Respiratory Failure in Children

Diagnosis

and

Management

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Objectives

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

Definition

- **Inability to meet one's need for tissue oxygenation and elimination of CO_2, 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**
- **Mechanics: Work, Compliance, Resistance, Time Constants, Visco-Elastic Properties, Surfactant**
- **Perfusion: Lung Zones, HPV,**
- **V/Q matching: Shunt, Venous Admixture, Virtual Shunt, Alveolar gas equation, A-a gradient.**

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 CO_2 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

$V_D = (PaCO2 - PECO_2)\ V_E / PaCO_2$

Normally $V_D / V_{tidal} = 0.3$

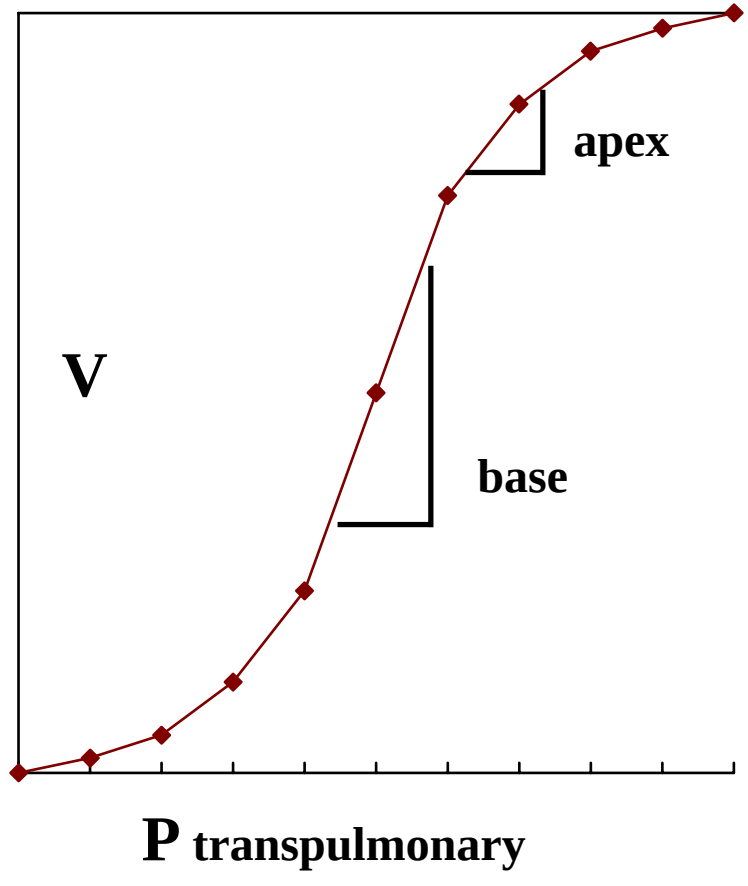
This increases in most disease states.

More on this under V /Q matching.

