A Pilot Study of Expiratory Flow Limitation and Lung Volume Reduction Surgery*

Ron Dueck, MD; Sheila Cooper, MD; David Kapelanski, MD; Henri Colt, MD, FCCP; and Jack Clausen, MD

**Study objectives:** To examine the relationships between changes in expiratory flow limitation (FL) during anesthesia and postoperative responses to lung volume reduction surgery (LVRS).

**Design:** Prospective consecutive case comparison.

**Setting:** University medical center.

**Patients:** Eight patients with severe emphysema.

**Interventions:** General anesthesia with muscle paralysis and thoracic epidural analgesia were provided for LVRS via median sternotomy.

**Measurements:** FEV₁, functional residual capacity (FRC), and total lung capacity (TLC) were measured preoperatively and 3 months postoperatively. Tidal volume (Vₜ) flow/volume (F/V) curves were obtained with a Pitot-type spirometer. Vₜ, expiratory flow rate at 0.25 Vₜ (V*ₜ,25%), and peak expiratory flow rate (V*ₜ,MAX) were obtained from Vₜ F/V curves to derive V*ₜ,25%/V*ₜ,MAX ratio as a measure of FL.

**Results:** Closed chest Vₜ F/V curves during anesthesia pre-LVRS showed four patients with FL (group A) whose V*ₜ,25%/V*ₜ,MAX ratio was 0.38 ± 0.06 (mean ± SD) and four patients without FL (group B) whose V*ₜ,25%/V*ₜ,MAX ratio was 0.82 ± 0.06 (p = 0.0001). Closed chest post-LVRS V*ₜ,25%/V*ₜ,MAX ratio during anesthesia increased by 0.48 ± 0.08 in group A, compared with a 0.19 ± 0.16 reduction in group B (p = 0.0001). Preoperative FEV₁ was 0.57 ± 0.10 L for group A vs 0.82 ± 0.13 L for group B (p = 0.02). Postoperative FEV₁ increased by 67 ± 40% for group A (p = 0.03) vs 29 ± 21% for group B (not significant). FRC decreased by 33 ± 3% for group A vs 17 ± 5% for group B (p = 0.0007), and FRC/TLC decreased by 0.14 ± 0.05 for group A vs 0.01 ± 0.07 for group B (p = 0.026). Post-LVRS V*ₜ,25%/V*ₜ,MAX ratio change during anesthesia correlated with postoperative reduction in FRC (r² = 0.89, p = 0.0004) and FRC/TLC (r² = 0.52, p = 0.045).

**Conclusion:** Post-LVRS change in V*ₜ,25%/V*ₜ,MAX ratio during anesthesia showed a linear relationship with 3-month postoperative improvement in dynamic hyperinflation. Thus, V*ₜ,25%/V*ₜ,MAX ratio change may help provide valuable insights into the interactions between chest wall recoil, dynamic hyperinflation, and Vₜ flow rates in patients with severe COPD and LVRS.

*(CHEST 1999; 116:1762–1771)*

**Key words:** COPD; dynamic hyperinflation; emphysema; expiratory flow limitation; flow/volume curve; lung volume reduction surgery

**Abbreviations:** V*ₜ,25% = expiratory flow rate when 25% of tidal volume is exhaled; Eₜ = lung elastic recoil; FL = flow limitation; FRC = functional residual capacity; F/V = flow/volume; PEEP = positive end-expiratory pressure; PEEPi = intrinsic PEEP; V*ₜ,MAX = peak expiratory flow rate; LVRS = lung volume reduction surgery; NS = not significant; PFT = pulmonary function test; TLC = total lung capacity; Vₜ = tidal volume; Vₜ1.0/ₜ,% = percent of tidal volume exhaled in 1 s

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Lung volume reduction surgery (LVRS) for severe emphysema can significantly improve maximal expiratory flow rates and ameliorate dyspnea on exertion, probably by reducing airway resistance and by reducing hyperinflation with corresponding improvement in inspiratory muscle function.\textsuperscript{1–4} The benefit derives from increased lung elastic recoil (E\textsubscript{L}) associated with reduced dynamic hyperinflation,\textsuperscript{1–3} outcomes that Hopkin\textsuperscript{5} proposed may be more dependent on the quantity of lung resected than on the severity of distension of these resected areas.

