Chest Wall Kinematics and Breathlessness During Pursed-Lip Breathing in Patients With COPD*

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Background: **Pursed-lip breathing (PLB) is a strategy often spontaneously employed by patients with COPD during distress situations. Whether and to what extent PLB affects operational lung volume is not known. Also, conflicting reports deal with PLB capability of decreasing breathlessness.**

Participants and measurements: **Twenty-two patients with mild-to-severe COPD were studied. Volumes of chest wall (CW) compartments (rib cage [RC] and abdomen) were assessed using an optoelectronic plethysmograph. Dyspnea was assessed by a modified Borg scale.**

Results: **Compared to spontaneous breathing, patients with PLB exhibited a significant reduction (mean ± SD) in end-expiratory volume of the CW (V_{CW}) [$V_{CW}ee$; − 0.33 ± 0.24 L, $p < 0.000004$], and a significant increase in end-inspiratory V_{CW} ($V_{CW}ei$; + 0.32 ± 0.43 L, $p < 0.003$). The decrease in $V_{CW}ee$, mostly due to the decrease in end-expiratory volume of the abdomen ($V_{A}bee$) [− 0.25 ± 0.21 L, $p < 0.00002$], related to baseline FEV_1 ($p < 0.02$) and to the increase in expiratory time (T_E) [$r^2 = 0.49$, $p < 0.0003$] and total time of the respiratory cycle (T_{TOT}) [$r^2 = 0.35$, $p < 0.004$], but not to baseline functional residual capacity (FRC). Increase in tidal volume (V_T) of the chest wall (+ 0.65 ± 0.48 L, $p < 0.000004$) was shared between V_T of the abdomen (0.31 ± 0.23 L, $p < 0.000004$) and V_T of the rib cage (+ 0.33 ± 0.29 L, $p < 0.00003$). Borg score decreased with PLB ($p < 0.04$). In a stepwise multiple regression analysis, decrease in $V_{CW}ee$ accounted for 27% of the variability in Borg score at 99% confidence level ($p < 0.008$).**

Conclusions: **Changes in $V_{CW}ee$ related to baseline airway obstruction but not to hyperinflation (FRC). By lengthening of T_E and T_{TOT}, PLB decreases $V_{CW}ee$ and reduces breathlessness.**

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Key words: breathing pattern; breathing retraining; COPD; dyspnea; hyperinflation

Abbreviations: a.u. = arbitrary units; CW = chest wall; FRC = functional residual capacity; OEP = optoelectronic plethysmography; Pga,ee = end-expiratory gastric pressure; PLB = pursed-lip breathing; Prcm,e = pressure developed by the expiratory rib cage muscles; QB = quiet breathing; RC = rib cage; Rf = respiratory frequency; T_E = expiratory time; T_I = inspiratory time; T_I/T_{TOT} = duty cycle; T_{TOT} = total time of the respiratory cycle; V_{Ab} = volume of the abdomen; $V_{A}bee$ = end-expiratory volume of the abdomen; V_{CW} = volume of chest wall; $V_{CW}ee$ = end-expiratory volume of the chest wall; $V_{CW}ei$ = end-inspiratory volume of the chest wall; $\dot{V}_E$ = minute ventilation; V_{RC} = volume of the rib cage; $V_{RC}ee$ = end-expiratory volume of the rib cage; $V_{RC}ei$ = end-inspiratory volume of the rib cage; V_{TAb} = tidal volume of the abdomen; V_{TCW} = tidal volume of the chest wall; V_{Tpn} = pneumotachographic tidal volume; V_T/T_I = mean inspiratory flow; V_T/T_E = mean expiratory flow

Pursed-lip breathing (PLB), is a breathing retraining strategy often spontaneously employed by patients with COPD to relieve dyspnea.[1–4] However, despite improvement in gas exchange[2,5] and efficiency of ventilation,[6] the efficacy of PLB in relieving dyspnea varies greatly among patients.[3,6–8]

Lung hyperinflation, by increasing the motor command to and reducing the strength of the respiratory muscles, is one of the physiopathologic mechanisms of dyspnea in patients with COPD.[9,10] In contrast, reduction of dynamic hyperinflation relieves dyspnea.[11,12]

