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RESPIRATORY ORGANS CLINICAL LABORATORY DIAGNOSTICS LITERATURE REVIEW

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Abstract
This article gives us the opportunity to consider the main methods, object and features of laboratory diagnostics of diseases of the respiratory system. The structure of the lungs provides the function of gas exchange. In the study of the anatomical structure of the lungs, it is necessary to give special attention to the study of gas exchange in the lungs (study of its activity at the cell level). Microscopic examination of sputum is relevant in the diagnosis and diphdiagnostics of most diseases.

Keywords: Lungs, sputum, breathing, diagnostics, laboratory, elastic fibers, differential diagnosis.

Introduction

The structure of the lungs provides the function of gas exchange. In the study of the anatomical structure of the lungs, it is necessary to give special attention to the study of gas exchange in the lungs (study of its activity at the cell level). The surfactant substance is involved in the exchange of gases in the lungs. It is synthesized in the lungs.

Energy processes in lung tissue

In order to maintain a functional system in the lungs, the energy generated during the exchange of substances is required. The main space of energy is characterized by mitochondria. Pulmonary mitochondria differ from mitochondria of other tissues in their enzyme activity and distribution of enzymes. Thus, pyruvate is present in the pulmonary mitochondria (90%) of phosphate transferase, and in the liver in the soluble fraction of the cytoplasm (90-96%). [1, 2, 8, 10-14, 17].

In lung mitochondria, 60% of pyridine nucleotides are NADH-shaped-NAD is 6-8 times slower compared to liver, and a-glycerophosphate and Malate are oxidized 5-10 times faster. The energy system of the pulmonary mitochondria responds to the speed of blood flow in the lung tissue and its replenishment with air. In cases of complete filling of the lungs with air, glycolysis occurs more intensively and more ATF is formed. With a low blood flow rate, the energy supply of cells, as well as the synthesis of adenyl nucleotides, decreases. In the case of severe hypoxia in the lungs, there is a decrease in mitochondrial superoxidismutase activity.

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A measure of metabolic activity indicates the level of oxygen use. The concentration of ATF in lung tissue is the same as in other tissues. The lungs synthesize 57 to 174 mmol ATF per 1 g of tissue in 1 hour. One of the main factors that determine the violation of biochemical processes in lung tissue in the case of bronchopulmonary diseases is hypoxia. Impaired blood flow and lymph flow cause oxygen starvation in the affected areas of the lungs resulting in the development of respiratory failure. In the case of oxygen deficiency, increased lactate production in lung tissue is not only a result of glucose breakdown, but also of amino acid catabolism.

As a result of hypoxia, LDG 5 increases from LDG isopherments. There are also significant changes in the isoenzyme spectrum of MDG. [3, 4, 9, 10,14,17].

Chronic hypoxia increases the activity of glycolysis and glycogenolysis; at the same time, the concentration of ATF decreases, in response to which the genetic apparatus increases the number of mitochondria to restore ATF production.

Hypoxic conditions cause changes not only in lung tissue, but also in red blood cells. Hypoxia results in a level I and II respiratory failure in patients with pneumonia . In patients with bronchial asthma with Level II respiratory failure, the maximum return of oxygen to the tissues occurs during snoring, and the interval between snoring decreases. An increase in 2,3-DFG reduces the activity of G-6-FDG, while increasing the activity of carbonate hydratase (EC 4.2.1.1), which leads to disruption of the transport of oxygen by red blood cells. Subsequent restructuring of metabolic processes in red blood cells leads to the emergence of compensatory mechanisms of oxygen transport. In patients with a disease duration of more than 3 years, high G-6-FDG activity and low levels of carbonate hydratase are often isoenzyme changes in bronchial asthma with the blood of recorded patients; LDG increases, LDG3 and LDG4 decrease. In addition, as a result of an increase in blood glucose levels,an increase in the amount of free fatty acids, lactic acids, disorders in gas exchange in the lungs are observed.

Features of protein metabolism in the lungs:

The lungs perform their functions thanks to the content of proteins – collagen and elastin. Compared to other parenchymal organs, the amount of collagen in the lungs is the highest. These proteins ensure the consistency of the lung shape and facilitate their gas exchange. In some diseases emphysema and pulmonary fibrosis - changes in the structure and properties of these proteins are observed. In the physiological processes of the lungs, the main achamiat; proteins in the composition of the surfactant and bronchogenic secretions. [2, 5, 8, 10,12,16].

