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Effect of Lung Volume on Lung Resistance and Elastance in Awake Subjects Measured during Sinusoidal Forcing

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Background: **Although lung volume may be changed by certain procedures during anesthesia and mechanical ventilation, dependence of the dynamic mechanical properties of the lungs on lung volume are not clear. Based on studies in dogs, the authors hypothesized that changes in lung mechanics caused by anesthesia in healthy humans could be accounted for by immediate changes in lung volume and that lung resistance will not be decreased by positive end-expiratory airway pressure if tidal volume and respiratory frequency are in the normal ranges.**

Methods: **Lung resistance and dynamic lung elastance were measured in six healthy, relaxed, seated subjects during sinusoidal volume oscillations at the mouth (5 mL/kg; 0.4 Hz) delivered at mean airway pressure from −9 to +25 cmH_2O. Changes in lung volume from functional residual capacity were measured with inductance plethysmographic belts.**

Results: **Decreases in mean mean airway pressure that caused decreases in lung volume from functional residual capacity comparable to those typically observed during anesthesia were associated with significant increases in both dynamic lung elastance and lung resistance. Increases in mean mean airway pressure that caused increases in lung volume from functional residual capacity did not increase lung resistance and increased dynamic lung elastance only above about 15 cmH_2O.**

Conclusions: **Increases in dynamic lung elastance and lung resistance with anesthesia can be explained by the accompanying, acute decreases in lung volume, although other factors may be involved. Increasing lung volume by increasing mean airway pressure with positive end-expiratory pressure will decrease lung resistance only if the original lung volume is low compared to awake, seated functional residual capacity. (Key words: Airway pressure. Compliance. Mechanics of breathing.)**

IT is important to understand the precise effects of varying mean lung volume on lung mechanical properties, *i.e.*, elastance and resistance. Decreases in functional residual capacity (FRC) commonly occur during the induction of anesthesia[1] and as a consequence of changes in patient positioning.[2] On the other hand, increases in FRC occur with application of positive end-expiratory pressure (PEEP). Although static elastance curves of the lung over the entire range of lung volume have been well described,[3] these curves may not be applicable to dynamic situations such as during spontaneous or artificial ventilation. For example, the static curves indicate that the elastance of the lung is constant as lung volume decreases below FRC. However, it has long been a common assumption[1] that lung elastance (E_L) increases in patients if lung volume decreases to less than FRC, as may occur during anesthesia. Also, how PEEP affects lung mechanical properties is not well characterized.

Studies of dynamically determined E_L in humans[4–7] found that elastance is increased at low or high lung volumes. However, the lung volumes at which E_L begins to increase from minimal as volume is increased or decreased from FRC are not well defined. Our previous study in humans using physiologic frequencies and tidal volumes indicated that slight (about 200 mL) decreases in lung volume around FRC can increase E_L significantly.[8] Other studies in humans have not controlled tidal volume and respiratory frequency as lung volume was changed. Because it has been shown that both of these factors affect E_L in excised lungs and that the effects depend on mean lung volume,[9,10] the relationship between E_L and lung volume is not clear.

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Airway flow resistance decreases with increasing lung volume.[11] However, the lung volume dependency of total lung resistance (R_L) is complex because it includes both airway flow and tissue contributions, and lung tissue resistance appears to increase at higher lung volume.[9,12] Moreover, the relative contributions of the tissues and airway flow in determining R_L depend on the type of forcing used for measurement: airway flow resistance increases with increasing flow,[13] whereas tissue resistance decreases with increasing frequency[9,14,15] and, to a lesser extent, with increasing tidal volume.[9,10] Although R_L, measured in different ways at various frequencies, has been shown to increase as lung volume decreases below FRC in humans,[5,6,16,17] R_L has been reported to increase,[6] decrease,[5,16] or not change[17] as lung volume increases above from FRC. None of these studies have kept frequency and tidal volume constant above FRC, and this could bias results if frequency and/or tidal volume change with lung volume. Thus, the behavior of R_L above FRC, *e.g.*, when PEEP is applied, needs to be resolved.

Based on recent measurements we made in anesthetized, paralyzed dogs,[18] we hypothesized that, in humans: (1) the changes in lung mechanics that accompany anesthesia could be due largely to the well known, acute, accompanying decreases in FRC; and (2) PEEP will not affect R_L if respiratory frequency and tidal volume are in the range of normal breathing. We systematically measured resistance and elastance of the lungs in awake human subjects during sinusoidal forcing (0.4 Hz) at a constant, physiologic tidal volume (about 500 mL) as mean airway pressure (P_{aw}) was changed to as low or as high a level as could be tolerated comfortably. Unlike most other studies, breathing pattern was constant as lung volume was changed. Results show the extent to which dynamically determined mechanics depend on lung volume in awake subjects, which has implications for anesthetized patients.

