ФГБОУ ВО «Северо-Осетинская государственная медицинская академия» Минздрава РФ
Federal State Budgetary Educational Institution of Higher Education "North Ossetian State Medical Academy",
the Ministry of Health of the Russian Federation

Bestaev G.G., Slepushkin V.D.

CLINICAL PHYSIOLOGY OF RESPIRATION

(Tutorial)

Bestaev GG, Slepushkin VD

CLINICAL PHYSIOLOGY OF RESPIRATION

(Tutorial)

Владикавказ

The authors:

Bestaev Georgy Givievich —Associate Professor of the Department, Candidate of Medical Sciences
Slepushkin Vitaly Dmitrievich- Head. department, doctor of medical sciences, professor

English translation:

Shulga Elena Vitalievna– Candidate of Philological Sciences, Associate Professor

Reviewer:

Lebedeva Elena Anatolievna —doctor of medical sciences, professor of Rostov medical university

Authors:

George Bestaev-dozen*t*
Vitaly Slepushkin-Head of the Department, Doctor of Medical Sciences, Professor

The English translation:
Elena Shulga – candidate of Philological Sciences, Associate Professor

Review:

Elena Lebedeva -professor of Rostov Medical University

The manual is intended for senior students, including foreign students studying in English, residents, graduate students.

CLINICAL PHYSIOLOGY OF RESPIRATION

Understanding the process of breathing is a key step to understanding the essence and tactics of managing acute respiratory failure. The control of respiratory function is represented by the close coordination of three groups of neurons in the medulla oblongata: the dorsal respiratory center, which controls inhalation; the ventral respiratory center, which controls expiration, and the pneumotaxic center, which controls the frequency and depth of breathing. In addition to brainstem neurons, there is a system of peripheral chemoreceptors located outside the brain in the form of carotid bodies and aortic bodies that respond to slight changes in PaO_2.
Nerve impulses from the central nervous system travel through the spinal cord and motor neurons, reaching and activating the diaphragm and other respiratory muscles. The contraction of the respiratory muscles creates a negative pressure in the pleural cavity due to the expansion of the chest and displaces the contents of the abdominal cavity downward. In the thorax, the negative pressure created during inhalation leads to a decrease in pressure in the alveoli below atmospheric pressure, forming a gradient for the flow of inhaled air towards the alveoli. The oxygen-enriched inhaled air allows diffusion of $O_2$from the alveoli to the blood through the alveolo-capillary membrane, where the deoxygenated hemoglobin is saturated with oxygen and becomes the form oxyhemoglobin.

Respiration in the broad sense of the word is the exchange of body gases with the external environment, which is necessary to maintain energy metabolism. The respiratory function is provided by three successive stages: external respiration (exchange of gases between the external environment and the alveoli of the lungs), transport of gases by blood from the alveoli to cell membranes and back, tissue respiration, during which oxygen is utilized by tissues and carbon dioxide is released.

The normal function of external respiration is determined by airway patency, alveolar ventilation, the state of blood flow in the lungs and diffusion. An essential point in maintaining airway patency is the drainage function inherent in all levels of the bronchial tree. Violation of this function leads to an increase in resistance, a violation of the movement of air flows and, ultimately, requires increased work of the respiratory muscles. The volume of ventilation is regulated by the respiratory center, which is sensitive to reflex,

humoral and mental stimuli. Alveolar ventilation is important, which is interconnected with the respiratory rate, since with an increase in the respiratory rate, the specific value of the ventilation of the dead space increases. It is convenient to control the adequacy of ventilation by voltage O_2 and $C0_2$in arterial blood. The nature of the circulation of the lungs (shunting from right to left) with the discharge of venous blood into the left heart determines the adequacy of blood arterialization. Due to the fact that SO_2can be released into the alveoli in unlimited volumes, and oxygen can be absorbed depending on the blood passing through the alveoli, the shunting mechanism cannot be compensated by the absorption of O_2. The mechanism of shunting is reflected in the change in the gas composition of the blood in the form of a decrease in PaO_2at normal or reduced voltage of carbon dioxide. The thickening of the alveolocapillary membrane impairs the permeability of gases through it, primarily O_2(diffusion capacity of $CO_2$20 times higher than O2). At the stage of gas transport, the determining processes are their solubility in plasma, the ability to enter into a chemical reaction with hemoglobin and diffusion. In transport $CO_2$the buffer system of the blood is involved. The solubility of gases in plasma does not play an important role in the transport of gases, but the tension of the gas is important for its diffusion transfers through permeable partitions.

