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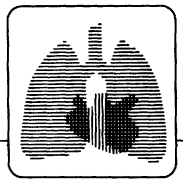

clinical investigations

Contribution of Lung and Chest Wall Mechanics Following Emphysema Resection*

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Objective: **To determine the contributions of (1) chest wall (Pcw) and (2) lung elastic recoil pressure (PL) to (3) total elastic recoil pressure exerted by the respiratory system (Prs) in 18 patients (12 men) aged 66±6 years (mean±1 SD) with severe emphysema who underwent video-assisted thoracoscopic bilateral lung volume reduction surgery under paralyzed (vecuronium) general anesthesia (isoflurane).**

Design: **We measured preoperative and 6-week postoperative lung function studies, and intraoperative inspiratory lung conductance (GL), PL, Pcw, and Prs (cm H_2O) at end-expiratory lung volume (EELV), EELV plus 0.60±0.0 L, and EELV plus 1.15±0.0 L. All values are mean±SEM.**

Results: **Preoperative vs postoperative FVC was 1.9±0.1 L vs 2.3±0.1 L (p=0.03); FEV_1 was 0.6±0.1 L vs 0.9±0.1 L (p<0.02); total lung capacity was 7.4±0.4 L vs 5.9±0.3 L (p<0.001); functional residual capacity was 5.7±0.4 L vs 4.4±0.2 L (p=0.001). At EELV preoperative vs postoperative, PL was 0.0±0.3 vs 1.1±0.05 (p=0.04), Pcw was 5.0±0.7 vs 2.4±0.9 (p=0.02), and Prs was 5.0±0.8 vs 3.5±0.7 (p=0.08). At EELV plus 0.60 L, PL was 3.2±0.6 vs 6.1±0.9 (p<0.001), Pcw was 8.8±0.8 vs 7.0±0.9 (p=0.12), and Prs was 12.0±0.8 vs 13.1±0.7 (p=0.80). At EELV plus 1.15 L, PL was 6.8±0.9 vs 10.3±1.1 (p<0.001), Pcw was 13.5±1.0 vs 11.2±1.2 (p=0.12), and Prs was 20±1.2 vs 21.5±1.0 p=0.93). At EELV plus 0.60 L, GL was 0.09±0.00 L/S/cm H_2O vs 0.16±0.01 (p<0.01). At EELV plus 1.15 L, GL was 0.12±0.01 vs 0.21±0.03 (p<0.05) with similar preoperative vs postoperative GL/PL slopes.**

Conclusion: **The increase in PL and decrease in Pcw following LVRS for emphysema may be responsible for the increase in spirometry and airway conductance.** ***(CHEST 1996; 110:11-17)***

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

Abbreviations: EELV=end-expiratory lung volume; FRC=functional residual capacity; Paw=airway pressure; Pcw=chest wall pressure; PEEP-I=intrinsic positive end-expiratory pressure; Pes=esophageal pressure; PL=lung elastic recoil pressure; Prs=total elastic recoil pressure of the respiratory system; SGaw=specific conductance; TLC=total lung capacity

Emphysema results in a physiologic loss of lung elastic recoil, causing decreased expiratory airflow due to loss of driving pressure and premature airway collapse due to reduced airway traction, *ie,* less airway distending forces.[1,2] Conversely, an increase in lung elastic recoil would result in increased expiratory airflow and airway conductance, *ie,* increased airway caliber offering less resistance to airflow. Chest strapping, despite overall reduction in lung volume, paradoxically causes an increase in airway conductance due to increased lung elastic recoil.[3]

Previous surgical attempts at lung volume reduction surgery, *ie,* bullectomy in isolated bullous lung disease[4,5] and bullous emphysema,[4,6-9] have resulted in variable short-term improvement in expiratory airflow and airway conductance. We[5,6] and others[4,7-9] have reported previously that this could be accounted for by an increase in lung elastic recoil. More recently, lung

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volume reduction surgery in *nonbullous* diffuse emphysema has also resulted in improvement in lung function.[10-12] Although the mechanism is unclear, recent preliminary results demonstrate an improvement in lung elastic recoil.[13,14]

The present study evaluates contributions of (1) lung and (2) chest to (3) total respiratory static elastic recoil pressures exerted in markedly dyspneic patients with severe airflow limitation due to extensive emphysema who undergo lung volume reduction surgery. Because measurement of these pressures except lung elastic recoil require total relaxation of the respiratory system, data were obtained during general anesthesia in paralyzed patients.

