ELATIONSHIP BETWEEN THE FUNCTION OF THE DIAPHRAGMATIC MUSCLES AND THE PERFUSION STATE OF THE LUNGS IN PATIENTS WITH CHRONIC OBSTRUCTIVE PULMONARY DISEASE COMPLICATED WHITH PULMONARY ARTERIAL HYPERTENSION

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Abstract

Study of the relationship between the function of the diaphragmatic muscles and the perfusion state of the lungs in patients with chronic obstructive pulmonary disease complicated for pulmonary arterial hypertension, progression of pulmonary hypertension and the development of chronic pulmonary heart disease in COPD patients are closely related to the development of endothelial dysfunction and affective symptoms. We noted improvement in the parameters of diastolic function of the right ventricle, mean pulmonary arterial pressure, the level of stable metabolites of nitric oxide, endothelium-dependent vasoregulation and psychoemotional status in patients with COPD complicated by HPH, after complex treatment with ozone therapy and amlopidine against standard therapy.

Keywords: chronic obstructive pulmonary disease, the function of the diaphragmatic muscles perfusion state, chronic obstructive pulmonary disease complicated pulmonary arterial hypertension

Based on the strategy of the World Health Organization "Practical approach to lung Health - PAL" [1], its current data on the diagnosis of respiratory diseases provide various points of view on the pathogenetic mechanism of vascular changes in respiratory diseases. The diagnosis of pulmonary artery hypertension, based on examinations, is that the systolic blood pressure in the pulmonary artery is >25 mmHg, and the average pressure in the pulmonary artery is >18 mmHg, where it is established [25,32].

The effect of the oxygen content in the alveolar air on the hemodynamics of the pulmonary artery was proven 60 years ago. Belhaj A., Devachter L., Kerbaul F. et al. [18] the experiment involved those who, in their studies, demonstrated the induction of central and peripheral vasoconstriction in acute hypoxic fracture on a model of pulmonary hypertension in cats.

Wonk-Nordegraaf A., Marcus J.T., Holverda S. et al. [30] It was noted that hypoxia of the maxillary part causes dilation of the systemic arteries. Alveolar hypoxia is a powerful vasoconstrictor that reduces perfusion to maintain an even oxygen level in arterial blood vessels. The phenomenon of vasoconstriction occurs in the unventilated parts of the lungs.

Karoli N.A., Rebrov A.P. [6], who divided 69 patients with BMS into 2 groups: 1st group of II degree and 2nd group of III - IV degree. It was found that the increase in pressure in the pulmonary artery in the systole due to hypoxia with an increase in the severity of the disease and bronchial obstruction increased by 20.6% and 41.9% in groups 1 and 2, respectively. There was a decrease in the cross-section of the pulmonary vessels, which indicates a spasm of the thin pulmonary arteries. It was reported that as a result of hypoxemia, increased endothelial dysfunction, hyperactivation of remodeling agents and diastolic dysfunction of the right ventricle of the heart increased by 47.8% and 72.1% in groups 1 and 2, respectively. In the course of studies, the detection of stable nitric oxide metabolites - SMNo - was evaluated as a predictor of pulmonary arterial hypertension. In patients of groups 1 and 2, a decrease in the level of stable metabolites of nitric oxide was recorded to 6.2 and 4.7 mmol/l (norm 9.5 ± 0.6 mmol/l). In the conclusions of those who confirmed that in patients with bcc, the pressure in the pulmonary artery in the systole increased due to hypoxia, cardiac arrhythmias and SMNo decreased, corresponded to the severity of the disease [9,17].

Currently, many researchers deny the development of vasoconstriction by reflex–nervous mechanism in hypoxia, explaining hypoxia by direct action on the smooth muscles of small arteries.

A number of clinical studies [11.10] have shown that antihypertensive drugs, steroid hormones, biogenic amines, prostaglandins stabilize pulmonary arterial hypertension in chronic obstructive pulmonary disease, bronchial asthma, coronary heart disease, hypertension and other diseases. In the conclusions, hypoxia is interpreted as the direct cause of vasoconstriction due to the fact that the pulmonary arteries reduce the supply of energy to smooth muscle tissue, the potassium-sodium pump.

