Patterns of chest wall kinematics during volitional pursed-lip breathing in COPD at rest

Roberto Bianchi, Francesco Gigliotti, Isabella Romagnoli, Barbara Lanini, Carla Castellani, Barbara Binazzi, Loredana Stendardi, Michela Grazzini, Giorgio Scano*

Fondazione Don C. Gnocchi (IRCCS), Via Imprunetana, 124, 50020 Pozzolatico, Firenze, Italy

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KEYWORDS
Chronic obstructive pulmonary disease (COPD);
Rehabilitation;
Chest wall;
Dyspnea;
Breathing exercises

Summary
Background: Analysis of chest wall kinematics can contribute to identifying the reasons why some patients benefit from pursed-lip breathing (PLB).
Material and methods: We evaluated the displacement of the chest wall and its compartments, the rib cage and abdomen, by optoelectronic plethysmography (OEP), during supervised PLB maneuver in 30 patients with mild to severe chronic obstructive pulmonary disease (COPD).
Results: OEP showed two different patterns. A first pattern characterized the 19 most severely obstructed and hyperinflated patients in whom PLB decreased end-expiratory volumes of the chest wall and abdomen, and increased end-inspiratory volumes of the chest wall and rib cage. Deflation of the abdomen and inflation of the rib cage contributed to increasing tidal volume of the chest wall. The second pattern characterized 11 patients in whom, compared to the former group, PLB resulted in the following: (i) increased end-expiratory volume of the rib cage and chest wall, (ii) greater increase in end-inspiratory volume of the rib cage and abdomen, and (iii) lower tidal volume of the chest wall. In the patients as a whole changes in end-expiratory chest wall volume were related to change in Borg score ($r^2 = 0.5$, $p < 0.00002$).
Conclusions: OEP helps identifying the reason why patients with COPD may benefit from PLB at rest.

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*Corresponding author. Tel.: +39 055 260 11; fax: +39 055 260 1272.
E-mail address: uopneumo.pozzolatico@dongnocchi.it (G. Scano).

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Introduction

Pursed-lip breathing (PLB) performed as nasal inspiration followed by expiratory blowing against partially closed lips is a breathing retraining strategy employed by patients with chronic obstructive pulmonary disease (COPD).\(^1\) Not all patients, however, employ PLB, or report benefiting from it. Some apply this technique spontaneously, whereas other patients do not use it even when they are taught.

Studies on patients who naturally incorporate PLB into their breathing pattern may provide additional information on whether changes in chest volume help identify the reasons why PLB relieves dyspnea in patients with COPD. It has been reported that the relief from dyspnea provided by PLB is related to its ability to promote a slower and deeper breathing.\(^2,3\) In contrast, an increase in tidal volume with unchanged end-expiratory-lung-volume variably affects dyspnea during exercise either by enhancing it or leaving it unmodified.\(^4\)

An increased tidal volume, however, may be obtained in two different ways: (i) by decreasing end-expiratory volume of the abdomen and limiting the increase in end-inspiratory volume of both the rib cage and abdomen. This would limit pressure production of rib cage muscles and the diaphragm to a small fraction of their maximal pressure-generating capacity, therefore, attenuating the sensation of dyspnea in symptomatic patients\(^3,5,6\). (ii) in contrast, without the abdominal contribution, the tidal volume of the chest wall is the result of a higher rib cage inspiratory muscle fractional pressure; this, conceivably, would not attenuate the dyspnea.\(^5,6\)

Thus, the question arises: do changes in operational chest wall volumes help identify the reason for dyspnea relief with PLB in patients with COPD?

We hypothesized that an increase in tidal volume of the chest wall promoted by PLB at the exclusive expense of tidal volume of the rib cage would not be associated with dyspnea relief in COPD patients.

