Ventilatory failure (pathophysiology)

Respiratory failure is classified based on the pathophysiological mechanisms that lead to **hypoxemia** and/or **hypercapnia**. In a simplified view, these are disorders of ventilation, distribution, diffusion and perfusion. The following abnormalities lead to hypoxemia and/or hypercapnia:

- V/Q ratio (ventilation-perfusion ratio);
- intrapulmonary R-L shunts (intrapulmonary shunt);
- hypoventilation;
- abnormal gas exchange at the alveolocapillary membrane;
- reduced concentration of inspired O_2;
- increased venous blood desaturation with cardiac dysfunction plus one or more of the above 5 factors.

Ventilation

Sufficient minute ventilation ensures physiological **normocapnia ($PaCO_2$ 4.5–6 kPa)**, hypoventilation causes hypercapnia, and hyperventilation, on the contrary, hypocapnia.

Hyperventilatory hypocapnia can worsen oxygen economy due to respiratory alkalosis by shifting the dissociation curve for hemoglobin to the left and inducing cardiac arrhythmias.

Hypercapnia is particularly undesirable due to arrhythmogenic and other negative effects on the circulatory system - vasodilation and subsequent compensatory tachycardia. Rapid correction of long-lasting hypercapnia with mechanical ventilation, which leads to post-hypercapnic metabolic acidosis, is also undesirable. The kidneys have a relatively slow ability to correct excess bicarbonate.

Ventilation disorders

Ventilation disorders can be divided according to the impairment of individual functional levels of the respiratory system:

Disorders of the respiratory center

- trauma/intracranial hemorrhage;
- neuroinfection;
- pharmacological depression;
- brain perfusion disorders;
- apnea in premature infants.

Disorders of innervation and neuromuscular transmission

- nerve and spinal cord damage;
  - Guillain-Barré syndrome;
  - trauma;
  - M. Werdnig-Hoffmann;
  - paresis *of the phrenic nerve*;
- neuromuscular blockade;
  - medicines;
  - myasthenic syndrome;
- systemic diseases;
  - DPM;
  - infection.

Peripheral disorders of the executive organ (= it is also a distribution disorder)

- involvement of the chest and pleura;
  - pneumothorax;
  - hemothorax;
  - chylothorax;
- impairment of respiratory muscles;
  - with excessive work of breathing;
  - cachexia;
- respiratory tract impairment;
  - epiglottitis;
  - acute laryngitis;
  - aspiration;
  - bronchial asthma;
  - bronchiolitis.
- lung parenchyma involvement;
  - pneumonia;
  - ALI/ARDS;

- pulmonary edema.

Distribution

The distribution represents the distribution of the inhaled mixture to individual areas of the lung. Distribution ratios can be influenced by choosing the ventilation mode and other adjustments to the breathing cycle (inspiratory delay, the introduction of the so-called *sigh*, etc.). Distribution disorders refer to the intrapulmonary distribution of inspired air. Gas exchange is affected differently in different components by 2 basic types of lung diseases: obstruction and restriction.

Distribution disorders - obstruction

Obstruction is characterized by an increase in airway **resistance** (expiratory stridor, wheezing, mixed stridor). The pathogenesis is dominated by unevenness of ventilation and perfusion (V/Q disorder), pulmonary shunt, increase in airway resistance. Spirometry demonstrates impaired dynamics of gas exchange and an increase in FRC.

Obstructive disorders are the prototype of ventilation failure, where the primary problem is insufficient elimination of CO_2.

- bronchial asthma
- bronchitis
- bronchiolitis
- COPD

Obstructive disorders are characterized by normal compliance, increased resistance (mainly airway resistance), increased RV, FRC and reduced FEV1.

Distribution disorders - restriction

The restriction is characterized by reduced chest and/or lung compliance. Alveolocapillary block, reduced diffusion capacity, pulmonary shunt and ventilation-perfusion imbalance are used in the pathogenesis. Spirometry demonstrates a reduction in lung volumes and capacities. Restrictive disorders are the prototype of hypoxic failure, i.e. failure of oxygenation. In severe disorders, hypercapnia is also associated.

