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Chest wall regional volume in heart failure patients during inspiratory loaded breathing

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ABSTRACT

Were evaluated individuals divided into two groups: we studied chronic heart failure (CHF) (19 patients with CHF plus cardiomegaly) and control (12 healthy volunteers) during performance of inspiratory loaded breathing (ILB). We evaluated: spirometry, functional capacity through the six-minute walk test (6MWT), and distribution of thoracoabdominal volumes via optoelectronic plethysmography (OEP), namely volume variations of pulmonary rib cage (Vrc,p), abdominal rib cage (Vrc,a), and abdomen (Vab). In each compartment, the percentage contributions of right and left sides were also calculated. During ILB, patients with heart failure were characterized by a significant reduction of the Vrc,a volume variations compared to the control group. Correlations were found between left %Vrc,a on the left side measured during ILB and left ventricular ejection fraction (r=0.468; p=0.049), and dyspnea after the 6MWT (r=-0.878; p<0.01).Then, patients with CHF and cardiomegaly are characterized by a reduced mobility in left part of the lower part of the rib cage, that contributes leading to increased perception of dyspnea during submaximal exercise.

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1. Introduction

The main symptoms of chronic heart failure (CHF) are dyspnea and fatigue (Jefferies and Towbin, 2010; Pina, 2003). Various studies have pointed out how these symptoms are related to abnormalities in respiratory muscles (Drexler et al., 1992; Coats, 1996) and the presence of cardiomegaly (Olson et al., 2006). Inspiratory muscle dysfunction has been reported as a reduction in the capacity to generate inspiratory muscle pressure and strength, a functional decline which can be attributed to histological and biochemical changes. Diaphragm biopsies from CHF patients have demonstrated the

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Studies on the role of cardiomegaly in respiratory symptoms of CHF patients are scarce in the literature (Olson et al., 2007). Expansion of the limited thoracic volume, where extra-pulmonary restriction may be caused by competition between the lungs and heart for intrathoracic space, can lead to imbalance in the thoracoabdominal system. As the disease progresses and worsens, associated with cardiomegaly, minor effort leads to more frequent and severe dyspnea episodes and early muscle fatigue sets in (Ulrik et al., 1999).

Optoelectronic plethysmography (OEP) is used to elucidate the influence of cardiomegaly in regional distribution of ventilation in the thoracoabdominal system of CHF patients (Aliverti and Pedotti,

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2003). No studies were found in the literature using used the technique for this population. Therefore, the hypothesis for this study is that individuals with CHF and cardiomegaly associated with diaphragmatic weakness exhibit volumetric differences in the thoracoabdominal system during the inspiratory loaded breathing (ILB) test when compared to healthy subjects.

The present study aimed to investigate whether alterations in regional chest wall displacement, reflecting abnormalities in respiratory muscle action, are present in CHF patients with cardiomegaly, and if these alterations are related to other functional parameters, namely dyspnea.

2. Methods

2.1. Participant characteristics

This was a cross-sectional cohort study in which a total of 31 individuals were evaluated and divided into two groups: CHF and control. In the CHF group, nineteen patients diagnosed with CHF were recruited from an outpatient clinic at a hospital cardiac center from May to December 2010, according to the following inclusion criteria: sedentary adults aged between 21 and 65 years; both sexes; diagnosed with CHF associated with cardiomegaly; functional class II and III; hypertensive, ischemic, and Chagas disease etiology; left ventricular ejection fraction (EF)<45%; inspiratory muscle weakness (predicted MIP < 70%) (Neder et al., 1999); clinical stability (>3 months); duration of symptoms > 1 year, body mass index (BMI) $< 35 \text{ kg/m}^2$ and non-smokers or former smokers with a smoking history <10 packs/year. Patients with the following characteristics were not considered: unstable angina; myocardial infarction or cardiac surgery in the three months prior to the start of the research; orthopedic diseases or respiratory comorbidities such as asthma and COPD. All patient medication was optimized for CHF throughout the study. The control group consisted of twelve volunteer participants with similar age, sex, and body mass index to the CHF group. Control participants displayed a left ventricular ejection fraction (EF) > 50% and had no cardiac chamber abnormalities, history of hypertension, lung disease, or cardiac ischemia; MIP 80% above (Neder et al., 1999) that predicted, in addition to being sedentary. All participants were instructed regarding the research and signed informed consent. This study was approved by the institutional Human Research Ethics Committee and its registration was accepted by ClinicalTrial.gov under registration NCT 01292902.

