

RESPIRATORY FAILURE IN CHILDREN

Critical Concepts Course

Objectives...

- ❑ Define respiratory failure
- ❑ Common causes of hypoxemia/hypercapnia
- ❑ Clinical signs/investigations

How is respiratory failure defined?

- Historically $\text{PaO}_2 < 60 \text{ mm Hg}$, $\text{PaCO}_2 > 50 \text{ mm Hg}$
- Obviously must take into account patient's anatomy (ie - cyanotic heart lesion)
- Can develop acutely or over days
- How the patient looks is usually incorporated into diagnosis/management
- Symptoms/Severity dependent on acuity

Adults vs. Kids

- Multiple differences from underlying airway anatomy to disease process
- Kids usually affected by congenital or infectious processes
- Adults inflicted by respiratory disease such as COPD, as well as infectious processes
- Review differences in vital sign normals such as resp. rate, HR etc... for children of different ages

Clinical decision making...

- ⦿ Acute vs. Chronic
 - Helps in deciding acuity of treatment
 - Progression of illness also important - history
- ⦿ Any underlying chronic disease?
 - i.e. Asthma, congenital heart disease...
- ⦿ Examine patient!!!
 - Work of breathing, Level of consciousness, Vitals
 - What tests might be helpful

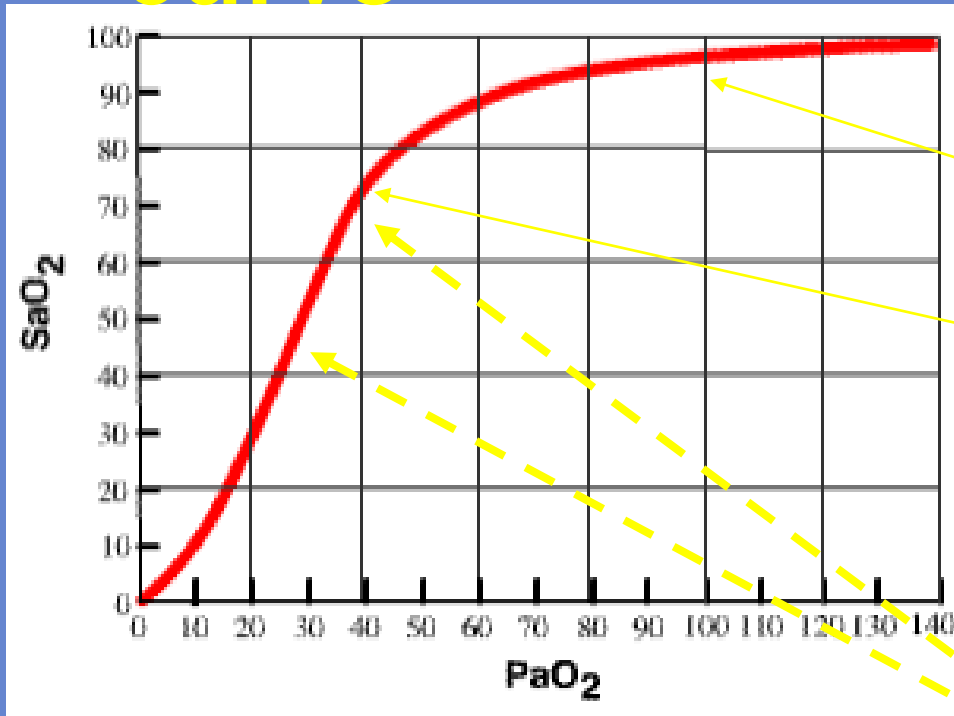
Laboratory investigations

- ⦿ Arterial blood gas (if possible)
 - *Gives info on oxygenation and ventilation status*
 - *Difficult to get in some patients*
 - *Obtaining and ABG should be part of resident skills*
- ⦿ Other blood gas – ventilation info but not oxygenation
 - *Venous – good only if obtained from free flowing site – no tourniquet*
 - *capillary – easiest to obtain*
- ⦿ Other blood work based on clinical scenario (ie WBC count, cultures if suspect infection)

Important points on blood gas interpretation

- ⦿ Know type of gas (ABG vs VBG vs CBG)
- ⦿ Only interpret PaO₂ on ABG
- ⦿ PaCO₂ slightly higher in VBG
- ⦿ Remember metabolic side (*base deficit, [HCO₃⁻]*)

