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Morphological and Functional Alterations of Respiratory Muscle Performance and Spirometry Parameters in Patients with Congestive Heart Failure

Vitaliy S. Shabaev*, Indira V. Orazmagomedova, Vadim A. Mazurok, Aelita V. Berezina, Andrei E. Bautin, Lyudmila G. Vasilyeva, Daria A. Aleksandrova

Almazov National Medical Research Centre, Ministry of Health of Russia,
2 Akkuratova Str., 197341 St. Petersburg, Russia

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***Correspondence to:** Shabaev Vitaliy Sergeevich, shabaev_vitaliy@mail.ru

Summary

The purpose of the study. To identify structural changes and functional modifications in respiratory muscle performance in patients with congestive heart failure.

Materials and methods. We conducted prospective observational study at the V. A. Almazov National Medical Research Center involving 118 subjects: 49 patients with congestive heart failure (CHF-group) and 69 healthy people (control group). NYHA functional classes of II to IV were taken as inclusion criteria in the CHF group, and respiratory diseases, abdominal pathology, morbid obesity, and anemia — as exclusion criteria.

Ultrasound imaging was used to assess the structural (thickness) and functional (thickening and excursion indices) diaphragmatic impairments during quiet (resting) and deep breathing. Facemask spirometry was used to assess pulmonary function.

Results. Patients with CHF were on average older than 59.0 years (53.0; 70.0) vs. 25.0 years (24.0; 26.0) in the control group, *P*=0.000001, had excessive body weight — 82.0 (73.0; 95.0) vs. 68.5 (55.0; 84.0) kg, *P*=0.000005 and higher body mass index — 28.4 (24.3; 31.3) vs 21.8 (19.9; 24.0) kg/m^2, *P*=0.000001, but did not differ in height 173.0 (166.0; 179.0) vs. 170.0 (165.0; 183.0) cm, 0.97.

Lower maximum inspiratory volume (MIV): 3000.0 (2300.0; 4000.0) vs. 3684.1 (3392.5; 4310.8) ml, *P*=0.0006, and negative inspiratory force (NIF) measured as max negative pressure generated by the respiratory muscles: 43.1 (–56.7; –33.0) vs. 53.5 (–58.8; –50.9) mBar, *P*=0.000082, respectively were found in patients with CHF. The diaphragm was significantly thicker (mm) in patients with CHF during quiet (eupnea) and deep breathing compared to healthy subjects. The thickness at the end of quiet inspiration was 3.0 (2.2; 3.6)/1.9 (1.5; 2.2) in the right hemi-diaphragm, *P*<0.001; and 3.0 (2.4; 3.5)/1.7 (1.4; 2.0) — in the left, *P*=0.000001; thickness at the end of quite expiration — 2.2 (1.8; 2.9)/1.5 (1.2; 1.7) in the right dome, *P*=0.000001; and 2.0 (1.7; 2.5)/1.4 (1.2; 1.5) — in the left, *P*=0.000001. Thickness at the end of deep inspiration was 5.1 (4.4; 6.1)/4.4 (3.6; 5.1) in the right dome, *P*=0.0005, and 4.9 (4.2; 6.2)/ 3.7 (3.1; 4.8) — in the left, *P*=0.000007.The diaphragm thickening index during deep breathing was lower in the CHF group than in the control group: 131.1 (82.5; 181.8) vs. 190.9 (150.0; 240.0) in the right dome, *P*=0.000004; and 148.8 (112.5; 190.3) vs. 175.2 (130.7; 227.7) — in the left, *P*=0.03, respectively.

Diaphragmatic excursions during quiet breathing were larger in patients with CHF than in healthy controls: 2.3 (1.6; 2.8)/1.7 (1.5; 1.9), *P*=0.0001 and 1.8 (1.5; 2.2)/1.5 (1.3; 1.9), *P*=0.03 of the right and left domes, respectively.

Conclusion. Congestive heart failure contributes to the development of structural and functional impairments of the diaphragm.

Keywords: congestive heart failure; ultrasound examination of the diaphragm; diaphragm; external respiration; diaphragm function; diaphragmatic dysfunction

Conflict of interest. The authors declare no conflict of interest.