The final stage of respiration consists in the transfer of electrons to molecular oxygen and oxidative phosphorylation. For the normal course of respiration, a sufficient amount of substrates and the active state of respiratory enzymes are necessary.

In addition to gas exchange between the blood and the atmosphere, the lungs participate in many metabolic processes only indirectly related to gas exchange or not at all related to it. The role of the non-respiratory functions of the lungs is to mechanically, physically and biochemically process the air entering the body and the blood circulating in it. Air purification is performed both by the respiratory tract and by the alveolar tissue. The air is purified from mechanical impurities, infections, toxic gases and allergens. The main role is played by mucociliary and cough mechanisms, as well as alveolar macrophages, which are involved in inflammatory reactions and secrete enzymes, modulators of immune responses, etc. Non-respiratory functions of the lungs include hemostasis and fibrinolysis. The lungs synthesize prostacyclin, thromboxane A2. The lungs are involved in the metabolism of proteins and fats. The production of surfactant by alveolar cells ensures normal

lung ventilation. Water-salt and heat exchange is one of the most important non-respiratory functions of the lungs. By perspiration, about 500 ml of water is removed from the lungs, which enters the alveoli from the pulmonary circulation. Thermal and liquid air conditioning is carried out in both the upper and lower airways. The degree of warming of the air in the respiratory tract depends on its temperature and ventilation mode. The lungs are actively involved in the metabolism of histamine and serotonin.

In the lungs, there are two circulatory systems: bronchial blood flow and pulmonary - "small circle" of blood circulation, significantly different from the "large". The driving force of pulmonary blood flow is the difference in pressure in the right ventricle and the left atrium, and the main regulatory mechanism is pulmonary vascular resistance. The normal value of pulmonary vascular resistance is 0.2 kPa/l min. Pulmonary blood flow disorders can be of three types:

1. Pulmonary blood flow may be impaired due to macro- and microemboli.

2. Pulmonary vasculitis causing lung ischemia.

3. Pulmonary arterial hypertension.

One of the most important pathological consequences of impaired pulmonary blood flow is pulmonary ischemia. It is known that the bronchial vessels feed the lung, and the pulmonary vessels are designed for gas exchange of the whole organism. Bronchial blood flow is only 1-3%. cardiac output, and for its own nutrition of the lungs, 1/7 of the total volume of pulmonary blood flow is consumed. A decrease in pulmonary blood flow by more than 7 times can be observed with pulmonary embolism, severe cardiogenic, anaphylactic, hemorrhagic, and other types of shock. When pulmonary blood flow is reduced so much that the lung's own metabolism is not ensured, tissue ischemia occurs, accompanied by a decrease in the formation of substances, the destruction of many biological substances suffers, tissue permeability increases and interstitial edema occurs, surfactant production decreases,

The diffusion of gases through the alveolo-capillary membrane is provided by the difference in their partial pressures on both sides of the membrane. The volume of diffusible gas is mainly affected by the diffusion surface area and the amount of pulmonary blood flow. The solubility of oxygen in

membrane tissue is 20 times smaller than CO_2, in connection with which$_2$diffuses more slowly.

