Respiratory Failure in Children

Diagnosis and Management

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Objectives

- Define Respiratory Failure.
- Review Physiology of Respiration.
- Categorize Respiratory Failure by Physiologic Mechanisms.
- Develop an approach to Etiologic Diagnosis.
- Outline Treatment Modalities based on Physiology and Etiology.
- Discuss Examples - i.e. Acute Upper Airway Obstruction.
Definition

- Inability to meet one's need for tissue oxygenation and elimination of CO2, often but not always associated with distress.

- Will focus on Pulmonary aspects of this process.

- 50% of pediatric ICU admissions.

- Produced by a wide variety of diseases.
Orientation

- Oxygenation and Ventilation are Essential to Living.
  # Two simultaneous goals in management.
- Diagnosis and treatment of underlying disease.
  # Amelioration of pathophysiology producing ARF, independent of diagnosis.
- Relative importance depends on degree of failure and rate of change.
  # Focus on Physiologic Approach
Respiratory Physiology

# Developmental aspects

# Ventilation: Dead Space, Distribution, Lung Volumes, FRC, Closing Capacity


# Perfusion: Lung Zones, HPV,

Developmental Physiology

- Conducting Airways relatively smaller 1st 5 years.
- Cartilage spread to segmental bronchus, 12 w gest.
- Alveoli: fewer, smaller, less surface area /BSA
- Neonates, Premies: Pause, Apnea, Flat CO2 response, Decrease V to hypoxia
- Chest wall compliant: deforms, wastes effort.
Dead Space

A. Anatomic = Conducting Airways, 2ml/kg

B. Alveolar = non perfused alveoli (PE, hypo tension, excess PEEP, CC > FRC)

Physiologic = A + B

VD = (PaCO2 - PE LGBTQ) × VE / PaCO2

Normally VD / V_tidal = 0.3

This increases in most disease states.

More on this under V/Q matching.
Distribution of Ventilation

# More ventilation to bases in healthy lungs due to less P-transpulmonary at end expiration.

# Shift in pressure-volume relationship can change this dramatically.
Lung Volumes

- Total Lung Capacity
- Vital Capacity
- Tidal Volume
- Functional Residual Capacity
- Residual Volume
- Closing Capacity

RESPIRATORY FAILURE
FRC

Volume in Lung at end expiration. Balance between factors favoring collapse, and those favoring expansion.

Represents gas volume available for exchange.

Faster desaturation at lower FRC.

Lower FRC favors atelectasis.
Closing Capacity

- Volume at which small airways begin to collapse, preventing further gas exchange with those lung units.
- Normally well below FRC.
- Closer to FRC in Infants.
- When CC exceeds FRC, this happens during normal tidal breathing with resultant air trapping and maldistribution of Ventilation.
## Convergence of FRC and CC

<table>
<thead>
<tr>
<th>Elevation of CC</th>
<th>Reduction of FRC</th>
</tr>
</thead>
<tbody>
<tr>
<td>Infancy</td>
<td>Supine Position</td>
</tr>
<tr>
<td>Bronchiolitis</td>
<td>Abdominal Distension</td>
</tr>
<tr>
<td>Asthma</td>
<td>Surgery, Atalectasis</td>
</tr>
<tr>
<td>BPD</td>
<td>Pulmonary edema</td>
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<tr>
<td>Smoke Inhalation</td>
<td>Near Drowning</td>
</tr>
<tr>
<td>Cystic Fibrosis</td>
<td>ARDS, Pneumonitis</td>
</tr>
</tbody>
</table>
Work of Breathing

# Done during inspiration.
# Overcome tissue viscoelastic resistance of lung and chest wall.
# Move air into lungs against resistance to flow.
# Tissue returns work to move air out.
Compliance

- Lung compliance
- Chest Wall compliance
- Total compliance
- Specific compliance is indexed to FRC.
Decreased Total Compliance

Decreased $C_L$

- Increased Recoil
  - ARDS, pneumonitis
  - edema, near drowning
- Overexpansion
  - Asthma, Bronchiolitis
  - Toxic or Thermal Inhalation
  - Excess PEEP or CPAP
- Volume loss
  - Atalectasis
  - Supine position

Decreased $C_w$

- Thoracic Trauma or Surgery
- Abdominal Surgery
- Diaphragmatic Loading
  - Abdominal Distension
  - PD, MAST
- Pneumothorax
- Pleural Effusion
- Thoracic Bony deformities
# Resistance

- Pressure change needed to produce Flow.

