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Diaphragmatic dysfunction in patients with chronic heart failure

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ABSTRACT

Objective. To detect diaphragmatic dysfunction in patients with chronic heart failure (CHF) via assessment of structural and functional state of external respiration.

Material and methods. The study was conducted at the Almazov National Medical Research Center and included 58 subjects. There were 40 patients with CHF and 18 ones without CHF (control group). Inclusion criterion for the main group was CHF NYHA class II—IV. Exclusion criteria for both groups were pulmonary diseases, morbid obesity, abdominal diseases and anemia. We performed ultrasonic assessment of structural (thickness) and functional (thickening fraction and excursion) state of diaphragm under calm and deep inhalation/exhalation. External respiration was assessed using spirometry functions of a ventilator.

Results. Patients with CHF had a thicker diaphragm on both sides and smaller volume of deep inhalation. Diaphragm thickening index was significantly less under deep inhalation on the right. Patients with CHF had a larger excursion of the right and left domes of the diaphragm under calm breathing, whereas no significant differences were found under deep breathing. We found a moderate positive linear correlation between CHF and diaphragm thickness on both sides under calm inhalation.

Conclusion. Structural and functional diaphragmatic dysfunction makes a contribution to respiratory failure in patients with CHF.

Keywords: *diaphragmatic dysfunction, chronic heart failure, sonography of diaphragm, diaphragm, lung function, diaphragmatic function.*

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Introduction

Diaphragmatic dysfunction is interesting for intensive care specialists, as it predisposes to prolonged ICU-stay [1] and can determine respiratory support option (from oxygen therapy to respiratory function replacement) [2, 3]. Progression of chronic heart failure (CHF) leads to dysfunction of respiratory muscles, and it is considered as a symptom of multiple organ failure [4]. There are ventilation disorders following sarcopenia (loss of muscle mass) and dynapenia (loss of muscle strength without decrease in muscle mass) [5, 6], as well as restrictive respiratory failure (RF) in CHF [6—9]. Reduced strength of respiratory muscles does not depend on etiology of CHF [10]. Many researchers emphasize an impaired respiratory function in CHF [7, 10—13], while no changes in neuro-respiratory drive are found in patients with CHF [10]. However, morphological changes in respiratory muscles, in particular, diaphragm, are described [7, 9, 14—16].

In general, ultrasound data on structural and functional state of respiratory muscles in patients with various diseases are interesting [14, 16, 17]. However, information about diaphragmatic function in patients with CHF is contradictory.

The purpose of this study was to detect diaphragmatic dysfunction in patients with CHF via assessment of structural and functional state of external respiration.

Material and methods

A prospective study was devoted to structural (ultrasound of diaphragm) and functional (spirometry) state of the respiratory apparatus in patients with CHF. The local ethics committee approved this study (protocol dated April 30, 2022, chairperson — Zagorodnikova K.A.).

The study included 58 patients (40 ones with CHF and 18 ones without CHF). The study group enrolled patients with CHF NYHA class II-IV, the control group — patients who underwent radiofrequency pulmonary vein isolation for paroxysmal atrial fibrillation following arterial hypertension. The last ones had good visualization of diaphragm excursion on both sides.

Exclusion criteria for both groups were comorbidities potentially changing function of diaphragm: pulmonary diseases, morbid obesity, abdominal diseases accompanied by increased intra-abdominal pressure, anemia (HGB <120 g/l).

The study group consisted of 8 women and 32 men aged 59.1±12.9 years. Body weight was 84.9±17.0 kg, height 172.9±8.3 cm, BMI 28.3±4.9 kg/m². All ones suffered from CHF for at least a year. There were 15 patients with CHF class II, 12 ones with class III and 13 ones with class IV upon admission.

All patients received combination of drugs (ACE inhibitors, beta-blockers, diuretics, aldosterone antagonists, statins). Patients with heart rhythm disturbances (atrial fibrillation) additionally received oral anticoagulants.

In the study group, visualization of diaphragm excursion on the left under calm breathing was available in 75% (*n*=30) of patients, under deep breathing — in 57.5% (*n*=23) of patients due to objective difficulties [18, 19, 20, 21].

The control group consisted of 12 women and 6 men. Age of patients was 60.3±11.1 years, body weight 81.7±15.5 kg, height 172.1±12.0 cm, BMI 27.6±4.7 kg/m². Both groups were comparable in anthropometric parameters and age.