Introduction

Chronic heart failure (CHF) can lead to a variety of symptoms, such as muscle dysfunction [1–3], including respiratory muscle dysfunction [2, 4, 5] that can be severe enough to cause ventilatory impairment [5]. Respiratory muscle dysfunction is considered a sign of multiorgan failure in advanced CHF [1] and is associated with increased dyspnea, reduced exercise tolerance and ultimately early death [1, 6–9].

Dysfunction of the diaphragm, as the most active respiratory muscle, is relevant to intensive care physicians and anesthesiologists because it may prolong ICU stay [10–12] and influence the choice of respiratory support [10, 13]. Meanwhile, current guidelines for preoperative assessment of respiratory function in patients with CHF include only lung auscultation [14].

The assessment of respiratory function in general and diaphragm performance in particular is especially relevant in CHF because it could help to determine the strategy of patient management in the ICU, including the choice of noninvasive and invasive ventilatory parameters [5, 10, 15].

While respiratory impairment in CHF is well known [2, 4, 5], diaphragmatic dysfunction, especially as assessed by ultrasound, has been poorly studied.

All the above provides a rationale for studying respiratory performance in critically ill patients with CHF, from both scientific and practical points of view.

Aim. To identify structural and functional changes in respiratory function in patients with chronic heart failure.

Materials and methods

A cross-sectional prospective descriptive study of structural and functional disorders of the respiratory system in patients with CHF was conducted at the Almazov National Medical Research Center from May 2022 to December 2022. The study followed the Helsinki Declaration of 2000 and was approved by the local ethics committee (protocol dated April 30, 2022).

Inclusion criterion for patients in the main (CHF) group was CHF II-IV NYHA functional class.

Exclusion criteria were respiratory diseases, morbid obesity, abdominal diseases, anemia (hemoglobin less than 120 g/l).

A total of 118 individuals were enrolled in the study (Figure 1), including 49 patients with CHF and 69 apparently healthy volunteers. The CHF group included 11 women and 38 men diagnosed with CHF at least one year prior to enrollment, with a mean age of 58.7±13.5 years, body weight of 84.7±18.3 kg, height of 172.8±8.6 cm, and BMI of 28.3±5.2 kg/m². Among all patients with CHF, 20 were diagnosed with NYHA class II, 15 with class III, and 14 with class IV. All patients were stable and receiving combination therapy including ACE inhibitors, beta-blockers, diuretics, aldosterone antagonists and statins. Patients with cardiac arrhythmias (atrial fibrillation) were on oral anticoagulants.

The control group consisted of 39 female and 30 male subjects with a mean age of 25.0±2.1 years, body weight of 68.56±15.62 kg, height of 173.4±10.7 cm, and BMI of 22.54±3.4 kg/m².

Ultrasound (US) examination of the diaphragm and measurement of respiratory function were performed in the supine position with the head end of the bed elevated 30 degrees, which is the basic position of patients in intensive care units. The structure (diaphragm thickness, DT) and function (diaphragm excursion, E; thickness fraction, DTF) of the diaphragm during quiet and deep inspiration/expiration were evaluated using a Philips CX50 ultrasound device (Philips Ultrasound, Inc., USA). Ultrasound parameters obtained only in patients with good visualization of the diaphragm were included in the final analysis.

Respiratory function was assessed using a Dräger Evita Infinity V500 ventilator (Germany) in noninvasive lung ventilation mode. A constant positive pressure of 0 mbar without pressure support and a FiO_2 of 0.21 were set on the device.

Tidal volumes during quiet (T_{vquiet}) and maximal deep (T_{vdeep}) breathing, inspiratory and expiratory times were measured. Neurorespiratory drive and respiratory muscle strength were estimated based on P0.1 (airway pressure at 100 ms of spontaneous attempt to breathe during occlusion of the breathing circuit) and NIF (negative inspiratory force, the minimum airway pressure during inspiration from a tightly closed circuit). Since NIF is conventionally used as a predictor of spontaneous breathing readiness with a weaning success threshold of less than –25 to –30 mBar [16], values above this threshold were considered indicative of weak respiratory muscles.

Hemoglobin oxygen saturation was assessed with a pulse oximeter.

The collected data were analyzed using STATISTICA 10.0 software and the Real Statistics Resource Pack Microsoft Excel add-on.

During the design phase, using the power analysis option of the STATISTICA-10 software, the sample size was estimated to be at least 100 subjects to achieve 80% power.