Materials and Methods

Patient Selection

We consecutively studied 18 patients (12 men) aged 66±6 years (mean±SD). The patients who underwent the procedure were markedly symptomatic with grade 3 dyspnea,[15] with severe fixed expiratory obstruction that had not improved despite appropriate therapeutic interventions, including physical conditioning, antibiotics, aerosol and oral bronchodilators, and corticosteroids.[14] Although no patient had $PaCO_2$ greater than 52 mm Hg, 13 patients required intermittent or continuous low-flow oxygen. In addition, high-resolution, thin-section CT of the lungs demonstrated emphysema scores[16] of 60 or greater with heterogeneous distribution, *ie,* predominantly emphysematous destruction of upper to midlung fields with relative less emphysematous destruction in the lower lung fields. Standard nuclear medicine ventilation and perfusion lung scans demonstrated similar heterogeneous distribution.

Operative Technique

After informed consent and approval of the Institutional Human Investigation Committee at Chapman Medical Center were obtained, all patients underwent sequential bilateral video-assisted thoracoscopic surgery (by R.J.M. and R.F.) at the same operative sitting under vecuronium paralysis and isoflurane (0.75 MAC) general anesthesia (Siemens Servo 900C anesthesia ventilator; Siemens Medical Systems Inc; Danvers, Mass) with fraction of inspired oxygen (FIo_2) of 1.0 using a left-sided 39F double-lumen endotracheal tube (Mallincrodt Anesthesia; St. Louis).[14] After single dependent lung ventilation had been achieved, the contralateral upside deflated lung was examined. Visually the most distended, destroyed, emphysematous areas previously targeted by the preoperative CT lung scan in the upper and midlung fields were excised and linear staple lines were reinforced with bovine pericardium as previously described[17] (Peri-Strips; Bio-Vascular Inc; St. Paul, Minn) or bovine collagen (Instat; Johnson and Johnson; New Brunswick, NJ) to minimize air leaks. It was visually estimated that the excised lung volume was approximately 15 to 20% of each lung. Actual weight of resected lung was 30 to 90 g per side. Following lung excision, apical pleural tents and/or talc pleurodesis were not required. Operative time ranged from 1 to 2 h.

Lung Function Studies

Outpatient lung function studies were performed after informed consent had been obtained. These included static lung volumes measured by plethysmographic techniques,[18] timed spirometry, and single-breath diffusing capacity in accordance with American Thoracic Society recommendations,[19,20] and values were compared with predictions.[21-23] All patients were considered to have fixed airflow limitation since the FEV_1 following 3 inhalations of aerosolized albuterol (670 µg) improved less than 12% and/or less than 200 mL.[19] Thoracic gas volume[18] and airway resistance[24] were measured in a plethysmograph (models 2800 and 6200; Sensormedics Inc; Yorba Buena, Calif) as previously described and compared with predicted values.[18,24] The reciprocal of airway resistance[24] is conductance and was divided by the thoracic gas volume at which it was measured and specific conductance (SGaw) calculated. Normal values are greater than 0.12 L/s/cm H_2O/L.[25] All outpatient lung function studies were obtained within 1 year and 2 weeks prior to and repeated within 6 weeks after surgery.

Intraoperative Static Elastic Recoil Pressure and Elastance

Inspiratory static elastic recoil pressure curves were obtained intraoperatively during paralyzed general anesthesia in the supine position just prior to and immediately after emphysema resection. An intraesophageal balloon inflated with 0.5 mL air was positioned in the stomach and then retracted 10 to 13 cm into the lower third of the esophagus where it most closely tracked the changes in airway pressure, usually with the least cardiac oscillations observed to yield reliable changes in pleural pressure.[26-28] Airflow, tidal volume, esophageal (Pes), and airway pressures were measured and recorded (Bicore Inc; Irvine, Calif), and graphic analysis was used for all calculations. The static pressure exerted by the total respiratory system (Prs) can be obtained by measuring static airway pressure (Paw) to reflect alveolar pressure relative to ambient pressure against a closed shutter (zero flow) at different lung volumes when the patient is totally relaxed.[29] By simultaneously measuring Pes to reflect pleural pressure, the static elastic recoil pressure exerted across the integrated chest and abdominal wall and diaphragm and muscles (Pcw) can be measured.[29] The Prs at any given lung volume is the sum of lung (PL) and Pcw as they are in series.[29] Since the patients are both anesthetized and paralyzed, the contribution of the muscle component is eliminated. The methods used are similar to previous techniques.[30-32]