Based on the varying changes in end-expiratory lung volume with PLB,[4,6] we hypothesized that the effect of PLB on breathlessness relies on its defla-

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tionary effect on the chest wall (CW). To verify this hypothesis, we carried out the present study on 22 patients with a large span of airway involvement. We tried to define the effect of PLB on volume changes of CW compartments, and whether the levels of baseline airway obstruction and hyperinflation are determinants for the effect of PLB on breathlessness. We applied a recently well-developed technique based on optoelectronic plethysmography (OEP) that allows the evaluation of volume changes of CW compartments.[13–16]

Materials and Methods

Patients

Twenty-two clinically stable patients with COPD and mild-to-severe airway obstruction participated in the study (Table 1). Eleven patients were truly hyperinflated (functional residual capacity [FRC]; range, 141 to 212% of the predicted value). They were selected to take part in an outpatient pulmonary rehabilitation program involving regular exercise on a bicycle and several evaluations of pulmonary function, and entered the pulmonary rehabilitation program after satisfying the following criteria: (1) long history of smoking and moderate-to-severe chronic dyspnea score (Medical Research Council dyspnea score > II); (2) clinically stable condition, with no exacerbation or hospital admission in the preceding 4 weeks; and (3) free from other significant disease potentially contributing to dyspnea.

Protocol

Routine function was measured first and then patients familiarized with procedures and scales for rating symptom intensity. Compartmental lung volumes were evaluated with subjects in a sitting position at rest during both quiet breathing (QB) and PLB. The patients were instructed to make a nasal inspiration followed by expiratory blowing against partially closed lips avoiding forceful exhalation.[1] QB was defined as habitual comfortable breathing. Both QB and PLB were recorded for at least 6 min, and then data were averaged. Several trials of each breathing condition were performed, and PLB maneuvers were supervised by a physiotherapist. Dyspnea sensation was evaluated before and after 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 according to European Respiratory Society guideline.[17] FRC was measured by volume-displacement body plethysmograph (Autobox DL 6200; SensorMedics; Yorba Linda, CA). The normal values for lung volumes are those proposed by the European Respiratory Society.[17]

CW Kinematics and Compartmental Volumes

The volume of the CW (VCW) was modeled as the sum of the volume of the rib cage (VRC), and volume of the abdomen (VAb). VCW and its compartments were assessed by applying a noninvasive OEP technique, used as previously described.[13] Briefly, 89 reflecting markers were placed front and back over the trunk from the clavicles to the anterior superior iliac spines along predefined vertical and horizontal lines. To measure the VCW compartments from surface markers defined in agreement with Kenyon and coworkers,[16] we defined the following: (1) the boundaries of rib cage (RC) 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 RC to a horizontal line at the level of the anterior superior iliac spine. The coordinates of the landmarks were measured with a system configuration of four infrared TV cameras, two placed 4 m behind and two placed 4 m in front of the subject, at a sampling rate of 50 Hz. Starting from these coordinates, the VCW was computed by triangulating the surface and then using Gauss theorem to convert the volume integral to an integral over this surface, as described previously.[13] End-expiratory and end-inspiratory volume of each compartment were 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 (VT) contribution by each compartment. Thus, VCW = VRC + VAb, and changes in VCW can be calculated as

$$\Delta VCW = \Delta VRC + \Delta VAb$$

assuming that the only factor causing VCW 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.

From OEP we derived the following: inspiratory time (TI), expiratory time (TE), total time of the respiratory cycle (TTOT), VT, mean inspiratory flow (VT/TI), mean expiratory flow (VT/TE), and duty cycle (TI/TTOT). Respiratory frequency (Rf) is calculated as 1/TTOT × 60); minute ventilation ($\dot{V}E$) is calculated as VT × Rf.

