Non-invasive assessment of inspiratory muscle performance during exercise in patients with chronic heart failure

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Aims The aim of this study was to assess inspiratory performance at rest and during exercise in patients with chronic heart failure in comparison with healthy controls using a non-invasive index: the tension-time index of inspiratory muscles (TTMUS).

Methods We studied 13 patients with chronic heart failure (57 ± 7 years) and 10 control subjects (58 ± 6 years) at rest and during an incremental maximal exercise test. Measurements included breathing pattern (inspiratory time, total time of respiratory cycle, minute ventilation, tidal volume and respiratory frequency), mouth occlusion pressure and mean inspiratory pressure (calculated as follows: 5 × mouth occlusion pressure × inspiratory time). The maximal inspiratory pressure was measured at rest. TTMUS was calculated from the equation: 

\[ TTMUS = \frac{P_I}{P_{IMAX}} \times \frac{T_I}{TTOT} \]

where \( P_I \) is the ratio of mean inspiratory pressure to maximal inspiratory pressure and \( T_I/TTOT \) is the ratio of mean inspiratory time to total time of the respiratory cycle.

Results At rest, the results in patients showed non-significantly higher mouth occlusion pressure, lower maximal inspiratory pressure (\( P < 0.001 \)), and a higher ratio of mean inspiratory pressure to maximal inspiratory pressure (\( P < 0.01 \)). There was no difference in the breathing pattern. TTMUS was thus significantly higher in the patients with chronic heart failure (\( P < 0.001 \)). At maximal exercise (77 ± 16 W for patients with chronic heart failure vs 142 ± 27 W for controls, \( P < 0.001 \)), the ratio of mean inspiratory time to total time of respiratory cycle, the mouth occlusion pressure and the ratio of mean inspiratory pressure to maximal inspiratory pressure were not different. TTMUS was thus comparable in the two groups. During exercise, at comparable workloads (20, 40 and 60 W), the patients showed higher mouth occlusion pressure (\( P < 0.01 \)) and a higher ratio of mean inspiratory pressure to maximal inspiratory pressure (\( P < 0.001 \)), whereas the ratio of mean inspiratory time to total time of the respiratory cycle was similar. TTMUS was thus higher in the patients at each workload (\( P < 0.05 \)).

Conclusion This study shows that the determination of TTMUS at rest and during exercise allows the observation of alterations in inspiratory muscle performance as a result of both reduced inspiratory strength, as measured by the maximal inspiratory pressure, and increased ventilatory drive, as reflected by the mouth occlusion pressure in patients with chronic heart failure. The non-invasiveness of this new index is an additional argument for its use in a clinical setting.

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Key Words: Chronic heart failure, inspiratory muscles, exercise, tension-time index.

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Introduction

The diminished exercise tolerance of patients with chronic heart failure has been extensively studied. The underlying abnormalities involve not only the heart, but also the pulmonary[1] and skeletal muscle systems[2,3]. Abnormalities in respiratory muscle function have been
Inspiratory muscle performance in chronic heart failure


Methods

Subjects
This study was carried out with 13 patients with chronic heart failure (12 males and one female) and 10 control subjects (nine males and one female). Each subject was informed of the purpose of the study and gave his/her consent. The patients with chronic heart failure (57 ± 7 years) had a history of daily exertional dyspnoea associated with mild to moderate stable chronic heart failure (classes II and III in the New York Heart Association classification) and left ventricular dysfunction, as assessed by the isotopic ejection fraction at rest <45% (32.9 ± 10%). This dysfunction was due to ischaemic or idiopathic dilated cardiomyopathy. All patients were in a clinically stable condition with no worsening of heart failure or change in cardiac medication in the previous 2 months. Peripheral oedema, ascites and pulmonary crackles were not present at the time of testing. None of the patients had a history of pulmonary disease, myocardial infarction or unstable angina (during the previous 2 months), myocardial ischaemia or arrhythmias during exercise, or exercise intolerance for any reason other than fatigue of dyspnæus. None had a history of smoking (past 5 years), amiodarone, beta-blocker or oral steroid treatment or had ever had an implanted pacemaker.

