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Serial Lung Function and Elastic Recoil 2 Years After Lung Volume Reduction Surgery for Emphysema*

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Study objective: **To evaluate serial lung function studies, including elastic recoil, in patients with severe emphysema who undergo lung volume reduction surgery (LVRS). To determine mechanism(s) responsible for changes in airflow limitation.**
Methods: **We studied 12 (10 male) patients aged 68±9 years (mean±SD) 6 to 12 months prior to and at 6-month intervals for 2 years after thoracoscopic bilateral LVRS for emphysema.**
Results: **At 2 years post-LVRS, relief of dyspnea remained improved in 10 of 12 patients, and partial or full-time oxygen dependency was eliminated in 2 of 7 patients. There was significant reduction in total lung capacity (TLC) compared with pre-LVRS baseline, 7.8±0.6 L (mean±SEM) (133±5% predicted) vs 8.6±0.6 L (144±5% predicted) (p=0.003); functional residual capacity, 5.6±0.5 L (157±9% predicted) vs 6.7±0.5 L (185±10% predicted) (p=0.001); and residual volume, 4.9±0.5 L (210±16% predicted) vs 6.0±0.5 L (260±13% predicted) (p=0.000). Increases were noted in FEV_1, 0.88±0.08 L (37±6% predicted) vs 0.72±0.05 L (29±3% predicted) (p=0.02); diffusing capacity, 8.5±1.0 mL/min/mm Hg (43±3% predicted) vs 4.2±0.7 mL/min/mm Hg (18±3% predicted) (p=0.001); static lung elastic recoil pressure at TLC (Pstat), 13.7±0.5 cm H_2O vs 11.3±0.6 cm H_2O (p=0.008); and maximum oxygen consumption, 8.7±0.8 mL/min/kg vs 6.9±1.5 mL/min/kg (p=0.03). Increase in FEV_1 correlated with the increase in TLC Pstat/TLC (r=0.75, p=0.03), but not with any baseline parameter.**
Conclusion: **Two years post-LVRS, there is variable clinical and physiologic improvement that does not correlate with any baseline parameter. Increased lung elastic recoil appears to be the primary mechanism for improved airflow limitation.** ***(CHEST 1998; 113:1497-1506)***

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

Abbreviations: FRC=functional residual capacity; Gaw=airway conductance; Gs=conductance of the S segment; LVRS=lung volume reduction surgery; Ptm′=critical transmural pressure in small airway collapsible segment; RV=residual volume; SGaw=specific airway conductance; TLC=total lung capacity

Despite aggressive medical therapy, including physical rehabilitation, the prognosis and palliative relief of dyspnea in COPD due to emphysema is poor. When the FEV_1 falls below 0.75 L or 30% predicted, survival at 3 years is only 50 to 60%.[1,2] Furthermore, patients admitted to a hospital ICU for exacerbation of COPD have a 1-year mortality rate of 30% and in patients aged >65 years, the 1-year mortality rate doubles.[3]

During the past several years, there has been emphasis in thoracic surgical procedures that attempt to provide palliative relief for markedly dyspneic patients with severe, diffuse (nongiant bullous) emphysema. Unilateral and bilateral video-assisted thoracoscopic[4,5] or median sternotomy[6-12] incisions are made, and the worst-targeted emphysematous areas are excised, *ie*, lung volume reduction surgery (LVRS). Following bilateral LVRS, results indicate variable improvement in relief from dyspnea, oxygen dependency, lung function, and exercise tolerance at 6 months,[4,5,7-11,13,14] 1 year,[6,15] and ≥2 years following surgery.[6,12]

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The present study evaluates the clinical and physiologic changes, including lung elasticity, in 12 markedly symptomatic patients with severe emphysema who have been followed up, both preoperatively and every 6 months, for 2 years post-LVRS. It extends our previously published results obtained immediately,[13] 6 months,[14] and 12 months[15] after bilateral LVRS. The thrust for this study was to analyze the physiologic mechanism(s) responsible for serial changes in lung function 2 years following LVRS.

Materials and Methods

Patient Selection

From February through June 1995, we evaluated 28 patients aged 67±8 years (mean±SD) who underwent LVRS. The intent was to obtain preoperative and postoperative lung function studies, including measurements of lung elasticity at 6-month intervals. Following LVRS, five patients died (at 1, 16, 17, 20, and 24 months) from respiratory failure, one patient was unavailable for follow-up, and three patients refused to be retested. Incomplete data were obtained in 7 of the remaining 19 patients since they refused repeated measurements of lung volumes, diffusing capacity, and elastic recoil post-LVRS, but did agree to spirometry. However, 12 (10 male) patients aged 68±9 years (mean±SD) satisfied our criteria and are described in detail. In the seven other patients, as well as the five patients who died post-LVRS and the one patient unavailable for follow-up and three patients who refused any additional testing, their preoperative clinical status and all lung function studies, including lung elastic recoil, were similar to the results obtained in the 12 presently reported patients. Furthermore, these seven patients demonstrated no significant increase in spirometry at 12 and 24 months post-LVRS compared with their baseline. Data from 8 of the 12 patients in this report were previously published at 6[14] and 12[15] months post-LVRS. The current data obtained at 24 months post-LVRS have not been previously reported. The cigarette-smoking history of the patients studied was 50±15 pack-years (mean±SD).