Pulmonary causes

- pulmonary fibrosis
- lung resection
- pulmonary edema
- pneumonia

Extrapulmonary causes

- ascites
- kyphoscoliosis
- burns
- high diaphragm condition

> Hypoxic pulmonary vasoconstriction leads to the development of *cor pulmonale* through the mechanism of pulmonary hypertension!

Restrictive disorders are characterized by reduced compliance, increased pulmonary resistance, increased work of breathing, decreased VA, RV, FRC and TV, increased R–L shunts, pulmonary hypertension, and low T (→ rapid lung units predominate).

Distribution disorders - combined disorders

Combined disorders of obstruction and restriction mean a combination of the above-mentioned pathophysiological mechanisms. A typical representative is cystic fibrosis. It is characterized by reduced compliance, increased resistance, increased work of breathing, increased R–L shunts, T can be low or high.

Diffusion

Diffusion is an important component of gas exchange. The transfer of blood gases is very fast (0.1 sec). It is given by the gradient of partial pressures on the alveolocapillary membrane. The solubility of carbon dioxide causes faster diffusion compared to oxygen, so hypoxemia precedes hypercapnia in many disease states. Sufficient diffusion of blood gases corresponds to the physiological value of the alveolar-arterial oxygen gradient A-aDO_2.

Interpretation of A-aDO_2 values :

- hypoxemia with normal A-aDO_2 indicates hypoventilation;
- hypoxemia with increased A-aDO_2 indicates a disorder of ventilation/perfusion, diffusion = alveolocapillary transport.

Diffusion disorders

Diffusion disorders affect the rate and extent of gas exchange. Oxygen transfer is always affected, and in more serious disorders, CO_2 transfer is also affected. In diffusion disorders, the following pathophysiological mechanisms are mainly applied: changes in gas pressure gradients between alveolar air and blood, functional or morphological limitation of the diffusion area, change in the diffusion path (alveolocapillary membrane disorder), changes in lung perfusion. Extensive disturbances are referred to as an alveolocapillary block.

- pulmonary fibrosis
- interstitial pneumonia
- ARDS

Perfusion

Ventilation-perfusion ratio and its disorders

Blood pressure determines the distribution of perfusion in the lungs. The lumen of the pulmonary capillaries determines the low-pressure circulation in the pulmonary basin and the pressure in the alveoli. Alveolar ventilation in adults normally reaches values of 4-5 liters/min., and minute cardiac output is 5 liters. So a normal V/Q value is 0.8-1.0.

🔎 *For more information see Pulmonary ventilation - perfusion ratio.*

The result of a pathological V/Q ratio is either a decrease in the V/Q ratio (the formation of a P–L shunt during perfusion of hypoventilated alveoli) or an increase in it (an increase in alveolar dead space during hypoperfusion of well-ventilated alveoli). Contradictory faults can also occur regionally. In an extreme case, V/Q is equal to infinity (non-perfusion, e.g. pulmonary embolization), or, on the contrary, in atelectasis (non-ventilation) it is equal to zero. Mild hypoxemia resulting from a V/Q imbalance responds well to oxygen therapy, a severe V/Q imbalance will only minimally affect the increase in FiO_2.

Short circuit Qz / Qco

A shunt can be defined as **the percentage of venous blood** in the total systemic flow that does not come into contact with a functional alveolocapillary membrane. The shunt is calculated as the ratio of the shunt flow Qz to the total flow, i.e. the cardiac output Qco. Increasing FiO_2 has only a small effect on pO_2 if the shunt value exceeds 30%.

Cause

The most common cause of shunt is the perfusion of non-ventilated lungs. The dynamics of changes in this indicator are typical for certain developmental stages of lung disorders. The increase in the pulmonary shunt correlates with the deterioration of the local findings and the general condition of the patient and has prognostic significance. Optimal conditions for gas exchange are achieved with the correct V/Q ventilation and perfusion ratio.

Even in completely healthy lungs, a short circuit must be expected. Functional shunts (approx. 2% of cardiac output) and anatomical shunts (also approx. 2% of cardiac output) contribute to it. Functional shunts are caused by the presence of very small regions of the non-ventilated lung, but with preserved perfusion. The anatomical shunt is caused by blood flow through bronchial veins, pleural veins, Thebesian veins and intracardiac shunt.

