Transpulmonary pressure...

mislead by esophageal pressure?

Marcelo B. P. Amato

Critical Care Canadian Forum

CCCF, Toronto, November 2013
## Conflict of Interest Disclosure

**Marcelo B.P. Amato**

I disclose the following financial relationships with commercial entities that produce healthcare-related products or services relevant to the content I am presenting:

<table>
<thead>
<tr>
<th>Company</th>
<th>Relationship</th>
<th>Content Area</th>
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</thead>
<tbody>
<tr>
<td>Covidien</td>
<td>Consultant</td>
<td>Mechanical Ventilation</td>
</tr>
<tr>
<td>Philips / Dixtal</td>
<td>Research grants</td>
<td>E.I.T.</td>
</tr>
</tbody>
</table>
Non-dependent (left b)

- low PSV
- strong effort

Dependent (left D6)
Pressure sensor: 1.2cm disk

Diaphragm (inner surface: the view from inside the thorax)

The dependent lung
Original Article

Mechanical Ventilation Guided by Esophageal Pressure in Acute Lung Injury


N Engl J Med
Volume 359(20):2095-2104
November 13, 2008
Esophageal pressures in acute lung injury: do they represent artifact or useful information about transpulmonary pressure, chest wall mechanics, and lung stress?

Stephen H. Loring,1,5 Carl R. O’Donnell,2,5 Negin Behazin,1,5 Atul Malhotra,3,5 Todd Sarge,1,5 Ray Ritz,1 Victor Novack,4 and Daniel Talmor1,5

1Department of Anesthesia, Critical Care, and Pain Medicine and 2Division of Pulmonary, Critical Care, and Sleep Medicine, Beth Israel Deaconess Medical Center, 3Divisions of Pulmonary and Critical Care and Sleep Medicine, Brigham and Women’s Hospital, 4Harvard Clinical Research Institute, and 5Harvard Medical School, Boston, Massachusetts

Submitted 29 July 2009; accepted in final form 14 December 2009

Loring SH, O’Donnell CR, Behazin N, Malhotra A, Sarge T, Ritz R, Novack V, Talmor D. Esophageal pressures in acute lung injury: do they represent artifact or useful information about transpulmonary pressure, chest wall mechanics, and lung stress? J Appl Physiol 108: 515–522, 2010. First published December 17, 2009; doi:10.1152/japplphysiol.00835.2009.—Acute lung injury can be worsened by inappropriate mechanical ventilation, and numerous experimental studies suggest that ventilator-induced lung injury is increased by excessive lung inflation at end inspiration or inadequate applied to the respiratory system and lung (1). Numerous experimental studies have shown that lung injury can also be reduced by maintaining sufficient positive end-expiratory pressure (PEEP) to prevent lung collapse at end expiration (8, 9, 11, 15, 22, 23, 26, 37–39, 49). Lung inflation depends on transpulmonary pressure (airway pressure – pleural pressure), which in turn depends on characteristics of the chest wall, as well as the lung. Unfortunately, pressures within the chest cavity are rarely
Lung Stress and Strain during Mechanical Ventilation for Acute Respiratory Distress Syndrome

Davide Chiumello¹, Eleonora Carlesso², Paolo Cadrinigher², Pietro Caironi¹,², Franco Valenza¹,², Federico Polli², Federica Tallarini², Paola Cozzi², Massimo Cressoni², Angelo Colombo¹, John J. Marini³, and Luciano Gattinoni¹,²

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Rationale: Lung injury caused by a ventilator results from nonphysiologic lung stress (transpulmonary pressure) and strain (inflated volume to functional residual capacity ratio).

Objectives: To determine whether plateau pressure and tidal volume are adequate surrogates for stress and strain, and to quantify the stress to strain relationship in patients and control subjects.

Methods: Nineteen postsurgical healthy patients (group 1), 11 patients with medical diseases (group 2), 26 patients with acute lung injury (group 3), and 24 patients with acute respiratory distress syndrome (group 4) underwent a positive end-expiratory pressure (PEEP) trial (5 and 15 cm H₂O) with 6, 8, 10, and 12 ml/kg tidal volume.

