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Distinctive Features of Systolic Function of the Left Ventricle in Patients with Chronic Obstructive Lung Disease in Extremely Cold Conditions

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Abstract

The purpose of this study was to identify the characteristics of systolic dysfunction and the formation of chronic pulmonary heart disease in patients with COPD living in the central zone of the Republic of Sakha (Yakutia). This article describes the echocardiographic indices of systolic dysfunction of the heart in 229 patients with chronic obstructive pulmonary disease of the Republic of Sakha (Yakutia). According to our data, severe decompensation of chronic pulmonary heart disease with symptoms of heart failure of right ventricular type in most cases combined with severe left ventricle systolic dysfunction, not related to the primary pathology of the left heart and leading to a further decline in the quality of life of patients and increase in the risk of fatal diseases.

Keywords

Echocardiography; Chronic obstructive pulmonary disease; Chronic pulmonary heart; Remodeling

Introduction

According to modern views, the term “heart remodeling” includes different, multiple malignant changes, which occur on two levels: (1) on the level of individual cells and myocardial interstitial space and (2) on the level of the whole ventricular chamber [1-4].

The main components of heart remodeling, which are independent of the characteristics of the cardiac pathological process, include [5-8] (1) changes in some of the cardiomyocytes (the disorder of the heart impulse-contraction process, β-adrenergic desensitization, hypertrophy of cardiomyocytes, myocyteolysis, impairment of the cytoskeleton proteins, etc.); (2) changes in the myocardium of the ventricles (hypertrophy, necrosis and apoptosis of cardiomyocytes, matrix deterioration, displacing fibroid heart); (3) changes in the ventricle geometry (spherisation, dilatation, thinning of the ventricle wall, AB-regurgitation); and (4) functional remodeling (local asynergy caused by the presence of scar, ischemia, hibernating myocardium, compensatory hyperfunctioning of the “intact” myocardium, and diastolic dysfunction of the myocardium).

The objective of the given study was to identify the specifics of systolic dysfunction and the formation of pulmonary heart in chronic obstructive pulmonary disease (COPD) patients who reside in the central zone of the Republic of Sakha (Yakutia), where the average winter temperature is ~50°C (Figure 1).

Materials and Methods

This work is based on the results of the clinical-laboratory and instrumental examinations of 229 COPD patients with complications.

COPD was diagnosed according to generally accepted criteria [9-11] on the basis of a typical disease pattern (long-standing moist cough with sputum discharge, intermittent fever, crescendo dyspnea accompanied by poor exercise capacity, which is preserved in the absence of disease exacerbation, typical auscultatory pattern in the lungs), medical history data (typical exacerbations of the disease), and X-ray findings (the presence of obstructive pulmonary emphysema, pulmonary fibrosis, change of the lung pattern, etc.), as well as on the basis of the examination results of the external respiration function (obstructive or mixed type of respiratory disturbance with a progressive decrease in forced expiratory volume). All of the examined patients demonstrated manifestations of the broncho-obstructive syndrome in combination with lesions of the respiratory areas of the lungs. In some cases (124 patients), the symptoms of compensated or decompensated pulmonary arterial hypertension and COPD were revealed (using clinical and echocardiographic data).

Thus, the main criteria for inclusion of patients in this study were

1. the presence of reliable clinical and instrumental symptoms of COPD (chronic cough, sputum discharge, crescendo dyspnea);
2. forced expiratory volume/forced inspiratory vital capacity of, at least, 70% of normal values; and
3. the patient’s informed consent.

The disease severity was assessed according to the COPD classification revised in 2003 (see below).

The diagnostic criteria of a chronic pulmonary heart (CPH) include

1. generally accepted clinical and instrumental indicators of right ventricular hypertrophy and/or dilatation of the cavities of the right ventricle and right atrium;
2. echocardiographic signs of increased mean pressure in the pulmonary artery >20 mm Hg;

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3. clinical signs of "congestion" in greater circulation (hepato-
megalia, edemas of lower limbs, jugular venous distention, 
hydrothorax, ascite, hepatojugular reflux, etc.); and 
4. echocardiography, to look for signs of systolic dysfunction 
of the right ventricle.

Importantly, in each specific case, the clinical-instrumental 
diagnostics of COPD were based on the detection of two or more of 
the previously described criteria.

Criteria for exclusion of patients from this study were the following:
1. Signs of severe bronchospasm and frequent asthma attack in 
   patients suffering from bronchial asthma.
2. Severe myocardial infarction or unstable angina.

Clinical-instrumental examination of COPD and CPH patients 
was performed during periods of COPD remission and in cases in 
which there were no signs of inflammatory process exacerbation in 
the lungs and bronchi.