The control group consisted of 10 sedentary normal subjects (58 ± 6 years) with no history of cardiovascular or pulmonary disease or smoking.

Spirographic measurements

Conventional spirographic measurements were performed on a spirometer (Pulmonet III, Sensormedics, Bilthoven, the Netherlands). The forced expiratory volume in one second and forced vital capacity were measured. Tiffeneau's ratio (ratio of forced expiratory volume in one second to forced vital capacity) was then calculated. The predicted values were those proposed by the European Community of Steel and Coal[13].

Exercise testing

The exercise test was performed on a bicycle ergometer (Ergometer 990, Bodyguard Jones AS, Sandnes, Norway). During the test, the subjects wore a noseclip and breathed through a low resistance (0.9 cm H2O l s−1), low dead space (50 ml) breathing valve (Warren E. Collins Inc., M.A., U.S.A.) with large calibre tubing (3.5 cm). The valve was connected by the expiratory circuit to a breath-by-breath automated exercise metabolic system (CPX, Medical Graphics Corp., M.N., U.S.A.). Expired gases were analysed for oxygen with a zirconia solid electrolyte analyser, and for carbon dioxide with an infrared analyser. The CPX continuously measured oxygen uptake, carbon dioxide output, and respiratory exchange ratio. Before each test, the gas analysers were calibrated with two gas mixtures of known oxygen and carbon dioxide concentration. The data were averaged during the last 20 s of each load over an integral number of breaths. Inspiratory airflow was measured with a Fleish no 3 pneumotachograph (Fleish, Lausanne, Switzerland) placed on the inspiratory line, and a differential pressure transducer with a measuring range of ±2 cmH2O (model M-45, Validyne, CA, U.S.A.). Tidal volume was obtained by integration of the flow signal. Heart rate was continuously recorded on a cardioscope (Personal 120, Esaote-Biomedica, Florence, Italy) and an electrocardiogram was periodically recorded.

Maximal inspiratory and expiratory pressures and mouth occlusion pressure

At rest, PIMAX and maximal expiratory pressure (PEMAX) at the mouth were measured at functional residual capacity with the Validyne M-45 transducer (±300 cmH2O) and a model CD 15 carrier transducer (12.5 cmH2O) using a differential pressure transducer with a measuring range of ±300 cmH2O. The mouth occlusion pressure measurement was calculated from the difference between PIMAX and PIMIN.

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demodulator, using the technique of Black and Hyatt\[14\].

The subjects were asked to make maximal inspiratory or expiratory effort against an occluded airway and to maintain maximal pressure for at least 1 s. Repeated measurements were made until three technically satisfactory and reproducible measurements were obtained (variation in PIMAX and PEMAX <10%). The repeated data represents the best values.

At rest and during exercise, P 0·1 measurement was performed with a silent electromagnetic valve that was closed during expiration and automatically opened about 150 ms after the onset of the occluded inspiration. Since closure was silent, the subjects were unable to anticipate which breath was going to be occluded. P 0·1 was measured with a Validyne MP-45 transducer (± 35 cmH$_2$O) and a model CD 15 carrier demodulator. P 0·1 is the pressure developed at the mouth 0·1 s after the beginning of inspiration against an occluded airway and it provides an indirect assessment of neural drive to the respiratory muscles\[15\].

**Derived parameters**

All signals were displayed on a Gould ES 1000 recorder (Gould Instruments, OH, U.S.A.). At rest and during exercise, the breathing pattern was determined from an average of 10 respiratory cycles: tidal volume, inspiratory time (TI), and total time of the respiratory cycle (TTOT) were measured. We then calculated respiratory frequency; minute ventilation; the ratio of mean inspiratory time to total time of the respiratory cycle (TI/ TTOT), which represents the time fraction during which the inspiratory muscles are in motion (index of the respiratory rhythm); and mean inspiratory flow (ratio of tidal volume to inspiratory time), which corresponds to an intensity index of the inspiratory activity (index of neural drive)\[\].