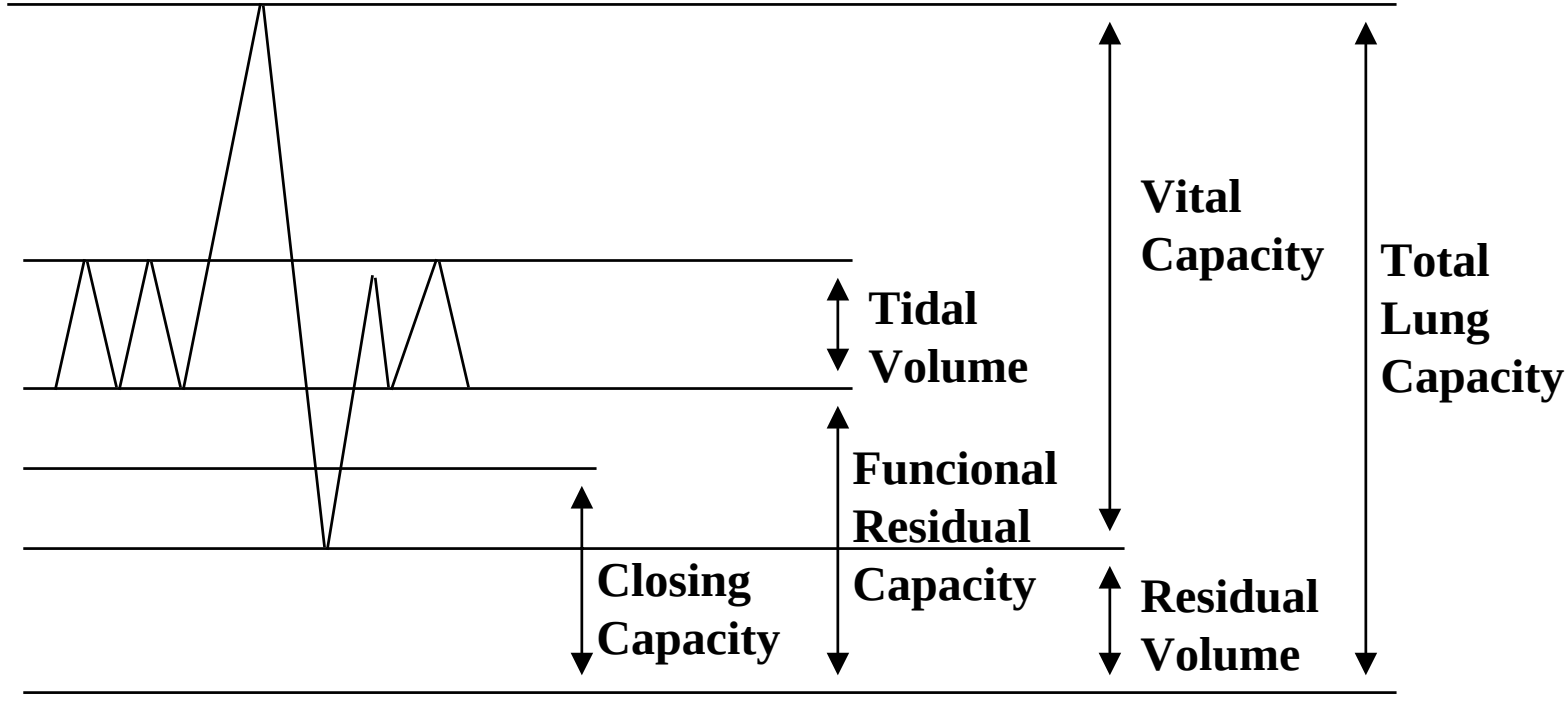
Distribution of Ventilation

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

Shift in pressure -volume relationship can change this dramatically.



Lung Volumes



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 atalectasis.

Closing Capacity

- **Volume at which small airways begin to collapse, preventing further gas exchange with those lung units.**
- **Noramlly 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