Collagen is a fibrillar protein that localizes in ribosomes, having a weight of M-270,000 nm, 290 nm, consisting of three helical molecules. The compound of 5-8 monomers forms fibrillar filaments. Proteoglycans help form collagen fibers and collagen filament bundles. Five types of lung collagens are distinguished, they differ in the composition of the last residues of the chains.

Elastin is a fibrillar protein-a special elastin with two components and is divided into structural glycoprotein. Ellastine is characterized by the presence of a large number of non-polyar amino acid residues, such as glycine (about 30%), alanine (24%), valine,phenylalanine, isoleucine, and leucine.Glycoprotein contains a large amount of carbohydrates and cysteine,while desmozine and oxypolin do not.

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In emphysema,the concentration of elastin in the lungs decreases(in the range of 9.0-9.9%), in healthy people it is 30-35%; in children, on average, 7.32%. When there is an imbalance in the system of proteolysis and ingibir enzymes,the breakdown of ellastine is observed. The protease inhibitor is gamma-antitrypsin.People who lack the gene responsible for the synthesis of Gamma-antitrypsin, then, with the development of emphysema, fall into the risk group for lung diseases.

The second protein is a protease inhibitor that belongs to α2-macroglobulin,the α2-globulin fraction, and weakens the activity of all four catalytic classes: these are siren-containing, thiol-containing, carboxyl-containing, and metaloprotease. The biological role of A2-macroglobulin consists in regulating the functioning of the complement system, regulating the tone of the vessels and inflammatory reactions. A decrease in the concentration of A2-macroglobulin is observed in lung disease, especially bronchial asthma.

In patients with chronic obstructive bronchitis in the acute period, phlegm levels of A2-macroglobulin range from 2.7 to 1009 mg / l, with a concentration of inhibitors of 15-35 mg / l (normal 22-14 mg / l). In whey, A2-macroglobulin enters the mucus as a result of increased cell wall permeability. Thus, the ratio of proteases and inhibitors is important in the development of pulmonary pathology. [3, 4, 9, 10,11].

Elastic fibers in the form of thin pink threads; painted with eosin (Figure 1)

The importance of proteoglycans and glycoproteins in the lungs;the substances between the connective tissue have a gel consistency. A typical proteoglycan molecule consists of a nuclear polypeptide chain with glycosaminoglycans attached to its sides. The carbohydrates contained in proteoglycans have a negative charge, which determines their main role in regulating water-salt metabolism, as well as the ability to enter complexes with collagen protein and calcium ions.

Heparin is a glycosaminoglycan synthesized by basophils of connective tissue, has an anticoagulant effect, thinning blood clotting due to its ability to form complexes with many proteins of the blood clotting system. The concentration of heparin in the lungs is much higher. Keratansulfate is involved in the formation of the skeleton of the lungs, the amount of which increases with age, which leads to a decrease in elasticity. The intercellular fluid also contains glycoproteins with up to 15% carbohydrate residues. They are poorly soluble but have high antigenic properties. They belong to fibronectin, many of which are located in the liquid on the surface of the cells. [3, 4, 9,7,14,10].

Lipids in pulmonary pathology

In diseases of the respiratory system, lipids are aimed at studying surfactant, lung tissue, bronchoalveolar lavage and serum lipids. Lipids play an important role in determining the maturation stage of surfactant. There are a number of enzymes for the synthesis of fatty acids, triglycerides and cholesterol in the lungs, which also contain lipolytic enzymes: phospholipases, lipoprotein lipases, diacylglycerol-triglycerolipase. Lipoprotein lipase in the lungs comes in two forms: soluble and membrane-bound, which differ from each other in pH (7.5 and 9) and are inhibited by protamine sulfate.Phospholipase activity in lung tissue is higher than in the liver. Phospholipase a 2 is mostly soluble and in an inactive state.

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The lung is an organ that is the first pathway of chylimicrons. The lungs act as a buffer that regulates lipids in the blood. In addition, lipids are necessary for the synthesis of active substances consisting of cholesterol (8%), mono, di and triglycerides (4%), phosphatidylcholine (66%), phosphatidylethanolamine (5%), phosphatidylglycerol, phosphatidylserine (4%). A feature of lipid synthesis in lung tissue is the formation of active lipids, especially phospholipids. The metabolism of other lipids occurs in the same way as in other organs. In hypoxia, the use of free fatty acids of lung tissue, phosphatidylcholine is reduced. Low oxygen partial pressure determines hypoxia in lung tissue, which leads to a decrease in the processes of transmethylation and acetylation and a violation of the synthesis of phospholipids. The Krebs cycle decreases and increases the concentration of predetermined fatty acid oxidation products, in the case of hypoxia, not only metabolic processes and ATF production decrease, but also damage to membranes and impaired cell activity.

