Respiratory Failure in Children
Assessing the need for Mechanical Ventilation

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Objectives...

- Define respiratory failure
- Overview of respiratory physiology
- Causes of hypoxemia/hypercapnia
- Clinical signs/investigations
- Ventilator management
Respiratory Failure

- Defined as the impairment of the lung’s ability to maintain adequate oxygen and carbon dioxide homeostasis.
Significance

- Respiratory failure has significant morbidity and mortality.
- Optimal ventilatory management will reduce morbidity and mortality.
- Optimal ventilatory management should be individualized and be based upon the pathophysiology and certain basic concepts of mechanical ventilation.
Physiologic functions of the lung

• **Ventilation**
  The movement of air between the atmosphere and the respiratory portion of the lungs

• **Perfusion**
  The flow of blood through the lungs

• **Diffusion**
  The transfer of gases between the air-filled spaces in the lungs and the blood.

• **Others**
Anatomy of the lungs

- Pulmonary system consists of 2 components:

  - **Airway**: conductive system
  - **Terminal respiratory unit**: alveolar capillary unit where gas exchange occur.
Terminal respiratory unit
Alveolar capillary unit

Airway
Conducting system
Ventilation

- **Depends on**
  - the conducting airways:
    - Nasopharynx and oropharynx
    - Larynx
    - Tracheobronchial tree
  - **Open Alveoli**

- **Function:**
  Moves air in and out of the lung, warms and humidifies. Airways do not participate in gas exchange.
Physiology

- Ventilation
- Perfusion
- Ventilation to perfusion
- Diffusion

Pathology

- Impaired ventilation
- Shunt
- V/Q mismatch
- Diffusion block
Function of the respiratory system
It is Conceptualized as pump consisting of:

1) CNS, Spinal nerves, peripheral nerves, neuro-muscular junction
2) Respiratory muscles, chest wall
3) Conducting airways

failure of respiratory pump leads to hypercapnia

4) Alveolar unit where gas exchange and diffusion occur

Alveolar disorders primarily cause hypoxia.
Causes of respiratory failure

- Respiratory Center in Brain
- Neuromuscular Connections
- Thoracic Bellows
- Airways (upper & lower)
- Lung parenchyma (alveoli)

*It only requires one disrupted “link” to cause respiratory failure!*
Definition continued...

- Historical definition includes “Type 1” vs. “Type 2” respiratory failure
- Basically hypoxic vs. hypercarbic respiratory failure
- Best way to think about it is oxygenation vs. ventilation failure
How is respiratory failure defined??

- Historically usually PaO2 <60 mm Hg, PaCO2 > 50 mm Hg.

- Obviously must take into account patient’s anatomy (i.e. ? cyanotic heart lesion).

- Can develop acutely or over days

- Symptoms/Severity dependent on acuity
Assessing respiratory failure

- Clinical signs and symptoms
- *How does the patient look?*
- Blood gas analysis
- O2 saturation
- Other parameters
- Interpretation should be taken in the context of:
  - Age
  - Presentation
  - Acuity
  - Etiology
Type 1 failure: Oxygenation failure

- Most common type of respiratory failure
- Occurs in wide variety of disease processes
- Will deal with main pathophysiologic derangements:
  1. **Hypoventilation (Alveolar hypoventilation)**
  2. **Shunt:**
     
     percentage of venous blood returning to systemic circulation bypassing the alveolar gas exchange unit
  3. **V/Q mismatch:**
     
     pulmonary edema, pneumonia......
  4. **Low inspired FiO₂**
     
     Smoke suffocation, high altitude......
  5. **Impaired diffusion**
1) Hypoventilation

• PO$_2$ of alveolar gas is balance of removal and replenishment

• In general, O$_2$ consumption varies little

• If the O2 replenishment is not adequate enough, the alveoli O2 will fall.

• **Therefore, alveolar PO$_2$ is determined mostly by level of alveolar ventilation**
Hypoventilation continues

• If ventilation falls, PAO$_2$ drops and PACO$_2$ will rise as well (hypoventilation will always lead to high PaCO$_2$).

• Example of alveolar hypoventilation: over-sedation which leads to hypoxia and hypercarbia.
Case Scenario

- A 2 month old boy underwent abdominal surgery. He came from OR extubated. 2 hours after surgery, he became agitated.

- He was given twice 0.5 mg/kg of morphine sulfate to alleviate pain.

- Subsequently, His breathing became shallow and slow (10/minute).

- ABG: pH 7.10, PaO2 52, PCO2 81, Sat 75% on RA.

- Is he in respiratory failure?

- What is the cause and management?
Hypoventilation continued...

• Hypoxia due to hypoventilation is easily overcome by increasing $\text{FiO}_2$

• May take a while for $\text{PCO}_2$ to equilibrate due to large amount of CO2 in body ($\text{HCO}_3^-$)
Other causes of hypoxia...
The concept of **shunt**...

- Blood entering the arterial system without entering ventilated lung

- **Intra- vs. extra-cardiac shunting**

- Always a small amount of shunt via bronchial vessels, coronary veins (< 5%)

- Most important feature is:
  
  Supplying: 100% O2 **does not** resolve hypoxemia

- PCO2 usually normal or low as minute ventilation usually increased by chemoreceptors
Intra-pulmonary V.S intra-cardiac shunt
A month old baby presented with deep cyanosis and saturation of 70%. O2 saturation did not improve with 02 ABG: pH 7.30, PCO2 30, PaO2 40, HCO3 15, BE - 9
What type of shunt does this patient have?

