

Lung Function 4 Years After Lung Volume Reduction Surgery for Emphysema*

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Study objectives: Current data for patients > 2 years after lung volume reduction surgery (LVRS) for emphysema is limited. This prospective study evaluates pre-LVRS baseline data and provides long-term results in 26 patients.

Intervention: Bilateral targeted upper lobe stapled LVRS using video thoracoscopy was performed in 26 symptomatic patients (18 men) aged 67 ± 6 years (mean \pm SD) with severe and heterogenous distribution of emphysema on lung CT. Lung function studies were measured before and up to 4 years after LVRS unless death intervened.

Results: No patients were lost to follow-up. Baseline FEV₁ was 0.7 ± 0.2 L, $29 \pm 10\%$ predicted; FVC, 2.1 ± 0.6 L, $58 \pm 14\%$ predicted (mean \pm SD); maximum oxygen consumption, 5.7 ± 3.8 mL/min/kg (normal, > 18 mL/min/kg); dyspneic class ≥ 3 (able to walk ≤ 100 yards) and oxygen dependence part- or full-time in 18 patients. Following LVRS, mortality due to respiratory failure at 1, 2, 3, and 4 years was 4%, 19%, 31%, and 46%, respectively. At 1, 2, 3, and 4 years after LVRS, an increase above baseline for FEV₁ > 200 mL and/or FVC > 400 mL was noted in 73%, 46%, 35%, and 27% of patients, respectively; a decrease in dyspnea grade ≥ 1 in 88%, 69%, 46%, and 27% of patients, respectively; and elimination of oxygen dependence in 78%, 50%, 33%, and 22% of patients, respectively. The mechanism for expiratory airflow improvement was accounted for by the increase in both lung elastic recoil and small airway intraluminal caliber and reduction in hyperinflation. Only FVC and vital capacity (VC) of all preoperative lung function studies could identify the 9 patients with significant physiologic improvement at > 3 years after LVRS, respectively, from 10 patients who responded ≤ 2 years and died within 4 years ($p < 0.01$).

Conclusions: Bilateral LVRS provides clinical and physiologic improvement for > 3 years in 9 of 26 patients with emphysema primarily due to both increased lung elastic recoil and small airway caliber and decreased hyperinflation. The 9 patients had VC and FVC greater at baseline ($p < 0.01$) when compared to 10 short-term responders who died < 4 years after LVRS.

(CHEST 1999; 116:1608-1615)

Key words: emphysema; lung elastic recoil; lung function; lung volume reduction surgery; transdiaphragmatic pressures

Abbreviations: DLCO = diffusing capacity of the lung for carbon monoxide; Gs = conductance of the small airway S segment; LVRS = lung volume reduction surgery; MEFV = maximum expiratory flow volume; MFSR = maximum expiratory airflow-static lung elastic recoil pressure; RV = residual volume; TLC = total lung capacity; VC = vital capacity

Following targeted bilateral stapled lung volume reduction surgery (LVRS) for emphysema, with and without α_1 -antitrypsin deficiency, variable im-

provement in relief of dyspnea, exercise tolerance, oxygen use and lung function, and mortality has been noted for 2 years following surgery.¹⁻⁶ Beyond 2 years after LVRS, there is very limited experience. The 2-year post-LVRS results are in contrast to the progressive deterioration in lung function in similar patients originally accepted, but denied LVRS by Medicare and followed for ≥ 2 years.⁶ Furthermore, historical data of patients with severe expiratory airflow limitation due to emphysema and FEV₁ < 0.75 L or 30% predicted indicates survival of 50 to 60% at 3 years.^{7,8} Additionally, patients admitted to an ICU for exacerbation of COPD have a 1-year mortality rate of 30% irrespective of the need for endotracheal intubation and mechanical ventilation; in patients > 65 years old, the mortality rate at 1 year doubles.⁹

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Supported by Department of Energy Grant No. DE-FG03-91ER61227, American Lung Association Grant No. CI-030-N, and California Tobacco Related Disease Research Program Grant No. 6RT-0158.

