

# ***ARF, Mechanical Ventilation and PFTs: ACOI Board Review 2018***

**Thomas F. Morley, DO, FACOI, FCCP, FAASM**  
**Professor of Medicine**  
**Chairman Department of Internal Medicine**  
**Director of the Division of Pulmonary, Critical Care  
and Sleep Medicine**  
**Rowan University - SOM**

***No Disclosures***

# ***Acute Respiratory Failure (ARF)***

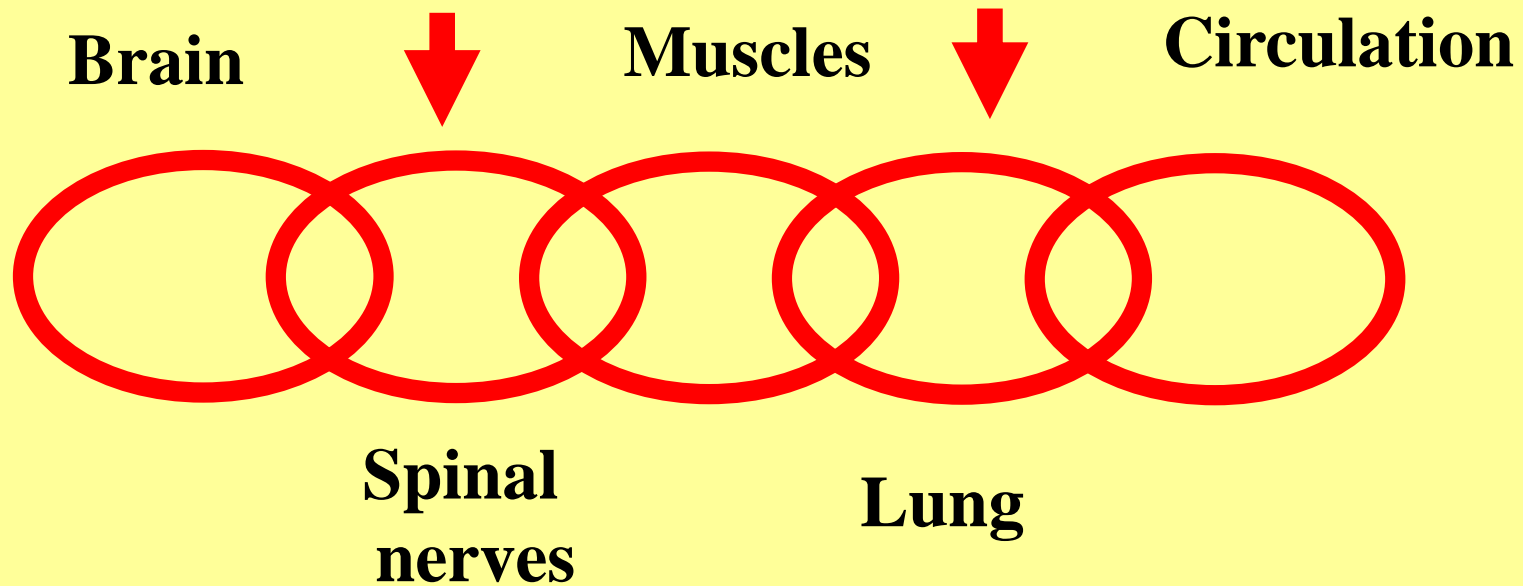
## **DEFINITION**

***ARF is the clinical state which occurs when the respiratory system (ie circulatory and lungs) is not able to meet the metabolic requirements of the organism.***

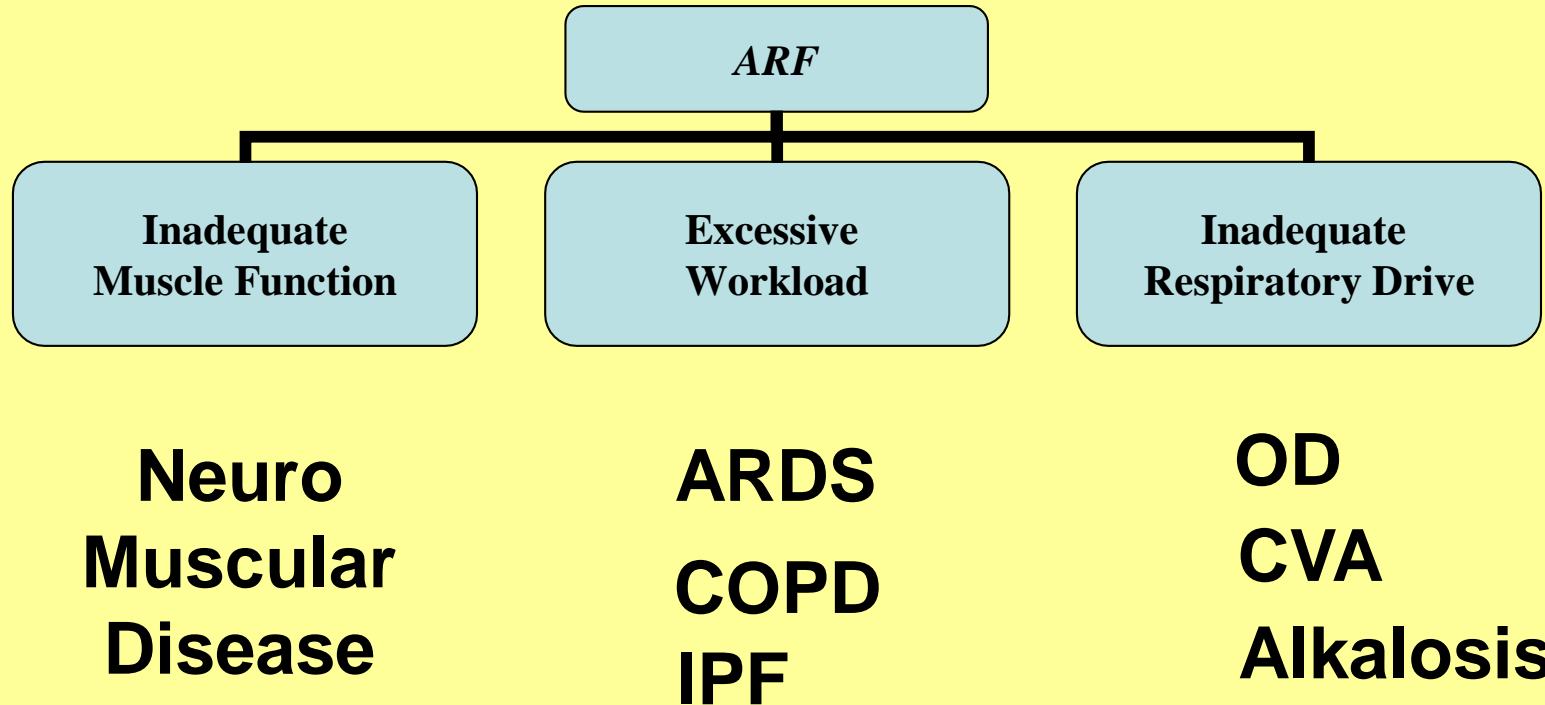
# ***Acute Respiratory Failure***

- ✓ ***Anatomic- Etiologic***
- ✓ ***Physiologic- Etiologic***
- ✓ ***Blood Gas***
- ✓ ***Radiologic***
- ✓ ***Tissue Oxygenation***

# ***Anatomic Etiologic Classification***



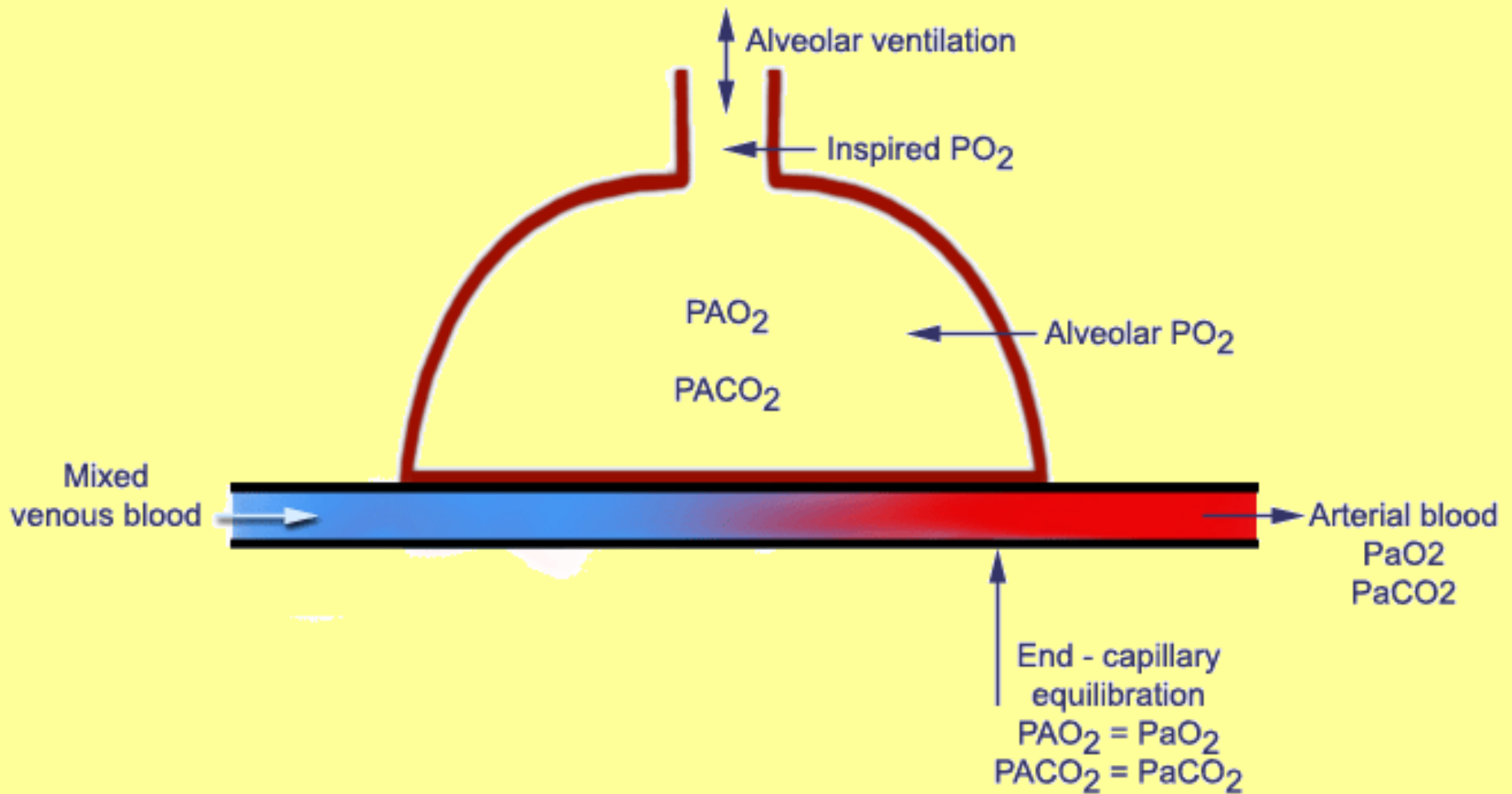
# *Physiologic Etiologic Classification*



# ***Blood Gas Classification***

## **Hypoxemic/Hypercapnic**

- ✓ **Clinically useful**
- ✓ **Can be used to divide patients into distinct ETIOLOGIC and TREATMENT groups**
- ✓ **Readily available**





