Mediastinal and chest wall limitations to asymmetry of lung inflation

Ken C. Lin, Anna Dizner-Golab, Robert L. Thurer, and Stephen H. Loring

Department of Anesthesia and Critical Care, and Division of Thoracic Surgery, Department of Surgery, Beth Israel Deaconess Medical Center, Harvard Medical School, Boston, Massachusetts 02215

Submitted 31 July 2003; accepted in final form 22 November 2003

Lin, Ken C., Anna Dizner-Golab, Robert L. Thurer, and Stephen H. Loring. Mediastinal and chest wall limitations to asymmetry of lung inflation. J Appl Physiol 96: 999–1004, 2004; 10.1152/japplphysiol.00807.2003.—The extent to which inflation of one lung increases pleural pressure around the contralateral lung could affect ventilatory function, e.g., after pneumonectomy or lung transplantation. The rise in contralateral pleural pressure is limited by mediastinal stiffness and other chest wall properties. To estimate these properties, we determined an elastance of asymmetric expansion (E_{Asym}) in 20 supine adults undergoing thoracic surgery requiring endobronchial intubation. Esophageal pressure, measured with a balloon catheter, was used as an estimate of pleural pressure for determining chest wall elastance during symmetric inflation. Pressures measured in the left and right lung airways during sequential symmetric inflations with known volumes were used to calculate E_{Asym} and elastances of left and right lungs by using a four-element mathematical model. Elastances (means ± SD) were 13.0 ± 8.7 (E_{Asym}), 14.0 ± 7.0 (left lung), 12.2 ± 6.1 (right lung), and 6.7 ± 2.1 cm H_{2}O/l (chest wall). E_{Asym} was high in three patients with prior cardiac surgery or mediastinal radiation therapy, suggesting that mediastinal stiffening due to scarring and fibrosis reduced pressure transmission between hemithoraces. Simulations with a previously published model showed that changes in E_{Asym} in the range of values observed could substantially affect lung ventilation after single-lung transplantation for emphysema.

METHODS

We studied 20 patients who were undergoing thoracic surgery requiring general anesthesia and a double-lumen endobronchial tube. Subjects gave informed consent for the study, which was approved by the Committee on Clinical Investigations. Characteristics and medical conditions of the subjects are listed in Table 1.

On induction of general anesthesia and pharmacological paralysis, a left double-lumen endobronchial tube (Mallinkrodt, St. Louis, MO) was placed and secured by the anesthesiologist. The endobronchial and tracheal cuffs were inflated to effect pneumatic separation of the left and right lungs, and mechanical ventilation was established with oxygen and a volatile anesthetic. An esophageal balloon catheter (Sensor Medics, Bilthoven, The Netherlands) was passed by mouth into the midesophagus, with its tip 40 cm from the incisors, and inflated with 0.5 ml of air. Pressure at the airway opening and esophagus were measured with variable reluctance transducers (Celesco model LCRV, Chatsworth, CA), and the digitized signals were displayed and recorded (WinDaq 220, DATAQ Instruments, Akron, OH).

First, we recorded esophageal pressure during 1 min of mechanical ventilation with a known tidal volume while the endobronchial airways were connected together. When the patient was oxygenated sufficiently to tolerate 1 min of apnea, the anesthesia circuit was disconnected for 10 s to allow the lungs to deflate to relaxation volume. Then, each endobronchial tube was connected to its own large syringe (AM Systems model CS-2000, Everett, WA) containing 700 ml of air. To avoid nonlinearity of the volume-pressure curve caused by airway closure at low lung volume, we initially inflated each lung with 100 ml from the syringes and allowed the pressure to equilibrate for 5 s. Left and right lungs in alternation were then asymmetrically inflated with 300-ml aliquots until a total of 700 ml had been injected into each lung (see Fig. 1). Each inflation was followed by a 5-s pause for pressure equilibration before measurement of airway pressures. The deflation and asymmetric inflations were repeated three times, with intervening periods of mechanical ventilation to restore normal end-tidal CO_{2} levels. The first and third series of inflations began with left lung inflation, and the second and fourth series began with right lung inflation. After the fourth series of inflations, the esophageal balloon was removed, and the scheduled surgery began. The entire protocol took ~10–15 min. Subjects were in the supine posture for all measurements.