Manuscript received June 25, 1999; revision accepted September 9, 1999.

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The present study prospectively evaluates annual clinical as well as physiologic changes in lung function, including mechanisms of expiratory airflow limitation following LVRS for non- α_1 -antitrypsin emphysema. Results indicate significant clinical and physiologic improvement in lung function in 9 of 26 patients and 7 of 26 patients at 3 years and at 4 years after LVRS, respectively. However, of all preoperative lung function tests, only vital capacity (VC) and FVC could identify these 9 long-term patients from 10 others who only had improvement for ≤ 2 years and died. The improvement in expiratory airflow and hyperinflation is related to the increase in lung elastic recoil pressure and its secondary effect on increasing small airways diameter.

MATERIALS AND METHODS

Patient Selection

The emphysematous patients were markedly symptomatic with grade ≥ 3 dyspnea¹⁰ (able to walk ≤ 100 yards), who had exhausted all medical therapy including antibiotics, aerosol and systemic bronchodilators (including β_2 -agonists and ipratropium bromide), aerosol and systemic corticosteroids, and repeated attempts at physical conditioning. As previously noted, high-resolution, thin-section CT of the lung demonstrated emphysema severity scores¹¹ ≥ 60 with heterogenous distribution, *ie*, severe emphysematous destruction predominantly in upper and middle lung fields. Nuclear medicine perfusion scans demonstrated similar heterogenous distribution. Smoking history was 52 ± 13 pack-years (mean \pm SD).

Operative Technique

From January to June 1995, after obtaining informed consent, 82 patients underwent sequential, bilateral stapled lung volume reduction for emphysema using video-assisted thoracoscopic surgery at the same sitting. The surgical technique and selection has been previously reported.^{2,5,12} It was estimated that approximately 20 to 30% of each lung was excised, and the resected lung weighed 30 to 90 g. Twenty-six of the 82 patients agreed to undergo additional studies, including lung elastic recoil, preoperatively and form the basis of this prospective study.

Lung Function Studies

As previously reported,² we obtained informed consent and measured lung function, including maximum inspiratory and maximum expiratory flow-volume (MEFV) curves, thoracic gas volume, airway resistance, single-breath diffusing capacity of the lung for carbon monoxide (DLCO), and static lung elastic recoil when the patients were clinically stable, using a pressure-compensated flow plethysmograph (Model 6200 Autobox; SensorMedics; Yorba Linda, CA), and compared the results with predicted values.² During panting, the cheeks were supported and the frequency was set at ≤ 1 Hz to avoid mouth pressure from spuriously underestimating alveolar pressure^{13,14} and overestimating thoracic gas volume. All studies were done after three inhalations of aerosolized albuterol, 670 μ g.

As previously noted,^{2,15} measurements of static lung elastic

recoil pressures were obtained in the open plethysmograph with the patient in a sitting position after placement of a 10-cm-long balloon inflated with 0.5 mL of air, initially in the stomach and withdrawn into the lower third of the esophagus. After at least two inspirations to total lung capacity (TLC), static transpulmonary (mouth-esophageal) pressures were recorded following stepwise, 3-s interruptions of exhalation against a closed shutter at different lung volumes, and the expired volume was measured at the mouth. A minimum of five deflation curves were obtained for each patient, and a plot of best visual fit of the pooled data was drawn. Balloon position and volume were similar in each patient before and after LVRS.

