Pulmonary, Chest Wall, and Lung-Thorax Elastances in Acute Respiratory Failure*

Jeffrey A. Katz, M.D.;† Steven E. Zinn, M.D.;†
Gerard M. Ozanne, M.D.;† and H. Barrie Fairley, M.B., B.S.‡

The usefulness of lung-thorax compliance (or elastance) as an index of pulmonary compliance (or elastance) was examined in 15 patients being ventilated for acute respiratory failure. Mean lung-thorax elastance ($E_{LT}$) was $27.9 \pm 2.6$ cm H$_2$O/L, and the chest wall accounted for 34 ± 2 percent of the mean total value. Changes in $E_{LT}$ caused by increments of positive end-expiratory pressure correlated only with changes in pulmonary elastance ($r = 0.96; P < 0.001$) and not with chest wall elastance, although individual patients varied as to the contribution of the chest wall component. Lung-thorax elastance increased in direct proportion (1:1) to increases in pulmonary elastance, whereas the changes in lung-thorax compliance were only half those in pulmonary compliance. We conclude that elastance is a more useful clinical index than compliance.

Lung-thorax or total compliance ($C_{LT}$) has been advocated as a measurement to assess the pulmonary status of patients with acute respiratory failure requiring mechanical ventilation. It has been suggested that $C_{LT}$ reflects pulmonary compliance ($C_L$) and that the reduction in both compliances may be due to decreased pulmonary volume. This hypothesis is based on the shape of the normal curve for chest wall compliance ($C_{CW}$), which is relatively linear at and above functional residual capacity (FRC) in the supine position. When $C_L$ is abnormally low, it then becomes the major numerical contribution to $C_{LT}$. Frequently, patients with acute respiratory failure have abnormal chest wall mechanics, eg, abdominal distention, chest wall edema, and pleural effusions. In such cases, low $C_{LT}$ may reflect a low $C_{CW}$ and not a pulmonary abnormality. The ability to differentiate between $C_L$ and $C_{CW}$ would therefore aid in differential diagnosis and ultimately better direct therapy specifically to the lung or chest wall.

The clinician commonly uses compliance to reflect elastic recoil; however, elastance (the reciprocal of compliance) reflects a more linear distribution of elastic recoil when examining the lung and chest wall, since these components are in series. Also, the relationship between elastance and compliance (Fig 1) is such that disorders causing a given increase in elastic recoil result in progressively smaller changes in units of compliance and larger increases in elastance. Thus, when the components of lung-thorax elasticity are evaluated, elastance is the more appropriate index.

The airway pressure (Paw) required to inflate the total respiratory system is the sum of the pressure required to inflate the lung plus the pressure required to inflate the chest wall. Two distinctly opposite clinical situations may occur. In one, the chest wall has a high elastance (low compliance); the abnormally high Paw that is required to inflate the chest wall is transmitted across the lung to the intrapleural and vascular spaces and contributes to

*From the Department of Anesthesia, University of California, San Francisco General Hospital, San Francisco. Supported in part by grant GM240223.
†Assistant Professor of Anesthesia.
‡Professor of Anesthesia.
Reprint requests: Dr. Katz, San Francisco General Hospital, 1001 Potrero Street, San Francisco 94110

![Figure 1. Reciprocal relationship of elastance and compliance. Mean (arrows) and ranges of lung-thorax, pulmonary, and chest-wall data.](image-url)
a decrease in ventricular filling and cardiac output. At the other extreme, where the lung has an increased elasstance, less of the Paw is transmitted to the pleural cavity, and cardiac output is less affected. In extreme cases, pulmonary vascular resistance is increased, and this may then be a factor limiting cardiac output. Additionally, in this group of patients with abnormal lungs, the high Paw may contribute to barotrauma. Thus, there are important clinical reasons to be able to distinguish between the components of lung-thorax elastance (ELT).

We examined the components of ELT in a group of patients requiring mechanical ventilation for acute respiratory failure, in order to determine if changes in ELT reflected changes in the lung, the chest wall, or both, when positive end-expiratory pressure (PEEP) was used to increase pulmonary volume.