Elevation of CC

Infancy

Bronchiolitis

Asthma

BPD

Smoke Inhalation

Cystic Fibrosis

Reduction of FRC

Supine Position

Abdominal Distension

Surgery, Atalectasis

Pulmonary edema

Near Drowning

ARDS, Pneumonitis

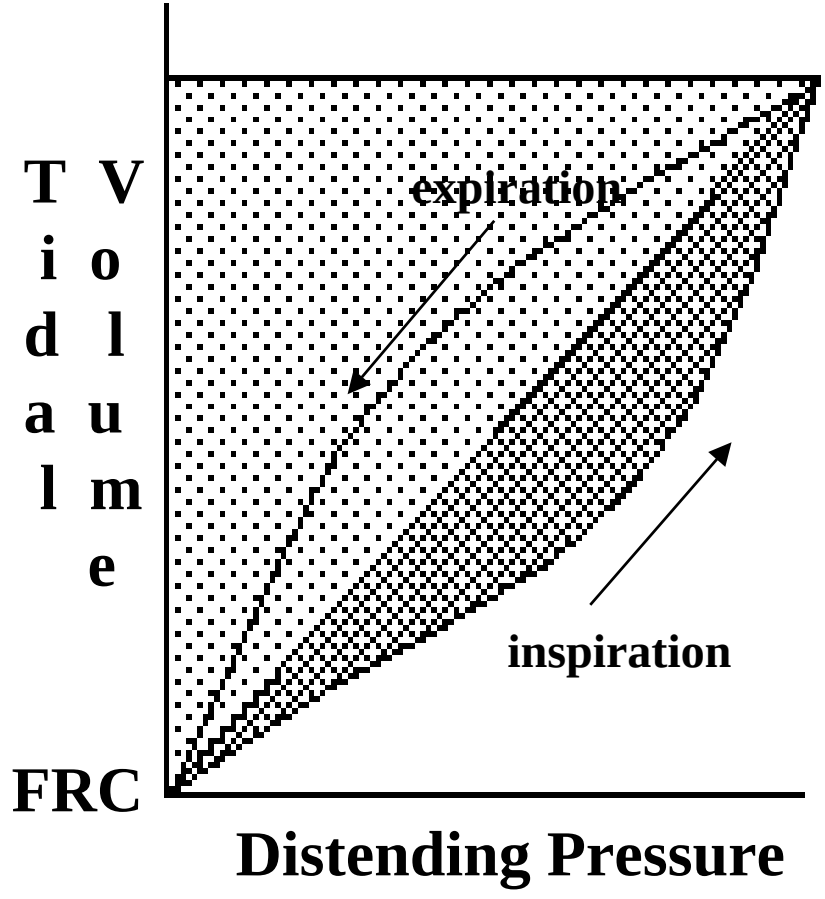
Work of Breathing

Done during inspiration.

Overcome tissue visco-elastic resistance of lung and chest wall.

Move air into lungs aginst resistance to flow.

#Tissue returns work to move air out.



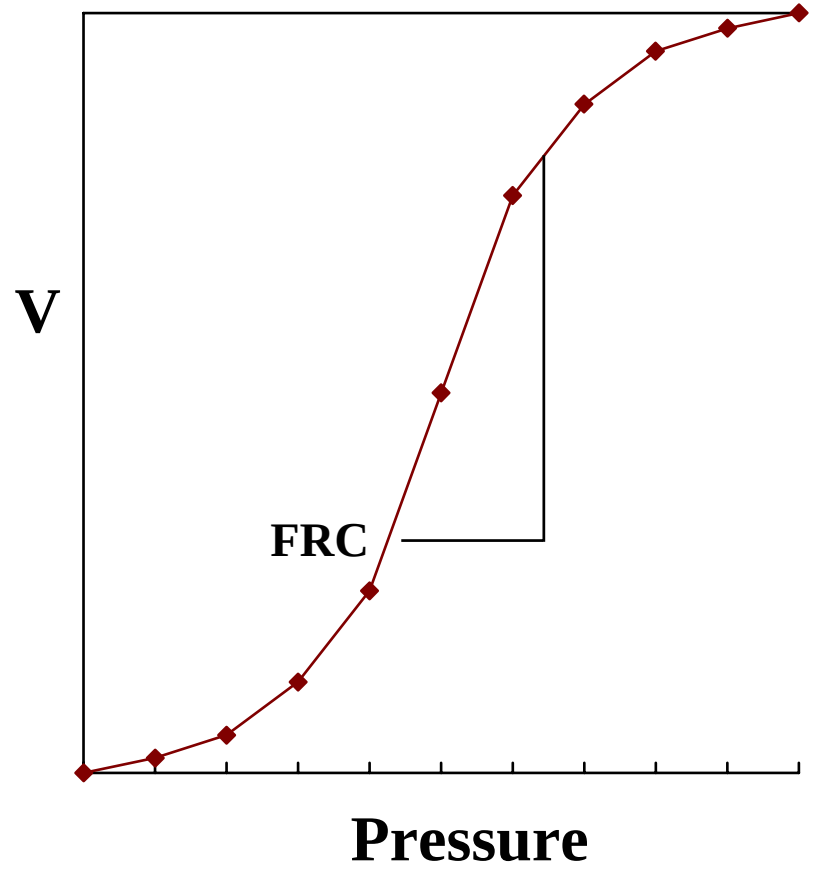
Compliance

Lung compliance

Chest Wall compliance

Total compliance

Specific compliance is indexed to FRC.



