Optimizing PEEP
Navigating Tough Choices

Focus Pittsburgh
September 28, 2017

John J. Marini, MD
University of Minnesota
Minneapolis / St.Paul
Optimizing PEEP

• History
  • Heterogeneity of ARDS
  • Physiologic workload on the baby lung
  • Increasing the size of the baby lung
    • Benefit
    • Hazards
  • The optimized trade-off
JANUS
God of Beginnings

Positive

Negative
In the Beginning…

Dr. Tom Petty
Permeability Edema Caused by ‘Rogue’ Inflammation

Ware & Matthay
NEJM 2000
Pathogenic Hallmark of ARDS

Diffuse Alveolar Damage
ARDS Follows an Evolving Time Course

Edema & Hyaline Membranes  →  Resolution or Fibrosis
Does ARDS Share a Common Link With IRDS?

HYPOTHESIS:

Endothelial/Alveolar Epithelial Damage

Surfactant Abnormalities in ARDS

Flooded Alveoli (Proteolysis)

Hydrostatic Forces Favoring Edema Formation

Increased Elastic Recoil

Surfactant Damage
Clements et al. (1958) described the theoretical considerations for a surface-active agent (surfactant). Several workers have described a decrease in surfactant in respiratory distress in the newborn (Avery and Mead 1959) and postperfusion lung (Gardner et al. 1962). Others have postulated that the surface-active agent is produced in the granular pneumocyte (Klaus et al. 1962). Two of our patients had decreased surface activity.

The inability to measure the surface-active agent directly is a serious obstacle in the effort to link this agent with clinical and pathological states. However, the theoretical basis for its presence is convincing, and indirect measurements seem to indicate that its loss is associated with the development of the clinical, physiological, and pathological conditions seen in the 12 patients in this series.
Little Concern Regarding the Causal Role of Mechanical VILI

<table>
<thead>
<tr>
<th>Complications Associated with ARDS</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Respiratory complications</strong></td>
</tr>
<tr>
<td>pulmonary emboli</td>
</tr>
<tr>
<td>pulmonary barotrauma</td>
</tr>
<tr>
<td>(pneumothorax, pneumomediastinum,</td>
</tr>
<tr>
<td>subcutaneous emphysema)</td>
</tr>
<tr>
<td>oxygen toxicity</td>
</tr>
<tr>
<td>pulmonary fibrosis</td>
</tr>
<tr>
<td><strong>Renal insufficiency</strong></td>
</tr>
<tr>
<td>Cardiac complications</td>
</tr>
<tr>
<td>decreased cardiac output</td>
</tr>
<tr>
<td>arrhythmias</td>
</tr>
<tr>
<td><strong>Hematologic complications</strong></td>
</tr>
<tr>
<td>anemia</td>
</tr>
<tr>
<td>thrombocytopenia</td>
</tr>
<tr>
<td>disseminated intravascular</td>
</tr>
<tr>
<td>coagulation</td>
</tr>
<tr>
<td><strong>Consequences of endotracheal</strong></td>
</tr>
<tr>
<td>intubation</td>
</tr>
<tr>
<td>laryngeal ulceration</td>
</tr>
<tr>
<td>tracheal ulceration</td>
</tr>
<tr>
<td>tracheal malacia</td>
</tr>
<tr>
<td>tracheal stenosis</td>
</tr>
<tr>
<td><strong>Infection</strong></td>
</tr>
<tr>
<td>nosocomial pneumonia</td>
</tr>
<tr>
<td>catheter-related infection</td>
</tr>
<tr>
<td>sepsis (bacteremia)</td>
</tr>
<tr>
<td><strong>Gastrointestinal complications</strong></td>
</tr>
<tr>
<td>stress ulceration and hemorrhage</td>
</tr>
<tr>
<td>ileus</td>
</tr>
<tr>
<td>pneumoperitoneum</td>
</tr>
</tbody>
</table>
Contribution of VILI to ARDS

Fowler 1984
The Myth of ARDS

Diverse Etiologies

Shared Pathophysiology

Well Defined Clinical Syndrome

Unified Clinical Approach
The Original *Clinical Recognition* ‘ARDS’ Referred To...