In chronic obstructive pulmonary diseases and bronchial asthma, primary pulmonary hypertension develops due to hypoxia, and an increase in SAO2 from 87% to 95-100% as a result of oxygen therapy has been confirmed. The average pressure in the pulmonary artery decreases from 28 to 22 mm.cm . ($p<0.05$) [24.23] when eating ham in very low concentrations of Hatto from an air-oxygen mixture for a short period of time.

Researchers [11] believe that hypoxia and endothelial dysfunction limit the processes of ventilation and perfusion in the lungs as vasoconstriction develops. Due to increased chest pressure in the right ventricle of the heart, a decrease in the ratio of the maximum frequency of early and late venous blood transfusions (E/a from 1.42+0.09 to 0.94+ 0.07) (R<0.01) was observed.

Brockhuizen R., Wouters E.F., Kreutzberg E.K., Shols A.M. [19] in studies that differ from those presented, pulmonary arterial hypertension in chronic obstructive pulmonary disease and bronchial asthma is a compensatory process that aims not to disrupt the state of ventilation-perfusion, metabolic processes in the early stages of development.. They claim that the blood circulation rate per minute increases from 4.80± 0.23 to 6.10± 0.26 liters, that is, changes in hemodynamics are only an adaptive, compensatory reaction.

Added to the above data, but changes in maxillary respiration disrupt the activity of central and general hemodynamics Viktorova I.A., Trukhan D.I. Recognized [26] that the maximum index of peripheral hemodynamics corresponding to a proportional shift towards a decrease in pulmonary ventilation OFV1 by 38.2±0.5%, FVC by 56.8±1.1%, compared with the control The accuracy of the difference in changes was analyzed by a group with a 1.05-fold decrease in Vmax in the brachial artery.

Smith B.M., Kavut S.M., Blumke D.A. et al. [29] pulmonary arterial hypertension significantly increased during examinations (SAD more than 32 mmHg), the left ventricle of the heart (LVH) correlates with diastolic dysfunction (75.7%), the supply of arterial blood with oxygen is observed at the level of 83-85%, and the oxygen pressure is below 60 mmHg.

Based on the interpretations of many authors [20,27], in chronic obstructive pulmonary disease and bronchial asthma, the work of the heart depends on the diastolic function of the left and right ventricles (RVH), changes in heart rhythm, levels of respiratory failure - i.e., when the activity of external respiration is OFV1<60% and OFV1<30%, the final size of the hemodynamics of the pulmonary The arterial pressure in LVH and pancreatic diastole is 3.65 ± 0.3 and 3.87± 0.3 cm, respectively. This correlates with variations. With a decrease in hypoxemia by 85.3 and 76.4%, the systolic index indicates an increase in vasoconstriction from 2.72±0.12 to 3.27±0.16 and 3.38±0.12 l/m2.

Hypercapnia RSO2 55 mm, which supports narrowing of the pulmonary vessels.s.who. in acute and chronic pulmonary arterial hypertension during decompensation of the pulmonary heart, the acid-base state of the blood shifts towards acidosis, increasing the narrowing of the pulmonary vessels. Not only hypercapnia, but also cardiac contraction: the volume of ejection ranges from 79.9 ± 3.5 to 70.5± 2.1 ml. It decreases to (r<0.05) when pathogenetic changes are predicted, hypercapnia, vasoconstriction and hyperventilation are caused as a response [23].

The study [26] showed that the wall thickness of the right ventricle of the heart ranges from 0.48±0.01 to 0.65±0.01 cm. It was noted that an increase in the amount of oxygen in the blood correlates not with partial pressure, but with the level of hypercapnia (R=0.52). The conclusions state: pulmonary artery remodeling and arterial hypertension are caused by morphological changes, such as stabilization, increased pressure in the pulmonary artery in most cases, hypertrophy of the intima of the middle ventricle, fibrosis and a decrease in the diameter of blood vessels.

Arterial hypertension (esophagus >30 mmHg) was observed in patients with pulmonary hypoxia living in high-altitude areas (2400-3500 meters above sea level). Hypertrophic and pseudonormal types were identified in patients with type 2 heart failure [12]. In these patients, the "reserve" in the diastole was assessed during acute hypoxic fracture, the effect of latent dysfunction in the systole and treatment of OAG with vasodilators were predicted. According to research, blood clotting due to erythrocytosis is the main cause of pulmonary artery hypertension and right ventricular hypertrophy in healthy people living in mountainous areas.

