ARF, Mechanical Ventilation and PFTs: ACOI Board Review 2013

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UMDNJ-SOM
ARF is the clinical state which occurs when the respiratory system (ie circulatory and lungs) is not able to meet the metabolic requirements of the organism.
Acute Respiratory Failure

- Anatomic- Etiologic
- Physiologic- Etiologic
- Blood Gas
- Radiologic
- Tissue Oxygenation
Anatomic Etiologic Classification

Brain → Muscles → Circulation

Spinal nerves → Lung
Physiologic Etiologic Classification

**ARF**

- **Inadequate Muscle Function**
  - Neuro Muscular Disease

- **Excessive Workload**
  - ARDS
  - COPD
  - IPF

- **Inadequate Respiratory Drive**
  - OD
  - CVA
  - Alkalosis
Blood Gas Classification

Hypoxemic/Hypercapnic

- Clinically useful
- Can be used to divide patients into distinct ETIOLOGIC and TREATMENT groups
- Readily available
Calculation of the A-a Gradient

PAO2 = FIO2 \( (P_b - 47) \) - 1.25 PaCO2

PaO2 = measured

A-a gradient should be less than 20 mmHg breathing room air OR
Less than 100 mmHg on 100 % O2
100% Oxygen and Pulmonary Shunt

Room Air Ventilation

Ideal alveolar
PO$_2$ = 109
PCO$_2$ = 35

Mixed venous blood

PO$_2$ = 33
PCO$_2$ = 44
Sat = 59%
Content = 11.6

1.75 liters/min
### Causes of Hypoxemia

<table>
<thead>
<tr>
<th>CAUSE</th>
<th>A-a Gradient</th>
<th>PaCO2</th>
<th>Response to 100 % Oxygen</th>
</tr>
</thead>
<tbody>
<tr>
<td>Low FIO2</td>
<td>Normal</td>
<td>Normal</td>
<td>Improved</td>
</tr>
<tr>
<td>Hypoventilation</td>
<td>Normal</td>
<td>Increased</td>
<td>Improved</td>
</tr>
<tr>
<td>Diffusion Impair</td>
<td>Increased</td>
<td>Normal</td>
<td>Improved</td>
</tr>
<tr>
<td>Low V/Q</td>
<td>Increased</td>
<td>Normal</td>
<td>Improved</td>
</tr>
<tr>
<td>Shunt</td>
<td>Increased</td>
<td>Normal</td>
<td>NOT Improved</td>
</tr>
<tr>
<td>Low PvO2</td>
<td>Increased</td>
<td>Normal</td>
<td>? Improved</td>
</tr>
</tbody>
</table>
Mechanisms of Hypercapnia

\[ \text{PaCO}_2 \, = \, \frac{\text{VCO}_2}{\text{K} \cdot \text{Va}} \]

- \text{PaCO}_2 = \text{arterial CO}_2 \text{ tension}
- \text{K} = \text{proportionality constant}
- \text{VCO}_2 = \text{CO}_2 \text{ production}
- \text{Va} = \text{Alveolar ventilation}
Causes of Hypercapnia

1. Alterations in CO2 production

2. Disturbances in the Gas Exchanger (the lungs)

3. Abnormalities in the mechanical system (the bellows)

4. Changes in ventilatory control
<table>
<thead>
<tr>
<th>WHITE LUNG</th>
<th>BLACK LUNG</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pneumonia</td>
<td>Asthma</td>
</tr>
<tr>
<td>Pulmonary edema</td>
<td>emphysema</td>
</tr>
<tr>
<td>Atelectasis</td>
<td>PE</td>
</tr>
<tr>
<td>Interstitial disease</td>
<td>microatelectasis</td>
</tr>
<tr>
<td></td>
<td>R to L Shunt</td>
</tr>
<tr>
<td></td>
<td>Ventilatory failure</td>
</tr>
</tbody>
</table>
pulmonary fibrosis due to RA
76 yo
Female