- Non-Cardiac, High Permeability Edema
- *Stiff* Lungs
- *Abrupt* Onset
- *Diffuse* Infiltrates
- Refractory hypoxemia often responsive to PEEP

“*A favorable blood gas response to PEEP* should be part of the definition of this syndrome”  T.L. Petty
The Berlin Definition of ARDS

<table>
<thead>
<tr>
<th>Timing</th>
<th>Within 1 week of a known clinical insult or new or worsening respiratory symptoms</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chest imaging(^a)</td>
<td>Bilateral opacities—not fully explained by effusions, lobar/lung collapse, or nodules</td>
</tr>
<tr>
<td>Origin of edema</td>
<td>Respiratory failure not fully explained by cardiac failure or fluid overload. Need objective assessment (eg, echocardiography) to exclude hydrostatic edema if no risk factor present</td>
</tr>
<tr>
<td>Oxygenation(^b)</td>
<td></td>
</tr>
<tr>
<td>Mild</td>
<td>200 mm Hg &lt; (P_{aO_2}/F_iO_2) (\leq) 300 mm Hg with PEEP or CPAP (\geq) 5 cm H(_2)O</td>
</tr>
<tr>
<td>Moderate</td>
<td>100 mm Hg &lt; (P_{aO_2}/F_iO_2) (\leq) 200 mm Hg with PEEP (\geq) 5 cm H(_2)O</td>
</tr>
<tr>
<td>Severe</td>
<td>(P_{aO_2}/F_iO_2) (\leq) 100 mm Hg with PEEP (\geq) 5 cm H(_2)O</td>
</tr>
</tbody>
</table>

What Are We Talking About Now?
Refractory Hypoxemia Originally Was Not Required to Define ARDS

<table>
<thead>
<tr>
<th>Case</th>
<th>Respiratory support</th>
<th>Frequency (min.⁻¹)</th>
<th>( \dot{V}_E ) (L/min.)</th>
<th>( \text{S}_aO_2 )</th>
<th>( \text{P}_{\text{O}<em>2} - \text{P}</em>{\text{A}_2\text{O}_3} ) gradient (mm. Hg)</th>
<th>( \text{P}_{\text{CO}_2} ) (mm. Hg)</th>
<th>pH</th>
<th>Compliance (L/cm. water)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Nasal oxygen (8 L/min.)</td>
<td>40</td>
<td>16·0</td>
<td>85</td>
<td>.</td>
<td>45·0</td>
<td>7·360</td>
<td>0·016</td>
</tr>
<tr>
<td>2</td>
<td>Bennett respirator (P.R.I.) (100% oxygen)</td>
<td>28</td>
<td>16·8</td>
<td>72</td>
<td>.</td>
<td>62·0</td>
<td>7·245</td>
<td>0·016</td>
</tr>
<tr>
<td>3</td>
<td>Bennett respirator (P.R.I.) (100% oxygen)</td>
<td>48</td>
<td>.</td>
<td>78</td>
<td>.</td>
<td>40·0</td>
<td>7·410</td>
<td>.</td>
</tr>
<tr>
<td>4</td>
<td>Bennett respirator (P.R.I.) (100% oxygen)</td>
<td>48</td>
<td>.</td>
<td>73</td>
<td>.</td>
<td>47·0</td>
<td>7·330</td>
<td>.</td>
</tr>
<tr>
<td>5</td>
<td>Engstrom respirator (70% oxygen)</td>
<td>48</td>
<td>8·0</td>
<td>85</td>
<td>.</td>
<td>63·0</td>
<td>7·270</td>
<td>.</td>
</tr>
<tr>
<td>6</td>
<td>Room air</td>
<td>36</td>
<td>14·4</td>
<td>84·4</td>
<td>.</td>
<td>37·0</td>
<td>7·338</td>
<td>0·017</td>
</tr>
<tr>
<td>7</td>
<td>Oxygen mask (3 L/min.)</td>
<td>64</td>
<td>.</td>
<td>79</td>
<td>.</td>
<td>22·0</td>
<td>7·420</td>
<td>.</td>
</tr>
<tr>
<td>8</td>
<td>Room air</td>
<td>44</td>
<td>20·0</td>
<td>41</td>
<td>536</td>
<td>29·5</td>
<td>7·395</td>
<td>0·009</td>
</tr>
<tr>
<td>9</td>
<td>Bennett respirator (vol.) (80% oxygen)</td>
<td>20</td>
<td>8·0</td>
<td>84</td>
<td>(100% oxygen) 320</td>
<td>57·5</td>
<td>7·270</td>
<td>0·016</td>
</tr>
<tr>
<td>10</td>
<td>Nasal oxygen (7 L/min.)</td>
<td>48</td>
<td>48·0</td>
<td>87</td>
<td>(100% oxygen) 30·5</td>
<td>7·420</td>
<td>0·019</td>
<td>.</td>
</tr>
<tr>
<td>11</td>
<td>Bennett respirator (P.R.I.) (40% oxygen)</td>
<td>36</td>
<td>25·2</td>
<td>74</td>
<td>.</td>
<td>29·5</td>
<td>7·480</td>
<td>0·017</td>
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<tr>
<td>12</td>
<td>Bennett respirator (P.R.I.) (40% oxygen)</td>
<td>34</td>
<td>12·8</td>
<td>72</td>
<td>(100% oxygen) 220</td>
<td>30·0</td>
<td>7·450</td>
<td>0·013</td>
</tr>
</tbody>
</table>
Optimizing PEEP