MATERIALS AND METHODS

We studied 25 patients (20 men and five women, ages 21 to 73 years of age) requiring mechanical ventilation for acute respiratory failure. Respiratory failure followed massive trauma in 11 patients, a major surgical procedure in four, and either a metabolic or infective process in ten. All patients were stable with respect to cardiopulmonary variables, and no patient was studied within 24 hours of starting mechanical ventilation.

All patients were ventilated with a volume or time preset ventilator (Monaghan 225 or Siemens Servo 900B) and received periodic morphine intravenously to facilitate mechanical ventilation. The tidal volume (VT) was adjusted to deliver 14 ± 0.2 percent of total lung capacity (TLC), as predicted from height and age. The inspiratory flow, respiratory frequency, and inspired oxygen concentration were held constant throughout the period of study. All patients were in the supine position.

It was not possible to collect all data on all patients, due to various clinical constraints. Therefore we separated the data into two groups.

Relation of ELT to FRC

In 25 patients, ELT and FRC were measured at 3 cm H2O of PEEP (3 cm H2O of PEEP was used as the initial level, since most ventilators do not return to zero end-expiratory pressure). In addition, in six of these patients (randomly selected), we examined the effect of two different histories of pulmonary volume on FRC and ELT, as follows: in three, we determined the effect on FRC and elastance of two hyperinflations to an end-inspiratory plateau pressure (Paw) greater than 50 cm H2O. In three other patients, we determined the effect of constant ventilation with a large TV (approximately 20 percent of TLC).

Pulmonary Elastances (ELT)CHEST, 80: 3, SEPTEMBER, 1981

PULMONARY ELASTANCES IN ACUTE RESPIRATORY FAILURE 395

FIGURE 2. Continuous esophageal trace with spontaneous ventilation. Arrow signifies point of reversal of pressure, indicating that top of esophageal balloon had entered esophagus from stomach.
supine subjects was assumed to be 80 percent of the value for subjects in the sitting position.12,13

The study was approved by our committee on human
research, and informed consent was obtained for each
patient. Statistical analysis was performed with methods
of least squares linear regression and two-way analysis of vari-
ance, with differences determined between groups using the
Newman-Keuls test.14

RESULTS

Relation of $C_{LT}$ and $E_{LT}$ to FRC

Table 1 lists the compliances, elastances, FRC,
correlated with changes in the chest wall component (patients 3 to 5, and 7) (Table 4).

**Discussion**

Lung-thorax compliance or pulmonary compliance (or elastance) may be altered by pulmonary volume, surface tension, disease, recent ventilatory history, size of TV, chest wall mechanics, pulmonary blood volume, and posture.15

Our finding of a strong relationship of both CLT and C_l to FRC agrees with data from studies in normal man16 and observations made in prior studies on patients with acute respiratory failure.6,12,17 Petty et al18 recently demonstrated an increased surface compressibility of fluid from bronchoalveolar lavage, a reduced pulmonary volume, and a reduced static C_l in patients with acute respiratory distress syndrome, as compared with normal subjects. The observed decreases in FRC and compliance in our series may have been due to such a mechanism.

Hyperinflating the lungs of normal and anesthetized man increases compliance by as much as 30 to 50 percent.19-21 This increase is temporary, and baseline values return after several breaths.22,23 Furthermore, this effect is mainly seen when the lungs have previously been ventilated with low TVs, and is less effective after ventilation with large TVs.23,24 The increase in compliance presumably results from recruitment of collapsed alveoli or a change in the surface properties of alveolar surfactant (or both).19,25 In our study, we found little or no change in ELT or FRC when two separate patterns of ventilation were used. The apparent discrepancy is probably related to a small lag in time in this study (eight breaths) between hyperinflation and measurement of FRC and to the inability of patients with acute respiratory failure to maintain recruited lung without the application of PEEP. Also, associated pathologic changes may have involved consolidative (space-occupying) processes.

