Partitioning of the elastic work of inspiration in patients with COPD during exercise

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ABSTRACT: During exercise, dynamic hyperinflation-induced intrinsic positive end-expiratory pressure (PEEPi) and decreased dynamic lung compliance (CL_{dyn}) of patients with chronic obstructive pulmonary disease (COPD) increase the elastic work of inspiration (W_i) more than would be predicted from the increase in tidal volume (VT). This contributes significantly to their exertional breathlessness.

In 10 stable patients with COPD, the dynamic W_i was measured during incremental bicycle exercise to exhaustion. The total W_i was then partitioned into the portion required to overcome PEEPi ($W_{i,PEEPi}$) and nonPEEPi elastic load ($W_{i,nonPEEPi}$). The latter is used to overcome the increase in the total respiratory system elastance during inflation.

From resting breathing to peak exercise, W_i more than doubled ($p < 0.001$). This increase was largely due to $W_{i,PEEPi}$, which significantly rose from 1.7 ± 0.3 to 5.3 ± 0.8 L·cmH₂O⁻¹ ($p < 0.001$). In comparison, $W_{i,nonPEEPi}$ increased from only 3.0 ± 0.4 to 5.1 ± 0.5 L·cmH₂O⁻¹ ($p < 0.01$). Consequently, $W_{i,PEEPi}$ as a fraction of total W_i increased from 35.5 ± 5.6 to $51.0 \pm 3.3\%$ ($p < 0.02$). In addition, the measured $W_{i,nonPEEPi}$ at peak exercise, when expressed as a percentage of its value during resting breathing, was 25% more than that predicted from the increase in VT alone. Assuming a constant chest wall compliance, this can be attributed to the exercise-induced decrease in CL_{dyn} , which was 0.27 ± 0.04 and 0.17 ± 0.02 L·cmH₂O⁻¹ ($p < 0.01$), respectively, during resting breathing and peak exercise.

In conclusion, the dynamic hyperinflation-induced intrinsic positive end-expiratory pressure is more important than the increase in tidal volume in raising the work of inspiration during exercise in patients with chronic obstructive pulmonary disease; the decrease in dynamic lung compliance plays a definite but less important role.

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During exercise, the elastic work of inspiration (W_i) in healthy subjects increases mainly due to an increase in tidal volume (VT). This is partly compensated for by the fall in end-expiratory lung volume (EELV) as a result of exercise-induced recruitment of expiratory muscles [1–3]. In patients with chronic obstructive pulmonary disease (COPD), the increase in W_i is more than would be predicted from the increase in VT during exercise [4], and contributes significantly to the sense of breathing effort and reduced exercise capacity. Two additional factors play a crucial role in increasing W_i in these patients. Firstly, due to expiratory airflow limitation, dynamic hyperinflation is almost inevitable during exercise, leading to an increased EELV and intrinsic positive end-expiratory pressure (PEEPi) [5–8] in spite of expiratory muscle activity. PEEPi behaves as an inspiratory threshold load and needs to be overcome before inspiratory flow can be generated [9], therefore, it significantly increases W_i for a given ventilation. Secondly, the increased frequency of breathing during exercise results in a decreased dynamic lung compliance (CL_{dyn}) in patients with COPD [10, 11], which also contributes to the increase in W_i under dynamic conditions.

How PEEPi and changes in CL_{dyn} in patients with COPD quantitatively contribute to an increase in W_i during exercise has not been described in detail. The purposes of the present study were, therefore, to quantify, in a group of clinically stable patients with COPD, the increase in W_i during exercise, and to partition it into the contributions from the increased PEEPi and from the decreased CL_{dyn} , in order to better understand inspiratory mechanics during exercise in this patient population.

Methods

Patients

A total of 10 patients with severe COPD (nine males and one female) in clinically stable conditions volunteered for the experiment. Clinically stable COPD is defined as having the medical history and signs of chronic airway obstruction but without acute exacerbation within 1 month prior to the study. Patients with evidence of cardiovascular disease, systemic disease and neuromuscular disease which are likely to affect exercise performance