Static total respiratory (Prs), and chest wall (Pcw) elastic recoil pressures were measured (Paw, Pes) after 3 deep inhalations of 1,000 mL followed by suspended inspiration against a closed shutter (zero flow) for at least 5 s at end-expiratory lung volume (EELV) and EELV plus inspiratory volume of 600 mL and 1,150 mL. Lung static elastic recoil pressure (PL) was calculated from Prs minus Pcw (or Paw-Pes). Lung, chest wall, and total respiratory system elastance[30-32] were measured at EELV plus 600 mL and EELV plus 1,150 mL at suspended inspiration against a closed shutter (zero flow) for at least 5 s. Positive values for Paw and Pes at EELV reflect intrinsic positive end-expiratory pressure (PEEP-i) due to increased intrathoracic pressures. Data were obtained 10 min after paralyzed anesthesia was achieved, and 2 to 3 measurements were made in each patient and averaged.

Postresection, at the time of measurement, all chest tubes were clamped and there were no air leaks; chest radiograph revealed no pneumothorax.

We could not measure functional residual capacity (FRC) using plethysmographic methods in the anesthetized, paralyzed supine patient, whereas a gas dilution technique would grossly underestimate lung volume in the presence of severe obstructive lung disease. To construct preoperative and postoperative inspiratory static elastic recoil pressure volume curves, as a compromise we used EELV as the baseline.

In each patient, static inspiratory elastic recoil pressures were measured with the esophageal balloon similarly positioned from the nares and with the same lung volume history prior to and after lung resection.

Inspiratory Lung Resistance

Intraoperatively, inspiratory nonelastic lung resistance was also measured[33] at similar inspiratory lung volumes above EELV before

and after lung resection from analysis of (Bicore) generated graphics. We used the Mead and Whittenberger[33] technique with decelerating airflow and not airway occlusion at the end of constant-flow inflation.[30-32] The isolated resistance of the 39F double-lumen left endotracheal tube was 7.8 cm H_2O/L/s at airflow of 0.83 L/s and the in-line flow transducer (Bicore) and connector was 1.2 cm H_2O/L/s at airflow of 0.83 L/s and were subtracted from the measured inspiratory lung resistance. We report conductance as the reciprocal of lung resistance.

Statistical Methods

Comparison of the difference between patients before and after surgery was determined using 2-tailed paired *t* test with values ≤0.05 being significant.

Results

Results of all lung function studies appear in Tables 1 and 2 and Figures 1 and 2. Diffusing capacity was markedly abnormal; in every patient it was less than 40% predicted prior to surgery. The average hospital stay was 10±2 days (mean±SD).

Six weeks after surgery, there was marked improvement in results of both static and dynamic lung function studies (Table 1) and dyspnea was improved in every patient by 1 grade or more.[15] While 13 of the 18 patients required supplemental oxygen preoperatively, only 5 patients still required it 6 weeks postoperatively. Total lung capacity (TLC) and FRC decreased significantly (p≤0.01), yet there was a significant (p<0.001) increase in airway conductance, FVC (p=0.03), and FEV_1 (p<0.02). In 12 patients, results of spirometry obtained up to 1 year prior to surgery were similar to the preoperative values obtained 2 weeks prior to surgery.