- History
- *Heterogeneity of ARDS*
- Physiologic workload on the baby lung
- Increasing the size of the baby lung
  - Benefit
  - Hazards
- The optimized trade-off
Diffuse Infiltrates → Globally Stiff Lungs
Appearances Are Often Deceiving
Is the Functional ARDS Lung Just *Smaller*?
Reduced Compliance Reflects Primarily **Reduced Number of Open Units**

…But Not Exclusively
The baby’s lung is not functionally ‘normal’
Stage, ARDS Etiology, RM Technique & Severity Influence Recruitability
Optimizing PEEP

• History
• Heterogeneity of ARDS
  • *Physiologic workload on the baby lung*
  • *Increasing the size of the baby lung*
    • Benefits
    • Hazards
• The optimized trade-off
The *Real* Cause of Dead Space in ARDS!
The Baby Lung of the ARDS Adult Has A Lot of Work to Perform!

For Any Given Minute Ventilation

- Multiples of Stretch
- Multiples of Gas flow
- Less than Proportional Blood Flows
Opening the Lung Reduces Shunt and Alveolar Deadspace

Tusman

Return to ‘Zone 3’ Conditions Improves RV Strain
Twenty-five years ago...

Editorial

Open up the lung and keep the lung open

B. Lachmann

Department of Anesthesiology, Erasmus University Rotterdam, The Netherlands
‘Open Up the Lung and Keep it Open’
Closed Units & Surfactant Loss Encourage Terminal Airway Damage
Opening the Lung Initially Placed Highest
Emphasis on Avoiding ‘Atelectrauma’

- Over Stretch
- Expanding Alveolus
- Junctional Stress and Shear
- Opening / Closure
- Collapsed Alveolus
Benefits From the ‘Open Lung’

• Increased membrane surface for gas exchange (less shunt & $V_D/V_T$)
• Surfactant renewal & protection
• Prevention of ‘atelectrauma’
• Improved uniformity of driving pressures
• Reduced infection
  – Lung
  – Bloodstream
The Big Trade-Off

- Better gas exchange
- Fewer ‘interface units’
  - Less stress focusing
  - Less tidal opening and closure
- Improved surfactant functioning & repair
- Increased global lung stress
- More tension on unopened interface units
- Greater ‘ergotrauma’?
- Greater hemodynamic stress
The ‘Surfactant-Central’ Hypothesis of IRDS Was Translated First to ARDS, then to VILI

- Surfactant depletion accentuates collapse stress focusing, and VILI.
- Surfactant regeneration requires aeration.
- Repetitive tidal cycles of opening and collapse deplete and degrade surfactant.
- *Sustained opening of lung units is key to lung protection.*
Open Lungs Allow Renewal and Repair Of Degraded Surfactant
Surfactant-Centric Rationale for Keeping An ‘Open Lung’ for Preventing ARDS

Am J Respir Crit Care Med Vol 185, Iss. 7, pp 702-708, Apr 1, 2012
Should We Embrace the *Fully “Open Lung”* Approach?
ARIES “Open Lung” Trial...
The lung was not fully open!

Crit Care Med
May 2006
Embrace?...Let’s Not Get Carried Away!
Why *Shouldn’t* We Crank the PEEP?