Oxyhemoglobin dissociation curve



Two key points on curve:

1. PO₂ 100 mm Hg= SpO₂ of 97%
2. PO₂ 40 mm Hg= SpO₂ of 75% (mixed venous blood)

**Note the steep part of the curve in this area
Small changes in clinical status will
produce large swing in SpO₂**

Key points about the oxyhemoglobin saturation curve

- ⦿ Remember how flat the slope is above $PO_2=60$ mm Hg
- ⦿ Any small drop in PO_2 below this will cause precipitous fall in saturation

Oxygenation failure:

- ⦿ Most common type of respiratory failure
- ⦿ Occurs in wide variety of disease processes
- ⦿ Main pathophysiologic derangements:
 - I. V/Q mismatch*
 - II. Shunt*
 - III. Hypoventilation*

Hypoventilation

- FiO_2 of air is 21%
- PaO_2 of air is $(.21 \times (760 \text{ mm Hg} - 47 \text{ mm Hg} \text{ (water vapor))})$
- PO_2 of alveolar gas is balance of removal and replenishment
- O_2 consumption varies little
- Therefore, alveolar PO_2 is determined mostly by *level of alveolar ventilation*
- If ventilation falls, PO_2 drops and PCO_2 will rise (this is key, hypoventilation will **always** lead to **high $PaCO_2$**)

Shunting

- ⦿ 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
- ⦿ Most important feature is 100% O₂ does not resolve hypoxemia
- ⦿ PCO₂ usually normal or low as minute ventilation usually increased by chemoreceptors

Ventilation Perfusion Mismatch

- 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 Mismatch

- Think of V/Q ratios varying from little to no ventilation ($V/Q=0$) to little to no blood flow ($V/Q=\text{infinity}$)
- Those lung units with low V/Q ratios cause hypoxemia
- Units with high V/Q ratios do not compensate for low O₂ content of others due to shape of dissociation curve

VQ mismatch continues...

- ⦿ Mismatch occurs in healthy lungs, difference is accounted for by regional blood flow/ventilation
- ⦿ Ventilation / Perfusion both increase slowly from top to bottom of the lung
- ⦿ Blood flow increases more rapidly than ventilation
- ⦿ VQ ratio subsequently different as you move from 1 lung segment to the other
- ⦿ Lungs with significant VQ mismatch cannot sustain the same levels of PaO₂ /PaCO

What are the important clinical points?

- ⦿ Is there an oxygenation defect?
 - Check A-a gradient

$$= P_A O_2 - P_a O_2 (\text{arterial})$$

$$PAO_2 = FiO_2 - (PaCO_2/0.8) \text{ (alveolar gas equ'n)}$$

- ⦿ Normal value 5-30 mm Hg (age dependent)
- ⦿ If elevated then **almost always** V/Q mismatch

Clinical examples of V/Q imbalance

- ⦿ Asthma
- ⦿ Pulmonary edema
- ⦿ ARDS

How do you follow response to therapy?

- Options include:

- PaO₂/FiO₂ ratio
- Oxygenation index (OI)

$$= \frac{\text{Mean airway pressure (MAP)} \times \text{FiO}_2 \times 100\%}{\text{PaO}_2}$$

- Both validated but OI better when ventilated with positive pressure

CO₂ and respiratory failure

- ⦿ Ventilation = the air moving in and out of lungs
- ⦿ Minute ventilation is amount moving in and out per minute (V_E)
- ⦿ Alveolar ventilation is the volume of air that takes part in gas exchange. Dead space ventilation does not take part in ventilation
- ⦿ PaCO₂ is only measurement that reflects alveolar ventilation and the relationship to CO₂ production
- ⦿ CO₂ production is continuous, elimination is through lungs predominantly

Why we care about hypoxemia/hypercarbia?

