Orthopnea and inspiratory effort in chronic heart failure patients

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Abstract Study objective: Orthopnea is a typical feature of patients with chronic heart failure (CHF), the factors contributing to it are not completely understood. We investigated changes in dyspnea and other respiratory variables, induced by altering posture (from sitting to supine) in 11 CHF patients (NYHA classes II–IV) and 10 control subjects.

Methods and Results: We measured dyspnea (Borg scale), the diaphragm pressure time product per minute (PTPdi/m, index of metabolic consumption), and mechanical properties of the lung (lung compliance (C_L) and resistances (R_L)). CHF patients also underwent a trial of non-invasive mechanical ventilation (NIMV) in the supine position in order to ascertain whether unloading the inspiratory muscles could somehow relieve dyspnea. While sitting the PTPdi/min was significantly higher in CHF patients than in controls (181 ± 54 cm H_2O × s/min vs. 96 ± 32; P < 0.05). Assuming a supine position caused no major changes in controls, whereas CHF patients showed a significant worsening in dyspnea, a rise in PTPdi/min (243 ± 97 p < 0.01) and R_L (4.7 ± 1.2 cm H_2O/L × s sitting vs. 7.9 ± 2.5 supine; P < 0.01) and a decrease in C_L (0.08 ± 0.02 L/cm H_2O sitting vs. 0.07 ± 0.01 supine; P < 0.05). Applying NIMV to supine CHF patients significantly reduced the PTPdi/min into 81 ± 42 (P < 0.001). Changes in dyspnea, produced by varying position or applying NIMV, were significantly correlated with PTPdi/min (r=0.80, P < 0.005 and r=0.58, P < 0.01, respectively).

Conclusions: CHF patients had a higher PTPdi/min than controls when sitting, and assuming a supine position induced severe dyspnea, a large rise in R_L, and a reduction in C_L so that PTPdi/min increased further. Orthopnea was strongly correlated with the increased diaphragmatic effort.

Keywords CHF; dyspnea; diaphragm; inspiratory muscle; non-invasive ventilation.

INTRODUCTION

Orthopnea is a common feature in patients with chronic heart failure (CHF). Surprisingly, however, little is known about the mechanisms underlying this symptom. A recent study has shown that airflow resistance, as assessed by the oscillation technique, increases significantly in CHF patients, but not in normal subjects, when they assume a supine position (1). In another study, performed on patients with acute heart failure, Duguet et al. (2) demonstrated a relationship between expiratory flow limitation and orthopnea, exaggerated by changing posture from sitting to supine. Whether or not this mechanism is also responsible for orthopnea in stable CHF is not known. Indeed, in CHF patients, increased blood venous return might also reduce lung compliance by increasing pulmonary hydrostatic pressure and causing interstitial edema (3–5). Both increased airflow resistance and decreased lung compliance may lead to greater energy expenditure by the diaphragm and other respiratory muscles, an event which has been shown to be linked to the genesis of dyspnea (6). Indeed, regardless of the specific cause, a sudden increase in respiratory impedance provoked by lying down should produce, together with an abrupt rise in inspiratory effort, the onset of orthopnea (2). In order to evaluate this hypothesis we measured lung mechanics, inspiratory muscle effort, and sensation of dyspnea in the sitting and supine position in II stable CHF patients and compared these results with those obtained in a control group of normal subjects. Furthermore, we applied inspiratory pressure support plus continuous positive airway pressure to the supine CHF patients to verify whether unloading the inspiratory muscles was somehow associated with a relief of dyspnea.
MATERIAL AND METHODS

Subjects

Eleven in-patients with stable CHF with no history of neurological or chronic respiratory disease were recruited. Two patients were in New York Heart Association functional class II, seven in class III and two in class IV. All of them were receiving diuretics and ACE-inhibitor drugs, which were continued on the day of the study. They were in a phase of clinical stability and were admitted to the hospital for a medical control and eventually for a short period of rehabilitation. Patients with X-ray evidence of pulmonary edema or congestion were excluded from the study. Data from a control group of 10 matched subjects were used for comparison. Spirometric and hemodynamic data on patients and controls are shown in Table 1. Written consent was obtained from all subjects, and the protocol was approved by the Fondazione Maugeri Ethics Committee.