The general ratio of ventilation and blood flow, which ensures the normal gas composition of arterial blood, should be 4:5 (0.8). The normal state of pulmonary functions is the unevenness of their intrapulmonary distribution. It depends on the action of gravity and other causes and can be regional, layered, parallel, asynchronous, etc. JB West systematized the data of many researchers and showed that regional differences in ventilation-perfusion ratios in the lungs depend on a combination of four types of intrapulmonary pressure: alveolar (P_A), arterial (Pa), venous (P_V), interstitial (Pi). Conventionally, four zones of pulmonary blood flow are distinguished, more or less passing one into another. In zone I (the uppermost) the blood pressure is lower than the alveolar one and conditions can be expressed as P_A> Pa > P_V. There is practically no blood flow in this zone, since easily compressible vessels under the action of alveolar pressure do not allow blood to pass through. Below zone I, arterial pressure already exceeds alveolar pressure, and the conditions for pulmonary blood flow in zone II are determined by the ratio Pa > P_A>P_V. The driving force of pulmonary blood flow in this zone is the difference between arterial and alveolar pressure, which increases rapidly, accompanied by an increase in pulmonary blood flow. In zone III, the rate of increase in blood flow decreases, since the venous pressure in it has increased so much that it exceeds the alveolar one, and the conditions of pulmonary blood flow depend on the ratio P_A>P_V> pa. The capillaries in this zone are dilated, the shunting effect is pronounced. In zone IV, the same relationships exist, but perfusion decreases again due to a local increase in interstitial pressure on the precapillary sphincters. Regional differences in lung ventilation cause a more frequent development of the inflammatory process and atelectasis in the lower lung zones. Not only the so-called stagnant blood flow plays a role, but mainly the occurrence of expiratory closure of the airways, mainly in the lower zones. Due to this, when coughing at this position of the patient, the lung zones are not emptied of sputum, and airway obstruction occurs in these zones. Taking into account regional differences in lung function with lesions of one lung, it is recommended that the position on the side is when the diseased lung is on top.

1. Effective alveolar volume, in which there is an adequate ratio of ventilation and blood flow, optimal figures for PaO_2; 2. Aerodynamic component associated with the timing of ventilation of individual alveoli due to different lengths of the airways, their unequal resistance, differences in intrapulmonary gaseous diffusion and convection;

3. Diffusion component, depending on the thickening of the alveolar capillary membrane and similar in nature to the alveolar shunt;

4. Alveolar dead space, the gas of which is not exchanged with capillary blood, but enters the total tidal volume entering the lungs;

5. Alvolar shunt, which transmits venous blood into a large circle, which does not exchange with alveolar air;

6. Anatomical dead space, which is filled in front of the entrance with exhaust air and introduces this air into all components except the alveolar shunt. All lung processes, pressures, volumes and volumetric velocities have a vertical gradient for a given body position.

In pathological conditions, alveoloarterial differences are associated with three physiological mechanisms: uneven ventilation-perfusion ratios, violation alveolocapillary diffusion of gases and shunting of venous blood. The ultimate expression of uneven lung function is the magnitude of respiratory dead space (DMP) and alveolar shunt. Respiratory dead space (DMP) - part of the ventilated gas that has not entered into gas exchange with the blood of the pulmonary capillaries. DMP consists of anatomical and alveolar volumes. Anatomical DMP is the volume of airways in which there are no pulmonary capillaries and therefore there is no gas exchange with the pulmonary circulation. Alveolar DMP is a part of the volume of alveolar ventilation that has not entered into gas exchange with the blood of the pulmonary capillaries. Physiological DMP is a calculated value obtained from the Bohr-Enghof equation and characterizing the part of the ventilation volume that has not entered into gas exchange with blood.

Alveolar shunt - shunting of blood from right to left (veno-arterial shunting) occurs through direct anastomoses between the veins and arteries of the lungs, the confluence of the Tebesian veins into the left atrium and

bronchial veins into the pulmonary artery through pathological fistulas in the heart and large vessels and through the blood flow preserved in atelectasis. The predominance of blood flow over ventilation can be a source of admixture of venous blood from a small circle into a large one. Normal values of the alveolar shunt do not exceed 5-7% of the minute volume of blood circulation.

Table 1.

Ideal values of partial gas pressure in mm Hg.

Index	Inhaled air	Alveolar gas	Exhaled air
RaO_2	159	100	116
$PaCO_2$	0.3	40	32

The oxygen reserves of the body are small: in the lungs - 370 ml, in the arterial blood - 280 ml, in the venous - 600 ml, in the muscles - 240 ml, etc. tissue - 56 ml. The total oxygen capacity of the body is 1.5 liters. Since at rest the body consumes 250 ml of O2 per minute, the maximum lifespan does not exceed 7 minutes. If 100% 02 is inhaled beforehand, then the 02 reserves in the lungs are 2352 ml, in the arterial blood - 297 ml, in the venous blood - 608 ml, that is, only 3257 ml (only twice as much). The change in gases in the lungs is ideally presented in Table. 1. Thus, the inadequacy of the lungs as a gas exchange device or damage to transport 0_2system of blood and circulation will lead to hypoxia in a matter of minutes.