- Laminar flow defined by Hagen-Poiseuille equation:
  \[
  \text{Resistance} = \frac{P}{V} = \frac{8 \pi \eta l}{r^4}
  \]

- Turbulent flow increases resistance, and makes resistance flow dependent, such that \( P \) is proportional to \( V^2 \) and density.

- \( \frac{1}{\text{Resistance}} = \text{Conductance} \).

- Specific Conductance = Conductance / Lung Volume. Similar in infants and adults.
Sites of Increased Airway Resistance

# In Adults -- Upper Airway, Nose.

# In Children -- Peripheral Airways.

# Dynamic Airway Compression: Increased Intrapleural pressure during forced exhalation augments collapse of intrathoracic airways.

# Worse with BPD, alpha-1-antitrypsin deficiency due to poor cartilege.

# Extrathoracic airway effected on inhalation.
Time Constants

# Time required for lung unit to fill to 63% of final volume.
# Time constant = Resistance x Compliance
# Those alveoli with shorter time constants fill faster.
# Local variation in resistance and compliance effect gas distribution.
# e.g. overall TC is increased in Asthma
Surfactant

# LaPlace's Law  \( P = \frac{2T}{r} \)

# This would predict that small alveoli would empty into large ones.

# However Surfactant allows a decrease in surface tension as the radius decreases.

# Therefore Pressure stays the same.

# Made by type II pneumocytes.

# Surfactant deficiency occurs in many disease states.
Pulmonary Circulation

# Development closely follows airway / alveolar development.

# Limited in pulmonary hypoplasia (eg CDH)

# Muscular wall actively remodels during development.

# Smooth muscle gradually extends more distally, but may extend faster with ensuing Pulm. Ht'n.

# Pulm circulation receives the entire C.O.
West Zones I

# Define by relationship of pressures affecting local pulmonary blood flow.

# Upstream pressure is $P_{PA}$ (pulmonary aretery)

# Downstream pressure is the greater of:

1. $P_{PV}$ (pulmonary veins) $\approx$ (left atrium)

2. $P_{A}$ (alveolus)

# Note the latter increases with Positive Pressure Ventilation.
West Zones II

1

2

3

PA

PPA

PPV

PA > PPA > PPV

PPA > PA > PPV

PPA > PPV > PA
HPV

- Alveolar Hypoxia leads to local pulmonary vasoconstriction.
- Usually useful to match perfusion to ventilation.
- With whole lung hypoxemia it produces pulmonary hypertension, and possible R to L shunt via PFO.
- Chronically leads to increased musculature and chronic pulmonary hypertension
**V / Q Matching**

# $V / Q = 0.6$ at bases; $= 3$ at apices

# True shunt is blood with no contact with aerated alveoli. (eg cardiac, atelectasis)

# Venous admixture (virtual shunt) amount of mixed venous blood to add to pulmonary end capillary blood to produce observed arterial O2 content.

# $PAO_2 = (FiO_2 \times (PB - 47)) - (PaCO_2 / R)$

# Normally A-a DO2 is small due to obligate shunt.
Lung Units

Idealized alveoli

Shunt

Matched V / Q

Dead Space
Virtual Shunt Lines

Hb
10-14 g/dl

PaCO₂
25-40 mmHg

a-v O₂ difference
5ml/100ml

Arterial
PaO₂

Inspired O₂ concentration (%)
0 5% 10%

15%
20%
25%
30%
50%
Alveolar-Capillary Membrane

May contribute to "diffusion" block of O2 movement. But this mechanism is rarely the sole cause of significant hypoxemia.

However, transudation of fluid across the membrane is a major cause of respiratory failure.

Function of 1. Pressure gradient. 2. Oncotic forces. 3. Filtration Coefficient.