Table 1. – Patient characteristics and lung function test results

Pt No.	Age yr	W kg	H cm	FEV ₁ L	FEV ₁ /FVC %	R _{aw} cmH ₂ O·L ⁻¹ ·s ⁻¹	TLC L	FRC L	RV L	P _a O ₂ kPa	P _a CO ₂ kPa
1	58	76	160	0.76 (33.3)	0.42	10.1	4.37 (90.4)	3.66 (137.4)	2.72 (146.6)	10.2	5.9
2	62	68	168	0.92 (31.0)	0.37	12.8	8.50 (134.0)	6.49 (191.4)	5.56 (240.2)	8.0	6.7
3	67	68	164	0.44 (16.0)	0.25	7.2	7.89 (131.0)	6.72 (200.1)	5.40 (223.7)	10.1	5.9
4	64	78	180	1.32 (38.6)	0.46	4.6	5.60 (76.7)	4.27 (115.7)	2.69 (106.9)	7.7	6.9
5	57	87	169	1.10 (34.0)	0.31	3.6	5.12 (73.3)	2.25 (67.8)	1.55 (85.8)	10.8	5.6
6	56	55	158	0.84 (37.1)	0.35	7.0	6.41 (138.1)	4.45 (171.4)	3.69 (211.8)	8.2	5.6
7	41	54	169	0.80 (32.0)	0.30	7.7	9.22 (143.6)	7.86 (242.4)	6.65 (348.7)	8.2	5.6
8	47	65	170	0.88 (25.6)	0.29	8.6	7.80 (121.4)	6.30 (192.1)	5.08 (254.7)	7.2	5.9
9	62	83	172	0.96 (30.9)	0.25	7.7	8.40 (126.1)	6.24 (178.5)	4.71 (197.3)	9.0	5.9
10	59	70	170	0.68 (21.9)	0.25	9.4	8.87 (136.4)	7.33 (214.4)	6.23 (271.6)	7.7	6.3
Mean	57	70	168	0.87 (30.0)	0.32	7.9	7.22 (117.1)	5.56 (171.1)	4.43 (208.7)	8.8	6.0
SEM	8	3	2	0.07 (2.2)	0.02	0.8	0.54 (8.4)	0.56 (16.2)	0.53 (25.0)	0.4	0.1

Values are presented as absolute values with percentages of predicted values in parenthesis. Pt: patient; W: body weight; H: height; FEV₁: forced expiratory volume in one second; FVC: forced vital capacity; R_{aw}: airway resistance; TLC: total lung capacity; FRC: functional residual capacity; RV: residual volume. P_aO₂: arterial oxygen tension; P_aCO₂: arterial carbon dioxide tension. Lung functions were measured in a body plethysmograph. Patients No. 4 and 5 also have restrictive pulmonary changes. 1 kPa=7.52 mmHg.

were excluded. Patient characteristics and lung function test results are given in table 1. All patients gave their informed consent to the protocol which had been explained to them in detail necessary for their understanding. The experimental protocol was approved by an appropriate institutional research ethics committee.

Measurements

Respiratory flow was measured by a Fleisch No. 2 pneumotachograph (Fleisch, Lausanne, Switzerland) and a differential pressure transducer. Mouth pressure (P_m) was measured by a differential pressure transducer which was connected to the mouthpiece. Oesophageal and gastric pressures (P_{oes} and P_{ga} , respectively) were measured conventionally by two additional differential pressure transducers attached to balloon-tipped catheters. One was placed in the lower third area of the oesophagus and the other in the stomach, to reflect the changes in pleural and abdominal pressures (P_{pl} and P_{ab}), respectively. The oesophageal balloon was inflated with 0.5 mL of air and its position was carefully adjusted so that the amplitudes of P_{oes} and P_m were equal during an airway occlusion test and cardio-genic artifacts were minimized. The gastric balloon was inflated with 1.0 mL of air. End-tidal carbon dioxide tension (P_{ET,CO_2}) was measured using a CO₂ analyser whose inlet was inserted into the mouthpiece. During exercise, arterial oxygen saturation (Sa_{O_2}) and electrocardiogram (ECG) measurements were continuously monitored. Arterial blood pressure was measured at the end of each workload. Motions of the rib cage and abdomen during breathing were measured by two respiration bands placed on the upper rib cage and the abdomen. These signals were used for estimating expiratory muscle contribution to P_{oes} baseline during quiet breathing [12, 13].