All the patients who had LVRS were markedly symptomatic with grade ≥3 dyspnea,[16] tolerance limited to walking <100 yd, with severe, fixed airflow limitation that had not improved despite antibiotics, oxygen, corticosteroids, aerosol, and oral bronchodilators. Thin-section (2-mm) high-resolution CT of the lungs[17,18] demonstrated visual emphysema scores ranging from 60 to 80 in the upper third lung fields and scores ranging from 40 to 70 in the lower third lung fields. Heterogeneity of visually scored emphysema distribution between upper and lower lung fields was present in every patient. We obtained standard nuclear medicine-perfusion lung scans (six view) in all patients, and in five patients, ^{99m}Tc macroaggregated albumin single-photon emission CT scans. Results demonstrated vascular distribution abnormalities corresponding to lung CT scans. There was relatively well-preserved perfusion in the lower third of lungs and none or markedly decreased perfusion in the upper third of lungs.

Operative Technique

As previously described,[4,13-15] after obtaining informed consent and approval of the Institutional Human Investigation Committee at Chapman Medical Center, patients underwent sequential bilateral upper lung fields video-assisted thoracoscopic stapled lung resectional surgery at the same operative sitting.

Lung Function Studies

As previously described,[14,15] dyspnea evaluation,[16] arterial blood gases at rest, and lung function, including elastic recoil studies, were measured after obtaining informed consent in a pressure-compensated flow plethysmograph (model 6200 Autobox; SensorMedics; Yorba Linda, Calif) and compared with predicted values. Maximum expiratory flow and airway conductance (Gaw) were plotted against static lung elastic recoil pressure curves, as previously described.[14,15] Studies in patients were obtained within 6 to 12 months and 2 weeks prior to and repeated every 6 months for 2 years after LVRS. The studies at 6 to 12 months prior to LVRS were originally obtained as yearly follow-up studies in patients with severe airflow limitation.

Exercise Studies

Progressive exercise testing to symptom-limited maximum was obtained using cycle ergometry (Tunturi; Turku, Finland) with 2-min increases of 10 to 20 W at pedaling cycle of 40 to 50 rpm. Subjects breathed room air through a mouthpiece with nose clips using a low-resistance two-way nonrebreathing valve. Expired gases were collected and analyzed (Vmax 29; SensorMedics Inc). A subset of only 7 of the 12 patients agreed to and were evaluated preoperatively and every 12 months post-LVRS. Lung function studies in the 7 patients were not significantly different from the 12 patients, either pre-LVRS or post-LVRS.

Statistical Methods

Comparison of the difference between patients before and after surgery was determined using two-tailed paired or two-sample unpaired t test with p values <0.05 being significant. Because of the small sample size, the degree of linear association between two continuous variables was assessed using the nonparametric Spearman correlation coefficients based on ranks. Each patient served as his or her own control subject for comparison with end points post-LVRS.

Results

Results of serial complete lung function and resting arterial blood gas studies in 12 patients appear in Table 1. Spirometry, lung volumes, and diffusion studies were available in patients 6 to 12 months prior to surgery, and results (data not shown) were similar when compared with 2-week preoperative baseline values, despite aggressive therapeutic intervention, including physical rehabilitation. The average hospital stay was 10.7±1.0 days (mean±SD). Dyspnea[16] was improved in every patient by ≥1 grade at 12 months post-LVRS and ≥1 grade in 10 patients 24 months post-LVRS. Oxygen dependence, full or part time, because of resting or postexercise PaO_2 <59 mm Hg was eliminated in two of seven patients up to 24 months post-LVRS. Up to 2 years preoperatively, two patients each required four hospitalizations for exacerbation of their COPD. Within

Table 1—*Physiologic Results in 12 Patients Who Were Studied Before and After LVRS for Emphysema*