Expiratory airflow limitation as seen in the MEFV curves does not distinguish whether the source is primarily due to loss of lung elastic recoil (emphysema),^{16,17} intrinsic small airway disease,^{16,17} or asthma,¹⁵ with no significant loss of elastic recoil or both. Therefore, to determine the mechanisms of expiratory airflow limitation in COPD before and after LVRS, we plotted the maximum expiratory airflow obtained from the MEFV curve against static lung elastic recoil pressure at corresponding lung volumes and constructed maximum expiratory airflow-static lung elastic recoil pressure (MFSR) curves^{2,15-17,19,20}. The slope of the MFSR curve between 50% and 25% of the FVC represents the conductance of the small airway S segment (Gs)¹⁹ and provides quantitative assessment of small airway caliber. Normal values were obtained previously in seven healthy subjects 61 to 74 years old in whom the Gs was 0.6 ± 0.1 L/s/cm H₂O (mean \pm SD) and static lung elastic recoil pressure at TLC was 25 ± 7 cm H₂O.²¹

Exercise Studies

As previously described,² progressive exercise testing to symptom-limited maximum was obtained using electronically braked cycle ergometry (Ergometrics 800; Sensor Medics; Yorba Linda, CA) with increases of 10 to 20 W at 2-min intervals at a pedaling cycle of 40 to 50 revolutions per minute. The patients breathed room air or oxygen-enriched air through a mouthpiece with nose clips using a low-resistance two-way nonrebreathing valve. Expired gases were collected and analyzed using a pulmonary function analyzer (Vmax 29; SensorMedics).

Follow-up

All patient were followed for up to 4 years after LVRS unless death intervened. No patient was lost to follow-up.

Statistical Methods

Comparison of differences between patient groups included paired and unpaired *t* tests, and analysis of variance was tested using a statistical software package (Systat 7.0 for Windows; SPSS; Chicago, IL). Values were considered significant at $p < 0.05$.

RESULTS

The results of preoperative lung function studies in the 26 patients (18 men) aged 67 ± 6 years (mean \pm SD) are reported in Table 1. Preoperative spirometry, lung volumes, and DLCO in the 26 patients were not significantly ($p > 0.05$) different from the other 56 patients (data not shown) who underwent LVRS during the same study period, but were not studied in greater detail.⁵

Table 1—Baseline Data on all 26 LVRS Patients; and Pre- and > 3-Yr Post-LVRS Data on 9 Patient Responders (FEV₁ > 0.2 L, FVC > 0.4 L, or Both); and Baseline in 10 Short-term Responders Who Improved ≤ 2 Yr and Died*

Variables	Baseline Data, n = 26	% Predicted or Normal Value	Short term responders		Responders, n = 9	
			Died, n = 10	Pre-LVRS	Pre-LVRS	> 3 Yr Post-LVRS
VC, L	2.4 ± 0.7	67 ± 6%	59 ± 5%‡	73 ± 13%	85 ± 14%†	
FVC, L	2.1 ± 0.6	58 ± 14%	52 ± 4%‡	64 ± 12%	79 ± 13%†	
FEV ₁ , L	0.7 ± 0.2	29 ± 10%	24 ± 9%	33 ± 10%	47 ± 15%†	
TLC, L	8.6 ± 1.8	147 ± 17%	150 ± 15%	146 ± 16%	128 ± 18%†	
RV, L	6.0 ± 1.4	268 ± 46%	275 ± 40%	254 ± 34%	186 ± 42%†	
RV/TLC, %	71 ± 6	176 ± 21%	185 ± 19%	170 ± 17%	141 ± 19%†	
DLVA, mL/min/mm Hg/L	1.1 ± 0.5	29 ± 15%	26 ± 4%	30 ± 21%	51 ± 23%†	
Pst at TLC, cm H ₂ O	11 ± 1.7	25 ± 7	10.8 ± 0.5	10.9 ± 1.9	12.9 ± 1.8†	
Raw, cm H ₂ O/L/s	5.1 ± 1.9	< 2.5	5.7 ± 0.6	5.1 ± 2.0	3.6 ± 1†	
SGaw, L/s/cm H ₂ O/L	0.032 ± 0.01	13 ± 6%	11 ± 2%	17 ± 10%	25 ± 9%†	
Coefficient retraction	1.3 ± 0.4	> 3.10	1.2 ± 0.3	1.2 ± 0.7	2.0 ± 0.6†	
Pst at TLC/TLC, cm H ₂ O/L						
Gs, L/s/cm H ₂ O	0.20 ± 0.10	0.6 ± 0.1	0.18 ± 0.04	0.20 ± 0.11	0.31 ± 0.08†	
ṠO ₂ max mL/kg/min	6.7 ± 3.8	> 18	5.0 ± 2.0	4.5 ± 1.5	9.5 ± 1.4†	
Dyspnea Score	3.2 ± 0.05	0	3.3 ± 0.05	3.2 ± 0.05	2.2 ± 0.05†	