Methods

We studied six healthy, nonsmoking adult subjects after obtaining their informed consent and approval for the study from the University of Maryland Human Volunteers Committee. Table 1 lists the physical characteristics and pulmonary functions of each subject. Sinusoidal volume changes were delivered to the mouth from a piston pump driven by a linear motor. Airway flow was measured with a pneumotachograph (Fleisch #2) and a differential pressure transducer (Celesco LCVR). Similar differential pressure transducers, with one port open to atmosphere, were used to measure P_{aw} 2.0 cm from the mouth end of a rubber mouthpiece and esophageal pressure (P_{es}) *via* a polyethylene catheter attached to a latex balloon. We inserted a 2.5-cm inner diameter plastic tube inside most of the length of the mouthpiece to prevent mechanical distortion; resistance of the measuring system was too small to be measured by our methods and was considered negligible. Placement of the balloon was checked in each subject in the following way with a method described by Baydur *et al.*[19] Esophageal pressure and P_{aw} were displayed X–Y with equal gains on an oscilloscope during spontaneous breathing efforts with occluded airways: the balloon was positioned so that changes in P_{es} and P_{aw} during the efforts were equal. This test was repeated in each subject at FRC, near total lung capacity, and near residual volume. A fourth Celesco LCVR transducer was used in differential mode to measure transpulmonary pressure, the difference between P_{aw} and P_{es}. The P_{aw} signal also was passed through a 0.1-Hz low-pass filter (Rockland, Series 2000) to provide continuous measurement of mean P_{aw}.

Table 1. Physical Characteristics and Pulmonary Functions of the Subjects

Subject No.	Sex	Age (yr)	Height (cm)	Weight (kg)	Forced Vital Capacity (L)	Forced Expiratory Volume—1 s (L)
1	M	32	185	84	6.2 (109)	5.4 (119)
2	M	40	178	97	5.6 (112)	4.8 (118)
3	F	29	170	58	3.4 (86)	3.3 (102)
4	M	31	173	77	5.3 (109)	4.6 (118)
5	M	30	185	86	7.1 (124)	6.6 (142)
6	M	49	180	82	4.6 (91)	3.9 (97)

Percent predicted values in parenthesis.

Inductance plethysmographic belts (Respitrace, Ambulatory Monitoring) placed around the rib cage and abdomen were used to estimate changes in lung volume from FRC caused by increases and decreases in mean P_{aw}. For these measurements, the Respitrace amplifier was used in DC mode, and the gains of the rib cage and abdominal signals were set equal by isovolume calibration.[20] We found that, in a given subject, this isovolume calibration did not change as mean lung volume was varied from lowest to highest levels achievable. Although the rib cage and abdominal signals from the belts tends to drift in DC mode, we adjusted the signals before each measurement of lung mechan-

ics, adjusting FRC to equal zero voltage. Only occasional, minor adjustments were needed to ensure that the signals were repeatably at zero at FRC. Thus, the summed signal from the belts was a reasonable approximation for changes in mean lung volume in the relatively brief periods of respiratory mechanics measurements described below.

Each subject sat in a chair, reclined slightly from vertical to facilitate relaxation of the trunk muscles and to maintain a constant body position. After a few deep breaths, including one to total lung capacity, the subjects exhaled passively to FRC by completely relaxing their respiratory muscles. They then closed their mouths around the mouthpiece, and the pump was started. The subjects' cheeks were tightly compressed by hand to remove the variable contribution of the tissue of the cheeks to the measurements. The pump delivered a sequence of breaths at 0.4 Hz and a constant tidal volume (5 mL/kg). A flow of oxygen was added to a "T" connection close to the pump side of the pneumotachograph, and a vacuum source was used to draw out the flow from an opening in the housing of the piston. By adjusting the flows in and out of the pump–subject system, we could adjust mean P_{aw} at the mouth at desired positive and negative, steady-state levels without affecting the measured flow to the lungs caused by the imposed oscillations. This arrangement also flushed the pump–subject system to replenish the oxygen and minimize carbon dioxide rebreathing. When mean P_{aw} was constant, three consecutive breaths were measured by computer; typically, the subject was connected to the pump for about 40 s. The subject was disconnected from the mouthpiece and allowed to sit quietly for a few minutes. Then, we repeated measurements at different mean P_{aw} in a pseudorandom order. In one subject, we repeated the measurements on separate days to assess the reproducibility of the methods.

Data Analysis

The three measured breaths (sampling rate 100/cycle) at each mean P_{aw} were computer-averaged and analyzed by discrete Fourier transform at the fundamental (*i.e.*, respiratory) frequency only, to give R_L and E_L. Elastance was calculated by multiplying the imaginary part of each impedance by $-2\pi f$, thereby containing effects contributed by inertia. These effects should be negligible at the frequency used.[21,22]

Each elastance and resistance measured in a subject was normalized to the corresponding value at the mean P_{aw} that most likely would occur during normal spontaneous breathing, *i.e.* 5 cmH_2O. For each subject, these normalized data were averaged into 1-cmH_2O bins of mean P_{aw}. Then, data from all subjects were averaged. Using stepwise multiple regression, we found that the relationships of average elastance and average resistance to mean P_{aw} closely followed the third-order polynomial form. Polynomial regression was used to characterize the effects of mean P_{aw} on the average change in end-expiratory lung volume from FRC, normalized to each subject's vital capacity (VC).