Measurements

Static and dynamic lung volumes were assessed by body plethysmography (MasterLab-Jaeger, Hochberg, Germany).

Flow was measured with a pneumotachograph (Screenmate Box 0586, Jaeger GmbH, Hochberg, Germany), connected to a rigid mouthpiece.

Airway pressure was measured through tubing inserted in the mouthpiece and connected to a differential pressure transducer (Honeywell 300 cm H2O, Freeport, IL, U.S.A.). VT was obtained by integration of the flow signal. The inspiratory (TI), expiratory (TE) and total breathing cycle (T Tot) duration, respiratory frequency (RR), and duty cycle (TI/T Tot) were calculated as average values of 10 consecutive breaths, after 5 min of breathing.

Baseline from peak changes in esophageal (Pes), gastric (Pga), and transdiaphragmatic (Pdi) pressures were measured using the balloon-catheter technique (7). Pressure at the airway opening (Paw) was measured via a side port.

Respiratory mechanics were assessed using Mead and Wittenberger’s technique (8). Inspiratory pulmonary resistance (Ri) and elastance (Ei) were calculated by fitting the equation of motion of a single-compartment model using multilinear regression.

Dynamic PEEPi (PEEPi,dyn) was measured according to Appendini et al. (9). The pressure time integrals of the diaphragm and the other inspiratory muscles were calculated per breath (PTPdi/b and PTPes/b, respectively) and per minute (PTPdi/min and PTPes/min) (10).

Relative changes in lung volumes were recorded by means of inductive plethysmography. A rib cage band was placed at the level of the axilla and an abdominal one was positioned just above the umbilicus. Calibration was performed with the isovolume maneuver (II).

Subjective ratings of dyspnea were made during the various trials using Borg’s scale.

Arterial blood gases (BG) were measured in samples taken from the radial artery (ABL 550 Radiometer, Copenhagen, Denmark).

The above-mentioned variables, excluding arterial blood gases and body plethysmography, were recorded in the CHF patients and control subjects both in sitting and supine positions after 15 min of spontaneous breathing, unless there was the onset of severe (Borg scale > 6) dyspnea, so that the measurements were recorded once reached that dyspnea value. At the end of this part of the study, the CHF patients were administered non-invasive mechanical ventilation (NIMV) through a nose mask for 20 min while they were in a supine position. The following pressures were used: 10 cm H2O of inspiratory aid and 5 cm H2O of expiratory-positive airway pressure. ECG, SaO2 and end-tidal CO2 (ETCO2) were monitored continuously.

All signals were collected using a personal computer equipped with an A/D board, and stored at a sampling rate of 100 Hz. The mean value of each physiological variable during the 5 min of recording was used for analyses. Results are presented as mean±standard deviation (SD). Differences between physiological parameters recorded in sitting and supine positions were analysed by paired Student’s t-test, both for CHF patients and normal subjects. Differences between patients and controls were analyzed using unpaired Student’s t-test. Pearson’s coefficient was calculated to assess correlation between variables. A P value < 0.05 was chosen as the threshold of statistical significance.

RESULTS

The participants completed the protocols without difficulty except one patient who was able to tolerate
the supine position for only 2 min. Data from that particular trial were, therefore, discarded from analysis.

**Ventilatory pattern**

Table 2 illustrates the ventilatory patterns of the CHF patients and controls in both the sitting and supine positions and during NIMV administration. In the sitting position VT was smaller and breathing frequency higher, albeit not statistically significantly so, in the CHF patients, so that minute ventilation was similar in the two groups. The change to a supine position resulted in a decrease in VT in both groups, but this decrease achieved statistical significance only in the CHF patients ($P < 0.05$). During NIMV, minute ventilation was statistically higher. This was due to a significant increase in VT while respiratory rate decreased; at the same time $\text{SaO}_2$ increased and $\text{ETCO}_2$ decreased.

**Inspiratory muscle strength**

In the sitting position diaphragm strength, measured by sniff Pdi, was statistically significantly lower than in normal subjects ($92.15 \pm 14.2 \text{ cm H}_2\text{O} \text{ CHF sit vs. } 120.3 \pm 16.1 \text{ Controls sit; } P < 0.01$). Assuming a supine position produced a significant decrease, but only in CHF patients ($81.6 \pm 13.7 \text{ cm H}_2\text{O} \text{ CHF sup vs. CHF sit; } P < 0.01$).