Analysis of the Static Lung, Chest Wall, and Total Respiratory System Elastic Recoil Pressures and Elastance

Following surgery, despite a significant reduction in lung volume (Table 1), there was a significant (p<0.04) increase in elastic recoil pressure exerted by the lung (P_L) at all lung volumes. The significant increase in P_L is in contrast to the significant (p=0.02) reduction in chest wall elastic recoil (Pcw) only at EELV. There was no change in total elastic recoil of the respiratory system (P_{RS}). Preoperatively, at EELV, the increase in total respiratory PEEP-I could be accounted for by PEEP-I exerted by the chest wall. Postoperatively, there was a reduction in PEEP-I of the total respiratory system due to a significant decrease in PEEP-I of the chest wall. Static elastance of the lungs and total respiratory system increased significantly following surgery.

After surgery, however, expiratory airflow, airway conductance, and P_L remain abnormal consistent with underlying diffuse emphysema.

Table 1—*Results of Pulmonary Function Studies Prior to and 6 Weeks After Surgery*

	Preoperative	Postoperative	p Value
FVC, L	1.9±0.1*	2.3±0.1	0.03
FVC, % predicted	51±2.0	59±2.0	0.001
FEV_1, L	0.6±0.05	0.9±0.07	<0.02
FEV_1, % predicted	23±2.0	33±2.0	<0.001
FRC, L	5.7±0.4	4.4±0.2	0.001
FRC, % predicted	174±8.0	127±7.0	0.001
TLC, L	7.4±0.4	5.9±0.3	<0.001
TLC, % predicted	126±5.0	98±5.0	<0.001
SGaw, L/s/cm H_2O/L	0.04±0.00	0.06±0.00	<0.001

*Mean±SEM.

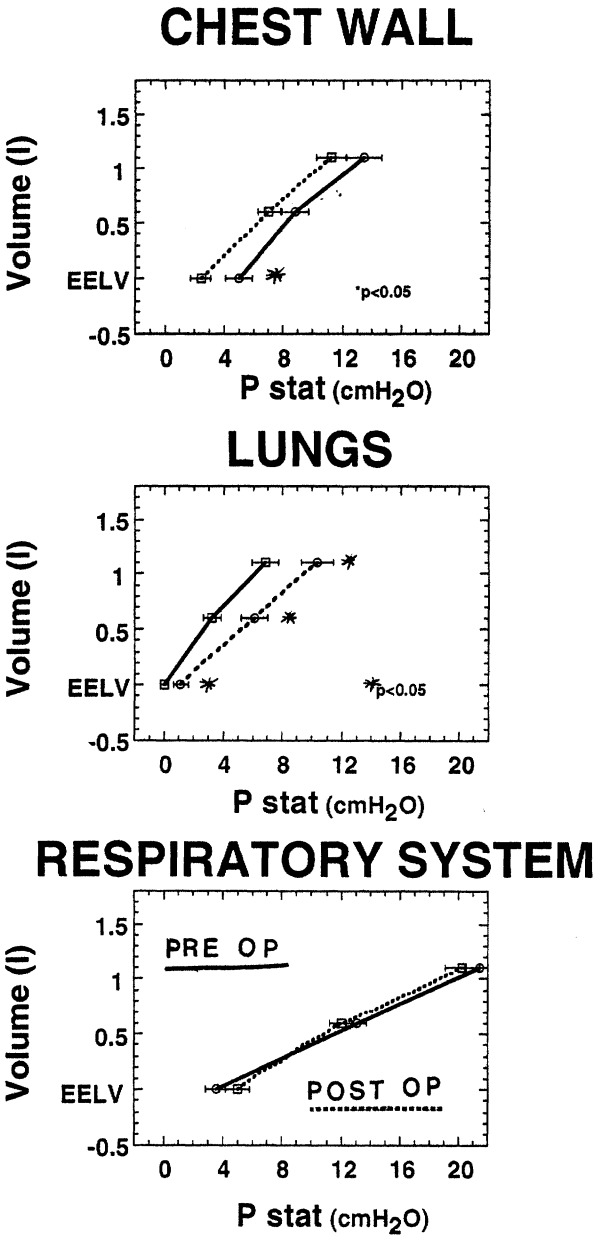

FIGURE 1. Results of static elastic recoil pressures of lung, chest wall, and total respiratory system. Immediately following lung volume reduction surgery for emphysema, there is a significant increase (p<0.05) in lung elastic recoil pressure at all lung volumes and decrease in chest wall elastic recoil pressure at EELV with no change in total respiratory system pressure. Solid line is preoperative and dashed line postoperative values.