All of the patients were divided into three groups. The first group 
consisted of 105 COPD patients having no reliable signs of CPH. In the 
second group, there were 71 COPD patients with signs of compensated 
CPH. In addition, 53 patients with signs of decompensated CPH and 
right ventricular CCF (chronic cardiac failure) were placed in the third 
group.

**Echocardiography and Doppler echocardiography**

For the echocardiographic examination, echocardiographs were 
obtained from Acuson-128 XP (USA) and Sonoage 4800 (Southern 
Corea). These devices were used to perform ultrasound heart 
examination in two-dimensional mode and M-mode as well as Doppler 
examination of blood circulation in the pulse mode and continuous 
wave mode. The study was performed according to the standard 

The examination was performed simultaneously with ECG 
registration to synchronize the phases of the echocardiogram cardiac 
cycle with the ECG data. Measurements of the thickness of the walls 
of the heart and sizes of its cavities in the systolic and diastolic phases 
were performed according to the recommendations of the American 
Committee of Echocardiography experts. Residual volumes of 
the left ventricle (end-diastolic volume and end-systolic volume), stroke 
output, and ejection fraction were calculated using the Simpson 
method. The mean pressure in the pulmonary artery was found 
using the Kitabatake method (1983). A quantitative evaluation of the 
parameters of the systolic blood flow in the outflow tract of the right 
ventricle was performed, and the ratio between the flow acceleration 
time and the overall duration of the right ventricle emptying was 
calculated.

The diastolic function of the right ventricle and the left ventricle 
was assessed on the basis of the results of the Doppler examination, 
which included an examination of the transtricuspid and transmitial 
diastolic blood flows in pulse, color, and continuous wave modes.

**Results and Discussion**

As shown in Table 1, patients in the first and second groups who had 
no signs of CCF did not manifest any serious impairments in the systolic 
function of the left ventricle. The ejection fraction of the left ventricle 
was 65.6 ± 2.1% and 61.9 ± 2.0%, and the degree of shortening of 
the anteroposterior size of the left ventricle was 40.2 ± 1.3% and 
37.2 ± 1.3%. The stroke output (SO) and stroke volume index (SVI) 
were significantly higher compared with the control group (p < 0.05).

Table 1: Comparison of Cardiac Function Parameters in Patients with COPD

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Control Group</th>
<th>First Group</th>
<th>Second Group</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ejection Fraction (%)</td>
<td>65.6 ± 2.1%</td>
<td>61.9 ± 2.0%</td>
<td>60.6 ± 2.3%</td>
</tr>
<tr>
<td>Stroke Output</td>
<td>55.2 ± 2.9%</td>
<td>61.9 ± 3.2%</td>
<td>59.3 ± 3.1%</td>
</tr>
<tr>
<td>Stroke Volume Index</td>
<td>61.9 ± 2.0%</td>
<td>60.6 ± 2.3%</td>
<td>58.5 ± 2.2%</td>
</tr>
</tbody>
</table>

There was also a tendency toward an increase in minute volume (MV) 
and cardiac index (CI), which was most likely due to the compensatory 
intensification of blood circulation. Such intensifications were not 
infrequent in COPD patients and in patients suffering from respiratory 
disturbance (RD).

The systolic and diastolic sizes of the left ventricle (end-systolic 
volume of the left ventricle, index of the end-systolic volume of 
the left ventricle, end-diastolic volume of the left ventricle, index of 
the end-diastolic volume of the left ventricle, end-diastolic dimension 
of the left ventricle) in patients in the first group (with no signs of CPH) 
were nearly similar to those of the control group. The average values of 
the end-diastolic volume of the left ventricle, index of the end-diastolic 
volume of the left ventricle, and end-diastolic volume of the left ventricle 
in the second group of patients (compensated CPH) were higher compared 
with the control group (p < 0.05-0.01), but these values still did not exceed the upper 
boundary of the normal range of values: end-diastolic volume of the 
left ventricle (100-125 ml), index of the end-diastolic volume of the left ventricle 
(55-73 ml/m²), end-diastolic dimension of the left ventricle (38-56 mm).

Thus, no noticeable growth in the systolic size of the left ventricle 
occurred, which confirms the absence of an impairment in the systolic 
function of the left ventricle in patients with compensated CPH. It is 
probable that a moderate increase in the end-diastolic volume of the left 
ventricle resulted from an increase in MV and consequently an increase 
in the amount of the preload and left ventricle filling pressure. This is 
a typical result of the majority of COPD patients and in patients with 
RDs. Under such conditions, an increase in the end-diastolic volume 
of the left ventricle is of a compensatory nature, and according to the 
Frank–Starling mechanism, this results in an increase in the SO and 
MV of blood circulation.