*Data are presented as mean ± SD unless otherwise indicated; DLVA = diffusing capacity per liter of alveolar volume; Pst = static lung elastic recoil pressure; RAW = airway resistance; SGaw = specific airway conductance; ṠO₂max = maximum oxygen consumption.

†p < 0.05 comparing pre- and > 3 year post-LVRS results in nine responders (FEV₁ ≥ 0.2 L, FVC ≥ 0.4 L, or both ≥ 3 years from baseline).

‡Statistical difference (p < 0.01) at baseline pre-LVRS in 10 short-term responders who died within 4 years of LVRS when compared to 9 patient responders. The short-term responders showed physiologic improvement ≤ 2 years after LVRS.

Results in the 26 patients indicate at baseline very severe expiratory airflow limitation, hyperinflation at TLC, reduction in DLCO, loss of static lung elastic recoil at TLC, increase in inspiratory airway resistance, and severe reduction in maximum oxygen consumption. Resting room air oxygen saturation was 86 ± 6% (mean ± SD). Eighteen patients required part- or full-time nasal oxygen supplementation, and there was only a mild increase in resting PaCO₂ (46 ± 9 mm Hg.) All patients were markedly dyspneic with grade 3.2 ± 0.05 (able to walk ≤ 100 yards; mean ± SD).

Actual survival at 0.5, 1, 2, 3, and 4 years after LVRS was 96%, 96%, 81%, 69% and 54%, respectively (See Fig 1). All deaths were related to respiratory failure, although concomitant lung malignancy was noted in two of four patients autopsied. Improvement in FEV₁ > 0.2 L, FVC > 0.4 L, or both was 88%, 73%, 46%, 35%, and 27% respectively, and these patients are considered responders (See Fig 1). Six of nine patients at 3 years and five of seven patients who demonstrated this physiologic improvement at 4 years after LVRS had both FEV₁ > 0.2 L as well as FVC > 0.4 L when compared to baseline values.

In the nine patients with long-term (> 3 years) physiologic improvement (See Table 1), there were only two deaths (at 37 months and 45 months after LVRS). In the other 17 patients, there were 10 deaths at 26 ± 13 months after LVRS (mean ± SD). If we eliminate the one postopera-

tive death at 2 months, the mean survival for the 16 non-long-term physiologic responders was 29 ± 10 months.

There was a decrease in dyspnea grade ≥ 1 in 88%, 69%, 46% and 27% of the 26 patients at 1, 2, 3, and 4 years after LVRS. Oxygen dependence (part- or full-time) initially present in 18 patients was eliminated in 78%, 50%, 33% and 22% of patients at 1, 2, 3, and 4 years after LVRS.

Maximum Expiratory Airflow

At > 3 years after LVRS, we analyzed the mechanism of improvement in expiratory airflow in the nine long-term responder patients who increased FEV₁ > 0.2 L, FVC > 0.4 L, or both following LVRS. Compared to the preoperative baseline, the MEFV demonstrated a reduction in both TLC and residual volume (RV), but more so in the latter, such that FVC increased (See Table 1 and Fig 2). Furthermore, maximum expiratory airflow at any lung volume was increased when compared to the same lung volume prior to LVRS, but was still far below normal values. FEV₁ increased 0.30 ± 0.1 L compared to baseline (mean ± SD). FVC increased 0.48 ± 0.25 L.

