Lung elastic recoil during breathing at increased lung volume

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Rodarte, J. Joseph R., Gassan Noredin, Charles Miller, Vito Brusasco, and Riccardo Pellegrino. Lung elastic recoil during breathing at increased lung volume. J. Appl. Physiol. 87(4): 1491–1495, 1999.—During dynamic hyperinflation with induced bronchoconstriction, there is a reduction in lung elastic recoil at constant lung volume (R. Pellegrino, O. Wilson, G. J. Enouri, and J. R. Rodarte. J. Appl. Physiol. 81: 964–975, 1996). In the present study, lung elastic recoil at control end inspiration was measured in normal subjects in a volume displacement plethysmograph before and after voluntary increases in mean lung volume, which were achieved by one tidal volume increase in functional residual capacity (FRC) with constant tidal volume and by doubling tidal volume with constant FRC. Lung elastic recoil at control end inspiration was significantly decreased by ~10% within four breaths of increasing FRC. When tidal volume was doubled, the decrease in computed lung recoil at control end inspiration was not significant. Because voluntary increases of lung volume should not produce airway closure, we conclude that stress relaxation was responsible for the decrease in lung recoil.

hyperinflation; elastic recoil; asthma

IN A RECENT STUDY OF INDUCED bronchoconstriction by Pellegrino et al. (13), when maximal flow imposed on the control tidal flow-volume curve, functional residual capacity (FRC) increased. The breathing pattern remained essentially constant except for the increase in lung volume (13). Before the increase in FRC, bronchoconstriction produced an increase in elastance with no consistent effect on lung elastic recoil at control FRC. Bronchoconstriction severe enough to produce an increase in FRC was associated with a further increase in elastance, but lung recoil at the elevated FRC was systematically less than predicted than the elastance at the initial FRC. This phenomenon occurred in both asthmatic patients and normal subjects who achieved sufficient bronchoconstriction to increase their FRC. The decreased lung elastic recoil reduced the increase of elastic work of breathing produced by the hyperinflation. There was no statistically significant change in lung elastic recoil from static deflation pressure-volume (P-V) curves from total lung capacity (TLC). The less-than-expected increase in lung recoil that occurred with the increase in mean lung volume could have been caused by airway closure induced by bronchoconstrictor agents or by stress relaxation produced by the increased mean lung volume. Voluntary increases in FRC should not produce airway closure. If voluntary increases in lung volume produced a reduction in lung elastic recoil, it would suggest that stress relaxation was responsible. Therefore, we studied the effects on lung recoil of voluntary increases of FRC and mean lung volume.

METHODS

Eight normal men, age 29–39 yr, were studied in a pressure-corrected, integrated flow-volume displacement plethysmograph. Anthropometric data are shown in Table 1. The frequency response of this plethysmograph is adequate up to 10 Hz. Volume measurements were obtained by measuring the pressure difference across a resistance element located in the wall of the plethysmograph with an MP45 Validyne pressure transducer (±2 cmH2O). This signal was then integrated and corrected for the phase lag because of the pneumatic capacitance of the plethysmograph to obtain volume. The characteristics of this type of plethysmograph are described elsewhere (11, 14). Respiratory flow was measured by a no. 3 Fleisch pneumotachograph connected to an MP45 Validyne pressure transducer (±2 cmH2O). Transpulmonary pressure (Ptp) was measured by a 10-cm-long thin latex balloon positioned in the lower one-third of the esophagus, 38–45 cm from the nostril, and connected to a Statham 131 pressure transducer (±5 psi). The balloon was filled with ~1 ml of air. Ptp was estimated as the difference between mouth and esophageal pressure. Placement of the balloon was considered correct if Ptp remained constant while subjects made gentle respiratory efforts against a small orifice. Oral pressure changes confirmed respiratory efforts. Signals of flow, volume, and Ptp were recorded on a strip-chart recorder (HP-7758A) and digitally collected with a computer (DEC 11/73) at a sample rate of 50 Hz for subsequent analysis.

Protocol. Subjects were instructed to breathe through the mouthpiece continuously throughout the study to achieve a steady-state heat transfer and to minimize thermal drift during measurements. Subjects breathed quietly for 3–5 min without specific instructions, except to avoid taking a deep breath while the integrator was adjusted to minimize thermal drift. The integrator was adjusted so that the reset button would return the volume to control end inspiration, which was recorded as zero volume by the on-line computer. The gain of a time-based oscilloscope was adjusted so that volume during normal tidal breathing fell between the first and second of three equally spaced lines on the display. At the beginning of data collection, this oscilloscope was placed so that subjects could see it, and they were instructed to
to "breathe between the first two lines" for 1 min [normal tidal volume (VT)] and then perform one of two experimental maneuvers. 1) VT was held constant, but FRC was increased so that the initial end-inspiratory volume became the new FRC and volume excursion was from lines 2–3 on the oscilloscope (VT1 pattern; Fig. 1A). 2) The VT was doubled so that VT excursion on the oscilloscope was from lines 1–3 rather than 1–2 (2VT pattern; Fig. 1B). The sequence of the maneuvers was random, and each lasted for 1 min. At the end of each imposed breathing pattern, the airway was occluded at the volume corresponding to control end inspiration, which was end expiration for the VT1 pattern and mid-VT for the 2VT pattern. Thoracic gas volume was measured, and the subjects performed a maximal inspiration to TLC.