Furthermore, the first group of patients (COPD without CPH) 
demonstrated a significant increase in the sphericity index of the left 
ventricle. This increase confirms the onset of a long-standing process 
of structural-functional changes in this compartment of the heart. It is 
known that an increase in left ventricle sphericity is accompanied by 
a decrease in the effectiveness of ventricle contraction and is one of the 
most important indicators of ventricle remodeling in response to a 
specific hemodynamic overload or to primary failure of the cardiac 
muscle.


Analysis of echocardiographic examination results revealed a moderate increase in the left atrium size (p < 0.05) in COPD patients without CCF (first and the second groups). The left atrium size (30.1 ± 1.6 mm) in the first group (without CPH) remained at the upper boundary of the normal range (19-33 mm), while in the case of the patients with uncompensated CHP (second group), the average sizes of the left atrium cavity were up to 36.2 ± 1.5 mm (p < 0.05).

Moreover, the first group of patients demonstrated a significant (p < 0.05) increase in the mean values of the systolic myocardial stress, 131 ± 3.2 dyne/cm², compared with 121.9 ± 2.6 dyne/cm² in the control group. Furthermore, an even greater increase in systolic myocardial stress was observed in patients in the second and the third groups (151 ± 3.5 dyne/cm² and 188 ± 4.1 dyne/cm², respectively). According to modern views, this integral indicator indirectly reflects the value of intramyocardial tension and, accordingly, the intensity of metabolic processes in the cardiac muscle. An increase in this indicator is currently considered to be one of the most important characteristics of heart remodeling in different cases of cardiac pathology.

Finally, the average values of the myocardial mass of the left ventricle and the index of the myocardial mass of the left ventricle increased in the second group of patients with specific CPH diagnosis. The left ventricular posterior wall and the interventricular septum were also thicker in this group. With regard for the above parameters, the mean values significantly differed from the values in the control group and in the first group of patients with no signs of CPH (p < 0.05). Nevertheless, the mean values of the index of the myocardial mass of the left ventricle in the case of these patients did not exceed the threshold values (which were 104 g/m² for female patients and 116 g/m² for male patients), which are currently used for echocardiographic diagnostics of left ventricle hypertrophy. Thus, despite the significant increasing tendency of the myocardial mass of the left ventricle in patients with compensated CPH, there was no formal basis for the diagnosis of left ventricle hypertrophy.

Thus, a significant increase in SO, SVI, left atrium sizes, sphericity index, and systolic myocardial stress was revealed in the first group of patients (COPD patients with no reliable symptoms of CPH) during the echocardiographic examination. Such an increase confirmed

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### Table 1: Changes in the systolic and diastolic functions of the left ventricle in patients suffering from COPD and chronic pulmonary heart disease

