Acute Respiratory Failure

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Respiratory Failure

Physiologic Definition:
Inability of the lungs to meet the metabolic demands of the body
Can't take in enough $O_2$
or
Can't eliminate $CO_2$ fast enough to keep up with production
Respiratory Failure

- Failure of Oxygenation: $P_{a}O_2<60$ mmHg
- Failure of Ventilation*: $P_{a}CO_2>50$ mmHg

* $P_{a}CO_2$ is directly proportional to alveolar minute ventilation

Acute Respiratory Failure

**Physiologic Classification**

<table>
<thead>
<tr>
<th>Mechanism</th>
<th>Type 1 Hypoxemic</th>
<th>Type 2 Hypercarbic</th>
<th>Type 3 Post-op</th>
<th>Type 4 Shock</th>
</tr>
</thead>
<tbody>
<tr>
<td>Etiology</td>
<td>Shunt</td>
<td>↓Va</td>
<td>Atelectasis</td>
<td>Cardiac Output</td>
</tr>
<tr>
<td>Airspace Flooding</td>
<td>Increased Respiratory load, Decreased ventilatory drive</td>
<td>Decreased FRC and increased Closing Volume</td>
<td>Decreased FRC and increased Closing Volume</td>
<td></td>
</tr>
<tr>
<td>Clinical Setting</td>
<td>Water, Blood or Pus filling alveoli</td>
<td>CNS depression, Bronchospasm, Stiff respiratory system, respiratory muscle failure</td>
<td>Abdominal surgery, poor insp effort, obesity</td>
<td>Sepsis, MI, acute hemorrhage</td>
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</tbody>
</table>
Ventilatory Failure

Inbalance between load on the lungs and the ability of bellows to compensate

Acute Hypoxemic Respiratory Failure

• Shunt disease - intracardiac or intrapulmonary
• Severe V/Q mismatch - asthma, PE
• Venous admixture due to low cardiac output states, severe anemia coupled with shunt and/or V/Q mismatch
Acute Respiratory Distress Syndrome (ARDS)

Leaky alveolar capillaries
Plasma fluid and leukocytes leak into the airspace
Shunt
Hypoxemia
Acute Respiratory Distress Syndrome (ARDS)

American-European Consensus Definition:

- Refractory hypoxemia
  \[ P_{a}O_{2}/F_{i}O_{2} \text{ (P/F ratio)} \]
  - <300 for ALI
  - <200 for ARDS

- A disease process likely to be associated with ARDS
- No evidence of elevated left atrial pressure elevation (by clinical exam, echo or PA catheter)
- Bilateral airspace filling disease on X-ray


Acute Respiratory Distress Syndrome

Each year in the U.S.:

75,000-150,000 cases
Causes of ARDS

**DIRECT LUNG INJURY**
- Pneumonia
- Aspiration of gastric contents
- Pulmonary contusion
- Near-drowning
- Inhalation injury (Cl-, smoke)
- Reperfusion pulmonary edema after lung transplantation or pulmonary embolectomy

**INDIRECT LUNG INJURY**
- Non-pulmonary sepsis/SIRS
- Severe trauma with shock
- Cardiopulmonary bypass
- Drug overdose (Narcotics)
- Acute pancreatitis
- Transfusion (TRALI)
- Drug reaction (ARA-C, nitrofurantoin)
- fat/air/amniotic fluid embolism, bypass
ARDS

Fundamental Pathophysiology:

**Increased alveolar permeability due to direct neutrophil-mediated injury to the alveolar epithelium**

Not a distinct disease - rather a sequelae of activation of lung and systemic inflammatory pathways

Infiltration of the alveolar septum with neutrophils, macrophages, erythrocytes.

