered why the raindrop is globular or tear shaped? Similarly, the droplet of the water which is coming from the tap, why it is in a tear shaped or a globular shape? Have you wondered this? Have you ever seen a mosquito or any insect walking on the surface of the water? We know that insect is heavier than the water, but still it walks on the surface of water. This is all possible because of the concept of surface tension. What is this surface tension? Whenever there is an air-water interface, there occurs the attraction between the molecules of the water, especially at this surface. Each molecule exerts some intermolecular attraction between each other. The surface molecules, tend to have some attractive forces between each other and to the molecules downwards, but there is no molecule above this layer. Hence all the forces, they tend to drag downwards. This exerts a pressure and that pressure is called as surface tension. We have the air-water interface in our alveoli too. There is a thin layer of fluid which is present in the inner wall of the alveoli and the alveolus is filled with air. So there occurs air-fluid interface and there develops a surface tension. This surface tension reduces the surface area in the alveoli and they tend to collapse the alveoli causing collapse of the lungs as such.

According to Laplace's Law, pressure in any sphere with a hollow inside, will be equal to twice the surface tension divided by its radius. Similarly, this pressure in the alveoli, which is referred as collapsing pressure or distending pressure is equal to twice the tension (surface tension) over the radius. As per this equation, whenever the surface tension increases, the collapsing pressure also increases. Whenever the radius increases, the collapsing pressure decreases. During expiration, the alveoli becomes deflated, it becomes smaller. When the pressure remains constant, if the tension is not reduced, the alveoli will completely close. That causes complete closure of the alveoli and the lung collapse. As a result, the effort for the next inflation cycle becomes very difficult to open up the completely closed alveoli. This is the effect of surface tension.

Imagine there are two alveoli - One is a smaller alveolus and the other is a relatively larger alveolus. The smaller alveoli have got a mucus plug that is obstructing in which air can come inside with efforts. The surface tension will be acting and it causes the pressure to collapse the alveoli. With this high pressure, the air will get rushed out of the alveoli and moreover it gets diverted to this alveolus which is larger. In the larger alveoli also, the surface tension is the same. But since the radius is larger, the distending pressure or the collapsing pressure will become lesser. As a result, air coming from the smaller alveoli adds to the air which is coming from the atmosphere, the larger alveolus tends to grow larger and larger whereas the smaller alveoli tend to grow smaller and smaller. This causes instability of the alveoli and there develops different sizes of the alveoli.

Surfactant

Physiologically, this collapsing of the alveoli and increasing distension of the alveoli is prevented. The substance doing this is called surfactant. This surfactant is a chemical combination of proteins and the lipids, they are secreted from the type II alveolar epithelial cells. The alveoli, has got a basement membrane and it has got two types of cells (pneumocytes). One is a type I cell and the other one is a type II cell. This type I cell is predominantly involved in the gas exchange whereas the type II cell is secreting the surfactant molecule. The surfactant is produced in the type II cells, they get stored in the lamellar bodies and whenever needed they get released by exocytosis and they also get removed by the pulmonary macrophages. The chief component of surfactant is dipalmitoylphosphatidyl choline. Other constituents are phosphatidyl glycerol, other phospholipids and neutral lipids, surfactant proteins A, B, C and D, and few carbohydrates are also present.

Mechanism of action of surfactant: Surfactant prevents the air-fluid interface. Surfactant layer is made of phospholipid which is having a globular head and two tails. The head is hydrophilic in nature which is facing the fluid side whereas the tail is hydrophobic in nature which is facing the air side. At the end of expiration, when the alveoli is smaller, the surfactant molecules becomes thickly or densely distributed. There is increase in the surfactant to alveolar surface area ratio. Thus, it covers the entire alveoli and effect of surface tension to collapse the alveoli is prevented. This prevents the complete closure of the alveoli during expiration. During end inspiration, the alveoli is inflated and it is larger. The surfactant becomes widely distributed, as they move apart. Because of this the surface tension lowering is little bit lesser but it prevents the over distention of the alveoli.

The physiological effects of the surfactant:

1. Surfactant reduces the tendency of the alveoli to collapse by reducing the surface tension to almost 7 to 14 times. If the surface tension of a pure water molecule is 70 dynes per centimeter square, alveolar fluid without surfactant is 50 dynes per centimeter. With surfactant the surface tension is reduced to just 5 to 30 dynes per centimeter square. At the end of expiration, the alveoli are just only partially closed with the help of the surfactant. Without surfactant, it will be completely closed. During the next inflation of the same alveoli, it is much easier, only little transmural pressure is required to open the partially closed alveoli when compared to the completely closed alveoli. Thus, surfactant reduces the work of breathing.
2. Surfactant helps to prevent the pulmonary edema. This is a very important function because surface tension is an inward drawing force, so it draws fluid, it is a retracting force which can draw fluid with the pressure of around 20 mm of Hg. With this force, some amount of the fluid which is present in the lung interstitium

can enter into the alveoli resulting in the pulmonary edema that is prevented with the help of the surfactant.

3. ***Stabilizes the alveolar size:*** and makes it uniform the gaseous exchange also happens effectively. These are the functions of the surfactant in our lungs.

Factors affecting surfactant synthesis:

Whenever the main bronchus is occluded or a main pulmonary artery is occluded or because of cigarette smoking or after inhalation of 100 percent oxygen for a long term the surfactant secretion decreases.

Whereas thyroid hormones and the steroid hormones increase the surfactant synthesis. Thyroid hormones causes increase in number and size of the inclusion bodies which are present in the type 2 cells whereas steroid hormones accelerate the maturation of the surfactant.

Deficiency of this surfactant happens in preterm babies is a very important cause of the respiratory failure in such babies. Usually, surfactant synthesis starts from 24 weeks onwards and get completed by 28 to 30 weeks. So if they suspect any preterm deliveries the mother will be given glucocorticoid injection so that it will fasten the maturation of the surfactant. This syndrome is called as respiratory distress syndrome of the newborn or hyaline membrane disease.

Work of Breathing

In order to inflate the alveoli we need to overcome the resistance offered by the surface tension of the alveoli. We need to overcome the resistance offered by the lung parenchymal tissues that is elastic and the collagen tissues. We need to overcome the pleural fluid and its pressures and we need to overcome the chest wall elastic recoil tendency. All these requires work to be done. Thus, work is required for breathing to happen normally which is done to overcome the resistance. There are two types of resistance - one is the tissue resistance and the other one is the airway resistance.

Tissue resistance is offered by the elastic and the viscous resistance whereas airway resistance is offered by the friction between the gas molecules themselves as well as between the walls. The total work done by the respiratory muscles during quiet breathing includes the work done to overcome the elastic resistance is 65%, the work done to overcome the viscous resistance is 7% and by the airway resistance is 28%. This is the work done to for the normal breathing; it will be much greater during exercise.