PATTERNS OF DYNAMIC HYPERINFLATION DURING EXERCISE AND RECOVERY IN PATIENTS WITH SEVERE CHRONIC OBSTRUCTIVE PULMONARY DISEASE

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Abstract

**Background:** Not all patients with severe chronic obstructive pulmonary disease (COPD) progressively hyperinflate during symptom-limited exercise. We investigated the pattern of change in chest wall volumes (Vcw) in patients with severe COPD who progressively hyperinflate during exercise and those who do not.

**Methods:** Twenty patients with FEV1 of 35 (2) % predicted were studied during a ramp-incremental cycling test to the limit of tolerance (Wpeak). Changes in Vcw at the end of expiration (EEVcw), inspiration (EIVcw) and at total lung capacity (TLCVcw) were computed by Optoelectronic Plethysmography (OEP) during exercise and recovery.

**Results:** Two significantly different patterns of change in EEVcw were observed during exercise in our patients. Twelve patients exhibited a progressive significant increase in EEVcw during exercise (early hyperinflators, EH) amounting to 750 (90) ml at Wpeak. In contrast, in all 8 remaining patients EEVcw remained unchanged up to 66% Wpeak, whilst it significantly increased by 210 (80) ml at Wpeak (late hyperinflators, LH). Although at the limit of tolerance the increase in EEVcw was significantly greater in EH, both groups reached similar Wpeak and breathed with a tidal EIVcw that closely approached TLCVcw: [EIVcw/TLCVcw (%): 93 (1) and 93 (3), respectively]. EEVcw was increased by 254 (130) ml above baseline 3-min post-exercise only in EH.

**Conclusions:** Our patients with severe COPD exhibited two patterns during exercise, namely early and late hyperinflation. In the EH group, hyperinflation may take several minutes before is fully reversed after termination of exercise.

**Key words:** COPD, exercise tolerance, dynamic hyperinflation
Introduction

Progressive dynamic hyperinflation (DH) leads to intolerable sensations of breathlessness that contribute importantly to the limitation of symptom-limited exercise in the majority of patients with chronic obstructive pulmonary disease (COPD).[1] In these patients changes in end-expiratory lung volume (EELV) constitute an important outcome in assessing the effects of therapeutic interventions on the development of DH during exercise.[2][3][4] Today, assessment of dynamic changes in EELV is routinely carried out by serial inspiratory capacity (IC) manoeuvres [5][6] assuming that in patients with COPD total lung capacity (TLC) does not change appreciably during exercise. [7] [8]

On the other hand, there is also a significant number of COPD patients who do not progressively hyperinflate during exercise [6] [9][10][11][12] but still claim dyspnoea as the main cause of exercise limitation. [6] The results reported for this category of COPD patients are, however, discrepant as EELV has either been reported to remain constant with increasing intensity [12] or actually fall [11], as commonly seen in healthy subjects. [13] Accordingly, exercise limitation in these patients is not associated with end-expiratory DH. This implies that simply tracking changes in EELV during exercise is not informative of all the factors that intensify dyspnoea and reduce exercise capacity in these patients. In COPD patients there is also variability in the response of the end-inspiratory lung volume (EILV) to exercise: most studies report a progressive increase in EILV [1] [5] [6] [12], while Aliverti and co-workers [11] have found that some patients exhibit a stable EILV. Assessment of all dynamically modified operational lung volumes during exercise is, therefore, important for understanding which factors contribute to exercise limitation. Accordingly, the present study was primarily undertaken in order to identify possible differences in the pattern of response in operational volumes during exercise in patients with severe COPD.