Hypoxia-induced polycythemia increases blood clotting and pulmonary artery hypertension due to the effects of hypertrophied and expanded axial compression of the heart, the full range of left ventricular velocities ranges from 0.73±0.05 to 0.52±0.03 m/s.It decreases to (r<0.05) [31].

A number of authors [15,22,30] have shown that in patients with BCC, cardiac arrows decrease as a result of endothelial dysfunction and increased mortality, while in the right department they do not notice changes in pressure. Other researchers have made contradictory assumptions that blood clotting and endothelial dysfunction are not of great importance in the genesis of pulmonary arterial hypertension, since the pulmonary artery is removed from this condition by compensatory expansion. It was concluded that changes in the properties of cones are limited only by an increase in resistance in the thin circulatory system.

The direction that scientific sources pay attention to in later times, the causes that aggravate pulmonary arterial hypertension are: endothelial dysfunction, apoptosis processes, pathological changes in the membrane lining of the pulmonary artery wall.

In experiments, it was found that the endothelial cells of the pulmonary artery have 3 strong vasodilating properties: prostacycline, endothelial relaxing factor (nitric oxide is not included in its composition) and endothelial hyperpolarizing factor. The fact that the endothelium is a relaxing factor, and prostacyclin not only opens blood vessels, but also reduces platelet aggregation and adhesion, is indicated in many scientific sources.Agusti A.G.N., Neff T.A. [15] in their studies, the pulmonary artery was removed with jarrochia in 17 of 29 patients "in vitro", as indicated in 12 patients without OAG. The pulmonary artery was monitored using various vasoconstrictors et-1, NE, Phe, 5-HT, Ang II. Studies have shown increased vasoconstriction when exposed to O'A et-1 during O'AG and decreased compression of O'a during exposure to NE, Phe, 5-HT, Ang II. It was found that in patients with ulcers, the overall elasticity of the vessels remains unchanged, while in small vessels there is severe endothelial dysfunction and vasoconstriction. The conclusion states that neuroendocrine activity is central to the pathogenesis of OAG development.

During dopplerography [14], endothelium-dependent vasodilation in patients with bronchial asthma of stage III – IV, respectively: Vmax= 3.0±0.6 m/s and 2.2±0.3 m/s.gacha ($r<0.05$) changed. A correlation ($R=0.55$; $R=0.67$; $R<0.05$) with a decrease in Vmax, an increase in dd in the right ventricle of the esophagus and heart was found with compression synapse conducted in the brachial artery. That is, it is claimed that endothelium-dependent relaxation and increased overload of the right ventricle of the heart into the diastole are accompanied by an increase in the severity of AD.

Vasoconstriction and pulmonary artery hypertension are one of the causes of the vascular intima structure, there is a decrease in endothelial activity due to pathological changes. Researchers have shown that pulmonary arterial hypertension is a proportional disorder between endothelins and endothelial relaxation factor in patients with ad, depending on the level of hypoxia [32].

In a clinical study by Oksana Nalivaiko [8], chronic obstructive pulmonary disease in 75% of 87 patients with pulmonary arterial hypertension was diagnosed with grade II-III severity, while 25% of patients were diagnosed with acute grade IV CK. Severe hypercapnia raso2, which develops at severe levels, was found in 71.43% of patients, hypercapnia raso2 - in 28.57% of patients. The dimensions of the diastole in 58 examined patients: the axis of the heart is 30.41 ± 0.63 mm, the right span is 46.03 ± 0.67 mm.to. SAD 46.16 ± 1.54 mmHg. In 26.92% of patients with heart failure, the left ventricular diastole increased from 59.3 ± 1.7 mm to 50.3±1.0 mm.

Tests have shown an increase in blood loss per minute, increased blood circulation in the pulmonary artery in patients with chronic obstructive pulmonary disease and so-called pulmonary arterial hypertension. However, in healthy people, daily blood consumption per minute increases with physical activity and does not affect pulmonary arterial hypertension. Therefore, in the early stages of pulmonary artery hypertension, it is important to assess the minute blood flow. Later, in medical practice, hemodynamic changes are observed in the early stages of chronic obstructive pulmonary disease using invasive and non-invasive methods, namely pulmonary arterial hypertension in the circulatory system and increased systolic resistance of the pulmonary artery [1,32].

It is known that in chronic obstructive pulmonary disease and severe bronchial asthma, the right ventricle of the heart (colon) is in the most unfavorable conditions, since it is subject to constant hemodynamic stress. The left ventricle of the heart is thin compared to the wall thickness, which makes it difficult to adapt to increased pressure. In healthy people, the right ventricle of the heart acts as a digestive pump, not as a pressure pump.