Dyspnea

Subjects were asked to quantify the sensation of dyspnea that was described to them as a nonspecific discomfort associated with the act of breathing. Patients quantified dyspnea by pointing to a score on a large Borg scale from 0 (none) to 10 (maximal) arbitrary units (a.u.).[18]

Table 1—*Anthropometric, Clinical, and Functional Data of 22 Patients

Variables	Age, yr	Height, cm	Weight, kg	VC, % Predicted	FEV_1, % Predicted	FRC, % Predicted	TLC, % Predicted	RV, % Predicted	MRC, a.u.
Mean ± SD	71 ± 7	171 ± 8	76 ± 10	84 ± 19	43 ± 16	134 ± 37	109 ± 14	151 ± 52	3.0 ± 0.8
Range	54–81	151–186	62–99	45–109	20–79	84–212	84–133	86–277	2–4

*VC = vital capacity; RV = residual volume; TLC = total lung capacity; MRC = Medical Research Council Dyspnea Scale.

Table 2—*Effects of PLB on Breathing Pattern and VCW Compartments

Variables	QB	PLB-QB	p Value
V̇E, L/min	10.26 ± 3.07	+ 0.96 ± 2.70	NS
Rf, min^{-1}	15.60 ± 5.51	− 6.85 ± 5.56	< 0.00001
VT, L	0.72 ± 0.28	+ 0.65 ± 0.48	< 0.000004
TI, s	1.57 ± 0.56	+ 1.02 ± 1.11	< 0.0004
TE, s	2.82 ± 1.06	+ 2.63 ± 2.59	< 0.0002
TTOT, s	4.38 ± 1.53	+ 3.65 ± 3.49	< 0.00008
VT/TI, L/s	0.48 ± 0.15	+ 0.10 ± 0.16	< 0.01
VT/TE, L/s	0.27 ± 0.09	+ 0.01 ± 0.07	NS
TI/TTOT	0.37 ± 0.06	− 0.03 ± 0.06	< 0.02
VCWei, L	31.15 ± 3.98	+ 0.32 ± 0.43	< 0.003
VRCei, L	19.44 ± 2.51	+ 0.26 ± 0.29	< 0.001
VAbei, L	11.71 ± 2.25	+ 0.07 ± 0.22	NS
VCWee, L	30.43 ± 3.96	− 0.33 ± 0.24	< 0.000004
VRCee, L	19.17 ± 2.49	− 0.08 ± 0.15	< 0.03
VAbee, L	11.25 ± 2.22	− 0.25 ± 0.21	< 0.00002
VTRC, L	0.27 ± 0.16	+ 0.33 ± 0.29	< 0.00003
VTAb, L	0.46 ± 0.18	+ 0.31 ± 0.24	< 0.000004

*Values are presented as mean ± SD. VAbei = abdomen end-inspiratory volume; VTRC = VT of the RC; NS = not significant.

Data Analysis

Values are means ± SD. A nonparametric statistical procedure was used to test differences (Wilcoxon test for paired samples). Individual regression analysis was performed using Pearson correlation coefficient. Least-square linear regression analysis was used to analyze the individual relationship between change in Borg score as dependent variable, and the concurrent changes in end-expiratory VCW (VCWee), end-expiratory VAb (VAbee), VT of the CW (VTCW), VT of the abdomen (VTAb), TE, TTOT, FEV_1, and FRC as independent variables. The level of significance was set at $p < 0.05$. When appropriate, multiple regression analysis with stepwise selection of the independent variables was carried out relating Borg score to the above variables. The proportion of total variance in the dependent variable, accounted for by the independent variables, is reported as the square of the correlation coefficient (r^2). All statistical procedures were carried out using the Statgraphics Plus 5.0 statistical package (Manugistics; Rockville, MD) and the Intercooled Stata 6.0 for Windows (Stata Corporation; College Station, TX).

RESULTS

Breathing Pattern

With PLB, VT, TI, TE, TTOT, and VT/TI all increased, whereas Rf and TI/TTOT decreased ($p < 0.02$ to 0.000004) [Table 2].

CW Kinematics

The time course of VCW compartment changes during QB and PLB in a representative patient is shown in Figure 1. With PLB, changes in VCWee, VAbee, end-expiratory VRC (VRCee), and in end-inspiratory VCW (VCWei) and end-inspiratory VRC (VRCei) were significant ($p < 0.03$ to 0.000004) [Table 2]. These changes are shown in Figure 2.