Optoelectronic Plethysmography (OEP) is a technique capable of accurately measuring breath-by-breath changes in the volumes of the entire chest wall (V_CW), and its rib cage and abdominal chest wall compartments. [13][14][15] In addition, OEP can measure breath-by-breath variations in end-inspiratory and end-expiratory V_CW and volume variations of the different chest wall compartments. These measures are crucial for the understanding of the different ventilatory strategies adopted during exercise between different patients. Additionally, OEP can track any changes in V_CW at TLC (V_CW,TLC) if maximal inspirations are repeatedly made during exercise. Thus, one can determine if tidal volume is restricted when end-inspiratory volume is at or near TLC. As the literature is lacking research investigating changes in operational lung volumes following the cessation of exhaustive exercise in patients with COPD, we additionally investigated the pattern of change in V_CW during recovery from exercise, since this could be an important issue for the patients when dealing with activities of daily living.
Materials and Methods

Subjects

Patients included 15 men and 5 women with stable COPD who satisfied the following criteria: 1) post-bronchodilator FEV₁ <50% predicted and FEV₁/FVC <65% without significant reversibility (< 12% change of the initial FEV₁ value), 2) optimized medical therapy and 3) no clinical evidence of exercise-limiting cardiovascular or neuromuscular diseases. Patients signed an informed consent and the protocol was approved by the University Ethics Committee.

Pulmonary function assessment

Spirometry and lung diffusion capacity for carbon monoxide (TLCO) were performed by a Spirometer (Masterlab; Jaeger, Wurzburg, Germany), whereas subdivisions of lung volumes were measured by body plethysmography (Medgraphic Autolink 1085D, Medical Graphics, St Paul, MN) according to ATS standards.[16]

Exercise Protocol

The following incremental protocol was performed on an electromagnetically braked cycle ergometer (Ergoline 800; Sensor Medics, Anaheim, CA): after 3-min of measurements during quiet breathing (QB), followed by 3-min of unloaded pedalling, the work rate was increased every min (increments of 5 or 10 W) to the limit of tolerance (Wpeak) while patients maintained a pedalling frequency of 60 rpm. The following gas exchange and ventilatory variables were recorded breath-by-breath (Vmax 229; Sensor Medics, Anaheim, CA): oxygen uptake ($\Delta$O₂), carbon dioxide output ($\Delta$CO₂), respiratory exchange ratio (RER), minute ventilation (VE), tidal volume (VT), and breathing frequency (fB). Cardiac frequency (fc) and percentage oxygen saturation (SpO₂%) were determined using the R-R interval from a 12-lead on line electrocardiogram (Marquette Max; Marquette Hellige GmbH, Germany) and a pulse oximeter (Nonin 8600; Nonin Medical, USA), respectively. The modified Borg Scale [17] was used to rate the magnitude of dyspnoea and leg discomfort every 2-min throughout exercise.

Operational lung and chest wall volume measurements

At baseline, during unloaded cycling and incremental exercise, patients performed IC manoeuvres at QB, every 2 min during exercise and in recovery. Patients were instructed after three to four regular tidal breaths to make maximal IC efforts from EELV to TLC according to previously described methods. [1] Simultaneously chest wall kinematics were measured by OEP as previously described.[13][14][15] In brief, the movement of 89 retro-reflective markers placed front and back over the chest wall was recorded. Each marker was tracked by six video cameras (Smart system BTS, Milan), three in front of the subject and three behind (Diagram 1). Subjects grasped handles positioned at mid-sternum level which lifted the arms away from the rib cage, so that lateral markers could be visualized. Dedicated software reconstructs the 3D co-ordinates of the markers in real time by stereophotogrammetry and calculates total and compartmental chest wall volume and volume variations by using Gauss’s theorem. As in the study by Aliverti and co-workers [11] the chest wall was modelled as being composed of two compartments: the rib cage and the abdomen. $V_{CW}$ was the sum of the rib cage volume ($V_{rc}$) and abdominal volume ($V_{ab}$). [11] $V_{CW}$ data are reported during QB, unloaded cycling (0 Watts), at 33, 66 and 100% of peak exercise workload (Wpeak) and one (R1) and three minutes (R2) into the recovery.

