Pursed-Lips Breathing Improves Inspiratory Capacity in Chronic Obstructive Pulmonary Disease

Frank J. Visser\textsuperscript{a} Sunil Ramlal\textsuperscript{a} P. N. Richard Dekhuijzen\textsuperscript{b} Yvonne F. Heijdra\textsuperscript{b}

\textsuperscript{a}Department of Pulmonology, Canisius Wilhelmina Hospital, and \textsuperscript{b}Department of Pulmonology, Radboud University Nijmegen Medical Centre, Nijmegen, The Netherlands

Abstract

Background: In patients with severe chronic obstructive pulmonary disease (COPD), pursed-lips breathing (PLB) improves the pulmonary gas exchange and hyperinflation measured by electro-optic coupling. The response to PLB in inspiratory lung function tests is not known. Objectives: The purpose of this study was to measure the effect of PLB on inspiratory parameters. Methods: Thirty-five subjects with stable COPD and a forced expiratory volume in first second (FEV\textsubscript{1}) <50\% of the predicted value were tested for the following primary parameters before and immediately after PLB, and 5 min later: forced inspiratory vital capacity, inspiratory capacity (IC), forced inspiratory volume in first second, maximal inspiratory flow at 50\% of vital capacity, and peak inspiratory flow. Patients were also tested for the following secondary parameters: vital capacity, FEV\textsubscript{1}, breathing frequency, end-tidal CO\textsubscript{2} tension, and oxygen saturation. Results: Of all the primary parameters only IC (p = 0.006) improved significantly; with regard to the secondary parameters, the mean oxygen saturation was improved by 1\% (p = 0.005) and the mean end-tidal CO\textsubscript{2} tension and breathing frequency decreased significantly (p < 0.0001 for both) to 3.2 mm Hg and 3.1 breaths/min, respectively. After 5 min the effects diminished. Conclusion: Improved IC after PLB indicates less hyperinflation in patients with severe COPD; there was no effect on parameters of flow.

Introduction

Chronic obstructive pulmonary disease (COPD) is a preventable and treatable disease but is still the fourth leading cause of death in the world [1]. The severity of COPD is defined by the degree of expiratory airflow limitation. Airflow obstruction is essential for diagnosis, and forced expiratory volume in first second (FEV\textsubscript{1}) provides a useful description of the severity of the pathological changes in COPD [1]. However, FEV\textsubscript{1} is not very well correlated with changes in dyspnea. Inspiratory parameters may be more sensitive in relation to dyspnea as was published by Taube et al. [2]. They are also sensitive to bronchodilators in patients with COPD [2–4].
Pursed-lips breathing (PLB) is a breathing exercise and an item of patient education in rehabilitation programs [5, 6]. PLB may improve pulmonary gas exchange [7, 8] and reduce the breathing frequency (BF) and end-expiratory volume measured by optoelectronic plethysmography (OEP), thereby decreasing hyperinflation [9, 10]. A decrease in dyspnea and an increase in tidal volume are other consequences of PLB in patients with moderate-to-severe COPD [6]. Additionally, a faster recovery from dyspnea and a slower respiratory rate were found after walking with PLB [11].

The physiologic changes induced by PLB cause an increased intrabronchial pressure during expiration and, as a consequence, may increase the bronchial diameter and thus improve the inspiratory and expiratory flow. The positive intrabronchial pressure prevents the collapse of the bronchi upon expiration and may therefore decrease the closing volume and improve the inspiratory capacity (IC) and vital capacity (VC).

We do not know how long this effect remains after PLB; however, we think it is maintained for approximately 5 min during quiet breathing (except when the patient performs a forced expiration). We wonder whether FEV\textsubscript{1} changes at all during PLB because of the compression due to a negative intrabronchial pressure causing airway collapse. We hypothesized that inspiratory parameters could be improved by PLB resulting in a decrease in dyspnea.

The aim of this study was to evaluate the effect of PLB in patients with severe-to-very-severe COPD (GOLD stages 3 and 4) on the following inspiratory parameters: forced inspiratory volume in first second (FIV\textsubscript{1}), IC, maximal inspiratory flow at 50% of VC (MIF\textsubscript{50}), and peak inspiratory flow (PIF); secondary outcome parameters included FEV\textsubscript{1}, forced VC (FVC), oxygen saturation, end-tidal CO\textsubscript{2} tension (ET-CO\textsubscript{2}), BF, and dyspnea.