- Animal Models Are More Recruitable Than ARDS
  - Surfactant depletion & Oleic Acid
- ARDS is a Heterogeneous Disease
  - Some areas will be overstretched
- Open Lungs Generate Zone 2
  - Deadspace
  - VILI (due to *vascular* stress)
- Timing / Severity
  - “Utility of Approach May Be Limited”--*Early* and/or *Severe*
- Pressure Cost of Lung Opening
  - Hemodynamic Compromise**
  - Heightened Tissue Strain
- ARDSnet, Express, & LOVS Trial Results Were Discouraging (?)
Why is the ‘Fully Open’ Lung the Wrong Target for ARDS?

• Most ARDS lungs *cannot* be fully recruited.
• The rationale for this objective is defective.
  – ARDS etiology, severity and stage affect its relevance
  – Small $V_T$ and low driving pressure now reduce risk due to tidal opening and collapse
• The required airway pressures have bad consequences.
  – Diminishing returns
    • Hemodynamics, stress amplification, dead space, power
• Strategies with this objective have shown no consistent benefit.
  – HFO and APRV
  – Exogenous surfactant
Exogenous Natural Surfactant for Treatment of Acute Lung Injury and the Acute Respiratory Distress Syndrome

Jozef Kesecioglu¹, Richard Beale², Thomas E. Stewart³, George P. Findlay⁴, Jean-Jacques Rouby⁵, Laurent Holzapfel⁶, Peter Bruins⁷, Edmee J. Steenken⁸, Ole K. Jeppesen⁸, and Burkhard Lachmann⁹

Am J Respir Crit Care Med Vol 180. 989–994, 2009

418 patients with ALI/ARDS randomly assigned to receive usual care either with or without instillation of exogenous natural porcine surfactant HL 10 as large boluses at 0, 12 and 36 h..

The study was prematurely terminated because safety analysis showed a trend toward higher mortality in the treatment group
ARDS is Mechanically Heterogeneous
Some units recruit as others over-expand
Experimental ‘Full Recruitment’ May Be Possible But Requires High Pressure

Is ‘more open’ better …or worse?

From Pelosi et al AJRCCM 2001
Opening and Closing Pressures in
*Established* ARDS

Some units can’t be kept open by any tolerable PEEP

High PEEP Offers Diminishing Returns

5 pts. Primary ALI / ARDS

Opening pressure

Closing pressure
Will Some Lung Units **Always** be Opening and Closing?

From Crotti et al AJRCCM 2001

Driving Pressure

Opening pressure

Closing pressure

Cyclic Collapse?
Early Emphasis on Avoiding Atelectrauma...
Later the emphasis shifted to reducing overstretch, junctional stress and shear.
Editorial

Should the Lung Be Rested or Recruited?
The Charybdis and Scylla of Ventilator Management

Didier Dreyfuss & Georges Saumon

AMERICAN JOURNAL OF RESPIRATORY AND CRITICAL CARE MEDICINE VOL 149 1994
Consequences of Striving for the ‘Fully Open’ Lung

- Hemodynamics
- Gas Exchange
- Stress Amplification in Unopened Units
- Raised Mechanical Power
Lung Expansion May Increase Vascular Stresses
Higher PEEP Often Increases Right Ventricular Strain
ARDS is Mechanically Heterogeneous

Heterogeneity amplifies stress with or without tidal recruitment.
PEEP Reduces *Number of Junctional* Units at Risk From VILI with a Fixed Driving Pressure...

...but raises the strain on those *NOT* recruited.
Machine Energy Per Cycle During Inflation

PEEP increases total machine work, but stores most of it until this potential energy is released during exhalation.
Should Tidal Compliance Guide Us To “Optimal PEEP” for the Individual?

Peter Suter
Compliance and Driving Pressure May Help Select *Best PEEP*

Tidal mechanics have disappeared from the ARDS criteria...
‘Optimum’ PEEP is a Compromise…
The ‘Fully Open’ Lung is Not!