Leads to 1. Decreased Compliance 2. Alveolar collapse -> Shunt -> Increased Aa O2 gradient.
Exclusions

# Physiology review has focused on lung physiology.
# Also important, but not included in this review are:

1. CNS control of breathing.
2. Neuromuscular transmission.
4. Toxicology
5. Cardiac Function and O2 delivery.
RESPIRATORY FAILURE

Sorting it Out 1

Won't Breath
(lack of Drive)

CNS
Toxic

Can't Breath
(strength inadequate for work required)

Airways
Lungs
Respiratory Pump

# Remember, a child with chronic respiratory disease can present in acute failure due to an exacerbating process.
| **Airway** | Extrathoracic large airway  
Intrathoracic large airway  
small airways |
| **Lung** | Increased closing capacity  
Decreased FRC  
Dead Space  
Shunt |
| **Pump** | Intrapleural  
Chest wall  
Neuromuscular |
### RESPIRATORY FAILURE

#### Sorting it Out 3

<table>
<thead>
<tr>
<th>Stridor</th>
<th>Wheeze</th>
<th>Rales</th>
<th>BS</th>
<th>Retract</th>
<th>IncCO2</th>
<th>DecO2</th>
<th>CXR</th>
</tr>
</thead>
<tbody>
<tr>
<td>X A/W Obs</td>
<td>I &gt; E</td>
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<td>I A/W Obs</td>
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<td>Small A/W</td>
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<td>Inc CC</td>
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<td>Toxic</td>
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<td>++++</td>
<td>+</td>
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</table>
Hypoxia

# The four basic mechanisms which can produce hypoxia.

1. Inadequate FiO2.
2. Decreased Ventilation.
3. Shunt (pulmonary or cardiac).
4. Decreased Cardiac Output.
Treatment

# Provide supplemental Oxygen
# Judge severity, decide if immediate intervention needed.
# Monitors: Pulse oximeter, Respiratory rate, ABG
# Get Assistance if needed.
# Maintain Airway.
# Maintain Breathing
# Treat underlying cause and pathophysiology
# ECMO, Hyperbaric O2
Oxygen

# Simple masks, Nasal Cannula, impossible to know FiO2. Better with Venturi mask.

# Non-Rebreather mask or Hood for infant provide known FiO2 from a mixer.

# High FiO2 may accelerate collapse of closed segments.

# O2 is toxic, Don't use high FiO2 for long periods unless necessary.

# O2 is life-saving, Always use high FiO2 in acute emergency.
Severity 1

The patients in trouble when:

1. Inadequate ventilation: PaCO2 > 50-55
2. Apnea, respiratory pauses (fatigue)
3. Rising PaCO2
4. Vital Capacity <15 ml/kg
5. Dead Space / Tidal Volume > 0.6
6. Change in Level of Consciousness
Severity 2

7. Cyanosis or \(\text{PaO2} < 70\) with \(\text{FiO2} > 0.6\).

8. \(\text{A-a DO2} > 300\) with \(\text{FiO2 at 1.0}\).

9. Shunt Fraction > 15 - 20%.
Airway

# Natural

# Supported: Jaw Lift, Suctioning, OPA, Nasal A/W,

# Artificial: ETT

Size:  
3.0  Newborn
3.5  3-8 months
4.0  9-24 months

Size = (Age / 4) + 4

Cuff adds half a size.
Intubation

# Suction Available.

# Preoxygenate generously. Fill FRC with O2 may take a long time in diseased lungs.

# Monitoring

# Vascular access preferred.

# Sedative / hypnotic, and neuromuscular blockade.

# Cricoid pressure.

# Laryngoscopy and Intubation, Gently

# Confirm: BS, CO2, Chest movement, CXR

# SECURE IT.
Acute Upper Airway Obstruction in Children

Differential Diagnosis

- Epiglottitis
- Croup (viral laryngotracheobronchitis)
- Bacterial Tracheitis, Pharyngeal Abscess
- Foreign Object, Thermal or Chemical Injury
- Diphtheria
- Angioneurotic Edema
- Acute exacerbation of chronic obstruction
Any of these may require emergency airway management if severe.

In Epiglottitis you need to secure the airway ASAP regardless of the patients current level of distress.
Children with Stridor

155 children presenting to the emergency room with acute stridor. 
Supraglottitis

- Acute infection of the Epiglottis and Aryepiglottic folds.
  
  # Sudden onset of sore throat, dysphagia, often with stridor and shortness of breath.
  
  - May result in severe, rapidly progressive airway obstruction in 6 to 12 hours.
  
  # Patients sit forward and drool, don't talk.
  
  - Usually with high fever and bacteremia.
  