Decreased Total Compliance

Decreased C_L

Increased Recoil

ARDS, pneumonitis

edema, neart 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:
  $\text{Resistance} = P / V = 8 h l / r^4$
- Turbulent flow increases resisitance, and makes resistance flow dependent. such that P is proportional to V^2 and density.
- 1 / Resisitance = 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 $P = 2T / r$
- 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 recieves the entire C.O.

West Zones I

Define by relationship of pressures affecting local pulmonary blood flow.

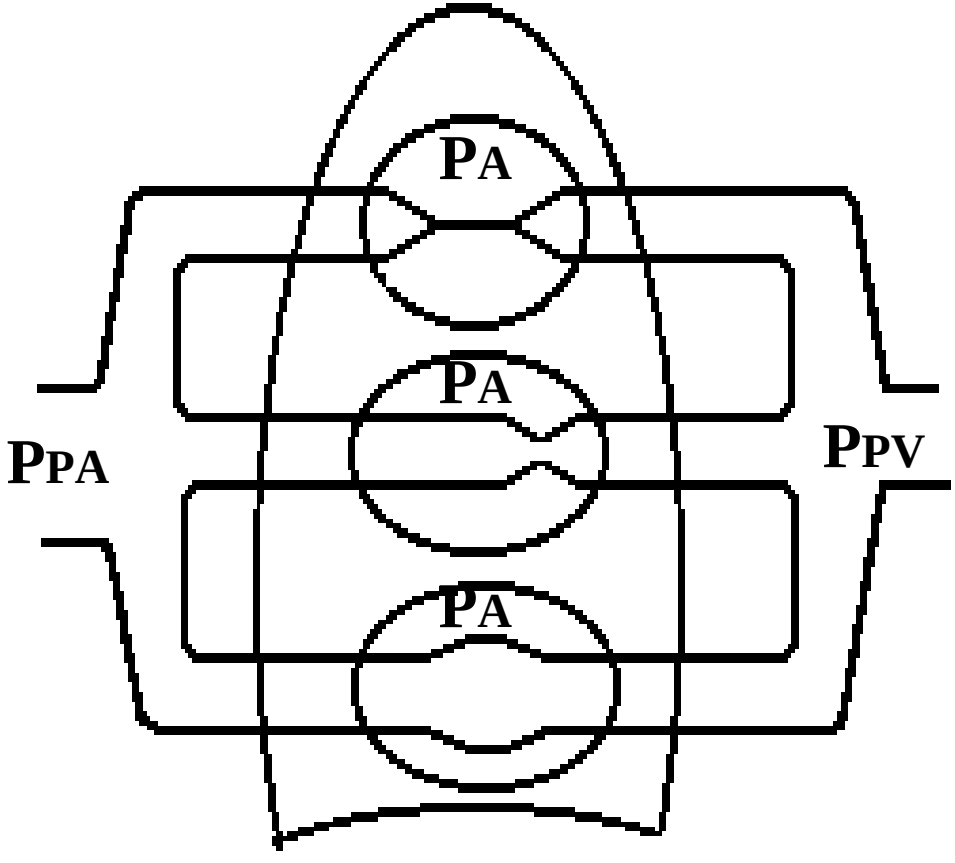
Upstream pressure is P_{PA} (pulmonary aretery)

Downstream pressure is the greater of:

1. P_{PV} (pulmonary veins) ~= (left atrium)
2. P_A (alveolus)

Note the latter increases with Positive Pressure Ventilation.

West Zones II



$P_A > P_{PA} > P_{PV}$

$P_{PA} > P_A > P_{PV}$

$P_{PA} > P_{PV} > P_A$

HPV

- Alveolar Hypoxia leads to local pulmonary vasoconsstriction.
- 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 muscularity and chronic pulmonary hypertension