Lung Elastic Recoil

Prior to LVRS, all nine patients had a marked reduction in static lung elastic recoil pressure at TLC (10.9 ± 1.9 cm H₂O; See Fig 3). At > 3 years after

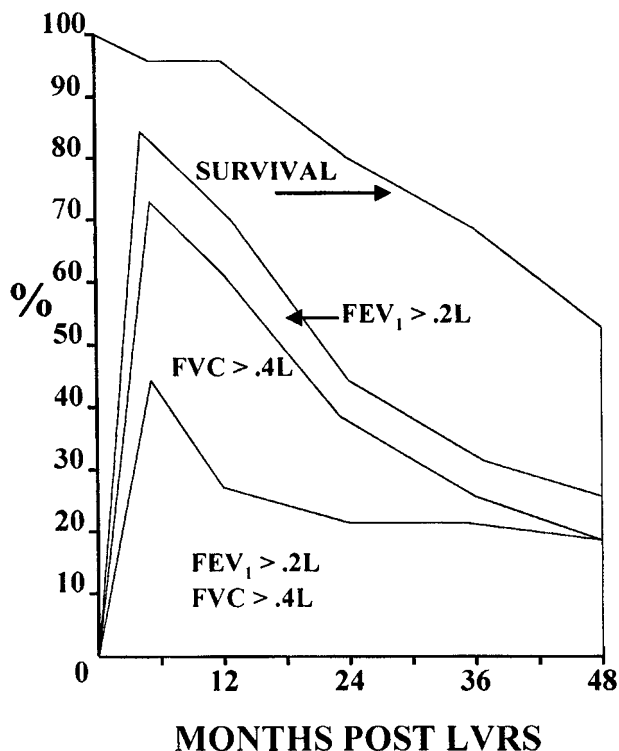


FIGURE 1. Results of survival and lung function studies at 1, 2, 3, and 4 years after LVRS.

LVRS, elastic recoil pressures remained increased at TLC (12.9 ± 1.8 cm H₂O) and at all lung volumes compared to baseline, but were still below normal values.

Mechanism of Expiratory Airflow Limitation

Preoperatively, the slope (Gs) of the MFSR curve was reduced compared to normal²¹ (See Table 1 and

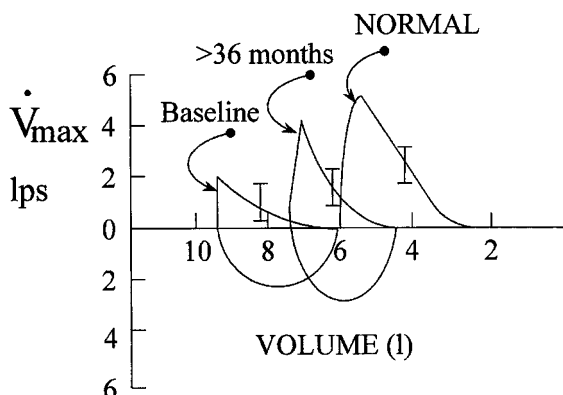


FIGURE 2. In the 9 patients who increased FEV₁ > 0.2 L and/or FVC > 0.4 L or both > 3 years after LVRS, their MEFV curve was shifted to the right, such that both TLC and RV decreased. The decrease in RV was greater, and FVC increased. Maximum expiratory airflow at any lung volume was greater compared to preoperative baseline but was still below age-matched normal subjects.²¹ Bar is mean \pm SD. \dot{V}_{max} = maximum oxygen consumption; lps = liters per second.