---

**Table 3—Effect of TV on FRC**

<table>
<thead>
<tr>
<th>Patient</th>
<th>TV, L</th>
<th>FRC, L</th>
<th>TV, L</th>
<th>FRC, L</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>0.99</td>
<td>1.28</td>
<td>1.58</td>
<td>1.30</td>
</tr>
<tr>
<td>24</td>
<td>0.73</td>
<td>1.08</td>
<td>1.40</td>
<td>1.07</td>
</tr>
<tr>
<td>25</td>
<td>0.71</td>
<td>1.56</td>
<td>1.22</td>
<td>1.58</td>
</tr>
</tbody>
</table>

---

**Table 2—Effect of Hyperinflation on FRC and ELT**

<table>
<thead>
<tr>
<th>Patient</th>
<th>Before Hyperinflation</th>
<th>After Hyperinflation</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>FRC, L</td>
<td>ELT, cm H₂O/L</td>
</tr>
<tr>
<td>11</td>
<td>1.50</td>
<td>21.3</td>
</tr>
<tr>
<td>22</td>
<td>0.75</td>
<td>23.3</td>
</tr>
<tr>
<td>23</td>
<td>1.96</td>
<td>15.9</td>
</tr>
</tbody>
</table>

---

**Figure 3. Relationship of CLT to FRC.**

and clinical diagnosis for each patient. Mean FRC at 3 cm H₂O of PEEP was 1.46 ± 0.13 L (53 ± 5 percent of predicted FRC for supine subjects) and correlated with CLT (r = 0.78; P < 0.001; Fig 3). This correlation was mainly the result of C_l (r = 0.73; P < 0.002) and not Ccw (r = 0.5; not significant). The ELT correlated inversely with FRC (at 3 cm H₂O of PEEP) (r = -0.65; P < 0.001), as did both its components (for EL, r = -0.59 and P < 0.02; for ECW, r = -0.54, and P < 0.05). The FRC did not change in three patients before and after hyperinflation (Table 2). In three other patients, ventilation with large TVs also did not alter FRC (Table 3).

**EL, ECW, and ELT**

Mean ELT at 3 cm H₂O of PEEP (N = 15) was 27.9 ± 2.6 cm H₂O/L. The EL was 18.5 ± 2.1 cm H₂O/L, and ECW was 9.4 ± 0.8 cm H₂O/L. Mean values for CLT, C_l, and CCW were, respectively, 0.036, 0.054, and 0.106 L/cm H₂O. The chest wall component accounted for 34 ± 2 percent of the total ELT (Table 1).

Stepwise changes in EL with increments of PEEP correlated directly with stepwise changes in ELT (r = 0.96; P < 0.001; Fig 4A and Table 4). In addition, changes in CLT correlated with changes in CLT (r = 0.92; P < 0.001; Fig 4C). Although changes in both EL and CL correlated well with changes in ELT and CLT, their regression slopes dif-
Our data are consistent with those of a prior study that showed no increase in dynamic compliance with periodic sighs.26