	Preop 2 wk	Postop 6 mo	Postop 12 mo	Postop 18 mo	Postop 24 mo
FVC, L	2.2±0.2	3.1±0.2	2.7±0.2	2.8±0.2	2.7±0.1
	(60±5)	(77±5)	(73±6)	(74±7)	(73±6)
p value		0.000	0.001	0.001	0.001
FEV_1, L	0.72±0.05	1.19±0.13	1.02±0.1	0.93±0.1	0.88±0.08
	(29±3)	(45±7)	(41±5)	(38±6)	(37±6)
p value		0.001	0.002	0.006	0.02
TLC, L	8.6±0.6	7.6±0.4	7.7±0.5	8.0±0.5	7.8±0.6
	(144±5)	(122±8)	(129±5)	(138±5)	(133±5)
p value		0.002	0.002	0.002	0.003
FRC, L	6.7±0.5	5.3±0.4	5.3±0.5	5.8±0.5	5.6±0.5
	(185±10)	(141±7)	(151±9)	(162±8)	(157±9)
p value		0.000	0.000	0.000	0.001
RV, L	6.0±0.5	4.4±0.4	4.7±0.5	5.2±0.5	4.9±0.5
	(260±13)	(184±13)	(201±15)	(220±13)	(210±16)
p value		0.000	0.000	0.000	0.000
SGaw, L/s/cm H_2O/L	0.03±0.00	0.07±0.02	0.05±0.00	0.04±0.00	0.04±0.006
	(12±2)	(18±3)	(20±2)	(16±2)	(17±3)
p value		0.12	0.01	0.07	0.03
DcoSB, mL/min/mm Hg	4.2±0.7	7.3±0.9	9.2±1.0	8.1±1.0	8.5±1.0
	(18±3)	(36±5)	(47±3)	(40±3)	(43±3)
p value		0.02	0.004	0.006	0.001
PaO_2, mm Hg	63±4.0		73±2.0		60±5.0
p value			NS		NS
$PaCO_2$, mm Hg	46±3.0		40±5		45±3
p value			NS		NS
PstatFRC, cm H_2O	1.3±0.2	2.9±0.1	3.1±0.2	2.3±0.2	2.8±0.4
p value		0.000	0.001	0.004	0.009
PstatTLC, cm H_2O	11.3±0.6	16.3±0.7	14.8±0.8	13.4±0.7	13.7±0.5
p value		0.000	0.01	0.04	0.008
PstatTLC/TLC, cm H_2O/L	1.4±0.2	2.1±0.2	1.9±0.2	1.8±0.2	1.9±0.2
p value		0.000	0.002	0.005	0.001
Gs, L/s/cm H_2O	0.17±0.03	0.28±0.04	0.27±0.04	0.23±0.04	0.19±0.04
p value		0.02	0.03	NS	NS
Ptm', cm H_2O	3.1±0.23	2.4±0.20	2.5±0.20	2.7±0.19	2.8±0.19
p value		0.005	0.01	NS	NS

*Preop=preoperative; postop=postoperative; DcoSB=single breath diffusing capacity for carbon monoxide; PstatTLC=static transpulmonary pressure at TLC; NS=not significant. All physiologic studies obtained during the year before surgery were similar to preoperative studies. All values are means±SEM. Values shown in parentheses are percent predicted values, and p values refer to comparison with preoperative study.

2 years post-LVRS, these same patients required one and three hospitalizations.

Lung Function Studies

In the 12 patients described in detail at 24 months post-LVRS, there was still significant improvement in most physiologic studies except resting arterial blood gases when compared with preoperative values. Compared with baseline, the FEV_1, specific airway conductance (SGaw), diffusing capacity, static lung elastic recoil pressure at both functional residual capacity (FRC) and total lung capacity (TLC), and coefficient of retraction (static lung elastic recoil pressure at TLC/TLC) remained significantly improved 24 months post-LVRS, despite reduction in all static lung volumes. Spirometric and lung volumes were most improved at 6 months post-LVRS.

Exercise Studies

Results of exercise studies appear in Table 2, and pre-LVRS, all patients had severe exercise intolerance. The increase in oxygen consumption, minute ventilation, tidal volume, and resting oxygen saturation peaked at 1 year post-LVRS. However, even at 2 years post-LVRS, exercise performance and resting oxygen saturation remain above pre-LVRS baseline values.

Maximum Flow Volume Loops

Analysis of the mean maximum expiratory and inspiratory flow volume loops in 12 patients demonstrates severe airflow limitation and hyperinflation at baseline (Fig 1). Compared with preoperative LVRS baseline, there was a continued downward shift on the volume axis toward lower lung volumes even at

Table 2—*Results in Preoperative and Postoperative Exercise Studies in Seven Patients**

Study	Preoperative 2 wk	Postoperative 12 mo	Postoperative 24 mo
$\dot{V}O_2$, mL/min/kg	6.9±1.5	9.5±0.8†	8.7±0.8†
$\dot{V}E$ max, L/min	25.8±4.4	29±3.0†	26±3.0
f, min	27.1±4.0	21±2.0†	25±3
VT, L	0.88±0.10	1.26±0.1†	1.1±0.10
O_2 sat rest, %	91±4	94±0.7†	94±0.5†
O_2 sat peak exercise, %	87±2	91±0.6	86±1.3
O_2 pulse, $\dot{V}O_2$, mL/min/pulse rate	5.2±1.0	6.4±0.7	6.7±1.0

*$\dot{V}O_2$=oxygen consumption; $\dot{V}E$ max=maximum ventilation; f=respiratory rate per minute; VT=tidal volume; sat=saturation. All values are mean±SEM.
† $p<0.05$.