Protocol

The patients were seated on an electronically braked ergometer (Ergo-Metrics 800; Ergo-Line GmbH + Co KG, Bitz, Germany) with their body tilted slightly forward and their arms supported by the handle of the ergometer in

order to maintain an unchanged posture throughout the experiment. The patients breathed through a mouthpiece while wearing a noseclip. No patient took long-lasting bronchodilators and all refrained from taking coffee or bronchodilators for at least 4 h before the experiment. At the beginning of the experiment, patients were instructed to breathe quietly on the bicycle without pedalling for ~5 min to become accustomed to the breathing circuit. In the last 2 min of this adaptation, quiet breathing data were recorded. The patients then performed an incremental bicycle exercise starting at a workload of 10 or 20 W. The workload was progressively increased every minute by 10 or 20 W until symptom limitation. At the end of quiet breathing and the end of each level of exercise, an inspiratory capacity (IC) inspiration was performed by "making a further maximal effort on top of a maximal inspiration" [6, 14].

Data analysis

The respiratory flow, P_{oes} , P_{ga} , respiration signals, Sa_{O_2} , and P_{ET,CO_2} were all amplified, digitized at 100 Hz, and stored in a computer. The actual sampling frequency was sometimes increased up to 1,000 Hz when necessary by linear extrapolation, in order to analyse fast events, such as the development of P_{oes} during the initial part of inspiration. The signals for the last five breaths at a given workload were averaged for further analysis. VT, breathing frequency, minute ventilation (V_E), and duty cycle were derived from the flow signal. Change in EELV ($\Delta EELV$) was measured by calculating the magnitude of IC, assuming the decrease in IC equals the increase in EELV during exercise [5–8]. The change in end-inspiratory lung volume ($\Delta EILV$) was calculated as $\Delta EELV + V_T$. CL_{dyn} was measured by dividing V_T by the difference in P_{oes} values at the beginning and end of inspiratory flow.

Figure 1 shows schematically the method used to calculate and partition the elastic work of inspiration. In this figure, the static volume-pressure relationship of the chest wall (the straight line passing points F, E, D) is shown. It was assumed that this relationship is linear [15] and its slope in patients with COPD is comparable to that obtained in healthy subjects [16–18]. Thus, the slope of

Fig. 1. – Diagrammatic presentation of the partitioning of the elastic work of inspiration during dynamic hyperinflation. Please see text for further explanation.

the static volume-pressure relationship of the chest wall was obtained from the literature, taking the age and sex of each patient into consideration [19]. In order to position the static volume-pressure relationship of the chest wall properly, we first determined the end-expiratory P_{oes} value during quiet breathing, to represent the chest wall elastic recoil pressure, shown at point F in figure 1. Then, we positioned the static volume-pressure relationship of the chest wall by passing it through point F. It needs to be pointed out that: firstly, since most of our patients with COPD had dynamic hyperinflation and PEEP_i during quiet breathing, P_{oes} started to fall before inspiratory flow began [20, 21]. End-expiration, therefore, was defined as the point at which P_{oes} began to fall for the next inspiratory effort rather than the beginning of inspiratory flow; secondly, due to dynamic hyperinflation during quiet breathing, the volume at point F was often already above the relaxation volume (V_r); and thirdly, expiratory muscles were active at end-expiration during quiet breathing in some patients with COPD, resulting in elevation of end-expiratory P_{oes} independent of chest wall elasticity [13]. This was confirmed by an expiratory rise in P_{ga} converted to an abrupt inspiratory fall with outward displacement of the abdomen [13]. To circumvent this problem, the P_{oes} value at point F was carefully corrected by subtracting the expiratory rise in P_{ga} whenever applicable, as recently suggested by LESSARD *et al.* [22]. Figure 2 shows the tracings of expiratory muscle recruitment during quiet breathing and defines the method used to correct the effect of this phenomenon on end-expiratory P_{oes} estimation. Five of our patients displayed the pattern shown in figure 2 during quiet breathing. The mean correction for these patients was 2.1 cmH₂O (range: 0.8–3.6 cmH₂O).