$\dot{V}$ / $\dot{Q}$ Matching

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

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

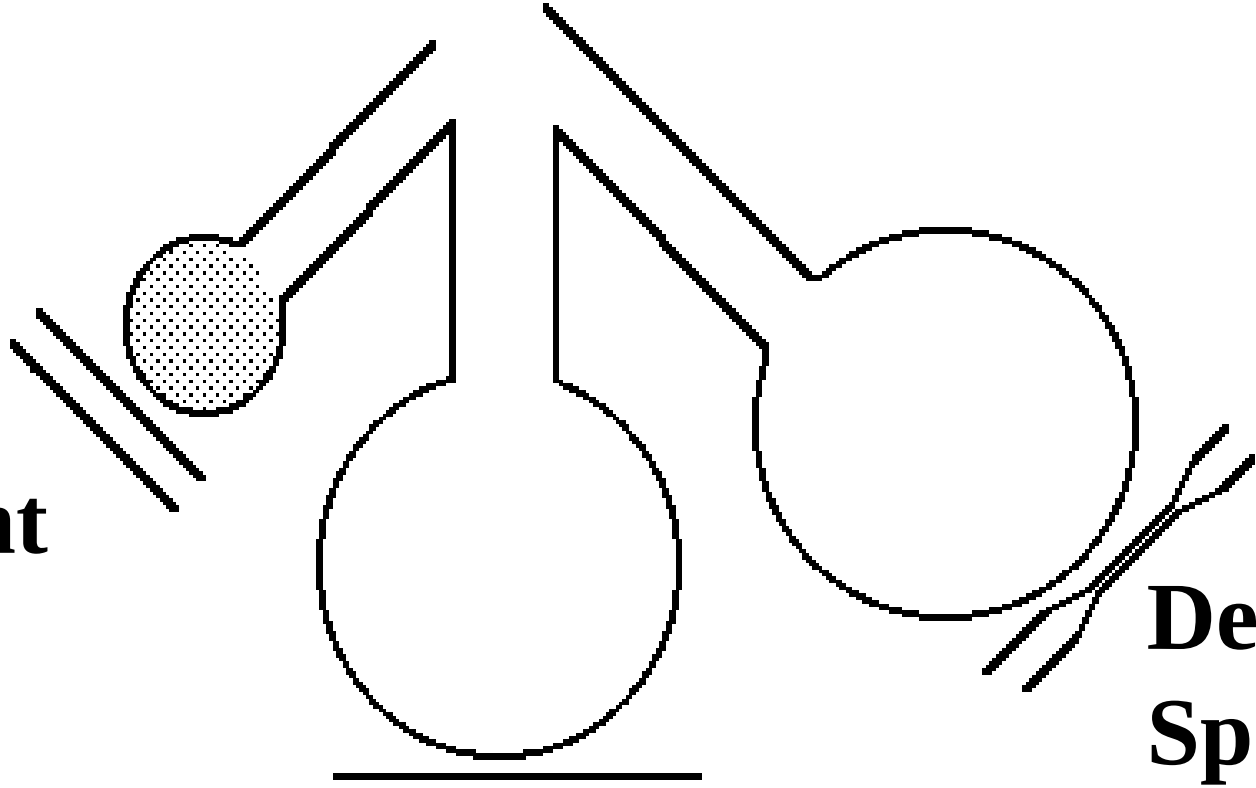
Venous admixture (virtual shunt) amount of mixed venous blood to add to pulmonary end capillary blood to produce observed arterial O2 content.

