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ACUTE RESPIRATORY FAILURE. CLASSIFICATION, CLINIC. MECHANISMS OF DEVELOPMENT OF HYPOXEMIA, HYPERCAPNIA. GENERAL PRINCIPLES OF RESPIRATORY THERAPY

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ABSTRACT

This article provides a detailed understanding of acute respiratory failure, classification, clinic, hypoxia and its types. In addition, information on the mechanisms of hypoxemia, hypercapnia development, general principles of respiratory therapy was given, and the above topics were discussed.

Tissue respiration is the process by which it absorbs oxygen. Blood circulation, blood and external respiratory systems participate in tissue oxygen supply. Disruption of the activity of each of these systems to a different degree is certainly reflected in tissue respiration. However, the malfunction of one of these systems is compensated by the increase in the activity of the other, thus ensuring the continuity of respiration in the tissue. In anemia, the rate of blood flow increases, and even when the oxygen capacity of the blood decreases, the tissue is oxygenated in the required amount per unit of time. In the case of blood circulation failure, the blood flow rate decreases, the demand for oxygen in the tissue increases (at the expense of erythrocytes from the blood depot), and the oxygen capacity of the blood increases. When the partial pressure of oxygen decreases, the blood flow rate in the alveoli increases and the oxygen capacity of the blood increases due to erythrocytosis. When these adaptation processes are insufficient and when the tissue's oxygen usage is disturbed, the tissue becomes hypoxic.

This type of hypoxia develops when the partial pressure of oxygen in the inhaled air decreases. An example of this is mountain sickness, which manifests itself as a person climbs higher. Exogenous hypoxia can be created experimentally by using a barocamera and by using oxygen-poor breathing mixtures. Examples of the latter include malfunctions of the oxygen supply system in mines, underground wells, submarines and aircraft, and malfunctions of anesthesia equipment during surgical operations.

Respiratory hypoxia is caused by a lack of gas exchange in the lungs. This hypoxia can be caused by:

1. Decreased ventilation in respiratory tract disorders (bronchospasm, inflammatory processes, bronchitis, tracheitis), obstructions to lung alignment (pneumothorax, accumulation of exudates in the pleural cavity) and other respiratory diseases.

2. Violation of the ventilation-perfusion ratio as a result of blood flow in the lungs and gas exchange in the alveoli. When venous blood enters the alveoli through arteriovenous anastomoses (shunts) in the lungs and passes into the arterial system of the greater blood circulation, the blood passes from the pulmonary artery to the pulmonary vein, thus deoxygenated blood is poured into the left ventricle. In all of the above, the delivery of oxygen to the body does not meet the body's requirements. The amount of oxygen in the blood passing through the lungs decreases, resulting in a significant decrease in oxygen in the arterial blood. Hypercapnia is usually added to hypoxia, and gas acidosis is added to metabolic acidosis.

Circulatory hypoxia develops in disorders of local and general blood circulation, and it can be divided into ischemic and immobile forms of blood stasis.

If hemodynamic disturbances develop within the larger blood circulation, the oxygen saturation of the blood in the lungs may be sufficient, but there is a possibility that it will be difficult for the oxygen to reach the tissue. If the hemodynamics is disturbed in the small blood circulation, the oxygenation of the arterial blood is lost. Circulatory hypoxia can occur not only from absolute, but also from relative lack of circulation. This condition can be observed when the tissue's demand for oxygen exceeds its supply. An example of this is the expansion of coronary arterioles under the influence of adrenaline in the heart muscle in an emotional state, at the same time, a certain increase in the oxygen demand of the myocardium. This type of hypoxia also includes tissue oxygen starvation as a result of microcirculation disorders. In this case, oxygen passage is disturbed due to swelling of tissues, disruption of permeability of cell membranes.

Pathological changes in the blood system of this type of hypoxia occur mainly due to a decrease in the effective oxygen capacity of the blood. Hemic hypoxia is divided into hypoxias resulting from anemia and hemoglobin dysfunction. Anemia causing hypoxia is discussed in detail in the Pathological Physiology of the Blood System section. In pathological conditions, such compounds of hemoglobin can be formed that cannot carry out the function of oxygen transport. An example of these is carboxyhemoglobin, i.e. the combination of hemoglobin with carbon dioxide (CO). The ability of hemoglobin to bind to CO is 300 times higher than that of oxygen, which means that carbon dioxide is very toxic; poisoning occurs in the air, even in small

amounts. In this case, not only hemoglobin, but also the activity of respiratory enzymes rich in iron is disturbed. In poisoning with nitrate, nitrite and aniline products, methemoglobin is formed, which contains trivalent (Fe3+) iron and cannot bind oxygen.