Comparison of OEP with spirometric data

$V_T$ measured by the OEP ($V_{T\ OEP}$) was calculated as the difference between end-inspiratory and end-expiratory $V_{CW}$ volume (i.e.: EIVcw - EEVcw). As in a previous study [15] in which we assessed the ability of the OEP to measure changes in lung volumes during exercise, we compared $V_{T\ OEP}$ to $V_T$ obtained spirometrically ($V_{T\ SP}$) over periods of 20-s throughout all stages. IC was calculated by the OEP ($IC_{OEP}$) as the difference between TLCVcw and the EEVcw; the latter value was derived by averaging the EEVcw over a period of 20-s prior to the IC effort (fig 1). $IC_{OEP}$ and EEVcw values recorded at QB, exercise and
recovery were compared to IC measured by the spirometer (IC_SP) according to previously described methods. [1]

**Statistical Analysis**

Data are presented as means (SE). Linear regression analysis was performed using the least square method. Two-way analysis of variance (ANOVA) with repeated measures was used to identify statistically significant differences in chest wall volumes across different time points between groups. Within groups one way ANOVA with repeated measures was performed to examine statistical differences, followed by paired t-tests when necessary. For all analyses a statistical significance of 0.05 was used, with appropriate Bonferroni corrections for multiple comparisons.
**Results**

*Patient characteristics*

Patients were characterized by severe airway obstruction and reduction of TLCO, with increased TLC, FRC and RV (Table 1). Exercise capacity was severely compromised (Table 2).

**Table 1** Demographic and post-bronchodilator data of the study population and subgroups

<table>
<thead>
<tr>
<th></th>
<th>COPD (n=20)</th>
<th>Early hyperinflators (n=12)</th>
<th>Late hyperinflators (n=8)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>62 (2)</td>
<td>61 (3)</td>
<td>64 (2)</td>
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<tr>
<td>Height (cm)</td>
<td>167 (2)</td>
<td>168 (3)</td>
<td>165 (3)</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>66 (2)</td>
<td>65 (3)</td>
<td>67 (4)</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>23.7 (0.7)</td>
<td>22.9 (1.0)</td>
<td>24.7 (0.7)</td>
</tr>
<tr>
<td>FEV₁ (L)</td>
<td>0.94 (0.07)</td>
<td>0.93 (0.10)</td>
<td>0.94 (0.09)</td>
</tr>
<tr>
<td>FEV₁ (%pred)</td>
<td>35 (2)</td>
<td>33 (4)</td>
<td>37 (7)</td>
</tr>
<tr>
<td>FVC (L)</td>
<td>2.70 (0.13)</td>
<td>2.65 (0.19)</td>
<td>2.76 (0.15)</td>
</tr>
<tr>
<td>FVC (%pred)</td>
<td>79 (4)</td>
<td>76 (5)</td>
<td>84 (5)</td>
</tr>
<tr>
<td>FEV₁/FVC (%)</td>
<td>35 (2)</td>
<td>35 (3)</td>
<td>35 (3)</td>
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<tr>
<td>TLCO (%)</td>
<td>43 (6)</td>
<td>37 (4)</td>
<td>49 (13)</td>
</tr>
<tr>
<td>TLC (%pred)</td>
<td>120 (22)</td>
<td>123 (13)</td>
<td>119 (16)</td>
</tr>
<tr>
<td>FRC (%pred)</td>
<td>156 (14)</td>
<td>157 (11)</td>
<td>149 (10)</td>
</tr>
<tr>
<td>RV (%pred)</td>
<td>216 (10)</td>
<td>220 (12)</td>
<td>212 (9)</td>
</tr>
<tr>
<td>IC (L)</td>
<td>2.07 (0.08)</td>
<td>2.03 (0.12)</td>
<td>2.15 (0.08)</td>
</tr>
<tr>
<td>IC (%pred)</td>
<td>70 (4)</td>
<td>69 (7)</td>
<td>71 (5)</td>
</tr>
</tbody>
</table>

