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 PaO_2 <60 mm Hg, $PaCO_2$ > 50 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

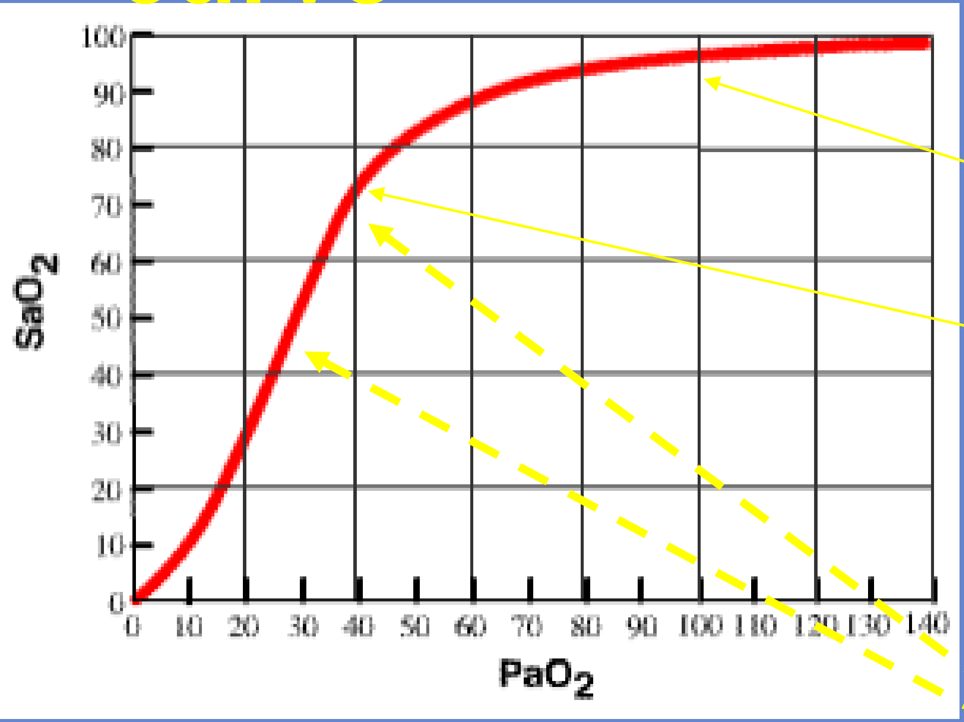
Laboratory investigations

- 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 PaO_2 on ABG
- $PaCO_2$ slightly higher in VBG
- Remember metabolic side *(base deficit, [HCO_3^-])*

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%
- PaO2 of air is (.21 X (760 mm Hg - 47 mm Hg (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 PCO2 will rise (this is <u>key</u>, hypoventilation will **always** lead to high PaCO2

