

## HEMODYNAMIC PROFILE OF PATIENTS WITH CHRONIC OBSTRUCTIVE PULMONARY DISEASE AND OBSTRUCTIVE SLEEP APNEA SYNDROME

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### ABSTRACT

*Chronic obstructive pulmonary disease (COPD) is one of the most common diseases and one of the leading causes of death worldwide. The progressive course of the disease is accompanied by worsening of respiratory symptoms, a decrease in pulmonary functional parameters, quality of life, a decrease in working capacity and leads to terminal respiratory failure.*

**KEYWORDS:** *Obstructive Sleep Apnea Syndrome, Respiratory Failure, Chronic Obstructive Pulmonary Disease*

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### INTRODUCTION

Chronic obstructive pulmonary disease (COPD) is one of the most common diseases and one of the leading causes of death worldwide. The progressive course of the disease is accompanied by worsening of respiratory symptoms, a decrease in pulmonary functional parameters, quality of life, a decrease in working capacity and leads to terminal respiratory failure [1,2].

Recently, the concept of hypopnea has also become widely used in clinical practice – a decrease in airflow or thoracoabdominal movements by 30% or more compared to the basal level for 10 c or more, accompanied by a decrease in oxygen saturation of blood hemoglobin (desaturation) by 4% or more [3,4].

Obstructive sleep apnea syndrome (OSA) is a life-threatening respiratory disorder, defined as a period of asphyxia during sleep, leading to the development of excessive daytime sleepiness, hemodynamic disorders and instability of cardiac activity. OSA and COPD are two fairly common diseases in clinical practice, and cardiovascular comorbidity is very often present in each of them. The development of COPD predisposes to the occurrence of nocturnal hypoventilation, especially in the stage of rem sleep, and even contributes to increased resistance of the upper respiratory tract during sleep. Patients with a combination of OSA and COPD have

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worse indicators of nocturnal hypoxemia and hypercapnia, accelerated development of pulmonary hypertension and chronic respiratory failure [5].

In patients with COPD, OSA significantly worsens the course of the main suffering, increases hypoxemia, promotes the development of secondary erythrocytosis, pulmonary hypertension and the formation of a chronic pulmonary heart with right ventricular insufficiency. The so-called overlap syndrome - a combination of COPD and OSA, in fact, is a state of mutual aggravation. The prevalence of this syndrome among people with COPD is estimated at 2%, and among patients with OSA – at 10% [6,7].

Pathophysiological factors that develop in patients with COPD during the course of the disease can negatively affect the state of the cardiovascular system. Currently, it has been proven that the development of sleep apnea syndrome in humans increases the risk of cardiovascular catastrophes [8].

**Objective of the study:** To assess the functional state of the cardiovascular system in patients with COPD, depending on the degree of risk of OSA

**Materials and methods:** On the basis of the Bukhara regional multidisciplinary medical centre (BOMC) examined 84 patients aged 45-70 years, 1-the control group consisted of 24 healthy individuals, held a planned medical examination and does not make any complaints at the time of the survey, 2-group consisted of 32 patients with COPD without OSA and 3 group consisted of 28 patients with COPD with OSA. All patients underwent general clinical, functional and laboratory methods of examination (general blood test, biochemical blood test), analysis of blood gas composition, pulse oximetry, measurement of morning blood pressure, EchoCG, determination of respiratory function, 6-minute walking test. The obtained results were processed statistically using Excel 7.0 and Statistica 6.0 programs using nonparametric and parametric criteria. The average values and standard deviations were calculated. The differences were considered significant at  $p < 0.05$ .

## RESULTS AND DISCUSSION

In our study, all patients with COPD were at the II-38 (63.3%) and III-22 (36.7%) clinical stages of the disease. Measurement of blood gas composition and respiratory function in patients tended to significantly decrease all parameters of groups 2 and 3 compared to the control. The determination of the initial dyspnea index before and after the 6-minute walk test was based on summing up the answers to questions assessing: • functional disorders associated with the presence of dyspnea; • the complexity of the activity causing dyspnea; • the degree of effort causing dyspnea. The severity of each symptom is assessed on a five-point scale (from 4 to 0); the maximum number of points is 12 (characterizes the most pronounced shortness of breath).

Hypoxemia is known to play an important role in the pathogenesis of COPD, in particular in the development of pulmonary hypertension, hypertrophy of the right heart, systemic inflammation and myocardial damage.

In our study, the greatest severity of dyspnea was observed in the group of COPD patients with OSA  $4.30 \pm 0.56$  compared to the group of COPD without OSA  $3.94 \pm 0.57$ . Pulse oximetry of patients performed to evaluate the 6-minute walk test indicated a decrease in blood oxygen

saturation by 2% after the test in all groups of subjects, however, the lowest values were in Group 3  $91.7 \pm 3.81$  (Table 1).