24 months post-LVRS. However, the shift at 24 months post-LVRS was not as marked as at 6 and 12 months post-LVRS. Even at 24 months post-LVRS, mean maximum expiratory flow was increased at the same lung volume when compared with baseline.

Static Lung Elastic Recoil Pressure Curves

Preoperatively, there was marked loss of lung elastic recoil (Fig 2). The peak mean increase in static lung elastic recoil occurred 6 to 12 months postoperatively. However, even at 24 months after LVRS, values were significantly greater when compared with baseline.

Maximum Expiratory Flow-Static Lung Elastic Recoil Pressure Curves

At baseline, the critical transmural pressure in small airway collapsible segment (Ptm′) was shifted toward higher pressures than age-matched normal subjects, and conductance of small airway S segment (Gs) was markedly reduced (Fig 3). There was a significant increase in small airway Gs, and decrease in Ptm′ only up to 12 months post-LVRS when compared with baseline. After 1 year post-LVRS, values for Gs and Ptm′ were similar to baseline preoperative values. However, the increased driving pressure (elastic recoil) increased maximum expiratory flow at isovolume points.

Gaw-Lung Elastic Recoil Pressure Curve

Initially, all patients had reduced airway conductance that could not be accounted for solely by loss of lung elastic recoil (Table 1 and Fig 4). Up to 12 months following LVRS, despite the *reduction* in FRC, total Gaw measured at FRC increased significantly due to the significant increase in lung elastic recoil.

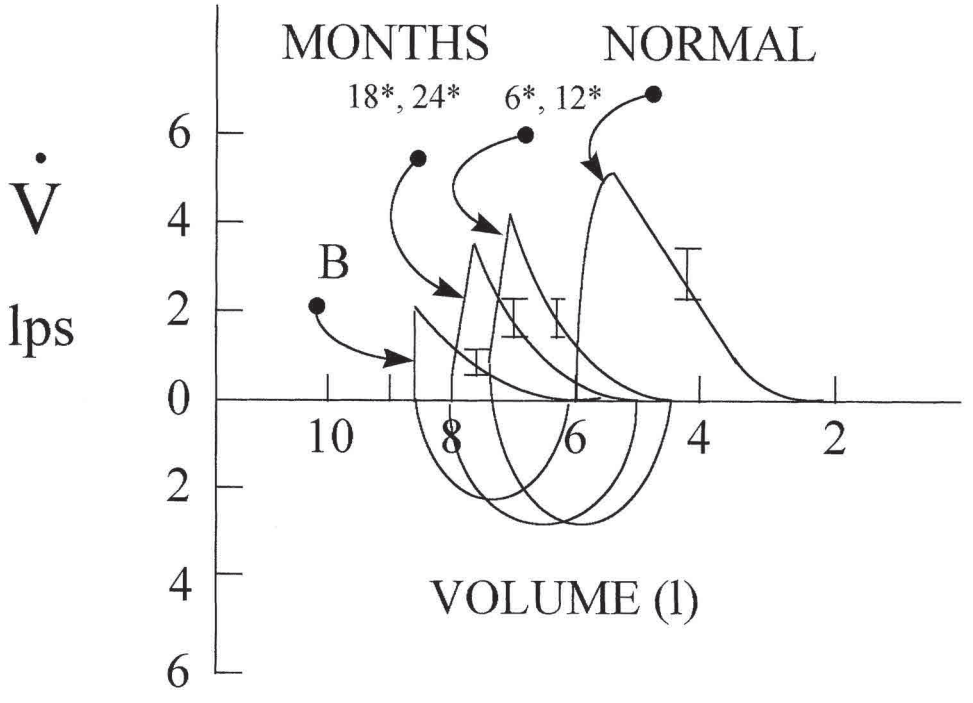

FIGURE 1. Mean maximum expiratory and inspiratory flow-volume loops at baseline (B) and 6, 12, 18, and 24 months post-LVRS in 12 patients compared with age-matched normal subjects. There is marked reduction in lung volume that peaks 6 to 12 months post-LVRS and increased maximal flow at 50% FVC compared with baseline at 6, 12, 18, and 24 months. Bars=±SEM; asterisk=$p<0.05$. Normal values are from references 19-21.