To determine the extent to which respiratory structures limit asymmetric lung inflation, we calculated E_{Asym}, defined as the left-

THE MECHANICAL PROPERTIES of the mediastinum and other chest wall structures limit the degree to which left and right lungs will inflate asymmetrically under pathological conditions, such as after pneumonectomy or lung transplantation. In such conditions, the limitation to asymmetric inflation could affect individual lung ventilation. In a previous study (6), our laboratory used a mathematical model to explore mechanical factors causing respiratory dysfunction after single-lung transplantation for emphysema. In that study, the degree to which the native emphysematous lung expands asymmetrically to fill the thorax, limiting expansion of the graft, was shown to depend in part on the compliance of the mediastinum. A relatively noncompliant (stiff) mediastinum could limit the unequal inflation of transplanted and native lungs, whereas a compliant mediastinum could allow progressive expansion of the emphysematous native lung and underinflation of the mechanically normal transplanted lung. It is not known to what extent chest wall properties such as mediastinal compliance limit asymmetric inflation of lungs in humans. Therefore, in anesthetized patients, we measured an elastance (E_{Asym}) that reflects limitation of asymmetric lung expansion. E_{Asym} depends on mediastinal stiffness, the mechanical coupling of left and right sides of the chest wall that limits its asymmetric expansion, and the stiffness of the diaphragm and upper abdominal contents to displacement by a pressure difference between the two hemithoraces. E_{Asym} was comparable to elastances of lung and chest wall (E_{CW}), and thus could potentially affect the distribution of lung ventilation. Using values of E_{Asym} found in our subjects, we used the previously published model (6) to show how E_{Asym} could affect lung ventilation after single-lung transplantation.

Address for reprint requests and other correspondence: S. H. Loring, Dept. of Anesthesia and Critical Care, Beth Israel Deaconess Medical Center, 330 Brookline Ave., Dana 717, Boston, MA 02215-5491 (E-mail: sloring@bidmc.harvard.edu).

http://www.jap.org 8750-7587/04 $5.00 Copyright © 2004 the American Physiological Society

999
right difference in pleural pressures divided by the left-right difference in lung volumes, using a four-element analytic model. The calculations for each subject were done twice. In the first analysis, elastances were based on the individual’s measured value of $E_{CW}$ and, in the second analysis, on the average value of $E_{CW}$ from all subjects.

**Analytic model.** To determine the elastic impedance to asymmetric lung inflation, we used a four-element model of the respiratory system, which consisted of two compliant lungs separated by a compliant structure within a compliant chest wall. The pressure acting on the chest is the average of the pleural pressures within the hemithoraxes. In the model, the chest wall expands symmetrically; the effects of asymmetric chest expansion, which reduce $E_{Asym}$, are attributed to displacement of the compliant structure between the hemithoraxes. In the following, all volumes and pressures are changes from those at relaxation volume with the airway open.

Because the chest wall expands symmetrically, when the volumes of the right ($V_{right}$) and left lungs ($V_{left}$) are unequal, the difference in volumes must be accommodated by the mediastinal structure, whose volume displacement is $(V_{left} - V_{right})/2$. Under static conditions, the pressures expanding the lungs and chest wall are $P_{left} - P_{pl left}$, $P_{right} - P_{pl right}$, and $(P_{pl left} + P_{pl right})/2$, respectively, where $P_{left}$ and $P_{right}$ are (measured) pressures in the endobronchial airways (under static conditions, $P_{left}$ and $P_{right}$ are also the alveolar pressures), and $P_{pl left}$ and $P_{pl right}$ are the (unmeasured) pleural pressures in the hemithoraxes. The pressure displacing the mediastinum is simply $P_{pl left} - P_{pl right}$. The elastance of lungs, $E_{CW}$, and compliant dividing structure ($E_{Asym}$) are defined by the following equations

\[
E_{left} = (P_{left} - P_{pl left})/V_{left} \quad (1)
\]