Data analysis. Ptp, flow, and volume for each breath were analyzed by the least squares method. The start of inspiratory flow (V˙I) was determined by searching back from a definite inspiration to the first flow point.0. End-expiratory flow is taken as the last point,0 in the expiratory phase of the breath. Each breath was examined with one elastance term and a separate pulmonary resistance for inspiration (RLi) and expiration (RLe) (12). After the zero flow points are determined, the V˙I is forced to zero during expiration, the expiratory flow (V˙E) is forced to zero during inspiration, and the data are fit to the following equation

\[
P_{\text{tp}} = P_0 + E_{\text{dyn}} \cdot V + R_L \cdot \dot{V}_I + R_L \cdot \dot{V}_E
\]

where \(P_0\) is Ptp at zero volume, which is control end inspiration; \(E_{\text{dyn}}\) is dynamic elastance; and \(V\) is volume relative to zero. Lung elastic recoil for any volume within the tidal-breathing range was computed by solving the equation with both flows set to zero. The line formed by this computation is indistinguishable from the line connecting points of zero flow, but the slope has much less breath-to-breath variability. We fitted the data for each of the four breaths before and after changing the ventilatory pattern for each subject.

Statistics. \(P_0\), RLi, RLe, and Edyn for breaths before and after the breathing pattern of each subject was changed were analyzed by repeated-measures ANOVA at each of the breathing patterns. TLC at the end of the two breathing patterns was compared by paired \(t\)-test.

RESULTS

There were no differences in TLC measured after the two maneuvers or \(P_0\), RLi, RLe, and Edyn before and after the change in breathing pattern. Figure 1A illustrates both the change in breathing pattern and changes in Ptp for a representative subject when FRC was increased. The changes in lung volume were not associated with proportionate changes in Ptp. This effect occurred within the first few breaths. Corresponding transpulmonary P-V loops and the fit for the dynamic pulmonary elastance for representation breaths of this same subject are shown in Fig. 2A. For the VT1 pattern, the computed elastic component of the dynamic loop was reduced after the increase of FRC relative to the extrapolation of the control breath. The effect of doubling VT in the same subject is shown in Fig. 2B. For the 2VT pattern, the amplitude of the pressure doubled, and there was essentially no change in pressure at zero volume in this subject. There were no differences in RLi and RLe between any of the breathing patterns. Elastic recoils and Edyn for each subject are shown in Table 2. The VT1 pattern was associated with a 10.6% decrease in mean elastic recoil at the common volume, control end inspiration (\(P < 0.001\)). The 2VT pattern was associated with a smaller decrease in elastic recoil by 10.2 + 0.3 on June 9, 2017 http://jap.physiology.org/ Downloaded from

Table 1. Anthropometric characteristics of subjects

<table>
<thead>
<tr>
<th>Subject</th>
<th>Gender</th>
<th>Age, yr</th>
<th>Height, cm</th>
<th>Smoker</th>
<th>FVC, % Pred</th>
<th>FEV1, % Pred</th>
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<tr>
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<td>M</td>
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<td>29</td>
<td>173</td>
<td>N</td>
<td>5.75</td>
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</tbody>
</table>

M, male; N, no; Y, yes; FVC, forced vital capacity; % Pred, percent predicted; FEV1, forced expiratory volume in 1 s.

Fig. 1. Representative relative volume and transpulmonary pressure (Ptp) over time during control and 2 experimental breathing patterns. A: tidal volume (VT1); B: 2VT. See METHODS for details. Note that Ptp does not shift by same magnitude as volume.
recoil at the same volume and was not statistically significant (P, 0.27). There were small but statistically significant increases in elastance with both VT1 and VT2 patterns.

**DISCUSSION**

Comments on the methodology. Separating RL and RLe provides a better fit for the dynamic P-V relationship than a single resistance. RLe is greater than RLi, as would be expected from the changes of intrathoracic airway transmural pressure and glottic aperture. If a single resistance is used when RLe and RLi are quite different, the regression analysis will accommodate by shifting the computed elastic P-V relationship away from the side of the loop with the higher resistance. Therefore, using both RL and RLe provides a better estimate of the elastic pressure. The elastic component of the dynamic P-V loop over the tidal-breathing range is estimated by plotting PLFRC, where PLFRC is Ptp at FRC. This line is essentially indistinguishable from the straight line connecting the P-V relationship at instants of zero flow but has less breath-to-breath variability. Because zero flow on every breath during the control and VT1 pattern did not exactly occur at our mean control end-inspiratory volume, zero volume, this methodology allows us to estimate the dynamic elastic pressure at that volume with reduced variability. However, when we computed elastic recoil at end inspiration during control breathing and end expiration during the VT1 pattern, virtually identical results were obtained because our subjects were quite

| Table 3. Effects of doubling tidal volume (2VT pattern) |
|-----------------|----------|----------|-----------------|-----------------|
| Elastic Recoil, cmH2O | Elastance, cmH2O/l | Inspiratory Resistance, cmH2O·l−1·s | Expiratory Resistance, cmH2O·l−1·s |
| Before            | 6.3      | 3.8      | 1.5             | 1.7             |
| After             | 5.9      | 4.0      | 1.4             | 2.1             |
| Difference        | -0.4     | 0.21     | -0.06           | 0.4             |
| P                 | 0.27     | 0.04     | 0.74            | 0.12            |

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skilled at accomplishing the requested respiratory pattern.