AT A GLANCE COMMENTARY

Scientific Knowledge on the Subject

Lung stress and strain are the primary determinants of ventilator-induced lung injury. Their surrogates are airway pressure and tidal volume normalized for ideal body weight (V̇̇T IBW). Prevention of ventilator-induced lung injury is primarily based on recognizing the “harmful” threshold for these surrogates (30 cm H₂O airway plateau pressure and 6 ml/kg V̇̇T IBW).

\[ \Delta P_L \text{ (stress)} = \Delta P_{aw} \times E_L / (E_L + E_{CW}) \]  (3)
Fig. 7. Lung stress calculated using Eq. 2 of Chiumello et al. (7), $\Delta P_L$ (stress), plotted against directly measured transpulmonary pressure ($P_L, e_{S_{EIO}}$). $\Delta P_L$ (stress) does not account for prestress before inflation and is much greater than $P_L, e_{S_{EIO}}$. 
Fig. 3. Transpulmonary pressure at end-expiratory occlusion ($P_{LEsEEO}$) vs. simultaneously measured airway pressure [i.e., total positive end-expiratory pressure ($PEEP_T$)]. $P_{LEsEEO}$ is usually substantially less than $PEEP_T$. Solid line, line of regression.
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Chest Wall Elastance

Ecw was, on average, slightly greater than normal, 8.7 ± 4.7 cmH₂O/l, but it ranged widely from 1.3 to 21.8 cmH₂O/l. Perhaps surprisingly, Ecw was not correlated with Pes_EEO, implying that the slope of the pressure-volume curve of the chest wall was not correlated with the position of the curve.
Lung Stress and Strain during Mechanical Ventilation for Acute Respiratory Distress Syndrome

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Getting more information

X

Introducing noise
sensor
sensor

C balloon = ?
sensor

C balloon = ?

C esophagus = ?
$P_{es}$ catheter
$\text{FiO}_2 = 100 \%$; \hspace{1cm} $V_T = 4 \text{ mL/kg}$; \hspace{1cm} PEEP = 5 cmH$_2$O

Collapse: 55.6 %

Patient # 9

Pneumocystis carinii pneumonia
\[ \text{FiO}_2 = 100\%; \quad V_T = 4\; \text{mL/kg}; \quad \text{PEEP} = 25\; \text{cmH}_2\text{O} \]

( after recruitment maneuver )

Collapse: 0.9 %

Patient #9
*Pneumocystis carinii* pneumonia
LOCAL TRANSPULMONARY PRESSURE MEASUREMENT

P_pl
NON-DEPENDENT

P_pl
MIDDLE

P_pl
DEPENDENT

LOCAL TRANSPULMONARY PRESSURE MEASUREMENT
CHEST-WALL P-V CURVES
(super-syringe)

PLEURAL PRESSURES (cmH\textsubscript{2}O)

VOLUME (plethysmograph - mL)

Normal
CHEST-WALL P-V CURVES
(super-syringe)

PLEURAL PRESSURES (cmH₂O)
0 4 8 12 16 20

VOLUME (plethysmograph - mL)
0 300 600 900 1200 1500 1800

Injury
CHEST-WALL P-V CURVES
(super-syringe)

PLEURAL Pressures (cmH\(_2\)O)
0 4 8 12 16 20

VOLUME
(plethysmograph - mL)
0 300 600 900 1200 1500 1800
Airway pressure (cmH₂O)

Pleural pressure (cmH₂O)

Occlusion test

Tube clamp at the end of expiration

A

\( \Delta \) Airway pressure (cmH₂O)

\( \Delta \) Pleural pressure (cmH₂O)

Slope: 0.91

B
$P_{es}$ as a surrogate for $P_{pl}$

Baydur A. *Am Rev Respir Dis* 1982;126:788
diaphragmatic contraction uniformly lowers $P_{pl}$ at all points on the lung surface
Ipsilateral transpulmonary pressures during unilateral electrophrenic respiration

VU-DINH MINH, PAUL J. FRIEDMAN, NAOTSUGU KURIHARA, AND KENNETH M. MOSER
Pulmonary Division, Department of Medicine, University of California, San Diego, School of Medicine,
San Diego, California 92103
### TABLE 3. Pressure distribution between apex and base during spontaneous breathing