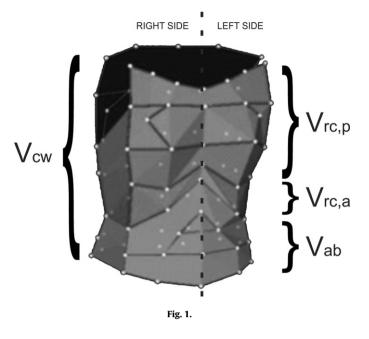
2.2. Functional data

2.2.1. Pulmonary function and strength measurement

Inspiratory muscle strength was evaluated using a digital manometer (MVD-300, *Globalmed*, Brazil) connected to a mouthpiece with a 2 mm opening. Each patient performed three maneuvers with maximum variation of up to 10% between them to achieve MIP (Neder et al., 1999), from residual volume (RV) to total lung capacity (TLC). The best of the three maneuvers was recorded. A portable spirometer (*Micro Medical, Microloop*, MK8, England) was used for pulmonary function testing. Forced vital capacity (FVC) and forced expiratory volume in one second (FEV1) were evaluated in accordance with recommendations of the American Thoracic Society (American Thoracic Society, 2002).

2.2.2. Functional capacity evaluation

The six-minute walk test (6MWT) was used to assess functional capacity in terms of distance covered (6MWD) in accordance with protocol established by the American Thoracic Society (ATS)(2002). The following resting parameters were evaluated before testing: arterial pressure (Pa), heart rate (HR), oxygen saturation (SpO2



measured by Onyx 9500 portable pulse oximeter), respiratory rate (RR), and dyspnea scale (Borg Scale).

2.3. Protocol for assessment during inspiratory loaded breathing

Inspiratory loaded breathing testing was performed with a threshold device (Threshold Inspiratory Muscle Trainer, Healthscan Products Inc., Cedar Grove, New Jersey), mostly used for inspiratory muscle training in healthy subjects (Hostettler et al., 2011) and in patients with various pathologies such as CHF (Dall'Ago et al., 2006; Chiappa et al., 2008). This device was connected the mouthpiece. During the three-minute-long test (De Andrade et al., 2005), patients breathed through the mouthpiece with their noses occluded by a noseclip, using 30% MIP. An inspiratory load of 30% was chosen taking into consideration several studies of inspiratory muscle training for this population (Laoutaris et al., 2004; Dall'Ago et al., 2006; Chiappa et al., 2008). During the test, the participants were encouraged to maintain respiratory frequency between 12 and 16 bpm. Testing was interrupted if HR increased more than 20% and/or SpO2 <88%.

2.4. Assessment of regional chest wall volumes by optoelectronic plethysmography

Optoelectronic plethysmography (BTS Bioengineering, Italy) measures volume changes in the thoracoabdominal system through the placement of 89 markers formed by hemispheres covered with retro-reflective paper. The location of each hemisphere is determined by anatomical references in the anterior and posterior regions of the thorax and abdomen. Markers were placed on the skin using hypoallergenic bioadhesives. Eight cameras were placed around the patient and recorded images were transmitted to a computer, where a three-dimensional model is formed based on the markers OEP capture software (BTS Bioengineering, Italy). The chest wall was divided into the following compartments (Fig. 1), each separated into its right and left parts: pulmonary rib cage (RCp – portion of the rib cage opposed to the lung); abdominal rib cage (RCa – portion of the rib cage opposed to the diaphragm); and abdomen (AB) (Aliverti and Pedotti, 2003). OEP data acquisitions were performed while individuals were seated with their arms at their sides. Data were gathered on two separate occasions:

Table 1

Demographics data and clinical features.