We calculated TTMUS from the equation:

\[ TTMUS = P_l / PIMAX \times TI / TTOT, \]

where $P_l$ is the mean inspiratory pressure developed by the respiratory muscles during inspiration. $P_l$ was estimated as follows:

\[ P_l = 5 \times P 0·1 \times T_l^{12,16}, \]

where P 0·1 was measured from signals displayed at a paper speed of 100 mm . s$^{-1}$ over an average of three to five measurements. For the TTMUS equation, $P_l$ and TI/TTOT were determined at each workload and PIMAX was measured at rest.

**Protocol**

The entire experiment was performed in the afternoon in all subjects. The subjects first underwent spirometric measurements at rest. They were then seated and, after a period of familiarization with the experimental equipment (mouthpiece and noseclips), ventilatory and pressure parameters were recorded for 5 min. At least 10 occlusions were performed for each subject, at the rate of 2-3 per minute. After 5 min of rest, PIMAX and PEMAX were measured.

The subjects then performed an incremental exercise test on a calibrated cycle ergometer in the sitting position. Resting measurements were obtained over 5 min at rest on the cycle. This was followed by a 3 min 20 W warm-up period. The workload was then increased by 10 W every 90 s for the patients with chronic heart failure and by 20 W every 90 s for the control subjects, until exhaustion. All subjects were vigorously encouraged to perform maximal exercise until they felt unable to continue or they reached their maximal oxygen uptake. For each workload, a period of 90 s was needed to obtain all respiratory gas exchange measurements, and breathing pattern and P 0·1. Maximal symptom-limited oxygen uptake was determined for patients with chronic heart failure. All patients stopped exercise because of fatigue or dyspnoea, or both. For the control subjects, at least three of the following four criteria had to be observed to demonstrate that they had reached their maximal oxygen uptake: (1) stability of heart rate at a value close to the theoretical maximal heart rate; (2) stability of oxygen uptake despite the increase in workload; (3) respiratory ratio >1·10; and (4) the inability of the subject to maintain a pedalling rate of 50 rpm.

**Statistics**

The values are reported as mean ± standard deviation (SD). The data at rest and maximal exercise were compared between the two groups using an unpaired Student’s t-test or the Kruskall-Wallis test when variables were non-parametric. Exercise data were compared at the same workload (20, 40, 60 W) using a two-way analysis of variance (ANOVA) for repeated measures. When the distribution of variables was not normal, we transformed them by using the Box’s Cox transformation ($(x^\lambda - 1)/\lambda$), with $\lambda$ varying. We kept the value of $\lambda=0·001$, which gave the greater P for the Shapiro-Wilk’s test. After variables were normalized, we tested for a group effect and an intensity effect using a two-way ANOVA for repeated measures: a fixed factor (group) and a repeated factor (intensity). In the presence of an intensity effect and an interaction, we compared means using a Student’s t-test (for small samples) while penalizing the P value by way of Bonferroni’s method. The comparison of ordinal quantitative or non-normalized quantitative variables between the two independent groups was done using the Kruskall-Wallis test. A P value <0·05 was considered statistically significant.

The data processing was performed using the 3100 computer of IURC (Institut Universitaire de Recherche Clinique, Montpellier, France) and BM DP
Table 1 Anthropometric, spirometric and respiratory muscle performance parameters at rest in patients with chronic heart failure and control subjects. Values are reported as mean ± SD