\[
E_{right} = (P_{right} - P_{pl right})/V_{right} \quad (2)
\]

\[
E_{CW} = (P_{pl left} + P_{pl right})/[2(V_{left} + V_{right})] \quad (3)
\]

\[
E_{Asym} = 2(P_{pl left} - P_{pl right})/(V_{left} - V_{right}) \quad (4)
\]

where $E_{left}$ and $E_{right}$ are the elastances of the left and right lungs, respectively. Rearrangement of the equations above yields expressions for the measured variables in terms of elastances and known volumes

\[
P_{left} = E_{CW}(V_{left} + V_{right}) + E_{Asym}(V_{left} - V_{right})/4 \quad (5)
\]

\[
P_{right} = E_{CW}(V_{left} + V_{right}) + E_{Asym}(V_{left} - V_{right})/4 \quad (6)
\]

$E_{CW}$ could not be reliably determined from pressures measured during sequential inflation, so it was found first from tidal volume.

---

**Fig. 1.** Representative raw data, with esophageal pressure ($P_{es}$) and pressures in the left and right airways ($P_{left}$ and $P_{right}$, respectively) during an initial period of tidal ventilation of both lungs (while $P_{es}$ and $P_{right}$ were not recorded) followed by 4 series of asymmetric left and right lung inflations (arrows). Chest wall elastance ($E_{CW}$) was calculated from $P_{es}$ and tidal volume during tidal ventilation. All other elastances were calculated from airway pressures at the end of 5-s pauses after asymmetric inflations. Dashed vertical lines indicate intervening periods of mechanical ventilation (not recorded).
ELASTANCE OF ASYMMETRICAL INFLATION

Fig. 2. Elastance of asymmetric expansion (E_{Asym}) in 20 subjects from the first analysis, in which values are based on E_{CW} measured in each subject (A), and the second analysis, in which values are based on the average E_{CW} from all subjects (B). *Subjects with prior mediastinal radiation therapy or cardiac surgery, who, as a group, had higher E_{Asym} values than the others (P < 0.01, ANOVA).

Table 2. Elastances of left and right lungs, E_{Asym}, and E_{CW} in all subjects

<table>
<thead>
<tr>
<th>Patient no.</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>7</th>
<th>8</th>
<th>9</th>
<th>10</th>
<th>11</th>
<th>12</th>
<th>13</th>
<th>14</th>
<th>15</th>
<th>16</th>
<th>17</th>
<th>18</th>
<th>19</th>
<th>20</th>
</tr>
</thead>
<tbody>
<tr>
<td>Left lung elastance</td>
<td>19.6</td>
<td>27.8</td>
<td>22.4</td>
<td>10.4</td>
<td>12.0</td>
<td>9.7</td>
<td>12.4</td>
<td>-1.8</td>
<td>30.0</td>
<td>6.8</td>
<td>23.7</td>
<td>13.0</td>
<td>9.0</td>
<td>9.1</td>
<td>16.6</td>
<td>12.3</td>
<td>12.7</td>
<td>12.0</td>
<td>7.9</td>
<td>14.7</td>
</tr>
<tr>
<td>Right lung elastance</td>
<td>21.0</td>
<td>16.6</td>
<td>22.9</td>
<td>11.6</td>
<td>14.9</td>
<td>9.3</td>
<td>11.6</td>
<td>6.7</td>
<td>11.4</td>
<td>5.8</td>
<td>23.9</td>
<td>10.0</td>
<td>6.9</td>
<td>4.6</td>
<td>5.7</td>
<td>12.9</td>
<td>12.7</td>
<td>10.6</td>
<td>7.7</td>
<td>16.0</td>
</tr>
<tr>
<td>E_{Asym}</td>
<td>6.1</td>
<td>12.8</td>
<td>18.9</td>
<td>2.7</td>
<td>1.9</td>
<td>19.0</td>
<td>3.4</td>
<td>25.7</td>
<td>6.0</td>
<td>14.1</td>
<td>24.3</td>
<td>29.0</td>
<td>20.1</td>
<td>10.8</td>
<td>20.4</td>
<td>5.6</td>
<td>19.3</td>
<td>7.6</td>
<td>13.9</td>
<td>-2.0</td>
</tr>
<tr>
<td>E_{CW}</td>
<td>4.9</td>
<td>6.1</td>
<td>8.5</td>
<td>4.9</td>
<td>4.4</td>
<td>8.7</td>
<td>4.0</td>
<td>11.4</td>
<td>4.5</td>
<td>8.3</td>
<td>7.9</td>
<td>8.7</td>
<td>8.2</td>
<td>7.1</td>
<td>8.0</td>
<td>4.8</td>
<td>7.9</td>
<td>3.9</td>
<td>7.4</td>
<td>3.5</td>
</tr>
</tbody>
</table>