**Inspiratory muscle effort**

Changes induced by posture were negligible for controls, but statistically different in the CHF patients. Table 3 shows the tidal Pdi (Pdi$_T$), the PTPdi/b, the PTPdi/min, the PTPes/b and PTPes/min values in the two groups recorded in both postures. All the parameters of muscle effort in the sitting position were significantly higher in CHF patients than in the control subjects, while assuming a supine position induced a further significant increase. NIMV, applied only to the CHF patients, significantly reduced the energy expenditure of the diaphragm and the other inspiratory muscles. Figure 1 illustrates the percentage changes from sitting position in the PTPdi and PTPes per minute in the CHF and control subjects during unassisted breathing.

**Lung mechanics**

Figure 2 upper part is a box-whisker plot comparing resistances in the two groups. In the sitting position control subjects and CHF patients had similar resistances, although they were slightly higher in the latter group. Changing to a supine position significantly increased the resistances only in CHF patients. Lung compliance was significantly lower in supine CHF patients than in sitting ones. PEEPi,dyn was similar in both groups in the sitting position ($1.06 \pm 1.22 \text{ cm H}_2\text{O} \text{ CHF sit vs. 0.5+0.1 for controls } \text{sit; } P=\text{n.s.}$), but significantly worsened with a change in posture only in the CHF patients ($1.64 \pm 1.35 \text{ cm H}_2\text{O} \text{ CHF sup vs. CHF sit; } P < 0.05$).

Posture-induced changes in end-expiratory lung volumes, as assessed by inductive plethysmography, were $-138 \text{ ml in patients with CHF vs. } -583 \text{ ml in control subjects}$.

**Dyspnea**

The passage from a sitting to supine position induced a significant deterioration in Borg’s score of dyspnea in CHF patients (from $1.3 \pm 1$ to $4.8 \pm 2.1; P < 0.01$), while the application of NIMV to the already supine patients was associated with a significant improvement in the score. Of interest the increasing in Borg score recorded from the sitting to supine position was significantly ($P < 0.005$) correlated to the percent increase in PTPdi (Fig. 3 upper part). Similarly, the decrease in Borg score observed during NIMV was significantly ($P < 0.01$) correlated to the percent decrease in PTPdi (Fig. 3 lower part).

**Table 2.** Ventilatory pattern during the two postures and during non-invasive mechanical ventilation (NIMV) in the two groups.

<table>
<thead>
<tr>
<th>Variables</th>
<th>CHF sitting</th>
<th>CTR sitting</th>
<th>CHF supine</th>
<th>CTR supine</th>
<th>CHF NIMV</th>
</tr>
</thead>
<tbody>
<tr>
<td>VT (ml)</td>
<td>499 ± 146</td>
<td>591 ± 91</td>
<td>451 ± 108</td>
<td>555 ± 102</td>
<td>758 ± 138*</td>
</tr>
<tr>
<td>Respiratory rate (breath/min)</td>
<td>18.1 ± 5.3</td>
<td>14.7 ± 2.8</td>
<td>18.9 ± 5.0</td>
<td>15.0 ± 2.6</td>
<td>14.6 ± 4.5†</td>
</tr>
<tr>
<td>Ve (l/min)</td>
<td>8.63 ± 1.45</td>
<td>8.53 ± 0.94</td>
<td>8.09 ± 1.29</td>
<td>8.32 ± 2.05</td>
<td>11.1 ± 3.2*</td>
</tr>
<tr>
<td>Ti/TOT (%)</td>
<td>374 ± 5.6</td>
<td>36.3 ± 3.5</td>
<td>398 ± 7.4</td>
<td>35.5 ± 2.9</td>
<td>34.6 ± 3.3</td>
</tr>
<tr>
<td>ETCO$_2$ (mmHg)</td>
<td>34.5 ± 5.0</td>
<td>NA</td>
<td>35.1 ± 4.7</td>
<td>NA</td>
<td>31.7 ± 4.4‡</td>
</tr>
<tr>
<td>SaO$_2$ (%)</td>
<td>94.6 ± 1.2</td>
<td>NA</td>
<td>92.5 ± 34</td>
<td>97.2 ± 1.3†</td>
<td></td>
</tr>
</tbody>
</table>

* $P < 0.01$ CHF supine vs. CHF NIMV.
† $P < 0.05$ CHF supine vs. CHF NIMV.
‡ $P < 0.01$ CHF sitting and supine vs. CHF NIMV.
The two groups, during spontaneous breathing.