$P_AO_2 = (F_iO_2 (P_B - 47)) - (P_aCO_2 / R)$

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

Lung Units

Idealized alveoli



Shunt

Dead Space

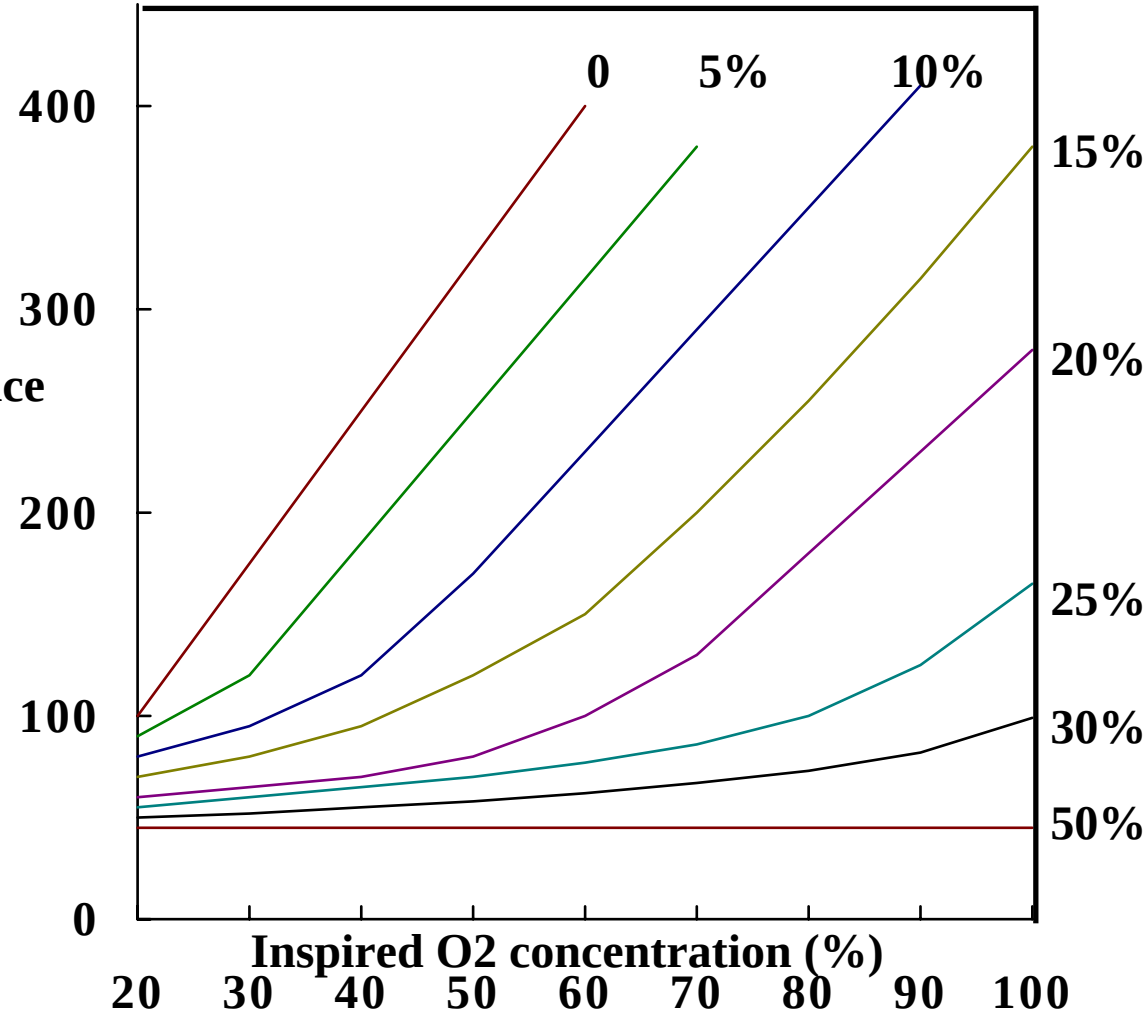
Matched V / Q

Virtual Shunt Lines

Hb
10-14 g/dl

PaCO2
25-40 mmHg

a-v O2 difference
5ml/100ml



Alveolar-Capillary Membrane

May contribute to "diffussion" block of O2 movement. But this mechanism is rarely the sole cause of signifigant 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.
3. Muscular function.
4. Toxicology
5. Cardiac Function and O2 delivery.

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.

Sorting it Out 2

Airway	**Extrathoracic large airway** **Intrathoracic large airway** **small airways**
Lung	**Increased closing capacity** **Decreased FRC** **Dead Space** **Shunt**
Pump	**Intrapleural** **Chest wall** **Neuromuscular**