Table 4—FRC, E_L, E_CW, and E_LT at Differing Levels of PEEP

<table>
<thead>
<tr>
<th>Case</th>
<th>FRC, cm H_2O/L</th>
<th>E_LT, cm H_2O/L</th>
<th>E_L, cm H_2O/L</th>
<th>E_CW, cm H_2O/L</th>
<th>FRC, cm H_2O/L</th>
<th>E_LT, cm H_2O/L</th>
<th>E_L, cm H_2O/L</th>
<th>E_CW, cm H_2O/L</th>
<th>FRC, cm H_2O/L</th>
<th>E_LT, cm H_2O/L</th>
<th>E_L, cm H_2O/L</th>
<th>E_CW, cm H_2O/L</th>
<th>FRC, cm H_2O/L</th>
<th>E_LT, cm H_2O/L</th>
<th>E_L, cm H_2O/L</th>
<th>E_CW, cm H_2O/L</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>1.08</td>
<td>19.2</td>
<td>11.3</td>
<td>7.9</td>
<td>1.42</td>
<td>21.3</td>
<td>14.4</td>
<td>6.9</td>
<td>1.82</td>
<td>23.0</td>
<td>16.3</td>
<td>6.7</td>
<td>2.50</td>
<td>23.0</td>
<td>16.3</td>
<td>6.7</td>
</tr>
<tr>
<td>2</td>
<td>1.31</td>
<td>24.4</td>
<td>16.1</td>
<td>8.3</td>
<td>1.48</td>
<td>19.2</td>
<td>12.0</td>
<td>7.2</td>
<td>1.83</td>
<td>20.0</td>
<td>12.7</td>
<td>7.3</td>
<td>2.31</td>
<td>30.3</td>
<td>21.3</td>
<td>8.9</td>
</tr>
<tr>
<td>3</td>
<td>1.54</td>
<td>24.2</td>
<td>13.5</td>
<td>10.7</td>
<td>1.76</td>
<td>27.4</td>
<td>14.0</td>
<td>13.4</td>
<td>1.95</td>
<td>24.7</td>
<td>12.7</td>
<td>12.0</td>
<td>2.28</td>
<td>23.8</td>
<td>11.6</td>
<td>12.2</td>
</tr>
<tr>
<td>4</td>
<td>2.70</td>
<td>17.5</td>
<td>10.0</td>
<td>7.5</td>
<td>3.18</td>
<td>15.6</td>
<td>10.0</td>
<td>5.6</td>
<td>3.57</td>
<td>19.7</td>
<td>12.7</td>
<td>7.1</td>
<td>4.23</td>
<td>35.0</td>
<td>28.3</td>
<td>6.7</td>
</tr>
<tr>
<td>5</td>
<td>0.80</td>
<td>40.1</td>
<td>29.3</td>
<td>10.8</td>
<td>0.96</td>
<td>38.7</td>
<td>29.3</td>
<td>9.4</td>
<td>1.15</td>
<td>44.0</td>
<td>31.6</td>
<td>12.4</td>
<td>1.31</td>
<td>51.5</td>
<td>37.9</td>
<td>13.6</td>
</tr>
<tr>
<td>6</td>
<td>1.07</td>
<td>32.3</td>
<td>18.9</td>
<td>13.5</td>
<td>1.15</td>
<td>27.0</td>
<td>14.1</td>
<td>12.9</td>
<td>1.39</td>
<td>26.5</td>
<td>14.9</td>
<td>11.6</td>
<td>1.81</td>
<td>27.3</td>
<td>15.9</td>
<td>11.4</td>
</tr>
<tr>
<td>7</td>
<td>1.44</td>
<td>20.8</td>
<td>14.0</td>
<td>6.8</td>
<td>1.64</td>
<td>22.7</td>
<td>16.9</td>
<td>5.8</td>
<td>1.88</td>
<td>25.6</td>
<td>19.2</td>
<td>6.3</td>
<td>2.36</td>
<td>29.2</td>
<td>20.0</td>
<td>9.1</td>
</tr>
<tr>
<td>8</td>
<td>0.88</td>
<td>39.1</td>
<td>25.3</td>
<td>13.8</td>
<td>0.99</td>
<td>41.4</td>
<td>28.5</td>
<td>12.9</td>
<td>1.30</td>
<td>41.3</td>
<td>28.0</td>
<td>13.3</td>
<td>1.43</td>
<td>57.5</td>
<td>45.6</td>
<td>11.8</td>
</tr>
<tr>
<td>9</td>
<td>1.07</td>
<td>24.3</td>
<td>13.5</td>
<td>10.8</td>
<td>1.37</td>
<td>22.5</td>
<td>14.8</td>
<td>7.7</td>
<td>1.78</td>
<td>23.6</td>
<td>17.0</td>
<td>6.5</td>
<td>2.22</td>
<td>24.9</td>
<td>18.0</td>
<td>6.9</td>
</tr>
<tr>
<td>10</td>
<td>1.30</td>
<td>25.6</td>
<td>17.1</td>
<td>8.5</td>
<td>1.58</td>
<td>24.9</td>
<td>17.3</td>
<td>7.6</td>
<td>1.95</td>
<td>28.9</td>
<td>20.2</td>
<td>8.5</td>
<td>2.31</td>
<td>45.4</td>
<td>36.3</td>
<td>7.1</td>
</tr>
<tr>
<td>Mean</td>
<td>1.32*</td>
<td>26.8</td>
<td>16.9</td>
<td>9.9</td>
<td>1.55*</td>
<td>26.1</td>
<td>17.1</td>
<td>8.9</td>
<td>1.86*</td>
<td>27.7</td>
<td>18.5</td>
<td>9.2</td>
<td>2.28*</td>
<td>34.6**</td>
<td>25.1**</td>
<td>9.5</td>
</tr>
<tr>
<td>SE</td>
<td>0.17</td>
<td>2.5</td>
<td>1.9</td>
<td>0.8</td>
<td>0.20</td>
<td>2.6</td>
<td>2.1</td>
<td>1.0</td>
<td>0.20</td>
<td>2.6</td>
<td>2.1</td>
<td>0.9</td>
<td>0.25</td>
<td>3.9</td>
<td>3.6</td>
<td>0.8</td>
</tr>
</tbody>
</table>