- A shunt < 10 % during UPV is a picture of normal cardiopulmonary system function.
- A shunt between 20-30 % represents the upper physiological limit - the patient can breathe spontaneously if no other organ involvement is present.
- With a shunt > 30 %, the patient can no longer be burdened with spontaneous ventilation. Hypoxemia persists even with oxygen therapy because the blood flowing through the shunt does not come into contact with the high concentration of O_2 in the alveoli. In this situation, increasing FiO_2 no longer brings benefit, but it is necessary to offer recruitment maneuvers and maximize lung volume by increasing positive pressure (PIP, PEEP). With the increase in intrapulmonary shunt, pO_2 decreases proportionally, and pCO_2 remains constant due to the increase in pulmonary minute ventilation until the shunt is > 50 %.
- An increase in the proportion of shunted blood to > 50 % causes life-threatening hypoxemia.

Secondary perfusion disorders arise from pulmonary vasoconstriction (hypoxia, acidosis), a mechanism of hypoxic pulmonary vasoconstriction when ventilation is reduced.

Pathological conditions leading to an increase in R–L shunts

- atelectasis
- PNO
- pulmonary edema
- pneumonia
- ARDS
- RDS from immaturity

Alveolar dead space

If ventilation exceeds the capabilities of capillary flow, the V/Q ratio is > 1. In this situation, alveolar dead space is increased. The alveolar dead space together with the anatomical dead space forms the so-called ventilation dead space, which under normal circumstances represents up to 30% of the total ventilation. An increase in dead space leads to hypoxemia, and later hypercapnia. Alveolar dead space increases due to pulmonary perfusion disorders due to hypotension, pulmonary embolism, but especially high pressures (PIP, PEEP) during UPV by the mechanism of alveolar overdistension. The increase in anatomical dead space is mainly represented by the ventilation circuits during UPV.

At steady state, the pCO_2 value is directly proportional to CO_2 production and inversely proportional to alveolar ventilation. It follows that a decrease in alveolar ventilation and/or increased production of CO_2 leads to hypercapnia.

Under pathological conditions, with excess ventilation and inadequate perfusion, the proportion of dead space ventilation increases. Conversely, insufficient ventilation of a well-perfused area causes an increase in the short circuit!

For an indicative assessment of the ratio between the size of the functional dead space and the size of the respiratory volume (Vd/Vt), the determination of **the difference between the arterial tension of CO_2 and the tension of CO_2 in the exhaled mixture** at the end of exhalation ($etCO_2$) is used. Under normal circumstances, this difference is minimal (2–5 torr), under pathological circumstances it rises significantly (the rise is mainly caused by a decrease in the $etCO_2$ value).

In normal subjects, the Vd/Vt value is in the range of 0.2–0.3. An increase in Vd/Vt is first associated with the development of hypoxemia, hypercapnia usually occurs when Vd/Vt is greater than 0.5.

Division of respiratory failure according to blood gas values

1. **Hypoxic respiratory failure**, which is caused by a ventilation-perfusion imbalance with an increase in pulmonary shunt or a diffusion disorder on the alveolocapillary membrane = alveolocapillary block. This type leads to intrapulmonary mixing of venous and arterial blood. The result is hypoxemia with normocapnia, because, as already mentioned above, the solubility of CO_2 is significantly higher than that of O_2. Even with a compensatory increase in ventilation (in children, especially an increase in respiratory rate), we can initially find hypocapnia. However, with the deepening of this disorder, the value of pCO_2 gradually rises, and in severe conditions, we already find global respiratory insufficiency.
2. The second type is **hypoxic-hypercapnic respiratory failure**. It also occurs when alveolar ventilation is reduced in relation to the organism's physiological needs. The result is hypoxia and hypercapnia at the same time.

- diseases affecting the lung parenchyma and leading to a decrease in the V/Q ratio initially lead to hypoxic respiratory failure.
- diseases affecting the airways and respiratory control units lead to hypoxic-hypercapnic respiratory failure. Hypercapnia is quite typical for diseases affecting the respiratory pump.

Links

Source

- HAVRÁNEK, Jiří: Ventilační selhání (edited)

Related Articles

- Binding of oxygen to hemoglobin
- Pulmonary ventilation - perfusion ratio
- Ventilatory failure (pediatrics)