Because the P-V relationship of the lung is not linear, elastance is an approximation of the P-V relationship. If the P-V relationship were a single exponent, then elastance would be a linear function of the mean lung volume. Thus the increase in elastance in both the VT+1 and 2VT patterns is expected. Use of a linear relationship slightly underestimates the lung recoil at both extremes of volume and should not systematically bias our comparison between the control and VT+1 data. Linear analysis would overestimate the elastic pressure at mid-VT of an exponential P-V relationship slightly underestimates the lung recoil at zero volume when VT was doubled.

Effects of increased VT. Doubling VT produced the same end-inspiratory lung volume but a lower mean volume than did increasing FRC (Table 3). The decrease in recoil at control end inspiration was smaller and more variable and did not achieve statistical significance. The increase in elastance was also smaller but was still significant. As noted above, linear analysis of an exponential P-V curve tends to overestimate lung recoil at mid-VT. Because zero volume is end inspiration under control conditions and is mid-VT during increased VT, there is a systematic bias that would underestimate a decrease in elastic recoil associated with this breathing pattern.

It has been appreciated for sometime that reductions in FRC caused by chest strapping increased lung recoil (1, 3, 16). This increase in recoil is functionally significant and is associated with an increase in maximal VE at constant volume (16). The preponderance of evidence suggests that it is not due to atelectasis but may be due to changes in lung surface tension (9, 15). Changes in lung surface tension are one of the potential mechanisms for stress adaptation that would produce an increase in lung recoil when lung volume is reduced below its usual value and a reduction in lung recoil when mean lung volume is increased.

We are unaware of any previous study reporting elastic recoil during voluntary increases in mean lung volume. In a previous study of static deflation P-V curves from TLC after subjects breathed for 45–60 s at very high mean lung volumes, one of three normal subjects showed a decrease in the static lung recoil pressure, but the other two did not (8). Several studies have shown decreases in TLC and increases in static deflation elastic recoil at constant lung volume after relief of bronchoconstriction with beta agonist (4, 7). However, in these studies, TLC and airway resistance were determined by body plethysmography before the demonstration that high-frequency panting causes overestimates of lung volume in patients with severe airway obstruction (2). Therefore, artifact may contribute to these results. However, high doses of beta agonist cause decreases in static lung elastic recoil on deflation from TLC in normal individuals, presumably due to relaxation of contractile tissue rather than changes in surfactant (4, 10). Lung stretch is associated with a release of surfactant in excised rat and dog lungs inflated proportionately to the degree of inflation, and stretch causes release of surfactant from alveolar type II cells in vitro (5, 6). The present study provides strong evidence that, when mean lung volume is increased, there is a rapid decrease in lung recoil due to stress relaxation. It provides no evidence as to whether this is due to a release of surfactant onto the alveolar surface, relaxation of contractile elements, or a viscoelastic deformation of other parenchymal elements.

In one subject, we administered methacholine in a dose sufficient to increase mean resistance by 64%. This dose was not sufficient to cause flow limitation in the tidal-breathing range or to increase FRC. The computed elastic components of the dynamic P-V loops are shown in Fig. 3. In the control case, both VT+1 and 2VT patterns were associated with a decrease in lung elastic recoil and a minimal increase in elastance. With bronchoconstriction, lung recoil at FRC and lung elastance increased relative to control. The VT+1 breathing pattern was associated with a larger decrease in lung recoil at zero volume than during the previous study. There was also a decrease in resistance with the increased mean lung volume because of the bronchodilator effect of increased mean lung volume in normal subjects during bronchoconstriction. We cannot exclude the possibility that increased elastance during bronchoconstriction with a normal breathing pattern in the present or previous study (13) was due to parallel inhomogeneity up to and including airway closure. In contrast to the situation in which FRC is increased because of the airway constriction, the changes occurring with voluntary increase in lung volume, both before and after bronchoconstriction, cannot be due to airway closure. The data in this study are most consistent with stress adaptation causing the reduction in recoil.

Asthmatic patients with severe bronchoconstriction both increase their elastance and become dynamically hyperinflated. If the dynamic P-V relationship followed the trajectory of the P-V curve before the increase in FRC, there would be a substantial increase in the elastic work of breathing, which could contribute to respiratory muscle fatigue and failure. The shifts in the P-V curve do not eliminate, but substantially reduce, this increased elastic work.

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