According to the physiological mechanism, hypoxia is usually divided into 4 forms:

1. Hypoxic (respiratory) - the result of disorders of the pulmonary apparatus (pathology of ventilation, diffusion, shunt, etc.), a characteristic feature is low PaO_2;

2. Circulatory - occurs when there is a violation of transport 0_2 blood flow, i.e. with ischemia or congestive plethora of organs, a characteristic sign is an increase in the arteriovenous difference O_2.

3. Hemic - associated with a lack of hemoglobin (anemia) or its inability to bind 0_2(poisoning with cyanides, carbon monoxide, etc.); characteristic feature - high $P0_2$at low content 0_2.

4. Histotoxic (tissue) - the inability of tissues to utilize 0_2in connection with the defeat of enzymatic or energy systems; a characteristic feature is a sharp reduction in the arteriovenous difference O_2. Thus, already by comparing the voltage and the content of 0_2in arterial and venous blood, one can judge the type of hypoxia and correctly prescribe treatment.

Relationship between RO_2(voltage) and HNO_2(hemoglobin saturation) is expressed by the oxyhemoglobin dissociation curve (ODC), which has an S-shape and characterizes the affinity of hemoglobin for oxygen. This ability is expressed by the value P50 - partial voltage O_2at which 50% Hb is bound to O_2at pH 7.4 and temperature - 37 °C. The normal value of P50 is 27 mm Hg. Art. Shift of KDO to the right means a decrease in the ability of Hb to bind O_2 and, therefore, is accompanied by an increase in P50; when mixing DRC to the left, the phenomena are reversed. Hb in the body is about 600 g. The affinity of hemoglobin and oxygen depends mainly on pH: the higher it is, the less the ability of Hb to bind $O_2$and the more P50. changes in pH by 0.1 less than P50 by 2.5 mm Hg. Art. In tissues Hb easily gives $0_2$and actively absorbs it in the lungs. The higher the temperature, the lower the affinity of Hb and $O_2$and the more oxygen hemoglobin gives to the tissues. During hyperthermia, tissues receive more $O_2$and vice versa for hypothermia.

Under the influence of hypoxia, the permeability of the brain membranes increases, and its edema develops. Clinical manifestations - euphoria, irritability, convulsions, coma.

In the myocardium, the main part 0_2spent on its reduction. During hypoxia, energy decreases, excitability, conductivity, myocardial contractility are disturbed, necrobiosis and fatty degeneration of the myocardium occur.

In the lungs, vasoconstriction, interstitial edema occur, surfactant production decreases, and extensibility decreases. Hypertension in the small circle leads to right ventricular failure.

In the liver, central necrosis develops, ferritin is released, which increases the resistance to portal blood flow.

AT kidneys arises ischemic necronephrosis due to catecholaminemia, which spasms the arteries and disrupts blood flow in the microcirculation systems. This is where rheological disturbances begin.

blood properties that lead to hypovolemia and, consequently, to circulatory hypoxia, closing the vicious circle - respiratory hypoxia - circulatory hypoxia. The growth of underoxidized products leads to an increase in the number of H+ ions, and metabolic acidosis develops. The main products of metabolites in the cell are acids that dissociate with the release of active H+ ions, the intracellular fluid is oxidized, and some of the ions are neutralized by the cell's buffer system. When the concentration of hydrogen ions exceeds the power limit of the cellular buffer system, they leave the cell together with Na+ and HCO3- ions (the "sodium pump" mechanism). In the intercellular environment, H + ions come into contact with the buffer system of the tissue fluid, then the pulmonary and renal compensation mechanisms are activated,$_2$, i.e. metabolic acidosis is accompanied by respiratory alkalosis. Ventilation maintains the partial tension of carbon dioxide in arterial blood at the level of 35-45 mm Hg. Art.

