Mediastinal and chest wall limitations to asymmetry of lung inflation

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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 thoracic structures limit asymmetric expansion of the lungs is not well understood. The purpose of our study was to define the extent to which mediastinal and chest wall properties, such as mediastinal compliance, affect asymmetric lung inflation. We studied 20 patients who were 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 compliance during inflation. Pressures were measured in the left and right lungs during sequential symmetric and asymmetric inflations with known volumes. We calculated 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.

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 LCVR, 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 anesthesiologist disconnected the left and right lungs 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-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.

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. A mathematical model was used to explore mechanical factors causing respiratory dysfunction after single-lung transplantation for emphysema. E Asym in the range of values observed could substantially affect lung ventilation after single-lung transplantation for emphysema.
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

$$E_{left} = (P_{left} - P_{pl,left})/V_{left}$$

$$E_{right} = (P_{right} - P_{pl,right})/V_{right}$$

$$E_{CW} = (P_{pl,left} + P_{pl,right})/[2(V_{left} + V_{right})]$$

$$E_{Asym} = 2(P_{pl,left} - P_{pl,right})/(V_{left} - V_{right})$$

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

$$P_{left} = E_{CW}(V_{left} + V_{right}) + E_{left}V_{left} + E_{Asym}(V_{left} - V_{right})/4$$

$$P_{right} = E_{CW}(V_{left} + V_{right}) + E_{right}V_{right} + E_{Asym}(V_{right} - V_{left})/4$$

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

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F, female; M, male; COPD, chronic obstructive pulmonary disorder; +, with the condition.

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**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 $P_{left}$ 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).
measured by the anesthesia machine and esophageal pressure excursions during tidal ventilation, assuming that, when the left and right airways are connected together, Ppl\textsubscript{left} and Ppl\textsubscript{right} are equal to each other and to esophageal pressure. In the first analysis, we used each individual’s measured value of E\textsubscript{CW}, and in the second analysis we used the average value of E\textsubscript{CW} as an input parameter, and the other elastances were found by minimizing the sum of squared errors of Eqs. 5 and 6 during sequential inflations. (In 2 subjects, the final inflation in the first series was rejected because the lungs were not pneumatically isolated.)

**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\textsubscript{CW} (first analysis), E\textsubscript{Asym} was 13.0 ± 8.7 cmH\textsubscript{2}O/l (mean ± SD), similar to the elastance of one lung, and ranged from ~0 to 30 cmH\textsubscript{2}O/l (Fig. 2A, Table 2). Other elastances were 14.0 ± 7.0 (E\textsubscript{left}), 12.2 ± 6.1 (E\textsubscript{right}), and 6.7 ± 2.1 cmH\textsubscript{2}O/l (E\textsubscript{CW}). The average E\textsubscript{Asym} corresponds to a compliance of 77 ml/cmH\textsubscript{2}O and implies that, if the two lungs were inflated to volumes differing by 1 liter, the Ppl\textsubscript{left} and Ppl\textsubscript{right} would differ by 6.5 cmH\textsubscript{2}O.

When calculations were based on the average value of E\textsubscript{CW} (6.7 cmH\textsubscript{2}O/l, second analysis), E\textsubscript{Asym} was 12.6 ± 4.2 cmH\textsubscript{2}O/l and other elastances were 14.0 ± 7.0 (E\textsubscript{left}) and 12.2 ± 6.1 cmH\textsubscript{2}O/l (E\textsubscript{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\textsubscript{Asym} was only one-half that in the first analysis, suggesting that much of the variation in E\textsubscript{Asym} among subjects in the first analysis was due to the variation in measured E\textsubscript{CW}.

One subject with prior cardiac surgery (patient 12) had the highest E\textsubscript{Asym} value, and two subjects with prior mediastinal radiation therapy (patients 11 and 15) had among the highest four values of E\textsubscript{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\textsubscript{Asym} we found were on average similar to the elastance of one lung or E\textsubscript{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\textsubscript{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

![Fig. 2](http://jap.physiology.org/)

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

**Table 2. Elastances of left and right lungs, E\textsubscript{Asym}, and E\textsubscript{CW} in all subjects**

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E\textsubscript{Asym}, elastance of asymmetric expansion; E\textsubscript{CW}, chest wall elastance.

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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 ECW 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 ECW, which was measured during mechanical ventilation and was used for computation of all other elastances. Small changes in ECW, 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 ECW cause large changes in the calculated values of the other elastances. Because E asym was calculated from data obtained at different times and with different volumes in both lungs than were the other elastances, the values of E asym used may have been inappropriate. Furthermore, E asym was based on measurements of esophageal pressure, which is subject to artifact in supine subjects (3). This artifact would decrease the apparent value of E asym and thus decrease the calculated value of E sym. In 10 healthy subjects, we have found that the supine artifact was variable among subjects, resulting in an apparent increase in lung elastance in the supine posture of 16 ± 23% (unpublished observations). In part to avoid variability due to these effects, in the second analysis we used the average ECW value from all subjects for calculation of elastances in each subject. Use of the average ECW 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 hemithorax and the upper abdominal visceria 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,
EAsym in our subjects was probably of negligible importance in the anatomy. EAsym is probably of negligible importance in the mediastinum is the most compliant and, therefore, the most important of these structures in permitting asymmetric lung inflation. EAsym is probably of negligible importance in the mediastinum itself is the most compliant pathway determining the extent of asymmetric lung inflation in humans. A similar conclusion was reached recently by DeGroot 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.

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 EAsym 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.

Fig. 5. Simulation showing how EAsym affects ventilatory function in a patient after single-lung transplantation for emphysema. Shown are transplanted lung (Left) and native emphysematous lung (Right) volume excursions during sustained hyperpnea with 2 EAsym values (2 and 20 cmH2O/L). With the lower EAsym (higher compliance), the transplanted lung is smaller and less well ventilated, and total ventilation is 40% less than with the higher elastance.
with $E_{\text{Asym}}$ values of 2 and 20 cmH$_2$O/l, which are within the range observed in our subjects. In this model, the more compliant mediastinum (lower $E_{\text{Asym}}$) 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 $E_{\text{Asym}}$, 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 $E_{\text{Asym}}$, would make it more prone to atelectasis and ventilation-perfusion abnormalities. This simulation shows how a low value of $E_{\text{Asym}}$ could adversely affect ventilatory function in patients after transplantation and raises the question of whether surgery to stiffen the mediastinum could be therapeutic.

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