Biologically active substances exchange

Special pulmonary metabolic processes that do not perform the main respiratory function in the lungs. The endogenous pulmonary filter or pulmonary barrier is cisobized. They are associated with a number of biologically active substances (BAMS). They include serotonin, cotecholamine, acetylcholine, histamine, and vasoactive peptide.

The study of the metabolic function of the lungs with their various pathologies makes it possible to distinguish three different metabolic changes: the first is associated with an increase in the concentration of biologically active substances in tissues, which is accompanied by an increase in the activity of their catabolism enzymes. This type occurs in cases of acute stress; the latter - with an increase in the concentration of biologically active substances, which is accompanied by a decrease in the activity of catabolic processes. This type is observed in the case of hypoxia and chronic bronchopulmonary processes. The third type is characterized by a lack of biologically active substances in the lung tissue, which is accompanied by a decrease in the activity of catabolic enzymes. This is observed during a long (more than 20 years) period of bronchoectatic disease.

When observed in some pathological conditions, there will be changes in the metabolism of monoamines and acetylcholinan. Hypoxia is accompanied by increased activity of monoamine oxidase (MAO). Ischemia of short-term organs causes an increase in enzyme activity and determines long-term ischemia. Increased oxidative deamination has been reported in patients with bronchoectasis.

Thus, a decrease in MAO (monoaminooxydase) activity is one of the reasons why the metabolic function of the lungs is disrupted by serotonin and norepinephrine, leading to an increase in their concentration in the blood. Such cases include ischemia of the lungs, a chronic inflammatory bronchopulmonary process, in which the activity of this enzyme decreases sharply.

Vasoactive peptides; the most studied kinins belong to them: bradykinin, calidine, methionyl Lysyl bradykinin. They are all formed from the kininogenic group represented by glycoproteins of the A2-globulin fraction. By Origin, kininogens are divided into plasma and tissue types. The lungs have sufficiently high quininogenic activity and contain sufficient amounts. Vasoactive peptides give a wide range of processes of biological action and relax smooth muscles, dilate the artery, affect microcirculation and maxillary circulation, affect bronchial matorics. Kinins not only

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affect microcirculation, but they also dilate arterioles and capillaries and determine the spasm of arteriovenous strains and venules, while increasing the permeability of the vascular walls. Thus, bradykinin can affect the smooth muscles of the bronchi not only directly, but also through the tickling property of adrenoreceptors located in the smooth muscles.

Kinins in the body are activated very quickly. A powerful enzyme system that breaks down bradykinin is located in the lungs. The pulmonary enzyme system can eliminate bradykinins or participate in the conversion of angiotensin I to angiotensin II. Active dipeptidyldipeptidase, is involved in this process.

In people with bronchial asthma,the accumulation of kinins increases, and with a decrease in kinase enzymes, the active inhibitors in the blood decrease in ham. [3, 4, 9, 10,14,17].

Histamine (3-imidazoldiethylamine) belongs to the group of biogenic amines and is formed from histidine. The site of histamine synthesis is located in the skin, the mucous membrane of the digestive canal and the lungs, which is present in tissue basophils. Unlike serotonin, norepinephrine, acetylcholine and radioquinine, which circulate in the blood, histamine does not disappear when passing through the lungs. The lungs contain enzymes that oxidize and methylate this Amine. Histamine inactivation can occur partially in the lungs because histidine methyltransferase activity is much higher than in other organs. Histamine is an unstable compound and quickly disappears. This increases the tone of the pulmonary vessels and, to a lesser extent, the tone of the pulmonary arteries. Histamine plays an important role in the development of bronchial asthma. This increases the tone of the bronchial muscles,provokes afferent vagus valocna or H1 receptors. In addition, it can enhance cholinergic and a-adrenergic bronchospastic effects or worsen smooth muscle relaxation that occurs when β-adrenergic receptors are excited.