Values are means (SE).
<table>
<thead>
<tr>
<th></th>
<th>COPD (n=20)</th>
<th>Early hyperinflators (n=12)</th>
<th>Late hyperinflators (n=8)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Wpeak (Watt)</td>
<td>45 (4)</td>
<td>44 (7)</td>
<td>47 (5)</td>
</tr>
<tr>
<td>Wpeak (%pred)</td>
<td>37 (3)</td>
<td>34 (4)</td>
<td>40 (5)</td>
</tr>
<tr>
<td>Exercise Tolerance (min)</td>
<td>6.7 (0.4)</td>
<td>6.4 (0.5)</td>
<td>7.2 (0.6)</td>
</tr>
<tr>
<td>VO₂ (L/min)</td>
<td>0.83 (0.06)</td>
<td>0.81 (0.09)</td>
<td>0.84 (0.08)</td>
</tr>
<tr>
<td>VO₂ (%pred)</td>
<td>50 (4)</td>
<td>47 (5)</td>
<td>53 (7)</td>
</tr>
<tr>
<td>RER</td>
<td>1.06 (0.05)</td>
<td>1.08 (0.02)</td>
<td>1.07 (0.07)</td>
</tr>
<tr>
<td>fc (beats/min)</td>
<td>115 (2)</td>
<td>114 (4)</td>
<td>116 (5)</td>
</tr>
<tr>
<td>fc (%pred)</td>
<td>73 (2)</td>
<td>72 (2)</td>
<td>75 (5)</td>
</tr>
<tr>
<td>SpO₂ (%)</td>
<td>92 (1)</td>
<td>93 (2)</td>
<td>91 (1)</td>
</tr>
<tr>
<td>Ve (L/min)</td>
<td>30.8 (2.0)</td>
<td>31.1 (2.6)</td>
<td>30.1 (2.7)</td>
</tr>
<tr>
<td>VT (L)</td>
<td>1.24 (0.08)</td>
<td>1.22 (0.10)</td>
<td>1.28 (0.12)</td>
</tr>
<tr>
<td>fb (breaths/min)</td>
<td>26 (2)</td>
<td>26 (3)</td>
<td>25 (2)</td>
</tr>
<tr>
<td>Dyspnoea (Borg)</td>
<td>4.1 (0.3)</td>
<td>4.1 (0.5)</td>
<td>3.6 (0.3)</td>
</tr>
<tr>
<td>Leg fatigue (Borg)</td>
<td>4.1 (0.3)</td>
<td>4.3 (0.3)</td>
<td>3.4 (0.7)</td>
</tr>
<tr>
<td>ΔEIVcw (L)</td>
<td>0.97 (0.10)</td>
<td>1.17 (0.17)</td>
<td>0.66 (0.08) *</td>
</tr>
<tr>
<td>ΔEEVcw (L)</td>
<td>0.53 (0.09)</td>
<td>0.75 (0.09)</td>
<td>0.21 (0.08) *</td>
</tr>
<tr>
<td>VT OEP/IC OEP (%)</td>
<td>86 (2)</td>
<td>88 (2)</td>
<td>83 (6)</td>
</tr>
<tr>
<td>IRVcw (L)</td>
<td>0.19 (0.04)</td>
<td>0.14 (0.05)</td>
<td>0.26 (0.08)</td>
</tr>
<tr>
<td>IRVcw/ TLCVcw (%)</td>
<td>7 (1)</td>
<td>7 (1)</td>
<td>7 (3)</td>
</tr>
</tbody>
</table>

Values are means (SE). Delta indicates changes from quiet breathing. Asterisks indicate significant differences between groups.
Comparison of OEP with spirometric data

The relationship between $V_{T\text{OEP}}$ and $V_{T\text{SP}}$ calculated simultaneously over a period of 20-s during quiet breathing, exercise and recovery, is shown in fig 2. The linear regression analysis yielded the following equation: $V_{T\text{OEP}} = 1.20 V_{T\text{SP}} - 0.18$ ($r^2=0.97$, $p<0.001$). The mean percentage difference between $V_{T\text{OEP}}$ and $V_{T\text{SP}}$ was -2.8 (1.2) % or 31(14) ml. The difference between the two systems at maximum workload (100%peak) was 8.4 (4.5) % or 93 (17) ml, with the $V_{T\text{OEP}}$ values being larger.

Changes in IC from quiet breathing measured by the spirometer ($\Delta IC_{SP}$) were in good relationship with the EEVcw calculated by the OEP ($\Delta EEVcw_{OEP}$) throughout exercise and recovery (fig 3 top panel). The linear regression analysis provided the following equation: $\Delta EEVcw_{OEP} = 0.82 \Delta IC_{SP} +0.03$ ($r^2=0.91$, $p<0.001$). The mean percentage difference between $\Delta EEVcw_{OEP}$ and $\Delta IC_{SP}$ throughout all stages was 7.0 (5.8) % or 35 (24) ml.