To assess whether the volume changes of chest wall can help identify the reason why some patients benefit from PLB while others do not, we applied a recently well-developed technique based on optoelectronic plethysmography (OEP) which allows the evaluation of volume changes of chest wall compartments.\(^7,9\) We performed direct measurements of end-expiratory and end-inspiratory chest wall volumes during natural breathing while avoiding measurements based on wearing a mouthpiece and nose clip. Breathing through a mouthpiece with a nose clip in place is known to alter the pattern of breathing, primarily by increasing tidal volume.\(^10,11\)

Methods

Patients

Thirty COPD patients with moderate to severe airway obstruction and mild to moderate hyperinflation and hypoxemia participated in the study (Table 1). They were selected from a pulmonary rehabilitation program if they satisfied each of three criteria: (i) long history of smoking and moderate to severe chronic dyspnea score (MRC > II),

<table>
<thead>
<tr>
<th>Group (30 subj.)</th>
<th>Age (years)</th>
<th>BMI (kg m(^{-2}))</th>
<th>Smoke (p.p.y.)</th>
<th>FEV(_1) (l)</th>
<th>FEV(_1)/VC (%)</th>
<th>FRC (l)</th>
<th>FRC (p.v.)</th>
<th>T.I.C. (l)</th>
<th>T.I.C. (p.v.)</th>
<th>RV (l)</th>
<th>RV (p.v.)</th>
<th>PaCO(_2) (Torr)</th>
<th>PaCO(_2) (p.v.)</th>
<th>MRC (a.u.)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Subgroup 1 (19 subj.)</td>
<td>71 ± 7</td>
<td>26 ± 3</td>
<td>46 ± 15</td>
<td>87 ± 21</td>
<td>45 ± 16</td>
<td>38 ± 11</td>
<td>138 ± 38</td>
<td>111 ± 20</td>
<td>156 ± 56</td>
<td>118 ± 11</td>
<td>118 ± 13</td>
<td>152 ± 18</td>
<td>152 ± 28</td>
<td>113 ± 27</td>
</tr>
<tr>
<td>Subgroup 2 (11 subj.)</td>
<td>71 ± 8</td>
<td>26 ± 3</td>
<td>46 ± 15</td>
<td>87 ± 21</td>
<td>45 ± 16</td>
<td>38 ± 11</td>
<td>138 ± 38</td>
<td>111 ± 20</td>
<td>156 ± 56</td>
<td>118 ± 11</td>
<td>118 ± 13</td>
<td>152 ± 18</td>
<td>152 ± 28</td>
<td>113 ± 27</td>
</tr>
</tbody>
</table>

Values are mean ± SD; VC, vital capacity; FEV\(_1\), forced expiratory volume in 1 s; FEV\(_1\)/VC, Tiffeneau ratio; FRC, functional residual capacity; RV, residual volume; MRC, Medical Research Council Dyspnea Scale; p.v., predicted value; a.u., arbitrary units.
Respiratory Society. for lung volumes are those proposed by the European Sensor Medics; Yorba Linda, CA, USA.). The normal values previously described. Briefly, 89 reflecting markers were assessed by applying a noninvasive OEP technique, used as previously. Flow signal was obtained by integrating volume integral to an integral over this surface, as described previously. The volume of the chest wall was computed by triangulating the surface and then using Gauss’s theorem to convert the volume of the chest wall into a surface integral over this surface, as described previously.

Protocol

Routine lung function was measured first and then patients were familiarized with procedures and scales for rating symptom intensity. Compartamental lung volumes were evaluated with subjects in a seated position at rest during both quiet breathing (QB) defined as a patient’s habitual comfortable breathing, and PLB. A physiotherapist gave every patient the same instructions about PLB execution. Patients were instructed to make a nasal inspiration followed by expiratory blowing against partially closed lips, avoiding forceful exhalation.

No patient had difficulty in learning this technique. Three trials of both QB and PLB maneuvers, performed correctly and with care in random order, were recorded for at least 6 min with the exclusion of sighing and coughing, and then averaged. Dyspnea sensation was measured before, during and after QB and PLB. The study was approved by the Ethics Committee of the Institution and informed consent was obtained from subjects.

Lung function

Routine spirometry obtained with subjects in a seated position was measured as previously reported. FRC (functional residual capacity) was measured by a constant volume whole-body plethysmograph (Autobox Di; 6200 Sensor Medics; Yorba Linda, CA, USA.). The normal values for lung volumes are those proposed by the European Respiratory Society.

