

Evaluation of Respiratory Muscle Strength and Pulmonary Function in Heart Failure Patients

Luiz Alberto Forgiarini Junior, Angélica Rubleski, Douglas Garcia, Juliana Tieppo, Rafael Vercelino, Adriane Dal Bosco, Mariane Borba Monteiro, Alexandre Simões Dias

Centro Universitário Metodista - IPA, Irmandade Santa Casa de Misericórdia de Porto Alegre, Fundação Faculdade Federal de Ciências Médicas de Porto Alegre - Porto Alegre, RS - Brazil

Summary

Background: Heart failure (HF) is the inability of the heart to pump enough blood to supply the necessities of the body. Pulmonary function and respiratory muscles can be affected and typical symptoms presented by the patients include discomfort at a minimal exertion.

Objective: To verify pulmonary function and respiratory muscle strength in patients with class II and III HF as defined by the New York Heart Association (NYHA).

Methods: The study was descriptive and observational, and comprised 12 class II and III HF patients in follow-up at the out-patient. Pulmonary function assessments [Forced Expiratory Volume in the first second (FEV1) and Forced Vital Capacity (FVC)] were performed using microspirometry and respiratory muscle strength [Maximal Expiratory Pressure (MEPmax) and Maximal Inspiratory Pressure (MIPmax)] were evaluated using a pressure transducer (Globalmed™).

Results: Differences were found between the functional classes II and III in relation to pulmonary function: FEV1 (II: 91.17 ± 19.87 ; III: 68.17 ± 21.78); FVC (II: 68.17 ± 21.78 ; III: 73.67 ± 22.94); and respiratory muscle strength: MIPmax (II: 71.67 ± 40.70 ; III: 53.33 ± 29.27) and MEPmax (II: 98.83 ± 34.56 ; III: 58.33 ± 15.06). The class II were higher for all study parameters, only MEPmax revealed a statistically significant difference.

Conclusion: The pulmonary function and respiratory muscle strength are impaired in heart failure patients class III patients, particularly in relation to MEPmax. (Arq Bras Cardiol 2007; 89(1) : 32-36)

Key words: Heart failure; cardiac output, low; muscle strength, pulmonary function, respiratory insufficiency.

Introduction

Heart failure (HF) is the inability of the heart to pump enough blood to supply the necessities of the body. It is a result of reduced cardiac output or an accumulation of blood in the veins supplying the right and left atriums (upper and lower vena cava and pulmonary veins). In response to this situation, compensatory mechanisms are induced by the cardiovascular system, such as the increase of heart rates, end diastolic pressures and ventricular mass. However, if the disease continues to develop, it will lead to a loss of ventricular function¹.

Data from the Ministry of Health report that HF is the main cause of hospital admissions, with 450 thousand new cases each year². The main symptom presented by the patients is exertion fatigue which limits their ability to perform daily activities (DA).

Respiratory muscle function can be affected by heart

diseases when the patients present weakness and respiratory muscle deterioration³.

HF causes hemodynamic alterations mainly as a result of pressure and volume overloads in the alveolar capillary region, indicating the acute phase that can be reversed. Remodeling occurs in the pulmonary capillaries and tissue membrane, increasing the density of the cellular matrix and compromises endothelial permeability. The removal of the active capillary fluid hinders gas exchanges⁴.

Hammond et al⁵, demonstrated that cardiopathy patients have weak respiratory muscles, which reduces blood flow to these muscles and causes generalized muscle atrophy.

A specific respiratory muscle training program improves muscle strength, functional capacity and quality of life for HF patients with weak inhalation muscles⁶.

Therefore, the objective of this study was to evaluate pulmonary function and respiratory muscle strength in patients with class II and III HF, as well as compare pulmonary function with respiratory muscle strength.

Methods

The design of the present study was descriptive and

Mailing address: Alexandre Simões Dias •

Rua Hipólito da Costa, 583 - casa 18 - Morro Santa Teresa - 90840-110

- Porto Alegre, RS - Brazil

E-mail: simoesdias@terra.com.br

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observational. The study population consisted of individuals with class II and III HF in follow-up at the Cardiology Heart Failure Outpatient Clinic of the *Santa Clara* Polyclinic at the *Santa Casa de Misericórdia* Hospital Complex in Porto Alegre. The patients had been clinically stable for more than three months and were in follow-up with the hospital's medical team. Exclusion criteria included advanced pulmonary disease, pulmonary tumors, HF functional classes I and IV, alterations during data collection, such as severe dyspnea or oxygen saturation below 80%, inability to understand the instructions to perform the test and patient refusal to participate.