Table 2—*Intraoperative Results of Static Elastance (Est) Elastic Recoil of Lung (PL), Chest Wall (Pcw), and Total Respiratory System (Prs) and PL, Pcw, Prs, and Conductance (GL) at Different Lung Volumes Just Prior to and After Lung Volume Reduction Surgery

Lung Volume	PL, cm H_2O	EstL, cm H_2O/L	Pcw, cm H_2O	Estcw, cm H_2O/L	Prs, cm H_2O	Estrs, cm H_2O/L	GL, L/s/cm H_2O
Preoperative							
EELV plus 1.15±0.0 L	6.8±0.9*	5.9±0.7	13.5±1.0	7.4±0.9	20.3±1.2	13.3±1.0	0.12±0.01
EELV plus 0.62±0.0 L	3.2±0.6	5.2±0.6	8.8±0.8	6.1±0.8	12.0±0.8	11.3±1.0	0.09±0.01
EELV	0.0±0.3		5.0±0.7		5.0±0.8		
Postoperative							
EELV plus 1.04±0.1 L	10.3±1.1	8.8±1.0	11.2±1.2	8.5±0.9	21.5±1.0	17.5±1.2	0.21±0.03
p value	<0.001	<0.01	0.12	>0.10	0.93	<0.01	<0.05
EELV plus 0.58±0.0 L	6.1±0.9	8.6±1.0	7.0±0.9	7.9±0.9	13.1±0.7	16.5±1.2	0.16±0.02
p value	<0.001	<0.01	0.12	>0.10	0.80	<0.01	<0.01
EELV	1.1±0.5		2.4±0.9		3.5±0.7		
p value	0.04		0.02		0.08		

*Values are mean±SEM. P is static elastic recoil pressure, GL is lung inspiratory conductance and elastance of lung (EstL), chest wall (Estcw), and total respiratory system (Estrs) is reported.

Analysis of Inspiratory Lung Conductance

Following surgery, despite a significant reduction in lung volume (Table 1), there was a significant increase in inspiratory lung conductance ($p \leq 0.05$). The mean slope of the tangent describing the relationship between mean change in inspiratory lung conductance per mean change in inspiratory static lung elastic recoil pressure was 0.009 L/s/cm H_2O/cm H_2O prior to lung resection and 0.013 L/s/cm H_2O/cm H_2O after lung resection. This insignificant ($p > 0.05$) change suggests that the improvement in airway lumen and lung conductance is due to increased lung elastic recoil and not elasticity of the airway wall. However, the decreased lung conductance-elastic recoil pressure relationship, when compared to the normal mean value 0.10 L/s/cm H_2O/cm H_2O, suggests that intrinsic airways disease and/or bronchial compression is present and that loss of lung elastic recoil by itself does not account for the decreased airway conductance and caliber.