Ultrasound of diaphragm and assessment of external respiration were performed in supine position with head raised by 30° (main position of patients in intensive care units). Structural (thickness, T) and functional (excursion, E; thickening fraction, TF) state of the diaphragm under calm and deep inhalation/exhalation was assessed using a Philips CX50 scanner (Philips Ultrasound, Inc, USA).

We analyzed pulmonary parameters using non-invasive lung ventilation machine (Dräger, model Evita Infinity V500, Germany). Device settings: constant positive pressure 0 cm H2O without pressure support, inspiratory oxygen fraction 21%.

Tidal volumes under calm and deep breathing, as well as inhalation and exhalation times were analyzed. We assessed neuro-respiratory drive considering decrease in airway pressure within 100 ms of spontaneous breathing attempt after respiratory circuit occlusion. Negative inspiratory force (NIF) was also estimated (minimum airway pressure during inhalation from closed circuit). NIF < −25 — −30 cm H_2O determined respiratory muscle weakness [22].

Statistical analysis was carried out using the STATISTICA-10 software (StatSoft, USA) and external analysis package (Real Statistics Resource Pack) supplementing standard capabilities of Microsoft Excel.

Study power was 0.83. Distribution normality was tested by using of Shapiro-Wilk and Kolmogorov-Smirnov tests. We compared independent groups using Mann-Whitney test. Relationship between certain variables were assessed by using of rank-biserial coefficient and Spearman's rank correlation coefficient with assessment of strength by Chaddock scale (weak — 0.1—0.3; moderate — 0.3—0.5; significant — 0.5—0.7; high — 0.7—0.9; very high — 0.9—1.0).

Data are presented as medians and quartiles (25; 75). Differences were significant at p-value < 0.05.

Results

There were no between-group differences in blood gases and hemoglobin level, as well as laboratory signs of oxygen debt (normal serum lactate).

The results are presented in **Tables 1—4**.

CHF patients had systolic dysfunction of the right and left ventricles and higher pulmonary hypertension. Despite normal values, there were significant between-group differences in peripheral oxygen saturation.

Patients with CHF had smaller inhalation volume under deep breathing, while respiratory drive (P0.1) and NIF were comparable **(Table 2)**.

CHF patients had a thicker diaphragm on both sides in each phase of respiratory cycle both during calm and deep breathing

Table 1. **Characteristics in both groups**

Value	No CHF	CHF	*p*-value
SpO_2, %	99,0 (99,0;100,0)	98,0 (97,0;99,0)	0,003*
APACHE II score	10,5 (8,0;11,0)	13 (11,5;15,0)	0,001*
LV EF, %	60,0 (56,0;62)	30,0 (21,5;37,5)	0,001*
TAPSE, mm	19,5 (18,0;24,0)	17,0 (14,0;19,0)	0,001*
sPAP, mm Hg	31,8 (26,0;38,0)	39,5 (34,5;50,0)	0,001*

Note. APACHE II — Acute Physiology and Chronic Health Evaluation II, LV EF — left ventricular ejection fraction, TAPSE — tricuspid annular plane systolic excursion, sPAP — systolic pulmonary artery pressure, SpO2 — peripheral oxygen saturation.

Table 2. **Spirometry parameters in both groups**

Value	No CHF	CHF	*p*-value
Breathing rate	14,0 (11,0;16,0)	15,0 (13,0;17,0)	0,19
IV_{cbr}, ml	570,5 (443,0; 696,0)	555,0 (450,0; 764,0)	0,87
IT_{cbr}, s	1,6 (1,3;2,1)	1,2 (1,1;1,6)	0,002*
ET_{cbr}, s	2,8 (1,8;3,4)	2,8 (2,4;3,3)	0,79
IV_{db}, ml	4022,0 (3800,0;4500,0)	3000,0 (2300,0;4000,0)	0,002*
P0.1 cm H_2O	−1,4 (−1,7;−1,0)	−1,3 (−1,8;−0,9)	0,84
NIF, cm H_2O	−47,2 (−58,0−43,0)	−44,0 (−58,0;−33,4)	0,29

Note. IV — inhalation volume, cbr — calm breathing, db — deep breathing, IT — inspiratory time, ET — expiratory time, NIF — negative inspiratory force, P0.1 — occlusion pressure at 100 ms.