Limitation of tidal volume (V\textsubscript{T}) increases during exercise because of expiratory flow limitation (FL) during tidal breathing is an important factor for exercise limitation in cases of COPD.\textsuperscript{6} LVRS enables increased exercise V\textsubscript{T} as well as work capacity and maximal expiratory flow rates and decreased dynamic hyperinflation and dyspnea during exercise.\textsuperscript{7} Based on the increased V\textsubscript{T} flow rates during exercise after LVRS\textsuperscript{8} and other observations, we suggest that improvement in expiratory FL during tidal breathing is an important outcome of LVRS. Reduced dynamic hyperinflation plays a major role in improving dyspnea after LVRS because of the recognized relationship between dynamic airway compression and sensations of dyspnea. However, we believe that reduced dynamic airway compression during exercise after LVRS may also contribute to reduced dyspnea. The importance of the interrelationships between hyperinflation and limitation of tidal flow has also been demonstrated during methacholine-induced bronchoconstriction\textsuperscript{9} and in the response to bronchodilators.\textsuperscript{10,11}

Expiratory FL has also been recognized as an important determinant of dynamic hyperinflation during assisted ventilation for patients with COPD and helps identify patients who will be difficult to wean.\textsuperscript{12} Chest wall recoil is substantially elevated in such patients.\textsuperscript{13} Because of these interrelationships between the positive pressures exerted by the chest wall during expiration, dynamic hyperinflation, and limitations in tidal flow during rest and exercise, we reasoned that the study of flow/volume (F/V) curves during anesthesia may provide important insights into changes in FL after LVRS. We sought to determine whether the post-LVRS changes in severity of FL during tidal breathing, anesthesia, and muscle paralysis would correlate with postoperative improvement in lung function. Pre- and post-LVRS closed chest V\textsubscript{T} F/V curves during anesthesia were analyzed for the presence and severity of FL. Anesthesia post-LVRS expiratory flow rate and FL changes were then compared with 3-month postoperative static lung volumes (functional residual capacity [FRC], total lung capacity [TLC]) and maximum FEV\textsubscript{1} changes. These findings were used for a preliminary test of the hypothesis that post-LVRS FL improvement during anesthesia with muscle paralysis correlated with 3-month postoperative improvement in dynamic hyperinflation.

**Materials and Methods**

**Participants**

Informed consent was obtained according to University of California, San Diego Internal Review Board guidelines. Eight consecutive patients selected for bilateral LVRS and available for 3-month postoperative pulmonary function tests (PFTs) were included in this pilot study (see Table 1 for anthropometric data). All had severe emphysema (as determined by history, physical examination, radiographic imaging, and PFTs) and severely emphysematous lung regions identifiable on chest radiographs, CT scans, and pulmonary ventilation-perfusion images. PFTs included FVC and static lung volume measurements before and 3 months after bilateral LVRS.

**Anesthesia and Surgery**

A thoracic epidural catheter was placed for intraoperative analgesia with lidocaine and postoperative analgesia with Dilau-

<table>
<thead>
<tr>
<th>Table 1—Subject Anthropometric Data*</th>
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<td>No.</td>
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<td>Group A</td>
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<td>Mean ± SD</td>
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<td>7</td>
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<td>Mean ± SD</td>
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*BMI = body mass index; DLT = double-lumen tube. Participant number refers to the sequence in which patients received LVRS.
Checks for air leaks were made with gradual sustained manual ventilation while surgery was performed on the opposite lung. Lung regions were identifiable, the lung was reinflated and 100% O₂ were provided at a rate of eight breaths per minute. The post-LVRS. V<sub>1</sub>/V<sub>T</sub>,MAX and V<sub>T</sub>/V<sub>R</sub> were measured for each surgical side ventilation. Bovine emphysematous lung tissue identified during each surgical side deflation was removed using a surgical stapling device. Bovine pericardium was used to buttress the suture line in an attempt to prevent air leaks. When no further overstretched lung regions were identifiable, the lung was inflated and ventilated while surgery was performed on the opposite lung. Checks for air leaks were made with gradual sustained manual inflation to 20 cm H₂O after lung immersion in normal saline solution and with comparison of inspired vs expired V<sub>T</sub> on V<sub>T</sub>/V<sub>F</sub> curves.

All patients were extubated after post-LVRS anesthesia V<sub>T</sub> F/V measurements were obtained, after neuromuscular blockade reversal was achieved with the IV administration of glycopyrrolate (0.8 to 1.0 mg) and neostigmine (4.0 to 5.0 mg), and after recovery of upper airway reflexes was achieved.