In the present study we have first shown that energy expenditure of the diaphragm increases significantly when the one hand to reduce the elastic load and that due to the other hand NIMV, able to unload the inspiratory muscle by about three-fold, had a salutary effect on dyspnea which was significantly reduced. O'Donnell et al. (17) showed in these patients that during the early stage of cycle ergometer exercise, inspiratory aid alone did not appreciably affect dyspnea, while CPAP alone had a modest effect, despite the endurance time improving in both trials. In the present study we have applied a combination of the two ventilatory aids, likely on the one hand to reduce the elastic load and that due to PEPPi, dym (using CPAP) and on the other the resistive loads (using Pressure Support) (18), so that the net effect on dyspnea may have been enhanced. Respiratory muscle weakness has been demonstrated in stable CHF patients, (14,15,19), so it has also been claimed that reduced strength of these muscle may play a role in the development of exertional and resting dyspnea. Most studies examining this aspect were, however, performed using volitional maneuvers and particularly static maximal mouth pressure, during which it may be difficult to exclude that the subject’s muscle activation is submaximal.

**DISCUSSION**

Dyspnea and orthopnea in particular are common clinical features in patients affected by chronic heart failure (12). It has been claimed that dyspnea is influenced by the central nervous system’s perception of inspiratory motor output (13), that may increase with a reduction in respiratory muscle strength (14,15). As a matter of fact, in patients affected by chronic obstructive pulmonary diseases (COPD), dyspnea seems to be well correlated with the portion of the maximal inspiratory pressure that each patient has to face during inspiration rather than the maximal output that can be generated (16). In the present study we have first shown that energy expenditure of the diaphragm increases significantly when a CHF patient lies down, due to an increase of the elastic (i.e. decrease in lung compliance) and resistive (i.e. increase in lung resistance) loads, and that this rise is very strictly correlated with orthopnea. Conversely, orthopnea was alleviated by the application of mechanical ventilation which, by reducing inspiratory muscle effort, decreased the sense of breathlessness.

The sensation of dyspnea has been linked to several factors. One of the most important of these is the burden that the inspiratory muscles have to face (6,16). While this may be not the case in patients affected by a “chronic” load, such as COPD patients, any “acute change” in this load may induce a profound variation in the sense of breathlessness (2). In CHF patients orthopnea often occurs very quickly, and in this study it was accompanied by a simultaneous increase in energy expenditure by the diaphragm and other inspiratory muscles. On the other hand NIMV, able to unload the inspiratory muscle by about three-fold, had a salutary effect on dyspnea which was significantly reduced. O’Donnell et al. (17) showed in these patients that during the early stage of cycle ergometer exercise, inspiratory aid alone did not appreciably affect dyspnea, while CPAP alone had a modest effect, despite the endurance time improving in both trials. In the present study we have applied a combination of the two ventilatory aids, likely on the one hand to reduce the elastic load and that due to PEPPi, dym (using CPAP) and on the other the resistive loads (using Pressure Support) (18), so that the net effect on dyspnea may have been enhanced. Respiratory muscle weakness has been demonstrated in stable CHF patients, (14,15,19), so it has also been claimed that reduced strength of these muscle may play a role in the development of exertional and resting dyspnea. Most studies examining this aspect were, however, performed using volitional maneuvers and particularly static maximal mouth pressure, during which it may be difficult to exclude that the subject’s muscle activation is submaximal.

**Table 3.** Diaphragm and inspiratory muscle function during the two postures and during non-invasive mechanical ventilation (NIMV) in the two groups.