Presence of hyaline membranes, and protein-rich edema fluid in the alveolar spaces, capillary injury, and disruption of the alveolar epithelium.
Fibroliferative Phase
fibrosing alveolitis, increased dead space (CO2 retention), decreased lung compliance, pulmonary HTN/right heart failure

Exudative Phase
rapid onset of respiratory failure, refractory hypoxemia, pulmonary edema on CXR (indistinguishable from CHF)

Adapted from: A. Katzenstein
Optimal V/Q matching

Shunt
Severe Hypoxemia

Therapeutic Goals

Maintain reasonable oxygen delivery

Find & fix the primary cause
“Baby Lungs”

FRC can be reduced by 80% or more in ARDS


ARDS Network Trial

Day 1 Ventilatory Characteristics

<table>
<thead>
<tr>
<th>Low VT Group n=432</th>
<th>Traditional VT Group n=429</th>
</tr>
</thead>
<tbody>
<tr>
<td>VT: 6.2 ± 0.9</td>
<td>11.8 ± 0.8</td>
</tr>
<tr>
<td>PEEP: 9.4 ± 3.6</td>
<td>8.6 ± 3.6</td>
</tr>
<tr>
<td>FIO2: 0.56 ± 0.19</td>
<td>0.51 ± 0.17</td>
</tr>
<tr>
<td>Pplat: 25.7 ± 7</td>
<td>33 ± 9</td>
</tr>
<tr>
<td>Peak: 32.8 ± 8</td>
<td>39 ± 10</td>
</tr>
<tr>
<td>PaO2/FIO2: 158 ± 73</td>
<td>176 ± 76</td>
</tr>
<tr>
<td>Paco2: 40 ± 10</td>
<td>35 ± 8</td>
</tr>
<tr>
<td>pH: 7.38 ± 0.08</td>
<td>7.41 ± 0.07</td>
</tr>
</tbody>
</table>

NEJM 342:1301-1308, 2000
ARDS Network Trial

Mortality: 39.8% in traditional tidal volume group, 31% in low tidal volume group (P=0.007)

Also: @ 28 days: more ventilator free days (12 vs. 10), more days without organ failure (15 vs 12), higher rate of liberation from ventilation rate (65.7% vs 55%)
What happens to alveoli in ARDS?

Edema accumulates in alveoli

Diluting & disaggregating surfactant

Surface tension increases

Alveoli collapse

Alveolar collapse decreases FRC and contributes to hypoxemia
Positive End-Expiratory Pressure (PEEP)

• Beneficial Effects
  - Increases FRC, CI, P_aO_2
  - Recruits Atelectatic Units
  - Decreases Qs/Qt
  - Allows Reduction in F_1O_2

• Detrimental Effects
  - Volutrauma
    - Alveolar Overdistention
  - Hemodynamic Derangements
**PEEP**

**Oxygen is:**
- A) good for you
- B) bad for you
- C) all of the above

\[ F_{\text{O}_2} > 0.6 \text{ for 24 hours or more may cause lung injury} \]

PEEP recruits collapsed alveoli,
improves FRC and
improves oxygenation

*An essential therapy for patients with ARDS*

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**ARDS Network Trial**

Assist Control
\[ V_t \text{ 6 cc/kg ideal body weight} \]
PEEP of \( \approx 8-10 \)
**Generally not due to respiratory failure**

**Does Mechanical Ventilation Contribute to MSOF?**


**Controls (n=19):** Rate 10-15 bpm, Vt targeted to maintain PaCO2 35-40 mmHg (mean: 11 ml/kg), PEEP titrated to SaO2 (mean: 6.5), Pplat maintained <35 cmH₂O

**Lung protective ventilation (n=18):** Rate 10-15 bpm, Vt targeted to keep Pplat less than upper inflexion point (mean: 7 ml/kg), PEEP 2-3 cmH₂O above LIP (mean: 14.8)

Plasma and BALF levels of IL-1β, IL-6, IL-8, TNFα, TNFα-sr 55, TNFα-sr 75, IL-1ra, measured within 8 hrs of intubation and again @24-30 hours & 36-40 hours after entry

Mechanical ventilation can induce a cytokine response that may cause or contribute to multiple organ system failure.

The lung is not just an innocent bystander - it functions as an immunomodulatory organ that may participate in the systemic inflammatory response that leads to multiple organ system dysfunction syndrome.

**Biotrauma**
Goals for Management of ARDS

The American-European Consensus Conference on ARDS, Part 2

- Ensure appropriate $O_2$ delivery to vital organs
- Minimize oxygen toxicity/tolerate mediocre ABG’s
- Reduce edema accumulation
- Minimize airway pressures
- Prevent atelectasis/Recruit alveoli
- Use sedation and paralysis judiciously


Survival from “pure” ARDS

1979: 20-50%
2002: 50-90%