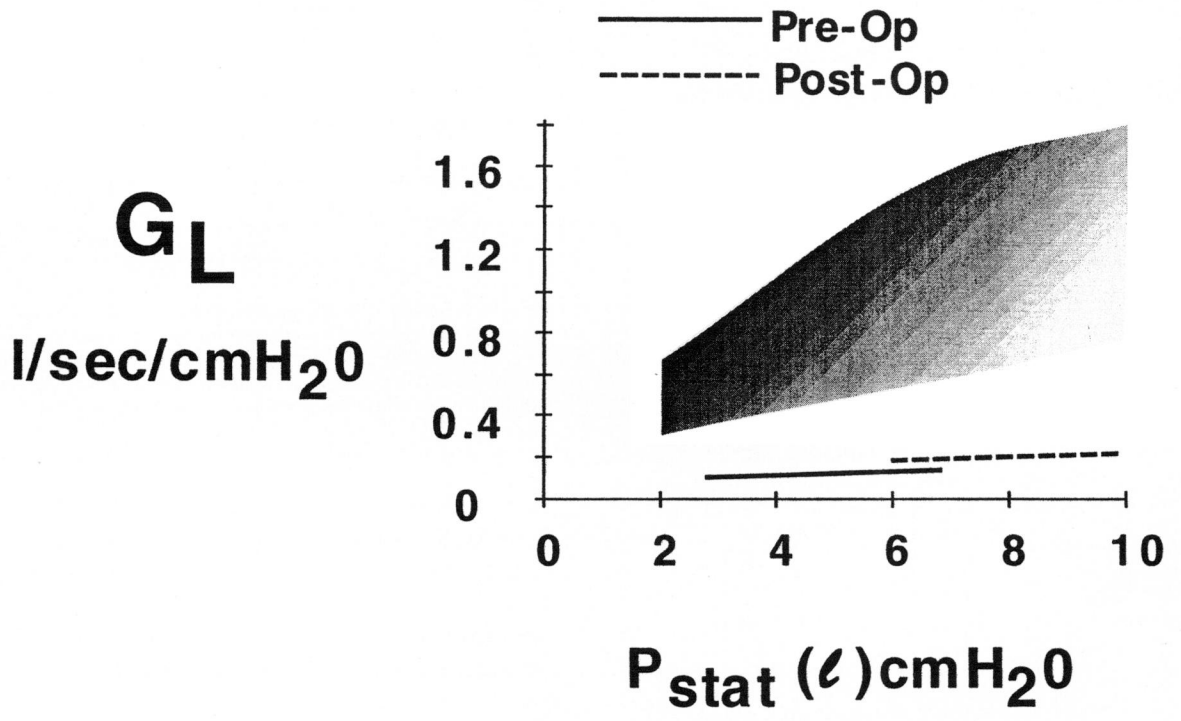

FIGURE 2. Results of lung conductance and elastic recoil following surgery. The insignificant change in the conductance recoil pressure slope suggests that the increase in lung conductance following lung volume reduction surgery for emphysema is due to increased lung elastic recoil and not elasticity of the airway wall.

Discussion

We have demonstrated that immediately following lung volume reduction surgery in patients with emphysema, there is no overall change in the total static elastic recoil pressure exerted by the respiratory system. However, there is an increase in static lung elastic recoil pressure and a decrease in static chest wall elastic recoil pressure. We believe these changes are responsible for the increased expiratory airflow and inspiratory nonelastic lung conductance observed following lung volume reduction surgery in patients with emphysema. The present study confirms and extends our results that demonstrated increased lung elastic recoil using different techniques following bilateral stapled lung volume reduction surgery.[14] Following removal of the most severe emphysematous lung, the remaining lung provides increased transmission and generation of driving pressure to increase expiratory airflow and increased stability of airways due to increased lung elastic recoil.

When the elastic recoil pressure of the total respiratory system is zero (Prs=0), the outward recoil of the chest wall (negative Pcw) is equally balanced by the inward recoil of the lung (positive P_L) and FRC is established. Normally at higher lung volumes as Prs is greater than 0 and Pcw is greater than 0, both the chest wall (Pcw) and lung (P_L) recoil inward. However, unlike the situation in patients with severe airflow limitation without severe emphysema, the relative contribution of Pcw to Prs remains considerable despite the increased compliance of the Pcw because of the marked loss of lung elastic recoil (P_L) in severe emphysema. In patients with emphysema in the anesthetized paralyzed supine position, Prs is positive at EELV despite marked loss of P_L, reflecting increased intrinsic intrathoracic pressure (PEEP-I) at Pcw, and hence, EELV is greater than FRC. Furthermore, TLC is increased because of an overall reduction in Prs, primarily by a loss of P_L and infrequently due to secondary loss of Pcw attributed to reduced inspiratory muscle pressure.[34,35]

In the present study, inspiratory static elastic recoil pressures were measured and would be increased relative to expiratory recoil pressures at any given lung volume because of hysteresis.[36-38] Furthermore, the elastic recoil pressures were measured in the supine position with a partial loss of vertical pleural pressure gradient, and the normally subatmospheric intra-abdominal pressure becomes more positive in the dependent position because of gravity. This supine positional change primarily influences the Pcw rather than P_L because of a rightward shift of the abdominal wall pressure volume curve.[29] This causes an overall expiratory effect with a shift of the Pcw and Prs volume curve to the right.[29] In addition, in normal subjects, induction of general anesthesia with muscle paralysis may result in a reduction in lung volume below FRC by shifting the Pcw volume curve to the left with a reduction in the outward recoil of the chest wall at lower lung volumes.[39-44] However, the predominant observation after induction of anesthesia in normal subjects is a reduction in lung volume and a variable increase in Prs due primarily to a variable small increase in P_L[27,41,42] that is unaffected by time beyond 10 min of anesthesia[27,42] and depth of anesthesia,[41] and cannot be prevented by lung inflation with high airway pressures.[42]