Table 3. **Diaphragm thickness in both groups**

Value	No CHF	CHF	*p*-value
$T_{c\text{-}inh}$ on the right, mm	2,0 (1,7;2,3)	2,9 (2,2;3,7)	0,00021*
$T_{c\text{-}exh}$ on the right, mm	1,6 (1,3;1,8)	2,1 (1,8;2,9)	0,0001*
$T_{d\text{-}inh}$ on the right, mm	4,2 (3,6;5,4)	5,2 (4,2;6,2)	0,07
$T_{d\text{-}exh}$ on the right, mm	1,3 (1,0;1,5)	1,7 (1,3;1,9)	0,007*
$T_{c\text{-}inh}$ on the left, mm	2,0 (1,8;2,1)	2,8 (2,4;3,5)	0,0002*
$T_{c\text{-}exh}$ on the left, mm	1,5 (1,4;1,7)	2,0 (1,7;2,5)	0,0002*
$T_{d\text{-}inh}$ on the left, mm	4,0 (3,2;4,4)	4,9 (4,1;6,9)	0,02*
$T_{d\text{-}exh}$ on the left, mm	1,2 (1,0;1,5)	1,6 (1,3;2,0)	0,0004*
$TF_{c\text{-}inh}$ on the right, %	20,9 (17,3;33,8)	29,5 (18,3;44,1)	0,17
$TF_{d\text{-}inh}$ on the right, %	151,8 (114,6;282,5)	118,5 (81,2;180,9)	0,04*
$TF_{c\text{-}inh}$ on the left, %	30,6 (22,5;43,6)	38,1 (29,1;60,1)	0,17
$TF_{d\text{-}inh}$ on the left, %	169,8 (100,0;245,8)	150,0 (119,0;192,7)	0,87

Note. T — thickness, TF — thickening fraction, c — calm, d — deep; inh — inhalation, exh — exhalation, * — significant differences ($p<0.05$, Mann-Whitney test).

Table 4. **Diaphragm excursion in both groups**

Value	No CHF	CHF	*p*-value
Number, *n*	18	40	-
E_{cbr} on the right, cm	1,8 (1,3;1,9)	2,3 (1,6;2,8)	0,003*
E_{db} on the right, cm	7,0 (5,4;8,5)	7,2 (5,9;9,1)	0,69
Number, *n*	18	30	-
E_{cbr} on the left, cm	1,58 (1,39;2,14)	1,87 (1,53;2,31)	0,27
Number, *n*	18	23	-
E_{db} on the left, cm	6,5 (5,2;7,4)	5,6 (4,5;7,3)	0,15

Note. E — excursion, cbr — calm breathing, db — deep breathing, * — significant differences ($p<0.05$, Mann-Whitney test).

(Table 3). Thickening fraction was significantly lower, but only under deep breathing on the right.

CHF patients have higher excursion of the right ($p<0.05$) and left ($p>0.05$) domes of diaphragm under calm breathing **(Table 4)**. There were no significant differences in diaphragm excursion on both sides under deep breathing.

Correlation analysis revealed moderate and significant negative relationship between chronic heart failure and tidal volume under deep inspiration (rs –0.41, $p<0.05$), as well as peripheral oxygen saturation (rs –0.55, $p<0.05$).

CHF affects many structural and functional parameters of the diaphragm, but this effect is not significant. A linear direct noticeable correlation is found only between CHF and diaphragm thickness on both sides under calm breathing (on the right rs 0.56, on the left rs 0.55, $p<0.05$). Correlations between diaphragm thickness and CHF are presented in **Fig. 1**—4.

No significant correlations were found between cardiac function and external respiration, as well as between spirometry parameters and diaphragm ultrasound data among patients with CHF.

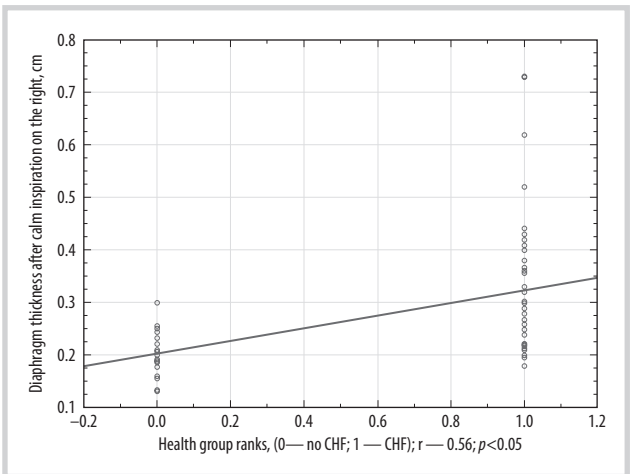


Fig. 1. **Correlation between thickening of the right dome of diaphragm and CHF.**