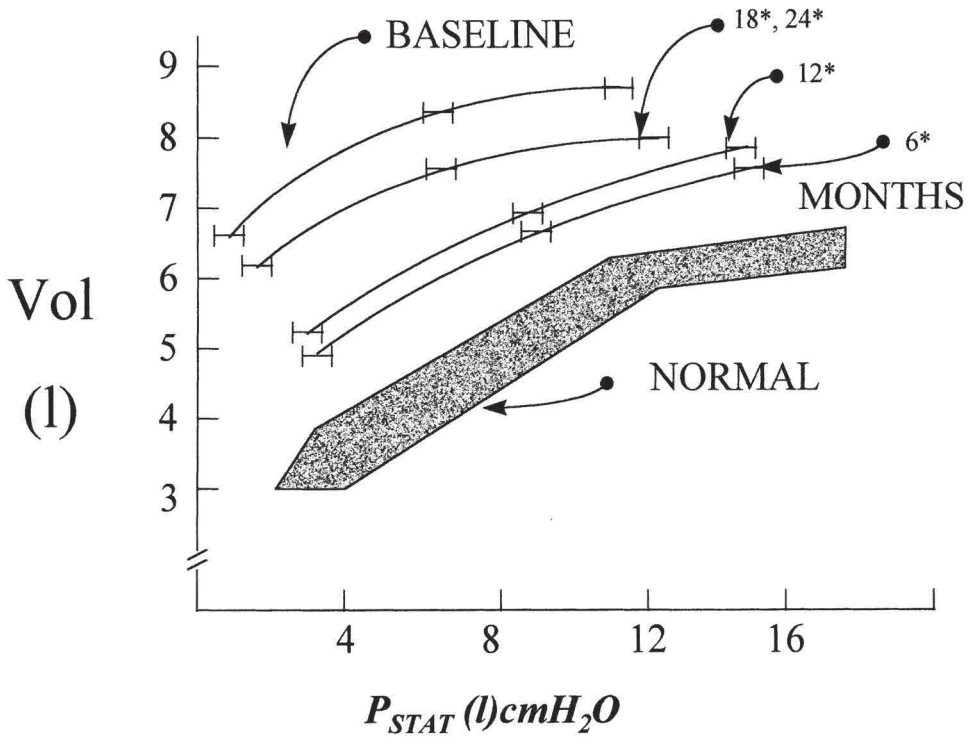

FIGURE 2. Mean static lung elastic recoil pressure (Pstat) curves at baseline and 6, 12, 18, and 24 months post-LVRS in 12 patients compared with range of age-matched normal subjects. The peak increases in Pstat at TLC and FRC occurred at 6 and 12 months (Table 1), but remain significantly increased even at 24 months post-LVRS. Bars=±SEM; asterisk=$p<0.05$. Normal values are from references 19-21.

Predictors of Post-LVRS Increase in FEV_1

At 12 and 24 months post-LVRS, the increase in FEV_1 was poorly correlated with baseline preoperative static lung elastic recoil at TLC ($r=0.3$; $p=0.3$), coefficient of retraction ($r=0.28$; $p=0.3$), SGaw, Gaw, and Gs ($r=0.4$; $p=0.2$), and with extent and heterogeneity of visually scored emphysema ($r=0.3$; $p=0.3$).

At 24 months post-LVRS, the increase in FEV_1 correlated best with the increase in coefficient of retraction ($r=0.75$; $p=0.03$) and increase in Gaw ($r=0.89$; $p=0.001$). This emphasizes post-LVRS the importance of increased lung elastic recoil despite reduction in lung volume to increase maximum expiratory flow and airway caliber. However, relief from dyspnea, oxygen independence, and improved exercise tolerance did not correlate with increased FEV_1.

DISCUSSION

Results in the present study reveal that at 24 months after targeted bilateral stapled LVRS for severe, nonbullous generalized emphysema, 12 selected patients maintained significant improvements in lung function, with variable relief from dyspnea, improved oxygen independence, and increased exercise tolerance when compared with baseline. This is primarily due to increased lung elastic recoil despite the reduction in lung volume. However, preoperative clinical, physiologic, and CT lung studies could not identify those individual patients who had optimal clinical improvement and increases in FEV_1 post-LVRS.

Lung Elastic Recoil

We have previously reported[19,20] that expiratory airflow limitation in clinically unsuspected and early physiologic (normal or near-normal FEV_1), but moderately advanced morphologic emphysema (mean visually scored anatomic grade 50) and bullous lung disease[21] without concomitant emphysema could be accounted for by loss of lung elastic recoil. This results in decreased driving pressure and loss of alveolar support to tether the airways during forced exhalation. We[21] and others[22-25] have also noted the increase in expiratory airflow and Gaw following bullectomy in isolated bullous lung disease, and bullous emphysema could be attributed to the increase in lung elastic recoil. The increase in lung elastic recoil described by Sciurba et al[26] 6 months