Results with the use of each individual's measured E_{CW}

<table>
<thead>
<tr>
<th>Patient no.</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>7</th>
<th>8</th>
<th>9</th>
<th>10</th>
<th>11</th>
<th>12</th>
<th>13</th>
<th>14</th>
<th>15</th>
<th>16</th>
<th>17</th>
<th>18</th>
<th>19</th>
<th>20</th>
</tr>
</thead>
<tbody>
<tr>
<td>Left lung elastance</td>
<td>16.1</td>
<td>26.8</td>
<td>25.3</td>
<td>6.9</td>
<td>7.5</td>
<td>13.9</td>
<td>7.1</td>
<td>7.7</td>
<td>25.8</td>
<td>10.2</td>
<td>26.2</td>
<td>17.0</td>
<td>12.1</td>
<td>10.1</td>
<td>19.2</td>
<td>8.7</td>
<td>15.2</td>
<td>6.4</td>
<td>9.4</td>
<td>8.3</td>
</tr>
<tr>
<td>Right lung elastance</td>
<td>17.5</td>
<td>15.5</td>
<td>27.7</td>
<td>8.1</td>
<td>10.4</td>
<td>13.5</td>
<td>6.3</td>
<td>16.2</td>
<td>7.1</td>
<td>9.2</td>
<td>26.3</td>
<td>14.1</td>
<td>10.1</td>
<td>5.5</td>
<td>8.4</td>
<td>9.2</td>
<td>15.2</td>
<td>5.0</td>
<td>9.2</td>
<td>9.6</td>
</tr>
<tr>
<td>E_{Asym}</td>
<td>13.1</td>
<td>14.8</td>
<td>3.9</td>
<td>9.5</td>
<td>10.9</td>
<td>10.7</td>
<td>14.0</td>
<td>6.7</td>
<td>14.6</td>
<td>7.5</td>
<td>19.3</td>
<td>21.0</td>
<td>13.8</td>
<td>8.9</td>
<td>15.2</td>
<td>12.9</td>
<td>14.4</td>
<td>18.8</td>
<td>10.9</td>
<td>10.8</td>
</tr>
<tr>
<td>E_{CW}</td>
<td>6.7</td>
<td>6.7</td>
<td>6.7</td>
<td>6.7</td>
<td>6.7</td>
<td>6.7</td>
<td>6.7</td>
<td>6.7</td>
<td>6.7</td>
<td>6.7</td>
<td>6.7</td>
<td>6.7</td>
<td>6.7</td>
<td>6.7</td>
<td>6.7</td>
<td>6.7</td>
<td>6.7</td>
<td>6.7</td>
<td>6.7</td>
<td></td>
</tr>
</tbody>
</table>

E_{Asym}, elastance of asymmetric expansion; E_{CW}, chest wall elastance.

RESULTS

We characterize the mechanics of asymmetric lung inflation by elastance instead of compliance because elastances were distributed over a more limited range than compliances, which included very high values (corresponding to very low elastances). When calculations were based on measured values of E_{CW} (first analysis), E_{Asym} was 13.0 ± 8.7 cmH_{2}O/l (mean ± SD), similar to the elastance of one lung, and ranged from ~0 to 30 cmH_{2}O/l (Fig. 2A, Table 2). Other elastances were 14.0 ± 7.0 (E_{left}), 12.2 ± 6.1 (E_{right}), and 6.7 ± 2.1 cmH_{2}O/l (E_{CW}). The average E_{Asym} corresponds to a compliance of 77 ml/cmH_{2}O and implies that, if the two lungs were inflated to volumes differing by 1 liter, the Pp_{left} and Pp_{right} would differ by 6.5 cmH_{2}O.