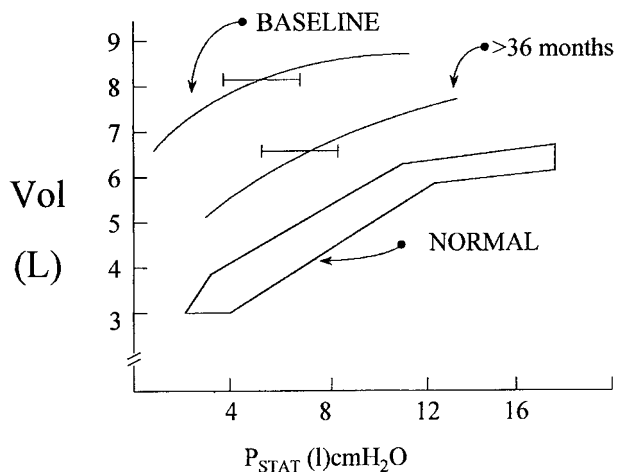


FIGURE 3. In the nine long-term patient responders, results of static lung elastic recoil pressure curve indicated increased lung elastic recoil at any given lung volume compared to baseline but still below age-matched normal subjects.²¹ Bar is mean \pm SD. P_{STAT} = static lung elastic recoil pressure; Vol = volume.

Fig 4). This indicates that maximum expiratory airflow was reduced, not only because of the loss of lung elastic recoil, but also due to suspected intrinsic small airways abnormalities and/or extrinsic collapse/obstruction of small airways. In long-term responders (≥ 3 years after LVRS), maximum expiratory airflow increased, both to greater lung elastic recoil as well as increased conductance of the S-segment

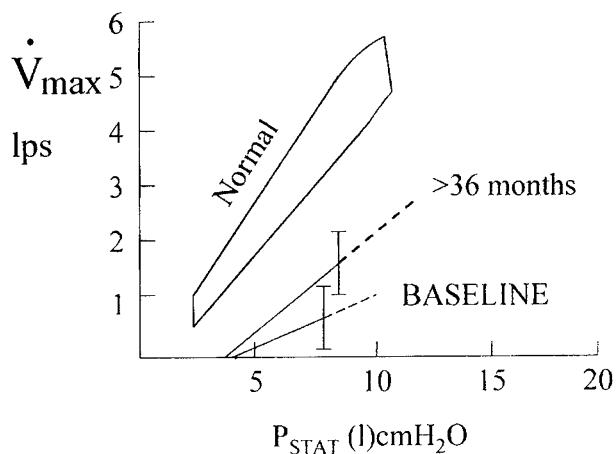


FIGURE 4. In the nine long-term patient responders, the improvement in maximum expiratory flow was due to both an increase in lung elastic recoil as well as increased slope (solid line) of flow-pressure relationships (Gs) of small airways. This indicates increased airway conductance (Gs) that could not be accounted for by the increase in lung elastic recoil and reflects increased airway caliber. The dashed line reflects the extension of Gs determined at effort-independent lung volumes to elastic recoil at TLC. Normal values were previously obtained.²¹ Bar is mean \pm SD. P_{STAT} = static lung elastic recoil pressure. See Figure 2 legend for other abbreviations.

slope, reflecting better airway stability with less collapse/obstruction of flow-limiting segments.

Baseline Physiologic Tests

There were significant differences ($p < 0.01$) for VC and FVC at preoperative baseline between 9 long-term responders at > 3 -year increase in $FEV_1 \geq 0.2$ L, $FVC \geq 0.4$ L, or both from baseline and 10 short-term responders who died within 4 years of LVRS (See Table 1). Short-term responders had increase in $FEV_1 \geq 0.2$ L, $FVC \geq 0.4$ L, or both ≤ 2 years after LVRS.

DISCUSSION

This prospective study, with no patients lost to follow-up, demonstrates that following bilateral LVRS for emphysema, durable clinical and significant physiologic improvement was achieved in 9 of 26 patients at 3 years, and in 7 of 26 patients at 4 years.