Variables	Groups	<i>p</i> -Value		
	Control	Heart failure		
n (% female)	12(58.4)	19(36.0)	0.06	
Age (years)	44.83 ± 12.67	52.83 ± 8.6	0.06	
BMI (kg/m ²)	23.77 ± 2.28	25.07 ± 3.7	0.3	
HR (bpm)	79.4 ± 8.7	72.9 ± 7.74	0.06	
RR (ipm)	15.75 ± 3.95	16.63 ± 2.57	0.53	
V ejection fraction (%)	64.41 ± 9.76	33.23 ± 8.91	< 0.01	
DDLV	44.33 ± 2.94	64.73 ± 10.05	<0.01	
SDLV	26.25 ± 2.56	55.93 ± 9.52	<0.01	
FVC (L/s)	3.66 ± 0.41	2.63 ± 0.98	0.03	
% Pred FVC	93.40 ± 13.16	76.07 ± 21.77	0.01	
FEV ₁ (L/s)	3.03 ± 0.72	2.09 ± 0.72	0.01	
۶ Pred FEV ₁	94.43 ± 15.22	73.85 ± 22.15	0.01	
Etiology [n(%)]				
Chagas Disease	-	10(52.63)	_	
Hypertensive	-	5(26.31%)	_	
schemic	-	4(21.05%)	-	
NYHA class [n(%)]				
Class II	-	9(47.36%)	_	
Class III	-	10(52.63%)	-	
Medications [number(% of total)]				
3-blocked	-	8(42.10%)	-	
Digitalis	-	6(31.57%)	_	
Diuretics	-	12(63.15%)	-	
MIP (cmH ₂ O)	95.55 ± 12.33	45.94 ± 15.17	<0.01	
%MIP	97.80 ± 16.08	47.30 ± 14.32	< 0.01	
Distance (m) in 6MWT	610.75 ± 73.95	426.47 ± 132.08	<0.01	
Distance (m) predicted for 6MWT	89.09 ± 16.64	72.02 ± 23.40	0.04	
Borg Scale before 6MWT	0.18 ± 0.02	0.06 ± 0.01	0.26	
Borg Scale after 6MWT	1.13 ± 0.35	4.27 ± 2.27	<0.01	

Values are given as the mean \pm SD. Level of significance p < 0.05. HR = heart rate; RR = respiratory rate; FVC = forced vital capacity; FEV₁ = forced expired volume in the first second; DDLV = diastolic diameter of left ventricle; SDLV = systolic diameter of left ventricle and MIP = maximal inspiratory pressure.

first, during three minutes of normal breathing and then during the inspiratory loaded breathing exercise with Threshold[®] ILB.

3. Statistical analysis

Statistical analysis was performed by SPSS 18.0 software. The following tests and analyses were conducted: Kolmogorov–Smirnov and Levene tests to assess sample normality and analyze intergroup homogeneity; *t*-test for independent samples for intragroup comparison of the right and left sides of compartmental chest wall volumes and same side compartmental volumes during normal breathing and inspiratory muscle training; *t*-test for dependent samples, for intragroup comparisons of chest wall volumes of the same side during normal breathing and inspiratory muscle training; Pearson's correlation analysis to evaluate the relationship between abdominal rib cage volume on the left side and predicted MIP, 6MWD, and EF. Data were described as mean \pm standard deviation (SD). Confidence intervals and differences were regarded as significant at 95% and *p* < 0.05, respectively.