**PFTs**

Preoperative and 3-month postoperative FVC and FEV₁ were obtained by pneumotach-based spirometry following standards recommended by the American Thoracic Society. FRC and TLC measurements were obtained by variable pressure body plethysmography (E. Jaeger, Inc; Warzburg, Germany) following the guidelines of the California Thoracic Society, except that panting/breathing frequency was approximately one cycle/s to avoid spurious overestimates of plethysmographic volumes.

**Anesthesia V<sub>T</sub> F/V Curves**

A Pitot-type (D-lite sensor; Datex-Ohmeda; Helsinki, Finland) flowmeter was used to measure anesthesia V<sub>T</sub>, V<sub>T</sub>/F<sub>V</sub>, percent of V<sub>T</sub> exhaled in 1.0 s (V<sub>T</sub>1.0/V<sub>T</sub>%, and inspiratory pressure. Calibration procedures were performed using a 1-L syringe (Hans Rudolph; Kansas City, MO) pumping through an identical size double-lumen endobronchial tube, adaptors, and connectors. When water vapor condensation produced visibly apparent water droplets, the D-lite sensor was removed, cleaned, and recalibrated and was then reintegrated into the patient's endotracheal tube adapter. Serial breath V<sub>T</sub>/F<sub>V</sub> curves were recorded during periods in which neither lung nor chest wall surgical manipulation was present, first during general anesthesia with muscle paralysis before sternotomy (pre-LVRS) and then after completion of LVRS, lung reexpansion, and sternotomy closure (post-LVRS). Any V<sub>T</sub>/F<sub>V</sub> loops with artifacts such as an irregular F<sub>V</sub>/pattern, V<sub>T</sub>/V<sub>T</sub>1.0/V<sub>T</sub>%, or airway pressure variation were rejected. The measured parameters for each study condition were then averaged from a minimum of three V<sub>T</sub>/F<sub>V</sub> loops.

### Table 2—Preoperative and 3-Month Postoperative Pulmonary Function Tests*

<table>
<thead>
<tr>
<th>Subject No.</th>
<th>Preop</th>
<th>3 mo Postop</th>
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<td>Group A</td>
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<td>2</td>
<td>0.60</td>
<td>1.13</td>
<td>5.15</td>
<td>3.53</td>
<td>6.63</td>
<td>5.13</td>
<td>0.777</td>
<td>0.688</td>
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<td>4</td>
<td>0.48</td>
<td>0.75</td>
<td>5.59</td>
<td>3.53</td>
<td>6.35</td>
<td>5.22</td>
<td>0.580</td>
<td>0.676</td>
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<td>5</td>
<td>0.62</td>
<td>0.78</td>
<td>6.20</td>
<td>4.26</td>
<td>7.30</td>
<td>5.73</td>
<td>0.849</td>
<td>0.743</td>
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<td>Mean ± SD</td>
<td>0.57 ± 0.10*</td>
<td>0.94 ± 0.20</td>
<td>5.77 ± 0.49</td>
<td>3.84 ± 0.37**</td>
<td>6.55 ± 0.44</td>
<td>5.46 ± 0.33</td>
<td>0.84 ± 0.05*</td>
<td>0.70 ± 0.03</td>
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<td>Pre- vs post-</td>
<td>+67.1 ± 40.4% (p = 0.03)</td>
<td>−33.4 ± 2.6% (p = 0.0004)</td>
<td>−20.3 ± 2.2% (p = 0.0007)</td>
<td>−13.9 ± 5.2% (p = 0.01)</td>
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<td>LVRS change</td>
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<td>1</td>
<td>0.95</td>
<td>1.16</td>
<td>4.86</td>
<td>4.17</td>
<td>6.95</td>
<td>5.82</td>
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<td>3</td>
<td>0.72</td>
<td>0.75</td>
<td>5.56</td>
<td>4.82</td>
<td>7.18</td>
<td>5.69</td>
<td>0.774</td>
<td>0.847</td>
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<tr>
<td>6</td>
<td>0.72</td>
<td>1.09</td>
<td>6.59</td>
<td>5.61</td>
<td>8.28</td>
<td>7.74</td>
<td>0.796</td>
<td>0.725</td>
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<tr>
<td>7</td>
<td>0.92</td>
<td>1.28</td>
<td>7.69</td>
<td>6.15</td>
<td>9.98</td>
<td>8.57</td>
<td>0.784</td>
<td>0.718</td>
</tr>
<tr>
<td>Mean ± SD</td>
<td>0.83 ± 0.13</td>
<td>1.07 ± 0.23</td>
<td>6.21 ± 1.29</td>
<td>5.19 ± 0.87</td>
<td>8.10 ± 1.38</td>
<td>6.96 ± 1.43</td>
<td>0.76 ± 0.04</td>
<td>0.75 ± 0.06</td>
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<td>Pre- vs post-</td>
<td>29.2 ± 20.6% (NS)</td>
<td>−16.0 ± 3.7% (p = 0.02)</td>
<td>−14.4 ± 5.9% (p = 0.01)</td>
<td>−1.2 ± 6.9% (NS)</td>
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<td>LVRS change</td>
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*Preop = preoperative; Postop = postoperative.
The Vt F/V curves were reviewed for evidence of FL, as described by Gottfried et al. They included criteria for evidence of FL, which were sharp initial expiratory flow peak followed by a slow expiratory flow pattern with convexity toward the volume axis (Fig 1). Participants whose closed chest anesthesia pre-LVRS Vt F/V curves satisfied these FL criteria constituted group A, whereas those whose closed chest pre-LVRS Vt F/V curves did not satisfy these FL criteria constituted group B (Fig 2, 3). In addition, we identified “overt auto-positive end-expiratory pressure (PEEP)” as an abrupt interruption of prolonged expiratory flow by onset of inspiratory flow for the next breath (Fig 2, participant 2, pre-LVRS). Post-LVRS disappearance of overt auto-PEEP was defined as a complete fall to zero expiratory flow before the onset of inspiratory flow for the next breath.