Chest wall kinematics and compartmental volumes

The volume of the chest wall ($V_{\text{CW}}$) was modeled as the sum of the volumes of the rib cage ($V_{\text{RC}}$), and abdomen ($V_{\text{Ab}}$). The volumes of the chest wall and its compartments were assessed by applying a noninvasive OEP technique, used as previously described. Briefly, 89 reflecting markers were placed front and back over the trunk from the clavicles to the anterior superior iliac spines along pre-defined vertical and horizontal lines. To measure the volume of chest wall compartments from surface markers we defined the following: (1) the boundaries of rib cage as extending from the clavicles to the costal margin anteriorly down from the xiphisternum, and to the level of the lowest point of the lower costal margin posteriorly; and (2) the boundaries of the abdomen as extending caudally from the lower rib cage to a horizontal line at the level of the anterior superior iliac spine. The landmark coordinates were measured with a system configuration of four infrared TV-cameras, two placed front and two 4 cm in front of the subject, at a sampling rate of 50 Hz. Starting from these coordinates the volume of the chest wall was computed by triangulating the surface and then using Gauss’s theorem to convert the volume integral to an integral over this surface, as described previously. Flow signal was obtained by integrating volume track. The end-expiratory and end-inspiratory volume of each compartment was measured at the beginning and end of inspiratory flow (zero-flow points). The difference between the end-inspiratory and end-expiratory volume of each compartment was calculated as the tidal volume ($V_T$) contribution by each compartment. Thus, $V_{\text{CW}} = V_{\text{RC}} + V_{\text{Ab}}$, and changes in $V_{\text{CW}}$ can be calculated as

$$\Delta V_{\text{CW}} = \Delta V_{\text{RC}} + \Delta V_{\text{Ab}},$$

assuming that the only factor causing chest wall volume changes is gas movement. OEP calculates absolute volumes and the absolute volume of each compartment at FRC in control conditions was considered as the reference volume. Volumes are reported either in absolute values or as changes from the volume at FRC in control conditions.

Dyspnea

Subjects were asked to quantify the following: (i) chronic exertional dyspnea by MRC questionnaire and, (ii) the sensation of nonspecific discomfort associated with the act of breathing by pointing to a score on a modified Borg scale from 0 (none) to 10 (maximal) arbitrary units (a.u.).

Data analysis

Values are means ± SD. Statistical procedure was used to test differences for paired and unpaired samples. Simple regression analysis was performed using Pearson’s correlation coefficient. The level of significance was set at $p < 0.05$. All statistical procedures were carried out using the Statgraphics Plus 5.1 statistical package (Manugistics, Rockville, MD, USA).

Results

Chest wall kinematics

The analysis of the time course of volume changes in chest wall compartments allowed us to identify two different PLB patterns. The first pattern (left panels of Fig. 1 and Table 2) characterized 19 patients we called euvolumics in whom the PLB maneuver decreased end-expiratory volumes of both chest wall ($V_{\text{CW}_{\text{ee}}}$) and abdomen ($V_{\text{Ab}_{\text{ee}}}$), and increased, to a lesser extent, end-inspiratory volumes of both chest wall ($V_{\text{CW}_{\text{ei}}}$) and rib cage ($V_{\text{RC}_{\text{ei}}}$), but not abdomen ($V_{\text{Ab}_{\text{ei}}}$). As shown in Fig. 2 expiratory deflation of chest wall ($V_{\text{CW}_{\text{ee}}}$) and abdominal compartment ($V_{\text{Ab}_{\text{ee}}}$) down to the FRC line (the dotted line in the fig), and mild inspiratory inflation of rib cage compartment ($V_{\text{RC}_{\text{ei}}}$) contributed to increasing the tidal volume of the chest wall ($V_{\text{TCW}} = V_{\text{RC}_{\text{ei}}} + V_{\text{RC}_{\text{ee}}}$; $0.63 ± 0.25\text{ L}$). Also, $V_{\text{CW}_{\text{ee}}}$ and $V_{\text{RC}_{\text{ei}}}$, but not $V_{\text{Ab}_{\text{ei}}}$, increased with PLB. This pattern was also associated with a decrease in Borg score (PLB vs QB, $p < 0.0007$) (Fig. 3).