Methods
A total of 35 consecutive patients who met the GOLD criteria for COPD were recruited from our outpatient clinic. Inclusion criteria were: GOLD stages 3 and 4, reversibility of FEV\textsubscript{1} <12% of the predicted normal value and <200 ml, age ≥40 years, smoker or former smoker (≥10 pack-years), and stable disease. Patients on oral corticosteroids or antibiotics in the month before inclusion and patients with symptomatic heart failure, respiratory diseases other than COPD, a history of asthma, allergic rhinitis, and active cancer (except basal cell carcinoma of the skin) or with spontaneous PLB were excluded. The study was approved by the hospital’s medical ethical committee and all patients gave their informed consent.

Study Design
Patients were asked not to use short-term bronchodilators 6 h prior to the study and long-term bronchodilators were stopped at least 12 h before the study. The use of tiotropium bromide and theophylline was not allowed 24 h prior to the spirometric test.

Patients were asked to rest and breathe quietly for at least 2 min before the start of the test, followed by the recording of basal values for BF, ET-CO\textsubscript{2}, and oxygen saturation (SO\textsubscript{2}) as well as lung function tests for the inspiratory and expiratory parameters FIV\textsubscript{1}, IC, forced inspiratory vital capacity (FIVC), PIF, MIF\textsubscript{50}, and FEV\textsubscript{1}.

After these measurements patients rested for 5 min and thereafter they learned the PLB procedure with the following instructions: ‘Sit straight and relax your neck and shoulders. Lean with your arms on the arm rests of your chair. Breathe quietly in through your nose and out by means of pursed lips. During the inspiration your mouth should be closed. The expiration should be about 2 times longer in duration than the inspiration.

After these instructions the patient was asked to demonstrate the PLB procedure; if the procedure was not adequately performed, the assistant corrected the patient by instructing him on what to do until the correct procedure was learned by the patient.

The values during PLB were recorded as follows: the patient practiced PLB for 2 min followed by 1 inspiratory maneuver to obtain the inspiratory parameters; this process was repeated until 5 adequate inspiratory flow curves were obtained (fig. 1). The largest FIV\textsubscript{1}, IC, FIVC, PIF, and MIF\textsubscript{50} were recorded. Responses to the visual analog scale (VAS), as well as SO\textsubscript{2}, ET-CO\textsubscript{2}, and BF were recorded just before the 5th inspiratory maneuver.
Thereafter, the patient practiced PLB for 2 min followed by the expiratory maneuver to obtain the expiratory parameters. This process was repeated until 3 adequate expiratory flow curves were obtained. The largest FEV1 and FVC were recorded. Five minutes after the last measurement the same parameters were recorded along with the responses to the VAS in order to obtain the post-PLB values.

Pulmonary Function Tests
Lung function was measured both at forced expiration and inspiration as follows: a 3-liter calibration syringe was used at 3 different emptying and filling speeds to check linearity, as recommended by American Thoracic Society (ATS) and European Respiratory Society (ERS) standards. The ambient (room) temperature was measured before each test session to allow body temperature, pressure, and saturation corrections to be applied to the flows and volumes.

To measure the basal and post-PLB values of FVC and FEV1, patients performed as many maneuvers as needed (with a maximum of 8) to achieve 3 adequate and acceptable flow-volume curves, according to conventional ATS/ERS criteria. For inspiratory parameters, 5 maximal forced inspirations after a slow and maximal expiration were obtained. Maximal inspiration was obtained when a plateau was reached or after at least 8 s of inspiration.

In order to obtain proper inspiratory parameters after a slow expiration, we started the measurement during slow expiration and stopped the procedure when the patient reached FVC, as otherwise the software of the V-MAX20 spirometer (SensorMedics) would reject the obtained values.

In the inspiratory maneuvers VC was reached before FIV1, then FIV1 = VC. The largest FVC, FEV1, and FIV1 were recorded. For the predicted FEV1 and FVC, the normal values of the European Community for Steel and Coal were used [12].

The flow-volume curves were measured with a V-MAX20 spirometer (SensorMedics). ET-CO2 was recorded with a Nellcor N1000 oximeter (Nellcor Puritan Bennett, Inc., Pleasanton, Calif., USA). SO2 was recorded with a Nellcor NP40 pulse oximeter (Nellcor Puritan Bennett).

Visual Analog Scale
The patients were asked to fill out a VAS [2, 13]. On the 10-cm long VAS scale the middle represents no change, and the left and right edges of the line represent the most dyspnea and least dyspnea, respectively.