Inspiratory and expiratory anesthesia Vt flow rates were derived off-line using computer software (Photoshop, version 3.0; Adobe; Mountain View, CA) from scanned Vt F/V curves. More specifically, X and Y gradations in Photoshop were used to derive the expiratory flow rate at 25% of Vt (Vt\textprime;25%) and the peak expiratory flow rate (Vt\textprime;MAX) (Fig 1); thus, the Vt\textprime;25%/ Vt\textprime;MAX ratio is a measure of FL pre-LVRS and post-LVRS.

Statistical analysis was performed with linear regression, repeated measures analysis of variance for a treatment effect, and factorial analysis of variance for group A vs group B differences, using computer software (Statview SE; SAS Institute; Cary, NC).

**RESULTS**

**Preoperative PFTs**

Preoperative FEV\textsubscript{1} for all eight participants was severely reduced at (mean ± SD) 0.70 ± 0.18 L (20.4 ± 8.5% of predicted FEV\textsubscript{1}). Plethysmographic FRC showed severe hyperinflation at 5.99 ± 0.93 L (207 ± 28% of predicted FRC). Individual data are shown in Table 2. Participants whose anesthesia pre-LVRS Vt F/V curves showed expiratory FL (group A, see below) had an average 0.26 L lower FEV\textsubscript{1} (p = 0.019) and an average 0.13 higher FRC/TLC ratio (p = 0.047) compared with those whose anesthesia Vt F/V curves did not show FL (group B). Preoperative FVC F/V curves did not show consistent shape or pattern differences between groups A and B.

**Anesthesia Closed Chest Pre-LVRS Vt F/V Curves**

Four participants from group A (participants 2, 4, 5, and 8) showed a pre-LVRS FL pattern during anesthesia (Fig 2, left) and failure to reach zero expiratory flow before interruption by the next breath’s inspiratory flow or overt auto-PEEP. Four participants from group B (participants 1, 3, 6, and 7) did not show an FL pattern pre-LVRS and clearly showed expiratory flow falling to zero before the onset of the next breath (no overt auto-PEEP, Fig 3, left).

Peak Vt inspiratory flow rate was 38 ± 5 L/min for group A vs 40 ± 8 L/min for group B, a difference that was not significant (NS). Pre-LVRS Vt1.0/ Vt,% was < 40% of Vt for all group A participants, whereas all group B Vt1.0/Vt,% values were > 40% of Vt (Table 3). The difference in peak expiratory flow rate (Vt\textprime;MAX) for group A vs group B was NS, whereas the Vt\textprime;25% was an average 2.6-fold higher for group B vs group A. Lower pre-LVRS Vt\textprime;25%/ Vt\textprime;MAX ratios were thus consistent with greater expiratory F/V convexity toward the volume axis in group A (more severe FL; Fig 2, 3, Table 3).