Relationships

ΔVCWee ($p < 0.02$) and ΔVAbee ($p < 0.03$) related to FEV_1 (percentage of predicted value), but not to FRC (percentage of predicted value), and to changes in TE and TTOT: the longer the TE, the greater the decrease in end-expiratory volume (Ta-

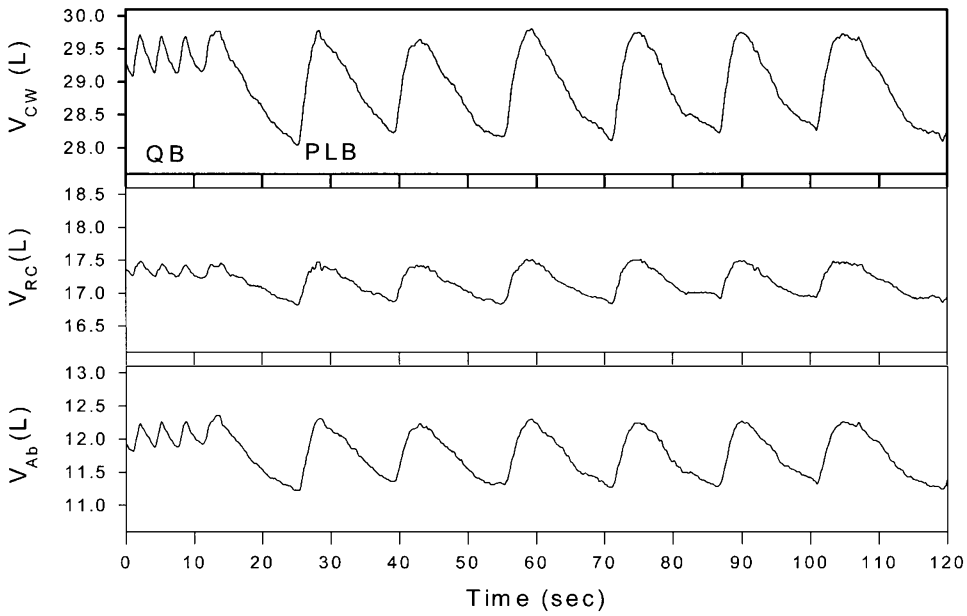

FIGURE 1. Time course of volume (V) changes of the CW compartments (RC and abdomen [Ab]) in a representative patient with severe obstruction.