Literature

1. Shik I.I. Guide to the clinical physiology of respiration. "Medicine", Leningrad, 1980

2. Satishur, O.E. Mechanical ventilation of the lungs. – M.: Med. lit., 2006. - 352 p.

3. Jean Louis Vincent. Guide to Critical Medicine. 7th edition. Volume #1. Publisher: Chelovek, 2019 - 951 p.

CLINICAL PHYSIOLOGY OF RESPIRATION

Understanding the process of breathing is a key step to understanding the essence and tactics of managing acute respiratory failure. The control of respiratory function is represented by the close coordination of three groups of neurons in the medulla oblongata: the dorsal respiratory center, which controls inspiration; the ventral respiratory center, which controls exhalation; and the pneumotactic center, which controls the frequency and depth of respiration. In addition to the brainstem neurons, there is a system of peripheral chemoreceptors located outside the brain in the form of carotid cells and aortic cells that respond to minor changes in PaO_2. Nerve impulses from the central nervous system travel through the spinal cord and motor neurons, reaching and activating the diaphragm and other respiratory muscles. The contraction of the respiratory muscles creates negative pressure in the pleural cavity due to the expansion of the chest and shifts down the contents of the abdominal cavity. In the chest, the negative pressure created during inspiration leads to a decrease in pressure in the alveoli below atmospheric pressure, forming a gradient for the flow of inhaled air towards the alveoli. The oxygen-rich inhaled air makes it possible to diffuse $O_2$from the alveoli into the blood through the alveolo-capillary membrane, where the deoxygenated hemoglobin is saturated with oxygen and becomes a form of oxyhemoglobin.

Breathing in the broadest sense of the word - the exchange of gases of the body with the external environment, necessary to maintain energy metabolism. The function of respiration is provided by three successive stages: external respiration (the exchange of gases between the external environment and the alveoli of the lungs), the transport of gases by blood from the alveoli to the cell membranes and back, tissue respiration, during which oxygen is utilized by the tissues and carbon dioxide is released.

The normal function of external respiration is determined by the patency of the airways, alveolar ventilation, the state of blood flow in the lungs, and diffusion. The essential point of maintaining airway patency is the drainage function inherent in all levels of the bronchial tree. Violation of this function leads to an increase in resistance, disruption of the movement of air flows and, ultimately, requires increased work of the respiratory muscles. The volume of ventilation is regulated by the respiratory center, which is sensitive to reflex, humoral, and mental stimuli. Important is alveolar ventilation, which is interrelated with the breathing rate, since with an increase in the breathing rate, the specific value of ventilation of the dead space increases. The adequacy of ventilation is conveniently monitored by the voltage of $O_2$and $C0_2$in the arterial blood. The nature of the blood circulation of

the lungs (right-to-left bypass surgery) with the discharge of venous blood into the left heart, it determines the adequacy of blood arterialization. Due to the fact that CO_2can be released into the alveoli in unlimited volumes, and oxygen can be absorbed depending on the blood passing through the alveoli, the bypass mechanism cannot be compensated for by the absorption of O_2. The mechanism of bypass surgery is reflected in the change in the gas composition of the blood in the form of a decrease in $RaO_2$with normal or reduced carbon dioxide voltage. The thickening of the alveolocapillary membrane disrupts the permeability of gases through it, primarily O_2(the capacity diffusion of $CO_2$is 20 times higher than that of O_2). At the stage of gas transport, the determining processes are their solubility in plasma, the ability to enter into a chemical reaction with hemoglobin, and diffusion. The blood buffer system is involved in the transport of CO_2. The solubility of gases in plasma does not play a large role in the transport of gases, but the gas voltage is important for its diffusion transport through permeable partitions.

The final stage of respiration consists in the transfer of electrons to molecular oxygen and oxidative phosphorylation. For the normal course of respiration, a sufficient amount of substrates and the active state of respiratory enzymes are necessary.