4. Results

4.1. Population characteristics

The sample was calculated based on a pilot study for a power of 90% and α = 0.05. A 40% increase in abdominal thoracic volume (Vrc,a) on the left side was observed for the control group compared to the group with heart failure. Clinical, demographic and medication characteristics are described in Table 1. Intergroup

differences include lower EF (p < 0.01) and higher left ventricle systolic diameter (LVSD) and left ventricle diastolic diameter (LVDD) (both with p < 0.01) for the CHF group compared with the control group. Controls were characterized by higher FVC%pred and FEV1%pred than the CHF group (p = 0.03 and p = 0.01, respectively). The control group also showed greater FVC and FEV1 in absolute values (p = 0.01 for both comparisons). In relation to MIP, control subjects exhibited higher absolute and %predicted values (p < 0.01 for both comparisons) compared to the CHF group. Subject belonging to the control group covered an higher 6MWD than CHF (p < 0.01).

4.2. Regional distribution of chest wall volumes

Table 2 shows the comparison of regional chest wall volume distribution between normal breathing and ILB on the same side of the thoracoabdominal system for each of the groups, as well as a between-group analysis. When analyzing each group separately, a significant increase was observed for all thoracoabdominal compartments, on both sides during ILB for the two groups. CHF patients showed significantly lower Vrc,a variations (both sides) compared to the control group during ILB. Table 3 displays the comparison between right and left percentages of volume variations for each compartment of the chest wall during normal breathing and IMT for each group. Significant differences between right and left sides for variations of Vrc,a, Vab, and Vcw were present during the performance of ILB in the CHF group (p < 0.01, p < 0.01, and p = 0.04, respectively). Percentage contribution of left and right parts, respectively, was: $45.30 \pm 9.10\%$ and $54.33 \pm 12.9\%$ in Vrc,a

Comparison of volume variations of the six compartments of the thoracoabdominal system during quiet breathing and during inspiratory loaded breathing.
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Lung volumes (compartments)	Control group			Heart failure group				Control group × heart failure group (left side)	Control group × heart failure group (right side)	
	Left side	р	Right side	р	Left side	р	Right side	р	р	р
Vrc,p (Liters)										
QB	0.08 ± 0.03	< 0.01*	0.09 ± 0.04	< 0.01*	0.09 ± 0.05	0.03*	0.09 ± 0.04	< 0.01*	0.639	0.85
ILB	0.25 ± 0.08		0.26 ± 0.09		0.17 ± 0.11		0.18 ± 0.09		0.08	0.06
Vrc,a (Liters)										
QB	0.09 ± 0.12	< 0.01*	0.07 ± 0.06	< 0.01*	0.04 ± 0.02	< 0.01*	0.04 ± 0.02	0.01*	0.06	0.06
ILB	0.17 ± 0.06		0.20 ± 0.15		0.08 ± 0.05		0.10 ± 0.08		0.01*	0.04*
Vab (Liters)										
QB	0.12 ± 0.01	0.03*	0.13 ± 0.01	0.02*	0.12 ± 0.06	0.04*	0.12 ± 0.04	0.01*	0.86	0.72
ILB	0.27 ± 0.02		0.28 ± 0.02		0.20 ± 0.01		0.23 ± 0.19		0.36	0.53
Vcw (Liters)										
QB	0.26 ± 0.08	< 0.01*	0.27 ± 0.08	< 0.01*	0.25 ± 0.10	< 0.01*	0.26 ± 0.08	< 0.01*	0.82	0.88
ILB	0.65 ± 0.3		0.67 ± 0.3		0.47 ± 0.25		0.52 ± 0.28		0.95	0.15

QB – quiet breathing; ILB – inspiratory loaded breathing (30%Pimax); Vrc,p – pulmonary rib cage volume; Vrc,a – abdominal rib cage volume; Vab – abdominal volume; Vcw – chest wall volume. Values are expressed as mean ± SD. Consider significant as *p* < 0.05^{*}. Test *t* for dependent samples for into the same group and Test *t* for independent samples between two groups.