*Significant compared with all other levels of PEEP (P<0.001).
**Significant compared with 3, 8, and 13 cm H_2O of PEEP (P<0.005).
Alterations of pulmonary blood volume and chest wall mechanics would also alter compliance by changing end-expiratory transpulmonary pressure and resting pulmonary volume. In this study, CLT and ELT were separated into their pulmonary and chest wall components by using measurements of Pes as an estimate of intrapleural pressure. Grimby et al measured CLT, CL, and CW using an esophageal balloon in normal volunteers who were anesthetized and mechanically ventilated in the supine position. The mean values from Grimby et al were as follows: CLT, 0.087 L/cm H2O; CL, 0.089 L/cm H2O; and CW, 0.167 L/cm H2O.

Figure 1 shows the mean values and ranges for CL, CW, CLT, EL, CW, and ELT in this study. A given change in compliance has grossly different implications, in terms of elastic recoil (of this in-series lung-thorax system), depending on the absolute value of that compliance and, in fact, whether one is evaluating lung, chest wall, or lung-thorax. When such an in-series system is being evaluated, elastance is the preferred unit of measurement for two reasons. First, it is more convenient to manipulate mathematically. Secondly, although changes in both EL and CL correlated well with changes in ELT and CLT, the implication of their respective changes, in terms of lung elasticity, were different, as demonstrated by their regression slopes (Fig 4A, 4C). It follows that the clinician may be misled quantitatively by using compliance as a measure of changes in elasticity in the lung-thorax.

In our study, increased EL was largely responsible for the increased ELT, and this is consistent with a previous report; however, our patients did vary as to the proportions of pulmonary and chest wall contributions. After abdominal aortic aneurysmectomy (a clinical situation in which the abdomen is distended and chest wall mechanics are therefore abnormal), three patients (patients 2, 6, and 14 in Table 1) had increased ELT, EL, and CW. The chest wall component was increased to approximately 173 percent of normal in these three patients (mean CW, 10.4 cm H2O/L). The increase for these patients is contrasted with no increase in patient 11, who had a clinical diagnosis of pulmonary contusion, high ELT and EL, but normal CW (4.7 cm H2O/L).

The mean CW for all patients was 9.4 cm H2O/L (CW, 0.106 L/cm H2O) and was increased from the normal range of 5 to 6 cm H2O/L (CW, 0.10 to 0.20 L/cm H2O). This increase was probably caused by abdominal distention, chest wall edema, pleural effusion, or bandages which may have contributed to the decreased pulmonary volume. Another possible cause of the increased CW is that decreased pulmonary volume and increased EL may pull the chest wall below its resting level and therefore to a steeper portion of its elastance curve. A direct relationship between measured FRC and intrathoracic volume may not apply in acute respiratory failure, since in acute respiratory failure more of the intrathoracic volume is occupied by intrathoracic blood volume, edema and pleural fluid. Consequently, our lung volume measurements may not be predictive of total intrathoracic volume ranges.

The chest wall did not contribute significantly to changes in measurements of ELT, and one might conclude that in most clinical situations, lung-thorax data can be used as an index of changing pulmonary status; however, our patients were relaxed after receiving sedation and were being passively ventilated. In any consideration of CW, the activity of respiratory muscles making up the chest wall is of great importance. Although sedation does not abolish respiratory activity, examination of each patient's continuous Paw and Pes data showed stable pressures at end-inspiration and end-expiration, suggesting that spontaneous respiratory efforts were not present. However, since some respiratory muscle tone may have been present, this could have influenced CW and therefore CLT but not CL. In situations in which patients are not relaxed or being passively ventilated, lung-thorax data cannot be assumed to reflect pulmonary status, and the esophageal balloon may be invaluable in determining transpulmonary pressures.

Measurement of Pes

One must be cautious in using measurements of Pes when the patient is in the supine position. The Pes, as measured from a balloon, will accurately reflect local pleural pressures if no difference in pressure exists across the balloon's wall, the esophageal wall, and various mediastinal structures. Therefore, the problems in interpreting Pes as a measure of intrapleural pressure involve distortions of the length of the balloon, its diameter, wall thickness, air volume, position in the esophagus, and mediastinal compression in the supine position.

