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Respiratory Pressures

Sravani Pragna. K[1], Kalyan Chakravarthi. C[1], Muralinath. E[1], Guru Prasad. M[2] , Tulasi Rukmini. K[1]

[1]College of Veterinary Science, Proddatur, Andhra Pradesh

[2]Vaishnavi microbial Pvt. Ltd, Hyderabad

Two types of pressures are exerted in the thoracic cavity and lungs during the process of respiration:

1. Intrapleural pressure (intrathoracic pressure)

2. Intraalveolar pressure (intrapulmonary pressure).

Intrapleural pressure: Intrapleural pressure is the pressure existing in pleural cavity, that is, in between the visceral and parietal layers of pleura. It is exerted by the suction of the fluid that lines the pleural is also called Intrathoracic pressure since it is exerted in the whole of thoracic cavity (Fig. 1).

Normal values of Intrapleural pressure: Respiratory pressures are always expressed in relation to atmospheric pressure, which is 760 mmHg.. Normally, intrapleural pressure is always negative.

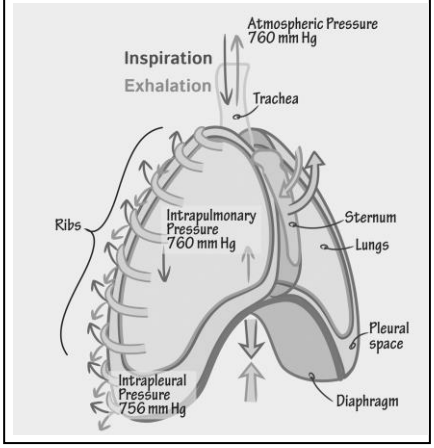
1. Intrapleural pressure at the end of normal inspiration: –6 mm Hg. (760 – 6 = 754 mm Hg.)
2. Intrapleural pressure at the end of normal expiration: –2 mm Hg. (760 – 2 = 758 mm Hg.)
3. Intrapleural pressure at the end of forced inspiration: –30 mm Hg..
4. Intrapleural pressure at the end of forced inspiration with closed glottis (Müller maneuver): –70 mm Hg..
5. Intrapleural pressure at the end of forced expiration with closed glottis (Valsalva's maneuver): +50 mm Hg..

Causes for Negativity of intrapleural pressure: Pleural cavity is always lined by a thin layer of fluid that is secreted by the visceral layer of pleura. This fluid is constantly pumped from the pleural cavity into the lymphatic vessels. Pumping of pleural fluid creates the negative pressure in the pleural cavity.

Causes for positivity of intrapleural pressure: Intrapleural pressure becomes positive in Valsalva's maneuver and in some pathological conditions such as pneumothorax, hydrothorax, hemothorax and pyothorax.

Measurement: Intrapleural pressure is measured by direct method and indirect method. In the direct method, the intrapleural pressure is determined by introducing a needle into the pleural cavity and connecting the needle to a mercury manometer. In indirect method, intrapleural pressure is measured by introducing the balloon, which is

Figure 1: Intrapulmonary pressure



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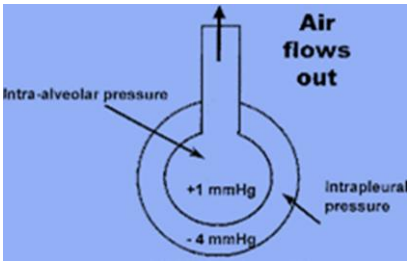
connected to a manometer. Intrapleural pressure is considered as equivalent to the pressure existing in the oesophagus.

Pathology of intrapleural pressure: If intrapleural pressure exhibits positivity, enhancing the effort (i.e., intrapleural pressure) leads no further enhancement in air flow. This effort independence leads to the conclusion that resistance to air flow is enhancing as the intrapleural enhances. Generally intrapleural pressure is negative regarding atmospheric and intrapulmonary pressure particularly during normal breathing. Unexpectedly, if intrapleural pressure becomes similar to atmospheric pressure, lungs will collapse. Generally the intrapleural pressure is the pressure within the pleural cavity. This pressure is essential for occurrence of pulmonary ventilation. If any deviation occurs from the normal value of intra pleural pressure, leads to abnormal pulmonary ventilation. Normally intrapleural pressure differs in various parts of the thorax due to natural elastic property of the lungs causing them to recoil inward and the movement of thoracic cavity outwards. If movement of lungs & thoracic cavity offers against natural momenta, an abnormality takes place. In pneumothorax air gains entry into the pleural space from outside the chest otherwise from the lung itself through mediastinal tissue planes of direct pleural perforation. So, intrapleural pressure enhances as well as volume also reduces. Normally, the negative intrapleural pressure is a prerequisite for the commencement of emphysema in a simple manner by the enlargement of the air space along with elastolytic enzymatic factors. The loss of intrapleural negative pressure leads occurrence of a lung collapse. Because of this vital capacity is reduced and reduction of PaO_2 which mostly leads to a condition known as pneumothorax.