Results

Lung Mechanics

In all subjects, E_L increased at low or high mean P_{aw}, although the range in which E_L was minimum varied (fig. 1). In the average data (fig. 2, upper curve) normalized to values obtained at 5 cmH_2O mean P_{aw} (average E_L 4.94 $cmH_2O/L \pm 0.40$ SE), the relationship between normalized E_L ($E_L/E_{L,control}$) and mean P_{aw} closely followed a third-order polynomial of the form:

$$E_L/E_{L,control} = 1.41 - 0.113(P_{aw}) + 0.0085(P_{aw})^2 + 0.00016(P_{aw})^3,$$

where r = 0.90, N = 36, and the SEs of the intercept and three regression coefficients are 0.086, 0.010, 0.0013, and 0.000044, respectively.

In all subjects, R_L increased as mean P_{aw} decreased below about 5 cmH_2O (fig. 1). At the lowest mean pressures, there was occasionally a tendency for the glottis to partly close (as judged subjectively and by large, transient increases in P_{aw}) during the expiratory stroke of the piston pump, which increased the measured resistance. This resulted in variability in R_L at low pressures in some subjects (*e.g.*, subjects 1 and 6). We elected to include this variability in the subsequent data analysis rather than delete it. Above 5 cmH_2O P_{aw}, there were no consistent changes in R_L. In the average data (fig. 2, lower curve) normalized to values obtained at 5 cmH_2O mean P_{aw} (average R_L = 2.04 $cmH_2O \cdot L^{-1} \cdot s \pm 0.36$ SE), the relationship between normalized R_L ($R_L/R_{L,control}$) and mean P_{aw} closely followed a third-order polynomial of the form:

$$R_L/R_{L,control} = 1.59 - 0.113(P_{aw}) + 0.0064(P_{aw})^2 - 0.0001(P_{aw})^3,$$

where r = 0.95, N = 36, and the SEs of the intercept and three regression coefficients are 0.06, 0.007, 0.001, and 0.00003, respectively. When only values

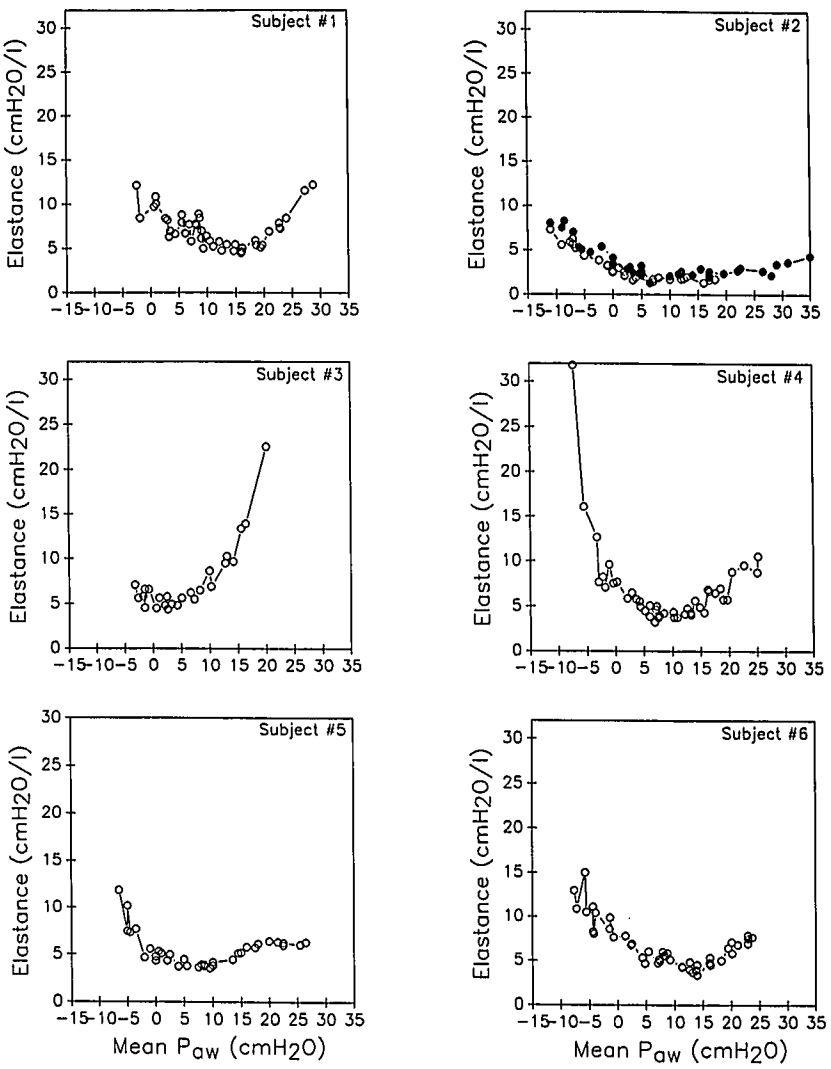

Fig. 1. Lung elastance of six healthy, awake, seated subjects during sinusoidal volume forcing at different mean airway pressures (P_{aw}). In subject 2, values from measurements repeated on different days are indicated by open and closed symbols.

above 5 cmH_2O were included in the analysis, average R_L was not affected by mean P_{aw} ($P > 0.1$).

In the subject (2) in whom measurements were repeated, values for E_L and for R_L on different days were nearly the same (figs. 1 and 3).