Tissue hypoxia is a violation of sufficient use of oxygen in tissues. In this case, while the tissue is supplied with oxygen in sufficient quantity, biological oxidation is disturbed. The main cause of tissue hypoxia is a decrease in the activity or amount of respiratory enzymes and a violation of oxidation and phosphorylation processes.

Poisoning by cyanide monoiodoacetate of cytochromoxidase, the last link in the respiratory chain, can be a vivid example of seed hypoxia. Dehydrogenases break down and tissue hypoxia occurs when poisoned by alcohol and other narcotic substances (ether, urethane). A decrease in the synthesis of respiratory enzymes, which causes tissue hypoxia, is also observed in avitaminosis. This condition is observed especially when riboflavin and nicotinic acid are low. Because they belong to the prosthetic group of flavin enzymes and dehydrogenases. As a result of the interruption of oxidation and reduction, the efficiency of biological oxidation decreases, energy as free heat disperses, the resynthesis of the macroergic compound decreases. Energy starvation and metabolic shifts occur. Activation of free radical oxidation may also be important in seed hypoxia. In this, organic substances undergo non-enzymatic oxidation by molecular oxygen. Fatty oxides cause membrane destabilization in mitochondria and lysosomes. An increase in free radical oxidation, as well as a lack of its natural inhibitors (tocopherol, rutin, glutathione, serotonin, some steroid hormones), under the influence of ion radiation, it is also observed in seed hypoxia caused by an increase in atmospheric pressure.

Depending on the speed of development of the adaptation process: a) rapid (emergency) adaptation; b) divided into long-term adaptations.

In hypoxia, first of all, compensatory - adaptive processes are observed in the oxygen transport and use systems. In addition, mechanisms of struggle for oxygen and adaptation of tissue to low respiratory conditions are activated. Rapid (emergency) adaptation is reflexive: increased lung ventilation, adaptation in the circulatory system, increased erythrocyte and hemoglobin content. An increase in lung ventilation is one of the compensatory reactions, which is formed as a result of reflex stimulation of the respiratory center under the influence of chemoreceptor impulses in blood vessels. Sinocarotid and aortic receptors are very sensitive to changes in the chemical composition of blood and the accumulation of carbon dioxide and hydrogen ions. The pathogenesis of gasping in hypoxic hypoxia is different - the activation of chemoreceptors occurs in response to a decrease in the partial pressure of oxygen in the blood. Obviously, hyperventilation is a positive reaction for the body at altitude, but it also has negative consequences. As long as the amount of carbon dioxide in the blood decreases due to the release of carbon dioxide, this process becomes more complicated.

Hypoxia causes a violation of the structure and functions of various organs.

Nervous system. The central nervous system is very sensitive to hypoxia, but not every part of it is damaged equally in hypoxia. The cerebral cortex is especially sensitive to it, while the brain stem, medulla oblongata, and spinal cord are less sensitive. 2.5-3 minutes after the oxygen supply is cut off, foci of necrosis are observed in the cerebral cortex and cerebrum. At the same time, in case of asphyxiation in the medulla, a few cells die after 10-15 minutes. When there is a lack of oxygen in the brain, first there is agitation, then inhibition, sleep, headache, movement control disorders are observed breath When oxygen deficiency develops acutely, breathing becomes frequent, shallow, and air exchange decreases. Cheyne-Stokes type peripheral respiration may occur.

Blood circulation. Tachycardia is observed in acute hypoxia. Systolic pressure remains unchanged or gradually decreases, pulse pressure does not change or increases. The minute volume of blood increases.

The amount of oxygen in the arterial blood decreases by 8-9% and it significantly increases blood circulation in the coronary artery. These changes are caused by the expansion of the coronary arteries and the increase in venous flow, which occurs as a result of the acceleration of heart contraction. Metabolism in M. Changes in metabolism in hypoxia depend on biological oxidation, starting with the violation of carbon water and energy exchange. Macroergy in the cell for hypoxia - reduction of ATF value and its breakdown products

The concentration of ADF, AMF and inorganic phosphate is characteristic. The content of creatine phosphate in brain tissue acid is reduced. As a result of the decrease in macroergy, glycolysis increases, pyruvate and lactate increase due to the decrease in glycogen content. An increase in pyruvate, lactate and several other organic acids ensures the development of metabolic (non-gaseous) acidosis, which is due to oxygen deficiency.

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