Pathophysiology of Respiratory Compromise

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Pathophysiology of Respiratory Compromise

• Determinants of Respiratory Homeostasis
  – Ventilatory pattern
  – Lung resting size (FRC)
  – Gas exchange
  – Right heart function

• Mechanisms and Physiologic Consequences of Compromise
  – CNS injury/depressants (incl sleep disorders)
  – Compliance loading
  – Resistance loading
  – Vascular loading

• Systemic Manifestations
Ventilatory Pattern

• Inputs into brainstem pacer network
  – Neural (consciousness, stress response)
  – Mechanical (stretch [loads, volume], irritant)
  – Gas exchange (PO2, PCO2, pH)

• Outputs into phrenic (and other) nerves
  – Rate, tidal volume, I:E timing (flow)

• Overall “goal”:
  – Maintain adequate PO2 and pH while minimizing loads and irritant activity
Corical Inputs
- Rhythm Generator

Outputs
- PO2, pH inputs
- Stretch, irritant inputs
Ventilatory Pattern

- For PAO2 to fully saturate hemoglobin, must approach 100 mmHg
\[ f = \frac{1 + 2a \times RC \times \frac{\text{Min Vol} - (f \times V_d)}{V_d}}{a \times RC} - 1 \]
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• Overall “goal”:
  – Maintain adequate PO2 and pH while minimizing loads and irritant activity
  – True in both health and disease but goals may compete
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Lung Resting Size (FRC)
Gas Exchange

- Alveolar ventilation
  
  \[ VA = VE - VD. \text{Anatomic } VD = 1\text{ml/lb} \]

- Ventilation-perfusion matching
- Diffusion
Gas transport impacted by alv-cap membrane (DM) and capillary blood volume (Vc)
Right Heart Function

• Pre-load
  – Fluid status, mean intrathoracic pressure

• Afterload
  – Mean intrathoracic pressure
  – Pulmonary vascular function – hypoxic vasoconstriction

• Adrenergic tone
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CNS Injury/CNS depressants  
(Improvised Ventilator Controller)

• Hypoventilation and/or erratic breathing leads to reduced VA
  – Hypoxemia, acidosis

• Supine position, hypoventilation leads to atelectasis and VQ mismatch
  – Hypoxemia

• May be complicated by poor airway protection and aspiration
  – Hypoxemia, acidosis, mechanical loading
Sleep Disordered Breathing
(Impaired Ventilatory Controller with Sleep)

• Central and/or obstructive apneas/hypopneas lead to reduced VA
  – Hypoxemia, acidosis

• Failure of arousal reflexes
  – Hypoxemia, acidosis, respiratory arrest
Compliance Loading

- Increased elastic WOB
  - Rapid shallow breathing pattern, dyspnea
- Reduced VA (reduced VE, increased VD)
  - Hypoxemia, acidosis
- Parenchymal inflammation/edema/collapse leads to VQ mismatch
  - Severe hypoxemia and RV impairment
  - Reduced FRC
ALI and Compliance Loading

50% of pts
- 42% ALI
- 15% mortality

50% of pts
- 10% ALI
- 41% mortality

Gatttinoni NEJM 2006
Lung Resting Size (FRC)
Terminal rise of RR due to severe metabolic (lactic) acidosis

Onset Potentially Mortal Event (e.g. Sepsis, CHF, PE)

Divergence Pattern of SpO₂ and RR

First SpO₂ Threshold Warning (breach - 85)

Potentially Fatal False Sense of Security (may exceed 12 hours)
Resistance Loading

• Increased resistive WOB and air trapping
  – Competing goals: VE vs air trapping
  – Longer expiratory time initially, then tachypnea with dyspnea, then bradypnea and ultimately arrest
  – Complicated by diaphragm malposition

• Reduced VA from reduced VE and markedly increased VD
  – Hypoxemia, acidosis,

• Intrinsic PEEP from Air Trapping
  – RV dysfunction
Ventilatory Pattern
Lung Resting Size (FRC)
NORMAL SUBJECT

A

PATIENT WITH COPD

B

- Ribcage
- Dome of diaphragm
- Zone of apposition
- Diaphragm with loss of zone of apposition
OAD: Chest radiograph

Posteroanterior

Lateral

Flattened Diaphragm

Enlarged Retrosternal Air Space

Flattened Diaphragm
Vascular Loading

• Increased RV afterload (mechanical, hypoxia)
  – Hypotension, dyspnea
• Increased VD
  – Tachypnea, dyspnea
• Impaired VQ
  – Hypoxemia
## Patterns of Respiratory Compromise

<table>
<thead>
<tr>
<th>Etiology</th>
<th>Vent Pattern</th>
<th>FRC</th>
<th>Hypercapnia</th>
<th>Hypoxemia</th>
<th>RV dysfunction</th>
</tr>
</thead>
<tbody>
<tr>
<td>CNS</td>
<td>Slow, erratic, dec VA</td>
<td>sl reduced</td>
<td>Yes</td>
<td>Later</td>
<td>Late</td>
</tr>
<tr>
<td>Crs Load</td>
<td>Rapid shallow dec VA late</td>
<td>reduced</td>
<td>Later</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>Raw Load</td>
<td>Short Ti, Incr R dec VA</td>
<td>increased</td>
<td>Yes</td>
<td>Later</td>
<td>Yes</td>
</tr>
<tr>
<td>Vasc Load</td>
<td>Tachypnea</td>
<td>no change</td>
<td>No</td>
<td>Yes</td>
<td>Pronounced</td>
</tr>
</tbody>
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Systemic Effects of Respiratory Compromise from Any Cause

• Hypoxemia affects CNS
  – Mental status changes, confusion, agitation
• Severe hypoxemia can affect all organs (MODS)
• Acidosis affects cardiovascular and hemodynamic function
• Dyspnea and muscle loading produces stress response and adrenergic stimulation
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