<table>
<thead>
<tr>
<th>Animal No.</th>
<th>Apical $\text{Ptp}_{E-I}$</th>
<th>Basal $\text{Ptp}_{E-I}$</th>
<th>Apical $\text{Ptp}_{FRC}$</th>
<th>Basal $\text{Ptp}_{FRC}$</th>
<th>Apical $\Delta \text{Ptp}$</th>
<th>Basal $\Delta \text{Ptp}$</th>
<th>Basal/Apical $\Delta \text{Ptp}$ Ratio</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>12.3</td>
<td>12.1</td>
<td>3.7</td>
<td>3.3</td>
<td>8.6</td>
<td>8.8</td>
<td>1</td>
</tr>
<tr>
<td>2</td>
<td>11.2</td>
<td>12.3</td>
<td>4.2</td>
<td>5.3</td>
<td>7.0</td>
<td>7.0</td>
<td>1</td>
</tr>
<tr>
<td>3</td>
<td>10.5</td>
<td>11.8</td>
<td>4.8</td>
<td>4.8</td>
<td>5.7</td>
<td>6.9</td>
<td>1.2</td>
</tr>
<tr>
<td>4</td>
<td>13.3</td>
<td>13.2</td>
<td>4.3</td>
<td>4.2</td>
<td>9</td>
<td>9</td>
<td>1</td>
</tr>
<tr>
<td>5</td>
<td>9.3</td>
<td>11.7</td>
<td>4.8</td>
<td>4.8</td>
<td>4.5</td>
<td>6.9</td>
<td>1.5</td>
</tr>
<tr>
<td>6</td>
<td>13.8</td>
<td>12.8</td>
<td>4.3</td>
<td>4.8</td>
<td>9.5</td>
<td>8</td>
<td>0.8</td>
</tr>
<tr>
<td>7</td>
<td>11.2</td>
<td>11.7</td>
<td>5.4</td>
<td>6.0</td>
<td>5.8</td>
<td>5.7</td>
<td>1</td>
</tr>
<tr>
<td>Mean</td>
<td>11.6</td>
<td>12.2</td>
<td>4.5</td>
<td>4.7</td>
<td>7.1</td>
<td>7.5</td>
<td>1.07</td>
</tr>
<tr>
<td>±SD</td>
<td>±1.4</td>
<td>±0.5</td>
<td>±0.5</td>
<td>±0.8</td>
<td>±1.8</td>
<td>±1.1</td>
<td>±0.20</td>
</tr>
</tbody>
</table>

$t = 1.37; t = 1.29; t = 0.65$

$P < 0.4; P < 0.4; P < 0.50$
$\text{FiO}_2 = 100 \%$; \hspace{1cm} V_T = 4 \text{ mL/kg}; \hspace{1cm} \text{PEEP} = 5 \text{ cmH}_2\text{O}$

Collapse: 55.6 \%

Patient # 9
Pneumocystis carinii pneumonia
was surgically exposed and cannulated with a catheter. A three lumen central venous and a pulmonary artery catheter were inserted through the right internal jugular vein and advanced under waveform guidance. Proper positioning of endovascular catheters (superior vena cava and pulmonary artery, respectively) was verified on lung computed tomography and confirmed at autopsy. A bladder catheter was positioned via cistostomy.

Animals were then turned prone. Stomach emptying by gastric suction preceded esophageal balloon placement. A latex thin walled, 5 cm long, esophageal balloon was advanced into the inferior third of the esophagus and filled-in with 1.5 ml of room air. Pressure transducers were connected to the endotracheal tube, the esophageal balloon and the endovascular catheters, zeroed at room air or heart level, as appropriate. Data were recorded and analyzed using a dedicated software (Colligo, www.elekton.it).
during mechanical ventilation. Placement of the balloon in the stomach was confirmed by a transient increase in pressure during a gentle compression of the abdomen and by a qualitative change in the pressure tracing (i.e., an increased cardiac artifact) as the balloon was withdrawn into the esophagus. In approximately one third of the patients, the balloon could not be passed into the stomach, and esophageal placement was confirmed by the presence of a cardiac artifact and the changes in transpulmonary pressure during tidal ventilation. The mixed expired partial
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ESSURE IN ACUTE LUNG INJURY

ratories, available from CooperSurgical.com). The balloon (9.5 cm long, 2 cm perimeter) was filled with 0.5–1.0 ml of air. Frequency response of the system was adequate (without significant delays) up to 15 Hz. Pressure, volume, and flow measurements were displayed and recorded using a custom-written program and analyzed with Windaq software (Dataq.com).

Protocol
Balloon volume (mL of room air)

Esophageal pressure (end-expiration)

Signal

Noise
Balloon volume (mL of room air)
Compliance Chest-Wall

60%
Getting more information

Introducing noise

X

X
\[ \Delta P \text{ or } \Delta P_{TP} ? \]

\[ (\Delta P_{AW} - \Delta P_{esoph}) \]
Lung Stress and Strain during Mechanical Ventilation
Any Safe Threshold?