Data collection instruments included the Globalmed™ analog pressure transducer to evaluate respiratory muscle strength, the Sherwood Medical Respiradyne II Plus spirometer for pulmonary function; the evaluation form was prepared by the researchers.

All patients included in the study were given an informed consent form explaining the study procedures. Afterwards, the evaluation form was completed and the muscle strength and pulmonary function tests were performed.

After approval from the *Irmandade Santa Casa de Misericórdia* Ethics and Research Committee, the respiratory muscle data were measured with the patient in a sitting position, elbows flexed and hands firmly securing the mouthpiece close to the mouth. To evaluate Maximal Inspiratory Pressure (P_{Imax}), the individual was asked to exhale until only the residual volume (RV) remained, and after adequately positioning the mouthpiece, to perform forced inhalation. To evaluate Maximal Expiratory Pressure (PE_{max}) the individual began with total pulmonary capacity (TPC), the mouthpiece was adequately positioned, a nose clip was placed to avoid air loss and the patient was asked to perform forced exhalation^{7,8}. The pulmonary function test was performed three times with an average duration of six seconds each. All data was collected by the same evaluator and the procedures were thoroughly explained to the patients. All tests were conducted with the patients in a sitting position, hands firmly securing the equipment in their mouth and nose obstructed with appropriated sized nose clips. The patients were then asked to inhale to TPC followed by maximum forced exhalation. The process was repeated three times and the highest value was selected. Differences between the repetitions could not exceed 10%. Values greater than 80% of the Forced Vital Capacity (FVC) were considered normal. The Forced Expiratory Volume in the first Second (FEV₁) was based on the patient's height and age⁹.

Respiratory muscle strength and pulmonary function

of the heart failure patients were analyzed using the study variables. Comparisons were made between the patients in the same functional class and between classes II and III, using the Student's t-test and a significance level of 5% ($p < 0.05$). Correlations between the study variables were evaluated using Simple Linear Regression.

Results

The sample was comprised of twelve individuals with heart failure of which six were functional class II (two females and four males) and six were functional class III (two females and four males) in accordance with the classification by the New York Heart Association (2001). Ages ranged from 47 to 67 years. Body weight ranged from 37 to 87 kilograms. All patients were white and the primary diseases found were cardiomyopathy (34%), hypertension (25%), alcoholism (17%), valve diseases (8%), ischemia (8%) and ischemia with associated valve diseases (8%) (Table 1).

Differences were found between the functional classes II and III in relation to pulmonary function and respiratory muscle strength. A statistically significant difference was only found for PE_{max} in comparisons between classes II and III (Table 2).

In comparison with the class II patients, pulmonary function (Table 3) and respiratory muscle strength were more compromised in the class III patients; however, this difference was statistically significant ($p < 0.042$) only for PE_{max} (Graphic 1).

Discussion

Santoro et al¹⁰ reported that HF is a highly prevalent heart disease in our country and is usually defined as physiopathological state with abnormal cardiac function, where the heart is not able to pump sufficient blood for tissue metabolism. HF causes the ejection fraction to

Table 1 - Demographic data of the study population

	Class II	Class III	p
Age (years)	58.2±8.8	65.2±11.3	p=0.59
Weight (Kg)	63.1±12.9	64.1±18.4	p=0.45
Height (cm)	167.7±12.8	164.2±10.3	p=0.64
Gender	4M/2F	4M/2F	-

M – male; F – female.

Table 2 - Pulmonary Function and Respiratory Muscle Strength for functional classes II and III