Statistics
The differences between inspiratory and expiratory parameter values before and after PLB were calculated with a 2-tailed paired Student t test. p < 0.05 was defined as a statistically significant difference. Correlations with the VAS scale (Spearman’s rank correlation test) were determined. VAS scores are presented as means and confidence intervals (CI) of the means. The D’Agostino-Pearson omnibus normality test was used to check whether the distribution of the VAS scores was normal. For statistical calculations we used GraphPad Prism5 for Windows (www.graphpad.com).

Table 1. Clinical and demographic characteristics

<table>
<thead>
<tr>
<th>Subjects, n</th>
<th>32</th>
</tr>
</thead>
<tbody>
<tr>
<td>Female/male ratio</td>
<td>8/24</td>
</tr>
<tr>
<td>Age, years</td>
<td>63.9 ± 7.5</td>
</tr>
<tr>
<td>Height, cm</td>
<td>170.8 ± 9.7</td>
</tr>
<tr>
<td>Weight, kg</td>
<td>75.9 ± 15.8</td>
</tr>
<tr>
<td>FEV1, liters</td>
<td>1.08 ± 0.37</td>
</tr>
<tr>
<td>FEV1 %pred</td>
<td>37.15 ± 11.6</td>
</tr>
</tbody>
</table>

Data are presented as means ± SD.

Results
Of the 35 patients in the study, 2 were not able to learn the PLB procedure and 1 was not able to perform the inspiratory lung function maneuver. Therefore, 32 patients were eligible for analysis. Twenty-five patients had GOLD stage 3 and 7 had GOLD stage 4 COPD. The clinical and demographic characteristics are summarized in table 1.

Change in Inspiratory Parameters during PLB and 5 min Later
During PLB, we found a significant improvement in IC with a mean increase of 89 ml (range –190 to +570); 6 patients had an increase of 200 ml or more. MIF50 showed a significant mean decrease of 170 ml/min. The other parameters were not significantly altered by PLB.

Five minutes later, none of the inspiratory parameters showed any significant improvement in relation to the basal values (before PLB). The IC was still 61 ml higher than at baseline but a 2-tailed paired t test showed that this difference lacked significance (p = 0.061). When we compared the changes in parameters during PLB and 5 min after PLB, we found a mean change in IC of 28 ml (p = 0.237, not significant). The results are summarized in table 2.

Change in Secondary Parameters during and 5 min after PLB
Expiratory lung function parameters FEV1 and FVC did not show significant differences (mean changes in differences –11 and +59 ml, respectively). However, SO2, end-tidal pCO2, and BF all showed small but significant improvements during PLB.

Five minutes after PLB the improvements diminished somewhat, except in FVC which showed a significant improvement compared to the basal value (mean change in
Effect of PLB on Inspiratory Parameters

Respiration 2011;81:372–378

Table 2. Changes in inspiratory parameters

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Differences in inspiratory parameters before and during PLB</th>
<th>Differences in inspiratory parameters before and 5 min after PLB</th>
</tr>
</thead>
<tbody>
<tr>
<td>FIV₁, liters</td>
<td>0.061, -0.004 to 0.073, 0.57 (n.s.)</td>
<td>0.025, -0.044 to 0.094, 0.47 (n.s.)</td>
</tr>
<tr>
<td>IC, liters</td>
<td>0.089, 0.038 to 0.140, 0.006**</td>
<td>0.061, -0.004 to 0.126, 0.061 (n.s.)</td>
</tr>
<tr>
<td>MIF₅₀, l/s</td>
<td>-0.17, 0.0 to 0.34, 0.049*</td>
<td>-0.15, -0.371 to 0.070, 0.087 (n.s.)</td>
</tr>
<tr>
<td>PIF, l/s</td>
<td>-0.084, -0.26 to 0.09, 0.34 (n.s.)</td>
<td>-0.007, -0.220 to 0.21, 0.946 (n.s.)</td>
</tr>
</tbody>
</table>

n.s. = Not significant; * p < 0.05 (significant); ** p < 0.01 (very significant); *** p < 0.001 (extremely significant).