**Anesthesia Closed Chest Post-LVRS**

Vt F/V curves showed improved Vt1.0/Vt,% disappearance of the FL pattern, and disappearance of overt auto-PEEP post-LVRS for all four participants in group A (Fig 2, right). One participant in group B (participant 6) did show an FL pattern post-LVRS (Fig 3, right), whereas his pre-LVRS Vt F/V pattern did not show FL. This participant’s post-LVRS Vt1.0/Vt,% was reduced to the group A pre-LVRS Vt1.0/Vt,% range (< 40% of Vt). Thus, our eight participants showed an anesthesia FL pattern only when Vt1.0/Vt,% was < 40% of Vt.

Peak Vt inspiratory flow rate post-LVRS was 32 ± 2 L/min for group A vs 36 ± 1 L/min for group B (p = 0.02), although pre- and post-LVRS inspiratory flow rate differences were NS for either group.

Expiratory flow rates showed substantial improvement for group A participants. Post-LVRS Vt1.0/ Vt,% was 100 ± 43% higher than pre-LVRS for group A, whereas group B showed an average 20 ± 19% reduction in post-LVRS Vt1.0/Vt,% (p = 0.0023). Vt\textprime;25% improved by 176% post-LVRS for group A (Table 3), whereas Vt\textprime;MAX showed a modest NS increase. In contrast, Group B showed a 32% Vt\textprime;25% reduction (significant difference, p = 0.0156) and NS Vt\textprime;MAX change. Thus, the Vt\textprime;25%/ Vt\textprime;MAX ratio improved by 128% for group A vs a 25% reduction for group B (p = 0.0001) (i.e., significantly different FL changes).

Assessment of the reproducibility of our Vt F/V measurements showed breath-to-breath Vt variation of 2.1 ± 0.9% of Vt pre-LVRS, and 1.8 ± 0.7% of Vt post-LVRS (NS difference). Vt1.0/Vt,% showed an average 2.3 ± 1.2% breath-to-breath variation pre-LVRS vs 2.5 ± 3.2% post-LVRS (NS difference). Inspired Vt was an average 6 ± 2 mL higher than expired Vt pre-LVRS (p = 0.25), vs 9 ± 4 mL higher than expired Vt post-LVRS, suggesting that post-LVRS staple line leaks were NS.

**3-Month Postoperative PFTs**

The post-LVRS increase in FEV\textsubscript{1} was significant only for group A (p = 0.03). Group A had an average 33 ± 3% FRC reduction, compared with a 16 ± 4% FRC reduction for group B (p = 0.0002, Table 2),
whereas post-LVRS change in FEV₁ and FRC showed modest correlation ($r² = 0.56$, $p = 0.03$). In addition, group A showed an average $0.14 ± 0.05$ FRC/TLC reduction vs a $0.01 ± 0.07$ FRC/TLC reduction for group B ($p = 0.026$).

**Anesthesia Vt F/V and Postoperative PFT Relationships**

Change in Vt1.0/Vt,% during anesthesia with muscle paralysis did not correlate with 3-month...

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**Figure 2. Left panels:** representative anesthesia closed chest pre-LVRS VtF/V curves from the four subjects in group A show inspiratory (negative) and expiratory (positive) flow on the ordinate, plotted clockwise from zero volume on the abscissa. Expiratory flow starts with a sharp upward peak, then falls immediately to a low flow rate with convexity toward the volume axis, suggesting FL and, thus, a low $V'\text{V}_t,25%/V'_\text{V}_t,\text{MAX}$ ratio (see Table 3, group A). Expiratory flow is eventually interrupted by inspiratory flow for the next breath before it falls to zero, suggesting “overt auto-PEEP.” **Right panels:** Anesthesia closed chest post-LVRS VtF/V curves show an improved expiratory flow rate with less convexity toward the volume axis and a higher $V'\text{V}_t,25%/V'_\text{V}_t,\text{MAX}$ ratio, with no FL and no “overt auto-PEEP.”
postoperative change in FEV\(_1\) \((r^2 = 0.45, p = 0.07)\). Similarly, \(V^{\prime}V_t,25%/V^{\prime}V_t,MAX\) ratio change during anesthesia did not correlate with 3-month postoperative change in FEV\(_1\) \((r^2 = 0.16, p = 0.32)\). However, post-LVRS change in \(V_t1.0/V_t,\%\) during anesthesia showed excellent correlation with 3-month postoperative percent FRC reduction \((r^2 = 0.94, p = 0.0001; \text{Fig 4})\) and with FRC/TLC reduction \((r^2\)
5.003; Fig 5). Failure to show improved post-LVRS V\textsubscript{t}1.0/V\textsubscript{t}%, during anesthesia was associated with 15% postoperative FRC reduction. Likewise, V\textsuperscript{*}V\textsubscript{t}25% change during anesthesia showed high correlation with postoperative percent FRC reduction (r\textsuperscript{2} = 0.96, p = 0.0001) and with change in FRC/TLC (r\textsuperscript{2} = 0.52, p = 0.045). Finally, V\textsuperscript{*}V\textsubscript{t}25%/V\textsuperscript{*}V\textsubscript{t},MAX ratio change during anesthesia also showed significant correlation with FRC and FRC/TLC reduction (Fig 6, 7).