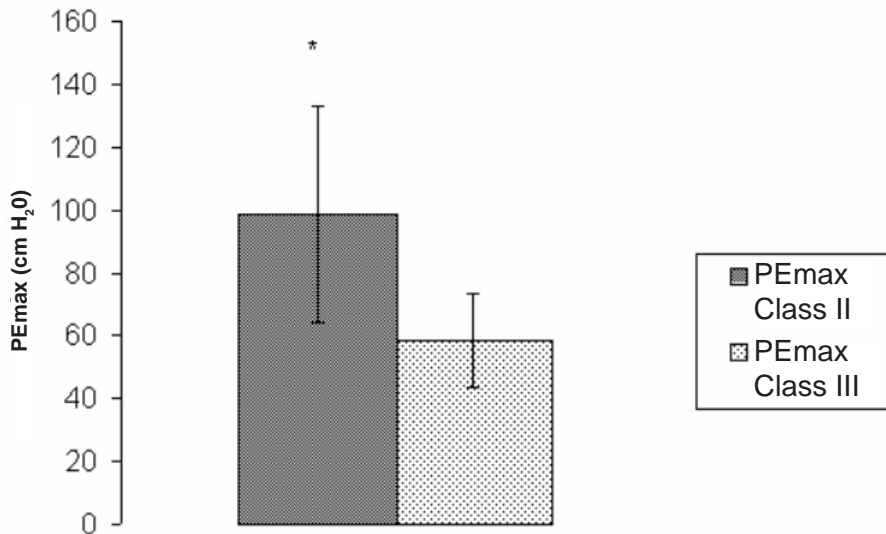
	FVC (%)	FEV ₁ (%)	P _{Imax} (cmH ₂ O)	PE _{max} (cmH ₂ O)
II	91.17 ± 19.87	87.00 ± 11.70	71.67 ± 40.70	98.83 ± 34.56*
III	68.17 ± 21.78	73.67 ± 22.94	53.33 ± 29.27	58.33 ± 15.06

* - $p = 0.042$ class II versus III.

Table 3 - Patient data for pulmonary function

Patient	Class II		Class III	
	FEV1 (%)	FVC (%)	FEV1 (%)	FVC (%)
1	75.1	48	40.2	75.1
2	69.5	51.4	70.9	69.5
3	95.1	55.6	68.8	95.1
4	86.9	68.2	73.7	86.9
5	96.7	101.2	87.3	96.7
6	98.6	89	102.6	98.6
Mean ± SD	87.00 ± 11.70	91.17 ± 19.87	73.67 ± 22.94	68.17 ± 21.78

Mean ± SD – mean ± standard deviation.



Graphic 1 - * - $p = 0.042$ Class III versus Class II.

diminish, contributing to an annual mortality rate of 30% to 50%¹¹.

The following signs and symptoms are commonly found in the advanced stage of heart failure and comprise the clinical manifestation of the disease: dyspnea, tachypnea, orthopnea, cold, pale and cyanotic extremities, weight gain, hepatomegaly, adventitious lung sounds (rales) and decreased tolerance for physical or daily activities¹².

Sleep apnea is a common finding, affecting close to 40% of the patients with severe conditions, marked by the symptoms of daytime drowsiness and fatigue, and if not diagnosed and treated in time could cause premature death¹³.

In the present study, analysis of the results revealed decreased pulmonary function and respiratory muscle strength in the class III patients in comparison to class II (Table 2) however a statistically significant difference was found only for PEmax (Graphic 1).

The patients with respiratory problems presented muscular

dysfunction, which contributes to exercise intolerance, dyspnea and hypercapnea. With adequate physical training, respiratory muscle function can be improved. With an effective respiratory muscle training program symptoms can be prevented or alleviated. Once the physiotherapists are aware of their respiratory, upper and lower limb muscle limitations they can develop an appropriate and effective training program for the patients¹⁴. This muscle training can be performed using respiratory exercises, aerobics or specific stimulants¹⁵.

The cardiovascular and pulmonary systems are essential for normal respiratory function, since they distribute atmospheric oxygen to the skeletal muscles. Therefore, cardiopulmonary system failure results in diminished pulmonary function and mainly, respiratory muscle strength; in the present study, PEmax was lower in the patients with more serious conditions¹⁶.

Altered respiratory muscle strength in HF patients

demonstrates that diminished pulmonary function is a result of reduced respiratory muscle strength. Hughes et al¹⁷ demonstrated that after respiratory muscle evaluation, P_Imax decreased 28% and P_Emax decreased 20%.

In a study conducted with NYHA (2001) class I, II, III and IV HF patients, significant pulmonary volume reductions were found, especially in FVC and FEV1¹⁸. Reductions in FVC and FEV1 were also found in the present study; however, no significant statistical difference was found.

Borst et al¹⁹, analyzed respiratory muscle dysfunction in HF patients and the class III patients presented compromised respiratory muscle strength (P_Imax and P_Emax) when compared to the class II patients. A greater difference in P_Imax in comparison to P_Emax was also observed in this study¹⁸. In the study of Borst et al¹⁹ was founded a greater difference in P_Emax (Table 2 and Graphic 1). Meyer et al²⁰ attributed the diminished respiratory muscle strength and pulmonary function to the reduced muscle mass. This fact could also be responsible for capillary density and oxidative enzyme activity²⁰. This could be the main factor responsible for diaphragm atrophy.