The second pattern (right panels of Fig. 1 and left panels of Table 3) characterized 11 patients, called hyperinflators, with lower $V_{\text{TCW}}$ ($p < 0.01$), $V_{\text{TRC}}$ ($p < 0.05$) and their sum $V_{\text{RC}_{\text{ee}}}$ ($p < 0.01$) than euvolumics at QB. In these patients, PLB increased end-expiratory volumes of the chest wall and rib cage up to FRC (dotted line) ($V_{\text{CW}_{\text{ee}}}$; $0.15 ± 0.21\text{ L}$; $V_{\text{RC}_{\text{ei}}}$; $0.11 ± 0.14\text{ L}$), and increased, to a greater extent than in the euvolumics, end-inspiratory volumes of the chest...
The OEP analysis of chest wall kinematics shows why not all patients with COPD obtain symptom relief from PLB at rest. Important clinical differences were associated with the two pattern groups. Euvolumics were more severely obstructed (FEV1/VC and FEV1) and hyperinflated (FRC, TLC, RV) at baseline (Table 1). They appeared to adopt PLB during common activities of the pulmonary rehabilitation program. In contrast, hyperinflators did not use PLB if not specifically requested. Moreover, a greater decrease in V_CWE was correlated with a greater level of airway obstruction ($r = 0.45$, $p < 0.02$). Finally, change in V_CWE positively correlated with change in Borg score ($r^2 = 0.50$, $p < 0.00002$) (Fig. 5).

Discussion

The OEP analysis of chest wall kinematics shows why not all patients with COPD obtain symptom relief from PLB at rest.

Breathing pattern

Tables 2 and 3 (right panels) show the effects of PLB on QB pattern in euvolumics and hyperinflators, respectively. Analysis of the variance indicated greater PLB increase in $V_T$ ($p < 0.0004$), inspiratory time ($T_I$) ($p < 0.005$), expiratory time ($T_E$) ($p < 0.0009$) and total time of the respiratory cycle ($T_{TOT}$) ($p < 0.003$) in euvolumics than in hyperinflators.

Table 2: Effects of PLB on volumes of chest wall compartments and breathing pattern in euvolumic patients.