SOB
Edema
Orthopnea
Male

SOB
Male 40 yo

Dyspnea
Male 40 yo

Dyspnea
<table>
<thead>
<tr>
<th>CONDITION</th>
<th>DEFINITION</th>
<th>EXAMPLE</th>
<th>ABNORMALITY</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ventilatory Failure</td>
<td>Abnormal CO2 elimination by lungs</td>
<td>Drug overdose</td>
<td>PaCO₂ &gt; 50 mm Hg</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Asthma</td>
<td></td>
</tr>
<tr>
<td>Failure of Arterial Oxygenation</td>
<td>Abnormal O2 uptake by lung</td>
<td>Pneumonia, ARDS</td>
<td>PaO₂ &lt; 50 mm Hg</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Failure of Oxygen Delivery</td>
<td>Abnormal O₂ delivery to the tissues</td>
<td>Cardiogenic shock, Anemia, CO poisoning</td>
<td>CvO₂ &lt; 18 cc/dl, PVO₂ &lt; 30 mm Hg, SvO₂ &lt; 60 %</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Failure of Oxygen Utilization</td>
<td>Failure of O₂ uptake by tissues</td>
<td>Cyanide poisoning, septic shock</td>
<td>CvO₂ &gt; 18 cc/dl, PVO₂ &gt; 60 mm Hg, SvO₂ &gt; 80 %</td>
</tr>
</tbody>
</table>
## Acute Respiratory Failure

### Treatment

<table>
<thead>
<tr>
<th>HYPOXEMIA (PaO2 &lt; 50 torr)</th>
<th>HYPERCAPNIA (PaCO2 &gt; 50 torr)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Increase FIO2</td>
<td>Increase alveolar ventilation</td>
</tr>
<tr>
<td>Reduce V/Q mismatch</td>
<td>Reduce V/Q mismatch</td>
</tr>
<tr>
<td>Eliminate Shunt</td>
<td>Haldane</td>
</tr>
<tr>
<td>Increase ventilation</td>
<td>Reduce VD/VT</td>
</tr>
<tr>
<td>Reduce Diffusion Impairment</td>
<td>Reduce CO2 Production</td>
</tr>
<tr>
<td>Increase PvO2</td>
<td></td>
</tr>
</tbody>
</table>
Objectives of Mechanical Ventilation
Tobin MJ. NEJM 1994; 330:1056-61

- Improve pulmonary gas exchange
  Reverse hypoxemia
  Relieve acute respiratory acidosis

- Relieve respiratory distress
  Decrease the O2 cost of breathing
  Reverse respiratory muscle fatigue

- Alter pressure-volume relations
  Prevent/reverse atelectasis
  Improve compliance
  Prevent further lung injury

- Permit lung and airway healing

- Avoid complications
**CPAP**

Pressure applied during entire respiratory cycle
Does NOT AUGMENT TIDAL VOLUME
Splint open the upper airway
Recruit collapsed alveoli

**BiPAP**

* Different pressure during Ins and Exp
I-PAP can AUGMENT tidal Volume
E-PAP can prevent airway closure and recruit collapsed alveoli

USEFUL FOR CHF, COPD, - May prevent need for INTUBATION
What volume do we want?

1. Shearing forces
2. Overdistention of normal lung

Ideal

Volutrauma
Classic Approach to MV in Acute Lung Injury

Use HIGH TV (10-15 cc/Kg) and HIGH PEEP (10 - 20 cm H2O)

Volutrauma

Inflection Point
How do we measure Plateau Pressure

Figure 1. Proximal airway pressure recording during an end-inspiratory airway occlusion and during an end-expiratory occlusion.
How should we approach MV in ARDS TODAY?

- TV smaller (5 cc/Kg)
- PEEP (above inflection point)
- Keep plateau pressure < 30 cm H2O
- THIS MAY RESULT IN HYPERCAPNIA!
• Until recently it was believed that alveolar rupture was due to excessive proximal airway pressure

• If peak airway pressure exceeded 50 cm H2O then the patient was considered to be at high risk for alveolar rupture.
Barotrauma

• If inspiratory resistance is HIGH, DISTAL ALVEOLAR PRESSURE may be LOWER than PEAK AIRWAY PRESSURE!