After the pilot study, calculations showed that the minimum number of subjects required in the main group was 28. In the end, 49 subjects were included in the study, giving a power of 0.9.

The pilot study was conducted according to the full study methodology to confirm the hypothesis of different diaphragm performance in patients with CHF, as well as to identify early the parameters with the greatest difference and to determine the minimum number of subjects required. First, the most different parameters, such as diaphragm thickness, were identified by taking the minimum number of observations necessary for statistical calculations according to the Mann–Whitney criterion, which was 3 healthy controls and 5 patients with CHF. Then, the group size was increased to 10 and the sample calculation for the whole study was performed. After obtaining

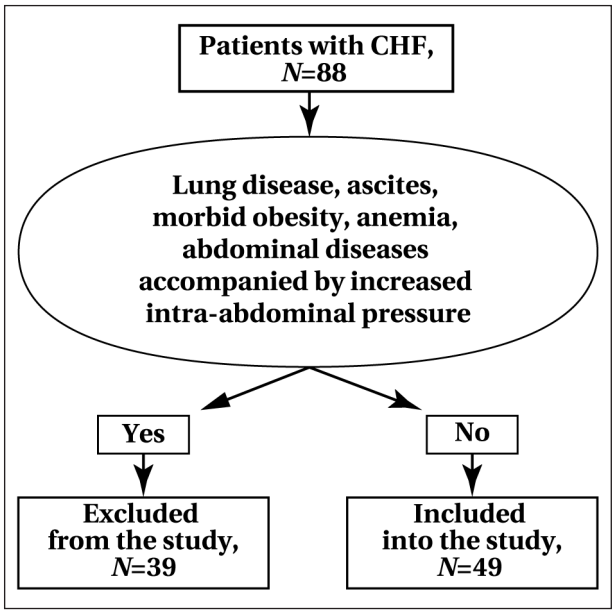

Fig. Patient inclusion flowchart.

the results and their statistical analysis in the initial stage, the study was continued in full length.

Normality of the distribution was assessed using the Kolmogorov–Smirnov and Shapiro–Wilk tests. Independent groups were compared using the Mann–Whitney test. Data were presented as median *(Me)* and interquartile range *(Q1; Q3)*. Differences were considered significant at $P \leqslant 0.05$.

Results

The results obtained are shown in Tables 1–6.

As shown in Table 1, patients with CHF were older and had higher body weight and BMI.

As shown in Table 2, patients with CHF had a higher respiratory rate, shorter inspiratory and expiratory times, lower V_{tdeep} and inspiratory force. In addition, patients with CHF had lower hemoglobin oxygen saturation, although SpO_2 values remained in the reference range.

As shown in Table 3, the diaphragm thickness of patients with CHF was significantly higher during quiet and deep breathing, whereas the thickening fraction was smaller during deep breathing.

As shown in Table 4, a higher diaphragm excursion was observed during quiet breathing in patients with CHF, whereas no differences were found during deep breathing. Visualization of the diaphragm on the left side was not always possible, which explains the smaller number of subjects included in the statistical analysis.

As Table 5 demonstrates, the times of craniocaudal (contraction) and caudal-cranial (relaxation) diaphragm motion during quiet and deep breathing

Table 1. Anthropometric parameters of the study groups, median (*Q1; Q3*).

Parameter	Values in groups		*P*
	Controls, *N*=69	Patients with CHF, *N*=49	
Age, years	25.0 (24.0; 26.0)	59.0 (53.0; 70.0)	0.000001*
Body mass, kg	68.5 (55.0; 84.0)	82.0 (73.0; 95.0)	0.000005*
Height, cm	170.0 (165.0; 183.0)	173.0 (166.0; 179.0)	0.97
BMI, kg/m²	21.8 (19.9; 24.0)	28.4 (24.3; 31.3)	0.000001*

Note. BMI — body mass index; CHF — chronic heart failure; * — significant difference ($P \leqslant 0.05$, Mann–Whitney test).

Table 2. Respiratory function parameters, median (*Q1; Q3*).