In the first four columns can observe the behavior of the distribution of total volume within the same group and same for right or left side of the thoracoabdominal compartment when it is compared, along the QB and ILB. The last two columns represent the behavior of these variables between the groups and to the same side of the thoracoabdominal system for both the QB and for the ILB.

 $45.00\pm6.52\%$ and $55.00\pm6.52\%$ in Vab, and $48.04\pm5.38\%$ and $52\pm5.31\%$ in total chest wall volume (Vcw).

4.3. Correlation of % Vra on the left side with the functional data

A significant negative correlation (r = -0.878 and p < 0.01) was found between Borg Scale after the 6MWT and the Vrc,a (left side) during ILB (Fig. 2). A linear correlation at the limit of significance (r = 0.468 and p = 0.049) was present between Vrc,a (left side) and LV ejection fraction during ILB (Fig. 3). No significant correlations were recorded between variations of Vrc,a (left side) during IMT

Table 3

Percentage contribution of the right and left side of each of the six compartments of the chest wall.

	Control group	p-Value	Heart failure group	p-Value
Quiet breath	ning			
Vrc,p (%)				
Left side	49.67 ± 3.64	0.348	50.20 ± 3.43	0.714
Right side	51.35 ± 4.86		49.80 ± 3.43	
Vrc,a (%)				
Left side	50.95 ± 4.70	0.329	47.52 ± 5.93	0.273
Right side	49.03 ± 4.7		50.77 ± 9.40	
Vab (%)				
Left side	48.40 ± 5.95	0.207	47.09 ± 12.7	0.219
Right side	51.56 ± 5.92		50.41 ± 5.80	
Vcw (%)				
Left side	49.38 ± 2.30	0.214	49.15 ± 2.79	0.072
Right side	50.58 ± 2.30		50.83 ± 2.79	
Inspiratory l	oaded breathing (3	30% MIP)		
Vrc,p(%)		, ,		
Left side	49.67 ± 3.64	0.348	49.09 ± 10.39	0.273
Right side	51.35 ± 4.86		52.88 ± 10.00	
Vrc,a (%)				
Left side	50.81 ± 5.11	0.583	45.30 ± 9.10	< 0.01*
Right side	49.60 ± 5.5		54.33 ± 12.97	
Vab (%)				
Left side	48 ± 3.86	0.053	45.00 ± 6.52	< 0.01*
Right side	51.46 ± 4.64		55.00 ± 6.52	
Vcw (%)				
Left side	49.67 ± 4.45	0.214	48.04 ± 5.38	0.043*
Right side	51.15 ± 3.18		52 ± 5.31	

Vrp – pulmonary rib cage volume; Vra – abdominal rib cage volume; Va – abdominal volume; Vcw – chest wall volume. Values are expressed as mean \pm SD. Consider significant as p < 0.05. Test t for independent samples.

and 6MWD (r = -0.064 and p = 0.79), LSVE (r = 0.03 and p = 0.89), and LVSD (r = -0.11 and p = 0.695).

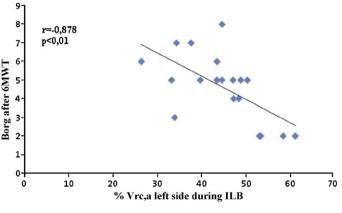
5. Discussion

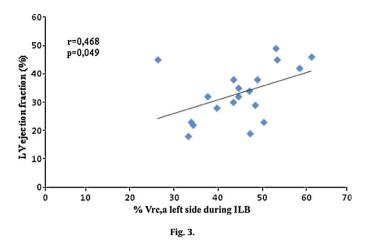
5.1. Major finding

The present study demonstrates significant differences in regional distribution of thoracoabdominal volumes between patients with heart failure associated with cardiomegaly and healthy controls. More specifically, the left side of the lower rib cage is characterized by lower displacement during ILB breathing. Regional distribution differences in chest wall volume are correlated with other functional parameters, namely left ventricular ejection fraction and dyspnea.