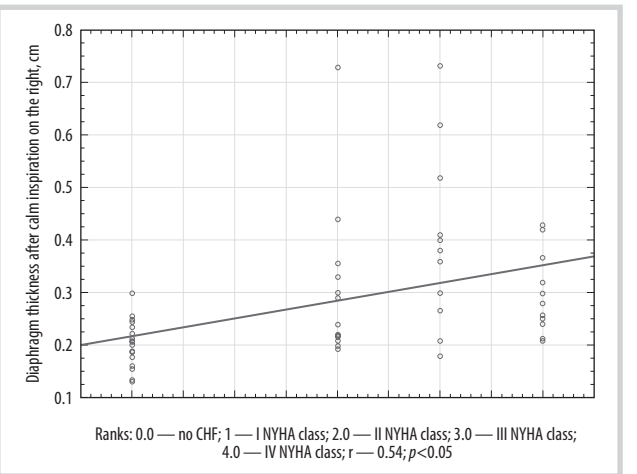


Fig. 2. **Correlation between thickening of the right dome of diaphragm and CHF depending on NYHA class.**

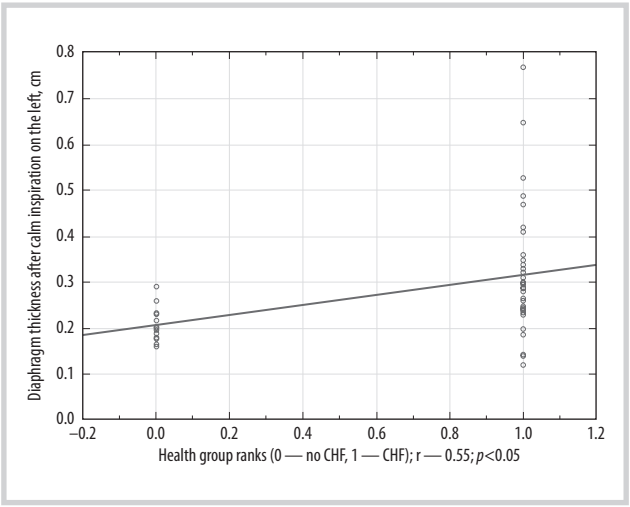


Fig. 3. **Correlation between thickening of the left dome of diaphragm and CHF.**

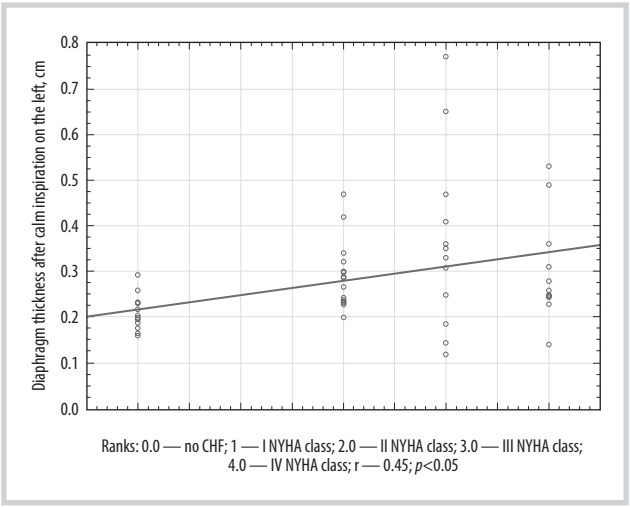


Fig. 4. **Correlation between thickening of the left dome of diaphragm and CHF depending on NYHA class.**

We did not perform regression analysis considering no significant correlations.

Discussion

The purpose of this study was substantiation of hypothesis on the role of diaphragmatic dysfunction in respiratory failure among patients with CHF. This assumption is justified by high oxygen consumption by diaphragm [23] and its vulnerability to reduced oxygen flow [24]. The last one increases with progression of heart failure [7] up to low cardiac output syndrome.

Patients with CHF had significantly worse cardiac parameters (LV EF, TAPSE, sPAP). SpO2 was normal but significantly lower in the same group that may be explained by reduced peripheral blood flow in these patients [25].

The most obvious difference of spirometry parameters in patients with CHF was significantly lower tidal volume under deep inspiration (3000 vs. 4000 ml, less by 25%, p=0.002). There were no between-group differences in tidal volumes under calm breathing. Thus, we can conclude decrease of functional reserves of external respiration in patients with CHF that is not a discovery per se [5, 6, 8]. A more interesting question is devoted to role of diaphragm.

Our data indicate that patients with CHF have a significantly thicker diaphragm on both sides in all phases of respiratory cycle under calm and deep breathing. Other authors also emphasize changes of diaphragm in patients with CHF [5, 9, 26], but these changes multidirectional.