When calculations were based on the average value of E_{CW} (6.7 cmH_{2}O/l, second analysis), E_{Asym} was 12.6 ± 4.2 cmH_{2}O/l and other elastances were 14.0 ± 7.0 (E_{left}) and 12.2 ± 6.1 cmH_{2}O/l (E_{right}) (Fig. 2B, Table 2). The average values in the second analysis were nearly identical to those in the first, but the standard deviation of E_{Asym} was only one-half that in the first analysis, suggesting that much of the variation in E_{Asym} among subjects in the first analysis was due to the variation in measured E_{CW}.

One subject with prior cardiac surgery (patient 12) had the highest E_{Asym} value, and two subjects with prior mediastinal radiation therapy (patients 11 and 15) had among the highest four values of E_{Asym} in both the first and second analyses (asterisks in Fig. 2), and the average in these three subjects differed from the rest (P < 0.01, ANOVA).

Critique of methods. The values of E_{Asym} we found were on average similar to the elastance of one lung or E_{CW}; however, there was wide variation among subjects. Possible sources of variation include the use of esophageal pressure measurements and the indirect method of estimating E_{Asym}. Pleural pressure is known to vary over the pleural surface due to gravity and to differences between the unstressed shapes of the lung and its container. Therefore, a single value of pleural pressure, such as that estimated by esophageal pressure, cannot reflect pressure over the entire lung. In this study, we assume that a single
value of pleural pressure in each hemithorax is representative of the stresses applied to the lung, determining its volume. This common assumption can be justified by the relative ease with which the lung is deformed by nonuniform surface pressures, so that changes in pressure at different locations over the pleural surface are usually similar during changes in lung volume.

In the first analysis, the model produced estimates of lung $E_{CW}$ and $E_{Asym}$ with coefficients of variation ranging from 0.32 to 0.67, raising the possibility that the model was unable to estimate elastances with precision. However, the model fit the data well, accounting for >97% of the variance in the data in all subjects. Furthermore, parameter estimates were consistent within subjects; $E_{Asym}$ values estimated from the first two serial inflations, last two inflations, and all four inflations were similar in all patients (Fig. 3). The likely source of this variability in $E_{Asym}$ is variation in the value of $E_{CW}$, which was measured during mechanical ventilation and was used for computation of all other elastances. Small changes in $E_{CW}$, which ranged from 0.003 to 0.011 with a coefficient of variation of 32%, caused large changes in estimates of other parameters, especially $E_{Asym}$. Figure 4 illustrates how small changes in the value of $E_{CW}$ cause large changes in the calculated values of the other elastances. Because $E_{CW}$ was calculated from data obtained at different times and with different volumes in both lungs than were the other elastances, the values of $E_{CW}$ used may have been inappropriate. Furthermore, $E_{CW}$ was based on measurements of esophageal pressure, which is subject to artifact in supine subjects (3). This artifact would affect variability due to these effects, in the second analysis we used the average $E_{CW}$ value from all subjects for calculation of elastances in each subject. Use of the average $E_{CW}$ reduced the coefficient of variation of $E_{Asym}$ by one-half without substantially changing its average value, suggesting that the average $E_{Asym}$ reflects the average elastic impedance to asymmetric lung inflation in our subjects. Other problems were caused by intermittent failure of separation of the two lungs by the endobronchial tube, which caused rejection of 1/10 of the data from two subjects and may have caused the negative lung elastance in another (Table 2).

Our measurements of $E_{Asym}$ were made acutely and may not predict pressure changes caused by unequal volume displacements that persist longer than a few days. It is likely that prolonged unequal volume displacements, such as after pneumonectomy, cause remodeling of the chest wall and mediastinum, accommodating asymmetric inflation and reducing $E_{Asym}$. $E_{Asym}$ might also be affected by contraction of skeletal muscle, which makes it stiffer to passive stretch. Thus contraction of the diaphragm during inspiration may reduce displacement of volume from one hemithorax to the other through the abdomen and thus could cause an increase in $E_{Asym}$.