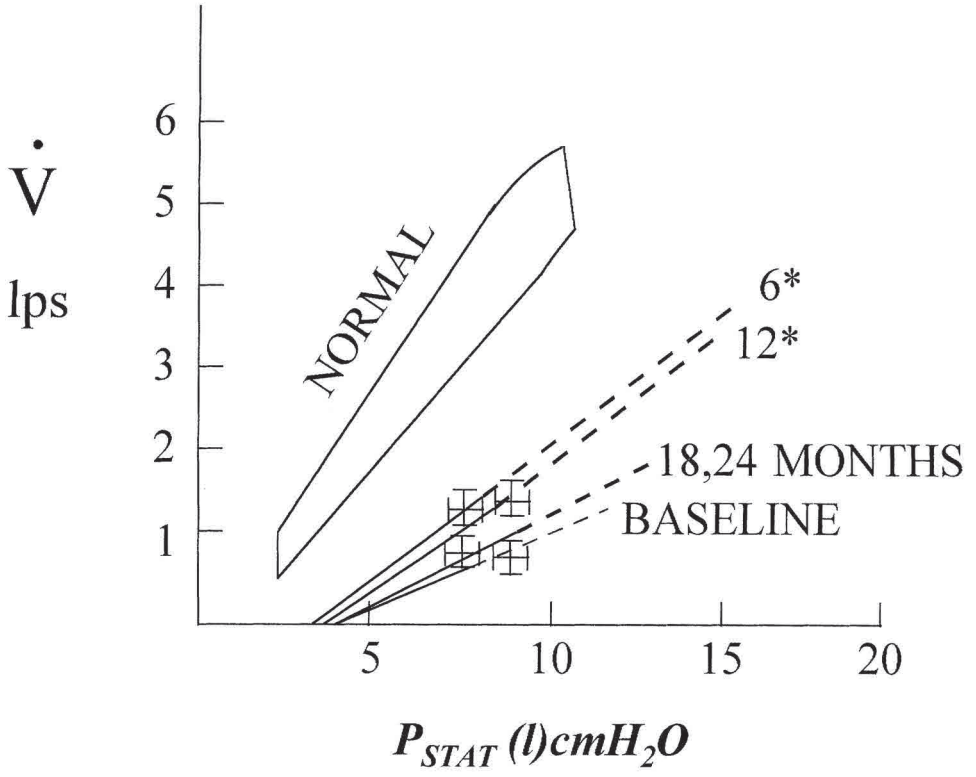

FIGURE 3. Mean maximum expiratory flow-static lung elastic recoil pressure curves in 12 patients compared with baseline and age-matched normal subjects. The solid slope line represents the Gs 80 to 50% FVC, and its intercept on the pressure axis is the Ptm'. The extrapolated dashed Gs slope line extends to Pstat at TLC. Gs and Ptm' are significantly increased at only 6 and 12 months post-LVRS. The greater lung elastic recoil increases maximum flow at 18 and 24 months post-LVRS by increasing the driving pressure, but not by increasing airway caliber or reducing airway collapse. Bars=±SEM; asterisk=$p<0.05$. Normal values are from references 19–21.

following unilateral LVRS for generalized emphysema, and our results immediately, 6 months, and 12 months[13-15] following bilateral LVRS, is probably the mechanism for improvement in expiratory airflow. The present report extends these conclusions 24 months post-LVRS.

Mechanical Changes

A physiologic consequence of emphysema is loss of lung elastic recoil, causing hyperinflation. This, together with dynamic airway collapse and intrinsic positive end-expiratory pressure, causes a shift in breathing to higher lung volumes. There is shortened diaphragm muscle and reduced surface area[27] with significant functional impairment in muscle strength with hyperinflation.[28] Furthermore, dyspnea may be better correlated with abnormal respiratory muscle dysfunction, breathing patterns, and hyperinflation than expiratory airflow limitation.[29-31]

Following LVRS, there is marked reduction in all static lung volumes, *eg*, residual, FRC, and TLC. After 6 months post-LVRS, there is a progressive increase in these volumes, although they remain significantly below baseline values even at 24 months. We believe this increasing hyperinflation corresponds to the subsequent loss of the lung elastic recoil that also occurs after 6 months following LVRS.

Despite the overall reduction in all static lung volumes, there are increases in FEV_1, FVC, Gaw, and maximum expiratory flow compared with baseline at isovolume points that peak at 6 months but persist 24 months post-LVRS. This also reflects the initial increase and subsequent loss of lung elastic recoil that is observed following LVRS. Moreover, even at 24 months post-LVRS, lung elastic recoil pressure at TLC and FRC remain significantly increased when compared with baseline values.