Parameter	Values in groups		*P*
	Controls, *N*=69	Patients with CHF, *N*=49	
Respiratory rate	13 (11; 15)	15 (13; 17.5)	0.0009*
$V_{t\ quiet}$, ml	560.0 (493.5; 678.0)	548,0 (450.0; 666.0)	0.37
IT, s	1.5 (1.3; 1.6)	1,3 (1.1; 1.4)	0.0001*
ET, s	3.0 (2.5; 3.7)	2,8 (2.3; 3.1)	0.008*
$V_{t\ deep}$, ml	3684.1 (3392.5; 4310.8)	3000.0 (2300.0; 4000.0)	0.0006*
SpO_2, %	99 (99; 99)	97.4 (97.0; 98.0)	0.000001*
P 0.1, mbar	–2.1 (–3.0; –1.5)	–1.3 (–1.8; –0.9)	0.000003*
NIF, mbar	–53.5 (–58.8; –50.9)	–43.1 (–56.7; –33.0)	0.000082*

Note. $V_{t\ quiet}$ — tidal volume in quiet breathing; $V_{t\ deep}$ — tidal volume in deep breathing; IT — inspiratory time; ET — expiratory time; NIF — negative inspiratory force; P0.1 — airway occlusion pressure at 100 ms; * — significant difference ($p \leqslant 0.05$, Mann–Whitney test).

Table 3. Diaphragm thickness, median (*Q1; Q3*).

Parameter	Values in groups		*P*
	Controls, *N*=69	Patients with CHF, *N*=49	
$DT_{quiet-insp}$, right , mm	1.9 (1.5; 2.2)	3.0 (2.2; 3.6)	0.000001*
$DT_{quiet-exp}$, right, mm	1.5 (1.2; 1.7)	2.2 (1.8; 2.9)	0.000001*
$DT_{deep-insp}$, right, mm	4.4 (3.6; 5.1)	5.1 (4.4; 6.1)	0.0005*
$DT_{deep-exp}$, right, mm	1.1 (1.0; 1.4)	1.7 (1.3; 1.9)	0.000001*
$DT_{quiet-insp}$, left, mm	1.7 (1.4; 2.0)	3.0 (2.4; 3.5)	0.000001*
$DT_{quiet-exp}$, left, mm	1.4 (1.2; 1.5)	2.0 (1.7; 2.5)	0.000001*
$DT_{deep-insp}$, left, mm	3.7 (3.1; 4.8)	4.9 (4.2; 6.2)	0.000007*
$DT_{deep-exp}$, left, mm	1.1 (0.9; 1.2)	1.6 (1.3; 2.0)	0.000001*
$DTF_{quiet-insp}$, right, %	27.8 (20.0; 35.0)	30.4 (17.9; 44.8)	0.38
$DTF_{deep-insp}$, right, %	190.9 (150.0; 240.0)	131.1 (82.5; 181.8)	0.000004*
$DTF_{quiet-insp}$, left, %	23.6 (18.3; 33.0)	40.9 (28.5; 59.8)	0.000002*
$DTF_{deep-insp}$, left, %	175.2 (130.7; 227.7)	148.8 (112.5; 190.3)	0.03*

Note. DT — diaphragm thickness; DTF — diaphragm thickening fraction; insp — inspiration; exp — expiration; * — significant difference ($P \leqslant 0.05$, Mann–Whitney test).

were shorter in patients with CHF than in healthy controls.

Table 6 demonstrates that during quiet inspiration, the rate of cranial-caudal and caudal-cranial diaphragmatic movements was higher on both sides in the group of patients with CHF. During deep exhalation, only the right hemisphere of the diaphragm moved faster, while only a tendency for faster kinetics was observed on the left side.

Discussion

The diaphragm, along with the myocardium, is an almost continuously working muscle, which explains its significant oxygen consumption [17] and determines a significant susceptibility to over- or under-exertion as well as to oxygen delivery [18]. Chronic heart failure is associated with reduced oxygenation of the respiratory muscles, especially the diaphragm [5]. The data obtained indicate a

Table 4. Diaphragm excursion, median (*Q1; Q3*).

Parameter	Values in groups		*P*
	Controls	Patients with CHF	
Number of patients tested	69	49	—
E_{quiet}, right	1.7 (1.5;1.9)	2.3 (1.6;2.8)	0.0001*
E_{deep}, right	6.9 (6.0;8.0)	6.9 (5.8;9.1)	0.67
Number of patients tested	29	34	—
E_{quiet}, left	1.5 (1.3;1.9)	1.8 (1.5;2.2)	0.03*
Number of patients tested	29	26	—
E_{deep}, left	6.0 (5.3;6.9)	5.8 (4.5;7.3)	0.41

Note. E — excursion; quiet — quiet breathing; deep — deep breathing; * — significant difference ($P \leqslant 0.05$, Mann–Whitney test).