**DISCUSSION**

Pressure changes in one hemithorax displace the mediastinum and chest wall structures to change pressure in the contralateral hemithorax. This communication between the hemithoraces is of two types. The first type is due to compliant structures between the hemithoraces that are displaced by pressure differences, allowing transmission of pressures between the two sides. Thus inflation of one lung would tend to increase the contralateral pleural pressure, reducing the inflation of the contralateral lung and promoting asymmetric lung expansion. These compliant structures include the mediastinum and the upper abdominal viscera and diaphragm. Displacements of these structures by gravity can be appreciated in chest roentgenograms of patients in lateral decubitus, in whom the dependent lung is less inflated than in the nondependent lung. Another type of communication between the hemithoraces is due to mechanical coupling between the left and right sides of the rib cage, tending to equalize expansion of the two hemithoraces and prevent asymmetric chest expansion. This coupling has an opposite effect, in that a rise in pressure in one hemithorax, by causing expansion of both sides of the chest,
lowers pressure in the contralateral hemithorax and promotes symmetric inflation of the lungs. \( E_{\text{Asym}} \) in our subjects was dominated by the first type of communication, so that asymmetric expansion of one lung caused pressure applied to the contralateral lung to be higher than it would otherwise be. For modeling purposes, we attribute the combined functional contributions of these several structures to a single compliant structure between the hemithoraces. This vision seems appropriate because there is reason to believe (see below) that the mediastinum is the most compliant and, therefore, the most important of these structures in permitting asymmetric lung inflation. \( E_{\text{Asym}} \) is probably of negligible importance in the formation of the normal subject but could be important in determining each lung’s ventilation when left and right lungs have markedly different sizes or mechanical properties such as might be expected after lobectomy or single-lung transplantation, or with extensive atelectasis or unilateral emphysema. \( E_{\text{Asym}} \) may also be important when two similar lungs are subjected to different pleural pressures because of extrapulmonary factors, such as pleural effusion, paralysis of one hemidiaphragm, or gravitational effects in the lateral posture.

Mechanical effects of asymmetric lung inflation have been studied in animals. In dogs with unilateral papain-induced emphysema, Margulies et al. (7) found that the distribution of volume between emphysematous and normal lungs was the same in vivo and in vitro, suggesting that the pleural pressures were equal in the two hemithoraces, despite asymmetric inflation volumes. That would correspond to an \( E_{\text{Asym}} \) near zero. Later, Hubmayr and Margulies (4) explored the effects of unilateral lung inflation in dogs and baboons. In dogs, pleural pressure in the two hemithoraces was estimated indirectly, as in our study, and measured directly via liquid-filled cannulas through the ribs. Both methods showed that, when one lung was inflated, the rise in pleural pressure next to the inflated lung was greater than that in the contralateral hemithorax; the ratio of contralateral to ipsilateral pressure change was \(~0.7\). In baboons, the indirect technique showed a corresponding ratio of 0.5. These authors did not estimate \( E_{\text{Asym}} \), so we could not compare our results directly with theirs. However, we did estimate the relative changes in pleural pressure in the two hemithoraces in our subjects, whose \( E_{\text{Asym}} \) values were near the median, and found ratios of 0.3–0.4. Taken together, these results show a progressive increase in values of left-right pleural pressure difference from dog to baboon to human. We speculate that, from dog to human, the progression to a relatively wider mediastinum and a greater ratio of transverse to dorsoventral thoracic diameters could cause a progressive increase in effective mediastinal stiffness (and perhaps increase the tendency for the expansion of one side of the chest wall to cause symmetric expansion of the other side), thus increasing \( E_{\text{Asym}} \).