**Discussion**

V\textsubscript{t} F/V curves during anesthesia with muscle paralysis showed that four of eight emphysema LVRS patients had an expiratory FL pattern pre-LVRS (group A), whereas four participants did not show an FL pattern (group B). Group A participants had significantly lower V\textsuperscript{*}V\textsubscript{t},25% and V\textsuperscript{*}V\textsubscript{t},25%/V\textsuperscript{*}V\textsubscript{t},MAX ratio pre-LVRS (Table 3). The finding that an FL pattern was seen only in participants with V\textsubscript{t}1.0/V\textsubscript{t},% < 40% was consistent with our observation that pre-LVRS FEV\textsubscript{1} was lower and FRC/TLC was higher in group A. FL on pre-LVRS anesthesia V\textsubscript{t} F/V curves was therefore consistent with more severe dynamic hyperinflation.\textsuperscript{10,22}

The severity of FL appeared appropriately quantified with the V\textsuperscript{*}V\textsubscript{t},25%/V\textsuperscript{*}V\textsubscript{t},MAX ratio (Table 3) to enable regression of post-LVRS FL changes during anesthesia with muscle paralysis and 3-month postoperative static lung volume changes. LVRS provided significantly greater anesthesia expiratory flow rate improvement, as well as significantly greater FRC and FRC/TLC reduction 3 months after LVRS in group A compared with group B participants. Linear regression suggested that V\textsuperscript{*}V\textsubscript{t},25%/V\textsuperscript{*}V\textsubscript{t},MAX ratio changes during anesthesia showed a continuous function related to postoperative FRC and FRC/TLC reduction. We therefore suggest that the distinction between group A vs group B participants as FL vs non-FL based on visual inspection of the expiratory V\textsubscript{t} F/V curve may be arbitrary, whereas the V\textsuperscript{*}V\textsubscript{t},25%/V\textsuperscript{*}V\textsubscript{t},MAX ratio may provide a more reproducible measure of FL.

Selection of patients for this study was not based on their likelihood of having FL vs not being FL. Patients were recruited in a consecutive manner after the decision was made to provide LVRS based...
on the presence of severe chronic dyspnea symptoms, severe expiratory obstruction, and severe hyperinflation.\(^1\) Possible concerns regarding participant selection bias might be raised in that all four participants in group A were female participants. However, there were also two female participants in group B, and we are not aware of a male/female difference in either FL or efficacy of LVRS. We were surprised to note that the four group B participants did not show FL on their anesthetized-paralyzed supine position pre-LVRS \(V_t\) \(F/V\) curves and that these four participants did not show significant improvement during anesthesia post-LVRS.

The assessment of greater severity of expiratory obstruction in group A was supported by the presence of overt auto-PEEP or interruption of prolonged expiratory flow by inspiratory flow for the next breath, despite a ventilator inspiratory/expiratory setting of 1:4.5. Bardoczky et al.\(^{19}\) recently showed that overt auto-PEEP had 78% sensitivity and 91% specificity for intrinsic PEEP (PEEPi). We note that these authors did not find a size of double-lumen endotracheal tube effect on presence

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**Figure 4.** Anesthesia post-LVRS change in \(V_t\)\(^{1.0}/V_t\)\(\%\) showed excellent correlation with 3-month postoperative percentage of FRC reduction (\(y = 7.0255 \times -133.21; \ r^2 = 0.939, p = 0.001\)).