  - Usually caused by *Hemophilus influenzae* type B.
# Epiglottitis vs. Croup

<table>
<thead>
<tr>
<th></th>
<th>EPIGLOTTITIS</th>
<th>CROUP</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Age</strong></td>
<td>All, peak 3-5 years</td>
<td>Younger, peak 3 m-3 y</td>
</tr>
<tr>
<td><strong>Etiology</strong></td>
<td>Bacterial (h. Flue B)</td>
<td>Viral (parainfluenza)</td>
</tr>
<tr>
<td><strong>Speed of Onset</strong></td>
<td>Rapid (&lt;24 hours)</td>
<td>Slow (1-4 days)</td>
</tr>
<tr>
<td><strong>Appearance</strong></td>
<td>Anxious, toxic</td>
<td>Frequently non-toxic</td>
</tr>
<tr>
<td><strong>Position</strong></td>
<td>Upright, forward</td>
<td>Variable</td>
</tr>
<tr>
<td><strong>Temperature</strong></td>
<td>Usually high (&gt;39)</td>
<td>Normal to high</td>
</tr>
<tr>
<td><strong>Resp. Distress</strong></td>
<td>Usually present</td>
<td>Variable</td>
</tr>
<tr>
<td><strong>Retractions</strong></td>
<td>Usually late</td>
<td>Progressive</td>
</tr>
<tr>
<td><strong>Voice/Cough</strong></td>
<td>Muffled or absent</td>
<td>Hoarse/ &quot;seal&quot; bark</td>
</tr>
<tr>
<td><strong>Stridor</strong></td>
<td>Yes, less with more obstruct.</td>
<td>Yes</td>
</tr>
<tr>
<td><strong>Mouth</strong></td>
<td>Open, forward, drooling</td>
<td>Closed, nasal flaring</td>
</tr>
</tbody>
</table>
Bacterial Tracheitis

- Rare
- Similar to croup at first,
- Patient becomes toxic appearing
- Progressive Respiratory distress
- At risk for acute life threatening airway obstruction
- Diagnosis usually made at intubation for presumed severe croup.
- Management similar to epiglottitis
Foreign object

- Should be considered in every child with acute upper airway obstruction.
- History may or may not help
- Age, usually under 4 years, can be any age
- Stridor may or may not be present
- Wheezing may be present
- Fever not common early
- Usually not toxic appearing
- Radiograph may be definitive, only if positive
Approach to the Patient with Acute Upper Airway Obstruction

1. Prepare.
2. Does the Patient have Extrathoracic Airway Obstruction?
3. Assess the severity.
4. Decide about immediate treatment vs further evaluation.
Extrathoracic Airway Obstruction

- Stridor, if present, is greater on Inspiration.
- Suprasternal, Supraclavicular Retractions
- Chest Wall Retractions in Infants
  -# Stridor may be less with worse obstruction
Severity of Distress

- Stridor without tachypnea
- Tachypnea without distress
- Retractions, Decreased Activity
- Increased work, Use of accessory muscles
- Irritability and air hunger
- Fatigue may develop
- Lethargy and cyanosis presage impending respiratory arrest.
RESPIRATORY FAILURE

UAO: An Algorithm

Airway Obstruction

I  II  III

Respiratory failure or moribund
Real distress
Air hunger
Access Musc.
Stridor with mild to moderate distress
Algorithm I

UAO & Respiratory Failure

# Oxygen
# Artificial Airway if immediately available
# Bag and Mask Ventilation, +Pressure
# Cricothyroidotomy
# Cardiac Assessment, and Recussitation
# To ICU or OR
Algorithm II

UAO & Severe Respiratory Distress

# Allow to remain sitting up
# Oxygen, preferably with humidity
# Pulse Oximeter
# Minimize Perturbation
# Arrange transfer to ICU or OR for controlled airway management i.e. Intubation
# No decrease in proximate expertise
Algorithm III

UAO & Mild to Moderate Distress

# Clinical Impression

# If Suspect epiglottitis -- Lateral Neck X-Ray

# Accompany by a physician

# If epiglottitis -- protocol

# If not -- further exam, other studies

# Hospital admission to appropriate unit.
X-Ray Features

# Find the epiglottis
    valecula, arytenoids, hyoid
# Enlarged epiglottis, lack of central lucency
# Balooning of hypopharynx
# Supraglottitis
    ary-epiglottic folds
# "Steeple" sign in croup
# Foreign bodies