In addition, a close correlation was found between IC OEP and IC SP throughout all stages (fig 3 bottom panel). The linear regression analysis provided the following equation: 

$$IC_{OEP} = 0.65 IC_{SP} +0.52$$

($r^2=0.89$, $p<0.001$). The mean percentage difference throughout all stages between IC OEP and IC SP was 3.8 (1.8) % or 73 (32) ml.

Changes in operational $V_{CW}$ during exercise and recovery

Two significantly different patterns of change in EEVcw were observed during exercise in our patients (fig 4). Twelve patients exhibited a progressive significant increase in EEVcw during exercise (early hyperinflators, EH) amounting to 750 (90) ml at Wpeak (fig 4, top right panel). In contrast, in all 8 remaining patients EEVcw remained unchanged from QB up to 66% Wpeak (fig 4, bottom right panel). In the latter group EEVcw was, however, significantly increased by 210 (80) ml compared to QB at Wpeak (late hyperinflators, LH).

Similarly, groups differ in terms of the recovery pattern since in the EH group 3-min into the recovery EEVcw was still higher compared to QB by 254 (130) ml. In contrast, in the LH group the EEVcw had returned to QB within 3-min of the recovery (fig 4).

In the EH group TLCVcw increased, albeit not significantly, compared to QB from 66% Wpeak onwards (fig 4). At Wpeak the increase in TLCVcw from QB amounted to 198 (95) ml, corresponding to an increase of 0.8 (0.1)% of TLCVcw measured at QB. At the first min of recovery, TLCVcw was still higher than during QB [by 153 (64) ml], whereas by the third min of recovery, TLCVcw reached values very close to QB (fig 4). Similarly, in the LH group TLCVcw increased by 72 (25) ml at Wpeak [0.3 (0.1) % of TLCVcw at QB] but it did not differ significantly to that recorded during QB (fig 4).

The pattern of change in EIVcw during exercise did not differ among groups. EIVcw increased significantly throughout exercise and remained significantly higher compared to QB during recovery (fig 4). At Wpeak patients in both groups breathed with a tidal EIVcw that closely approached TLCVcw (Table 2), thus restricting further expansion of $V_{T\text{OEP}}$. During exercise, $V_E$ and $V_{T\text{OEP}}$ in the EH group tended to be higher than in the LH group. Nevertheless, volume constraints on $V_{T\text{OEP}}$ expansion ($V_{T\text{OEP}}/IC_{OEP}$, IRVcw/TLCVcw) were similar between groups, whereas inspiratory reserve $V_{CW}$ (IRVcw) reached the same level in both groups (Table 2). Symptoms of dyspnoea and leg discomfort were not different between groups. Neither resting lung volumes, nor peak exercise workload or gas exchange were significantly different among groups (Tables 1 and 2).

Compartmental tidal volumes

The volume variations for the abdominal compartment were significantly different between groups during exercise and recovery (fig 4, middle panels). In the EH group the increase in EEVcw with increasing work rate was almost entirely attributable to the significant increase in end-expiratory Vrc with no significant contribution from Vab (fig 4, top left and middle panels). In contrast, in the LH group there was no significant change in EEVcw up to 66% Wpeak; this was attributed to the significant decrease seen in end-
expiratory Vab during exercise (fig 4, bottom middle panels). In the EH group, $V_{T_{OEP}}$ expansion was due to a progressive increase in end-inspiratory Vrc and Vab, whereas $V_{T_{OEP}}$ increase in the LH group was achieved by an increase in end-inspiratory Vrc and a decrease in Vab (fig 4).

In both groups, 3-min into recovery $V_{T_{OEP}}$ remained significantly higher compared to QB mainly as a result of elevated end-inspiratory Vrc in both groups. In contrast, within 3-min of recovery the end-expiratory Vrc in both EH and LH groups was not significantly different compared to QB.

Throughout incremental exercise sensations of dyspnoea and leg discomfort tended to be higher in the EH group than in the LH group (fig 5). However, differences between groups were not significant.
Discussion

The main findings of this study are: (1) In patients with severe COPD there are two distinct patterns of change in the chest wall volume response to exercise: in the EH group EEVcw progressively increases throughout exercise, while in the LH group it remains unchanged up to 66% Wpeak, but increases significantly at Wpeak; (2) Although at the limit of tolerance the increase in EEVcw was significantly greater in the EH than LH group, both reached similar values of Wpeak, IRVcw and dyspnoea; (3) Groups did not differ in terms of resting lung volumes or exercise tolerance measures; (4) Post-exercise the EEVcw did not return to the pre-exercise value by 3-min in the EH group only.