Table 3. Changes in secondary parameters

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Differences in secondary parameters before and during PLB</th>
<th>Differences in secondary parameters before and 5 min after PLB</th>
</tr>
</thead>
<tbody>
<tr>
<td>FEV₁, liters</td>
<td>-0.011, -0.056 to 0.024, 0.52 (n.s.)</td>
<td>-0.016, -0.052 to 0.019, 0.341 (n.s.)</td>
</tr>
<tr>
<td>FVC, liters</td>
<td>0.059, -0.029 to 0.148, 0.182 (n.s.)</td>
<td>0.105, 0.0285 to 0.183, 0.0093**</td>
</tr>
<tr>
<td>SO₂, %</td>
<td>0.97, 0.463 to 1.474, 0.0005***</td>
<td>0.375, -0.173 to 0.923, 0.172 (n.s.)</td>
</tr>
<tr>
<td>ET-CO₂, mm Hg</td>
<td>-3.281, -4.298 to -2.26, &lt;0.0001***</td>
<td>-2.59, -3.60 to -1.59, &lt;0.0001***</td>
</tr>
<tr>
<td>BF, breaths/min</td>
<td>-3.063, -4.066 to -2.059, &lt;0.0001***</td>
<td>-1.31, -2.421 to 0.204, 0.022*</td>
</tr>
</tbody>
</table>

n.s. = Not significant; * p < 0.05 (significant); ** p < 0.01 (very significant); *** p < 0.001 (extremely significant).

differences 105 ml; p = 0.009); however, there was no significant improvement compared to the value immediately after PLB (mean change in differences 46 ml; p = 0.143). The results are summarized in table 3.

Correlations between the Dyspnea Score and Improving Parameters with PLB

We correlated those parameters that showed significant improvements during or after PLB with the subjective change in the patients’ feelings of dyspnea. None of the parameters showed a significant correlation with the patients’ feelings of dyspnea. Only SO₂ showed a tendency to correlate weakly with the VAS score (−0.038, p = 0.08, not significant). The results are summarized in table 4.

VAS Score Immediately after PLB and 5 min after PLB

Patients recorded an improvement on the VAS scale immediately after PLB with a mean of 7.8 mm (CI 3.3–12.2) out of 50 mm. Patients recorded an improvement on the VAS scale 5 min after PLB with a mean of 7.6 mm (CI 2.6–12.5) out of 50 mm. The distribution of the VAS scores was normal.

Discussion

Change in Inspiratory Parameters Immediately after PLB

We found a significant improvement in IC following PLB. However, 9 patients showed a decrease in IC ranging from 10 to 190 ml; 4 of them had a decrease of more than 100 mm.

To our knowledge, no data exist on inspiratory pulmonary function tests after PLB in the literature, but of all the inspiratory parameters measured (FIV₁, IC, MIF₅₀ and PIF) only IC showed improvement. IC is a static lung function parameter that is also a marker of hyperinfla-
tion. OEP showed a significant reduction (mean ± SD) in the end-expiratory volume of the chest wall [9] during PLB (–0.33 ± 0.24 liters; p < 0.000004). This finding of a lower end-expiratory volume by OEP is supported by our finding of an increase in IC after PLB. However, the change in OEP volume of 0.33 liters was higher than our mean change of 0.098 liters. This difference may be partly attributed to patient selection, as we only included GOLD stages 3 and 4, and to the other types of measurements that were performed (change in chest wall dimensions). Our study also supports reduced hyperinflation (improved IC) following PLB as reported previously. This reduction in hyperinflation may also be responsible for the improved oxygen cost of breathing [14] and the faster recovery after walking [15].

MIF50 showed a significant mean decrease of 170 ml/s (p = 0.049). This change in MIF50 was the opposite of what we expected. We speculate that this effect may be caused by reflex bronchoconstriction (a stretching of the J receptor in the bronchial wall caused by higher intra-bronchial pressure during PLB). All other parameters of dynamic (forced) lung function were not significantly changed after PLB.

**Change in Inspiratory Parameters 5 min after PLB**

All inspiratory lung function parameters lacked significant improvement after 5 min in relation to the basal values (before PLB). The IC was still 61 ml higher than at baseline but a 2-tailed paired t test showed that this difference lacked significance (p = 0.061); when we compared this value to the IC during PLB we found no significant decrease either. Thus, after 5 min some of the initial improvement in IC had disappeared. We did not take any measurements later to see how long the improvement due to PLB lasted, nor did we find any clues in the literature to answer that question.

**Change in Secondary Parameters Immediately after PLB**

Expiratory lung function parameters like FEV1 and FVC did not show change after PLB. No studies are available on this subject; therefore, we are not able to compare our results with those of others.

In contrast to FEV1 and FVC, SO2, end-tidal pCO2, and BF showed small but significant improvements similar to those reported previously [16]. The reason for a better SO2 may be a lower cost of oxygen due to less hyperinflation (less work breathing). Less hyperinflation improves lung compliance which may be the explanation for the decrease in BF.

**Change in Secondary Parameters 5 min after PLB**

Five minutes after PLB the improvements in BF, end-tidal pCO2, and SO2 diminished again. FVC, however, improved compared to the basal value. This improvement in FVC surprised us, but might also reflect less hyperinflation. Why this change reached significance after 5 min and not immediately after PLB is a question we cannot answer. As stated earlier, we did not find any data on these parameters in the literature.