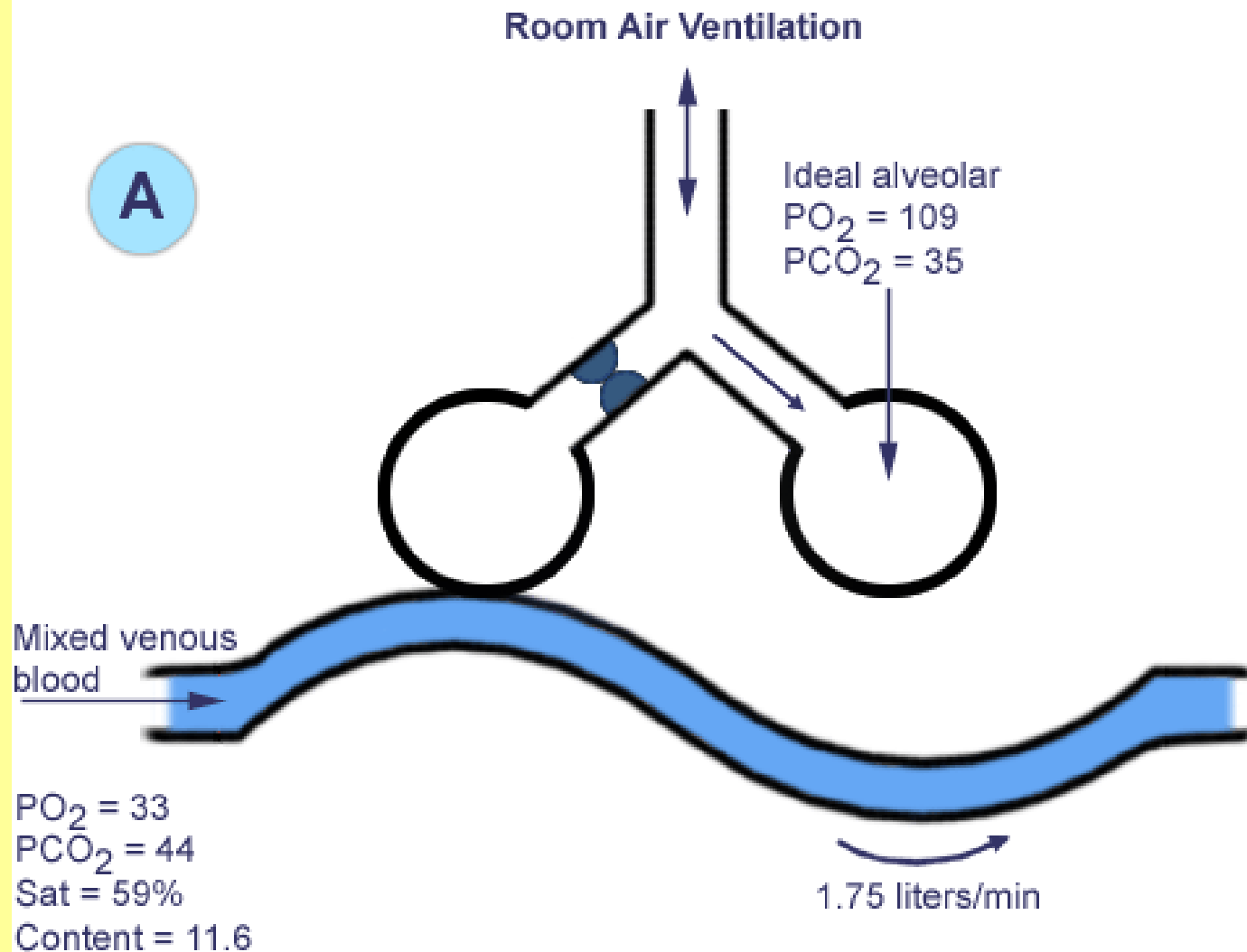
# ***Calculation of the A-a Gradient***

$$\text{PAO}_2 = \text{FIO}_2 (\text{Pb} - 47) - 1.25 \text{ PaCO}_2$$

**PaO<sub>2</sub> = measured**

**A-a gradient should be less than 20 mmHg  
breathing room air OR  
Less than 100 mmHg on 100 % O<sub>2</sub>**

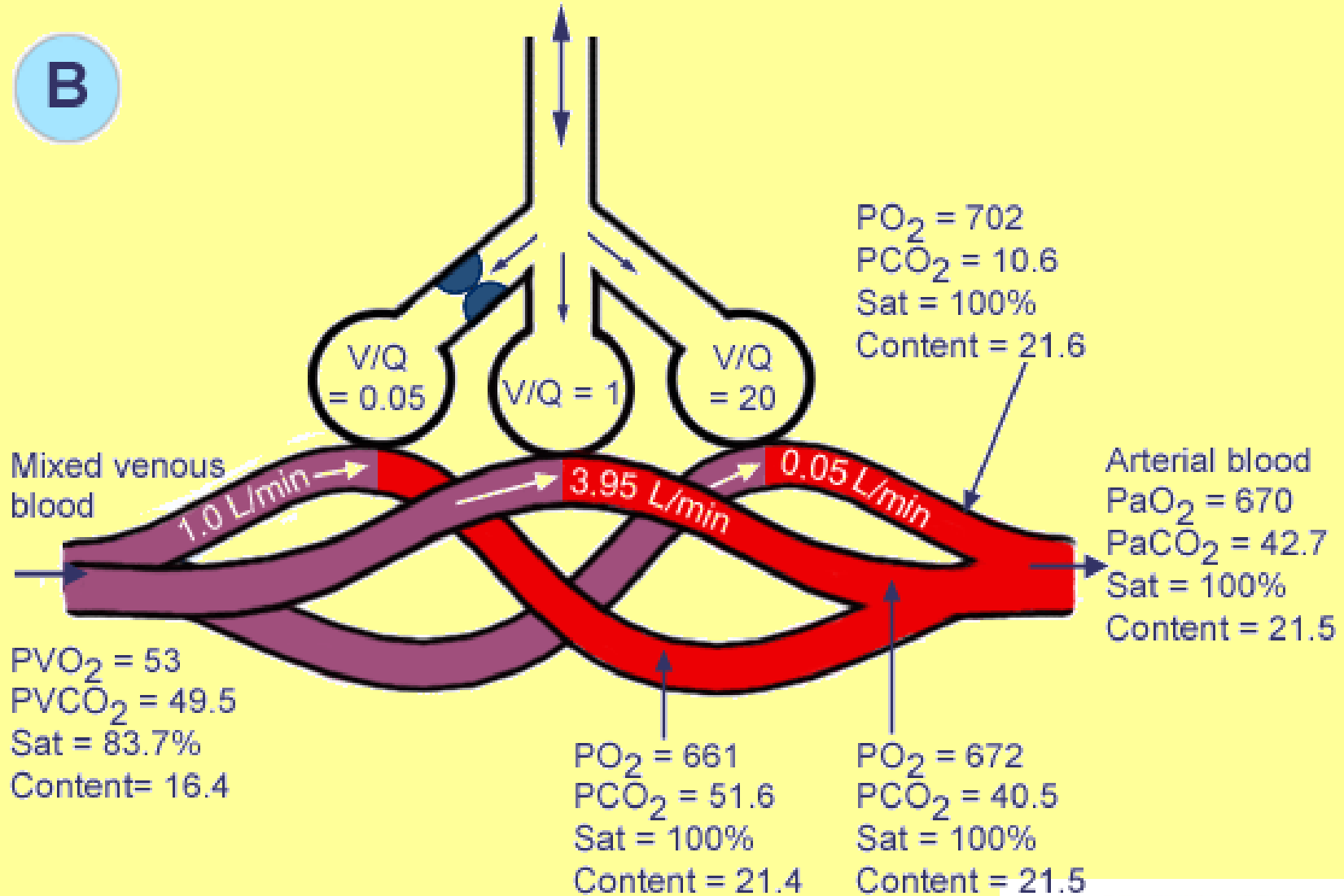
# 100% Oxygen and Pulmonary Shunt



**B**

### 100% Oxygen Ventilation

$$P_{I}O_2 = 713$$



# ***Causes of Hypoxemia***

<b>CAUSE</b>	<b>A-a Gradient</b>	<b>PaCO<sub>2</sub></b>	<b>Response to 100 % Oxygen</b>
<b>Low FIO<sub>2</sub></b>	<b>Normal</b>	<b>Normal</b>	<b>Improved</b>
<b>Hypoventilation</b>	<b>Normal</b>	<b>Increased</b>	<b>Improved</b>
<b>Diffusion Impair</b>	<b>Increased</b>	<b>Normal</b>	<b>Improved</b>
<b>Low V/Q</b>	<b>Increased</b>	<b>Normal</b>	<b>Improved</b>
<b>Shunt</b>	<b>Increased</b>	<b>Normal</b>	<b>NOT Improved</b>
<b>Low PvO<sub>2</sub></b>	<b>Increased</b>	<b>Normal</b>	<b>? Improved</b>

# ***Mechanisms of Hypercapnia***

$$\text{PaCO}_2 = K \frac{\text{VCO}_2}{\text{V}_a}$$

**PaCO<sub>2</sub>** = arterial CO<sub>2</sub> tension

**K** = proportionality constant

**VCO<sub>2</sub>** = CO<sub>2</sub> production

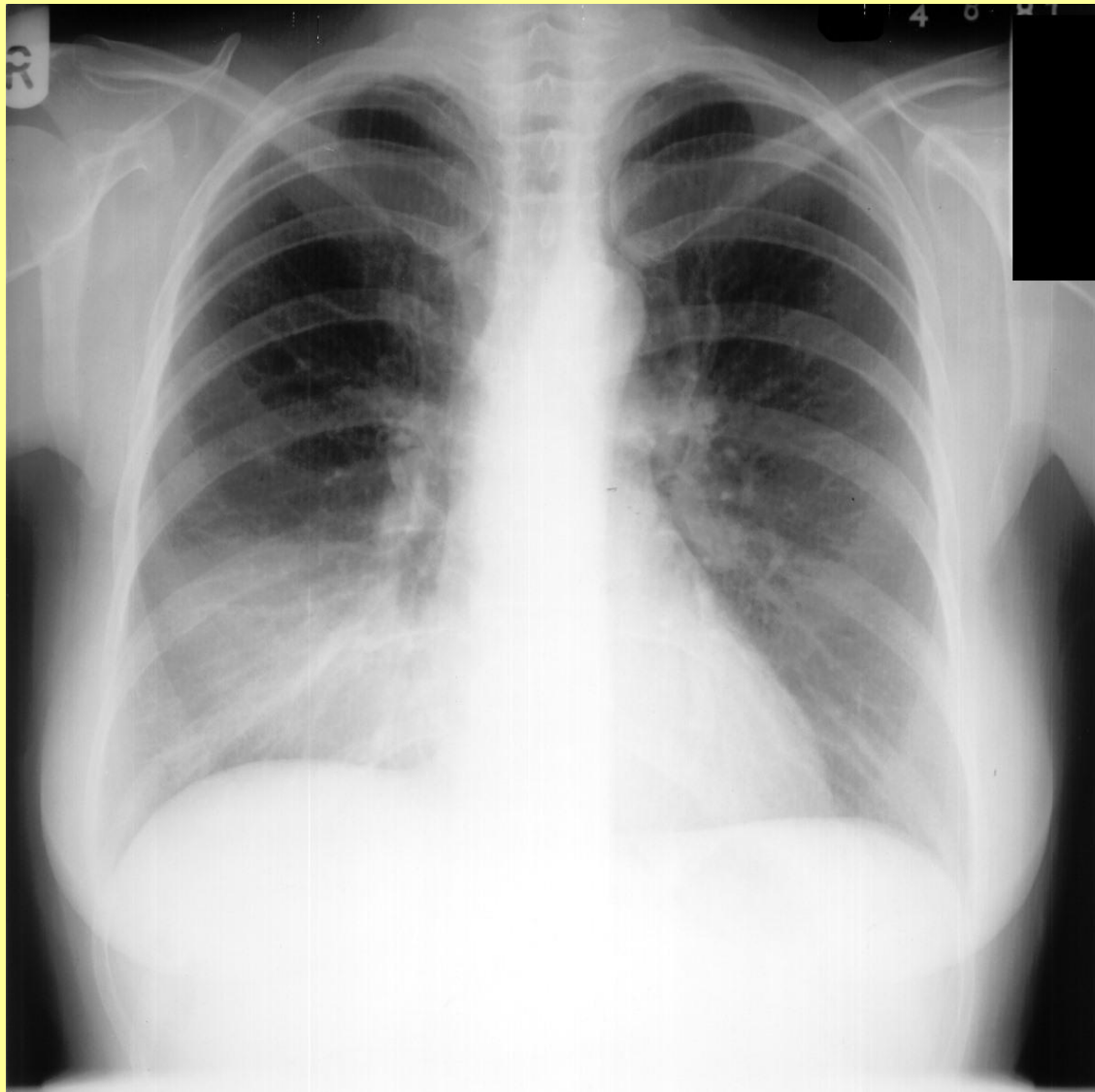
**V<sub>a</sub>** = Alveolar ventilation

# ***Causes of Hypercapnia***

- 1. Alterations in CO<sub>2</sub> production**
- 2. Disturbances in the Gas Exchanger  
(the lungs)**
- 3. Abnormalities in the mechanical system  
(the bellows)**
- 4. Changes in ventilatory control**

# ***Radiographic Classification of ARF***

<b>WHITE LUNG</b>	<b>BLACK LUNG</b>
<b>Pneumonia</b>	<b>Asthma</b>
<b>Pulmonary edema</b>	<b>emphysema</b>
<b>Atelectasis</b>	<b>PE</b>
<b>Interstitial disease</b>	<b>microatelectasis</b>
	<b>R to L Shunt</b>
	<b>Ventilatory failure</b>