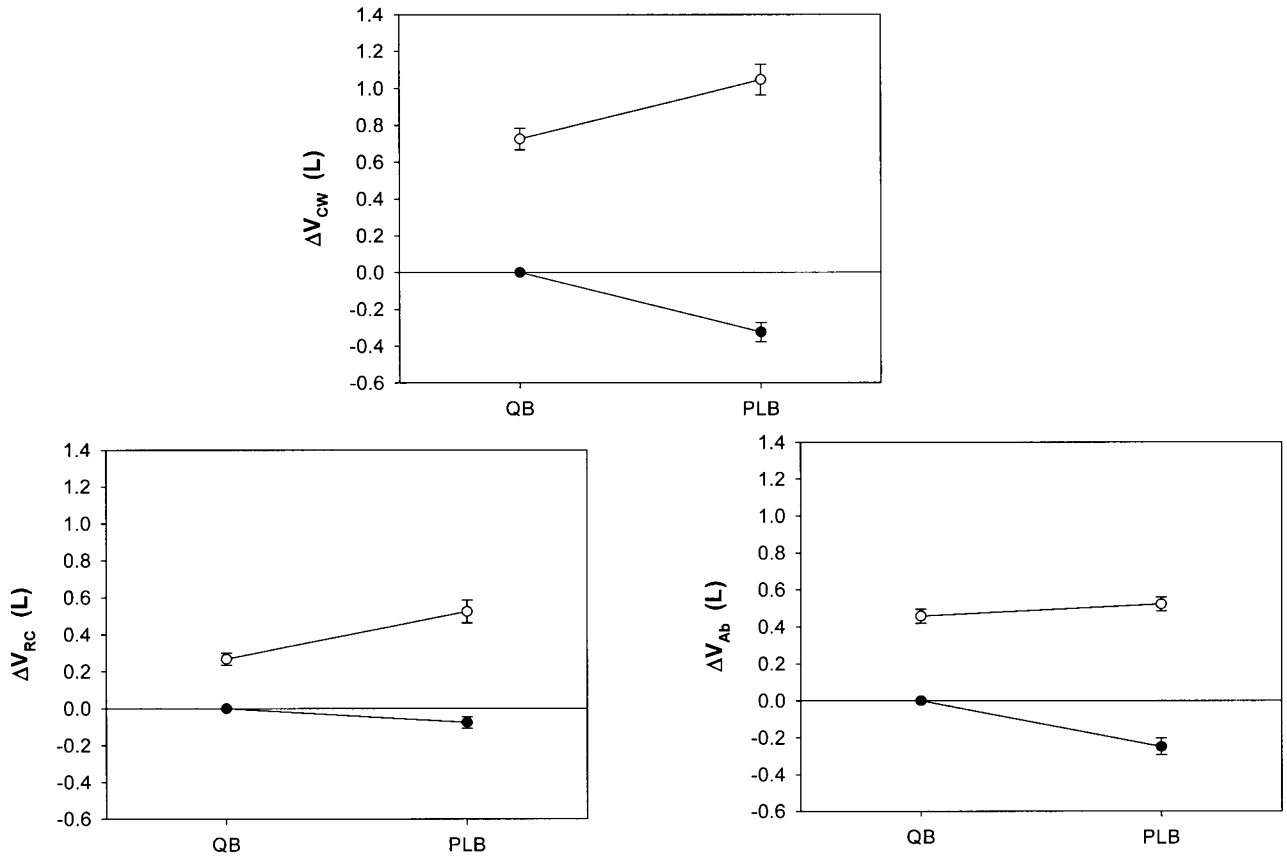

FIGURE 2. Changes in volumes of CW and its compartments with PLB. Closed symbols indicate end-expiratory volume; open symbols indicate end-inspiratory volume. Bars are means ± SEM.

ble 3; Fig 3). Changes in VTcw and VTAb related directly to both ΔTE ($p < 0.0003$ and $p < 0.0001$, respectively) and ΔTTOT ($p < 0.0001$ and $p < 0.0001$, respectively). Changes in Borg score (from 2.1 ± 1.0 to 1.8 ± 1.1 a.u., $p < 0.04$) related to ΔVCwee ($p < 0.008$), ΔVAbee ($p < 0.02$), ΔVTcw ($p < 0.04$), ΔVTAb ($p < 0.02$), ΔTE ($p < 0.008$), and ΔTTOT ($p < 0.02$), but neither to FEV_1 nor FRC baseline values.

In the multiple regression analysis, with Borg as the dependent variable and ΔVCwee, ΔVTcw, ΔVAbee, ΔVTAb, ΔTE, and ΔTTOT as the independent variables, stepwise forward regressions selected ΔVCwee as the major predictor of the variability in Borg score ($r^2 = 0.27$) at 99% confidence level ($p < 0.008$). The equation of the final fitted model was ΔBorg = 0.17103 + 1.43506 ΔVCwee.

DISCUSSION

As shown, ΔVCwee related to baseline FEV_1 but not to FRC. The increase in TE with PLB resulted in a decrease in VCwee, mostly at the abdominal level (VAbee). The decrease in Borg score significantly related to the decrease in VCwee.

Comments on Methodology

Ours is the first description of the actual volume changes in CW compartments with PLB. In particular, we performed direct measurements of end-expiratory and end-inspiratory lung volumes during natural breathing, and not indirect measurements as those based on spirometric inspiratory capacity. Although OEP requires substantial technical preparation, it provides a direct measurement of absolute volumes and variation of the CW compartments. Unlike magnetometers or respiratory inductance plethysmography, it requires neither calibration on the subject, nor depends on degree of freedom, nor yet does it require particular respiratory maneuvers involving the patient's cooperation. Moreover, it is noninvasive and does not require a mouthpiece.