<table>
<thead>
<tr>
<th></th>
<th>CHF</th>
<th>Controls</th>
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<tbody>
<tr>
<td>Subjects</td>
<td>n=13</td>
<td>n=10</td>
</tr>
<tr>
<td>Age (years)</td>
<td>57 ± 7</td>
<td>58 ± 6</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>170 ± 8</td>
<td>171 ± 6</td>
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<tr>
<td>Weight (kg)</td>
<td>77 ± 12</td>
<td>74 ± 10</td>
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<tr>
<td>FEV₁ (l)</td>
<td>2.7 ± 0.8</td>
<td>3.6 ± 0.6*</td>
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<tr>
<td>FEV₁ (% pred)</td>
<td>83 ± 23</td>
<td>112 ± 16**</td>
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<tr>
<td>FVC (l)</td>
<td>3.6 ± 0.7</td>
<td>4.5 ± 0.8*</td>
</tr>
<tr>
<td>FVC (% pred)</td>
<td>90 ± 16</td>
<td>111 ± 15**</td>
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<tr>
<td>FEV₁/FVC (%)</td>
<td>73 ± 12</td>
<td>79 ± 6</td>
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<tr>
<td>P 0·1 (cmH₂O)</td>
<td>1.9 ± 0.8</td>
<td>1.5 ± 0.5</td>
</tr>
<tr>
<td>PIMAX (cmH₂O)</td>
<td>69 ± 20</td>
<td>104 ± 17***</td>
</tr>
<tr>
<td>PI/PIMAX (%)</td>
<td>0.20 ± 0.07</td>
<td>0.10 ± 0.03**</td>
</tr>
<tr>
<td>TTMUS</td>
<td>0.08 ± 0.03</td>
<td>0.04 ± 0.01***</td>
</tr>
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CHF = chronic heart failure; FEV₁ = forced expiratory volume in one second; FVC = forced vital capacity; % pred = percentage of predicted value; P 0·1 = mouth occlusion pressure; PI/PIMAX = mean inspiratory pressure to maximum inspiratory pressure.

* = P < 0·05; ** = P < 0·01; *** = P < 0·001.

statistical software (Cork Technology Park, Model Farm Road, Cork, Ireland).

Results

Anthropometric and spirometric data

The anthropometric and spirometric data for the patients with chronic heart failure and the control subjects are listed in Table 1. There was no statistical difference between the two groups in age, height and body weight. Both forced expiratory volume in one second and forced vital capacity were significantly reduced in patients with chronic heart failure compared to the control subjects, but Tiffeneau’s ratio was not significantly different.

Gas exchange and breathing pattern parameters

At rest, no significant difference was found for minute ventilation, tidal volume, respiratory frequency, mean inspiratory flow, T₁/TTOT (0·43 ± 0·04 in chronic heart failure vs 0·41 ± 0·06 in controls), oxygen uptake and carbon dioxide output between patients with chronic heart failure and control subjects. At maximal exercise, T₁/TTOT and respiratory frequency were not significant difference between the two groups. In contrast, maximal workload (77 ± 16 W in chronic heart failure vs 142 ± 27 W in controls; P < 0·001), oxygen uptake (P < 0·001), carbon dioxide output (P < 0·001), tidal volume (P < 0·01), minute ventilation (P < 0·01), and mean inspiratory flow (P < 0·05) were significantly lower in the patients. The results are summarized in Table 2.

During exercise, the selected workloads (20, 40 and 60 W) were the common workloads reached by the two groups; at comparable and maximal workload, T₁/TTOT as comparable between the two groups (Fig. 1).

Respiratory muscles parameters

At rest, PEMAX was not significantly different between the two groups (90 ± 48 cmH₂O in chronic heart failure vs 109 ± 26 cmH₂O in controls), whereas PIMAX was significantly lower in the patients (69 ± 20 cmH₂O in chronic heart failure vs 104 ± 17 cmH₂O in controls, P < 0·001). At rest, P 0·1 was not significantly higher in the patients, whereas PI/PIMAX (P < 0·01) and TTMUS (P < 0·001) were both significantly higher (Table 1).

During exercise, at comparable workload, the pressure parameters (P 0·1, Fig. 2; PI/PIMAX, Fig. 3) and TTMUS (Fig. 4) were significantly higher in patients with chronic heart failure compared with the control subjects, but at maximal exercise, this difference was not statistically different.