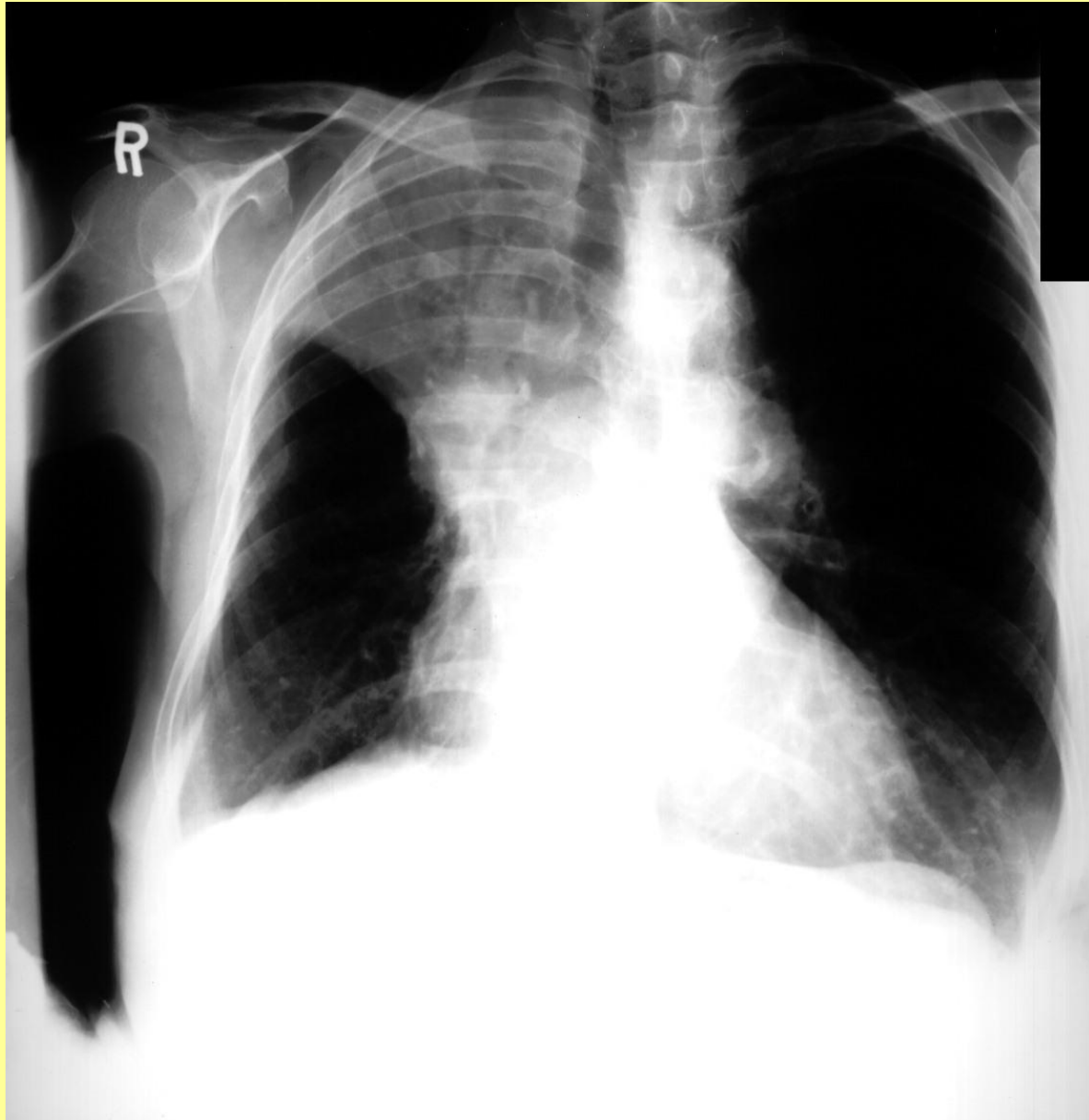


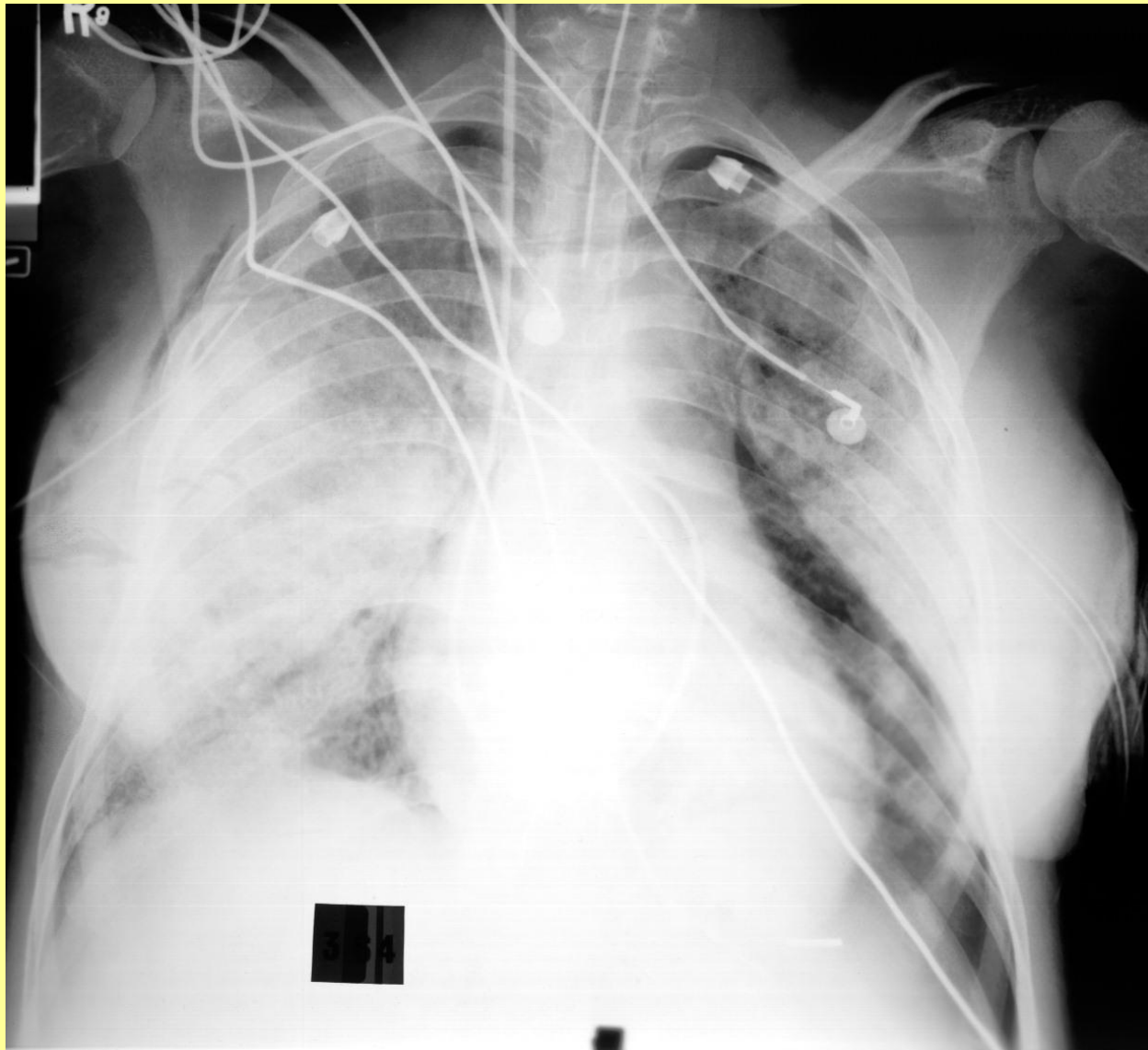


ROWAN UNIVERSITY



School of  
Osteopathic Medicine

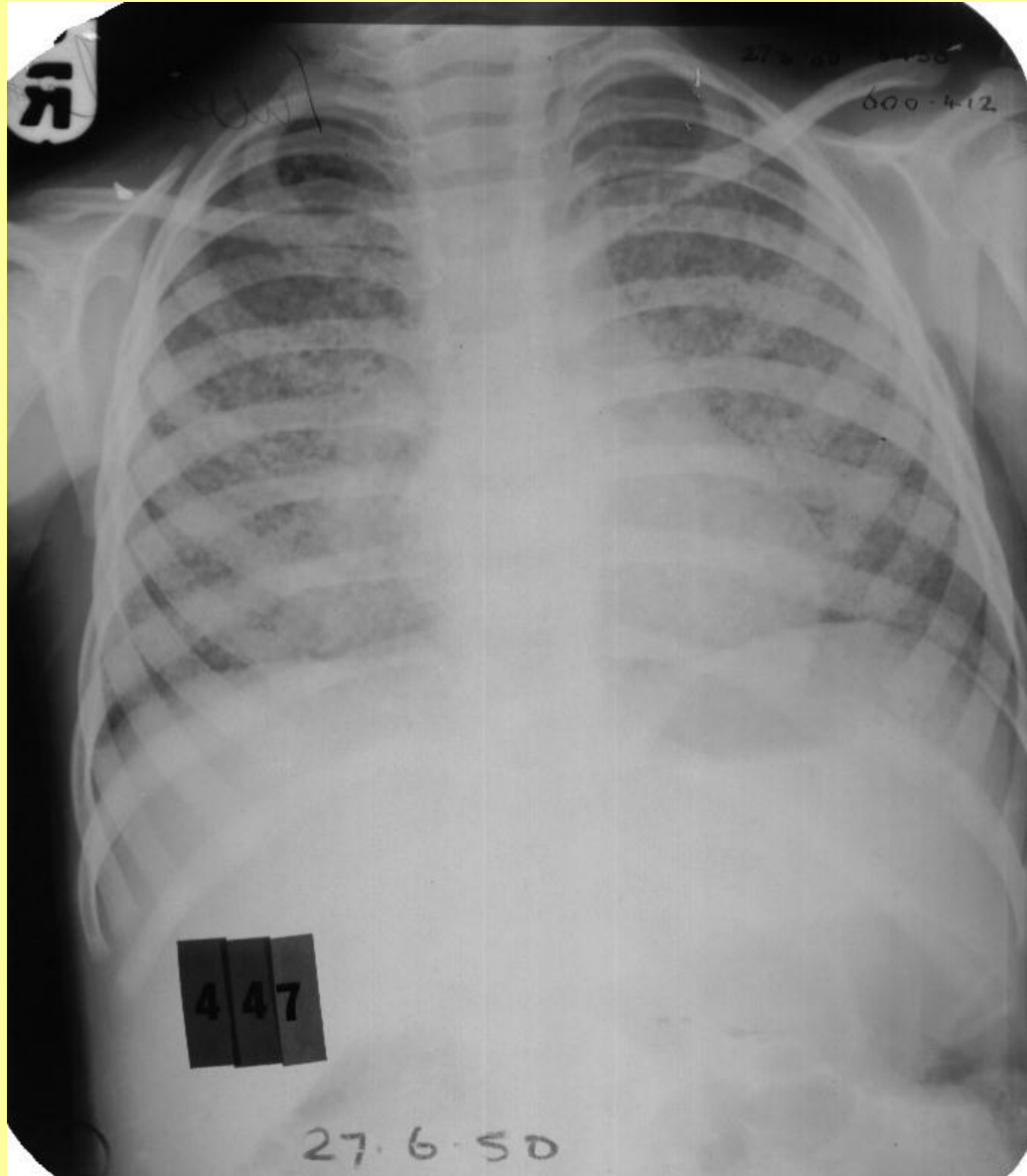




RSITY



School of  
Osteopathic Medicine

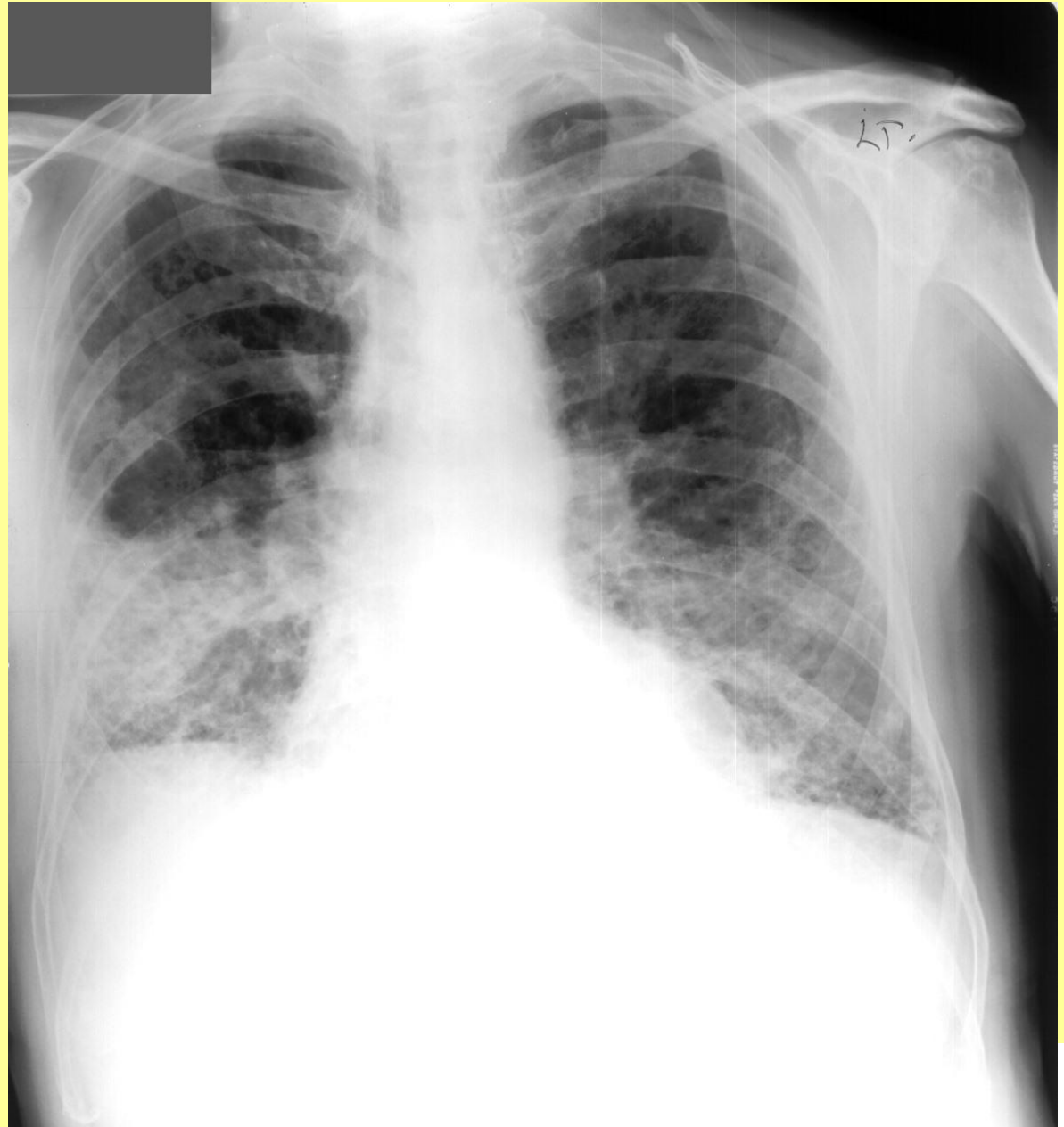


WAN UNIVERSITY



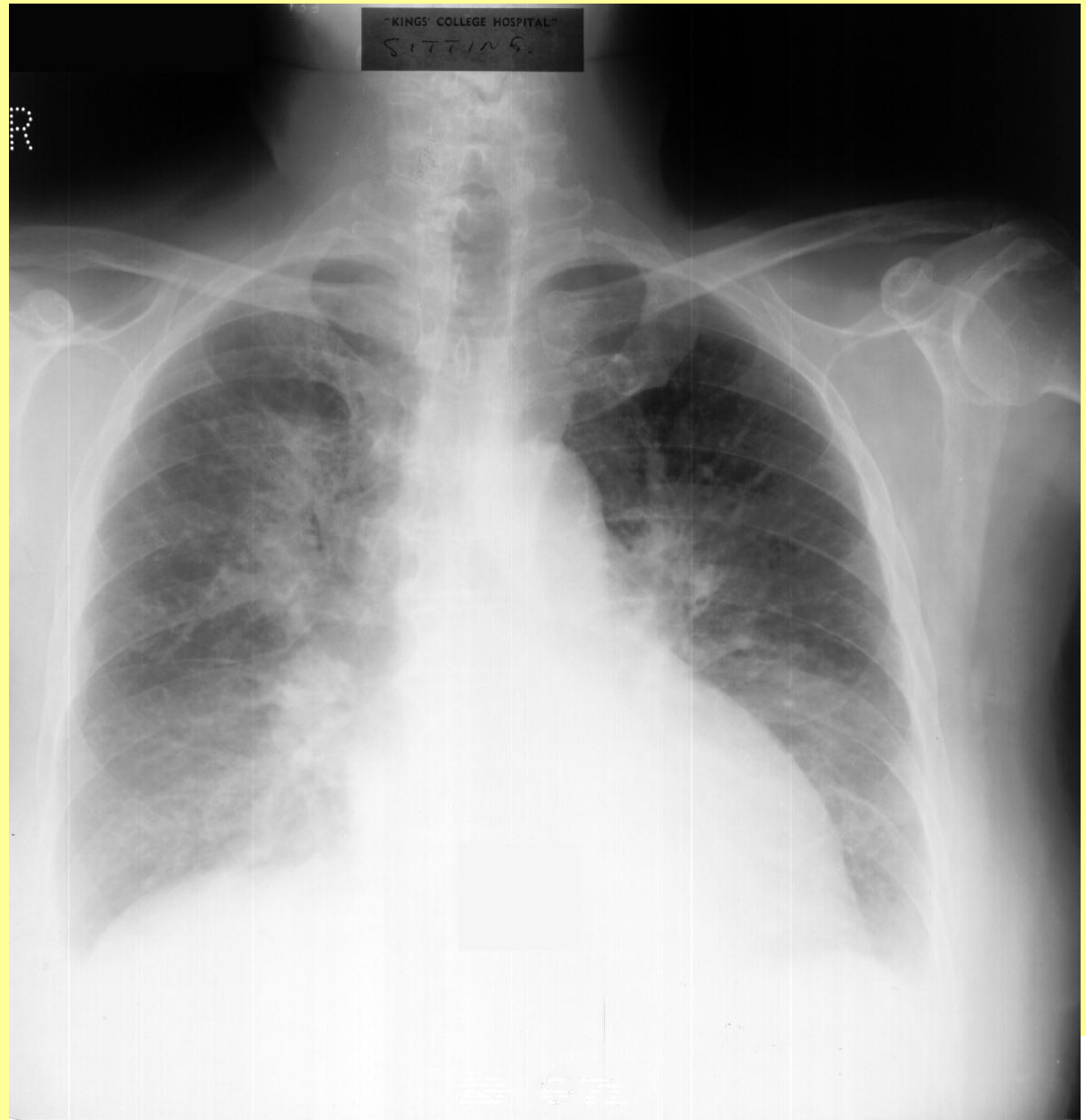
School of  
Osteopathic Medicine

*pulmonary  
fibrosis  
due to  
RA*



**76 yo**  
**Female**

**SOB**  
**Edema**  
**Orthopnea**



*Male*

*SOB*



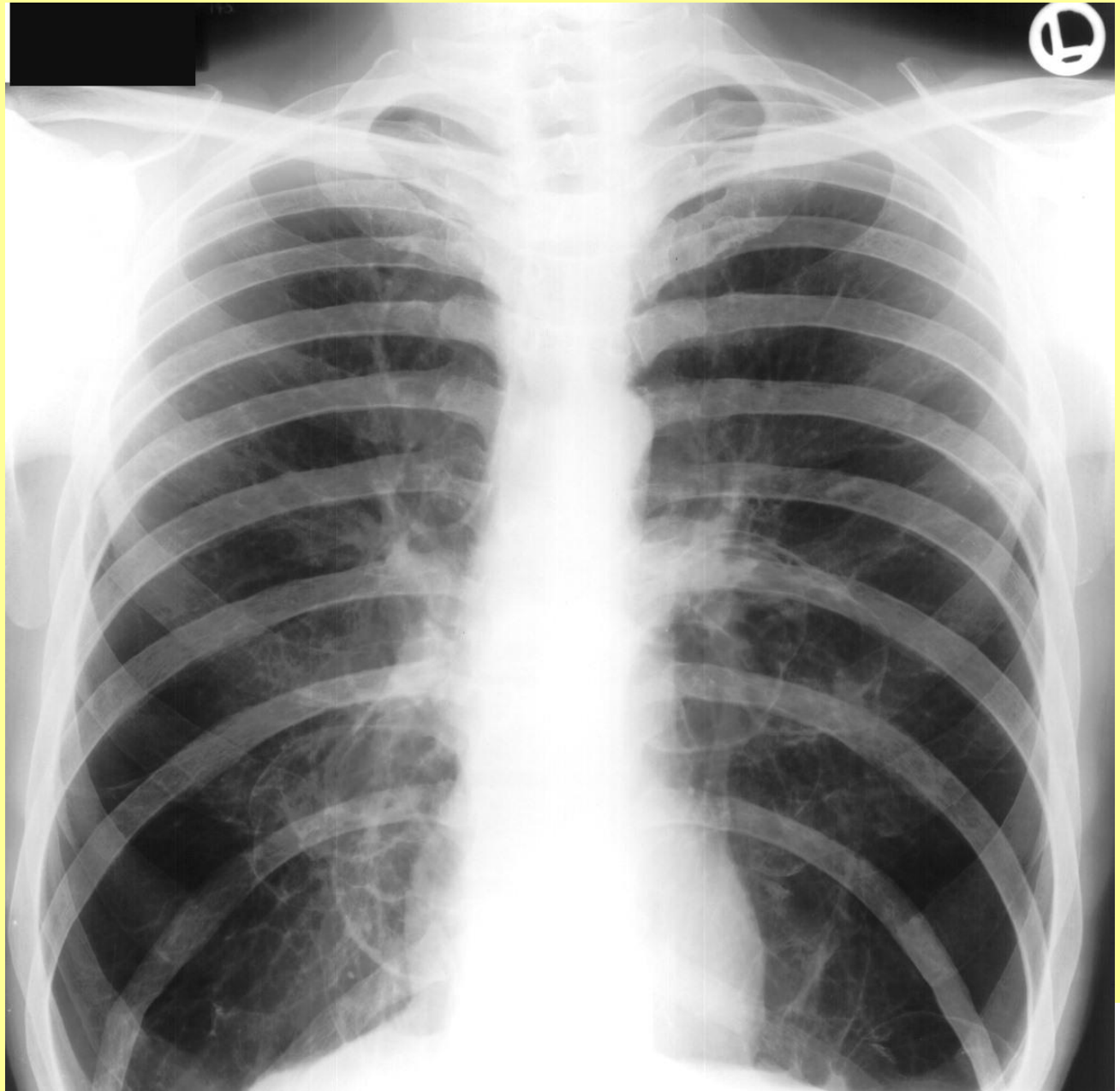
School of  
Osteopathic Medicine





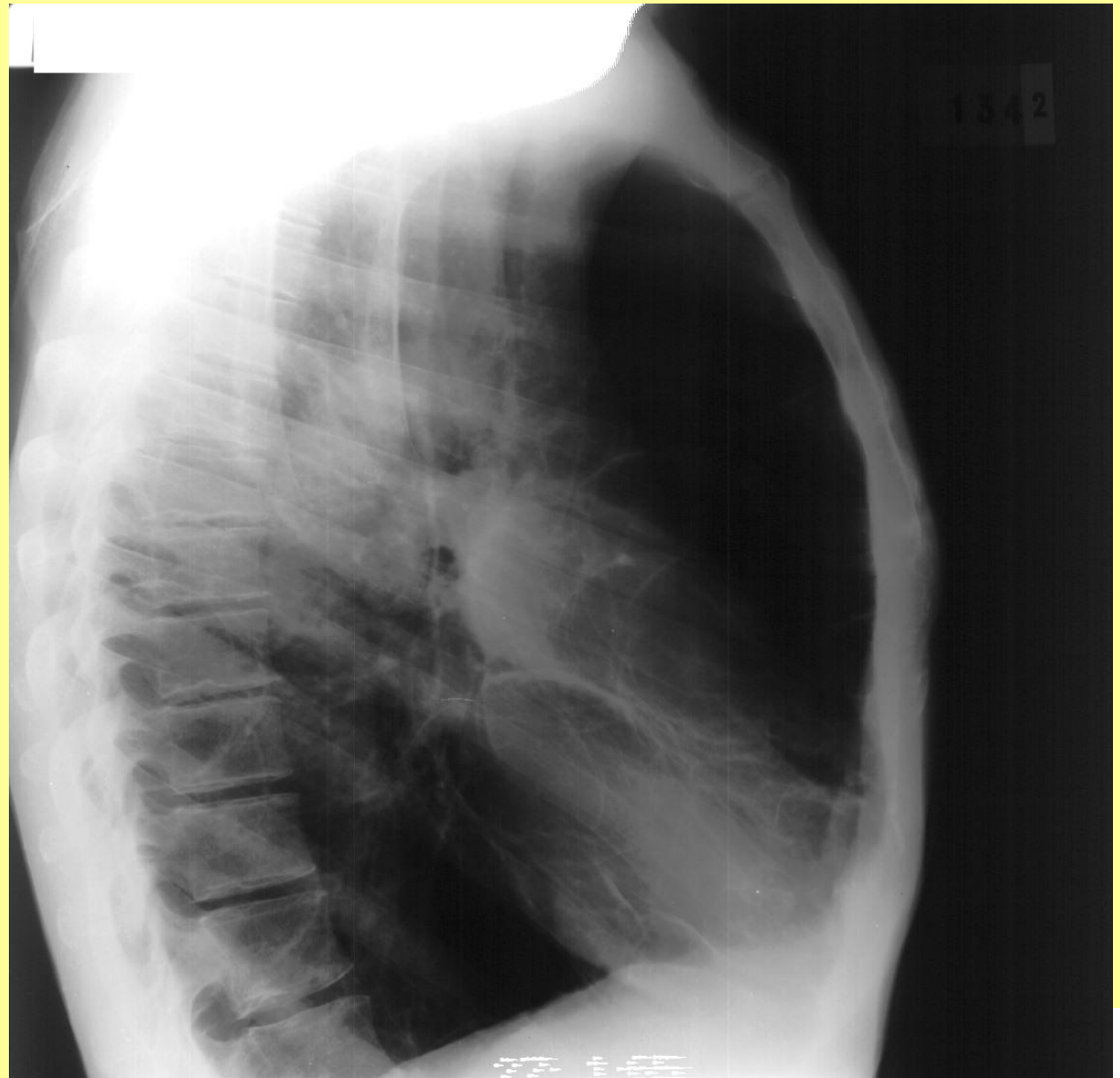
*Male 40 yo*

*Dyspnea*



*Male 40 yo*

*Dyspnea*





<b>CONDITION</b>	<b>DEFINITION</b>	<b>EXAMPLE</b>	<b>ABNORMALITY</b>
<b>Ventilatory Failure</b>	<b>Abnormal CO<sub>2</sub> elimination by lungs</b>	<b>Drug overdose Asthma</b>	<b>PaCO<sub>2</sub> &gt; 50 mmHg</b>
<b>Failure of Arterial Oxygenation</b>	<b>Abnormal O<sub>2</sub> uptake by lung</b>	<b>Pneumonia, ARDS</b>	<b>PaO<sub>2</sub> &lt; 50 mm Hg</b>
<b>Failure of Oxygen Delivery</b>	<b>Abnormal O<sub>2</sub> delivery to the tissues</b>	<b>Cardiogenic shock Anemia, CO poisoning</b>	<b>CvO<sub>2</sub> &lt; 18 cc/dl PvO<sub>2</sub> &lt; 30 mmHg SvO<sub>2</sub> &lt; 60 %</b>
<b>Failure of Oxygen Utilization</b>	<b>Failure of O<sub>2</sub> uptake by tissues</b>	<b>Cyanide poisoning septic shock</b>	<b>CvO<sub>2</sub> &gt; 18 cc/dl PvO<sub>2</sub> &gt; 60 mmHg SvO<sub>2</sub> &gt; 80 %</b>

# ***Objectives of Mechanical Ventilation***

***Tobin MJ. NEJM 1994; 330:1056-61***

- **Improve pulmonary gas exchange**  