**Dyspnea and Correlations with Changes in Parameter Values**

We found a mean difference of 4.85 mm in the VAS score after test-retest, which was significantly less than the 7.75-mm mean difference found immediately after PLB (Mann-Whitney test; p < 0.001). This is compatible with the improvement in dyspnea that we found in the literature [6, 16]. However, no significant correlations were found between significantly changed parameters after PLB and the feeling of change in dyspnea recorded by the patients. Furthermore, as this study was underpowered to find significant changes, very weak correlations

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Correlations between dyspnea (VAS) and change in parameter values</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>immediately after PLB</td>
</tr>
<tr>
<td></td>
<td>correlation  p value</td>
</tr>
<tr>
<td>FVC</td>
<td>0.249  0.17</td>
</tr>
<tr>
<td>IC</td>
<td>0.061  0.74</td>
</tr>
<tr>
<td>SO2 (%)</td>
<td>0.377  0.059</td>
</tr>
<tr>
<td>ET-CO2 (mm Hg)</td>
<td>–0.201  0.27</td>
</tr>
<tr>
<td>BF (breaths/min)</td>
<td>–0.044  0.81</td>
</tr>
</tbody>
</table>

Table 4. Correlations between dyspnea and improving parameters
remained; thus, we do not know which of the parameters contributed to a reduced feeling of dyspnea in patients after PLB. This issue was reviewed by Dechman and Wilson [16] and they found only 1 article, by Ingram et al. [17], which suggested that the higher collapsibility of bronchial airways in responders compared with nonresponders could be responsible.

Spahija et al. [18] found a strong and significant association between change in the end-expiratory lung volume and the VAS score during exercise. However, they only tested 8 patients with COPD, 6 of whom had an FEV₁ <50% of the predicted value. From their data, we calculated the association between the change in IC and the change in the VAS score and we also found a significant association; however, when we omitted patient 7 of their data (because this case had an outlying change in the dyspnea VAS score compared with the other 7 subjects), the significant association vanished. We think that much more data of patients with severe COPD are needed in order to obtain robust results. The VAS dyspnea did not change before or after PLB at rest (which is in line with what we found). Their method of VAS measurement was different; they used an absolute VAS scale (0–10) 2 times and we used a VAS scale 1 time to express the difference (less or more dyspnea).

Bianchi et al. [10] asked 30 patients with stable COPD to perform PLB at rest and found, in 19 patients, a reduction in the end-expiratory volume of the chest wall, corresponding with less hyperinflation after PLB and an increase in tidal volume. Overall, he found an association between a decrease in the end-expiratory volume of the chest wall and a change in the BORG scale. We did not find this association, but we used a VAS scale and not a BORG scale, and while we measured IC via spirometry they used OEP. Patients who hyperinflated during PLB had better FEV₁ as a percentage of the predicted values (FEV₁ %pred) in their basal values. In the 4 patients with a decrease in IC of more than 100 ml during PLB we found a mean FEV₁ %pred of 42% compared with 37% in the whole group; we also found a mean change of 8.2 mm in their VAS dyspnea scores (range 0–20) compared with 7.8 mm in the whole group.

In another study Bianchi et al. [9] analyzed 22 patients with COPD and found that the patients with a greater reduction in hyperinflation were the patients with more severe airway obstruction. They also described a longer breathing cycle after PLB (hence a lower BF). We also found a lower BF after PLB but this was not associated with less dyspnea in our sample of COPD patients.

In our study 10 patients with a decrease of 5 or more breaths/min in their BF had a mean change in VAS score of 9.3 mm compared with 7.8 mm for the whole sample. Bianchi et al. used OEP and a Borg scale in their study. Despite the fact that we were unable to associate less dyspnea with less hyperinflation at rest, Bianchi et al. found this association in their sample of patients and Spahija et al. found an association between PLB and less dyspnea during exercise but not at rest. We think a larger sample of severe COPD patients is needed to clarify the association between PLB and the decrease in dyspnea.

Conclusions

This study showed that there was an improvement in IC after PLB, supporting the idea of a decreased hyperinflation in patients with severe COPD and a possibly higher collapsibility of the bronchial airways. SO₂, end-tidal pCO₂, and BF also improved. We were not able to correlate these changes with a decreased VAS dyspnea score, however.

References


Effect of PLB on Inspiratory Parameters

Respiration 2011;81:372–378