As shown in figure 1, a clockwise dynamic volume-pressure breathing loop during exercise is superimposed on the static volume-pressure relationship of the chest wall. The EELV of this breath, as given by the level of A and E, is above the volume at point F, suggesting dynamic hyperinflation or further dynamic hyperinflation. Points A and B represent the beginning and end of inspiratory flow (zero flow points), respectively. If we assume that inspiratory effort begins from point E, the pressure difference between points E and A represents dynamic PEEP_i [23]. The pressure difference between points D and B represents the total

Fig. 2. – Example of flow, oesophageal and gastric pressure (P_{oes} and P_{ga} , respectively) tracings with expiratory muscle recruitment during quiet breathing. P_{ga} is biphasic with a large rise in inspiration and a small rise in expiration. The latter suggests active expiration. The amount of the expiratory increase in P_{ga} (as shown between the arrows) is subtracted from the P_{oes} baseline, to correct the effect of expiratory muscle contribution.

inspiratory pressure needed for the whole inspiration. The straight line passing points A and C (dashed line) is made parallel to the static volume-pressure relationship of the chest wall. Consequently, the total W_i is given by the area enclosed by points A, B, D and E. However, W_i can be partitioned into the portion required to overcome PEEP_i ($W_{i,PEEPi}$) as expressed by the area enclosed by points A, C, D and E, and the portion required to overcome the non-PEEP_i elastic load ($W_{i,nonPEEPi}$) as given by the area enclosed by points A, B and C. The latter is used to overcome the increase in the total respiratory system elastance during inflation.

The $W_{i,nonPEEPi}$ measured as described above was further compared with the calculated $W_{i,nonPEEPi}$ predicted from the increase in V_T alone during exercise [24]. As shown in figure 1, $W_{i,nonPEEPi}$ is given by the triangle ABC and can be expressed as $\frac{1}{2} \cdot V_T \cdot BC$. Since BC represents the increase in total respiratory system elastic recoil

pressure during inspiratory inflation, it can be expressed by V_T/C_{rs} where C_{rs} is the compliance of the respiratory system. Thus, assuming linearity, $W_{i,nonPEEPi}$ can be calculated as $\frac{1}{2} \cdot V_T \cdot V_T/C_{rs}$ or $V_T^2/2 \cdot C_{rs}$. If C_{rs} is constant, the increase in $W_{i,nonPEEPi}$ during exercise will be proportional to the increase in V_T^2 . Any discrepancy between the measured $W_{i,nonPEEPi}$ and the $W_{i,nonPEEPi}$ predicted from the increase in V_T alone will therefore indicate the contribution of changes in C_{rs} to $W_{i,nonPEEPi}$.

Results are presented as mean $\pm$ SEM. Analysis of variance and paired t-test were used, to compare the results under different experimental conditions. A p-value of <0.05 was considered to be statistically significant.

Results

At the symptom limitation, the exercise workload accomplished by the patients was $28 \pm 2.5\%$ pred. At this level of exercise, $\dot{V}_E$ was $27.9 \pm 1.8 \text{ L} \cdot \text{min}^{-1}$.

Figure 3 shows the relationship between lung volume (VL) and P_{oes} at the beginning (open circles) and end of inspiratory flow (closed circles). Therefore, the level of VL corresponding to each open and closed circle represents EELV and EILV, respectively for a given condition. Values were obtained during quiet breathing and at different intensities of exercise. In this figure, zero VL represents the level of EELV during quiet breathing. During exercise, all patients demonstrated increased EELV suggesting dynamic hyperinflation. Most patients reached the highest EELV and EILV at maximal exercise. From quiet breathing to peak exercise, EELV increased by $0.35 \pm 0.07 \text{ L}$. This resulted in an increase in dynamic PEEP_i from 2.5 ± 0.4 – $6.0 \pm 0.7 \text{ cmH}_2\text{O}$ ($p < 0.0001$). As shown in figure 3, the curvilinear aspect of the dynamic VL- P_{oes} relationship at zero flow likely reflected the combined effects of increasing curvature of the volume-pressure curve of the lung itself due to increasing volume and decreasing CL_{dyn} during exercise. From quiet breathing to peak exercise, CL_{dyn} significantly decreased from 0.27 ± 0.04 – $0.17 \pm 0.02 \text{ L} \cdot \text{cmH}_2\text{O}^{-1}$ ($p < 0.01$).