Alessandro Protti¹, Massimo Cressoni¹, Alessandro Santini¹, Thomas Langer¹, Cristina Mietto¹, Daniela Febres¹, Monica Chierichetti¹, Silvia Coppola¹, Grazia Conte², Stefano Gatti², Orazio Leopardi³, Serge Masson³, Luciano Lombardi⁴, Marco Lazzerini⁴, Erica Rampoldi⁵, Paolo Cadringher¹, and Luciano Gattinoni¹,⁶

¹Dipartimento di Anestesiologia, Terapia Intensiva e Scienze Dermatologiche, and ²Centro di Ricerche Chirurgiche Precliniche, Università degli Studi di Milano, Milan, Italy; ³Department of Cardiovascular Research, Istituto Mario Negri, Milan, Italy; and ⁴Dipartimento di Radiologia, ⁵Unità Operativa Laboratorio Centrale, and ⁶Dipartimento di Anestesia, Rianimazione (Intensiva e Subintensiva) e Terapia del Dolore, Fondazione IRCCS Ca’ Granda - Ospedale Maggiore Policlinico, Milan, Italy

<table>
<thead>
<tr>
<th></th>
<th>No Ventilator-induced Lung Edema (n = 15)</th>
<th>Ventilator-induced Lung Edema (n = 14)</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Strain, tidal volume/FRC</td>
<td>1.29 ± 0.57</td>
<td>2.16 ± 0.58</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Tidal volume/body weight, ml/kg</td>
<td>22 ± 8</td>
<td>38 ± 9</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Tidal volume, ml</td>
<td>461 ± 175</td>
<td>816 ± 203</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>End-inspiratory lung volume, ml</td>
<td>839 ± 194</td>
<td>1,205 ± 251</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Specific lung elastance, cm H₂O</td>
<td>5 ± 2 (n = 12)</td>
<td>6 ± 3 (n = 10)</td>
<td>0.54</td>
</tr>
<tr>
<td>Mean airway pressure, cm H₂O</td>
<td>Start of experiment 7 ± 3</td>
<td>12 ± 3</td>
<td></td>
</tr>
<tr>
<td></td>
<td>End of experiment 8 ± 3</td>
<td>17 ± 4</td>
<td></td>
</tr>
<tr>
<td>Δ (End − Start)</td>
<td>1 ± 3</td>
<td>6 ± 3</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Plateau pressure, cm H₂O</td>
<td>Start of experiment 16 ± 5</td>
<td>26 ± 7</td>
<td></td>
</tr>
<tr>
<td></td>
<td>End of experiment 20 ± 8</td>
<td>41 ± 7</td>
<td></td>
</tr>
<tr>
<td>Δ (End − Start)</td>
<td>4 ± 5</td>
<td>16 ± 5</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Δ Transpulmonary pressure, cm H₂O</td>
<td>Start of experiment 8 ± 3 (n = 13)</td>
<td>13 ± 5 (n = 13)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>End of experiment 13 ± 7 (n = 13)</td>
<td>29 ± 6 (n = 13)</td>
<td></td>
</tr>
<tr>
<td>Δ (End − Start)</td>
<td>5 ± 6 (n = 13)</td>
<td>17 ± 4 (n = 13)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Δ Esophageal pressure, cm H₂O</td>
<td>Start of experiment 9 ± 2 (n = 13)</td>
<td>13 ± 4 (n = 13)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>End of experiment 8 ± 2 (n = 13)</td>
<td>12 ± 3 (n = 13)</td>
<td></td>
</tr>
</tbody>
</table>
Lung Strain

No-VILI

VILI

Strain

Injury!!
Injury!!
Note that,

↑ Offset in Pleural press. ≠ ↓ Chest-wall Compliance
Chest Wall Elastance

$Ecw$ was, on average, slightly greater than normal, $8.7 \pm 4.7$ cmH$_2$O/l, but it ranged widely from 1.3 to 21.8 cmH$_2$O/l. Perhaps surprisingly, $Ecw$ was not correlated with $Pes_{EEO}$, implying that the slope of the pressure-volume curve of the chest wall was not correlated with the position of the curve.
Breathing Mechanics, Dead Space and Gas Exchange in the Extremely Obese, Breathing Spontaneously and During Anaesthesia with Intermittent Positive Pressure Ventilation

GÖRAN HEDENSTIERN and JOAKIM SANTESSON

Department of Clinical Physiology, Serafimer Hospital, and Department of Anaesthesiology, Karolinska Hospital, Stockholm, Sweden