<table>
<thead>
<tr>
<th>Variables</th>
<th>CHF sitting</th>
<th>CTR sitting</th>
<th>CHF supine</th>
<th>CTR supine</th>
<th>CHF NIMV</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pdi (cm H2O)</td>
<td>7.23 ± 2.72</td>
<td>4.47 ± 1.25*</td>
<td>8.96 ± 3.12</td>
<td>4.89 ± 1.59</td>
<td>3.68 ± 2.45†‡</td>
</tr>
<tr>
<td>PesT (cm H2O)</td>
<td>6.36 ± 7.2</td>
<td>3.85 ± 1.07*</td>
<td>7.38 ± 2.97</td>
<td>3.91 ± 1.44</td>
<td>2.97 ± 2.52††</td>
</tr>
<tr>
<td>PgaT (cm H2O)</td>
<td>1.48 ± 0.68</td>
<td>0.77 ± 0.48*</td>
<td>1.32 ± 0.37</td>
<td>0.93 ± 0.51</td>
<td>0.73 ± 0.32‡‡</td>
</tr>
<tr>
<td>PTPdi/min (cm H2O × s)</td>
<td>10.36 ± 4.96</td>
<td>6.54 ± 1.75*</td>
<td>13.15 ± 6.26*</td>
<td>6.85 ± 1.60</td>
<td>5.3 ± 2.5††</td>
</tr>
<tr>
<td>PTPes/min (cm H2O s)</td>
<td>9.33 ± 3.67</td>
<td>4.89 ± 1.42*</td>
<td>10.91 ± 5.54*</td>
<td>5.06 ± 1.22</td>
<td>4.2 ± 2.1††</td>
</tr>
<tr>
<td>PTPdi/min (cm H2O s/min)</td>
<td>181.2 ± 54.6</td>
<td>96.6 ± 32.8*</td>
<td>243.2 ± 970*</td>
<td>103.5 ± 44.2</td>
<td>81.8 ± 42.4††</td>
</tr>
<tr>
<td>PTPes/min (cm H2O s/min)</td>
<td>166.6±48.3</td>
<td>83.5±27.3*</td>
<td>226.9±84.3*</td>
<td>895±42.0</td>
<td>71.7±33.7††</td>
</tr>
</tbody>
</table>

*P < 0.01 CHF sitting vs CTR sitting.
†P < 0.001 CHF supine vs CHF NIMV.
‡P < 0.001 CHF supine vs CHF NIMV.
§P < 0.01 CHF supine vs CHF NIMV.
*P < 0.01 CHF sitting vs CHF NIMV.

**Fig. 1.** Percentage changes from sitting to supine position in the pressure– time product per minute of the diaphragm (PTPdi/min) and of the other inspiratory muscle (PTPes/min) in the two groups, during spontaneous breathing.

*=P < 0.01.
Using measurements that are less dependent on patients' aptitude, such as maximal sniff transdiaphragmatic pressure and cervical magnetic nerve stimulation, Hughes and coworkers (14) concluded that global respiratory muscle strength is well preserved in CHF patients and that only relatively mild weakness occurs in the diaphragm, but that this is unlikely to be clinically important. Indeed, the same authors in a further study showed that slowing of inspiratory muscle relaxation occurs with walking to severe breathlessness (20), directly suggesting that severe loading of the inspiratory muscles is a feature of dyspnea.

Lying for a few minutes resulted in a significant increase in the patients affected by CHF. The PTPdi per breath depends on the time of inspiration and the tidal Pdi generated during each inspiration (10). Since the two postures did not influence the respiratory timing in either group, the increase in the metabolic consumption was due to the use of a higher portion of Pdi, with respect to the maximal Pdi generated. Partitioning of PTPdi includes the threshold due to PEEPi, the component due to lung elastance and finally that due to the respiratory resistances (21). PEEPi, dyn did not significantly change in our patients. This is apparently in contrast with the results of a study by Duguet et al. (2) who reported the onset or exacerbation of flow limitation in patients with acute left heart failure. Flow limitation leads to dynamic hyperinflation, through incomplete lung emptying, with the persistence of an alveolar pressure above atmospheric pressure at the end of expiration not dependent on an increase in breathing frequency which was relatively constant despite the change in posture. In our study we found only a small, not significant increase in PEEPi,dyn on changing posture, probably related to the mild increase in breathing frequency. On the other hand, in CHF patients we have found only small (138ml) changes in end-expiratory lung volume, as assessed by inductive plethysmography, when they were lying supine, suggesting that flow limitation probably did not occur. This is in keeping with the findings of Yap et al. (1) The present study relies much on the recording of PTPdi when passing from the sitting to supine position in CHF patients (upper part) and between absolute changes in dyspnea (Borg) and percentage decrease in PTPdi when NIMV was applied in supine CHF (lower part).