⦿ Hypoxemia:

- Significant hypoxemia can lead to tissue hypoxia and anaerobic metabolism
- Different organ systems have different thresholds for tolerating hypoxemia (CNS and heart most vulnerable)
- Arterial PO₂ is only one component of oxygen delivery (DO₂), other important factors include hemoglobin level, cardiac output
- Rising serum lactate is an indicator of significant tissue hypoxia

Hypercarbia:

- ⦿ Controversial topic with emergence of permissive hypercapnia in treatment of ALI/ARDS
- ⦿ Definite CNS effects such as narcosis, mental clouding at high levels
- ⦿ Adverse effects of acidosis produced by hypercarbia may be overstated
- ⦿ Has demonstrated some protective effects against mechanical ventilation induced lung damage

Clinical Recognition










Recognition of Respiratory Problems Flowchart



Pediatric Advanced Life Support Recognition of Respiratory Problems					
Clinical Signs		Upper Airway Obstruction	Lower Airway Obstruction	Lung Tissue (Parenchymal) Disease	Disordered Control of Breathing
A	Patency	Airway open and maintainable/not maintainable			
B	Respiratory rate/effort	Increased			Variable
	Breath Sounds	Stridor (typically inspiratory) Seal-like cough Hoarseness	Wheezing (typically expiratory) Prolonged expiratory phase	Grunting Crackles Decreased breath sounds	Normal
	Air Movement	Decreased			Variable
C	Heart Rate	Tachycardia (early)		Bradycardia (late)	
	Skin	Pallor, cool skin (early)		Cyanosis (late)	
D	Level of Consciousness	Anxiety, agitation (early) Lethargy, unresponsiveness (late)			
E	Temperature	Variable			

Clinical Categorization

Pediatric Advanced Life Support Categorize Respiratory Problems by Severity

	Respiratory Distress		Respiratory Failure
A	Open and maintainable		Not maintainable
B	Tachypnea		Bradypnea to apnea
	Work of breathing (nasal flaring/retractions)		Increased effort
	Decreased effort		Apnea
	Good air movement		Poor to absent air movement
C	Tachycardia		Bradycardia
	Pallor		Cyanosis
D	Anxiety, agitation		Lethargy to unresponsiveness
E	Variable temperature		

Initial Management

Management of Respiratory Emergencies Flowchart <ul style="list-style-type: none"> • Airway positioning • Oxygen • Pulse oximetry • ECG monitor (as indicated) • BLS as indicated 		
Upper Airway Obstruction Specific Management for Selected Conditions		
<i>Croup</i>	<i>Anaphylaxis</i>	<i>Aspiration Foreign Body</i>
<ul style="list-style-type: none"> • Nebulized epinephrine • Corticosteroids 	<ul style="list-style-type: none"> • IM epinephrine (or auto-injector) • Albuterol • Antihistamines • Corticosteroids 	<ul style="list-style-type: none"> • Allow position of comfort • Specialty consultation
Lower Airway Obstruction Specific Management for Selected Conditions		
<i>Bronchiolitis</i>		<i>Asthma</i>
<ul style="list-style-type: none"> • Nasal suctioning • Bronchodilator trial 		<ul style="list-style-type: none"> • Albuterol ± ipratropium • Corticosteroids • SQ epinephrine • Magnesium sulfate • Terbutaline
Lung Tissue (Parenchymal) Disease Specific Management for Selected Conditions		
<i>Pneumonia/Pneumonitis</i>		<i>Pulmonary Edema</i>
<i>Infectious Chemical Aspiration</i>		<i>Cardiogenic or Noncardiogenic (ARDS)</i>
<ul style="list-style-type: none"> • Albuterol • Antibiotics (as indicated) 		<ul style="list-style-type: none"> • Consider noninvasive or invasive ventilatory support with PEEP • Consider vasocactive support • Consider diuretic
Disordered Control of Breathing Specific Management for Selected Conditions		
<i>Increased ICP</i>	<i>Poisoning/Overdose</i>	<i>Neuromuscular Disease</i>
<ul style="list-style-type: none"> • Avoid hypoxemia • Avoid hypercarbia • Avoid hyperthermia 	<ul style="list-style-type: none"> • Antidote (if available) • Contact poison control 	<ul style="list-style-type: none"> • Consider noninvasive or invasive ventilatory support

In conclusion

- ⦿ Think in terms of oxygenation and ventilation
- ⦿ Think WHY (ie physiology) the patient is hypoxic/hypercarbic...
- ⦿ Remember to follow patients closely as they can deteriorate quickly