<table>
<thead>
<tr>
<th>Indices</th>
<th>Control n = 30</th>
<th>1st group (without CPH) n = 105</th>
<th>2nd group (compensated CPH) n = 71</th>
<th>3rd group (decompensated CPH) n = 53</th>
<th>P&lt;sub&gt;23&lt;/sub&gt;</th>
<th>P&lt;sub&gt;44&lt;/sub&gt;</th>
</tr>
</thead>
<tbody>
<tr>
<td>Myocardial mass of the left ventricle, g</td>
<td>101.5 ± 6.3</td>
<td>120 ± 6.2</td>
<td>142 ± 5.6*</td>
<td>160 ± 6.7*</td>
<td>&lt;0.05</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>Myocardial mass of the left ventricle g/cm²</td>
<td>62.6 ± 2.4</td>
<td>70.4 ± 3.5</td>
<td>83.2 ± 2.5*</td>
<td>94.4 ± 3.0*</td>
<td>&lt;0.01</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>End-diastolic volume, ml</td>
<td>110.2 ± 3.3</td>
<td>116.6 ± 3.5</td>
<td>126.6 ± 3*</td>
<td>137.9 ± 2.3*</td>
<td>&lt;0.05</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>End-systolic volume, ml</td>
<td>42.1 ± 1.5</td>
<td>40.6 ± 2.0</td>
<td>43.1 ± 1.4</td>
<td>71.4 ± 1.83*</td>
<td>–</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>End-diastolic index, ml/m²</td>
<td>62.8 ± 2.3</td>
<td>67.4 ± 3.0</td>
<td>73.3 ± 2.5*</td>
<td>76.7 ± 2.1*</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>End-systolic index, ml/m²</td>
<td>22.9 ± 0.7</td>
<td>23.2 ± 0.5</td>
<td>24.6 ± 0.6</td>
<td>39.9 ± 1.9*</td>
<td>–</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>End-diastolic dimension, mm</td>
<td>48.1 ± 1.4</td>
<td>50.2 ± 1.3</td>
<td>54.6 ± 1.2*</td>
<td>61.6 ± 1.9*</td>
<td>&lt;0.05</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>End-systolic dimension, mm</td>
<td>29.3 ± 1.5</td>
<td>30.1 ± 1.4</td>
<td>33.2 ± 1.3*</td>
<td>46.2 ± 1.6*</td>
<td>&lt;0.01</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Stroke output, ml</td>
<td>72.7 ± 1.6</td>
<td>77.4 ± 1.5*</td>
<td>78.2 ± 1.3*</td>
<td>63.5 ± 1.4</td>
<td>–</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Stroke volume index, ml/m²</td>
<td>38.2 ± 1.2</td>
<td>42.3 ± 1.3*</td>
<td>42.7 ± 1.3*</td>
<td>36.2 ± 1.0</td>
<td>–</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>Ejection fraction, %</td>
<td>63.4 ± 2.3</td>
<td>65.6 ± 2.1</td>
<td>61.9 ± 2.0</td>
<td>46.6 ± 1.6*</td>
<td>&lt;0.001</td>
<td>–</td>
</tr>
<tr>
<td>Cardiac rate, strokes/min</td>
<td>68.2 ± 4.0</td>
<td>72.3 ± 3.6</td>
<td>74.1 ± 3.1</td>
<td>80.2 ± 3.2*</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>Minute volume, l</td>
<td>4.96 ± 0.3</td>
<td>5.59 ± 0.3</td>
<td>5.78 ± 0.3</td>
<td>5.09 ± 0.3</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>Cardiac index, l/min m²</td>
<td>2.7 ± 0.2</td>
<td>3.0 ± 0.3</td>
<td>3.25 ± 0.2</td>
<td>2.7 ± 0.2</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>ΔS% left ventricle</td>
<td>39.1 ± 1.4</td>
<td>40.2 ± 1.3</td>
<td>37.2 ± 1.3</td>
<td>25.5 ± 1.0*</td>
<td>–</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>The thickness of left ventricular posterior wall, mm</td>
<td>9.3 ± 0.2</td>
<td>9.6 ± 0.2</td>
<td>10.0 ± 0.2*</td>
<td>10.9 ± 0.2*</td>
<td>&lt;0.05</td>
<td>–</td>
</tr>
<tr>
<td>Interventricular septum thickness, mm</td>
<td>8.7 ± 0.2</td>
<td>9.0 ± 0.2</td>
<td>10.3 ± 0.1*</td>
<td>10.2 ± 0.2*</td>
<td>&lt;0.01</td>
<td>–</td>
</tr>
<tr>
<td>Left atrium, mm</td>
<td>25.2 ± 1.7</td>
<td>30.1 ± 1.6*</td>
<td>36.2 ± 1.5*</td>
<td>41.6 ± 1.2*</td>
<td>&lt;0.05</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>Systolic blood pressure, mm Hg</td>
<td>136 ± 2.8</td>
<td>135 ± 2.1</td>
<td>138 ± 2.6</td>
<td>136.9 ± 3.0</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>Sphericity index</td>
<td>0.57 ± 0.02</td>
<td>0.69 ± 0.02*</td>
<td>0.74 ± 0.01*</td>
<td>0.78 ± 0.02*</td>
<td>0.05</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>2h/D</td>
<td>0.37 ± 0.02</td>
<td>0.37 ± 0.01</td>
<td>0.36 ± 0.01</td>
<td>0.34 ± 0.01*</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>Myocardial stress, dyne/cm²</td>
<td>121.9 ± 2.6</td>
<td>131 ± 3.2*</td>
<td>151 ± 3.5*</td>
<td>186 ± 4.1*</td>
<td>&lt;0.001</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Left ventricle emptying, ms</td>
<td>316 ± 8.3</td>
<td>324 ± 5.7</td>
<td>326 ± 6.1</td>
<td>290 ± 6.2</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>V&lt;sub&gt;exp&lt;/sub&gt;, m/s</td>
<td>0.92 ± 0.02</td>
<td>0.89 ± 0.03</td>
<td>0.86 ± 0.02*</td>
<td>0.72 ± 0.02*</td>
<td>&lt;0.001</td>
<td>–</td>
</tr>
<tr>
<td>MV DT, ms</td>
<td>193 ± 6.0</td>
<td>201 ± 5.3</td>
<td>216 ± 5.2</td>
<td>174 ± 4.8*</td>
<td>&lt;0.05</td>
<td>–</td>
</tr>
<tr>
<td>MV IVRT, ms</td>
<td>74.3 ± 1.6</td>
<td>83 ± 1.8*</td>
<td>95 ± 2.2*</td>
<td>76.3 ± 2.8</td>
<td>&lt;0.05</td>
<td>0.01</td>
</tr>
<tr>
<td>MV Peak E, m/s</td>
<td>0.56 ± 0.02</td>
<td>0.58 ± 0.01</td>
<td>0.51 ± 0.02*</td>
<td>0.63 ± 0.02*</td>
<td>&lt;0.05</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>MV Peak A, m/s</td>
<td>0.35 ± 0.01</td>
<td>0.41 ± 0.01*</td>
<td>0.44 ± 0.02*</td>
<td>0.30 ± 0.02*</td>
<td>&lt;0.01</td>
<td>–</td>
</tr>
<tr>
<td>MV E/A</td>
<td>1.61 ± 0.02</td>
<td>1.42 ± 0.02*</td>
<td>1.15 ± 0.02*</td>
<td>2.10 ± 0.02*</td>
<td>&lt;0.001</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