We had predicted that \( E_{\text{Asym}} \) would be increased by scarring and fibrosis of the mediastinum. Scarring of the mediastinum is expected after cardiac surgery, and mediastinal fibrosis is a well-recognized late complication of radiation therapy (8). Although limited by sample size, our findings supported our prediction. The three subjects with prior coronary surgery or mediastinal radiation therapy had among the four highest \( E_{\text{Asym}} \) values. In our model, there are several ways for volume to be displaced from one hemithorax to another, thereby equalizing \( P_{\text{pl, left}} \) and \( P_{\text{pl, right}} \). The most compliant pathway is the one through which most of the displacement occurs, and thus it largely determines the elastance of all pathways together. If the mediastinum were not the most compliant pathway determining \( E_{\text{Asym}} \), one would have expected that mediastinal stiffening by fibrosis (reducing its compliance) would have had little effect on \( E_{\text{Asym}} \). Yet we found that \( E_{\text{Asym}} \) tended to be higher in subjects suspected of having mediastinal fibrosis and conclude that the mediastinum itself is the most compliant pathway determining the extent of asymmetric lung inflation in humans. A similar conclusion was reached recently by DeGroote et al. (2), who used optical techniques to measure displacements of the chest wall in subjects who had undergone single-lung transplantation for emphysema. During forced vital capacity and maximal breathing maneuvers designed to cause asynchronous, unequal volume changes in healthy transplanted and obstructed emphysematous lungs, the left and right sides of the thorax moved equally and synchronously, leading these investigators to conclude that asymmetric lung volume changes were accommodated by displacements of the mediastinum and not by asymmetric chest wall movements (Estenne M, personal communication).

Unequal lung inflation plays a major role in several complications of thoracic surgery. The most widely recognized is postpneumonectomy syndrome, a potentially fatal complication in which a severe shift of the mediastinum leads to bronchial obstruction and respiratory failure (1). Unequal lung inflation is also observed after single-lung transplantation, where it is thought to affect postoperative lung function (9, 10). To illustrate how different values of \( E_{\text{Asym}} \) could affect individual lung ventilation and volume, we used a previously published model of respiratory mechanics in patients after single-lung transplantation (6). In this model, left and right lung mechanical characteristics, passive characteristics of the chest wall, and inspiratory muscle function were initially specified by parameters derived from measurements of a patient with severe emphysema. To simulate mechanics after single-lung transplantation, the model was modified by substituting parameters of a healthy lung for those of one emphysematous lung. Ventilation was simulated by specifying a pattern of alternating inspiratory and expiratory muscle activation to simulate hyperpnea such as that in moderate exercise. Figure 5 shows individual lung volume excursions during hyperpnea.
with EA\textsubscript{sym} values of 2 and 20 cmH\textsubscript{2}O/l, which are within the range observed in our subjects. In this model, the more compliant mediastinum (lower EA\textsubscript{sym}) was easily displaced by the difference in the two pleural pressures, allowing the hypercompliant emphysematous lung and the normally compliant transplanted lung to expand unequally when exposed to nearly the same pleural pressures. Severe expiratory flow limitation in the native emphysematous lung compounded the problem by preventing it from emptying during expiration. With a lower EA\textsubscript{sym}, the native diseased lung is more hyperinflated and the healthy transplanted lung is less inflated, causing lower expiratory flow rates and tidal volumes in the transplanted lung and 20\% less ventilation of both lungs. Relative underinflation of the transplant, whose average volume was 413 ml or 22\% lower with the lower EA\textsubscript{sym}, would make it more prone to atelectasis and ventilation-perfusion abnormalities. This simulation shows how a low value of EA\textsubscript{sym} could adversely affect ventilatory function in patients after transplantation and raises the question of whether surgery to stiffen the mediastinum could be therapeutic.

ACKNOWLEDGMENTS

The authors thank Dr. Joseph Locicero for support with the study and Richard E. Brown for a helpful critique of the manuscript.

Present address of A. Dizner-Golab: Dept. of Anaesthesiology and Intensive Care, Warsaw Medical University, 02-005 Warsaw, Poland.

GRANTS

This work was supported by the Beth Israel Anesthesia Foundation and National Heart, Lung, and Blood Institute Grant HL-52586.

REFERENCES