These observations, based on very strict outcome criteria, are impressive in elderly patients with end-stage emphysema with a high mortality rate from respiratory failure. Preoperatively, they had very severe airflow limitation, hyperinflation, markedly impaired lifestyle with dyspnea limiting walking < 100 yards, and 18 of 26 patients required part- or full-time oxygen administration.

The mortality rate due to respiratory failure in 4%, 19%, 31%, and 46% of patients following LVRS at 1, 2, 3, and 4 years, respectively, is consistent or lower than previous nonsurgical data in similarly impaired patients with emphysema.⁶⁻⁹ The improvement in expiratory airflow limitation and decrease in hyperinflation is predominantly due to increased lung elastic recoil following LVRS^{2,15} with expansion of remaining lung.

Only VC and FVC of all preoperative clinical, physiologic, perfusion, and lung CT emphysema heterogeneity tests could identify the 9 individual patients who achieved significant improvement at > 3 years after -LVRS from the 10 patients whose physiologic improvement was < 2 years and who died within 4 years of LVRS.

Lung Elastic Recoil and Mechanisms of Airflow Limitation

Expiratory airflow at any given lung volume is directly proportional to the alveolar driving pressure (*ie*, elastic recoil) and inversely proportional to resistance offered by small airways < 2 mm in diameter.^{19,20} Elastic recoil also provides tethering support to small airways to reduce collapse during forced exhalation.

Emphysema destroys the alveolar-capillary surface area of the lung, which results in decreased DLCO and mechanical loss of lung elastic recoil, with increased collapse of small airways during exhalation even in early disease.^{16,17} This causes expiratory airflow limitation often best visualized on MEFV curves.^{16,17} However, there may be concomitant, independent, intrinsic small airway disease,^{16,17,22} especially in chronic cigarette smokers causing similar airflow limitation on MEFV^{11,16,17} curves. We have shown previously in clinically unsuspected emphysema, when the FEV_1 is normal or borderline^{16,17} despite significant parenchymal destruction, the reduction in expiratory airflow was predominantly accounted for by the loss of lung elastic recoil, implying small airway disease did not contribute significantly to airflow limitation. This is in contrast to results in the present study where severe expiratory airflow limitation is due to both loss of lung elastic recoil as well as suspected severe intrinsic small airway disease (reduced Gs). These results are consistent with the pathophysiologic studies by Hogg et al^{11,22,23} in chronic cigarette smokers with far-advanced emphysema.

Elastic Recoil Following Lobectomy and Pneumonectomy

Following lobectomy and pneumonectomy, previous studies²⁴⁻²⁶ have emphasized the reduction in both RV and TLC, but more so in the latter, causing a reduction in both VC and FEV_1 . The lungs become stiffer with decreased compliance over the tidal volume range, and lung elastic recoil at TLC is increased, especially following pneumonectomy. Because of the reduced lung volume, airway resistance increases despite the stiffer lungs. DLCO is maintained after lobectomy, but decreases following pneumonectomy.

Elastic Recoil Following Bullectomy and LVRS

Large lung bullae may occur in the presence or absence of emphysema. Rogers et al²⁷ initially showed long-term improvement in airway conductance as measured by plethysmography, following bullectomy in isolated bullous lung disease without emphysema, and short-term improvement in bullous emphysema. Subsequently, we²⁸ as well as other investigators²⁹⁻³¹ reported that bullectomy in the presence (LVRS equivalent) or absence³² of emphysema reduced hyperinflation and increased expiratory airflow and airway conductance by increasing lung elastic recoil.

By removing nonfunctioning lung, TLC and RV would be similarly reduced, and the lung pressure-volume curve would have a nearly parallel shift to

lower lung volumes. The lung elastic recoil pressure at TLC would be relatively increased. Alternatively, expansion of remaining near-normal functioning lung would result in increased VC, FEV₁, airway conductance, DLCO, and lung elastic recoil at TLC, such that RV would be decreased more than TLC.