5.2. Pulmonary function, inspiratory muscle strength, 6MWT and Borg Scale

Patients with CHF were characterized by impaired lung function, as shown by the lower FVC, FEV1, and FEF values compared to healthy individuals. Some authors attribute these findings to respiratory muscle weakness, lung fluid imbalance, and exaggerated neurohumoral activity (Rutten et al., 2006; Johnson et al., 2000; Daganou et al., 1999; Puri et al., 1994). Agostoni et al.





(2000) proposed an influence of cardiomegaly on pulmonary function. According to this study, patients with cardiomegaly, defined by an increase in cardiothoracic index, showed lower FEV1 and FVC. In the present study, cardiomegaly was determined by the increase in left ventricular systolic and diastolic diameters. This amplification in cardiac chambers could be considered a competing factor with pulmonary parenchyma, leading to deterioration in pulmonary function (Olson et al., 2006, 2007; Agostoni et al., 2000). In relation to inspiratory muscle strength, MIP < 70% was used as an inclusion criterion for the CHF group. Respiratory muscle weakness and physical deconditioning may be involved in the increase in respiratory work during hyperpnea at the time of task performance (Witte and Clark, 2005; Clark et al., 1995). Reduced functional capacity, assessed by the 6MWT, associated with less strength and endurance generated by inspiratory muscles are factors that worsen CHF patient prognosis and survival (Meyer et al., 2001). This study recorded a decrease in distance covered and a rise in the Borg index after the 6MWT for CHF group patients when compared to healthy subjects.

5.3. Distribution of chest wall volume variations

During ILB, the CHF group displayed smaller volume variations in the lower rib cage compared to controls. Since this compartment reflects direct action of the diaphragm on the rib cage, reduced values observed in CHF patients during ILB demonstrate its impaired action in developing inspiratory pressure. This is particularly evident during ILB, that is, a situation requiring a significant rise in inspiratory muscle pressure (Meyer et al., 2001). It is important to note that decreased lower rib cage displacement in CHF patients is not associated to reduced overall chest wall volume variations. This suggests the presence of compensatory mechanisms in the upper rib cage and abdominal compartments Aliverti et al. (1997) observed that, during exercise, abdominal and rib cage muscles play a double role of preventing costly rib cage distortions and unloading the diaphragm so that it acts as a flow generator. Furthermore, the rib cage and abdominal muscles assume the task of developing the pressures required to move the rib cage and abdomen, respectively.

This mechanism could be the base of similar compensatory mechanisms observed in the CHF group. Another original finding in the present study was that in both compartments submitted to the action of the diaphragm, namely the lower rib cage and the abdomen, during ILB displacement of the left side was significantly lower than the right in CHF patients, but not among controls. A possible explanation is that cardiomegaly would limit effective diaphragmatic displacement on the left side, where a heart with increased volume might represent a mechanical load for the diaphragm, altering its normal return to its relaxed position. This hypothesis is supported by Olson et al. (2006) who studied the relationship between cardiac and pulmonary volume in the thoracic cavity of 44 individuals with CHF compared to healthy individuals via radiographic analysis. These authors observed a strong correlation between heart size and pulmonary volume reduction for CHF patients. They also suggest that increased cardiac volume and reduced pulmonary volume could contribute to the rapid and shallow breathing frequently observed in this population, particularly during exercise. In another study, the same group (Olson et al., 2007) evaluated pulmonary function in CHF patients with cardiomegaly and observed lower values of FVC, FEV1, FEV1/FVC, and FEF 25-75%. More recently, Olson and Johnson (2011) studied the influence of cardiomegaly on respiratory disorder during exercise in patients with CHF and showed a strong correlation between cardiac volume in tidal volume changes and respiratory frequency during exercise.