We have previously reported that the relationship of VT measured by OEP and VT measured by a pneumotachograph, both in control subjects and during stimulated breathing, was highly significant,

Table 3—*Regression Coefficients (r^2) of Interrelationships Among FEV_1, Changes in Borg, Vcw Compartments, Respiratory Times With PLB***

Variables	ΔBorg	ΔVcwee	ΔVAbee	ΔVTcw	ΔVTAb	ΔTE	ΔTTOT	FEV_1
ΔBorg								
ΔVcwee	0.30							
ΔVAbee	0.26	0.63						
ΔVTcw	0.20	0.21	NS					
ΔVTAb	0.27	0.36	0.29	0.81				
ΔTE	0.30	0.49	0.31	0.49	0.54			
ΔTTOT	0.28	0.35	0.22	0.54	0.56	0.95		
FEV_1	NS	0.26	0.22	NS	NS	NS	NS	

*See Table 2 for expansion of abbreviation.

the slope and intercept being not significantly different from 1 and 0, respectively.[14] The absolute error of OEP in estimation of VT was never > 4%.

Comments on the Results

The analysis of CW kinematics showed that the decrease in end-expiratory volumes (Vcwee) mainly localized in the abdominal compartment (VAbee) was greater than previously reported.[4,6] ΔVcwee and ΔVAbee directly related to increase in TE such that the greater the latter, the greater the reduction in volumes (Fig 3). The deflation of VAb to a greater extent than that of VRC accounted for the deflation of Vcw. How can we explain the decrease in Vcwee in patients with airflow limitation? We found that the decrease in Vcwee was sustained by lengthening TTOT and TE, not by increasing the mean expiratory flow (VT/TE; Table 2). This mechanism is similar to that expected to reduce thoracic gas volume entrapment and exercise breathlessness after pulmonary rehabilitation in patients with COPD.[19]

Increase in TTOT through increases in both TE and TI accounted for the increase in VT of CW compartments. VTAb contributed to VTcw, even if to a relatively smaller extent as the VT of the RC. This strategy exploits the stores of elastic energy of the most compliant CW compartment, the abdomen.[20]

VRCei but not end-inspiratory VAb contributed to the increase in Vcwei. The close relationship of Vcwei with increase in VTAb ($r^2 = 0.45$, $p < 0.0006$) indicates the role of operational VAb to limit the increase in the end-inspiratory lung volume of the RC. In turn, exploiting the expiratory reserve volume with the contribution of the abdomen prevents end-inspiratory lung volume from reaching total lung capacity.

However, there may be an alternative explanation for changes in VRCei. Cala et al[13] have found that movements of the shoulder girdle by approximately 4 cm at closed-glottis FRC may result in several hundred milliliters of apparent artifactual volume changes. However, these conditions do not seem to apply to the conditions of our patients, in whom measurements were carried out at end-inspiration

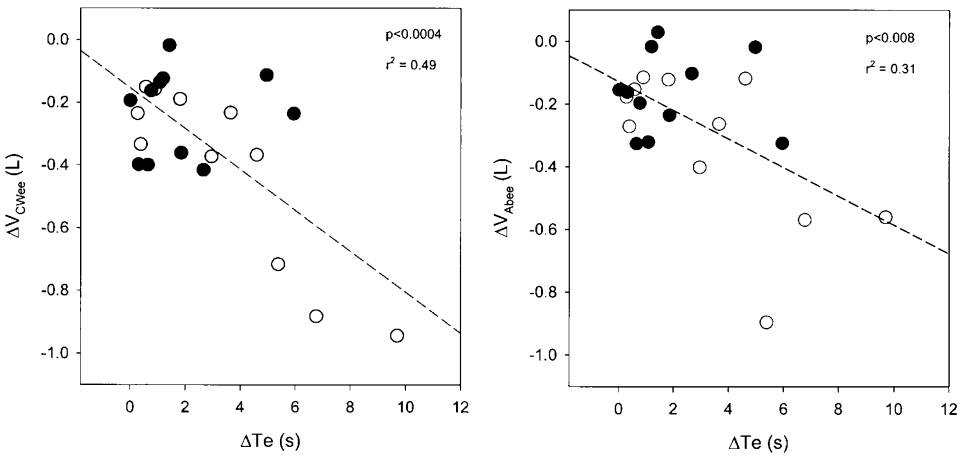

FIGURE 3. Relationships between changes in TE and changes in Vcwee and VAbee with PLB. Closed circles indicate normoinflated patients; open circles indicate hyperinflated patients.