Relation between P_{aw} and Lung Volume

We found that the average relationship (fig. 4) between mean P_{aw} and the change in end-expiratory lung volume from FRC (normalized in each subject to VC during sinusoidal forcing) followed a sigmoidal curve best described by the following:

$$(\text{Lung volume} - \text{FRC})/\text{VC} = -0.0416 + 0.0200(P_{aw}) + 0.000348(P_{aw})^2 - 0.0000164(P_{aw})^3,$$

where r = 0.992, N = 36, and the SEs of the intercept and three regression coefficients are 0.0073, 0.00087, 0.00011, and 0.0000038, respectively. The coefficient for P_{aw} indicates that respiratory system compliance is 2% VC/cmH_2O for most of the range in VC, whereas the other coefficients indicate that compliance decreases at the extremes of lung volume.

Discussion

The sigmoidal shape of the compliance curve of the respiratory system (fig. 4) compiled from inductance plethysmography compared favorably with the curve measured with traditional methods.[3] Compliance in the mid-range of lung volume was about 0.1 L/cmH_2O. At

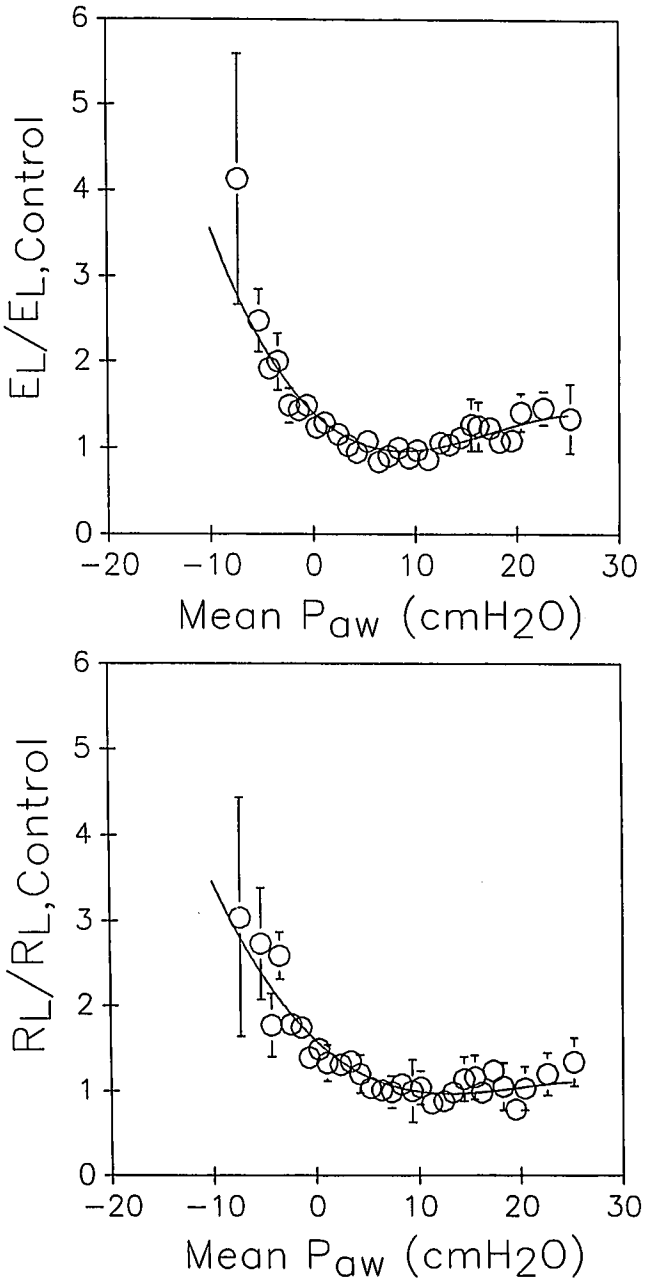

Fig. 2. Average lung elastance ($E_L/E_{L,control}$) and resistance ($R_L/R_{L,control}$) of six healthy, awake, seated subjects during sinusoidal volume forcing at different mean airway pressures (P_{aw}). Values are normalized in each subject to control values at 5 cmH_2O mean airway pressure and grouped into bins of 1 cmH_2O. SE for elastance, when larger than symbol, indicated by vertical bars; SE for mean P_{aw} was always smaller than symbol. Each point represents an average of 3–6 subjects. Polynomial regressions (see text) indicated by solid line.

about 5 cmH_2O mean P_{aw} in our study, end-expiratory lung volume equaled FRC, *i.e.*, the increase in mean lung volume from FRC was equal to about half the tidal volume. Thus, this P_{aw} represents the situation that would be observed during resting spontaneous breathing when seated. It should be noted that average E_L and R_L are minimal, or near minimal, at about 5 cmH_2O mean P_{aw}, and both increase if mean P_{aw} falls from this. Therefore, the data predict that, on average, lung impedance (which, at 0.4 Hz, comprises E_L and R_L) is minimal at the mean lung volume of normal, seated breathing. However, as seen in figures 1 and 3, there is individual variability among subjects in this respect.