**Reverse hypoxemia**  
**Relieve acute respiratory acidosis**
- **Relieve respiratory distress**  
**Decrease the O<sub>2</sub> cost of breathing**  
**Reverse respiratory muscle fatigue**
- **Alter pressure-volume relations**  
**Prevent/reverse atelectasis**  
**Improve compliance**  
**Prevent further lung injury**
- **Permit lung and airway healing**
- **Avoid complications**

# ***Treatment of ARF***

## ***Noninvasive Methods***

### ***CPAP***

Pressure applied during entire respiratory cycle

Does NOT AUGMENT TIDAL VOLUME

Splint open the upper airway

Recruit collapsed alveoli

***BiPAP*** \* Different pressure during Ins and Exp  
I-PAP can AUGMENT tidal Volume  
E-PAP can prevent airway closure  
and recruit collapsed alveoli

USEFUL FOR CHF, COPD, - May prevent  
need for INTUBATION

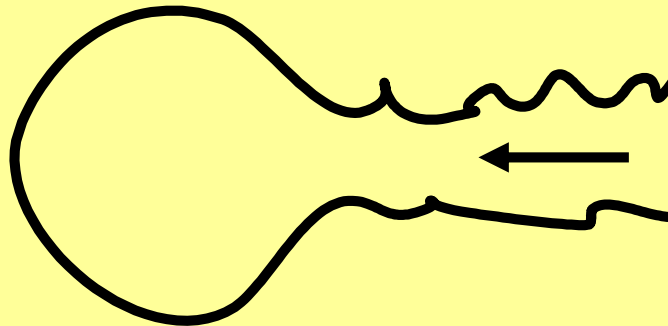
# ***Barotrauma***

- **Until recently it was believed that alveolar rupture was due to excessive proximal airway pressure**
- **If peak airway pressure exceeded 50 cm H<sub>2</sub>O then the patient was considered to be at high risk for alveolar rupture.**

# ***Barotrauma***

- If inspiratory resistance is **HIGH**  
**DISTAL ALVEOLAR PRESSURE** may be  
**LOWER** than  
**PEAK AIRWAY PRESSURE !**

Alveolar Pres =  
20 cm H<sub>2</sub>O

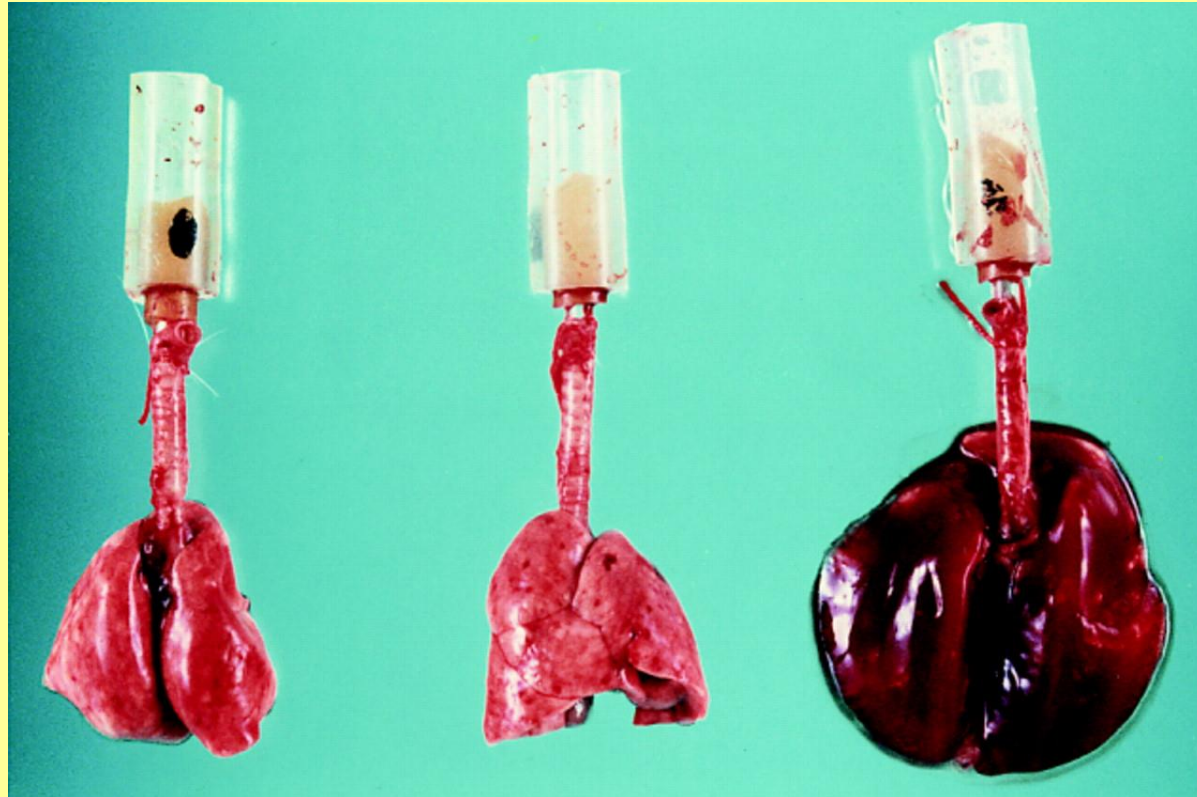


PAP = 50 cm  
H<sub>2</sub>O



# Ventilator-induced Lung Injury

Dreyfuss D, Saumon G. Ventilator induced lung injury: lessons from experimental studies. Am J Respir Crit Care Med 1998;157:294-323.  
Mead J, Takishima