In healthy subjects, in whom expiratory flow limitation (EFL) is absent, EELV decreases progressively during exercise. In contrast, the progressive increase in EELV that is typically observed in patients with severe COPD during exercise is mainly dictated by EFL. Koulouris and colleagues have shown that if EFL is present during resting breathing, any further increase in ventilation during exercise is associated with progressive DH. Therefore, the progressive increase in EEVcw observed in the EH group (12 patients) with increasing exercise level presumably reflects presence of EFL already at rest. On the other hand, 8 of our patients exhibited hyperinflation only at Wpeak. This suggests that in these subjects EFL started only after 66% Wpeak, in line with previous findings which indicated that some COPD patients do not become EFL up to 2/3 of Wpeak. Furthermore, it should be noted that in our LH patients, the end-expiratory Vab decreased progressively in the range of 0 to 66% Wpeak. The decreased end-expiratory Vab during exercise reflected increased abdominal muscle activity. It has been postulated that such a contraction is beneficial because of lengthening of the diaphragm, resulting in improved generation of a negative pleural pressure (better position of the length-tension relationship). Although, end-expiratory Vab was reduced over this exercise range, EEVcw did not change as there was a simultaneous increase in end-expiratory Vrc.

Aliverti et al. have also reported that not all COPD patients hyperinflate during exercise. They studied 20 patients during incremental exercise: 12 were EH similar to the present study, while 8 actually decreased the EEVcw from early exercise. These subjects were, however, different from the LH subjects of the present study and those of Koulouris et al. Their FEV1 averaged 50% of predicted compared to our value of 37%, and their exercise performance was very poor, with a Wpeak of only 20 W compared to 40 W in our LH group. In the present study the overall group of patients is comparable, at least in terms of FEV1, to that reported by Aliverti et al. for the hyperinflators (having an FEV1 of 39% predicted). This is probably the reason why we were not able to identify any “euvolumic” patients as previously described by Aliverti et al.

Furthermore, there were important differences in chest wall kinematics in their non-hyperinflators compared to the LH group we studied. They found no increase in EIVcw as exercise workload increased and hence the increase in VT OEP during exercise was solely due to the decrease in EEVcw, presumably reflecting absence of EFL throughout exercise. In addition, at Wpeak there was a large IRVcw, amounting to approximately 1.3 L, while in our subjects it was only 0.26 L. Neither Koulouris et al., nor O’Donnell et al found patients with such a high inspiratory reserve volume at the limit of tolerance as Aliverti et al. In our study, VT OEP increased with exercise entirely by an increase in EIVcw so that at Wpeak EIVcw was very close to TLCVcw and IRVcw was minimal. We found no decrease in EEVcw and the decrease in end-expiratory Vab was considerably less compared to their patients. It is possible, therefore, that besides the different degree of EFL experienced by patients in the two studies, expiratory muscle recruitment was more in their patients compared to ours, thus leading to a greater work of breathing.
Accordingly, considering the findings of the present study and those by Aliverti et al. [11], it can be suggested that during the natural history of COPD, patients pass through a stage with moderate impairment of expiratory flow rates so that exercise does not impose dynamic hyperinflation. With further disease progression, manifested by a decreasing FEV₁, dynamic hyperinflation might be accompanied by lesser degrees of expiratory muscle recruitment and increased dynamic hyperinflation. Longitudinal studies will be required to determine if this hypothesis is correct.

The present study provides for the first time simultaneous changes in VC at the end of inspiration, expiration and at TLC during symptom-limited exercise in patients with severe COPD. Interestingly, we found that at Wpeak tidal EIVc closely approached TLCVc in both EH and LH. Accordingly, exercise limitation was associated with the fixed mechanical constraint set by the reduced IRVc, rather than the magnitude of the change in EEVc, perse. Hence, the increase in DH during exercise is not the only mechanism limiting exercise capacity in patients with severe COPD.