During stabilization of pulmonary artery hypertension, in response to hypoxemia and the severity of pressure, a myocardial reaction develops - hyperfunction and hypertrophy, accompanied by an increase in blood volume and heart rate per minute. However, the development of hyperfunctions and hypertrophy of the right ventricle of the heart depends on the degree of hypoxemia, the mechanism of attachment of various responses to it. At this point, Strutinsky A.V., Sivtseva A.I., Bakaev R.G. [13] argue that in chronic hypoxia, dystrophy develops, not myocardial hypertrophy. The authors testify to the detection of hypertrophy of the right ventricle of the heart by Doppler echocardiography in patients with prolonged pulmonary artery hypertension.

In studies [5], patients with OOC and hypertension were diagnosed according to the type of hypertrophic remodeling of the left ventricle of the heart - normal (19.6% of patients), concentric type of remodeling (34.2% of patients). In the clinic, endothelial dysfunction increased during hypoxemia ($SaO2 <87\%$), hypertrophy of the right and left ventricles of the heart increased, and low levels of hypertrophy ($p >0.05$) were detected in the ventricles of patients who did not experience hypoxemia ($SaO2>88\%$).

Bates D.O. [17] in patients with remodeling of the right ventricle of the heart, SMNO decreased by 5.3% during deconpensation of blood circulation, thus registering OAG and right ventricular heart failure and increased blood clotting.

Researchers [24] found no correlation between emphysema levels and YUOK weight in patients with sook. They also note that the ventricles of the heart do not correlate with systole contraction and blood flow rate per minute. That is, they support the view that the number of blood cells per minute cannot be an insignificant factor in the contractile function of the heart. In case of serious heart changes, the ham heart rate may be normal in a patient's quiet time. On the contrary, many researchers confirm that the number of blood cells and heart rate per minute decrease as changes in lung ventilation and hemodynamics increase. In pulmonary arterial hypertension, its narrowing also increases with minor changes in pressure, reducing the incidence of stroke. The left ventricle of the heart, on the other hand, differs from the right ventricle of the heart by maintaining a pulse in the same rhythm, despite a strong increase in pressure in the large circulatory circle.

In patients with increased bronchial obstruction and pulmonary arterial hypertension, diastole determines early changes in right ventricular heart failure when the final pressure increases several times. According to some authors, there is an inverse correlation between the pressure in the pulmonary artery and the ejection fraction of the right ventricle of the heart [19,32].

In clinical studies, criteria for increased severity, recurrence of peptic ulcer disease and prognosis of chronic obstructive pulmonary disease were evaluated in patients. In this case, a decrease in the ejection fraction of the right ventricle of the heart from physical activity is detected, confirming the fact that in healthy people this indicator can be increased and a hidden deficiency of eggs can be detected by the response of the ejection fraction of the right ventricle of the heart [4,28].

Other experts [7] explain that in chronic obstructive pulmonary diseases, there is a relatively low proportion of acute eruption during exhalation with a change in venous blood flow to the right ventricle of the heart during the respiratory cycle.

In patients with chronic obstructive pulmonary disease and bronchial asthma, a decrease in the ejection fraction of the right ventricle of the heart was found without clinical symptoms of circulatory decompensation, and it was observed that such cardiac output was normal per minute.

Many authors have noted that in patients with severe chronic obstructive pulmonary disease and bronchial asthma, heart function does not correlate with right ventricular hypertrophy and pulmonary arterial hypertension, and with the level of quality of life, pulmonary arterial hypertension does not become too high, and systolic pressure in the pulmonary artery does not increase by 60 mmHg.

According to tests [16], patients with respiratory insufficiency with bronchial asthma have a low incidence of pulmonary artery hypertension, as well as very slow progression. The authors note that during the year the pressure in the pulmonary artery is 3-5 mm, which increases to. However, in primary pulmonary artery hypertension, such as pulmonary embolism, pulmonary artery pressure increases significantly during occlusion.

In conclusion, it should be noted that among the pathogenetic mechanisms of a qualitative response to systemic adaptation to chronic colds, in respiratory diseases: endothelial dysfunction and hemodynamic changes, cardiovascular pathology stimulates the development of remodeling of the right ventricle of the heart.

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