**Figure 6.** Anesthesia post-LVRS change in FL (\(V_tV_{25}\%)/V_t\)\(\text{MAX}\) ratio) showed excellent correlation with 3-month postoperative percentage of FRC reduction (\(y = 0.037 \times -0.739; \ r^2 = 0.889; p = 0.0004\)).

**Figure 5.** Anesthesia post-LVRS change in \(V_t\)\(^{1.0}/V_t\)\(\%\) showed significant inverse correlation with 3-month postoperative FRC/TLC change (\(y = -714.673 \times -13.713; \ r^2 = 0.796; \ p = 0.0029\)).

**Figure 7.** Anesthesia post-LVRS change in \(V_tV_{25}\%/\text{MAX}\) ratio showed significant correlation with the 3-month postoperative change in the FRC/TLC ratio (\(y = 3.077 \times -0.088; \ r^2 = 0.515; \ p = 0.0449\)).

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or absence of overt auto-PEEP, even though they used the same Vt, frequency, and inspiratory flow rates for one-lung vs two-lung ventilation. This observation was consistent with our present findings; female participants who were intubated with a 37-French double lumen endotracheal tube were present in both groups A and B, suggesting that there was not a sufficient extrinsic PEEP effect to prevent showing FL vs non-FL differences.

The disappearance of FL and overt auto-PEEP post-LVRS in group A participants was consistent with increased EL post-LVRS providing increased traction on the intrapulmonary airways to improve airway caliber and conductance and increased driving pressure to improve expiratory flow rate, Vt1.0/ Vt,%.3 In addition, reduced thoracic cage volume, and thus reduced chest wall recoil, may have contributed to reduced dynamic airway compression.3 Thus, the significantly greater post-LVRS V′Vt,25%/ V′Vt,MAX ratio improvement with the disappearance of FL in group A may also reflect a difference in the role of the chest wall, compared with group B, because of the significantly greater FRC reduction in group A. This difference should also be relevant to the loss of overt auto-PEEP post-LVRS in group A, which was consistent with the report presented by Tschernko et al.20 of reduced PEEPi, from 8.4 ± 1.1 cm H2O to 1.1 ± 0.4 cm H2O. However, the disappearance of overt auto-PEEP in our group A participants did not imply complete loss of PEEPi, because Bardoczky et al.21 showed that a small amount of PEEPi may be present even in the absence of visible auto-PEEP on the expiratory Vt F/V curve.

The absence of FL and overt auto-PEEP during anesthesia in group B patients suggests that their pre-LVRS expiratory obstruction was less severe, even though their preoperative forced maximum F/V curves showed no qualitative differences from group A participants. FL might have been more readily revealed in group B with application of negative airway pressure during Vt exhalation.12,22 However, the excellent correlation between anesthesia Vt1.0/ Vt,% and V′Vt,25%/V′Vt,MAX ratio changes with postoperative FRC and FRC/TLC reduction suggests that our measurements of FL during muscle paralysis provided a valid reflection of the mechanisms involved in the improvement of dynamic hyperinflation. Because expiratory obstruction and hyperinflation with emphysema are caused by severely reduced EL, we presume that pre-LVRS EL was higher in group B than in group A. This is consistent with the recent report by Barnas et al.,23 who observed the greatest increases in EL and lung resistance post-LVRS for participants with the highest preoperative FEV1, maximum voluntary ventilation, and lowest residual volume. Furthermore, because general anesthesia with muscle paralysis is associated with increased EL,24 we speculate that the increased EL during anesthesia with muscle paralysis may have been sufficient to enhance expiratory flow rate and thereby prevent FL on pre-LVRS anesthesia Vt F/V curves for group B.

**Conclusion**

We herein introduce a new measure of expiratory FL during Vt spirometry, the V′Vt,25%/V′Vt,MAX ratio. The eight participants in this pilot study showed a continuous relationship between their anesthesia post-LVRS change in V′Vt,25%/V′Vt,MAX ratio and postoperative improvement in dynamic hyperinflation. Our preliminary findings suggest that the V′Vt,25%/V′Vt,MAX ratio may help provide valuable insights into the mechanisms of improved lung function after LVRS. Further studies are warranted to confirm these observations and to examine their relationship to PEEPi, as well as pre- and postoperative measures of chronic dyspnea.

**References**

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