Figure 4 shows W_i , $W_{i,PEEPi}$, and $W_{i,nonPEEPi}$ during quiet breathing and at the first, the middle, and the peak exercise intensity (E1, E2 and E3, respectively). Although all three parameters progressively increased during exercise, the rates of increase in $W_{i,PEEPi}$ and $W_{i,nonPEEPi}$ differed. During quiet breathing, $W_{i,PEEPi}$ and $W_{i,nonPEEPi}$ were 1.68 ± 0.28 and $3.02 \pm 0.43 \text{ L} \cdot \text{cmH}_2\text{O}^{-1}$, respectively, with the former significantly less than the latter ($p < 0.05$). During exercise, $W_{i,PEEPi}$ increased more than $W_{i,nonPEEPi}$, so that at peak exercise, their values reach 5.34 ± 0.78 and $5.10 \pm 0.47 \text{ L} \cdot \text{cmH}_2\text{O}^{-1}$, respectively, and were no longer significantly different ($p > 0.75$). Consequently, when $W_{i,PEEPi}$ is expressed as a fraction of W_i , the ratio progressively increased with increasing exercise intensities from $35.5 \pm 5.6\%$ during quiet breathing to $51.0 \pm 3.3\%$ at peak exercise ($p < 0.05$).

Figure 5 plots the measured $W_{i,nonPEEPi}$ as a function of the predicted $W_{i,nonPEEPi}$ calculated from the increase in V_T alone during exercise. Both are expressed as a percentage of the value obtained during quiet breathing. The measured $W_{i,nonPEEPi}$ was slightly but consistently higher than the predicted $W_{i,nonPEEPi}$. At peak exercise (E3), the discrepancy was 25%.

Fig. 3. – The relationship between lung volume (VL) and oesophageal pressure (P_{oes}) at the beginning (○) and end (●) of inspiratory flow during quiet breathing and at different intensities of exercise in individual patients. Zero VL represents the end-expiratory lung volume during quiet breathing.

Fig. 4. – Elastic work of inspiration (W_i) during exercise. ●: total W_i ; ▲: the work required to overcome intrinsic positive end-expiratory pressure (PEEPi) ($W_{i,PEEPi}$); ◆: the work required to overcome nonPEEPi elastic load ($W_{i,nonPEEPi}$). QB: quiet breathing; E1, E2 and E3: first, middle and peak exercise intensity.

Fig. 5. – The measured elastic work of inspiration (W_i) required to overcome the nonintrinsic positive end-expiratory pressure (nonPEEPi) elastic load ($W_{i,nonPEEPi}$) plotted against the predicted $W_{i,nonPEEPi}$. Both are expressed as percentage of quiet breathing (QB). – – –: the line of identity. ○: QB; ■, □ and ▲: first, middle and peak exercise intensity (E1, E2 and E3, respectively). *: measured $W_{i,nonPEEPi}$ significantly greater than predicted.

Discussion

We measured the dynamic W_i in patients with severe COPD during incremental bicycle exercise, until symptom limitation. We partitioned the total W_i into $W_{i,PEEPi}$ and $W_{i,nonPEEPi}$ which corresponded to the work performed to overcome the PEEPi and nonPEEPi elastic load of the respiratory system, respectively. We found that: 1) $W_{i,PEEPi}$ became an increasingly important contributor to W_i as exercise proceeded to the maximum. At exhaustion, $W_{i,PEEPi}$ accounted for over 50% of total W_i in these patients; and 2) at peak exercise, the measured $W_{i,nonPEEPi}$ was 25% greater than the value predicted from the increase in V_T alone. This can be explained by a significant decrease in CL_{dyn} associated with exercise.

We used the method described by FLEURY *et al.* [17] to calculate W_i while taking into consideration of $\Delta EELV$ and the PEEPi-imposed inspiratory threshold load [25]. We used the end-expiratory P_{oes} baseline to represent the end-expiratory elastic recoil pressure of the chest wall during quiet breathing, and positioned the predicted static pressure-volume relationship of the chest wall at that