Evans et al²¹ found reduced P_Imax and P_Emax in HF patients and P_Imax was more accentuated. The study demonstrates the correlation between P_Imax and cardiac indexes, suggesting that muscle perfusion is involved in the etiology of the respiratory muscle pathologies²¹.

The inability to perform exercises or minimal exertion and the physiopathological process associated with HF considerably affect the skeletal muscles. The skeletal musculature of HF patients presents type I and II fibers with reduced diameters. Atrophy occurs in the type II fibers, and idiopathic myopathies and hypotrophy affect the type I fibers. However, the etiology of these primary (cardiac muscle or skeletal myopathies) or secondary alterations (related to HF) is still not clear. These alterations suggest that muscle fatigue in these patients could be related to the depletion of phosphocreatine to intracellular acidity, causing a reduction in muscle strength of up to 50%⁵.

Corra et al²² conducted a study with HF patients in order to evaluate pulmonary function and other variables. The sample consisted of 323 class II and III patients (NYHA), and the author's purpose was to understand the relationship between ventilation patterns and heart failure. The test consisted of evaluation on an exercise bicycle where the patients were submitted to maximum exertion. The study considered peak oxygen consumption and ventilation patterns. He observed that the test was interrupted for many of the patients that presented dyspnea which proved their diminished physical resistance. The study was conducted over a timeframe of eleven months and from the 323 individuals, 53 died as a result of a lower peak oxygen consumption (42% of the predicted value) and an abnormal ventilation pattern²². Therefore, it can be concluded that heart failure also affects other pulmonary system variables, as well as respiratory rhythm.

Johnson et al²³, also considering pulmonary function alterations during physical exertion, conducted a study with 11 patients (HF class II and III) who were submitted to a

treadmill test. Spirometry was conducted after the test, and in comparison to the control group, a reduction in FVC and FEV1 was observed in the patients. The patients attained an average of 76% of the predicted value for FVC and 78% of the predicted value for FEV1, whereas the control group presented values of 99% and 103% respectively²³.

Nanas et al⁸ were able to partially explain the relation between respiratory muscle performance and fatigue in HF patients. He studied 55 patients with functional classes I to III, and submitted them to a 15 minute treadmill test. Respiratory muscle strength using P_Imax and P_Emax, pulmonary function using spirometry and peak oxygen consumption (VO₂) were evaluated with the patients at rest and after the test (2, 5 and 10 minutes). None of the patients presented pulmonary function alterations, since all presented FVC and FEV1 greater than 80% of the predicted value and interestingly, 11 patients presented diminished P_Imax at rest after the test. Similar to the present study, the author observed a reduction in P_Imax and P_Emax, also at rest, in the patients that presented muscle weakness before the test. Mean P_Imax was 73% of the predicted value, whereas P_Emax was 53% of the predicted value. Observing the data released by the author, it can be said that the expiratory musculature was compromised at the end of the test, since the average values dropped to 49% of the predicted value while the P_Imax values dropped to 72% of the predicted value.

Knowing that HF provokes reduced respiratory musculature performance, mainly in relation to strength, Coirault et al²⁴ initiated treatment with angiotensin-converting enzyme (ACE) inhibitors in order to increase respiratory muscle strength. Eighteen patients were selected, with NYHA functional classes I, II and III and ACE inhibitors were administered to the patients for six months. Before the treatment, P_Imax and P_Emax were diminished by 57% and 62% of the predicted value, respectively²⁴. These values are close to those found in our study in relation to P_Emax.

In the present study, in comparison to the class II patients, all study parameters were reduced in the class III patients; however, only P_Emax was statistically significant. One limitation was the small study sample and therefore further studies with larger populations including variables to specify actual clinical conditions such as hypertension, diabetes mellitus, heart surgery, kidney failure and laboratory data are required.

Conclusion

Based on this study, it is possible to assume that pulmonary function and respiratory muscle strength are compromised in heart failure patients and that functional class III patients present significantly reduced P_Emax.

The research findings remind us of the importance of physiotherapeutic follow-up for HF patients, as specific respiratory muscle training could improve the symptoms and prognosis of these patients.

Potential Conflict of Interest

No potential conflict of interest relevant to this article was reported.

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Study Association with Graduate Work

This study is not associated with any graduation program.

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