Table 5. Time parameters of diaphragm excursion, median (*Q1; Q3*).

Parameter	Values in groups		*P*
	Controls	Patients with CHF	
Number of patients tested	69	49	
$T_{contr/quiet}$, right	1.4 (1.1; 1.7)	1.2 (1.0; 1.4)	0.029*
$T_{relax/quiet}$, right	1.4 (1.2; 1.4)	1.1 (0.9; 1.4)	0.003*
$T_{contr/deep}$, right	2.2 (1.9; 2.8)	1.9 (1.5; 2.3)	0.001*
$T_{relax/deep}$, right	2.4 (2.0; 2.7)	1.8 (1.2; 2.6)	0.00002*
Number of patients tested	29	34	
$T_{contr/quiet}$, left	1.4 (1.1; 1.6)	1.2 (1.0; 1.4)	0.02*
$T_{relax/quiet}$, left	1.4 (1.1; 1.7)	1.0 (0.9; 1.4)	0.007*
Number of patients tested	29	26	
$T_{contr/deep}$, left	2.2 (1.8; 2.8)	1.9 (1.5; 2.3)	0.08
$T_{relax/deep}$, left	2.4 (2.0; 2.7)	1.8 (1.2; 2.6)	0.01*

Note. Contr — cranio-caudal excursion (contraction); relax — caudo-cranial excursion (relaxation); quiet — quiet breathing; deep — deep breathing; T — time; * — significant difference ($P \leqslant 0.05$, Mann–Whitney test).

Table 6. Velocity parameters of diaphragm excursion, median (*Q1; Q3*).

Parameter	Values in groups		*P*
	Controls	Patients with CHF	
Number of patients tested	69	49	—
$R_{contr/quiet}$, right	1.0 (0.8; 1.3)	1.8 (1.3; 2.3)	0.000001*
$R_{relax/quiet}$, right	1.1 (0.9; 1.4)	1.8 (1.3; 2.3)	0.000001*
$R_{contr/deep}$, right	3.3 (2.4; 4.5)	2.7 (1.2; 3,4)	0.7
$R_{relax/deep}$, right	2.9 (2.0; 3.5)	3.4 (2.7; 5.2)	0.001*
Number of patients tested	29	34	
$R_{contr/quiet}$, left	1.2 (0.9; 1.7)	1,7 (1.3; 2.3)	0.03*
$R_{relax/quiet}$, left	1.3 (0.9; 1.8)	1,7 (1.3; 2.3)	0.02*
Number of patients tested	29	26	
$R_{contr/deep}$, left	2.6 (2.2; 3.0)	2.7 (1.9; 4.0)	0.7
$R_{relax/deep}$, left	2.8 (2.1; 2.9)	3.3 (2.0; 4.1)	0.08

Note. R — rate; contr — contraction; relax — relaxation; quiet — quiet breathing; deep — deep breathing; * — significant difference ($P \leqslant 0.05$, Mann–Whitney test).

significant difference in almost all parameters, both spirometric evaluation of respiratory function and ultrasound structural and functional characteristics of the diaphragm in patients with CHF compared to healthy controls.

The lower hemoglobin oxygen saturation found in patients with CHF was anticipated [19]. Increased respiratory rate during quiet breathing in the group of patients with chronic heart failure allowed to compensate the decreased tidal volume [20]. However, during deep breathing, more severe structural and time-velocity disturbances were found, indicating reduced respiratory reserves [19, 20–22], probably due to impaired muscle function [21, 22] and the development of restrictive respiratory failure in CHF [19, 22, 23].

Respiratory muscle strength on inspiration was significantly lower in the group of patients with CHF. P0.1 is considered an indicator of respiratory drive, which is not quite equivalent to respiratory muscle strength [22, 24], so its interpretation requires caution. Researchers have not found changes in P0.1 in patients with CHF [22, 23], which is to some extent confirmed by our data. Despite the fact that P0.1 in patients with CHF was significantly lower (modulo) than in healthy individuals, in absolute terms the parameter did not exceed the reference range, which can be interpreted as the absence of significant disorders of neurorespiratory drive. The observed reduction of NIF modulo values in patients with CHF compared to healthy controls was expected and is consistent with the data of other researchers [1, 21, 22, 25].