**TABLE 1 THE MAIN FUNCTIONAL INDICATORS OF THE RESPIRATORY SYSTEM**

Functional indicators		1 group, (n=24)		2 group, (n=32)		3 group, (n=28)		P
FEB	VCL,%	M	$\sigma$	M	$\sigma$	M	$\sigma$	
Blood gases	TFE1,%	82,10	14,36	78,93	13,86	76,40	12,87	P>0,05
6-minute walk test	TFE1,%	47,50	14,36	46,80	16,36	45,90	9,56	P>0,05
	pO2, mmHg.	68,43	7,88	66,52	6,23	64,43	4,52	P>0,05
	pCO2, mmHg.	38,00	2,94	41,60	3,17	43,91	3,33	P>0,05
	Distance traveled, m	376,40	61,09	353,05	84,35	332,70	95,67	P>0,05
	Shortness of breath on the Borg scale before the test	1,49	0,51	1,40	0,49	1,22	0,42	P>0,05
	Shortness of breath on the Borg scale after the test	4,09	0,82	3,94	0,57	4,30	0,56	P>0,05
	SpO2 before the test,%	96,20	1,99	94,70	0,98	93,10	1,06	P>0,05
	SpO2 after the test,%	94,40	2,56	93,10	2,35	91,70	3,81	P>0,05

As a result of basal blood pressure measurement, morning hypertension was detected in 18 (64.2%) patients of group 3 and in 15 (46.8%) patients of group 2, which is 4 times more common than in the control group (Table 2).

**TABLE 2 FREQUENCY OF MORNING HYPERTENSION**

Study groups	Morning arterial hypertension			Total
	Abs	M(%)	m	
Group1	4	16,7	7,85	24
Group 2	15	46,8	6,34	32
Group 3	18	64,2	10,34	28
P	Pearson's Chi-square = 3,603; p = 0,058			

Of considerable interest for assessing the state of the cardiovascular system was the study of echocardiographic parameters of the chambers of the heart, with a quantitative assessment of the following parameters: pulmonary artery pressure (PAP), interventricular septum thickness (IST

in systole (c) idiastole (d)), finite-diastolic size (FDS), finite-systolic size (FSS), finite-diastolic volume (CDR), finite-systolic volume (FSV), ejection fraction (EF), myocardial mass index (MMI).

**TABLE 3 THE MAIN ECHOCARDIOGRAPHIC PARAMETERS OF THE HEART CHAMBERS**

Indicators	1 group, (n=24)		2 group, (n=32)		3 group, (n=28)		P
	M	$\sigma$	M	$\sigma$	M	$\sigma$	
FDS,sm	5,10	0,56	5,11	0,52	5,21	0,67	P>0,05
FSS,sm	3,20	0,34	3,20	0,47	3,40	0,63	P>0,05
IST,sm	1,18	0,13	1,24	0,17	1,24	0,23	P>0,05
TS,sm	1,13	0,10	1,09	0,12	1,15	0,19	P>0,05
REL,u	0,46	0,11	0,45	0,09	0,46	0,07	P>0,05
MMI, g/m <sup>2</sup>	109,00	16,03	111,30	17,52	117,90	18,81	P>0,05
LVF according to Simpson, %	60,20	5,76	57,40	5,33	55,70	12,19	P>0,05
SLP, cm <sup>2</sup>	15,50	3,36	18,60	5,88	19,50	4,96	P<0,01
PAP, mmHg.	19,89	7,15	20,11	5,68	21,70	6,03	P>0,05

In COPD, the response of the cardiovascular system to hyperactivation of the respiratory system and compensation for respiratory failure can be used as an additional factor in assessing the functional state of the patient. However, according to the results of echocardiography, signs of left ventricular hypertrophy and an increase in the mean pressure in the pulmonary artery were found only in COPD patients with OSA, and expansion of the left atrium cavity was observed in both groups of COPD patients, compared to the control group. A difference in myocardial mass indices was revealed, which indicates changes mainly in the left chambers and may cause a worsening of the course of coronary artery disease in patients with COPD (Table 3).

## CONCLUSION

COPD patients with OSA had significantly higher scores for assessing the degree of dyspnea on the Borg scale after the test and low blood saturation values were observed after the 6-minute walk test of  $91.7 \pm 3.81$  ( $P < 0.01$ ).

The development of morning hypertension in COPD patients with OSA is associated with hypoxia, hypercapnia, changes in the state of blood gas composition, features of central and peripheral hemodynamics, endothelial function. A higher level of predictors of the development of chronic pulmonary heart disease (DLA and TMJ) was established in patients with COPD with OSA compared with patients without apnea, which indicates the need for more active monitoring in patients with COPD with OSA.

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