Prostaglandins (PG) are unsaturated compounds containing a chain of 20 carbon atoms, some of which have been incorporated into the cyclopentane core. They are divided into 4 main groups-A, B, E, G. They are formed by the action of the enzyme prostaglandin synthetase, which is bound to the multenzyme membrane located in the lungs. Its synthesis occurs in endothelial cells of the lungs. The inhibitors of this synthesis are glucocorticosteroids, which block the activation of phospholipase a 2, which cleaves phospholipids to form free fatty acids. This is how arachidonic acid, the main precursor of PG, is released. Prostaglandins are involved in the formation of the tone of the smooth muscles of the bronchi. PGE has a bronchodilator effect, PG 1-dilates the capillaries of the lungs and reduces pressure in the pulmonary artery, PG2 determines hypoventilation, hyperventilation. The latter leads to the synthesis of PG E, which is accompanied by vasodilation and increased ventilation / perfusion ratio. [1, 4, 9, 10,14,17].

The lung is the main site of activation of immunoglobulins. Thus, for one circulation, 90-95% of the PG group is neutralized by the introduction of their blood at a concentration of 0.5-1 mg / L. Neutralization of primary prostaglandins consists of oxidation of the hydroxyl group at position 15. They are converted into inactive metabolites and are quickly washed out, passing from the lungs to the liver.

Some PGs undergo complete metabolism in the lungs, others are partial and PG a, PG V and prostacyclin PGI 2 are not completely removed from circulation. It should be noted that the lungs not only activate the IGs that circulate in the blood, but also those that synthesize themselves. It manifests as a protective reaction of the body.

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Thus, one of the chains of the neurogumoral regulation system of the physiological functions of the body is the lungs, which are involved in the maintenance of homeostasis of many BAMS.

The immune chimneys system collects lymph fluid from bronchial pulmonary aparate lymphoid cells,lymph nodes, and lymph nodes.The lymphoid tissue will be located on a long path, starting from the nasopharynx and continuing to the alveoli. In the lungs, the specific structural unit of the immunocompetent system is characterized by bronchial-associated lymphoid tissue similar to group lymphatic intestinal follicles. Lymph nodes are located along the mucous membrane, most of which are located in the branching areas of the bronchi. They are a reserve of immunocompetent cells that can migrate through the epithelium to the bronchi. The small bronchi contain a large amount of lymphoid tissue. Such a division of lymphatic tissue,the protection of the upper respiratory tract is guaranteed by a nospesific mechanism;xavoli filter,mucous membrane,function of the lashes, enzyme activity and other bronchial secretions. In the lower parts, these mechanisms practically do not work, and therefore it becomes possible to contact the surface of the respiratory tract with antigenic substances, which explains the increase in the amount of lymphoid tissue in the airways.

Biological role of bronchopulmonary secretion:

The surfactant system consists of both cellular and non-cellular components. To the cell component; consists of alveolar macrophages and alveocytes (Type I-III). To the non-cellular component; contains the alveolar surfactant complex, the surfactant of the alveolar passages, and bronchioles of order 1-3. The surfactant alveolar complex consists of surfactant, pituitary, and glycocalyx. This surfactant subunit is made up of two layers of lipid, which are made up of a lipid layer and a glyco,lipoprotein membrane.

A hypophase is a liquid phase placed under a surfactant. It complements cellular alveolar inequality and contains products of secretion of macrophages, reserved surfactants, osmophilic Ray bodies and their fragments of Type II alveocytes (AG-II). Surfactant contains 90% lipid, of which 85% -

phospholipids, 10% - triglycerides, 8% - cholesterol, 8% - fatty acids.

Glucocorticoid hormones of the adrenal glands are involved in the regulation of the surfactant system of the lungs. The surfactan pulmonary system performs several important functions. Surfactants reduce surface tension and, as a result,the work necessary for the air circulation of the lungs, stabilizes the alveoli and prevents their atelectases, increasing surface tension in breathing and decreasing during exhalation, so it is practically zero.Surfactant supply has the property of adapting to external muxite effects. Hypoventilation of the lung leads to the destruction of the film, and the surfactant film is not fully restored when the havo exchange process continues. The properties of the surfactant change in the case of hypoxia. In lung diseases, surfactant properties vary depending on the inflammatory process. The extent of these diseases depends on the activity of the inflammation. [1, 4, 7, 10,14,15].

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Conclusion:

During pathological processes in these sections of the lungs, alveolar epithelial cells and alveolar macrophages are located in the alveolar epithelium and cannot be separated, so it is more correct to characterize them as alveolar cells in sputum analysis.

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