Diffusing Capacity

The increased diffusing capacity 6 to 12 months following LVRS[14,15] was maintained after 2 years and probably reflects a greater alveolar-capillary surface area due to less lung tissue compression and increased transpulmonary pressure. Another possibility (R. Hyatt, MD; personal communication; 1997)

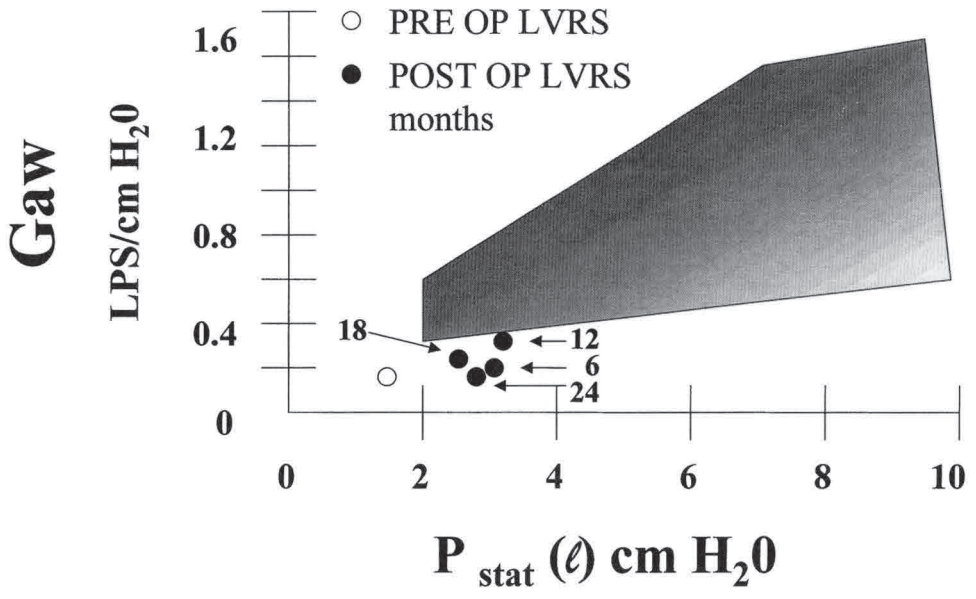

FIGURE 4. Relationship between Gaw and lung elastic recoil. Despite the increase in lung recoil, Gaw does not increase proportionately, probably because of severe intrinsic small airway disease resulting in reduced airway caliber. Normal values in shaded area are from reference 21.

may be more even distribution of ventilation/perfusion ratios to the areas with better potential for diffusion.

Mechanism of Airflow Limitation

Analysis of the maximum expiratory airflow-static lung elastic recoil pressure curve and the Gaw-static lung elastic recoil curves (Figs 3 and 4) indicates markedly abnormal airflow and Gaw, both before and after LVRS, that cannot be explained completely by the loss of lung elastic recoil and/or airway collapse. We attribute this to marked intrinsic small airway structural abnormalities that are associated with long-term cigarette smoking in patients with severe COPD and emphysema.[18,32] This physiologic profile was also seen in half the patients with α_1-antitrypsin deficiency, reported by Black et al,[33] as well as those patients with moderately severe airflow limitation, reported by Duffell et al.[34]

Leaver et al[35] noted in all 16 COPD patients with relatively severe airflow limitation (FEV_1, 1.02±0.4 L [mean±SD]) that maximum expiratory airflow was disproportionately reduced compared with the loss of lung elastic recoil. They attributed this to a combination of intrinsic airways disease and enhanced collapsibility of flow-limiting airways (decreased Gs with increased Ptm′). Hogg et al[36] demonstrated a predominant peripheral intrinsic small airways site to explain the elevated airway resistance in patients who died of emphysema. They described mucus plugging, narrowing, fibrosis, distortion, and obliteration of small airways.[36] Moreover, increasing the distending pressure (elastic recoil) failed to decrease the airway resistance, and there was no difference between inspiratory and expiratory airway resistance. They concluded that despite destruction of alveolar support for the airways and decreased lung elastic recoil in severe emphysema, airflow limitation is primarily due to intrinsic small airways abnormalities.[36]

Dyspnea

The relationship between post-LVRS improvement in FEV_1 and relief of dyspnea is poorly understood. Using multivariate analysis, we have previously noted that the increase in FEV_1 following unilateral stapled LVRS correlated statistically ($p<0.05$) only with smoking history and younger age, but not with preoperative thoracic gas volume, spirometry, or diffusing capacity.[37] Furthermore, analysis of 154 patients undergoing bilateral stapled LVRS noted that only the presence of a bilateral upper lobe heterogeneous pattern on lung CT and perfusion scan correlated with improvement in FEV_1 and relief of dyspnea. No other clinical factors or baseline lung function study could better refine patient selection criteria.[38] However, a weak correlation between baseline dyspnea scores and FEV_1 ($r=0.27$; $p=0.06$) was noted in 145 patients undergoing bilateral LVRS.[39] At 276±90 days (mean±SD) post-LVRS, follow-up in 84 patients

revealed that FEV_1 increased a mean of 50% (from 0.64±0.27 L [mean±SD] to 1.04±0.4 L), but was not correlated with improvement in dyspnea score (r=0.3; p=0.3).[39] The postoperative LVRS improvement in dyspnea score correlated weakly with preoperative plethysmograph calculated TLC (r=0.3; p=0.2), trapped gas volume (plethysmograph TLC-gas dilution TLC) (r=0.2; p=0.7), residual volume (RV)/TLC (r=0.4; p=0.05), and RV (r=0.4; p=0.03).[39]