5.4. Study limitations

A limitation of this study is the absence of an additional group for comparison, composed of patients with cardiomegaly related CHF without inspiratory muscle weakness, enabling effects for each of these variables to be evsluated in separadely. However, our data can be extrapolated for patients with CHF associated with muscle weakness, elements commonly found in patients with CHF functional class II or III (NYHA). In addition, the present study did not correlate thoraco-abdominal system volumes with variables from maximal cardiopulmonary exercise test (VO2-Oxygen uptake (Oxygen consumption) or VE/VCO 2, ventilatory equivalents for CO2) and with DLCO (Lung diffusion for carbon monoxide), which could better elucidate behavior of these volumes during maximum respiratory demand. This correlation is an important point to be consider in the future studies as well concomitant OEP assessment during submaximal exercise. The submaximal exercise selected in the present study was the six-minute walk test, since it corresponds to the demands of activities of daily living. As such, OEP evaluation of thoracoabdominal system volumes concomitant to this test would not be possible.

5.5. Clinical implications

Cardiomegaly and inspiratory muscle weakness are common in patients with CHF. However, the exact action mechanisms of these two associated or isolated factors in the determination of respiratory symptoms are still unknown. According to our study, lower chest wall expansion in the diaphragmatic region would lead to an increased perception of dyspnea during submaximal exercise in this population. Moreover, changes observed in the pattern of regional chest wall volume distribution in CHF patients compared to healthy individuals could serve as a base for other prospective studies using inspiratory muscle training (IMT) and analyzing its effects on redistribution of pulmonary ventilation in these patients.

In conclusion, in CHF patients with cardiomegaly, asymmetric expansion of the lower rib cage compartment is related to dyspnea and cardiac impairment. This suggests that significant interplay exists between cardiac and respiratory function, up to perceived effort sensation levels.

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Agostoni, P., Cattadori, G., Guazzi, M., Palermo, P., Bussotti, M., Marenzi, G., 2000. Cardiomegaly as a possible cause of lung dysfunction in patients with heart failure. Am. Heart J. 140, 24.

- Aliverti, A., Cala, S.J., Duranti, R., Ferrigno, G., Kenyon, C.M., Pedotti, A., Scano, G., Sliwinski, P., Macklem, P.T., Yan, S., 1997. Human respiratory muscle actions and control during exercise. J. Appl. Physiol. 83, 1256–1269.
- Aliverti, A., Pedotti, A., 2003. Opto-electronic plethysmography. Monaldi Arch. Chest Dis. 59, 12–16.
- American Thoracic Society, 2002. ATS statement: guidelines for sixminute walk test. Am. J. Respir. Crit. Care Med. 166, 111–117.
- Chiappa, G.R., Roseguini, B.T., Vieira, P.J., Alves, C.N., Tavares, A., Winkelmann, E.R., Ferlin, E.L., Stein, R., Ribeiro, J.P., 2008. Inspiratory muscle training improves blood flow to resting and exercising limbs in patients with chronic heart failure. J. Am. Coll. Cardiol. 51, 1663–1671.
- Clark, A.L., Chua, T.P., Coats, A.J.S., 1995. Anatomical dead space, ventilatory pattern and exercise capacity in chronic heart failure. Br. Heart J. 74, 377–380.
- Coats, A.J.S., 1996. The "muscles hypothesis" of chronic heart failure. J. Mol. Cell. Cardiol. 28, 2255–2262.
- Daganou, M., Dimopoulou, I., Alivizatos, P.A., Tzelepis, G.E., 1999. Pulmonary function and respiratory muscle strength in chronic heart failure: comparison between ischaemic and idiopathic dilated cardiomyopathy. Heart 81, 618–620.
- Dall'Ago, P., Chiappa, G.R., Guths, H., Stein, R., Ribeiro, J.P., 2006. Inspiratory muscle training in patients with heart failure and inspiratory muscle weakness. J. Am. Coll. Cardiol. 47, 757–763.
- De Andrade, A.D., Silva, T.N., Vasconcelos, H., Marcelino, M., Rodrigues-Machado, M.G., Filho, V.C., Moraes, N.H., Marinho, P.E., Amorim, C.F., 2005. Inspiratory muscular activation during threshold[®] therapy in elderly healthy and patients with COPD. J. Electromyogr. Kinesiol. 15, 631–639.
- Drexler, H., Riede, U., Munzel, T., Konig, H., Funke, E., Justet, H., 1992. Alterations of skeletal muscle in chronic heart failure. Circulation 85, 1751–1759.
- Hostettler, S., Illi, S.K., Mohler, E., Aliverti, A., Spengler, C.M., 2011. Chest wall volume changes during inspiratory loaded breathing. Respir. Physiol. Neurobiol. 175, 130–139.
- Jefferies, J.L., Towbin, J.A., 2010. Dilated cardiomyopathy. Lancet 375, 752–762.