- **Macroscopic aspect of rat lungs after mechanical ventilation at 45 cm H<sub>2</sub>O peak airway pressure. Left: normal lungs; middle: after 5 min of high airway pressure mechanical ventilation. Note the focal zones of atelectasis (in particular at the left lung apex); right: after 20 min, the lungs were markedly enlarged and congestive; edema fluid fills the tracheal cannula.**

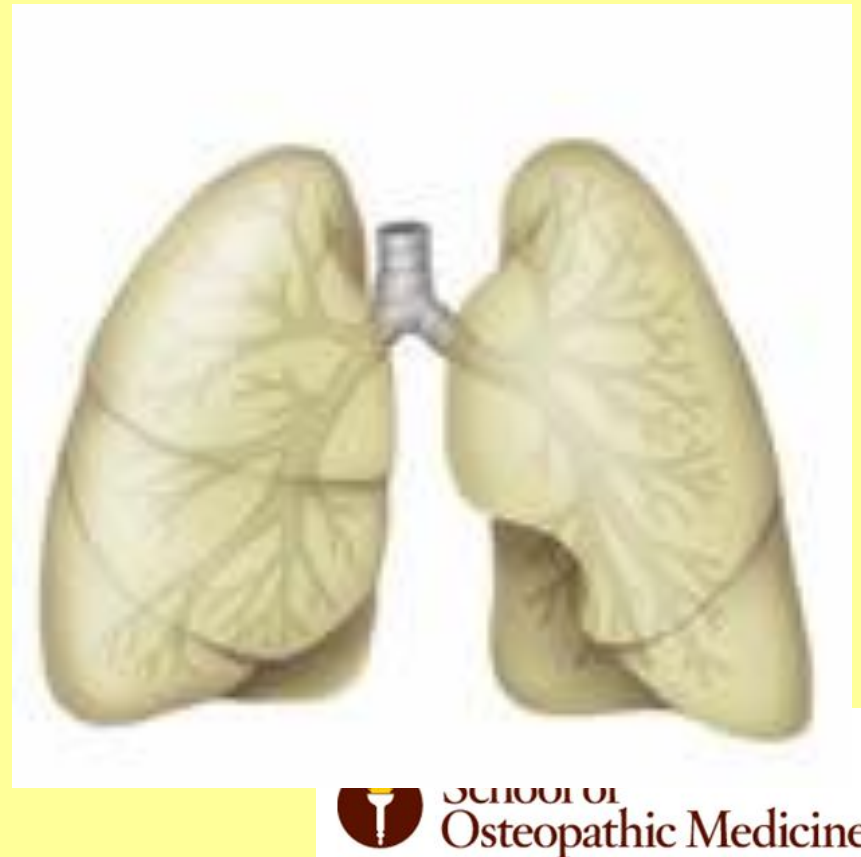


# ***Animal Experiment – Same pressure is applied to both animal lungs***

Banded lungs



Un-Banded lungs



# ***VOLUTRAUMA***

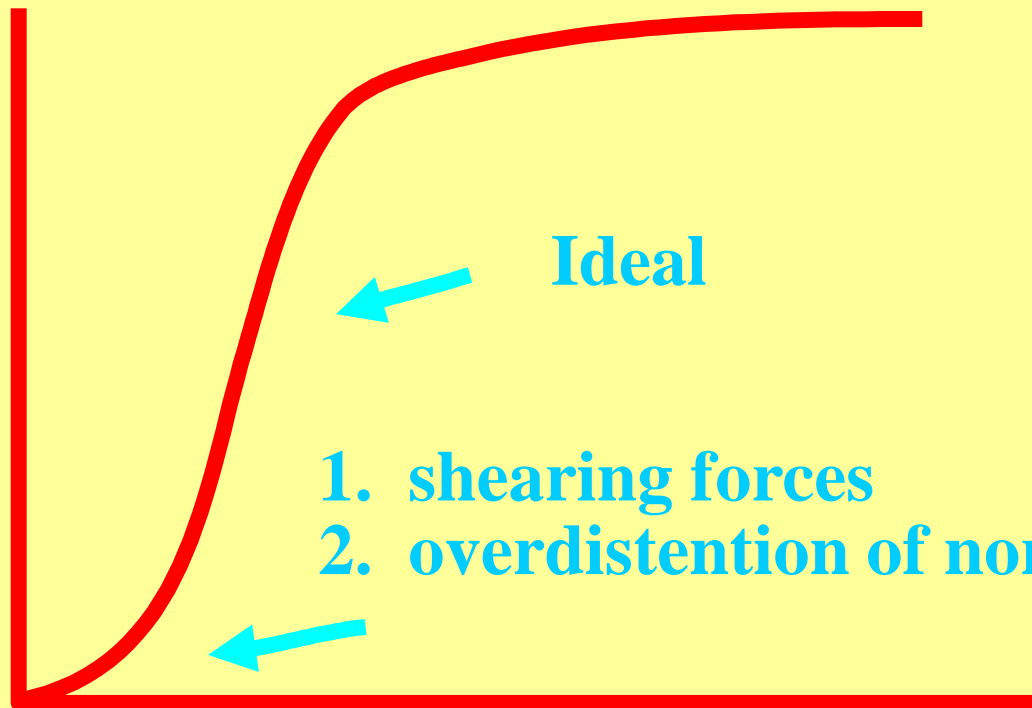
- **Recent studies in animals with normal and diseased lungs suggest that it is alveolar OVERDISTENTION and NOT EXCESSIVE PRESSURE which leads to alveolar rupture.**
- **VOLUME NOT PRESSURE Causes alveolar rupture**

# ***VOLUTRAUMA***

***What volume do we want ?***

***Volume***

***Volutrauma***

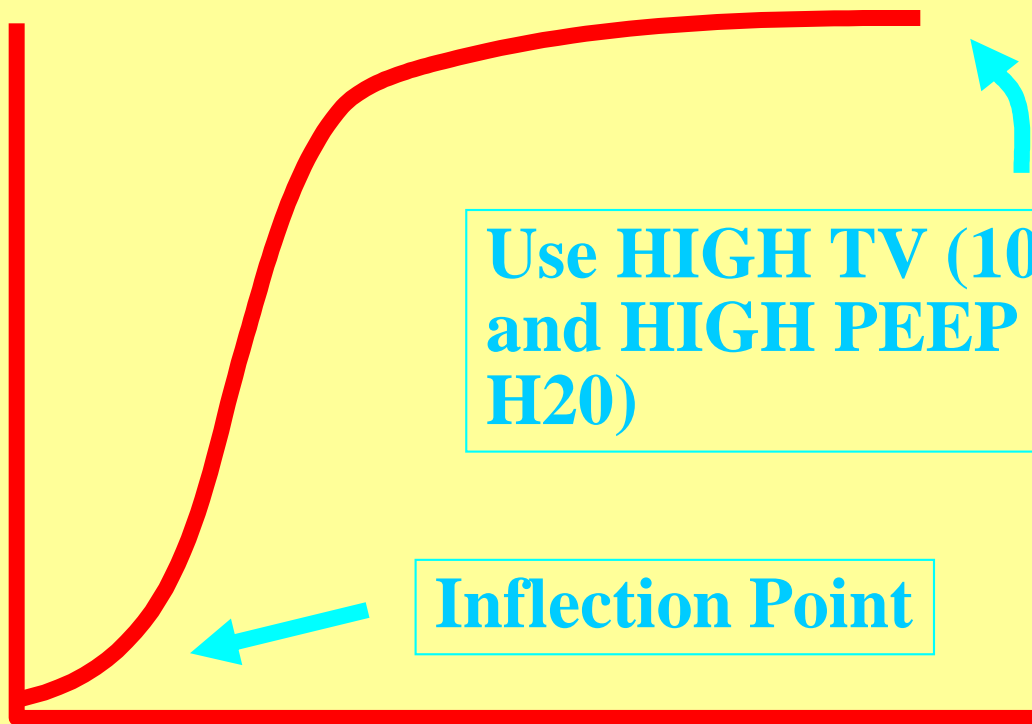


***Pressure***

# ***Classic Approach to MV in Acute Lung Injury***

**Volume**

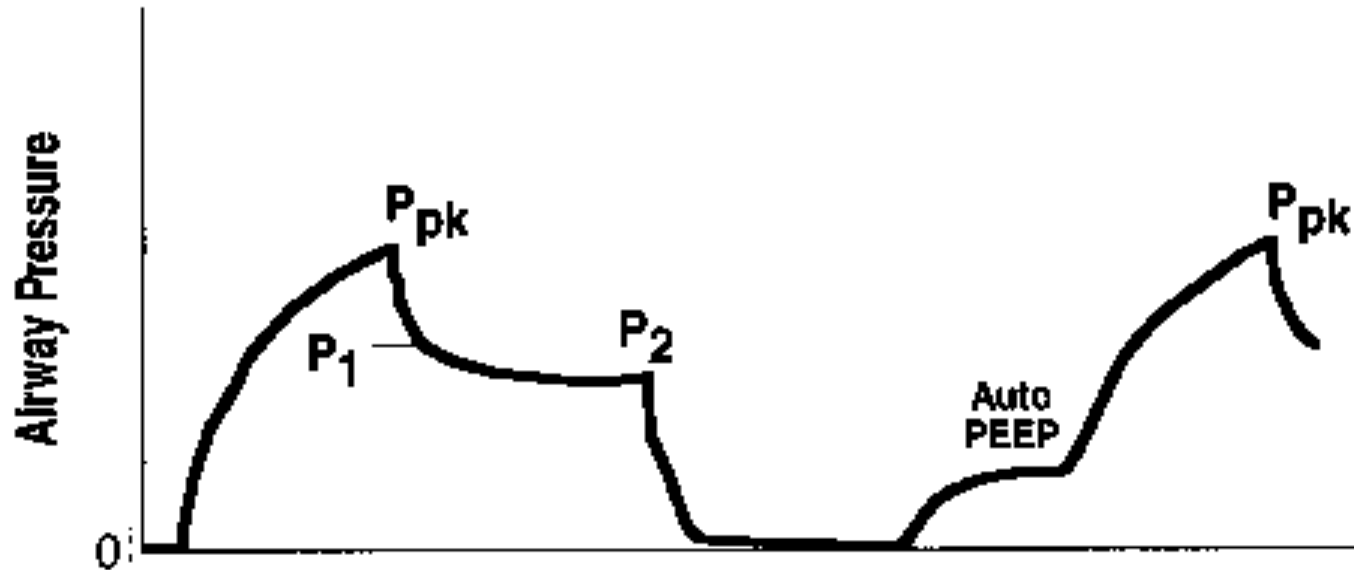
**Volutrauma**



**Inflection Point**

**Pressure**

# *How do we measure Plateau Pressure*



**Figure 1.** Proximal airway pressure recording during an end-inspiratory airway occlusion and during an end-expiratory occlusion.