In addition to the gas exchange between the blood and the atmosphere, the lungs participate in many metabolic processes only indirectly related to gas exchange or not related to it at all. The role of the non-respiratory functions of the lungs is to subject the air entering the body and the blood circulating in it to mechanical, physical and biochemical treatment. Air purification is performed by both the respiratory tract and the alveolar tissue. The air is cleaned of mechanical impurities, infections, toxic gases and allergens. The main role is played by mucociliary and cough mechanisms, as well as alveolar macrophages, which are involved in inflammatory reactions and secrete enzymes, modulators of immune responses, etc. Non-respiratory lung functions include hemostasis and fibrinolysis. The lungs synthesize prostacyclin, thromboxane A_2. The lungs are involved in the metabolism of proteins and fats. The production of surfactant by alveolar cells ensures normal ventilation of the lungs. Water-salt and heat exchange is one of the most important non-respiratory functions of the lungs. By perspiration, about 500 ml of water is removed from the lungs, which enters the alveoli from the small circle of blood circulation. Thermal and liquid air conditioning is carried out in both the upper and lower respiratory tracts. The degree of warming of the air in the respiratory tract depends on its temperature and ventilation mode. The lungs are actively involved in the metabolism of histamine and serotonin.

In the lungs, there are two circulatory systems: the bronchial blood flow and the pulmonary-the "small circle" of blood circulation, which is significantly different from the "large" one. The driving force of pulmonary blood flow is the difference in pressure in the right ventricle and left atrium, and the main regulatory mechanism is pulmonary vascular resistance. The normal value of pulmonary vascular resistance is 0.2 kPa / l min. Disorders of pulmonary blood flow, can be of three types:

1. Pulmonary blood flow may be impaired due to macro-and microembolism.

2. Pulmonary vasculitis, causing lung ischemia.

3. Pulmonary arterial hypertension.

One of the most important pathological consequences of pulmonary blood flow disorders is lung ischemia. It is known that the bronchial vessels feed the lung, and the lungs are designed for gas exchange of the entire body. Bronchial blood flow is only 1-3%. cardiac output, and for its own nutrition of the lungs, 1/7 of the total volume of pulmonary blood flow is consumed. A decrease in pulmonary blood flow by more than 7 times can be observed in pulmonary embolism, severe cardiogenic, anaphylactic, hemorrhagic, and other types of shock. When the pulmonary blood flow is reduced so much that the own metabolism of the lungs is not provided, there is tissue ischemia, accompanied by a decrease in the formation of substances, the destruction of many biological substances suffers, the permeability of the tissue increases and interstitial edema occurs,

The diffusion of gases through the alveolo-capillary membrane is ensured by the difference in their partial pressures on both sides of the membrane. The volume of diffused gas is mainly affected by the area of the diffusion surface and the amount of pulmonary blood flow. The solubility of oxygen in the membrane tissue is 20 times less than that of CO_2, and therefore O_2diffuses more slowly.

The total ratio of ventilation and blood flow, which provides a normal gas composition of arterial blood, should be 4:5 (0.8). The normal state of pulmonary functions is the unevenness of their intra-pulmonary distribution. It depends on the action of gravity and other causes and may be regional, layered, parallel, asynchronous, etc. West systematized the data of many researchers and showed that regional differences in ventilation-perfusion ratios in the lungs depend on a combination of four types of intra-pulmonary pressure: alveolar (P_A), arterial (Pa), venous (P_V), interstitial (Pi). Conventionally, there are four zones of pulmonary

blood flow, more or less passing into one another. In zone I (the uppermost), blood pressure is lower than alveolar and conditions can be expressed as P_A> Pa > P_V. In practice, there is no blood flow in this zone, since easily compressible vessels under the influence of alveolar pressure do not pass blood. Below zone I, the blood pressure already exceeds the alveolar pressure, and the conditions for pulmonary blood flow in zone II are determined by the ratio Pa > P_A>P_V. The driving force of pulmonary blood flow in this zone is the difference in arterial and alveolar pressure, which increases rapidly, accompanied by an increase in pulmonary blood flow. In zone III, the rate of increase in blood flow decreases, since the venous pressure in it has increased so much that it exceeds the alveolar one, and the conditions of pulmonary blood flow depend on the ratio P_A>P_V> pa. The capillaries in this area are dilated, and the shunt effect is pronounced. In zone IV, the same ratios exist, but perfusion is again reduced due to a local increase in interstitial pressure on the precapillary sphincters. Regional differences in lung ventilation cause more frequent development of the inflammatory process and atelectasis in the lower lung zones. Not only does the so-called stagnant blood flow play a role, but mainly the occurrence of expiratory closure of the airways mainly in the lower zones. Due to this, when coughing in this position of the patient, the areas of the lungs are not emptied of sputum, and there is an obstruction of the airways in these areas. Taking into account the regional differences in lung function in cases of lesions of one lung, it is recommended to have a position on the side, when the patient's lung is on top. The gas composition of the blood flowing from the lungs is the result of the mutual existence of several components of intra-pulmonary gas exchange:

1. The effective alveolar volume, in which there is an adequate ratio of ventilation and blood flow, the optimal figures of RaO_2; 2. The aerodynamic component associated with the different timing of ventilation of individual alveoli due to the different length of the airways, their unequal resistance, differences in intra-pulmonary gas diffusion and convection;

3. The diffusion component, which depends on the thickening of the alveolocapillary membrane and is similar in nature to the alveolar shunt;

4. Alveolar dead space, the gas of which is not exchanged with capillary blood, but enters the total respiratory volume entering the lungs;

5. Alveolar shunt, which transmits venous blood to the large circle, which does not exchange with the alveolar air;

6. Anatomical dead space, which is filled in front of the entrance with exhaust air and introduces this air into all components except the alveolar shunt. All lung processes, pressures, volumes, and volume velocities have a vertical gradient for a given body position.

In pathological conditions, alveoloarterial differences are associated with three physiological mechanisms: uneven ventilation-perfusion ratios, impaired alveolocapillary gas diffusion, and venous blood shunting. The final expression of the uneven lung function is the size of the respiratory dead space (RDS) and the alveolar shunt. The respiratory dead space (RDS) is a part of the ventilated gas that has not entered into gas exchange with the blood of the pulmonary capillaries. The DMP consists of anatomical and alveolar volumes. Anatomical RDS is the volume of airways in which there are no pulmonary capillaries and therefore there is no gas exchange with a small circle of blood circulation. Alveolar RDS is a part of the volume of alveolar ventilation that has not entered into gas exchange with the blood of the pulmonary capillaries.

Alveolar shunt-the discharge of blood from right to left (veno-arterial bypass) occurs through direct anastomoses between the veins and arteries of the lungs, the areas of the confluence of the tebesian veins in the left atrium and the bronchial veins in the pulmonary artery through pathological anastomoses in the heart and large vessels and through the blood flow preserved in atelectasis. The predominance of blood flow over ventilation can be a source of mixing of venous blood of the small circle in the large one. Normal values of the alveolar shunt do not exceed 5-7% of the minute volume of blood circulation.

Table 1.

Ideal values of the partial pressure of the gas in mmHg

indicator	Inhaled Air	Alveolar Gas	Exhaled Air
RaO_2	159	100	116
$PaCO_2$	0.3	40	32

Oxygen reserves of the body are small: in the lungs-370 ml, in arterial blood-280 ml, in venous-600 ml, in muscles-240 ml, and other tissues-56 ml. The total oxygen capacity of the body is 1.5 liters. Since the body consumes 250 ml of O_2 per minute at rest, the maximum life span does not exceed 7 minutes. If 100% 02 is pre-inhaled, then the reserves of 0_2 in the lungs are 2352 ml, in arterial blood - 297 ml, in venous blood-608 ml, ie only 3257 ml (only twice as much). The change in the gases in the lungs is ideally presented in Table 1. Thus, the inadequacy of the lungs as a gas exchange device or damage to the transport of oxygen by the blood and circulatory system will lead to hypoxia in a matter of minutes.

According to the physiological mechanism, hypoxia is usually divided into 4 forms:

1. Hypoxic (respiratory) - the result of disorders of the pulmonary apparatus (pathology of ventilation, diffusion, shunt, etc.), a characteristic feature-low RaO_2;

2. Circulatory-occurs when there is a violation of the transport of oxygen by the blood flow, ie, with ischemia or stagnant fullness of the organs, a characteristic feature is an increase in the arteriovenous difference of O_2.