- Johnson, B.D., Beck, K.C., Olson, L.J., O'Malley, K.A., Allison, T.G., Squires, R.W., Gau, G.T., 2000. Ventilatory constraints during exercise in patients with chronic heart failure. Chest 117, 321–332.
- Laoutaris, I., Dritsas, A., Brown, M.D., Manginas, A., Alivizatos, P.A., Cokkinos, D.V., 2004. Inspiratory muscle training using an incremental endurance test alleviates dyspnea and improves functional status in patients with chronic heart failure. Eur. J. Cardiovasc. Prev. Rehabil. 11, 489–496.
- Mancini, D.M., Henson, D., LaManca, J., Levine, S., 1994. Evidence of reduced respiratory muscle endurance in patients with heart failure. JACC 24, 972–981.
- Meyer, F.J., Borst, M.M., Zugck, C., Kirschke, D., Kubler, W., Haass, M., 2001. Respiratory muscle dysfunction in congestive heart failure: clinical correlation and prognostic significance. Circulation 103, 2153–2158.
- Mitch, W.E., Goldberg, A.L., 1996. Mechanisms of muscle wasting. The role of the ubiquitin-proteasome pathway. N. Engl. J. Med. 335, 1897–1905.
- Neder, J.A., Andreoni, S., Lerario, M.C., 1999. Reference values for lung function tests. II. Maximal respiratory pressures and voluntary ventilation. Braz. Med. Biol. Res. 32, 719–727.
- Olson, T.P., Beck, K.C., Johnson, B.D., 2007. Pulmonary function changes associated with cardiomegaly in chronic heart failure. J. Card. Fail. 13, 100–107.
- Olson, T.P., Beck, K.C., Johnson, J.B., Johnson, B.D., 2006. Competition for intrathoracic space reduces lung capacity in patients with chronic heart failure: a radiographic study. Chest 130, 164–171.
- Olson, T.P., Johnson, B.D., 2011. Influence of cardiomegaly on disordered breathing during exercise in chronic heart failure. Eur. J. Heart Fail. 13, 311–318.
- Pina, I.L., 2003. Exercise and heart failure: a statement from the American Heart Association Committee on Exercise, Rehabilitation, and Prevention. Circulation 107, 1210–1224.
- Puri, S., Baker, B.L., Oakley, C.M., Hughes, J.M., Cleland, J.G., 1994. Increased alveolar/capillary membrane resistance to gas transfer in patients with chronic heart failure. Br. Heart J. 72, 140–144.
- Rutten, F.H., Cramer, M.J., Lammers, J.W., Grobbee, D.E., Hoes, A.W., 2006. Heart failure and chronic obstructive pulmonary disease: an ignored combination? Eur. J. Heart Fail. 8, 706–711, 1996; 335:1897–1905.
- Ulrik, C.S., Carlsen, J., Arendrup, H., Aldershvile, J., 1999. Pulmonary function in chronic heart failure: changes after heart transplantation. Scand. Cardiovasc. J. 33, 131–136.
- Witte, K.K.A., Clark, A.L., 2005. Cycle exercise causes a lower ventilatory response to exercise in chronic heart failure. Heart 91, 225–226.