Suter NEJM 1975

- Least Deadspace
- Best Compliance
- Least Driving Pressure
- Maintained Cardiac Output
Did *Oscillate* Sail Too Close to the Danger Zones Of the Fully Open Lung?
Didier Dreyfuss
Jean-Damien Ricard
Stéphane Gaudry

Did studies on HFOV fail to improve ARDS survival because they did not decrease VILI? On the potential validity of a physiological concept enounced several decades ago

John J. Marini

Does high-pressure, high-frequency oscillation shake the foundations of lung protection?
Moderation in All Things, Please!
Does The ‘Fully Open’ Lung Objective Make Good Sense?

• *Not* for established or severe ARDS, especially when low driving pressures are used

• *Maybe* for patients ‘at risk’
  – Very early & Recruitable
  – Minimal disease / atelectasis
    • High ventilation requirements

• *Yes* for premature babies experimental animals
Regional Damage of Different Types
Supine Position High PEEP & Large $V_T$
More Uniform Trans-alveolar Pressures

*PEEP-like* Effect
Sustained Traction of “Supine Dependent” Units
Fig. 4 Examples of widening the baby lung by prone position (a, b) and by increasing PEEP from 5 cmH₂O (c) to 15 cmH₂O (d). Note that recruitment in general proceeds from dorsal to ventral regions in the prone position and from ventral to dorsal by pressure increase.
Prone Positioning May Also Improve Right Ventricular Dynamics

Jozwiak, Teboul, Anguel, et al.

Am J Respir Crit Care Med Vol 188, Iss. 12, pp 1428–1433, Dec 15, 2013
Experts’ opinion on management of hemodynamics in ARDS patients: focus on the effects of mechanical ventilation

A. Vieillard-Baron¹,²,³*, M. Matthay⁴, J. L. Teboul⁵,⁶, T. Bein⁷, M. Schultz⁸, S. Magder⁹ and J. J. Marini¹⁰

HEMODYNAMIC FAILURE

SIGNIFICANT PPV

YES

Hemodynamic effect of MV

RELATIVE, ABSOLUTE HYPOVOLEMIA
- Non-dilated RV
- SVC respiratory variations
- Low CVP
- Small IVC

- DISCUSS FLUID EXPANSION (benefits/risks balance)

NO

No hemodynamic effect of MV (consider norepinephrine)

RV OVERLOAD
- Dilated RV +/- ACP
- No SVC respiratory variations
- Elevated CVP
- Large IVC

- NO FLUID EXPANSION
- NOREPINEPHRINE
- RESPIRATORY SETTINGS ADAPTATION (Driving pressure, Pplateau, PEEP?)
- LIMIT HYPERCAPNIA
- PRONE POSITION?

Sinus Rhythm Passive VT > 8 ml/kg
Experts’ opinion on management of hemodynamics in ARDS patients: focus on the effects of mechanical ventilation

A. Vieillard-Baron¹,²,³*, M. Matthay⁴, J. L. Teboul⁵,⁶, T. Bein⁷, M. Schultz⁸, S. Magder⁹ and J. J. Marini¹⁰

PEEP Without Offsetting Recruitment Strains the Right Ventricle in ARDS

Figure 2. — Pressure gradient between diastolic pulmonary artery pressure (PAP) and pulmonary capillary wedge pressure (PCWP) induced by application of a high PEEP.
Right Ventricular Protection

Key points

- Acute cor pulmonale (ACP) occurs in 25% of cases of ARDS.
- ACP worsens prognosis when not taken into account to adapt respiratory settings.
- RV function assessment can be used to estimate the balance between lung recruitment and lung over-distension resulting from the respiratory strategy.
- The RV protective approach includes a plateau pressure below 27–28cmH₂O, the absence of intrinsic PEEP, controlled $P_{a\text{CO}_2}$, a ‘low’ PEEP, and routine use of prone positioning of patients with the most severe ARDS.

RV function, mechanical ventilation and ARDS Bouferrache and Vieillard-Baron

Curr Opin Crit Care 17:30–35
Physiological Proning Effects

- Alters conformation of the lung
- Reduces gradient of trans-lung pressure
  - Airway pressures better reflects overall stress when prone
- Improves matching of ventilation to perfusion
- Recruits and stabilizes atelectatic lung units
- Often Improves right ventricular afterload
- Encourages mouthward migration of secretions
- Attenuates VILI risk
The Myth of ARDS