We used each patient as his or her own control, obtaining initial measurements 10 min after paralyzed anesthesia was achieved. The patient was in the same supine position using similar lung volume history, and we used similar techniques that have previously validated Pes to reflect pleural pressure under paralyzed general anesthesia.[26-28]

Polese et al[31] have previously partitioned respiratory mechanics in mechanically ventilated patients with COPD who had a similar level of airflow limitation (FEV_1) as the patients in the present study. However, their patients' FRC and TLC were markedly lower when compared with the patient values in the present study, which suggests the etiology of the COPD was primarily airways disease rather than emphysema. While the value for PEEP-I of the respiratory system at EELV was similar to the present study, Polese et al[31] noted that lung PEEP-I accounted for most of the total PEEP-I. This contrasts with our results and is probably explained by the marked loss of P_L in our patients with emphysema. Polese et al,[31] rather than constructing pressure volume curves at varying inspiratory volumes, reported elastance at EELV plus 0.78 L. Mean values for respiratory, lung, and chest wall elastance reported in Table 2 in the present study increased after lung volume reduction surgery and reached values similar to those reported by Polese et al.[31]

The significant increase in airway and inspiratory lung conductance occurred despite a significant decrease in lung volume. It has been shown previously that following nonemphysematous surgical reduction in lung volume that included conducting airways, there was a shift of the lung pressure volume curve to the right[45] but a concomitant decrease in FEV_1 and airway conductance and little change in SGaw.[4] The increased FEV_1, airway conductance, and lung conductance in the present study can be explained by the increased P_L that we measured primarily by increasing driving pressure and its secondary effect on airway traction and caliber despite a reduction in lung volume. This mechanism is similar to account for improved lung function and diaphragm strength[46] observed following

bullectomy in patients with isolated bullous lung disease[4,5] and bullous emphysema[4,6-9,46] and in generalized emphysema following lung volume reduction surgery.[13,14] Increase in diaphragmatic strength,[46] while important, however, does not increase the FEV_1 because the diaphragm is primarily an inspiratory muscle and any effect during forced exhalation is negligible.

We assumed that the resistance of the endotracheal tube *in vivo* was similar to the *in vitro* value. However, previous studies by Wright et al[47] have reported higher *in vivo* values due to kinking, compression, and secretions. We used similar inspiratory flows in each patient and it is unlikely that *in vivo* factors in each patient would account for the changes in airway and lung conductance noted in the present study. However, more than likely, we overestimated values for lung conductance.

Criticism of Study

As discussed previously in the "Materials and Methods" section, we were unable to accurately measure FRC and we used EELV to construct pressure volume curves. Postoperatively, with reduction in lung volume, we assumed EELV is equal or lower than EELV preoperatively. A lower EELV postoperatively would yield a decrease in lung elastic recoil compared to preoperative values, yet just the opposite was observed following surgery. A reduced Pew could be caused by a lower EELV postoperatively. The effects of atelectasis postoperatively could surreptitiously increase lung elastic recoil. However, Caro et al[3] have demonstrated that the increased lung elastic recoil due to atelectasis that occurs following shallow breathing with a strapped chest can be completely reversed following a *single* deep breath to TLC. All of the elastic recoil measurements in the present study were made after 3 deep inhalations of 1,000 mL which would obviate the effects of changes in elastic recoil due to atelectasis.

In summary, following lung volume reduction surgery for emphysema, despite lower lung volumes, the improvement in expiratory airflow, inspiratory lung, and airway conductance is primarily due to increased P_L. There is also a decrease in chest wall elastic recoil. The increased P_L reduces hyperinflation, primarily provides greater driving pressures to improve expiratory airflow, and secondarily increases distending traction around the airways, to improve airway caliber and conductance.

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