Discussion

This study demonstrated that, at rest and at comparable workloads during exercise, TTMUS, a non-invasive index assessing the performance of all the inspiratory muscles, was significantly higher in the patients with chronic heart failure compared with control subjects, indicating lower inspiratory muscle performance at the same workload in the patients with chronic heart failure. In contrast, at maximal exercise, there was no difference in TTMUS between the two groups.
Methodology

For exercise testing, we used 90 s periods instead of the usual 60 s, as this longer duration was needed to obtain all gas exchange, breathing pattern and occlusion pressure measurements. Despite this longer duration, we obtained maximal symptom-limited oxygen uptake measurements in our patients that were comparable to those obtained in patients with chronic heart failure with similar disease severity [18]. Similarly, we were certain that the control subjects reached maximal oxygen uptake, as all fulfilled three of the four
standard criteria. Moreover, in the two groups, the mean exercise test duration was between 8 and 12 min, which is the optimal range for obtaining maximal oxygen uptake[19].

We assessed inspiratory performance by determining TTMUS, which was estimated from $P_{0.1}$. TTMUS takes into account the mean pressure developed by the muscles during inspiration in relation to their maximal capacity ($P_{I}/P_{IMAX}$), and the duration of contraction in relation to the total duration of the respiratory cycle ($T_{I}/TTOT$). Thus, the equation was: $TTMUS = P_{I}/P_{IMAX} \times T_{I}/TTOT[12]$, where $P_{I}$ is estimated as follows: $P_{I} = 5 \times P_{0.1} \times T_{I}$. This last equation assumes that inspiratory pressure linearly increases with time during inspiration. Since this linear relationship is not always observed in humans[15,20], this method leads to an over-estimation of transpulmonary pressure, as reported by Ramonatxo et al[12]. However, these authors showed the usefulness of $P_{0.1}$ to estimate $P_{I}$ at rest in normal subjects and chronic obstructive pulmonary disease patients[12], and during exercise in normal subjects with resistive loads, in order to put the entire inspiratory musculature in reference to its fatigue threshold[17].

Figure 3  Ratio of mean inspiratory pressure to maximal inspiratory pressure ($P_{I}/P_{IMAX}$) at comparable and maximal workloads in patients with chronic heart failure (n=13) and control subjects (n=10). The circles represent the comparable workloads reached by the two groups: $\circ$ = chronic heart failure patients at 20, 40 and 60 W and $\bullet$ = control subjects at 20, 40, 60, 80, 100 and 120 W. The triangles represent the mean of the maximal workload for chronic heart failure patients ($\triangle$) and for control subjects ($\Delta$). For this point, the SD on the horizontal axis represents the inter-subject variability of the maximal workload. $*=P<0.05$, $**=P<0.01$, $***=P<0.001$, NS = not significant at maximal workload between the two groups. The values are represented by mean $\pm$ SD.

Figure 4  Tension-time index of the inspiratory muscles (TTMUS) at comparable and maximal workloads in patients with chronic heart failure (n=13) and control subjects (n=10). The circles represent the comparable workloads reached by the two groups: $\circ$ = chronic heart failure patients at 20, 40 and 60 W and $\bullet$ = control subjects at 20, 40, 60, 80, 100 and 120 W. The triangles represent the mean of the maximal workload for chronic heart failure patients ($\triangle$) and for control subjects ($\Delta$). For this point, the SD on the horizontal axis represents the inter-subject variability of the maximal workload. $*=P<0.05$, $**=P<0.01$, NS = not significant at maximal workload between the two groups. The values are represented by mean $\pm$ SD.

Spirometric parameters and breathing pattern

The spirometric results obtained in the patients with chronic heart failure agreed with those usually reported. Indeed, there was a significant decrease in the forced expiratory volume in one second and in the forced vital capacity in the patients compared with the control subjects, and a similar Tiffeneau’s ratio, indicating a trend towards a restrictive handicap which could be partly attributed to pulmonary congestion and oedema[1,6,21–23].