Miyagi M. et al. [5] found thicker diaphragm in patients with lower left ventricular fraction. Spiesshoefer J. et al. [26] found no differences in diaphragm thickness at the end of calm exhalation in healthy people and CHF patients. Thickness of diaphragm under deep inspiration was greater in healthy people.

Considering edema of lung parenchyma with increased elastic recoil (i.e., decrease of compliance) of lungs and chest rigidity in patients with chronic heart failure [8, 15, 26], one can expect higher load on the diaphragm during inspiration with its hypertrophy. Similar pathophysiological mechanisms of increased load on the diaphragm under hypoxia and reduced blood supply in patients with non-cardiogenic respiratory failure lead to various structural changes of the diaphragm (thickening, atrophy, dystrophy and edema) [27].

According to our functional data, diaphragm excursion under calm breathing was higher in the CHF group (significant on the right). The same was true for thickening fraction (without statistical significance). These findings may be explained by increased work of respiratory muscles even under calm breathing. Literature data are also contradictory. Andriopoulou M.S. et al. [14] obtained smaller diaphragm excursion in patients with CHF, Spiesshoefer J.C. et al. [26] reported higher excursion.

The mechanisms of changes in our patients are presumably the following: respiratory muscles in CHF patients are forced to do a large work at rest to maintain effective tidal volume due to reduced lung compliance [8, 15]. This increases "metabolic and physiological cost" of breathing.

We found no significant between-group differences in diaphragm excursion under deep breathing. These findings contradict some data on less excursion under deep inspiration [14, 26]. Smaller diaphragm thickening and tidal volume under deep inspiration in our study are consistent with impossible maximum engagement of diaphragm muscles into deep breathing. This determines decrease of functional reserves.

The most likely explanation for "contradictory" results is between-group difference. The most severe (decompensated) CHF patients in our sample demonstrated reduction of diaphragm excursion and tidal volume presented in the literature.

Clinical example

A 67-year-old patient with respiratory failure requiring O2 insufflation 5 l/min due to decompensated CHF (LV EF 15%, TAPSE 11 mm, sPAP 65 mm Hg under infusion of dobutamine 5 μg/kg/min) was diagnosed with severe weakness of respiratory muscles. Spirometry data: IV_{cbr}/IV_{db} 278/1800 ml, P0.1 0.9 cm H_2O, NIF 20 cm H_2O.

Ultrasound of the diaphragm: thickness of the right dome under calm inhalation/exhalation — 4.3/3.8 mm; deep breathing — 4.7/2.3 mm. Thickening fraction under calm/deep breathing was 13/23%, excursion — 1.35/5.87 cm. Thus, increased thickness of the diaphragm was combined with low functional indicators (thickening fraction and excursion).

Complex therapy (beta-blockers, ACE inhibitors, diuretics, nitrates, inotropic support) compensated CHF (fluid balance in 5 days –16.950 ml). Respiratory failure regressed. Thickness of the diaphragm decreased, and its functional performance increased.

This case is remarkable in that the patient with decompensated CHF class IV had no indications for one or another mechanical respiratory support. This outlines the boundaries of functional classes, at least for intensive care specialist.

The actual, but insignificant difference in structural and functional ultrasonic parameters of the right and left domes of the diaphragm was previously revealed in healthy people [18]. We confirmed these data in the present study.

No strong correlations between spirometry parameters and structural/functional parameters of the diaphragm is confirmed in our findings [28] and literature data [19, 20, 29]. This fact is probably explained by unknown involvement of accessory muscles and insufficient data on diaphragm ultrasound.

Our study has a significant limitation. Although our study formally included patients with CHF class II—IV, almost all ones were in a compensated state without signs of respiratory failure prior to measurements. That is, higher functional classes did not allow identifying strong correlations between severity of myocardial dysfunction, spirometry and ultrasonic parameters.

Conclusion

Chronic heart failure causes some well-known and non-obvious changes in external respiration. For example, this is decrease of spirometric reserves (reduced maximum tidal volume and minute ventilation). Non-obvious result is thickening of the diaphragm with unclear nature (edema, hypertrophy?). In the most severe cases, generation of negative pressure is reduced.

Patients with cardiogenic respiratory failure, especially those requiring mechanical ventilation, are of the greatest interest regarding intensive care. Correction of diaphragmatic dysfunction in high-grade CHF can be an independent goal of setting the parameters of respiratory support. This may be necessary to minimize work of breathing and reduce oxygen consumption by respiratory muscles.

Further research will help to deepen and concretize the concept of diaphragm-protective mechanical ventilation.

The authors declare no conflicts of interest.

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