Sorting it Out 3

	Stridor	Wheeze	Rales	BS	Retract	IncCO2	DecO2	CXR
X A/W Obs	I > E	-	-	=	++++	+++	+	-
I A/W Obs	E > I	+	-	<>	++	+++	+	trap
Small A/W	-	++++	-	=	+	+++	++	trap
Inc CC	-	++++	-	=	+	+++	++	trap
Dec FRC	-	+	++	<>	++	+	++++	low vol
Dead Sp	-	+	-	-	+	+++	++	inc vol
Shunt	-	-	++	<>	++	+	++++	low vol
I Pleural	-	-	-	<>	++	+++	++	shift
Chest W	-	-	-	<>	+++	+++	+	+ / -
NeuroM	-	-	-	=	-	++++	+	bell
CNS	-	-	-	=	-	++++	+	-
Toxic	-	-	-	=	-	++++	+	-

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: $PaCO_2 > 50\text{-}55$**
2. **Apnea, respiratory pauses (fatigue)**
3. **Rising $PaCO_2$**
4. **Vital Capacity <15 ml/kg**
5. **Dead Space / Tidal Volume > 0.6**
6. **Change in Level of Consciousness**

Severity 2

7. Cyanosis or PaO_2 < 70with FiO_2 >0.6.

8. A-a DO_2 >300 with FiO_2 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 Abcess**
- **Foreign Object, Thermal or Chemical Injury**
- **Diphtheria**
- **Angioneurotic Edema**
- **Acute exacerbation of chronic obstruction**

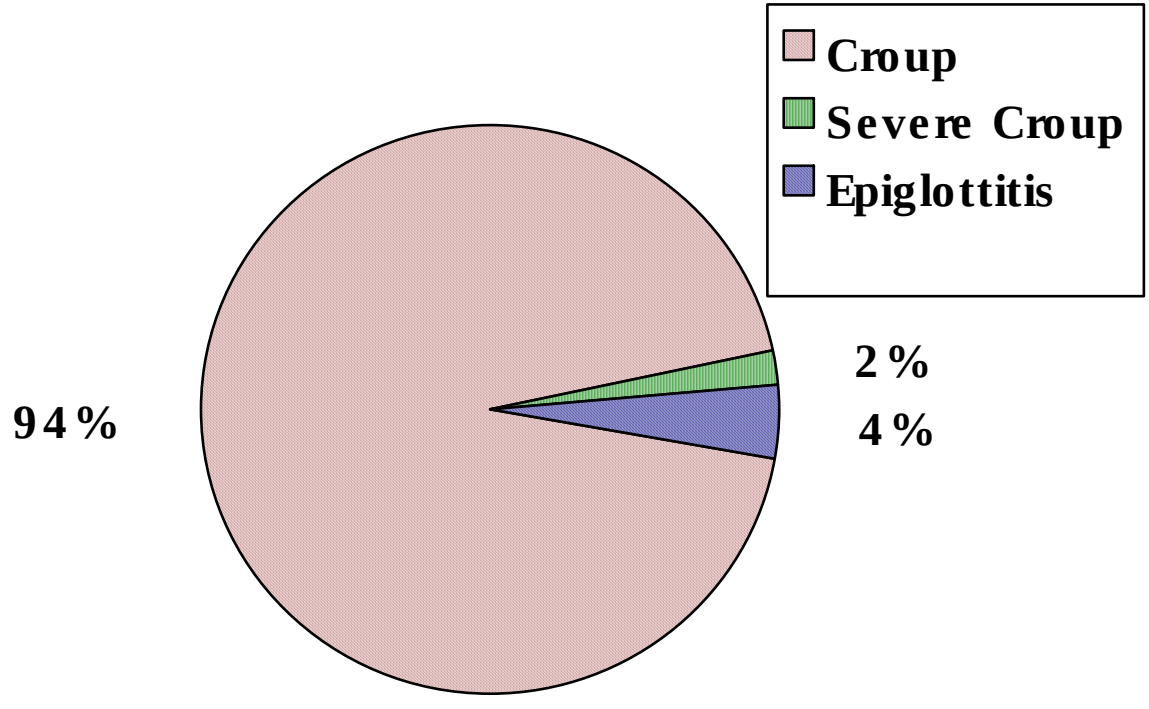
Any of these may require emergency airway management if severe.

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.