Intraalveolar pressure: Intraalveolar pressure, synonymously called as intrapulmonary pressure, is the pressure existing in the alveoli of the lungs. Normally, intraalveolar pressure is equal to the atmospheric pressure, which is 760 mm Hg. It becomes negative and positive during inspiration and expiration, respectively. Normal values intraalveolar pressure can be negative or positive, which as below (Fig. 2).

1. Intraalveolar pressure during normal inspiration: – 1 mm Hg. (760 – 1 = 759 mm Hg.)
2. Intraalveolar pressure during normal expiration: +1 mm Hg. (760 + 1 = 761 mm Hg.)
3. At the end of one cycle of inspiration and expiration: intraalveolar pressure is equal to atmospheric pressure (760 mm Hg.)
4. Müller manoeuvre: During this procedure of forced inspiration with closed glottis, intraalveolar pressure becomes negative (-80) mm Hg.).
5. Valsalva's manoeuver: During this procedure of forced expiration with closed glottis, intraalveolar pressure becomes positive (+100 mm Hg.).

Figure 2: Intraalveolar pressure



Measurement: Intra-alveolar pressure is measured by using plethysmograph.

Utility of intra-alveolar pressure measurements in the diagnosis of pulmonary diseases: The roentgen appearance of the lungs is, therefore, to a great extent dependent upon the degree of filling of these vessels. With an increase in filling, the lung pattern becomes more prominent; with a decrease, it is less pronounced. Even minor changes in the intra-alveolar

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pressure during normal respiration can affect the filling of the blood vessels. During inspiration, when the pressure falls, the filling of the vessels increases, and during expiration, when the pressure rises, it decreases. Valsalva's and Müller's experiments support the influence of the intraalveolar pressure on the filling of the vessels and on the structure of the lungs. In Valsalva's experiment the intra-alveolar pressure increases and the filling of the vessels in the lungs decreases. In roentgenograms made during this experiment we see a considerably decreased vascular structure in the hilum and the lungs. In Müller's experiment the intraalveolar pressure decreases and vascular filling increases. Roentgenograms acquired during this experiment reveals that the vascular shadows appear more pronounced than normal. Evidence that an increase in the intra-alveolar pressure causes the degree of filling of the vessels to decrease as seen in bronchostenotic emphysema, where there is an increased intra-alveolar pressure with anemia of the lungs. That decreased intraalveolar pressure causes increased filling of lungs is shown by observations made in obstructive atelectasis, where there is a reduced intra-alveolar pressure and hyperemia of the affected portion of the lung. The varying intraalveolar pressures during respiration significantly affects the blood flow to both the auricles as well as the ventricles. During inspiration, when the pressure falls, the blood is sucked through the vena cava to the thorax, and the right auricle is filled. During expiration the flow to the right auricle decreases or ceases. The blood flow to the left auricle, on the other hand, decreases during inspiration and increases during expiration. The outflow from the ventricles is rendered easier during inspiration and more difficult during expiration. The greater the magnitude of differences in the intra-alveolar pressure, the more significant is its influence on the circulation, and subsequently on the heart. In Valsalva's experiment, the flow of blood to the right auricle, and the outflow from the right ventricle, is even more difficult, and the volume of the heart can be observed to decrease during this experiment. In Müller's experiment the conditions are reversed. During the course of the usual roentgen study of the lungs, the intraalveolar pressure may vary from one patient to another or differ for the same patient from one examination to another.

References

1. West JB. Pulmonary Pathophysiology, 5th ed. McGraw-Hill, 1995.
2. Crystal RG, West JB. The Lung, Scientific Foundation, 2nd ed. Raven Press, 1997.
3. Fishman AP, Elias JA, Fishman JA, et al. Fishman's Pulmonary Diseases and Disorders, 4th ed. McGraw-Hill, 2008.