The data show that the immediate effects of decreases in lung volume are sufficient to cause the significant changes reported to occur in lung mechanics during

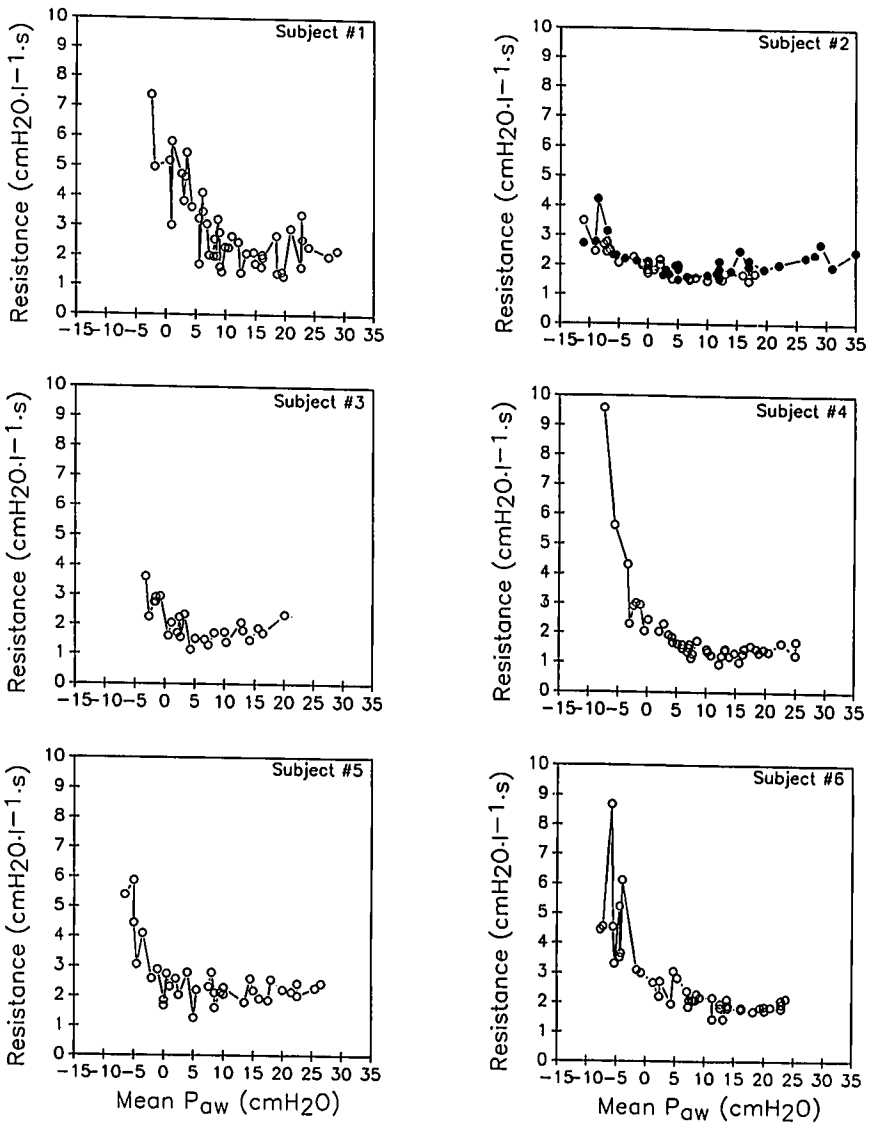

Fig. 3. Lung resistance of six healthy, awake, seated subjects during sinusoidal volume forcing at different mean airway pressures (P_{aw}). In subject 2, values from measurements repeated on different days are indicated by open and closed symbols.

anesthesia. Functional residual capacity decreases by about 16% of VC in adults changing from sitting to supine.[23] Data from a supine subject thus would be situated in the left portion of the curve in figure 4, and lung mechanics would correspond to the low mean P_{aw} portions of the curves in figure 2. In these ranges, both R_L and E_L increase if mean P_{aw} decreases. During induction of anesthesia in supine patients, lung volume further decreases by about 10% of VC,[24] and our data show that E_L and R_L could increase 50–100% with this decrease in lung volume. Although reports of measured changes in E_L and R_L induced by anesthesia have varied, most studies have found 50–100% increases.[24] Therefore, although we cannot rule out other factors, the immediate effects of decreases in FRC are sufficient to explain the changes in lung mechanics occurring during anesthesia. This is consistent with the common assumption, most recently suggested in a review by Wahba,[1] that changes in lung mechanics during anesthesia are secondary to a decrease in FRC due to changes in the chest wall. These changes are not predictable from static deflation curves of the lung, which usually indicate constant elastance below FRC.[3] One apparent exception was the curve measured by Caro *et al.*[4] showing that static E_L increased in humans at both high and low lung volumes, with or without rib cage strap-