point. This procedure has been used by many previous researchers [17, 25, 26]. In addition, we used the method described by LESSARD *et al.* [22] to correct the effect of expiratory muscle recruitment on determination of end-expiratory chest wall elastic recoil pressure during quiet breathing. It can be seen in figure 1 that once the static chest wall volume-pressure relationship was positioned, all subsequent measurements were referenced to this relationship and additional adjustments for expiratory muscle contribution were not necessary during exercise. However, there are still a number of concerns that need to be addressed. Firstly, end-expiratory P_{oes} could have changed due to changes in flow resistance independent of inspiratory muscle contribution, which would have influenced the reliability of point F determination in figure 1. After carefully checking our signals, we found that at end-expiration, the fall in P_{oes} almost always coincided with the rise in transdiaphragmatic pressure for the subsequent in-spiratory effort during quiet breathing, suggesting that the resistive pressure loss at end-expiration would be reasonably small. This is consistent with recent findings in patients with COPD that during spontaneous breathing, the rapid decrease in P_{oes} at end-expiration was in phase with the start of the electric activities of both the diaphragm and accessory muscles [22]. Secondly, since we used the predicted volume-pressure relationship of the chest wall as reference of the measurements for each individual, a certain degree of random error was unavoidable due to intersubject variability. We hope that this would be minimized by only reporting and interpreting the group results. Thirdly, it is obvious that the effect of any error of the slope or position of the chest wall volume-pressure curve on calculation of the W_i will be amplified by increasing V_T (fig. 1). However, V_T only increased from 0.72 L during quiet breathing to 0.88 L at maximal exercise. This makes any error of W_i or its components to be relatively constant and independent of exercise, and therefore less likely to have significantly influenced the major interpretations of our results based on comparisons between rest and at different levels of exercise.

As has been shown previously [6], in spite of severe expiratory airflow limitation, the increase in EELV in patients with COPD during exercise was modest. At symptom limitation, we found an average increase in EELV of 0.35 L. This, with the small increase in V_T of 0.16 L, however, leads to a reduction of the inspiratory reserve volume by 0.51 L or 54%. Meanwhile, $W_{i,PEEPi}$ more than tripled between quiet breathing and peak exercise and accounted for more than 50% of W_i at exhaustion, while $W_{i,nonPEEPi}$ only increased moderately (fig. 4). These results suggest that, in patients with COPD, the increase in W_i during exercise comes mainly from the increase in the magnitude of dynamic hyperinflation. Accordingly, our present work has added evidence to support the notion that dynamic hyperinflation is probably the most important factor contributing to exercise limitation in patients with COPD. In fact, even during quiet breathing, the effect of dynamic hyperinflation could be more significant than ever thought. We observed a dynamic PEEPi of 2.5 cmH₂O in our patients during quiet breathing, a magnitude consistent with other studies [20, 21]. However, 36% of W_i was required to overcome this "mild" PEEPi. Since respiratory controllers are able to detect very small increases in elastic load and respond to it to adjust ventilatory output [27], we would postulate that the PEEPi we observed at rest

might be sufficient to affect the control of spontaneous quiet breathing in patients with stable COPD.

In this study, we assumed that dynamic compliance of the chest wall ($C_{w,dyn}$) during exercise is constant and equal to its static compliance. This assumption may not be true. Our calculation of $W_{i,nonPEEPi}$ did not include influences from a possible change in $C_{w,dyn}$ [28], because we could not measure it in the present study. In other words, our results, showing that part of the increase in $W_{i,nonPEEPi}$ during exercise cannot be explained by the increase in V_T alone (fig. 5), were entirely attributable to changes in lung mechanics. As has been described previously [10], we showed a significant decrease in CL_{dyn} in our patients during exercise. This may be largely due to a significant frequency dependence of CL_{dyn} in patients with COPD [11] and, probably to a less extent, due to a decrease in the static lung compliance (CL_{stat}) at increased operating lung volume during dynamic hyperinflation. To what extent the latter contributed to the observed decrease in CL_{dyn} in our patients is unknown. MACKLEM and BECKLAKH [14] and STUBBING *et al.* [10] have shown that in patients with airway obstruction, the static pressure-volume relationship of the lung (CL_{stat}) was linear over a large part of the entire inspiratory capacity because of the loss of lung elastic recoil. Therefore, the contribution of the fall in CL_{stat} to the observed decrease in CL_{dyn} under our experimental conditions would have been small. Since the predicted $W_{i,nonPEEPi}$ was calculated by assuming a constant respiratory system compliance, and the measured $W_{i,nonPEEPi}$ was calculated by assuming a constant chest wall compliance, the discrepancy between the measured and predicted $W_{i,nonPEEPi}$ (fig. 5) was thus the direct consequence of the observed decrease in CL_{dyn} during exercise.

In summary, in patients with chronic obstructive pulmonary disease exercising to symptom limitation, dynamic hyperinflation is a major factor contributing to the increase in the elastic work of inspiration, while increasing tidal volume and decreasing the dynamic compliance of the lung during exercise plays a less important role. Therefore, without appropriate intervention for dynamic hyperinflation, the exercise limitation of these patients can hardly be improved.

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