Pulmonary ventilation ratio - perfuse

Pulmonary ventilation-perfusion ratio is a quantity that expresses the ratio of alveolar ventilation [V_A] = l/min and capillary flow in the lungs [Q] = l/min. This ratio can be applied to the lung as a whole or to specific parts of the lung. If we consider total alveolar ventilation and total lung flow, we get values of approximately $\frac{V_A}{Q} = \frac{5l/min}{5l/min} = 1$. This is the ideal situation, suitable for the most efficient transition of gases from the alveolar lumen to blood and vice versa. A suitable amount of air with suitable pO2 and pCO2 can transfer gases to a suitable amount of blood and vice versa, see below.

Ventilatory-perfusion imbalance

Of course, the ideal situation where $V_A/Q = 1$ does not always hold true even physiologically. **Ventilatory-perfusion imbalance** is a condition where the ratio V_A/Q is skewed away from 1, i.e. increased or decreased. This is usually due to a decrease in ventilation and/or perfusion. This imbalance may occur locally or throughout the lungs.

Increase in ventilation-perfusion ratio

- $\lim_{Q \to 0} \frac{V_A}{Q} = +\infty$
- Increase in V_A/Q ratio occurs, for example, in pulmonary embolism, when capillary flow decreases, even stops. The alveolus is ventilated properly, but there is no gas exchange, the hemoglobin in the capillary is already completely saturated with O_2, and no more unoxygenated blood is supplied. Gradually, pO_2 increases and pCO_2 decreases in the alveolus and in the blood.

Decrease in ventilation-perfusion ratio

- $\lim_{V_A \to 0} \frac{V_A}{Q} = 0$
- The decrease in the V_A/Q ratio occurs, for example, in airway obstruction, when ventilation decreases, pO_2 decreases and pCO_2 rises in the alveolus, and the flowing blood cannot oxygenate and get rid of CO_2. The outflowing blood has low pO_2. A similar situation can occur in right-to-left shunt, see below, where perfusion is normal but blood does not flow past the ventilated alveolus, does not oxygenate, and effective ventilation is zero.

Influence of gravity

Influence-on-ventilation

Gravity affects the amount of ventilation in different parts of the lung. At the base of the lung, due to the slightly greater atmospheric pressure, the difference between intrapleural pressure and atm. pressure is smaller - the pressure is "less negative". The alveoli are therefore less distended at the base than at the apex and then distend more on inspiration - they are more ventilated. However, the differences in ventilation are not nearly as great as the differences in perfusion.

Influence-on-perfusion

The perfusion pressure, the difference in pressure at the arterial and venous ends of the capillary (p_a - p_v) and the resistance of the capillary, related to its translucency, are critical to the rate of blood flow through the capillary. Frictional acceleration results in there being physiologically greater capillary pressure (p_a and p_v) at the base of the lung than at its apex. In the lung, however, the alveoli press on the capillaries. The alveolar pressure p_a is indeed different in different parts of the lung, see above, but for our purposes it may now be considered constant. The alveoli may in certain cases oppress the capillaries by their p_A, limiting their translucency, hence reducing the flow, or even stopping it altogether.

> According to some authors, gravity is not the cause of differences in flow through capillaries, but this cause is different. The rationale is that the difference in flow at the base and apex of the lung exists even in the weightless state of orbit

The lung can therefore be divided into 3 zones

- **zone at apex**; $p_A > p_a$; capillaries are collapsed due to low pressure and Q = 0 l/min;
- **middle zone**; $p_a > p_A > p_v$; blood flows but the capillary is partially occluded because the alveolar pressure is greater than the pressure at the venous end of the capillary;

- **zone at the base**; $p_a > p_v > p_A$; the capillary is distended, perfusion is not further restricted because the pressure at the venous end of the capillary (hence the pressure throughout the capillary) has exceeded the alveolar pressure.

Thus, the differences in local perfusion are very marked. At the base, perfusion is greatest, decreasing cranially. Flow in the upper zone may stop completely, but this zone represents a minimal part of the lung. The differences in perfusion are, as I have already stated, greater than in ventilation.

Management of ventilation-perfusion ratio

- Areas with a reduced ratio can in no way replace areas with an increased ratio. In an area with a reduced ratio, the blood is inadequately oxygenated and there is a normal amount of it due to normal perfusion.
- Conversely, areas with an increased ratio give more oxygenated blood, but since the transport capacity of the blood is normally almost exhausted, the increase is slight. But there is little blood because of the low flow.

If large amounts of poorly oxygenated blood and small amounts of highly oxygenated blood mix, the result is poorly oxygenated blood. It is therefore necessary to keep the ventilation-perfusion ratio in all areas of the lung close to 1 at all times.

The body has a mechanism to keep the ventilation-perfusion ratio within acceptable values. This is **hypoxic pulmonary vasoconstriction**. If ventilation is reduced locally, i.e., V_A/Q is reduced, pO_2 gradually decreases in the hypoventilated alveoli. The pulmonary arteries supplying blood to these alveoli react by vasoconstriction, thus reducing perfusion of this area. Blood is then diverted and conducted through other arteries that have not undergone vasoconstriction to areas of normal ventilation. The more ventilated alveoli are more perfused and the V_A/Q ratio is favourable for efficient gas exchange in all parts of the lung. If ventilation of a portion of the tissue returns to higher values, arterial smooth muscle relaxes, resistance decreases, and perfusion increases until a new equilibrium between ventilation and perfusion is established. The disadvantage of this mechanism is that during total hypoventilation, e.g. due to airway obstruction, postprandial pulmonary vasoconstriction occurs and pulmonary circulation resistance increases significantly. The right heart must then exert great force in its contractions to ensure sufficient flow. It is more susceptible to heart failure, among other reasons, because the blood in the coronary arteries in this situation has an increasingly smaller pO_2. The exact mechanism of hypoxic pulmonary vasoconstriction has not yet been fully explained.

- It is also worth noting the significance of hypoxic pulmonary vasoconstriction at birth, when an increase in pO_2 in the lungs results in a significant reduction in pulmonary blood resistance. According to some authors, the prenatal development of the fetus to maintain low lung flow and then to bring the lungs into function is the main reason why hypoxic pulmonary vasoconstriction has been maintained in human evolution.

Right-to-left shunts

Anatomical right-to-left shunts are situations where the arrangement of the vascular system is such that some venous blood from the bronchial and coronary circulation enters the pulmonary veins, or directly into the left atrium. It is therefore deoxygenated blood that does not pass through the pulmonary capillaries and enters directly into the systemic circulation, where it dilutes the normally oxygenated blood. The ventilation-perfusion ratio is 0 in the case of shunts. In a healthy person, they represent **about 2% of cardiac output**.

Additional shunts may occur in sick people (pneumonia) or in persons with congenital heart disease, and their effect on the overall ventilation-perfusion ratio should be taken into account.

References=

Resources

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