**Fig. 2.** Box–Whisker plot comparing resistances (upper part) and lung compliance (lower part) during the two postures in the two groups. Small square = mean Large square = ± 1st D. Vertical line = ± 1.96 st D. * = P < 0.01 CHF sitting vs. CHF supine. **Fig. 3.** Linear regression analysis between absolute changes in dyspnea (Borg) and percentage increase in PTPdi when passing from the sitting to supine position in CHF patients (upper part) and between absolute changes in dyspnea (Borg) and percentage decrease in PTPdi when NIMV was applied in supine CHF (lower part).
changes in body position were rather small as well as those in mean inspiratory flow. Moreover, we are also confident that PTdi is a rather good estimate of the metabolic cost of breathing since it increased with a concomitant increase in resistance and decrease in compliance with the supine position.

Lung resistances were fairly similar in sitting CHF patients and controls but increased significantly (approximately doubling) when the CHF patients assumed a supine position. This phenomenon was recorded a long time ago using a balloon-catheter technique (3) and has been very recently confirmed using the so-called forced oscillation technique applied at the mouth during tidal breathing (1). In fact, Yap et al. (1) demonstrated a very similar rise in total respiratory resistance to that observed in the present study. The present study was not aimed at investigating the possible mechanisms inducing a rise in lung resistances, but it is unlikely that this rise can be attributed to a reduction in FRC when supine that, if present, could cause an increased, gravity-determined closure of small airways. On the other hand, it has been shown that this increase in resistance is not related either to reflex bronchoconstriction, since it was only slightly attenuated by treatment with an inhaled muscarinic antagonist, or to the development of peri-airway edema and increased airway closure (1), so further studies are required to investigate the mechanisms of these changes.

Dynamic lung compliance in the sitting position was slightly, but not significantly, lower in CHF patients than in the controls subjects, but changing position resulted in a significant decrease in the former group. Evans and coworkers (12) also found a mild reduction in static lung compliance in CHF patients at rest, but this did not correlate with either treadmill exercise capacity or dyspnea. The recording of static lung compliance has the advantage, with respect to recording the dynamic one, of reflecting only the role of lung elasticity, and not of lung resistance as well. Unfortunately, the measurement of static compliance has the objective disadvantage of having a poor coefficient of repeatability (12), and of being quite difficult to perform in patients, especially when orthopneic develops, so that after a few attempts to measure it in the first 3 patients, we decided to record only the dynamic component. It has been claimed that the reduction in compliance is a result of a decrease in functioning alveoli, since the close relationship between absolute static lung compliance and FRC, and reduced regional ventilation in CHF patients, are both consistent with the above mentioned hypothesis (22).

The present study has some inherent limitations. We excluded all patients known to have COPD or to be at risk of it. As shown in Table I, spirometric evaluation ruled out significant obstruction despite about half the patients being smokers or ex-smokers.

In normal subjects, the supine posture induces a cephalic shift of the diaphragm and an increase in intrathoracic blood volume that reduces resting lung volume; this may account for the rise in resistances. It has, therefore, been postulated that to interpret changes in pulmonary mechanics induced by lying supine, changes in lung volume need to be monitored (1). Yap et al. (1) monitored absolute lung volumes during impedance measurements by integrating the flow signals, estimating FRC during the supine position, assuming that TLC did not change from sitting to supine position. We directly assessed end-expiratory lung volume (EELV), the main determinant to interpret changes in pulmonary mechanics, by using inductive plethysmography. As a matter of fact, our results for both normal subjects and CHF patients were very similar to those obtained by Yap et al. (1), so, since EELV changed only very slightly in CHF, it is unlikely that supine reduction in static lung volumes accounted for changes in pulmonary mechanics.

In conclusion, dyspnea worsened considerably in patients with chronic heart failure on adopting a supine posture. The present study showed that breathlessness was strictly related to a rapid increase in energy expenditure of the diaphragm, due in particular to a rise in the elastic and resistive loads against which the inspiratory muscle must work. The application of NIMV to these patients when they were lying supine significantly reduced the inspiratory effort and simultaneously the sense of dyspnea.

REFERENCES