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

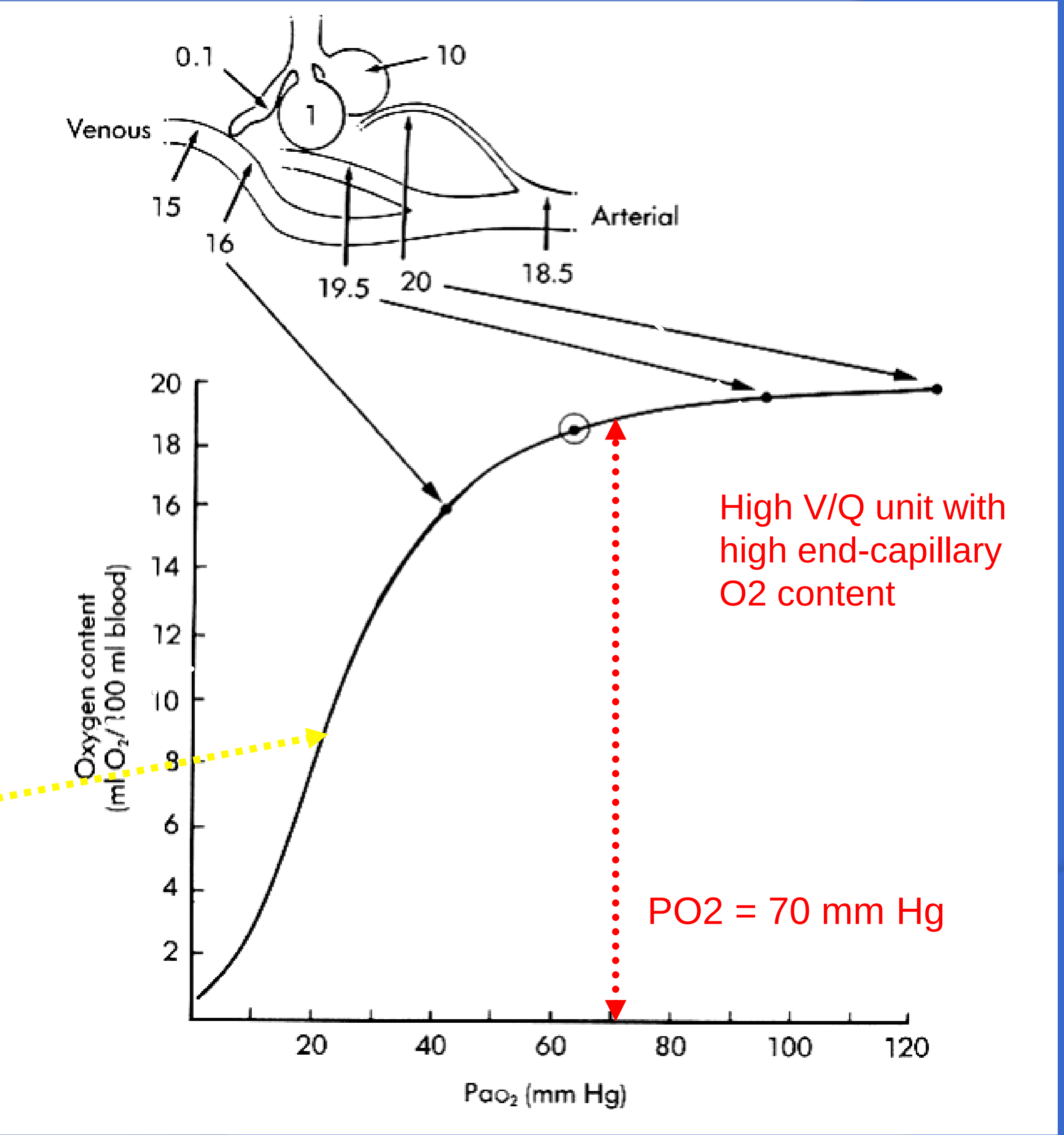
Ventilation Perfusion Mismatch

- 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=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

Low V/Q unit with low end-capillary O2 content



High V/Q unit with high end-capillary O2 content

PO2 = 70 mm Hg

NOTE: Steep part of curve in range of low VO2 units

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_AO2 - P_aO2(arterial)**

PAO2 = FiO2 - (PaCO2/0.8) ***(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

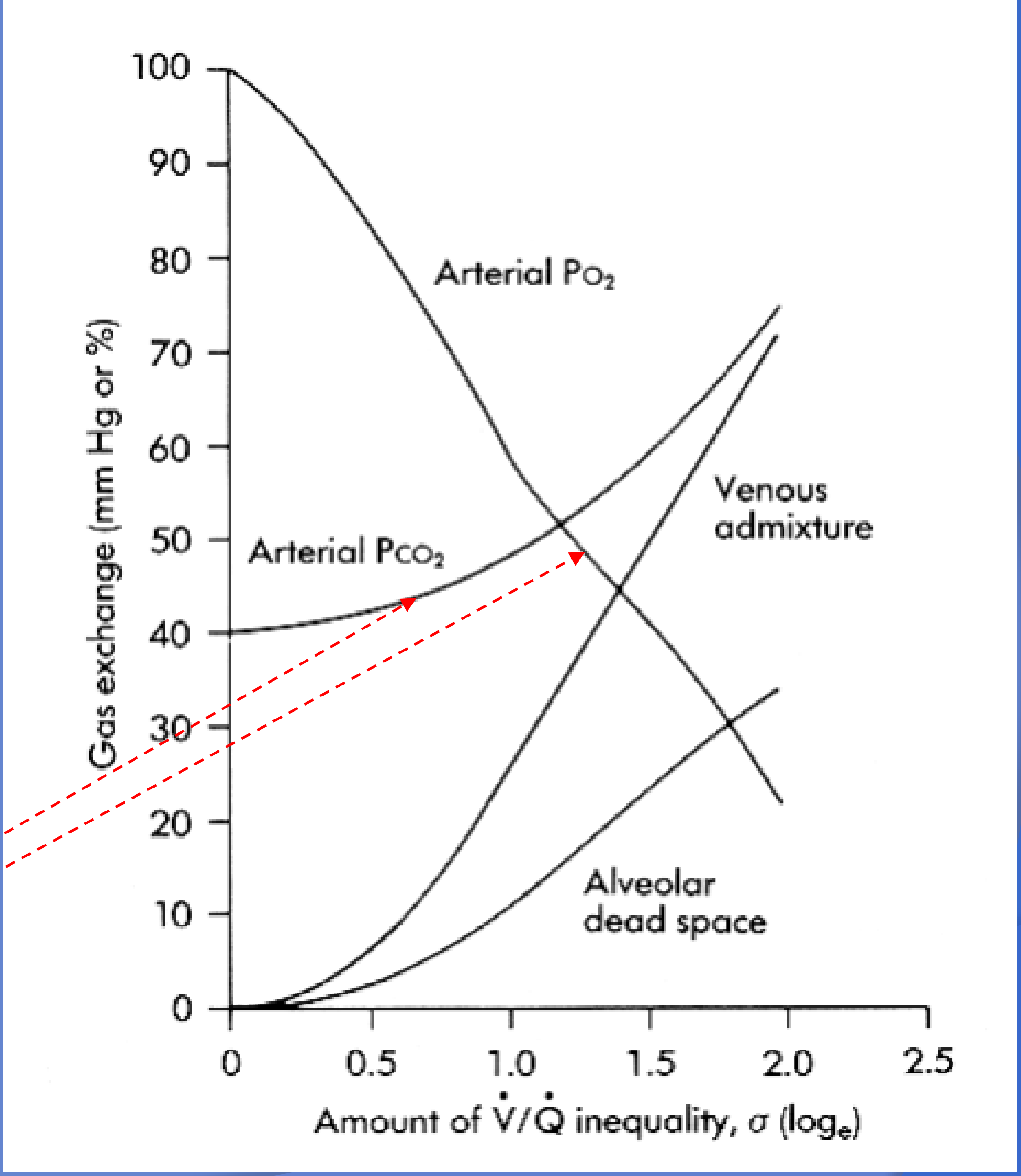
How do you follow response to therapy?