<table>
<thead>
<tr>
<th>Euvolumics (19 subj.)</th>
<th>QB</th>
<th>PLB-QB</th>
<th>p</th>
<th>QB</th>
<th>PLB-QB</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>$V_{CWE}$ (L)</td>
<td>30.24 ± 3.71</td>
<td>+0.25 ± 0.44</td>
<td>&lt; 0.05</td>
<td>10.12 ± 2.78</td>
<td>+0.30 ± 2.75</td>
<td>n.s.</td>
</tr>
<tr>
<td>$V_{RC}$ (L)</td>
<td>19.00 ± 2.31</td>
<td>+0.23 ± 0.31</td>
<td>&lt; 0.005</td>
<td>14.91 ± 4.91</td>
<td>−6.78 ± 4.40</td>
<td>&lt; 0.000003</td>
</tr>
<tr>
<td>$V_{Ab}$ (L)</td>
<td>11.24 ± 2.19</td>
<td>+0.02 ± 0.22</td>
<td>n.s.</td>
<td>0.74 ± 0.26</td>
<td>+0.63 ± 0.52</td>
<td>&lt; 0.00005</td>
</tr>
<tr>
<td>$V_{CW}$ (L)</td>
<td>29.50 ± 3.66</td>
<td>−0.37 ± 0.23</td>
<td>&lt; 0.00001</td>
<td>$T_I$ (s)</td>
<td>1.61 ± 0.56</td>
<td>−0.95 ± 1.13</td>
</tr>
<tr>
<td>$V_{RC}$ (L)</td>
<td>18.71 ± 2.28</td>
<td>−0.06 ± 0.18</td>
<td>n.s.</td>
<td>$T_E$ (s)</td>
<td>2.91 ± 1.03</td>
<td>−2.72 ± 1.57</td>
</tr>
<tr>
<td>$V_{AB}$ (L)</td>
<td>10.79 ± 2.16</td>
<td>−0.31 ± 0.22</td>
<td>&lt; 0.00001</td>
<td>$T_{TOT}$ (s)</td>
<td>4.52 ± 1.50</td>
<td>+3.67 ± 2.57</td>
</tr>
<tr>
<td>$V_{TCW}$ (L)</td>
<td>0.74 ± 0.26</td>
<td>+0.63 ± 0.52</td>
<td>&lt; 0.00005</td>
<td>$V_{I}/T_{TOT}$ (L s$^{-1}$)</td>
<td>0.48 ± 0.16</td>
<td>+0.10 ± 0.17</td>
</tr>
<tr>
<td>$V_{IR}$ (L)</td>
<td>0.28 ± 0.16</td>
<td>+0.29 ± 0.31</td>
<td>&lt; 0.001</td>
<td>$V_{I}/T_{I}$ (L s$^{-1}$)</td>
<td>0.27 ± 0.08</td>
<td>−0.01 ± 0.07</td>
</tr>
<tr>
<td>$V_{IC}$ (L)</td>
<td>0.46 ± 0.16</td>
<td>+0.33 ± 0.25</td>
<td>&lt; 0.00005</td>
<td>$T_{TOT}$</td>
<td>0.36 ± 0.06</td>
<td>−0.05 ± 0.05</td>
</tr>
<tr>
<td>Borg (a.u.)</td>
<td>1.61 ± 1.13</td>
<td>−0.42 ± 0.45</td>
<td>&lt; 0.0007</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Values are mean ± SD. $V_T$: minute ventilation; $R_T$: respiratory frequency; $V_I$: tidal volume; $T_I$: inspiratory time; $T_E$: expiratory time; $T_{TOT}$: total time of the respiratory cycle; $V_{I}/T_{TOT}$: mean inspiratory flow; $V_{I}/T_{TOT}$: mean expiratory flow; $T_{TOT}/T_{TOT}$: duty cycle. $V_{CWE}$: chest wall end-inspiratory volume; $V_{RC}$: rib cage end-inspiratory volume; $V_{Ab}$: abdomen end-inspiratory volume; $V_{CWE}$: chest wall end-expiratory volume; $V_{RC}$: rib cage end-expiratory volume; $V_{Ab}$: abdomen end-expiratory volume; $V_{TCW}$: tidal volume of the chest wall; $V_{IC}$: tidal volume of the rib cage; $V_{IC}$: tidal volume of the abdomen.

wall ($V_{CWE}$, $p < 0.000001$) and its compartments ($V_{Ab}$, $V_{RC}$). As shown in Fig. 4 the average increase in $V_{CWE}$ (+0.36 ± 0.30 L) was exclusively due to an increase in end-inspiratory compartmental volumes ($V_{Ab}$, $V_{RC}$). No change in Borg score was associated with this pattern (PLB vs QB, $p = n.s.$) (Fig. 3).

Figure 1: Time course of breathing pattern during QB and PLB in two representative patients. From top to bottom: volumes of chest wall ($V_{CWE}$), rib cage ($V_{RC}$) and abdomen ($V_{Ab}$). QB is quiet breathing; PLB is pursed lip breathing.

Tables 2 and 3 (right panels) show the effects of PLB on QB pattern in euvolumics and hyperinflators, respectively. Analysis of the variance indicated greater PLB increase in $V_T$ ($p < 0.0004$), inspiratory time ($T_I$) ($p < 0.005$), expiratory time ($T_E$) ($p < 0.0009$) and total time of the respiratory cycle ($T_{TOT}$) ($p < 0.003$) in euvolumics than in hyperinflators.