Example of Intra-cardiac shunt (TOF)
A full term baby developed tachypnea and desaturation after birth. He required intubation with increasing amount of FiO2 to keep saturation > 80%.

ECHO showed normal heart but a ductus arteriosus with R to L shunt and mild TR were observed.

Dx: persistent fetal circulation
A 4 years old child with ARDS, O2 Saturation of 60% while on 100 FIO2. Extra-Cardiac shunt \{Intra-pulmonary shunt \} as a result of severe lung disease \{pneumonia, ARDS....\}
Ventilation-Perfusion Inequality (mismatch V/Q)
• Ventilation / Blood flow are mismatched in different lung fields

• **Most common cause of hypoxemia**

• Usually exclude other causes before settling on V/Q mismatch
Ventilation perfusion relationship

**Figure 5.6.** Effect of altering the ventilation-perfusion ratio on the $P_{O_2}$ and $P_{CO_2}$ in a lung unit.
Ventilation / Perfusion mismatch

*It is most common cause of hypoxic failure*

- Blood
- Pus
- Air
- Water
- Atalectasis

Quantitate using $A - a$ Gradient
3 weeks old infant presented with fever, respiratory distress, Grunting and desaturation. Nasopharyngeal swab is + for RSV. CXR showed evidence of Lower Respiratory Tract Infection (LRTI) leading to **V/Q mismatch**

- Air Trapping
- RUL infiltrate
- Atelectasis
- Streaky Markings
How can we quantify the degree of V/Q mismatching objectively?
A – a Gradient \{Alveolar-arterial oxygen gradient\}

O2 Index \{Oxygen index\}

PaO₂ / FiO₂ Ratio

\[
\frac{[(P_b-P_{H2O}) \times FIO_2 - (PCO_2/.8)] - PaO_2}{PaO_2}
\]

normal value: < 10 (related to age)

• Other useful equations
  
  – PaO₂/FiO₂ Ratio (normal > 400)
  
  – OI = \( \frac{P_{aw} \times FIO_2 \times 100}{PaO_2} \)

  Mean airway pressure (MAP) X FiO₂ X 100%

  \( \frac{PaO_2}{PaO_2} \)

  OI Normally < 5
Example of calculation

Child with ARDS has
PaO2 60, PCO2 40, FiO₂: 0.6, MAP 20

• **A-a gradient:**

\[ \left( (P_b - P_{H_2O}) \times FIO_2 - (PCO_2/0.8) \right) - PaO_2 \]

\[ \left( (760 - 40) \times 0.6 - 40 \times 0.8 \right) - 60 \]

\[ 720 \times 0.6 - 50 \]

\[ 432 - 110 = 322 \quad \text{(NL 10 – 30)} \]

• **OI**

\[ OI = \left( \frac{P_{aw} \times FIO_2 \times 100}{PaO_2} \right) / 60 \]

\[ (20 \times 0.6 \times 100) / 60 \]

\[ = 20 \times 60 / 60 = 20 \quad \text{(NL < 5)} \]

• **PaO2 / FiO2 ratio**

\[ = 60 / 0.6 = 100 \quad \text{(NL > 400)} \]
Clinical examples of V/Q imbalance...

- Asthma
- Pulmonary edema
- ARDS
Ventilation perfusion mismatch secondary to RDS
A 10 days old infant was referred to our ICU because of respiratory distress and heart murmur. V/Q mismatch due to pulmonary edema.
Other causes of hypoxia (uncommon in children) Low inspired FIO2

• Deficiency in FIO2: high altitude
• Low oxygen inspired:
  – Inhalation injury
  – Smoke suffocation
Type II Respiratory Failure
Type 2 respiratory failure: Ventilation Failure

• The hallmark is CO2 retention
• There are 3 mechanisms for that:
  ➢ Hypoventilation
  ➢ Dead Space Ventilation
  ➢ Increased CO2:
    {Fever, High Carbohydrate load…..}
3 months old infant with progressive hypotonia, presented with respiratory failure due mainly to hypoventilation. ABG pH 7.1, PCO2 95, PaO2 60, HCO3 45
Increased Dead Space
(factor that decreases cardiac output to the lungs)

- Hypovolemia
- Low cardiac output
- Pulmonary embolus
- High airway pressures
- Short-term compensation by increasing tidal volume and/or respiratory rate
### Volume vs. Pressure Control Ventilation

<table>
<thead>
<tr>
<th>Volume Ventilation</th>
<th>Pressure Ventilation</th>
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</thead>
<tbody>
<tr>
<td>✓ Volume delivery constant</td>
<td>✓ Volume delivery varies</td>
</tr>
<tr>
<td>✓ Inspiratory pressure varies</td>
<td>✓ Inspiratory pressure constant</td>
</tr>
<tr>
<td>✓ Inspiratory flow constant</td>
<td>✓ Inspiratory flow varies</td>
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**When do you use pressure or volume control ventilation?**

- In general pressure control in babies and small infant < 6-8 kg
- In general volume control in children and adolescence > 8-10 kg
Initial Settings

- **Settings**
  - Rate: start with a rate that is somewhat normal; i.e., 15-20 for adolescent/child, 20-30 for infant/small child
  - FiO$_2$: 100% and wean down
  - PEEP: 3-5
  - TV 8-10 ml/kg, or PIP 14-20
  - Pressure support 5-10
  - Determine the mode: control every breath (A/C) or some (SIMV)
Summary

• The practice of the art of mechanical ventilation lies in the application of the underlying scientific and physiologic concepts to the specific clinical situation.

• An individualized flexible approach aimed at maintaining adequate gas exchange with the minimum of lung injury, should optimize the possible outcome.
THANK YOU