Similar mechanisms, but to a lesser extent, were observed following LVRS for severe emphysema with heterogenous distribution, such that the upper half of the lungs were more adversely affected. Scirba et al³³ reported increases in lung elastic recoil at 4 months after unilateral LVRS.

We have previously demonstrated^{2,15,34} that bilateral upper lobe LVRS for generalized nonbullous emphysema increases forced total capacity, FEV₁, expiratory airflow, and airway conductance, and reduces hyperinflation at TLC, all due to real increases in lung elastic recoil. The maximal increase in elastic recoil occurred 6 to 12 months after LVRS with subsequent loss to near baseline levels by 2 years after LVRS.² Furthermore, analysis of the mechanism of airflow limitation using MFSR curves prior to LVRS demonstrated, in addition to the severe loss of lung elastic recoil, suspected independent intrinsic small airway involvement (*ie*, reduced Gs),^{2,15} similar to the present study.

Following LVRS, we noted an increase in lung elastic recoil that was probably due to improved functioning of the remaining lung with less-extensive emphysema. The increase in Gs was attributed to reduced extrinsic compression and more effective tethering of small airways, thereby reducing collapse during forced exhalation and allowing for overall increased airway conductance.^{2,15} Subsequently, Martinez et al³⁵ and Jubran et al³⁶ also reported short-term increase in lung elastic recoil following bilateral LVRS.

Dyspnea and Exercise Tolerance After LVRS

Numerous investigators have reported improvement in dyspnea and exercise tolerance after LVRS.^{1-6,17,35,37-44} This best correlated with the reduction in hyperinflation and increase in transdiaphragmatic pressure due to recruitment of inspiratory respiratory muscles^{1,17,35,37-44} and subsequent increased neuromechanical coupling,⁴⁵ often irrespective of changes in FEV₁. However, we believe the reduction in hyperinflation and increased transdiaphragmatic pressure is consequent to the increase in lung elastic recoil following LVRS and repositioning of the diaphragm.

Selection Criteria

The results of this study identified preoperative significant VC and FVC differences between nine

patient responders with > 3-year increase in FEV₁ > 0.2 L and/or FVC > 0.4 L from short-term responders with physiologic improvement ≤ 2 years who died within 4 years of LVRS. This difference may reflect a global estimate of functioning lung tissue. Additional evaluation will be required to test the reliability of this observation.

A previous study by Ingenito et al⁴⁶ correlated 6-month increase in FEV₁ following LVRS with baseline lung elastic recoil at TLC and inspiratory total lung resistance. However, their range of lung resistance was much higher than what was measured in our patients. Furthermore, a review of the data of Ingenito et al⁴⁶ over the range of inspiratory airway resistance similar to our patients (< 9 cm H₂O/L/s) revealed no significant correlation with short-term increase in FEV₁. Inspiratory airway resistance in the present study was obtained using noninvasive plethysmography. This technique does not measure lung tissue resistance, which would not be expected to be significantly increased, and is a small component of total lung resistance. We found no significant correlation (*r* = 0.3) between preoperative airway conductance and subsequent long-term improvement in FEV₁ > 0.2 L and FVC > 0.4 L.

With the exception of one study,⁴⁷ most investigators^{48,49} have found preoperative mild pulmonary hypertension that improved following LVRS. Mild changes in gas exchange after LVRS have been reported, presumably from alternations in ventilation-perfusion heterogeneity.⁵⁰

In summary, LVRS provides significant clinical and physiologic improvement to a subset of patients with severe emphysema for up to 4 years. Baseline differences for VC and FVC separated these patients preoperatively from short-term responders who had physiologic improvement ≤ 2 years after LVRS and who died during the study. However, the importance of heterogenous distribution of emphysema on lung CT and perfusion scans in choosing potential LVRS candidates must still be emphasized.⁵¹

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