with an open glottis. Anyway, considering the inspiratory effort (V_T/T_I) the patients made, we interpret the increase in $V_{CW}ei$ cautiously.

One could also wonder whether abdominal muscles were contracting during expiration. To answer this question, we will give a brief description of a limited study we carried out with five patients assessing the pressure developed by the expiratory RC muscles (Prcm,e) and the end-expiratory gastric pressure (Pga,ee): Prcm,e is the distance between the greatest (less negative) expiratory esophageal pressure and the relaxed CW configuration. Prcm,e significantly increased to reduce $V_{RC}ee$, while Pga,ee increased less significantly. What explains this? Despite an unchanged Pga,ee, the abdominal muscle pressure—the distance between Pga,ee and the exponentially fitted pressure/volume relaxation line of the abdomen—may significantly increase in association with the reduction in $V_A$bee. The actual problem is to assess the abdominal pressure/volume relaxation line in these patients, as an eight-loop pattern may be present even during QB.

Aliverti et al[21] and Iandelli et al[22] showed that a significant increase in abdominal pressure during expiration may be transmitted to the RC. This results in a blood shift from the thorax to the periphery. Iandelli et al[22] found that in normal subjects during progressive strenuous cycling exercise with imposition of a Starling resistor, pneumotachographic V_T ($V_T$pn) is lower than V_T calculated with OEP. After subtracting the amount of gas compression (by means of alveolar pressure) from V_T calculated with OEP − $V_T$pn difference, the blood shift was assessed. In the circumstances of the present study, gas volume reduction could amount to 15 to 20% of the 250-mL decrease in $V_A$bee; however, because we decided not to assess changes in $V_T$pn, no data are provided on blood shifting. We hypothesize that PLB may have similar effect in exercising dyspneic patients with COPD as in normal subjects during incremental exercise with expiratory flow limitation.[22]

The issue of whether and to what extent PLB affects dyspnea is still a matter of debate. The efficacy of PLB in relieving dyspnea varies greatly among patients with COPD.[8] The starting hypothesis of this study was that the effect of PLB on modulating dyspnea could rely on a decrease in V_{CW}. Our finding of a decreased $V_{CW}ee$ associated with less Borg score appears to validate that hypothesis. It has been demonstrated that reduction in Rf[2,4,5] and $\dot{V}_E$[3] and increase in V_T[2,4,5] in patients with COPD during PLB are likely factors contributing to the improvement of dyspnea in some patients.[3,6,7] In addition, PLB leads to a decrease in T_I/T_{TOT},[2] an independent correlate of dyspnea.[9,10] In line with these data, we have shown that whatever the levels of hyperinflation, the increase in T_{TOT} and T_E modulated the level of resting breathlessness. In particular, our analysis of timing components of breathing pattern has confirmed that the decrease in Rf by allowing more time for expiration (T_E) was the reason for the decrease in $V_{CW}ee$ and $V_A$bee. Both variables, along with T_E, were related to Borg score in a univariate regression analysis.

One has also to consider that limiting the increase in end-inspiratory lung volume would prevent esophageal pressure from reaching a higher fraction of maximal inspiratory pressure, thus preventing dyspnea from increasing.[9,10] The increase in ΔV_Tab, which accounted for 45% of the variability in $\Delta V_{CW}ei$, probably had this purpose.

In conclusion, changes in $V_{CW}ee$ related to baseline airway obstruction but not to hyperinflation (FRC). By decreasing Rf and lengthening T_E, PLB decreases $V_{CW}ee$ and modulates breathlessness.

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