Figure 1: Time course of breathing pattern during QB and PLB in two representative patients. From top to bottom: volumes of chest wall ($V_{CWE}$), rib cage ($V_{RC}$) and abdomen ($V_{Ab}$). QB is quiet breathing; PLB is pursed lip breathing.

Discussion

The OEP analysis of chest wall kinematics shows why not all patients with COPD obtain symptom relief from PLB at rest.
The most severely affected patients who deflate the chest wall during volitional PLB reported improvement in their sensation of breathlessness. This was not the case in the group who hyperinflated during PLB.

The major difference between the two PLB patterns was the ability of euvolumics to decrease $V_{CW}$ by decreasing $V_{Abee}$, while limiting the increase in $V_{RCei}$. In line with previous data of ours, changes in $V_{CW}$ and $V_{Ab}$ are directly related to increase in $T_e$ in that the greater the latter the greater is the reduction in the volume. In particular, we explain the decrease in $V_{CW}$ in hyperinflated patients with lengthening $T_e$ and $T_{tot}$, not with increasing mean expiratory flow ($V_T/T_e$ see Tables 2 and 3). This mechanism is similar to that expected to reduce thoracic gas volume entrapment and exercise breathlessness after pulmonary rehabilitation programs in patients with COPD.17

The increased $V_{CW}$ was, therefore the result of both $V_{TAb}$, by exploiting the expiratory reserve volume, and $V_{TRC}$, by exploiting the inspiratory reserve volume. This strategy exploits the stores of elastic energy of the most compliant chest wall compartment, the abdomen,18 and prevents $V_{CW}$ from reaching total chest wall capacity.

In contrast, the increase in tidal volume of the chest wall in hyperinflators was due to an increase in the tidal volume of the rib cage without the contribution of abdominal tidal volume. End-inspiratory volume of the chest wall mainly resulted from rib cage inspiratory muscles; in these circumstances esophageal pressure may reach a higher fraction of maximal inspiratory muscle pressure capacity and generate a greater dyspnea score19 (see also fig. 34). Most importantly, in the conditions of the present study the lack of decrease in $V_{Abee}$ along with increase in $V_{Ab}$ suggest a lower abdominal muscle contribution, and higher
inspiratory diaphragmatic contribution to abdominal tidal volume,20 respectively.

The mechanisms of apparent symptomatic improvement with PLB have not been fully elucidated.2,21,22 Dynamic airway compression by itself may produceafferent sensory information that contributes to the sense of dyspnea experienced.23 Assumption of a slower, deeper-breathing pattern during PLB would reduce intrinsic end-expiratory-positive-alveolar-pressure (PEEPi) and, thereby, the inspiratory work of breathing.24 By using PLB, COPD patients breathe larger tidal volumes2,22 which correlate with symptomatic relief of dyspnea.26 Nonetheless, the issue of whether and to what extent PLB affects dyspnea is still a matter of debate, since the efficacy of PLB in relieving dyspnea varies greatly among COPD patients.4,25

Figure 4 Changes in volumes of chest wall (CW) and its compartments with PLB in hyperinflators. Left panel: changes in the volumes of the chest wall (VCW); middle panel: volumes of the rib cage (VRC); right panel: volumes of the abdomen (VAb). Closed symbols: end-expiratory volume; open symbols: end-inspiratory volume. The dotted line: functional respiratory capacity. Bars are means ± SEM. For explanation see Results.

![Figure 4](image_url)