The recommended length of the balloon is 10 cm. Longer balloons (15 cm) make it difficult to place the balloon in the midesophagus without part of the balloon entering the upper esophagus and thus possibly recording artifacts of tracheal compression or head movement. Shorter balloons (2 to 3 cm) are affected more by mediastinal compression. In addition, CL may be more reproducible with a 10-cm balloon.

Over a wide range of balloon volumes, the drop...
in pressure across the wall of the balloon in air should be negligible. When the balloon contains 0.5 ml of air, the pressure in the balloon should be between 0 and \(-0.5\) cm H2O.\(^7\) Esophageal pressures increase at all pulmonary volumes as the volume of the balloon is increased, but the effect is most marked at the extremes of vital capacity.\(^8\) This increase in pressure is presumably caused by distention and displacement of the esophageal wall and surrounding structures. Such distortions of the pressure-volume curves may be corrected by extrapolating the Pes to that of zero balloon volume or by using a technique with minimum balloon volume.\(^9,10\) Each balloon, depending on the thickness of the wall and circumference, would require different balloon volumes (0.2 to 0.6 ml).\(^10\)

Milic-Emili et al\(^9\) have recommended measuring Pes in the midesophagus. They found that the pressure was distorted in the upper and lower thirds of the esophagus. In the upper third (27 to 32 cm from nare to tip of the balloon), the pressure is altered by tracheal compression or head movement, ie, not by changes in transpulmonary pressure. In the lower third of the esophagus (greater than 45 cm from the nare), the pressure varies from point to point and with changes in body position. In the midesophagus (35 to 45 cm from nare), the pressure does not vary markedly, even with changes in posture.

In the supine position, measurements of Pes are affected by the weight of the heart and other mediastinal contents.\(^20\) Although this results in higher absolute pressures, the shape of the pressure-volume curve is not distorted over a wide range (20 to 80 percent of vital capacity [VC]) when compared with pressure-volume curves for subjects in the sitting position;\(^9,10\) however, there can be wide variations at the extremes of VC. In this study, some of the measurements of Pes were made at low pulmonary volume (<20 percent VC) and therefore may be influenced by this artifact. Such data are probably low estimates of the Cl\(_E\).\(^20,20\)

In conclusion, we have shown a correlation between FRC and both ELT and CLT in patients with acute respiratory failure. However, because of the many variables that might affect ELT (1/CLT), the relationship is not sufficiently close to predict pulmonary volume. We have also shown that ELT is preferable to CLT as an index of changes in pulmonary status in patients with acute respiratory failure for the following reasons: 1) while changes in both ELT and CLT usually reflect changes in EL and CL, there is a one-to-one relationship between the changes in ELT and EL and not between CLT and CL; 2) elastance is a more sensitive index of deteriorating lung status than is compliance (Fig 1); 3) should one wish to partition ELT into EL and EEW, the arithmetic manipulations are simpler.

ACKNOWLEDGMENT: The authors gratefully acknowledge the technical assistance of Patrick D. Pangburn, R.R.T., and the editorial advice of Pauline Snider.

REFERENCES

1 Bone RC. Diagnosis of causes for acute respiratory distress by pressure-volume curves. Chest 1976; 70:740-46
2 Suter PM, Fairley HB, Isenberg MD. Effect of tidal volume and positive end-expiratory pressure on compliance during mechanical ventilation. Chest 1978; 73: 158-62
7 Macklem PT, Procedures for standardized measurements of lung mechanics; and Leith DE, Mead J. Principles of body plethysmography. Bethesda, Md: National Heart and Lung Institute, Division of Lung Diseases, 1974
18 Petty TL, Silvers GW, Paul GW, et al. Abnormalities in lung elastic properties and surfactant function in adult

Chest, 80: 3, September, 1981
respiratory distress syndrome. Chest 1979; 75:571-74
22 Egbert LD, Laver MB, Bendixen HH. Intermittent deep breaths and compliance during anesthesia in man. Anesthesiology 1963; 24:57-60
26 Housley E, Louzada N, Becklake MR. To sigh or not to sigh. Am Rev Respir Dis 1970; 101:611-14