At rest, the breathing pattern (tidal volume, respiratory frequency, minute ventilation, mean inspiratory flow) observed in the patients compared with controls was in agreement with other reports[6,8,24].

At maximal exercise, the patients had a respiratory frequency similar to that of the controls, but a significantly lower tidal volume associated with a significantly lower mean inspiratory flow. These results confirm an abnormal breathing pattern that may be linked to limitations in pulmonary expansion in patients with chronic heart failure[1,21–26].

Respiratory muscle parameters

At rest, the patients with chronic heart failure demonstrated a tendency towards a decrease in P E M A X and a significant decrease in P I M A X compared with control subjects, in agreement with previous studies[4,6,7,27]. These results suggest a respiratory muscle weakness that may contribute to the dyspnoea experienced during daily activities, as suggested by M cParland et al.[5].

At maximal exercise (77 ± 16 W in chronic heart failure vs 142 ± 27 W in controls), respiratory muscle performance was decreased in both groups, with a high T T M U S (0.35 ± 0.18 in chronic heart failure vs 0.30 ± 0.11 in controls). These values of T T M U S appeared close to the respiratory muscle fatigue threshold (T T M U S=0.33) described at rest in healthy and chronic obstructive pulmonary disease patients[12]. M ancini et al.[9] also showed a dramatically increased tension-time index of the diaphragm, approaching the level of fatigue in patients with chronic heart failure but not in control subjects. This difference between the two indexes may be explained by the fact that the tension-time index of the diaphragm investigates only diaphragmatic work[9], whereas T T M U S assesses all the inspiratory muscles[12]. It therefore suggests that patients with chronic heart failure recruit different respiratory muscles compared with normal subjects: they may activate the diaphragm more extensively than other inspiratory muscles[8,28].

At rest and at comparable workload during exercise, T T M U S was higher in patients with chronic heart failure compared with control subjects, which indicates lower inspiratory muscle performance. These results were in agreement with those reported by M ancini et al.[9], who, using the tension-time index of the diaphragm, also demonstrated dramatically increased diaphragmatic work in patients with chronic heart failure. In our patients, the submaximal T T M U S was higher than in the controls because these patients showed a more elevated P 0-1, which resulted in higher P I. Thus, the increase in T T M U S was mainly due to an increase in the pressure parameters (P I/P I M A X) that represent inspiratory muscle load vs capacity, whereas T I/T T T O T was similar between the two groups.

P 0-1 is an index that reflects both neural drive to the inspiratory muscles and their resulting force output[15,20]. A t rest, we found a trend towards higher P 0-1 in patients with chronic heart failure, in agreement with Ambrosino et al.[6]. However, during exercise, we showed that P 0-1 was significantly greater in the patients, indicating an increased neural drive to the inspiratory muscles, which could be associated with both exercise hyperventilation and respiratory muscle weakness (decreased P I M A X)[6].

The mechanisms responsible for the decrease in inspiratory muscle performance observed at comparable workloads during exercise may be related to the under-perfusion due to the reduced cardiac output of patients with chronic heart failure. This reduced output has been demonstrated to limit the increase in blood flow in the limb muscles[29]. This hypothesis of an association between decreased performance and an under-perfusion was supported indirectly by M ancini et al.[6,30], who demonstrated accessory respiratory muscle deoxygenation during exercise in patients with chronic heart failure in comparison with control subjects, as assessed by near-infrared spectroscopy. This finding suggests an under-perfusion of these muscles in these patients. Nevertheless other mechanisms, such as metabolic and histological abnormalities may also be involved in this decreased performance[30,31].

Conclusion

T T M U S is an index that indicates a decrease in inspiratory muscle performance in patients with chronic heart failure at rest and particularly during exercise. This non-invasive and easily determined index may be useful in our understanding of the contribution of decreased inspiratory muscle performance to exercise intolerance in these patients, particularly in terms of their exercise dyspnoea. It could also provide a means of assessing the benefits of respiratory muscle training in chronic heart failure patients, which was recently proposed by M ancini et al.[32].

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