Breathing mechanics and gas exchange were studied in 10 extremely obese subjects (average weight 138 kg) prior to and during anaesthesia with mechanical ventilation. Breathing mechanics were analysed from measurements of transpulmonary pressure (during anaesthesia, trans-chest wall pressure as well) inspiratory gas flow and tidal volume. Gas exchange was studied by analysing inspired and expired gas as well as arterial blood samples. The total dead space was deduced from the Bohr equation, and the division into anatomical and alveolar dead space was arrived at by capnography. The patients were anaesthetised with neurolept agents and ventilated with an air-oxygen mixture. Lung compliance during spontaneous breathing was below normal and decreased further during artificial ventilation. Chest wall compliance measured during anaesthesia was within normal limits. Lung resistance was above normal during spontaneous breathing and increased further during mechanical ventilation. Total dead space was normal during spontaneous breathing and increased moderately during artificial ventilation, the increment coming mainly from alveolar dead space. A moderate hypoxaemia was recorded during spontaneous breathing, and the alveolar-arterial oxygen tension difference was slightly elevated. During anaesthesia this difference was markedly greater. It is concluded that the most probable reason for the relative hypoxaemia is right-to-left shunting.

Received 10 December, accepted 29 December 1975
Fig. 1. Lung compliance ($C_L$) during spontaneous breathing (SB) and artificial ventilation; chest wall compliance ($C_W$) and total compliance ($C_{tot}$) during artificial ventilation; and maximum inspiratory lung resistance ($R_L$) during spontaneous breathing and artificial ventilation (AV) (mean and s.d.).

Statistics SB–AV: $C_L$: $P < 0.05$

$R_L$: n.s.
...Chest-Wall compliance during anesthesia was comparable to that measured in non-obese, healthy subjects...
...This may be surprising, bearing in mind the voluminous fat layers over chest and abdomen...
Thus, a weight placed on the rib-cage does not affect chest-wall compliance, but simply displaces the pressure-volume relationship towards higher pressures...
+ 4.3 cmH$_2$O above average (P < 0.001)
High PEEP studies; N = 2259 pts.

\[ R^2 = 0.00 \]
Thank you!
$\text{FiO}_2 = 100\%$; \quad V_T = 4\, \text{mL/kg}; \quad PEEP = 5\, \text{cmH}_2\text{O}$

Collapse: 55.6 %

Patient #9

*Pneumocystis carinii* pneumonia
\( \text{FiO}_2 = 100 \% ; \quad V_T = 4 \text{ mL/kg} ; \quad \text{PEEP} = 25 \text{ cmH}_2\text{O} \)

( after recruitment maneuver )

Collapse: 0.9 %

Patient # 9

*Pneumocystis carinii* pneumonia
NON-DEPENDENT TRANSPULMONARY PRESSURES
( Oleic Acid Injury )

"Relief" zone

PEEP = 14.2

PEEP = 5

PEEP = 0.7

TOTAL LUNG VOLUME (mL)

REGIONAL PRESSURES (cmH₂O)
( AT END-INSPIRATION )

TRANSPPULMONARY PRESSURE AT ZEEP
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1Department of Anesthesia, Critical Care, and Pain Medicine and 2Division of Pulmonary, Critical Care, and Sleep Medicine, Beth Israel Deaconess Medical Center, 3Divisions of Pulmonary and Critical Care and Sleep Medicine, Brigham and Women’s Hospital, 4Harvard Clinical Research Institute, and 5Harvard Medical School, Boston, Massachusetts

Submitted 29 July 2009; accepted in final form 14 December 2009

Loring SH, O’Donnell CR, Behazin N, Malhotra A, Sarge T, Ritz R, Novack V, Talmor D. Esophageal pressures in acute lung injury: do they represent artifact or useful information about transpulmonary pressure, chest wall mechanics, and lung stress? J Appl Physiol 108: 515–522, 2010. First published December 17, 2009; doi:10.1152/japplphysiol.00835.2009.—Acute lung injury can be worsened by inappropriate mechanical ventilation, and numerous experimental studies suggest that ventilator-induced lung injury is increased by excessive lung inflation at end inspiration or inadequate applied to the respiratory system and lung (1). Numerous experimental studies have shown that lung injury can also be reduced by maintaining sufficient positive end-expiratory pressure (PEEP) to prevent lung collapse at end expiration (8, 9, 11, 15, 22, 23, 26, 37–39, 49). Lung inflation depends on transpulmonary pressure (airway pressure – pleural pressure), which in turn depends on characteristics of the chest wall, as well as the lung. Unfortunately, pressures within the chest cavity are rarely
sensor

C balloon = ?

C esophagus = ?