# ***How should we approach MV in ARDS TODAY ?***

- **TV smaller (5 cc/Kg) ideal body weight**
- **PEEP (above inflection point)**
- **Keep plateau pressure < 30 cm H<sub>2</sub>O**
- **THIS MAY RESULT IN HYPERCAPNIA !**

# ***AutoPEEP***

## ***Definition***

- **AutoPEEP is a pressure gradient between the alveoli and the central airways due to INSUFFICIENT EXPIRATORY TIME.**
- **Unlike applied PEEP which is deliberately set, AUTO-PEEP is inadvertent.**



# ***AutoPEEP***

## ***Incidence***

- **Reported in 47 % of patients in medical ICU's (Wright. Heart and Lung 1990; 19:352-357)**
- **Occurs in 100 % of MV patients with  $V_e$  above 20 L/min (Brown. Respir Care 1986; 31:1069-74)**

# ***AutoPEEP (AP)***

## ***Causes***

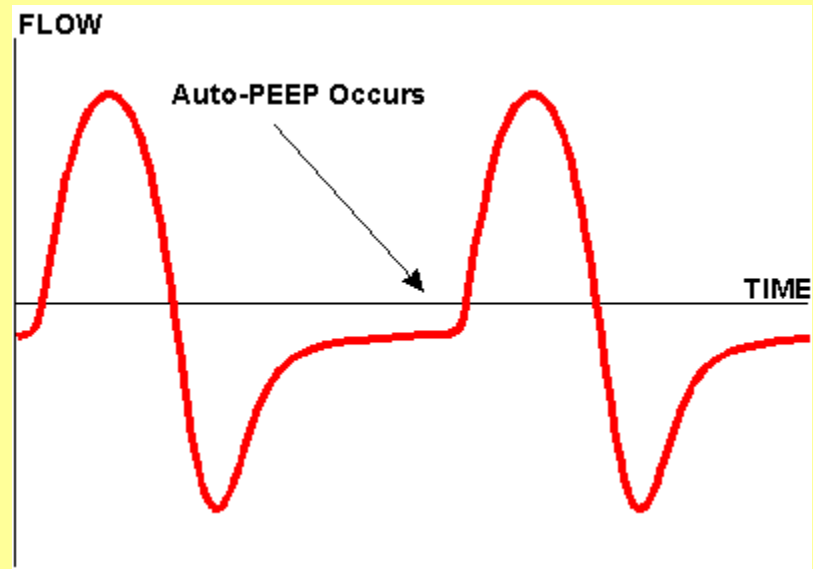
<b><i>Type of AP</i></b>	<b><i>Causes</i></b>
<b><i>AP with Hyperinflation and Airway obstruction</i></b>	<b><i>Dynamic airway closure</i></b>
<b><i>AP with Hyperinflation and NO Airway obstruction</i></b>	<b><i>High Ve vent circuitry, valves or filters which delay exhalation</i></b>
<b><i>AP with NO Hyperinflation and NO Airway obstruction</i></b>	<b><i>Forced exhalation</i></b>

# ***AutoPEEP***

## ***Methods for Detection***

- **Use of Flow Waveform (qualitative)**
- **Esophageal Balloon or inductive waveforms**
- **Block exhalation and allow alveolar and central pressures to equilibrate (Total PEEP)**

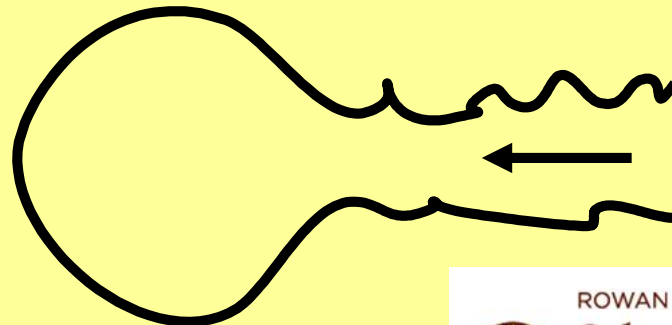
## Auto PEEP detection



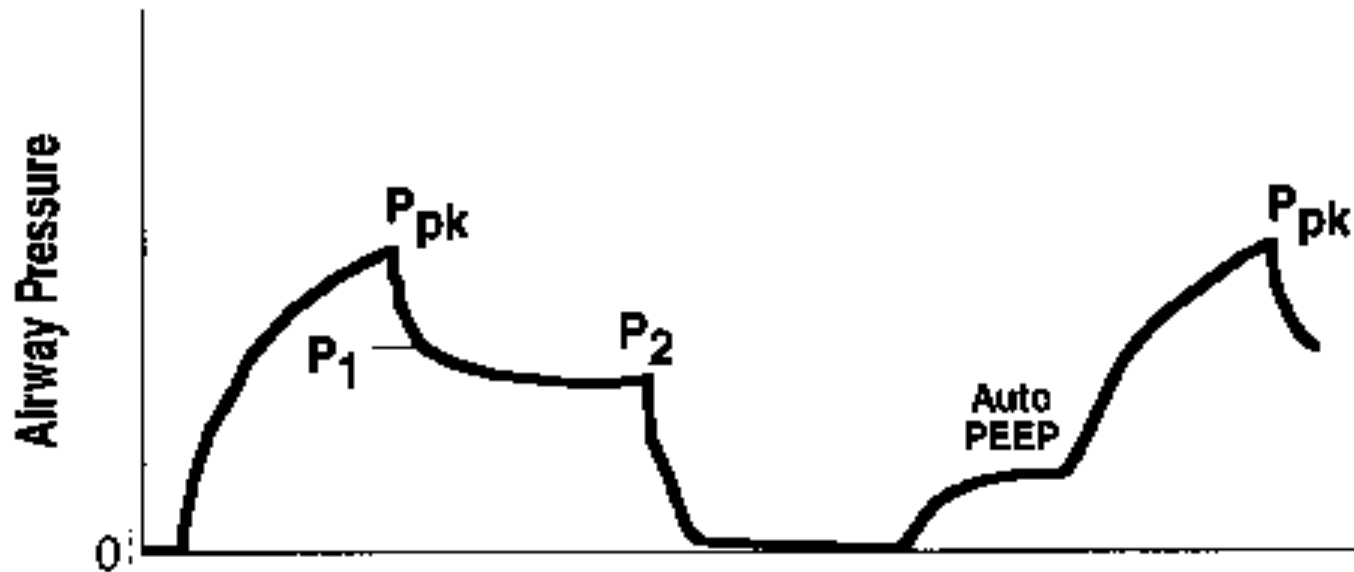
# ***AutoPEEP***

- **AutoPEEP can be measured by blocking the airway at the END OF EXHALATION**

- **This allows the distal alveolar pressure to equilibrate with the Proximal airway pressure**



# How do we measure AutoPEEP



**Figure 1.** Proximal airway pressure recording during an end-inspiratory airway occlusion and during an end-expiratory occlusion.

# ***AutoPEEP***

## ***Adverse Effects***

<i>Effect</i>	<i>Mechanism</i>	<i>Treatment</i>
<i>"Routine"</i>	<p><math>\uparrow</math> PVR, <math>\downarrow</math> CO</p> <p><math>\uparrow</math> Vd/Vt</p>	<p><i>Decrease RR</i></p> <p><i>Increase Vt/Ti</i></p> <p><i>Decrease Vt</i></p>
<i>Triggering</i>	<p><i>Patient has to create</i></p> <p><i>a - pressure greater</i></p> <p><i>than AP to trigger a</i></p> <p><i>MV breath</i></p>	<p><i>Extrinsic PEEP</i></p> <p><i>to = AP</i></p>

# AutoPEEP

## Methods to Reduce

<i>Increase Expiratory Time</i>	<i>Decrease Minute Ventilation</i>	<i>Decrease Expiratory Resistance</i>
<i>Increase peak flow</i>  <i>Square Wave</i>	<i>Decrease Rate</i>  <i>Decrease Tidal Volume</i>	<i>Medications</i>  <i>Remove kinks, secretions, casts</i>  <i>Larger ET tube</i>  <i>Change filters</i>



# ***“New Berlin definition”***

## ***ARDS***

- **Predicted mortality is slightly better than the existing definition (created at the 1994 American-European Consensus Conference/AECC), when applied to a cohort of 4,400 patients from past randomized trials.**

# ***New ARDS Definition***

<b>ARDS Severity</b>	<b>PaO<sub>2</sub>/FiO<sub>2</sub>*</b>	<b>Mortality**</b>
<b>Mild</b>	<b>200 – 300</b>	<b>27%</b>
<b>Moderate</b>	<b>100 – 200</b>	<b>32%</b>
<b>Severe</b>	<b>&lt; 100</b>	<b>45%</b>

**\*on PEEP 5+; \*\*observed in cohort**

# ***“Berlin definition”***

- **Onset of ARDS (diagnosis) must be acute, as defined as within 7 days**
- **Bilateral opacities may be detected on CT or chest X-ray**
- **“not fully explained by cardiac failure or fluid overload”**
- **JAMA online May 21, 2012.**

# ***Pulmonary Function Tests***

- 1. Spirometry**
- 2. Determination of Reversibility**
- 3. Lung Volume**
- 4. Bronchial Hyperreactivity  
(Methacholine Challenge)**
- 5. Diffusing Capacity for CO**
- 6. Exercise**

# ***Pulmonary Function Tests***

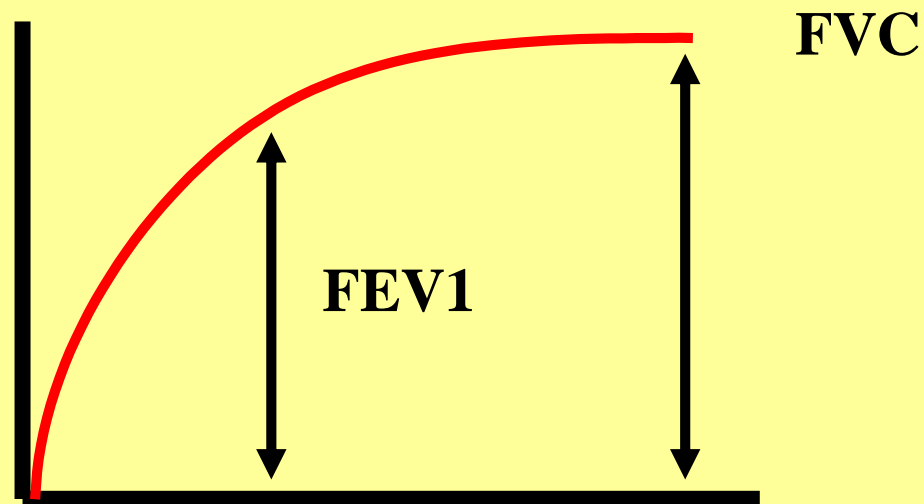
## ***WHY ?***

- 1. To determine if lung disease is present**
- 2. To screen for subclinical disease**
- 3. To determine severity of known disease**
- 4. To determine reversibility**
- 5. To follow disease course**
- 6. Pre-operative evaluation**