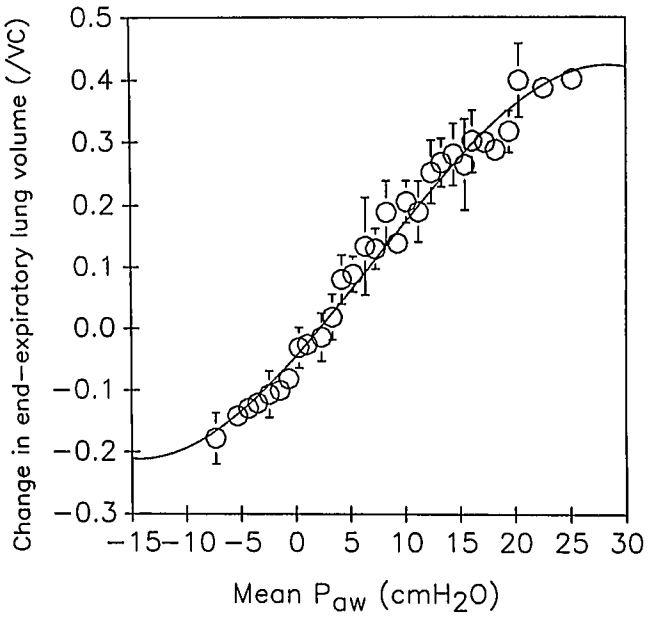

Fig. 4. Average change in end-expiratory lung volume from functional residual capacity (FRC), normalized to each subject's vital capacity (VC) during sinusoidal forcing plotted against mean airway pressure (P_{aw}). Values are grouped into bins of 1 cmH_2O. SE for lung volume, when larger than symbol, indicated by vertical bars; SE for mean P_{aw} was always smaller than symbol. Each point represents an average of 3–6 subjects. Polynomial regression (see text) indicated by solid line.

ping. However, for this curve, lung volume was plotted against mean P_{cs} during panting. Thus, the curve differs from other static curves in that the pressure contains a large dynamic component.

Anesthesia could have other effects on lung mechanics not directly caused by immediate changes in lung volume. For example, computer tomography reveals areas of lung "densities" 10–15 min after induction of anesthesia that are probably due to regional atelectasis in dependent parts of the lung.[25] These areas often are enhanced as anesthesia continues and often remain even after the anesthetic period.[25] No such lung densities are discernible in awake subjects with chest wall strapping to simulate the low lung volume measured during anesthesia.[26] Thus, anesthesia seems to cause a greater propensity toward atelectasis than occurs in awake subjects exposed immediately and transiently to a comparable lung volume. However, the present results are identical to those in dogs anesthetized with barbiturate,[18] and it is likely that the fundamental mechanical behavior of lung volume dependence is not greatly altered by anesthesia. Although inhalational anesthetics can affect bronchomotor tone,[27] it has been found that halothane does not affect the relationship between R_L and PEEP in open-chested dogs unless bronchoconstriction induced by vagal stimulation is present.[28] In summary, the present data represent the normal relationships between lung mechanical properties and fast, short-duration changes in lung volume in healthy, awake humans. It is reasonable to assume that analogous relationships exist in anesthetized humans, although this, and the effects of lung pathology, need to be studied more.

Why does E_L increase immediately at low lung volume? The increase is inconsistent with the virtually constant static E_L measured below FRC.[3] Our results indicate that this increase in E_L is immediate, already measurable after a few seconds at the low lung volume, and therefore not due to a gradual process, such as progressive atelectasis. Rather, the increase in elastance is likely due to recruitment and derecruitment of alveoli with each breath and changes in surface film kinetic behavior. In addition, at low lung volumes, airway closure probably will occur and there will be fewer open alveolar units throughout the breathing cycle. This will decrease ventilatable volume in the lungs, which will increase E_L without actually changing the mechanical properties of the lung tissue *per se.* Air trapping at low lung volumes also could lead to increases in E_L because the compressibility of the trapped gas is low, and this would tend to increase elastance. Elastance also could increase at low lung volume if lung tissue resistance increases, because the two properties seem to be closely coupled.[29] However, to our knowledge, tissue resistance below FRC has not been measured directly.

Although R_L clearly increases below FRC because of increases in airway flow resistance[11] and, possibly, lung tissue resistances, variable effects on R_L have been reported for increases in lung volume above FRC in previous studies in humans.[5,6,16,17] Lung tissue resistance, measured in excised lungs, increases at high lung volume.[9,12] Thus, there are competing tendencies for airway flow resistance to decrease and for tissue resistance to increase with increasing lung volume. Adding even greater complexity is the fact that the relative contributions of the airways and tissue to R_L will depend on the frequency and tidal volumes used for measurement. If low flows are used, tissue resistance is high[9,14,15] but airway flow resistance (which equals $k_1 + k_2 \times$ flow, where k_1 and k_2 are non-zero constants[13]) is low; at high flow rates, airway flow resistance increases but tissue resistance tends to decrease.[9,14,15] During me-

chanical ventilation, frequency and tidal volume are roughly within the normal range of spontaneous breathing. In one study in spontaneously breathing humans at relatively normal breathing pattern, there were large increases in R_L as lung volume increased above FRC.[6] However, because either frequency and/or tidal volume were not controlled in those experiments, the results are difficult to interpret. Our results, measured at constant forcing pattern, show that R_L does not consistently change with increases in lung volume above FRC in seated subjects with healthy lungs. In anesthetized supine subjects, though, lung volume may be low and a small amount of PEEP may decrease R_L, because the subjects' lung mechanics would correspond to the left portion of the curve in figure 3. Recently, interrupter resistance measured using the flow interruption technique (which supposedly represents the airway flow part of R_L), was found to not significantly increase with PEEP in patients with healthy lungs[30] or with adult respiratory distress syndrome.[30,31] However, interpretation of interrupter resistance is limited because it depends on a complex model and corresponds to an undetermined combination of forcing frequencies and amplitudes. Thus, comparisons are difficult.