Furthermore, we observed that at Wpeak, TLCVc in both EH and LH increased, albeit not significantly, from QB [EH: by 198 (95) ml; LH by 72 (25) ml respectively], or [EH: by 0.8 (0.1); LH: by 0.3 (0.1) % of TLCVc measured during QB, respectively]. The magnitude of these changes in TLCVc in both groups during exercise is in agreement with previous suggestions that small changes in TLC may occur during exercise because hyperinflation can cause an increase in lung distensibility. [21] [22] [23] Accordingly, changes in TLCVc tended to be larger in EH possibly because they were more hyperinflated than the LH. It should be noted, however, that changes in chest wall volumes are including changes in gas volume, gas compression, and blood volume. [13] Therefore the progressive increase in TLCVc seen in both groups could be due to all of these factors, which may in turn explain, at least in part, the small discrepancies found between the changes in the volumes recorded at the mouth by the spirometer and those calculated from the chest wall signals (fig 2 and 3).

In the EH group, EEVc was increased by 254 (130) ml above baseline 3-min post-exercise. This is in agreement with that recently reported by O’Donnell et al [24] who found that IC 3-min into the recovery from symptom-limited exercise was greater by 250 (35) ml than at baseline. The present study extends these findings by demonstrating that in the EH group the greater degree of DH and air trapping during exercise should have enhanced the threshold loading mainly of the muscles of rib cage compartment so as during recovery the function of these muscles would take longer to return to baseline. Furthermore, the delayed recovery of DH has important clinical implications when designing rehabilitative exercise training regimes for patients with severe COPD, especially if high-intensity interval exercise is chosen to be implemented.[25][26]

In conclusion, we found that in COPD patients there are two types of responders: those who hyperinflate early in exercise (EH) and those who hyperinflate late (LH). Despite this different pattern, exercise capacity is similar probably reflecting the fact that both groups closely approached their TLC at Wpeak.

This work was supported by the European Community CARED FP5 project (contract n. QLG5-CT-2002-0893) and by Thorax Foundation
Figure Legends

**Diagram 1.** Optoelectronic plethysmography: principle of measurement.

**Figure 1.** Typical experimental tracings of absolute chest wall volume measurements obtained from an early hyperinflator (EH: top panel) and a late hyperinflator patient (LH: bottom panel) during quiet breathing and peak exercise. A gradual shift in volumes during exercise occurred because of an increase in mean end-inspiratory (EI) and mean end-expiratory (EE) chest wall volumes indicated by the dashed line. Chest wall volumes at TLC are indicated by an arrow.

**Figure 2.** Regression line between tidal volume (VT) from the spirometer (VT SP) and the opto-electronic plethysmography (VT OEP) during quiet breathing (QB), exercise and recovery. Each point represents the mean value of all 20 patients. Line of identity is also shown.

**Figure 3.** (Top panel): Regression line between volume changes from quiet breathing (QB) in inspiratory capacity measured by the spirometer (ΔIC SP) and the end-expiratory chest volume calculated by opto-electronic plethysmography (ΔEEVCW OEP) throughout all stages. Each point represents the mean value of all 20 patients. (Bottom panel): Regression line between the inspiratory capacity measured by the spirometer (IC SP) and the inspiratory capacity calculated by the opto-electronic plethysmography (IC OEP) at QB, during exercise and recovery. Each point represents the mean value of all 20 patients.

**Figure 4.** Volumes of the rib cage and the abdominal compartments and of the total chest wall between the early hyperinflators (EY: top panels) and the late hyperinflators (LH: bottom panels) expressed in absolute values during quiet breathing (QB), exercise and recovery (R1 and R2). Open circles indicate end of inspiration, closed circles indicate end of expiration. Triangles indicate chest wall volumes at TLC. Crosses indicate significant differences in time points between groups. Asterisks denote significant differences across levels from QB within groups.

**Figure 5.** Perceptions of dyspnoea (upper panel) and leg discomfort (bottom panel) at quiet breathing (QB) and during exercise between the early (closed triangles) and late hyperinflators (open triangles).
References


Diagram 1. Optoelectronic plethysmography: principle of measurement.