Alveolar Pres = 20 cm H2O

PAP = 50 cm H2O
Animal Experiment – Same pressure is applied to both animal lungs

Banded lungs

Un-Banded lungs
Macroscopic aspect of rat lungs after mechanical ventilation at 45 cm H2O peak airway pressure. *Left:* normal lungs; *middle:* after 5 min of high airway pressure mechanical ventilation. Note the focal zones of atelectasis (in particular at the left lung apex); *right:* after 20 min, the lungs were markedly enlarged and congestive; edema fluid fills the tracheal cannula.
Recent studies in animals with normal and diseased lungs suggest that it is alveolar OVERDISTENTION and NOT EXCESSIVE PRESSURE which leads to alveolar rupture.

VOLUME NOT PRESSURE Causes alveolar rupture
AutoPEEP is a pressure gradient between the alveoli and the central airways due to INSUFFICIENT EXPIRATORY TIME.

Unlike applied PEEP which is deliberately set, AUTO-PEEP is inadvertent.
• Reported in 47 % of patients in medical ICU's (Wright. Heart and Lung 1990; 19:352-357)

• Occurs in 100 % of MV patients with Ve above 20 L/min (Brown. Respir Care 1986; 31:1069-74)
<table>
<thead>
<tr>
<th>Type of AP</th>
<th>Causes</th>
</tr>
</thead>
<tbody>
<tr>
<td>AP with Hyperinflation and Airway obstruction</td>
<td>Dynamic airway closure</td>
</tr>
<tr>
<td>AP with Hyperinflation and NO Airway obstruction</td>
<td>High Ve vent circuitry, valves or filters which delay exhalation</td>
</tr>
<tr>
<td>AP with NO Hyperinflation and NO Airway obstruction</td>
<td>Forced exhalation</td>
</tr>
</tbody>
</table>
• Use of Flow Waveform (qualitative)
• Esophageal Balloon or inductive waveforms
• Block exhalation and allow alveolar and central pressures to equilibrate (Total PEEP)
Auto PEEP detection

FLOW

Auto-PEEP Occurs

TIME
How do we measure AutoPEEP

**Figure 1.** Proximal airway pressure recording during an end-inspiratory airway occlusion and during an end-expiratory occlusion.
AutoPEEP can be measured by blocking the airway at the END OF EXHALATION.

This allows the distal alveolar pressure to equilibrate with the proximal airway pressure.
### AutoPEEP: Methods to Reduce

<table>
<thead>
<tr>
<th>Increase Expiratory Time</th>
<th>Decrease Minute Ventilation</th>
<th>Decrease Expiratory Resistance</th>
</tr>
</thead>
<tbody>
<tr>
<td>Increase peak flow</td>
<td>Decrease Rate</td>
<td>Medications</td>
</tr>
<tr>
<td>Square Wave</td>
<td>Decrease Tidal Volume</td>
<td>Remove kinks, secretions, casts</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Larger ET tube</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Change filters</td>
</tr>
<tr>
<td>Effect</td>
<td>Mechanism</td>
<td>Treatment</td>
</tr>
<tr>
<td>-------------------</td>
<td>----------------------------</td>
<td>----------------------------</td>
</tr>
<tr>
<td>&quot;Routine&quot;</td>
<td>↑ PVR, ↓ CO</td>
<td>Decrease RR</td>
</tr>
<tr>
<td></td>
<td>↑ Vd/Vt</td>
<td>Increase Vt/Ti</td>
</tr>
<tr>
<td></td>
<td>Patient has to create a - pressure greater than AP to trigger a MV breath</td>
<td>Decrease Vt</td>
</tr>
<tr>
<td>Triggering</td>
<td></td>
<td>Extrinsic PEEP to = AP</td>
</tr>
</tbody>
</table>
“New Berlin definition” ARDS

• Predicted mortality ever-so-slightly better than the existing definition (created at the 1994 American-European Consensus Conference/AECC), when applied to a cohort of 4,400 patients from past randomized trials.
## New ARDS Definition

<table>
<thead>
<tr>
<th>ARDS Severity</th>
<th>PaO2/FiO2*</th>
<th>Mortality**</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mild</td>
<td>200 – 300</td>
<td>27%</td>
</tr>
<tr>
<td>Moderate</td>
<td>100 – 200</td>
<td>32%</td>
</tr>
<tr>
<td>Severe</td>
<td>&lt; 100</td>
<td>45%</td>
</tr>
</tbody>
</table>

