Lung Protection

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Luca Gattinoni, MD, FRCP
Georg-August-Universität Göttingen
Germany

Excessive power

VILI comes from

Small, inhomogeneous lung

Power (Energy/Minute)
Includes ∆Pressure, ∆Volume, Rate, Flow.

Chest wall elastance

Slope $P_L/P_{aw} = E_w/E_{tot}$ [0.2 - 0.8]

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Chiumello et al, Am J Respir Crit Care Med. 2008
Energy/power

\[ \text{PL} \times \Delta V = \text{Energy (lung)} \]

Force/Area (cm\(^2\)) \(\times\) \(\Delta V\) (cm\(^3\)) = Force \(\times\) Movement

Tidal Energy \(\times\) RR = POWER

Are there thresholds for power?

1. The anatomical thresholds
2. The associated power

<table>
<thead>
<tr>
<th>FRC (ml)</th>
<th>TLC (ml)</th>
<th>Sp E (cm H(_2)O)</th>
<th>PL (TLC) (cm H(_2)O)</th>
<th>Strain at 6 ml/kg TV</th>
</tr>
</thead>
<tbody>
<tr>
<td>2.5</td>
<td>7.5</td>
<td>4</td>
<td>8</td>
<td>0.72</td>
</tr>
<tr>
<td>300</td>
<td>900</td>
<td>6</td>
<td>12</td>
<td>0.46</td>
</tr>
<tr>
<td>2000</td>
<td>6000</td>
<td>12</td>
<td>24</td>
<td>0.21</td>
</tr>
</tbody>
</table>
Time course of ventilator induced lung injury


Stress-strain curve of healthy pigs


Relative Risk of Death in the Hospital across Relevant Subsamples after Multivariate Adjustment — Survival Effect of Ventilation Pressures.


Tidal Strain

P*ΔV = Energy Input

Dissipated
Surface Tension
Sliding EM
Opening and Closing

Undissipated
Elastic System

Continuous Strain

PEEP *ΔV = Energy Input = 0

76 PIGS

76 PIGS

TOTAL VOLUME/kg SURVIVED
TOTAL VOLUME/kg DEAD
TV/kg SURVIVED
TV/kg DEAD
Global stress able to damage healthy (or “baby”?) lung in clinical practice is uncommon.

However, when the lung starts to deteriorate the rate of damage is impressively fast, why?

If global stress is so rare, how can we explain the following slide?

**Stress distribution:**
- High stiffness zone

**Voxel**
- Voxel gas fraction $= V_{gas}/V_{total}$
- Weighted gas ratio $= V_{gas}/V_{voxel}$, fraction of tissue

**Lung dishomogeneity and ARDS**

<table>
<thead>
<tr>
<th>Mild (N=62)</th>
<th>Moderate (N=71)</th>
<th>Severe (N=12)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dishomogeneity</td>
<td>1.49 ± 0.17</td>
<td>1.58 ± 0.29</td>
<td>1.75 ± 0.41</td>
</tr>
<tr>
<td>Dishomogeneity$^{2/3}$</td>
<td>1.30 ± 0.31</td>
<td>1.36 ± 0.44</td>
<td>1.45 ± 0.55</td>
</tr>
<tr>
<td>Extent</td>
<td>0.3 ± 0.1</td>
<td>0.36 ± 0.16</td>
<td>0.46 ± 0.18</td>
</tr>
<tr>
<td>Intensity</td>
<td>2.69 ± 0.27</td>
<td>2.76 ± 0.27</td>
<td>2.84 ± 0.41</td>
</tr>
<tr>
<td>Intensity$^{2/3}$</td>
<td>1.93 ± 0.42</td>
<td>1.97 ± 0.42</td>
<td>2.01 ± 0.55</td>
</tr>
</tbody>
</table>

*Am J Respir Crit Care Med. 2014 Jan 15;189(2):149-58*
Average ratio in normal subjects: 1.37±0.15

Hypothesis
Lesions should first occur where physiological stress risers are located

Before appearance first new densities
TIME 1: 5.7±6.5 hours

First CT scan with new densities
TIME 2: 8.4±6.3 hours

Last CT scan with distinguishable densities
TIME 3: 15±12 hours
First CT scan with one-field edema

**TIME 4:** 18±11 hours

First CT scan with all-field edema

**TIME 5:** 20±11 hours

VILI cumulative time course

<table>
<thead>
<tr>
<th>Severity trend</th>
<th>CT scan only</th>
</tr>
</thead>
<tbody>
<tr>
<td>T4-5</td>
<td>+ Gas Exchange</td>
</tr>
<tr>
<td>T3</td>
<td>+ Lung mechanics</td>
</tr>
<tr>
<td>T2</td>
<td>CT scan only</td>
</tr>
</tbody>
</table>

0 5 10 15 20 25 Hours

VILI prerequisite

- Anatomical limit: whole lung
- Anatomical limit: regions (stress risers)
- Stress risers with severity
- To decrease stress risers high PEEP and prone position

LUNG IMAGING

CT scan inflation

INHOMOGENEITY

PET FDG UPTAKE

Ki/lung inhomogeneity interaction and gas/tissue composition

MILD

MODERATE

SEVERE
Conclusions

- For VILI what matters is a tidal volume in a ventilatable lung
- Tidal strain ≥ 1.5 and total stress around 20 cmH₂O in man are possible thresholds.
- The stress risers allow to reach the threshold locally and a multiplication of pressure is nearly 2
- PEEP does not produce energy load and its positive effects may be due to the decreased tidal volume

Lung protective strategy

Less energy
+ More homogeneous lung