# Volume/Time Curves

## Definitions

*Volume*

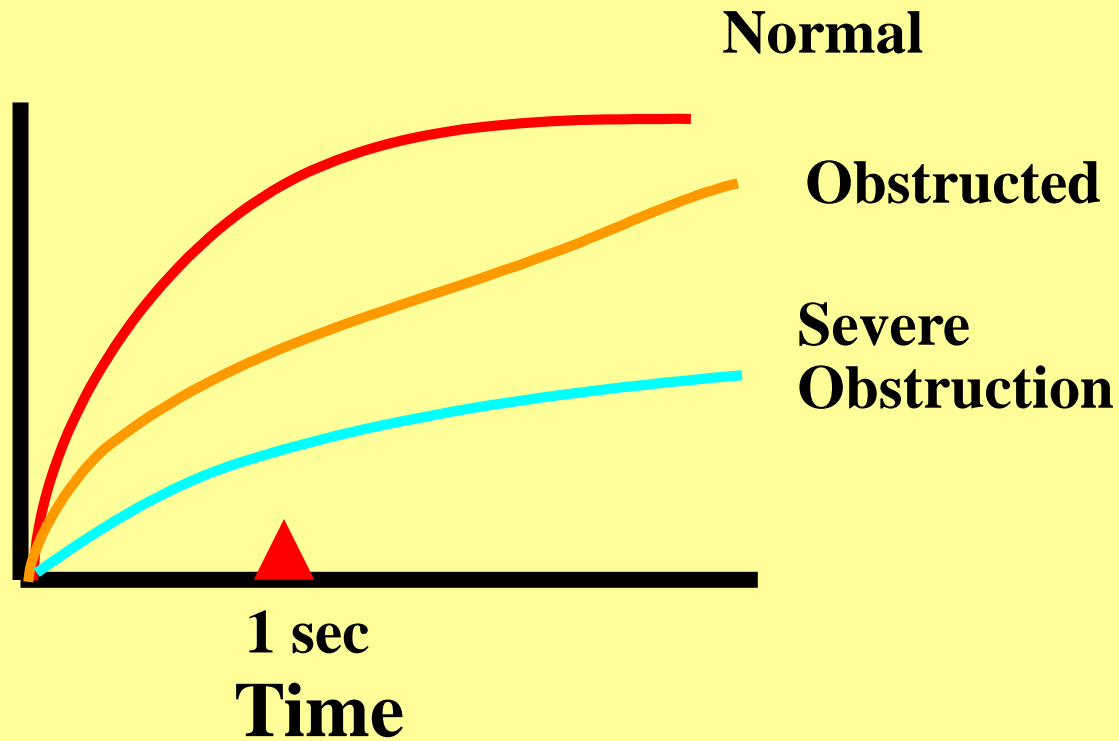


*Time*

# ***Volume/Time Curves***

## ***Obstruction***

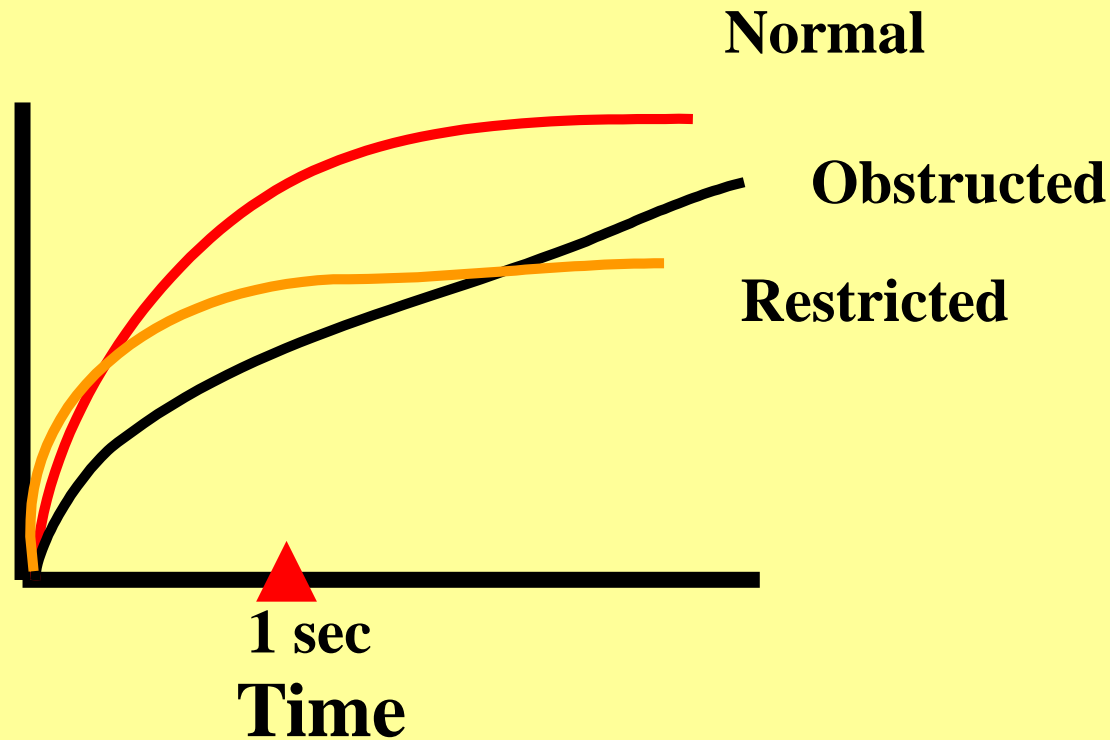
**Volume**



# ***Volume/Time Curves***

## ***Obstruction versus Restriction***

**Volume**



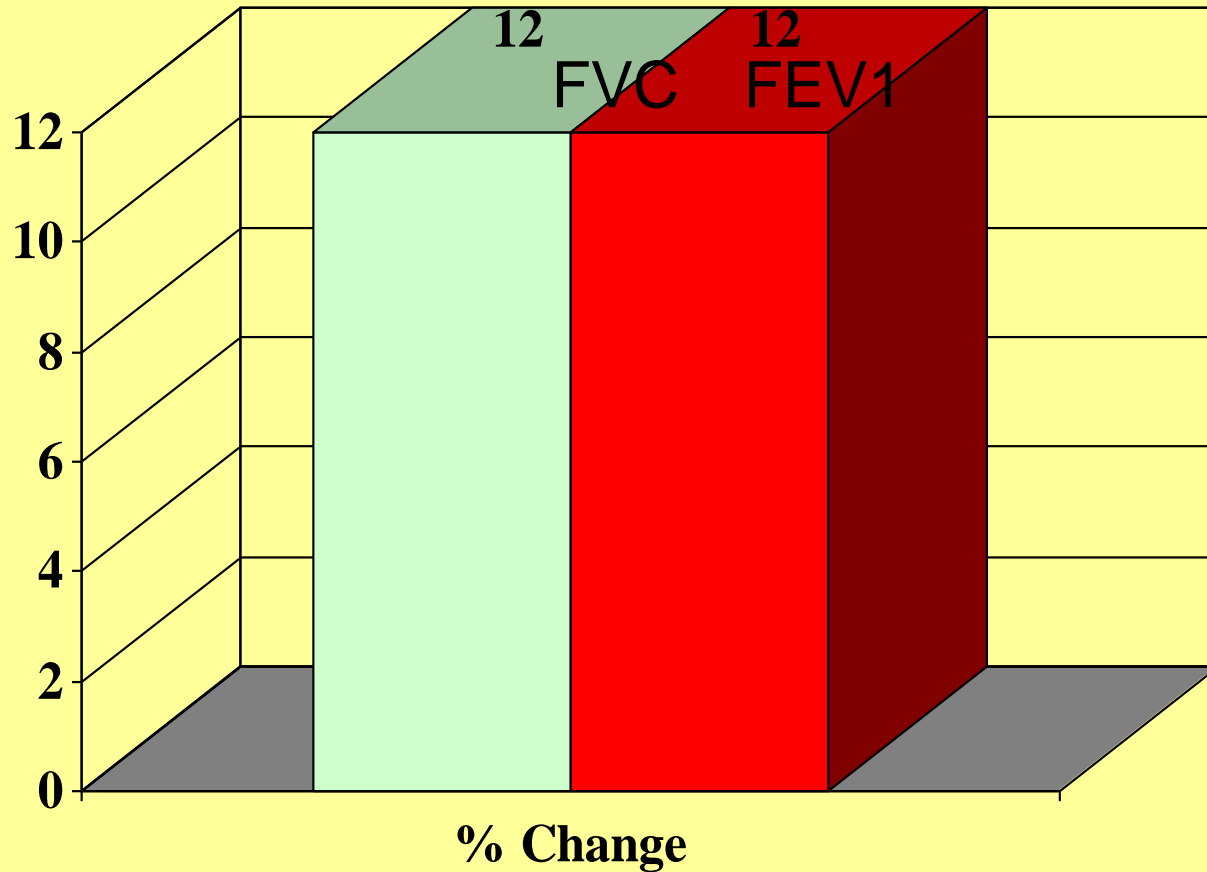
**FEV1 can be reduced by Obst or Rest disease**



# Differentiation of Obstruction from Restriction

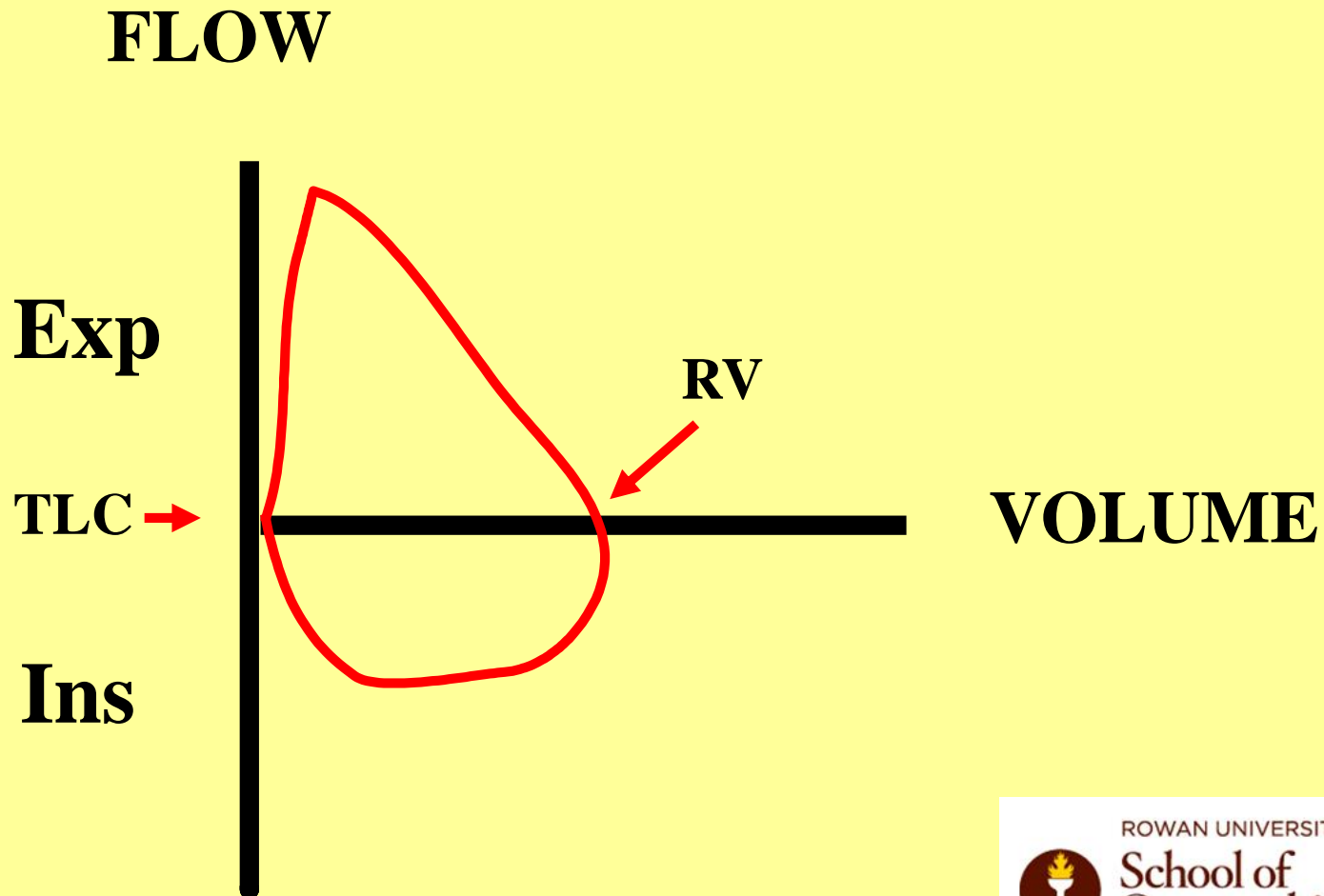
<i>VARIABLE</i>	<i>RESTRICTION</i>	<i>OBSTRUCTION</i>
<i>FVC</i>	<i>Reduced</i>	<i>N or Reduced</i>
<i>FEV1</i>	<i>Reduced</i>	<i>Reduced</i>
<i>FEV1/FVC</i>	<i>Normal</i>	<i>Reduced</i>
<i>TLC/RV/FRC</i>	<i>Reduced</i>	<i>N or Increased</i>