3. Hemic - associated with a lack of hemoglobin (anemia) or its inability to bind 0_2 (cyanide poisoning, carbon monoxide, etc.); a characteristic feature - high $P0_2$ with a low content of 0_2.

4. Histotoxic (tissue) - the inability of tissues to utilize 0_2 due to damage to the enzymatic or energy systems; a characteristic feature is a sharp reduction in the arteriovenous difference of O_2. Thus, already by comparing the voltage and the content of 0_2 in the arterial and venous blood, it is possible to judge the type of hypoxia and correctly prescribe treatment.

The relationship between PO_2 (voltage) and HBO_2 (hemoglobin saturation) is expressed by the oxyhemoglobin dissociation curve (BDO), which has an S - shape and characterizes the affinity of hemoglobin to oxygen. This ability is expressed by the value P50 – the partial voltage of O_2 at which 50% of H is bound to O_2 at a pH of 7.4 and a temperature of -37 °C. The normal value of P50 is 27 mm Hg. The shift of BWW to the right means a decrease in the ability of HB to bind O2 and, therefore, is accompanied by an increase in P50; when mixing BWW to the left, the phenomena are reversed. NI in the body about 600 g. The affinity of hemoglobin and oxygen depends mainly on the pH: the higher it is, the lower the ability of HH to bind O_2 and the greater the P50. changes in pH by 0.1 less than P50 by 2.5 mm Hg. In the tissues of HH, it easily gives off 0_2, and in the lungs it

actively absorbs it. The higher the temperature, the lower the affinity of H and O_2, and the more oxygen the hemoglobin gives to the tissues. With hyperthermia, the tissues receive more O_2, and with hypothermia-on the contrary.

Under the influence of hypoxia, the permeability of the brain membranes increases, its edema develops. Clinical manifestations - euphoria, increased excitability, convulsions, coma.

In the myocardium, the main part of the $0_2$is spent on its reduction. With hypoxia, energy decreases, excitability, conduction, contractility of the myocardium is disturbed, necrobiosis and fatty degeneration of the myocardium occur.

In the lungs, vasoconstriction, interstitial edema occur, the production of surfactant decreases, and the extensibility decreases. Hypertension in the small circle leads to right ventricular failure.

Central necrosis develops in the liver, and ferritin is released, which increases resistance to portal blood flow.

In the kidneys, ischemic necronephrosis occurs due to catecholaminemia, which spasms the arteries and disrupts blood flow in the microcirculation systems. With this, violations of the rheological properties of the blood begin, which lead to hypovolemia and, consequently, to circulatory hypoxia, closing the vicious circle - hypoxia respiratory - circulatory hypoxia. The growth of under-oxidized products leads to an increase in the number of H+ ions, and metabolic acidosis develops. The main products of metabolites in the cell are acids, which dissociate with the release of active H+ ions, the intracellular fluid is oxidized, and some of the ions are neutralized by the buffer system of the cell. When the concentration of hydrogen ions exceeds the power limit of the cell buffer system, they leave the cell together with Na+ and HCO_3-ions (the "sodium pump" mechanism). In the intercellular medium, H+ ions come into contact with the buffer system of the tissue fluid, then the pulmonary and renal compensation mechanisms are activated, and the concentration of H+ in the extracellular fluid is leveled While maintaining the ability of the lungs and kidneys to remove carbon dioxide, and the central nervous system - to the normal regulation of respiration. The shift in the pH of the blood to the acidic side is accompanied by stimulation of the respiratory center, hyperventilation and a decrease in $RaSO_2$, ie, respiratory alkalosis joins the metabolic acidosis. Ventilation maintains the partial voltage of carbon dioxide in the arterial blood at the level of 35-45 mm Hg.

Literature

1. Shik II Manual of clinical respiratory physiology. "medicine" Leningrad, 1980.

2. Satishur, OE Mechanical ventilation of the lungs. - M.: Med. lit., 2006 - - 352p.

3. Jean-Louis Vincent. A guide to Critical Care Medicine. 7th edition. Volume #1. Publisher: Man, 2019 - 951 p.