Diverse Etiologies

Common Pathophysiology

Unified Clinical Approach
ARDS History

• 1959 Surfactant Deficiency *Infant* RDS—IRDS
• 1967 *Adult* RDS described as ‘ARDS’
  – Permeability edema, diffuse alveolar damage
• 1975 Debate: Does a unifying entity ‘ARDS’ exist?
• 1975-1994 Multiple criteria for case selection used
• 1980-1990 Awareness of VILI grows
• 1990- (?) ‘Open Lung’ approaches & controversies
• 1980-1990 Value & need for EBM / clinical trials emphasized
• 1994, 2012 Consensus definitions of ARDS
• 1995—2013 Numerous *failed* clinical trials
• *1990-Present*—Awareness of VILI, heterogeneity, timing
The “baby lung” became an adult
The Baby Lung Became an Adult…
...or at least an adolescent

*Diverse* Etiologies

*Diverse* Pathophysiology

*Diverse* Clinical Approaches
Optimizing PEEP

- History
- Heterogeneity of ARDS
- Physiologic workload on the baby lung
- Increasing the size of the baby lung
  - Benefits
  - Hazards
- *The optimized trade-off*
Three Competing Objectives?

- Accomplish adequate gas exchange
- Protect the lung
- Maintain appropriate hemodynamics

One PEEP may not optimally fill each objective...
IMPEDANCE CHANGES

**Time (Volume)**

**CONSTANT FLOW**

**UPPER LUNG**

**TOTAL LUNG**

**LOWER LUNG**

Over distended

Under recruited
‘Optimum’ PEEP is a Compromise…
The ‘Fully Open’ Lung is Not!

Suter NEJM 1975

Least Deadspace

Best Compliance
Least Driving Pressure

Maintained Cardiac Output
Best PEEP Zone

Airway Pressures

Hemodynamics

Gas Exchange
Decremental PEEP After RM

Settle into ‘Best PEEP’ after opening recruited units.
Optimal PEEP’ may vary as selected $V_T$ changes.
How is the Injured Lung Best Recruited?

- Adequate PEEP
  - Decremental recruiting maneuvers
- Adequate tidal volume (and/or ‘sighs’?)
- Prone position
- Minimize edema (?)
- Lowest acceptable $F_iO_2$ (?)
- Spontaneous efforts (?)
Recruitment is Time-Dependent

Step Change of PEEP

~ 40 SECONDS
Decremental PEEP Setting After Recruitment

10-15 Minutes
Principles for PEEP Selection

• Use no more airway pressure than needed
  – Limit *passive* plateau to ~25-30cmH₂O* and Vₜ to <7 ml/kg*
  – Consider measurement of Pes

• Strategy should vary with stage of illness
  – Early
    • Prioritize Recruitment--PEEP and Proning
      – PV deflection (or oxygenation?)
    • Keep Ventilatory & Cardiac demands low
      – Deep sedation
    • Conservative fluids
  – After first phase (48-72 hours)
    • Attempt to wean PEEP and start reversing control
‘Best PEEP’ Compromise Strategy?

• Minimize effort and ventilation demand
• Choose the $V_T$ or driving pressure to be used in practice (e.g., 6 ml/kg)
• Use least acceptable FiO$_2$ to keep SaO$_2$ $\approx$ 92%
• **Perform a recruiting maneuver** using escalating PEEP to 50-60 mH$_2$O peak pressure (5 breaths at each PEEP level)
• Drop PEEP abruptly from its highest value to 20 cmH$_2$O and drop PEEP further in small steps every 2 min until O$_2$ sat falls or driving pressure rises.
• **Re-Recruit** and drop PEEP to that value plus one step
In Setting PEEP, What Surrounds the Lung is Important!
Aerated Lung Size is Proportional to Transalveolar Pressure
Proning Stiffens Chest Wall & Re-Distributes and Evens Lung Stress

Does Proning Change the Optimal PEEP Set Point?
Proning Does *Not* Consistently Shift ‘Optimum PEEP’

Keenan 2017

Lavaged Pigs

Steeper Slope

Normal & High Abd Pressure
Key Principles

• Raising PEEP poses benefits & hazards with diminishing returns.
  – Benefits are tied to lung recruitment
  – Hazards are tied to lung distention
• Proning maximizes benefit / risk ratio
• PEEP may be titrated using compliance or driving pressure.
• Higher PEEP helps most early on and in severe cases.
STOP!