Exercise and Dyspnea

At 1 year post-LVRS, the significant increase in maximum oxygen consumption and work performance we noted was achieved with increased minute ventilation and tidal volume with decreased respiratory frequency. At 2 years post-LVRS, the increase in maximum oxygen consumption may be related, in part, to improved cardiac output due to less mechanical constraints. However, the observed less-than-robust improvement in gas exchange and exercise tolerance at 2 years following LVRS emphasizes that there may be disproportionate improvement(s) in lung mechanics, exercising ability, perception of dyspnea, and gas exchange. Similar observations have been noted postlung transplantation[40] and LVRS.[41]

A recent study by O'Donnell and colleagues[42] investigated the potential mechanisms for short-term (3 months) relief of dyspnea in eight patients following unilateral LVRS. They attributed it to a combination of reduced thoracic hyperinflation, decreased breathing frequency, reduced mechanical constraints on tidal volume, and increased FVC. Keller et al[43] noted in 25 patients 4 months after LVRS that increased maximal oxygen consumption was accomplished by increased inspiratory and expiratory flows with larger minute ventilation and tidal volume, but no change in respiratory frequency and no correlation with clinical relief from dyspnea. Benditt et al[44] noted that following LVRS, improved exercise performance was associated with increased maximal ventilation.

Benditt et al[45] evaluated eight patients 3 months after bilateral LVRS and noted improvement in ventilatory muscle recruitment. There was a reduction in both end-expiratory resting and exercise esophageal and gastric pressures. Results were consistent with less recruitment of the abdominal and accessory muscles and a relatively greater contribution of the diaphragm in inspiratory muscle generation. Bloch et al[46] made similar observations studying patients before and after LVRS using respiratory inductive plethysmography. Martinez et al[47] reached similar conclusions after evaluating 17 patients at least 3 months after bilateral LVRS. In addition to increased lung recoil at TLC, they noted variable increases in maximal inspiratory mouth and transdiaphragmatic pressure, FEV_1, work performance, and less dynamic hyperinflation and breathlessness. Similar to Keller et al,[43] they noted little change in the ratio between inspiratory time and total respiratory-cycle duration. Teschler et al[48] also reported increases in transdiaphragmatic pressures at 3 months after unilateral stapled LVRS. However, the increase in transdiaphragmatic pressures reported[42,45,47,48] post-LVRS were not measured at lung isovolumes pre-LVRS.

Improvement in FEV_1

Roue et al[12] noted clinical and functional improvement in 4 of 11 patients at 2 years post-LVRS, in three patients at 3 years post-LVRS, and in none at 4 years post-LVRS. Cooper et al[6] followed up their initial 20 patients (mean age, 56 years) for a mean of 30 months post-LVRS (range, 25 to 39 months) and noted persistent clinical and physiologic improvement. This is in contrast to our experience in the present study. Furthermore, in 90 patients who had bilateral stapled LVRS, we noted a mean increase in FEV_1 of 0.39±0.03 L (mean±SEM) at 3 to 6 months postsurgery with subsequent decline in FEV_1 per year of 0.255±0.057 L (mean±SEM) over 420±15 days (mean±SEM) follow-up time.[49] A weak correlation was noted between 3- and 6-month post-LVRS incremental gain in FEV_1 and subsequent decline in FEV_1 (r=0.292; p=0.003), and individual response could not be predicted.[49]

Lung CT and Perfusion Lung Scans

All of the patients in the present study had a heterogeneous distribution of emphysema on lung CT with upper-third predominance and matching perfusion scan abnormalities. Wang et al[50] and Weder et al[51] have reported modest lung CT[51] and scintigraphic correlation of FEV_1 improvement with upper-lobe predominance (r=0.38; p=.001)[50] and heterogeneity (r=0.31; p=0.002).[50]

Conclusions

The results in the present study extend our earlier experience[13-15] and document the variable clinical and physiologic improvement in lung elastic recoil and expiratory airflow limitation observed 2 years after bilateral LVRS in 12 selected symptomatic patients with severe, generalized emphysema who had exhausted medical therapy. The increase in lung elastic recoil peaked at 6 months post-LVRS. We

urge caution in the interpretation and extension of the data because of lack of a control group and the small number of patients studied.

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