**Table 3** Effects of PLB on volumes of chest wall compartments and breathing pattern in hyperinflator patients

<table>
<thead>
<tr>
<th>Hyperinflators (11 subj.)</th>
<th>QB</th>
<th>PLB–QB</th>
<th>p</th>
<th>QB</th>
<th>PLB–QB</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>VC(Wa) (L)</td>
<td>31.42 ± 4.37</td>
<td>+0.51 ± 0.15</td>
<td>&lt;0.000001</td>
<td>9.85 ± 2.87</td>
<td>+0.30 ± 2.88</td>
<td>n.s.</td>
</tr>
<tr>
<td>VC(ri) (L)</td>
<td>18.40 ± 2.90</td>
<td>+0.34 ± 0.10</td>
<td>&lt;0.000001</td>
<td>22.29 ± 5.69</td>
<td>-9.06 ± 5.14</td>
<td>&lt;0.0002</td>
</tr>
<tr>
<td>VA(ri) (L)</td>
<td>13.02 ± 2.09</td>
<td>+0.17 ± 0.07</td>
<td>&lt;0.000005</td>
<td>0.47 ± 0.22</td>
<td>+0.36 ± 0.30</td>
<td>&lt;0.003</td>
</tr>
<tr>
<td>VC(Wa) (L)</td>
<td>30.95 ± 4.28</td>
<td>+0.15 ± 0.21</td>
<td>&lt;0.05</td>
<td>1.15 ± 0.33</td>
<td>+0.64 ± 0.68</td>
<td>&lt;0.02</td>
</tr>
<tr>
<td>VC(ri) (L)</td>
<td>18.25 ± 2.85</td>
<td>+0.11 ± 0.14</td>
<td>&lt;0.05</td>
<td>1.82 ± 0.77</td>
<td>+1.49 ± 1.28</td>
<td>&lt;0.0004</td>
</tr>
<tr>
<td>VA(ri) (L)</td>
<td>12.70 ± 2.02</td>
<td>+0.04 ± 0.10</td>
<td>n.s.</td>
<td>2.97 ± 1.06</td>
<td>+2.14 ± 1.82</td>
<td>&lt;0.003</td>
</tr>
<tr>
<td>Vr(CW) (L)</td>
<td>0.47 ± 0.22</td>
<td>+0.36 ± 0.30</td>
<td>&lt;0.005</td>
<td>0.41 ± 0.10</td>
<td>+0.07 ± 0.12</td>
<td>n.s. (&lt;0.06)</td>
</tr>
<tr>
<td>Vr(ri) (L)</td>
<td>0.15 ± 0.08</td>
<td>+0.23 ± 0.18</td>
<td>&lt;0.005</td>
<td>0.28 ± 0.10</td>
<td>-0.01 ± 0.09</td>
<td>n.s.</td>
</tr>
<tr>
<td>VT(ab) (L)</td>
<td>0.32 ± 0.19</td>
<td>+0.13 ± 0.14</td>
<td>&lt;0.05</td>
<td>0.40 ± 0.06</td>
<td>-0.05 ± 0.08</td>
<td>n.s.</td>
</tr>
<tr>
<td>Borg (a.u.)</td>
<td>0.96 ± 0.57</td>
<td>+0.09 ± 0.38</td>
<td>n.s.</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Values are mean ± SD. VE: minute ventilation; RF: respiratory frequency; Vt: tidal volume; Tt: inspiratory time; Te: expiratory time; Ttot: total time of the respiratory cycle; Vr/Tt: mean inspiratory flow; Vr/Te: mean expiratory flow; Te/Ttot: duty cycle. VC(Wa): chest wall end-inspiratory volume; VC(ri): rib cage end-inspiratory volume; VA(ri): abdomen end-inspiratory volume; VC(Wa): chest wall end-expiratory volume; VC(ri): rib cage end-expiratory volume; VA(ri): abdomen end-expiratory volume; Vr(CW): tidal volume of the chest wall; Vr(ri): tidal volume of the rib cage; Vr(ab): tidal volume of the abdomen.

Figure 5 Relationship between change in end-expiratory volume of the chest wall (VC(Wa)) and change in Borg score. Open circles: euvolumics; closed circles: hyperinflators.

![Figure 5](image_url)
length–tension relationship of the inspiratory muscles so that, for any given neural input, lung expansion \( (V_L) \) is commensurate with the degree of pressure generated when the muscle shortens. This is a physiological mechanism of neuromuscular coupling of the ventilatory pump. In contrast and for opposite reasons lung hyperinflation, which promotes the shortening of the inspiratory muscle along with a higher than normal neural motor output, results in neuromuscular dis coupling and more breathlessness.

In this connection, the significant difference in increased tidal volume we found in the two groups translates into a neuromuscular dis coupling and more breathlessness.

References