- *Options include*:
  - PaO2/FiO2 ratio
  - Oxygenation index (OI)

$$= \frac{\text{Mean airway pressure (MAP) X FiO2 X 100\%}}{\text{PaO2}}$$

- Both validated but OI better when ventilated with positive pressure

Relationship between V/Q mismatch and gas exchange



NOTE: Steep rate of decline in PaO2 compared to PaCO2

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

American Heart Association.
Learn and Live.

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
B	Breath Sounds	Stridor (typically inspiratory) Seal-like cough Hoarseness	Wheezing (typically expiratory) Prolonged expiratory phase	Grunting Crackles Decreased breath sounds	Normal
B	Air Movement	Decreased			Variable
C	Heart Rate	Tachycardia (early) Bradycardia (late)			
C	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

- Airway positioning
- Oxygen
- Pulse oximetry
- ECG monitor (as indicated)
- BLS as indicated

Upper Airway Obstruction
Specific Management for Selected Conditions

Croup	*Anaphylaxis*	*Aspiration Foreign Body*
• Nebulized epinephrine • Corticosteroids	• IM epinephrine (or auto-injector) • Albuterol • Antihistamines • Corticosteroids	• Allow position of comfort • Specialty consultation

Lower Airway Obstruction
Specific Management for Selected Conditions

Bronchiolitis	*Asthma*
• Nasal suctioning • Bronchodilator trial	• Albuterol ± ipratropium • Corticosteroids • SQ epinephrine • Magnesium sulfate • Terbutaline

Lung Tissue (Parenchymal) Disease
Specific Management for Selected Conditions

Pneumonia/Pneumonitis *Infectious Chemical Aspiration*	*Pulmonary Edema* *Cardiogenic or Noncardiogenic (ARDS)*
• Albuterol • Antibiotics (as indicated)	• Consider noninvasive or invasive ventilatory support with PEEP • Consider vasoactive support • Consider diuretic

Disordered Control of Breathing
Specific Management for Selected Conditions

Increased ICP	*Poisoning/Overdose*	*Neuromuscular Disease*
• Avoid hypoxemia • Avoid hypercarbia • Avoid hyperthermia	• Antidote (if available) • Contact poison control	• Consider noninvasive or invasive ventilatory suport

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

References, Recommended Reading, and Acknowledgements

- Up to Date: Emergent Evaluation of Acute Respiratory Distress in Children
- Nelson’s Textbook of Pediatrics

- Some slides based on work by Dr. Jeff Brusinski for PedsCCM

- American Heart Association PALS guidelines