Ultrasound structural (thickness) parameters of the diaphragm were significantly higher in patients with CHF on both sides during quiet and maximal deep inspiration and expiration. This does not agree with the results of Spiesshoefer J. et al [25], who found no differences in diaphragm thickness at the end of quiet expiration in healthy subjects and patients with CHF, whereas diaphragm thickness during deep breathing was greater in healthy subjects [25]. On the other hand, Miyagi M. et al [21] reported greater diaphragm thickness in patients with lower left ventricular ejection fraction, which is consistent with the results of our study.

Spiesshoefer J. et al. showed that the diaphragmatic thickening fraction during quiet breathing is lower in patients with CHF than in healthy subjects [25]. The data obtained are consistent with this finding, but only during maximal deep inspiration and with visualization of both right and left hemispheres. In general, it is rather difficult to compare the magnitude of diaphragmatic thickening because its calculation can be performed using different formulas [25, 26].

The assumption that diaphragm excursion decreases in the group of patients with CHF regardless of the depth of breathing has not been confirmed [27]. The amplitude of diaphragmatic motion during quiet breathing did not differ between healthy subjects and patients with CHF and preserved ejection fraction, whereas during deep breathing it was significantly lower in patients with reduced ejection fraction [25].

Our results differ from the above data: diaphragm excursion during quiet breathing was significantly greater in the group of patients with CHF than in the control group on both the right and left sides, whereas during deep breathing it did not differ significantly between the groups. The patients with CHF may already be doing relatively more work to maintain the effective tidal volume at rest, i.e., their metabolic and physiological «cost» of breathing increases.

The time-velocity parameters of diaphragmatic excursion are poorly studied. The authors of the previously mentioned study [25] did not find any changes in diaphragmatic kinetics during quiet inspiration in patients with CHF. According to our data, the time of diaphragmatic movements during inhalation and exhalation during quiet and deep breathing decreased on both sides and the velocity increased accordingly in the CHF group.

The observed increase in time-velocity characteristics during inspiration can be explained by the involvement of respiratory auxiliary muscles and the reduced ability to «hold the breath» due to dynapenia (decrease in muscle strength with preserved muscle mass) rather than by improved diaphragm performance. The increase in time-velocity characteristics during expiration can be explained by increased lung compliance.

All changes in respiratory function in CHF could be compensatory and explained by several pathophysiological mechanisms. Chronic heart failure associated with interstitial pulmonary edema causes increased elastic recoil (i. e., decreased compliance) of the lungs and chest wall stiffness [25, 28]. These changes are likely to increase the load on the diaphragm during inspiration as the reduced lung compliance must be overcome, ultimately leading to its hypertrophy.

However, these suggestions are not consistent with histologic data showing a decrease in muscle mass and its replacement by connective and adipose tissue [29]. Another hypothesis is that the diaphragm may be enlarged due to edema. In any case, the data in the literature [5, 6, 8, 25, 27] are controversial and limited by small samples, which calls for more research in this area.

Our study had several limitations. First, the patients with CHF were older and had higher body weight. Therefore, we cannot postulate that the results obtained were solely due to CHF. Second, all patients were relatively well compensated, whereas

the greatest changes in spirometric and ultrasound parameters would probably be expected in overt respiratory failure. Finally, functional class is a rather volatile indicator of severity in CHF patients, and changes in a patient's functional performance over a short period of time may influence the results obtained.

Conclusion

Anatomical and physiological evidence suggests that chronic heart failure is associated with impairment of the structure and function of the diaphragm, the main respiratory muscle. Its thickening, changes in the amplitude of cranio-caudal and caudal-cranial movements and time-velocity parameters indicate a decrease in the functional reserves of the respiratory system.

The strength of muscle contraction decreases, which leads to a decrease in tidal volume and, consequently, an increase in respiratory rate, i.e., the respiratory pattern changes to a more superficial and more frequent one.

Such a change in respiratory pattern, which is typical of patients with chronic heart failure, suggests that the structural and functional differences in diaphragm parameters found are also due to CHF rather than age. Further research into the impact of CHF on diaphragm performance is warranted.

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