From our results, we can predict changes in lung mechanics in certain clinical situations. In awake, spontaneously breathing patients with healthy lungs, FRC may be low because of a number of factors, such as posture,[2,3,23,32,33] obesity,[34] ascites,[35] pregnancy,[36] or water submergence for extracorporeal shock wave lithotripsy.[37] A combination of these factors may cause particularly low FRC. Our results show that E_L and R_L, and therefore work of breathing, will be higher than normal in these situations, to a degree that is roughly inversely proportional to FRC. However, in each condition, other, chronically developing factors may be present that affect lung mechanics in addition to those of acute changes in lung volume.

In mechanically ventilated patients, there is evidence that optimizing respiratory system mechanical properties with PEEP may be correlated to optimum oxygen delivery.[38] Use of PEEP may be an important consideration even in patients with normal lung mechanical properties if cardiovascular function and gas exchange are compromised. For example, in severe liver cirrhosis, there may be pronounced intrapulmonary shunt, ventilation/perfusion mismatch, and decreased affinity of oxygen to hemoglobin. The accumulation of ascitic fluid in this condition may produce low lung volumes in the absence of parenchymal or airways disease.[35] Our results predict that, when such patients (*i.e.*, with healthy lungs but poor oxygenation) are anesthetized and mechanically ventilated, immediate changes in R_L and E_L with PEEP will depend on the patient's baseline lung volume (*i.e.*, when no PEEP is used) relative to normal, awake FRC. This baseline will vary among patients. Lung volume probably will be low because of the anesthesia,[1] and the additional factors mentioned above may decrease FRC further. If FRC is very low, a moderate amount of PEEP will decrease both R_L and E_L. However, additional PEEP may not produce further decreases in R_L and E_L, because, as seen in figure 2, the relationships between these variables and mean P_{aw} are relatively flat as lung volume nears the range of awake, seated FRC. If baseline FRC in a patient is only slightly decreased compared to awake, seated FRC, moderate PEEP will not greatly affect R_L or E_L. In other words, it can be assumed from figure 2 that, if PEEP significantly decreases E_L and R_L, the patient's baseline FRC must have been very low. We also can predict that use of high levels of PEEP to increase lung volume more than awake, seated FRC will not change R_L and may increase E_L only slightly. In patients with lung disease or in patients in whom anesthesia may have large, additional effects besides those due to immediate decreases in lung volume, the relationships between lung mechanical properties and PEEP may differ from those in figure 2. In fact, such differences (*e.g.*, a large increase in E_L with moderate PEEP) may indicate abnormality. It will be useful to study these relationships in more detail.

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References

1. Wahba RWM: Perioperative functional residual capacity. Can J Anaesth 38:384–400, 1991

2. Lim TP, Luft UC: Alterations in lung compliance and functional residual capacity with posture. J Appl Physiol 14:164–166, 1959

3. Agostoni E, Mead J: Statics of the respiratory system, Handbook of Physiology: Respiration. Edited by Fenn WO, Rahn H. Washington, DC, American Physiological Society, 1964, pp 387–410

4. Caro CG, Butler J, DuBois AB: Some effects of restriction of chest cage expansion on pulmonary function in man: An experimental study. J Clin Invest 39:573–583, 1960

5. Mills RJ, Cumming G, Harris P: Frequency-dependent compliance at different levels of inspiration in normal adults. J Appl Physiol 18:1061–1064, 1963

6. Mueller E, Wuthe H, Petro W: Untersuchungen zur Abhaengigkeit atemmechanischer Parameter von Atemfrequenz, Dehnungslage und koerperlicher Belastung unter Einsatz der automatischen Messwerterfassung. Z Erkr Atmungsorgane 145:274–283, 1976

7. Petro W, v Nieding G, Boell W, Smidt U: Determination of respiratory resistance by an oscillation method. Respiration 42:243–251, 1981

8. Barnas GM, Campbell DN, Mackenzie CF, Mendham JE, Fahy B, Runcie C, Mendham GE: Lung, chest wall and total respiratory system resistances and elastances in the normal range of breathing. Am Rev Respir Dis 145:110–113, 1992

9. Hildebrandt J: Pressure-volume data of the cat lung interpreted by a plastoelastic, linear viscoelastic model. J Appl Physiol 28:365–372, 1970

10. Suki B, Hantos Z, Daroczy B, Alkayso G, Nagy S: Nonlinearity and harmonic distortion of dog lungs measured by low-frequency forced oscillations. J Appl Physiol 71:69–75, 1991

11. Briscoe WA, Dubois AB: The relationship between airway resistance, airway conductance and lung volume in subjects of different age and body size. J Clin Invest 37:1279–1285, 1958