Mauro et al, Am. J. Dis. Child., 142(6):679-82, June 1988



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

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

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 <u>Inspiration</u>.**
- **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

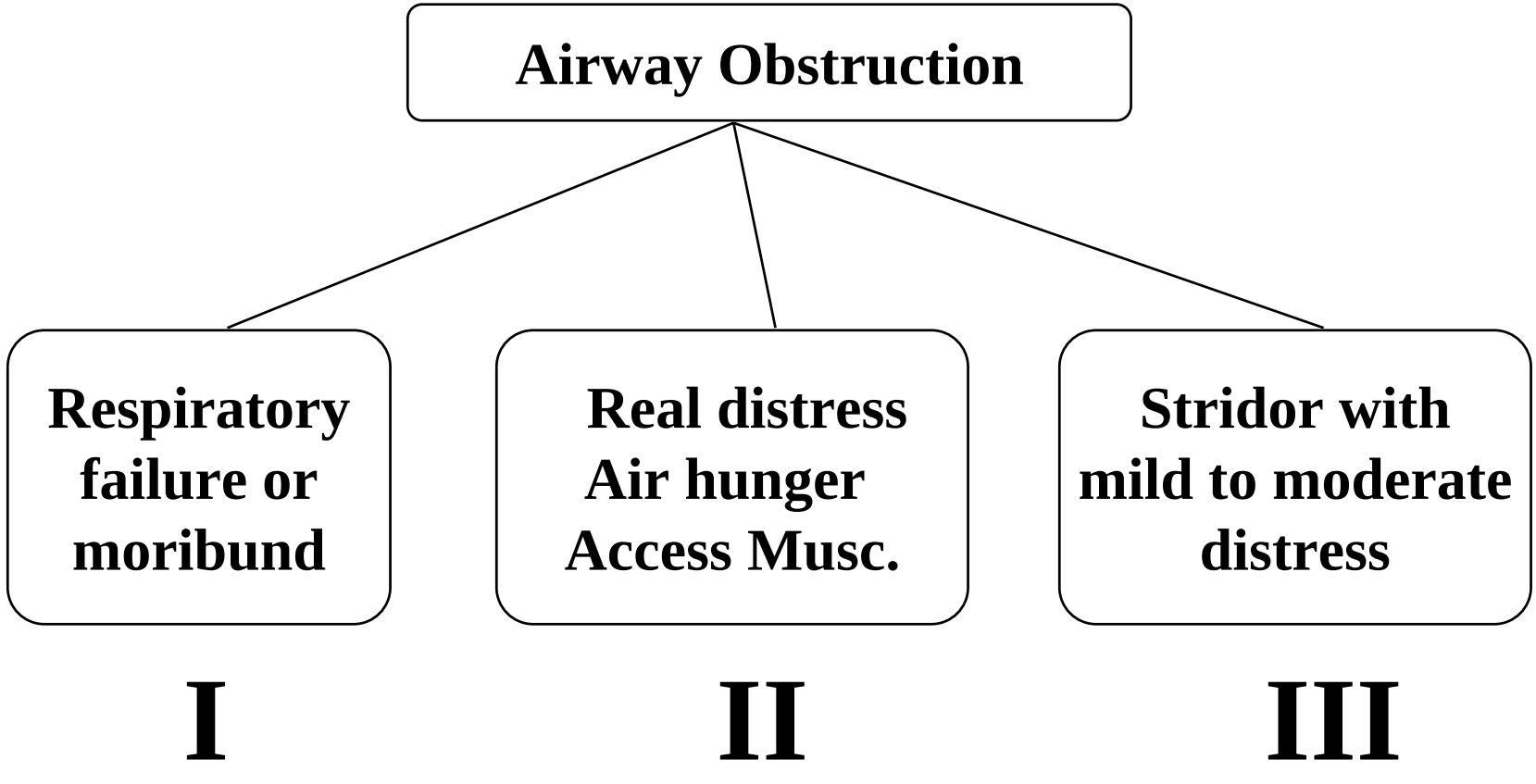
Increased work, Use of accesory muscles

Irritability and air hunger

Fatigue may develop

Lethargy and cyanosis presage impending respiratory arrest.

UAO: An Algorithm



Algorithm I

UAO & Respiratory Failure

- **Oxygen**
- **Artificial Airway if immediatly 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**