The parameters that significantly deviated from normal values (p < 0.05 and p < 0.01, respectively) are shown in bold or marked with * and #.
the onset of a long-standing process of left ventricle remodeling in COPD patients. Such changes were more pronounced in patients of the second group (compensated CPH) and were accompanied by an increase in the myocardial mass of the left ventricle and by a moderate increase in the end-diastolic volume and end-systolic volume. In most cases, these changes did not progress to the stage typical of left ventricle remodeling in cases of myocardial infarction, chronic forms of ischemic heart disease, arterial hypertension, etc. However, these changes convincingly confirm that pathological changes, which eventually result in the development of CCF, affect not only the right but also the left compartments of the heart in patients with COPD and CPH. Moreover, this occurs in the early stages of CPH development, long before the occurrence of compensatory changes.

The situation was somewhat different in the case of the third group of patients with decompensated CPH. These patients demonstrated a moderate decrease in ejection fraction, which averaged to 46.6 ± 1.6%. The degree of shortening of the left ventricle was up to 25.3 ± 1.0%, which was significantly (p < 0.001) different compared with the respective values of the patients in the first and second groups (61-65% and 40-37%, respectively). There was also a minor decrease in the SO (p < 0.001) and SVI (p < 0.05) and a decreasing tendency in the MV and CI (p > 0.05) compared with patients in the first and second groups.

The mean values of the end-systolic volume of the left ventricle, end-diastolic volume of the left ventricle, end-systolic dimension of the left ventricle, and end-diastolic dimension of the left ventricle in patients of the third group with decompensated CPH were also significantly higher compared with the respective values in patients in the first and the second groups (p < 0.001). Moreover, unlike the values in groups 1 and 2, the values in the third group were outside the range of values typical of the control group and are generally accepted as normal (permissible). These changes and the decrease in the ejection fraction, SO, SVI, and ΔS% indicate the presence of systolic dysfunction of the left ventricle in patients with decompensated CPH. Not surprisingly, indicators of functional depression of the pumping ability of the left ventricle were more pronounced in patients with severe CPH decompensation, which corresponded to the respective III-IV FC (FUNCTIONAL CLASS) according to NYHA. The mean values of these parameters in patients with less pronounced decompensation (CCF II FC) varied very little from the mean values in the second group, which consisted of patients with compensated CPH.

Moreover, a significant increase in myocardial mass of the left ventricle was observed in the third group of patients compared with patients in the second group (p < 0.05). The thickness of the left ventricular posterior wall (p < 0.05) and the thickness of the interventricular septum (p < 0.001) also increased. Moreover, the index of relative thickness of the left ventricle wall (2H/D) significantly decreased, which indicated the development of eccentric hypertrophy of the left ventricle.

Furthermore, there was a clear increase in the mean values of systolic myocardial stress (p < 0.001), which was higher compared with the respective index of patients with compensated CPH (second group) even in cases of moderate CCF II FC (FUNCTIONAL CLASS) according to NYHA.

Conclusions

Thus, severe CPH decompensation with signs of right-ventricular heart failure in most cases was accompanied by comorbid disorders of the systolic function of the left ventricle. These disorders were not correlated to the primary pathology of the left compartments of the heart, and they resulted in additional deterioration of the patients’ quality of life and increased the risk of lethal outcome. Engagement of the left ventricle in the remodeling of the heart is another factor that aggravates the course of disease in patients with COPD and CPH.

References