*on PEEP 5+; **observed in cohort
“Berlin definition”

- Onset of ARDS (diagnosis) must be acute, as defined as within 7 days
- Bilateral opacities may be detected on CT or chest X-ray
- “not fully explained by cardiac failure or fluid overload”
- [JAMA online May 21, 2012](https://doi.org/10.1001/jama.2012.527991)
Pulmonary Function Tests

1. Spirometry
2. Determination of Reversibility
3. Lung Volume
4. Bronchial Hyperreactivity (Methacholine Challenge)
5. Diffusing Capacity for CO
6. Exercise
Pulmonary Function Tests

**WHY?**

1. To determine if lung disease is present
2. To screen for subclinical disease
3. To determine severity of known disease
4. To determine reversibility
5. To follow disease course
6. Pre-operative evaluation
Volume/Time Curves

Definitions

- FEV1
- FVC
Volume/Time Curves

Obstruction versus Restriction

Volume

Time

1 sec

Normal
Obstructed
Restricted

FEV1 can be reduced by Obst or Rest disease
### Differentiation of Obstruction from Restriction

<table>
<thead>
<tr>
<th>VARIABLE</th>
<th>RESTRICTION</th>
<th>OBSTRUCTION</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>FVC</strong></td>
<td>Reduced</td>
<td>N or Reduced</td>
</tr>
<tr>
<td><strong>FEV1</strong></td>
<td>Reduced</td>
<td>Reduced</td>
</tr>
<tr>
<td><strong>FEV1/FVC</strong></td>
<td>Normal</td>
<td>Reduced</td>
</tr>
<tr>
<td><strong>TLC/RV/FRC</strong></td>
<td>Reduced</td>
<td>N or Increased</td>
</tr>
</tbody>
</table>
Response to Bronchodilator

% Change

FVC

FEV1

% Change
Flow-Volume Curve

Definitions

FLOW

VOLUME

Exp

TLC

RV

Ins
Flow-Volume Loop
Normal and Restrictive FVL
Obstructive FVL
Flow-Volume Curve
Sleep Apnea/ OHS

FLOW

Saw-toothing

Exp

Ins

VOLUME
Flow-Volume Curve

Severe Airway Obstruction

FLOW

Exp

Airway Collapse

Ins

VOLUME
**Effect of dynamic extrathoracic airway obstruction**  
Effects of forced expiration and inspiration in dynamic extrathoracic airway obstruction. Left, during forced expiration, intrathoracic pressure ($P_{tr}$) exceeds the pressure around the airway ($P_{atm}$), lessening the obstruction. Right, during forced inspiration, when intrathoracic pressure falls below the atmospheric pressure, the obstruction worsens resulting in flow limitation. (Redrawn from Kryger, M, Bode, F, Antic, R, et al, Am J Med 1976; 61:85.)
Subglottic Stenosis
Intra and Extra Thoracic Obstructions

<table>
<thead>
<tr>
<th>VARIABLE</th>
<th>VARIABLE</th>
<th>FIXED</th>
</tr>
</thead>
<tbody>
<tr>
<td>Extrathoracic</td>
<td>Intrathoracic</td>
<td>FIXED</td>
</tr>
</tbody>
</table>
Effects of dynamic intrathoracic airway obstruction  Left panel, during forced expiration, the intrathoracic intratracheal pressure ($P_{tr}$) is less than the pressure in the pleural pressure ($P_{pl}$), worsening the obstruction. Right, during forced inspiration, intratracheal pressure exceeds the pleural pressure, lessening the degree of obstruction. (Redrawn from Kryger, M, Bode, F, Antic, R, et al, Am J Med 1976; 61:85.)
Intrathoracic Tracheal Compression
Intra and Extra Thoracic Obstructions

VARIABLE

Extrathoracic

VARIABLE

Intrathoracic

FIXED
Bronchial Provocation Testing
Diseases associated with Nonspecific Bronchial Hyperresponsiveness

- Asthma
- COPD
- Bronchiolitis
- Viral URI
- Hay Fever
- Cystic Fibrosis
- Foreign body aspiration
- Near drowning
- Smoke inhalation
- Sarcoidosis
- Post ARDS