12. Suki B, Bates JHT: A nonlinear viscoelastic model of lung tissue mechanics. J Appl Physiol 71:826–833, 1991

13. Rohrer R: Der Stromungswiderstand in den menschlichen Atemwegen und der Einfluss der unregelmassigen Verweigung des bronchial Systems auf den Atmungsverlauf in verschiedenen Lungenbezirken. Pflugers Arch 162:225–299, 1915

14. Brusasco V, Warner DO, Beck KC, Rodarte JR, Rehder K: Partitioning of pulmonary resistance in dogs: Effect of tidal volume and frequency. J Appl Physiol 66:1190–1196, 1989

15. Vetterman J, Warner DO, Brichant JF, Rehder K: Halothane decreases both tissue and airway resistances in excised canine lungs. J Appl Physiol 66:2698–2703, 1989

16. Nagels J, Landser FJ, van der Linden L, Clement JP, van de Woestijne KP: Mechanical properties of lungs and chest wall during spontaneous breathing. J Appl Physiol 49:408–416, 1980

17. Vincent NJ, Knudsen R, Leith DE, Macklem PT, Mead J: Factors influencing pulmonary resistance. J Appl Physiol 29:236–243, 1970

18. Barnas GM, Sprung J: Effects of mean airway pressure and tidal volume on lung and chest wall mechanics in the dog. J Appl Physiol (in press)

19. Baydur A, Behrakis PK, Zin WA, Jaeger M, Milic-Emili J: A simple method for assessing the validity of the esophageal balloon technique. Am Rev Respir Dis 126:788–791, 1982

20. Konno K, Mead J: Measurement of the separate volume changes of the rib cage and abdomen during breathing. J Appl Physiol 22: 407–422, 1967

21. Dosman J, Bode F, Urbanetti J, Antic R, Martin R, Macklem PT: Role of inertia in the measurement of dynamic compliance. J Appl Physiol 38:64–69, 1975

22. Douglas NJ, Wraith PK, Brash HM, Millar J, Sudlow MF, Flenley DC: Computer measurement of dynamic compliance: Technique and reproducibility in man. J Appl Physiol 48:903–910, 1980

23. Lumb AB, Nunn JF: Respiratory function and ribcage contribution to ventilation in body positions commonly used during anesthesia. Anesth Analg 73:422–426, 1991

24. Rehder K, Marsh HM: Respiratory mechanics during anesthesia and mechanical ventilation, Handbook of Physiology: The Respiratory System. Edited by Macklem PT, Mead J. Bethesda, American Physiological Society, 1986, pp 737–752

25. Strandberg A, Tokics L, Brismar B, Lundquist H, Hederstierna G: Atelectasis during anaesthesia and in the postoperative period. Acta Anaesthesiol Scand 30:154–158, 1986

26. Tokics L, Hederstierna G, Brismar B, Strandberg A, Lundquist H: Thoracoabdominal restriction in supine men: CT and lung function measurements. Acta Anaesthesiol Scand 30:154–158, 1986

27. Vettermann J, Beck KC, Lindahl SG, Brichant JF, Rehder K: Actions of enflurane, isoflurane, vecuronium, atracurium, and pancuronium on pulmonary resistance in dogs. ANESTHESIOLOGY 69:688–695, 1988

28. Joyner MJ, Warner DO, Rehder K: Halothane changes the relationship between lung resistances and lung volume. ANESTHESIOLOGY 76:229–235, 1992

29. Fredberg JJ, Stamenovic D: On the imperfect elasticity of lung tissue. J Appl Physiol 67:2408–2419, 1989

30. Pesenti A, Pelosi P, Rossi N, Virtuani A, Brazzi L, Rossi A: Effects of positive end-expiratory pressure on respiratory resistance in patients with the adult respiratory distress syndrome and in normal anesthetized subjects. Am Rev Respir Dis 144:101–107, 1991

31. Eissa NT, Ranieri VM, Corbeil C, Chasse M, Braidy J, Milic-Emili J: Effects of positive end-expiratory pressure, lung volume and inspiratory flow on interrupter resistance in patients with the adult respiratory distress syndrome. Am Rev Respir Dis 144:538–543, 1991

32. Behrakis PK, Baydur A, Jaeger MJ, Milic-Emili J: Lung mechanics in sitting and horizontal body positions. Chest 83:643–646, 1983

33. Nunn JF: Elastic forces and lung volumes, Applied Respiratory Physiology. London, Butterworths, 1987, pp 23–45

34. Fisher A, Waterhouse TD, Adams AP: Obesity: Its relation to anaesthesia. Anaesthesia 30:633–647, 1975

35. Krowka MJ, Cortese DA: Pulmonary aspects of chronic liver disease and liver transplantation. Mayo Clin Proc 60:407–418, 1985

36. Weinberger SE, Weiss ST, Cohen WR, Weiss JW, Johnson TS: Pregnancy and the lung. Am Rev Respir Dis 121:559–581, 1980

37. Liu WS, Wong KC: Anesthesia for genitourinary surgery, Clinical Anesthesia. Edited by Barash PG, Cullen BF, Stoelting RK. Philadelphia, JB Lippincott, 1989, pp 1105–1115

38. Suter PM, Fairley HB, Isenberg MD: Optimum end-expiratory airway pressure in patients with acute pulmonary failure. N Engl J Med 292:284–289, 1975