# *Response to Bronchodilator*

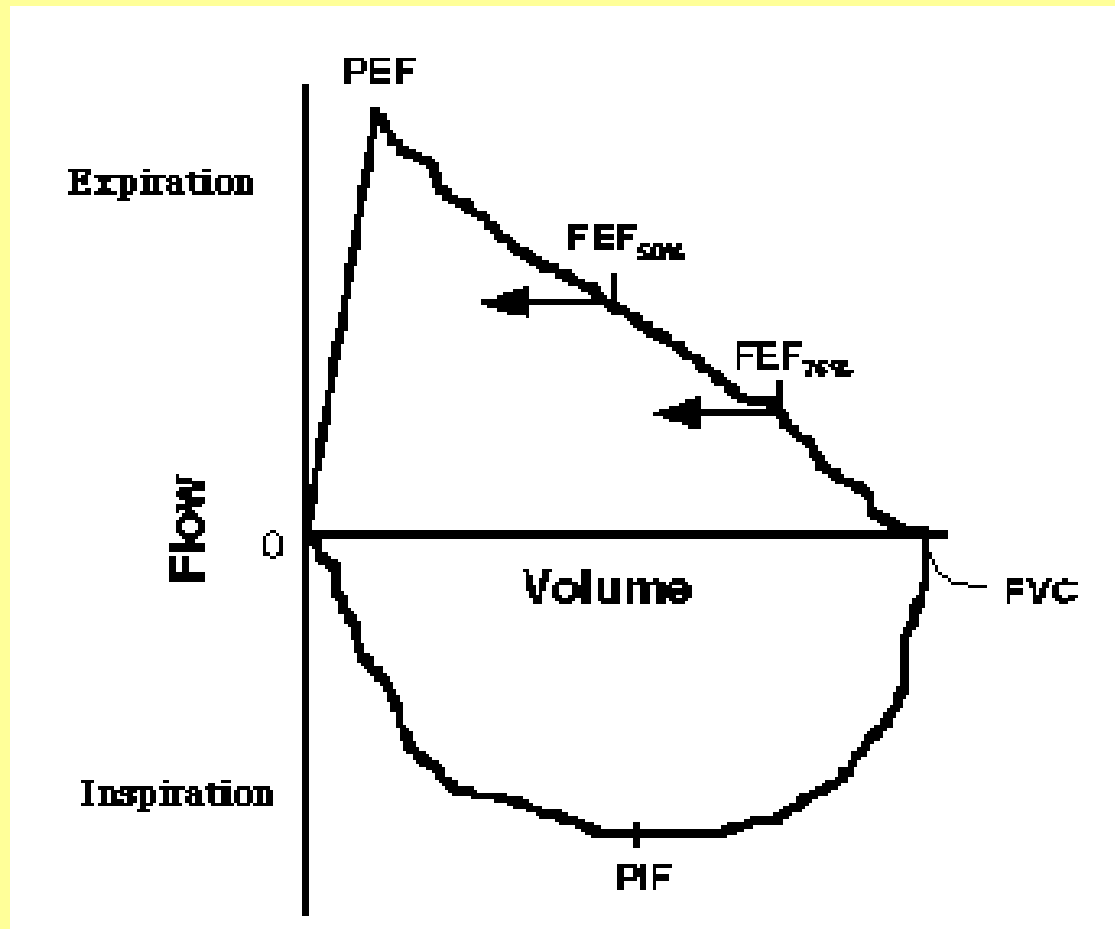


# Flow-Volume Curve

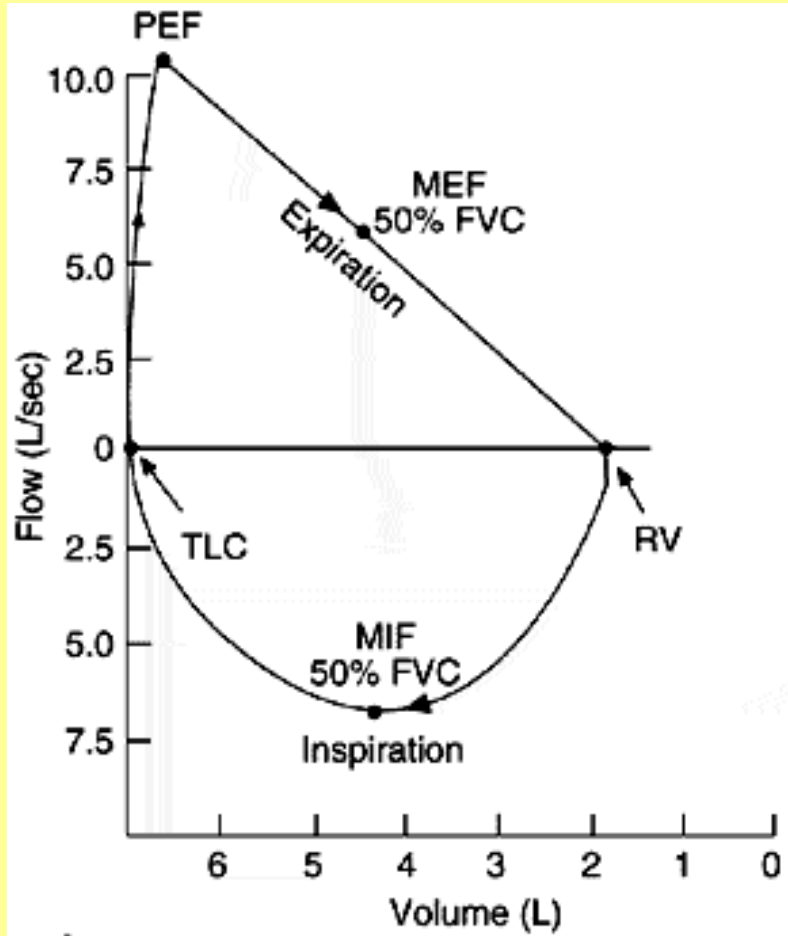
## Definitions



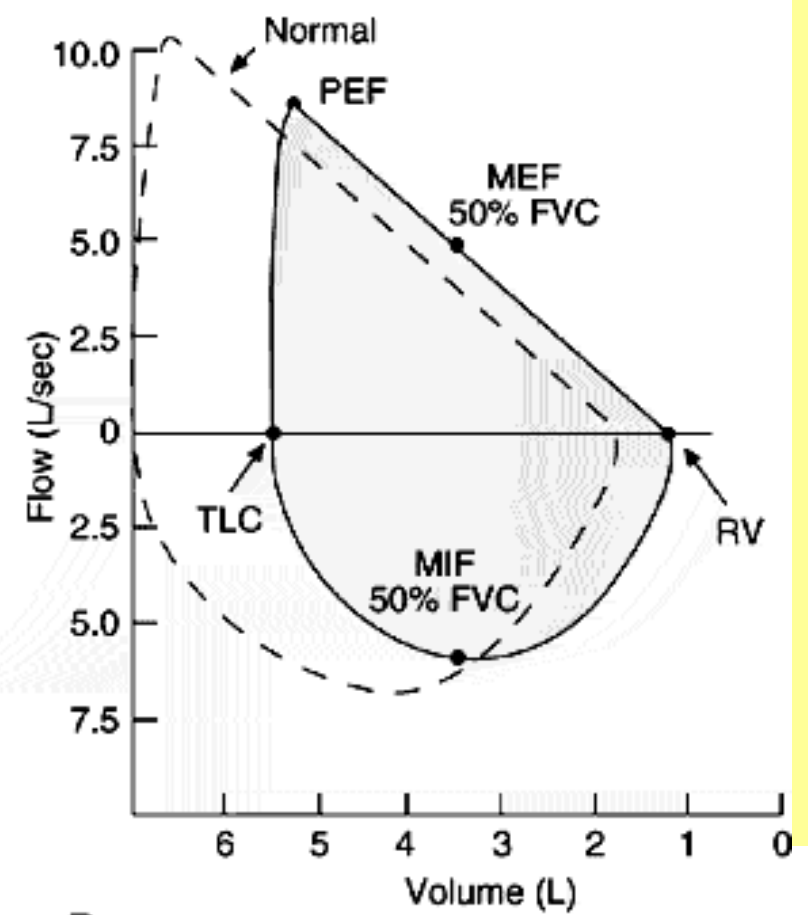
# Flow-Volume Loop



# Normal and Restrictive FVL

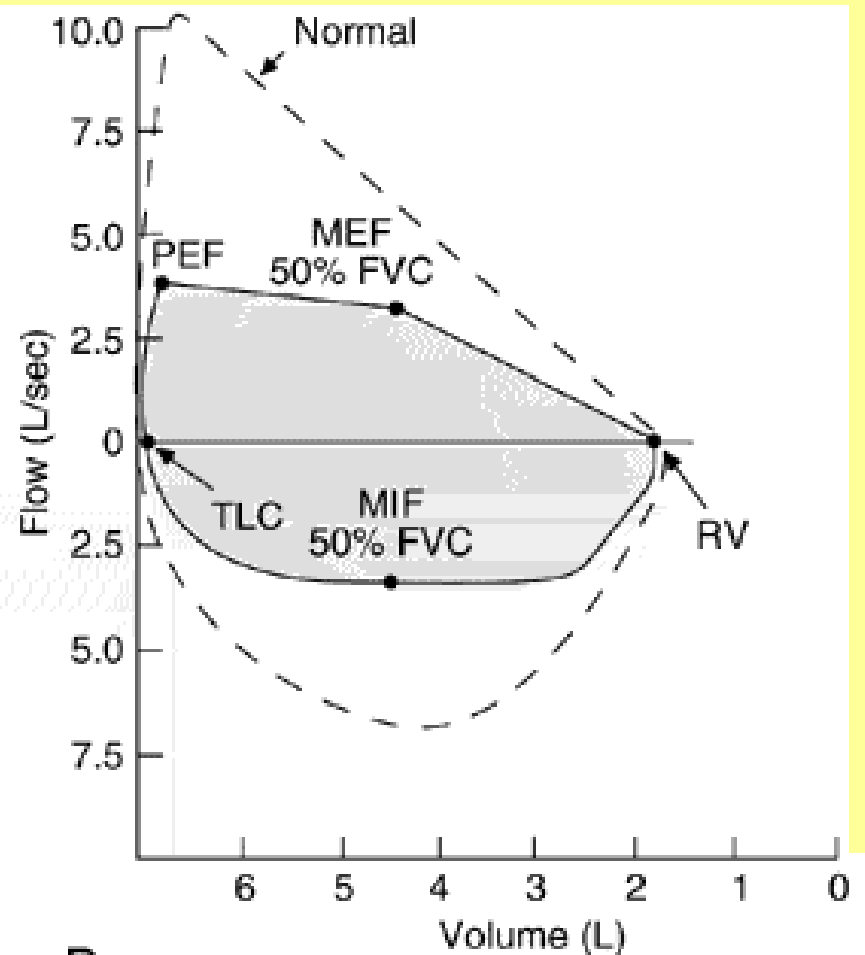
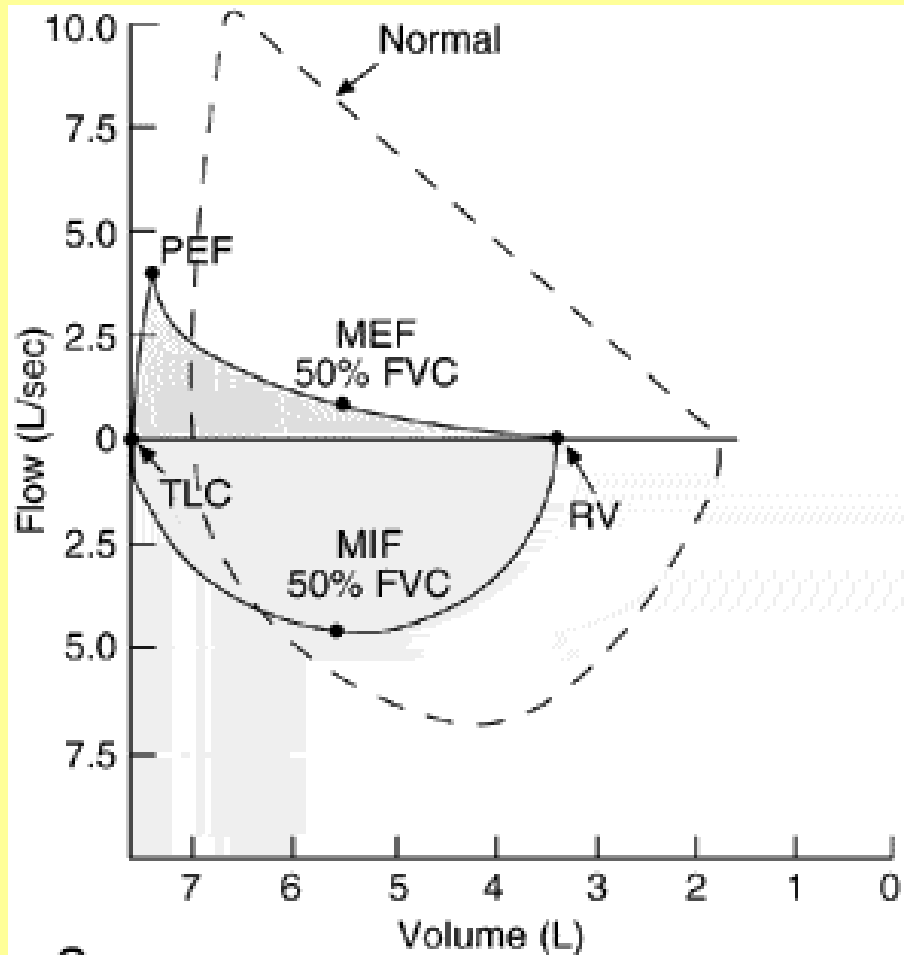


A



B

# Obstructive FVL

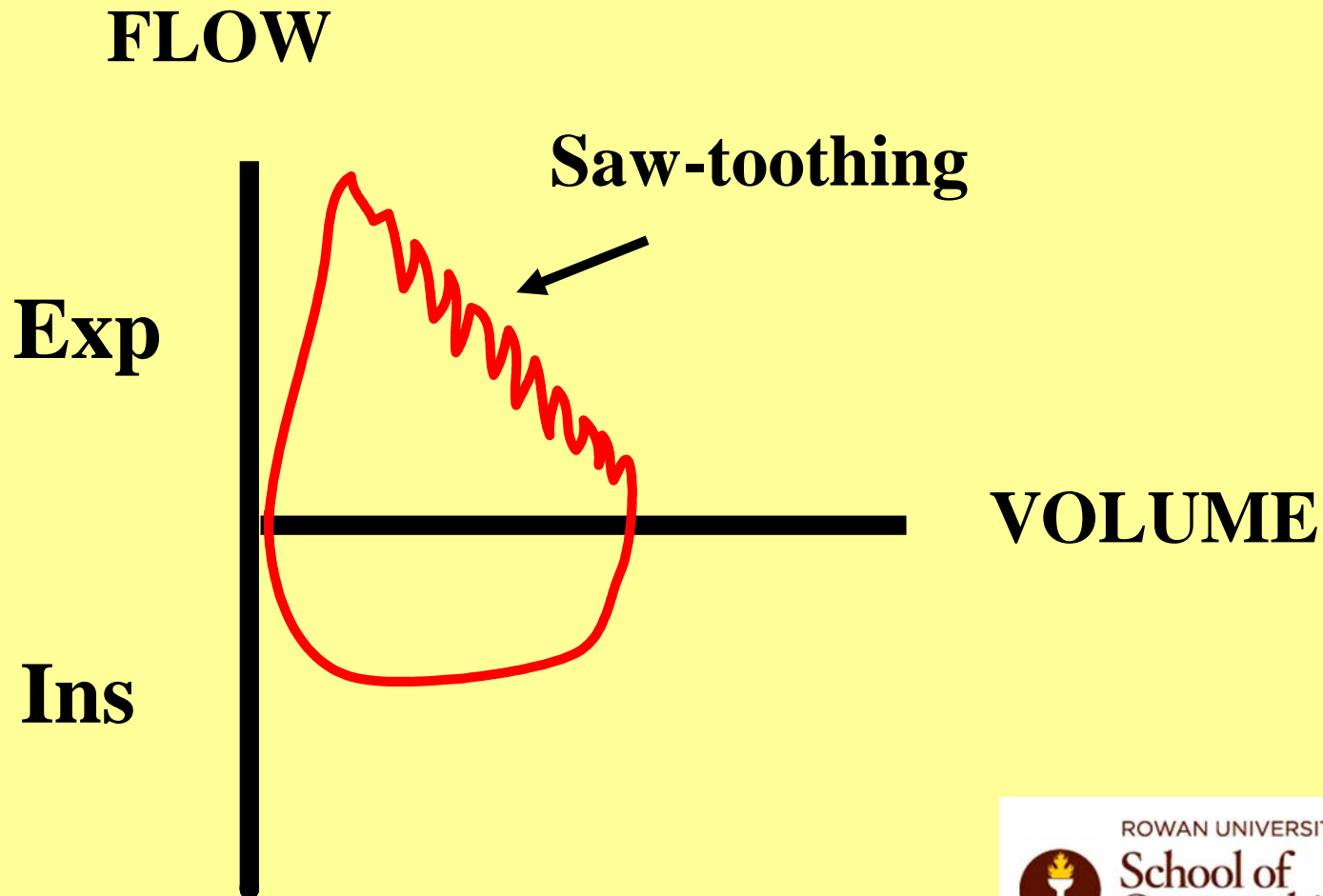


C

D

# ***Flow-Volume Curve***

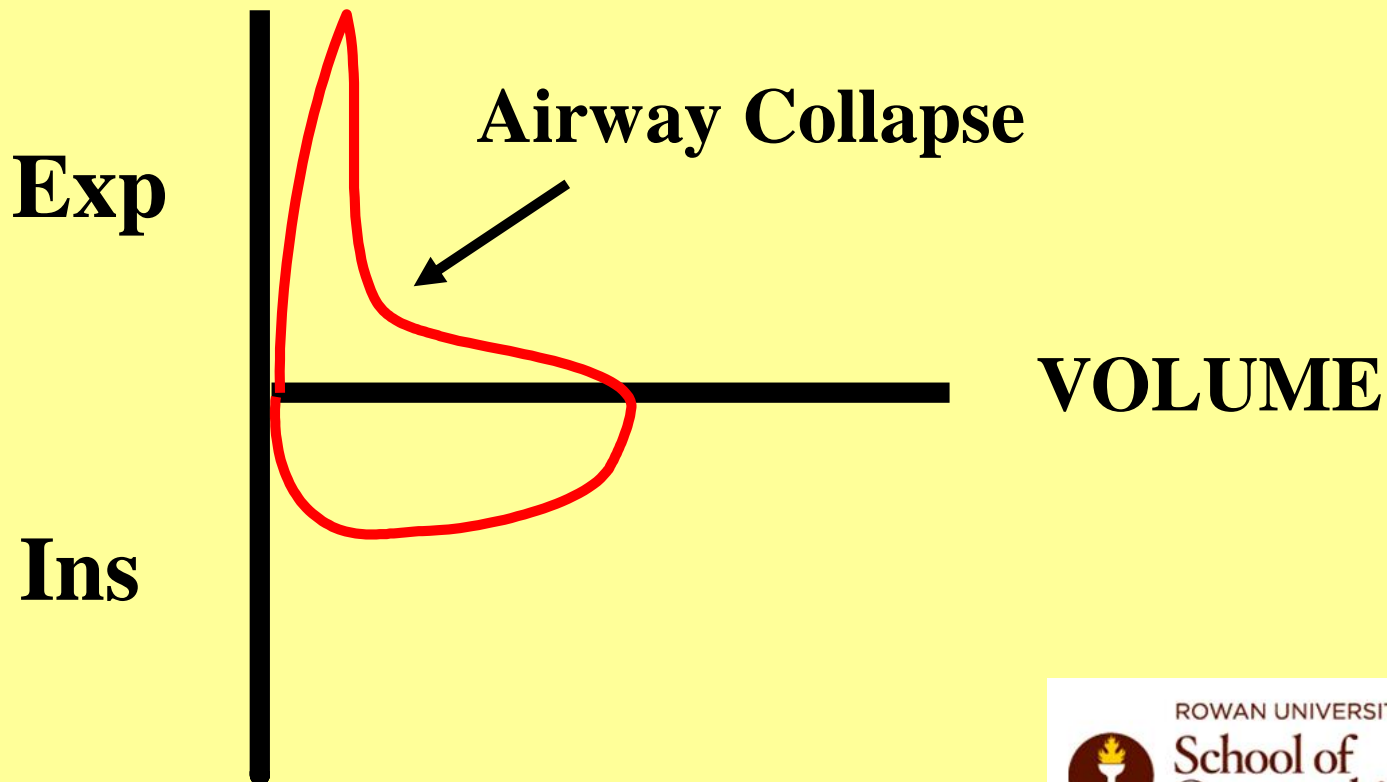
## ***Sleep Apnea/ OHS***



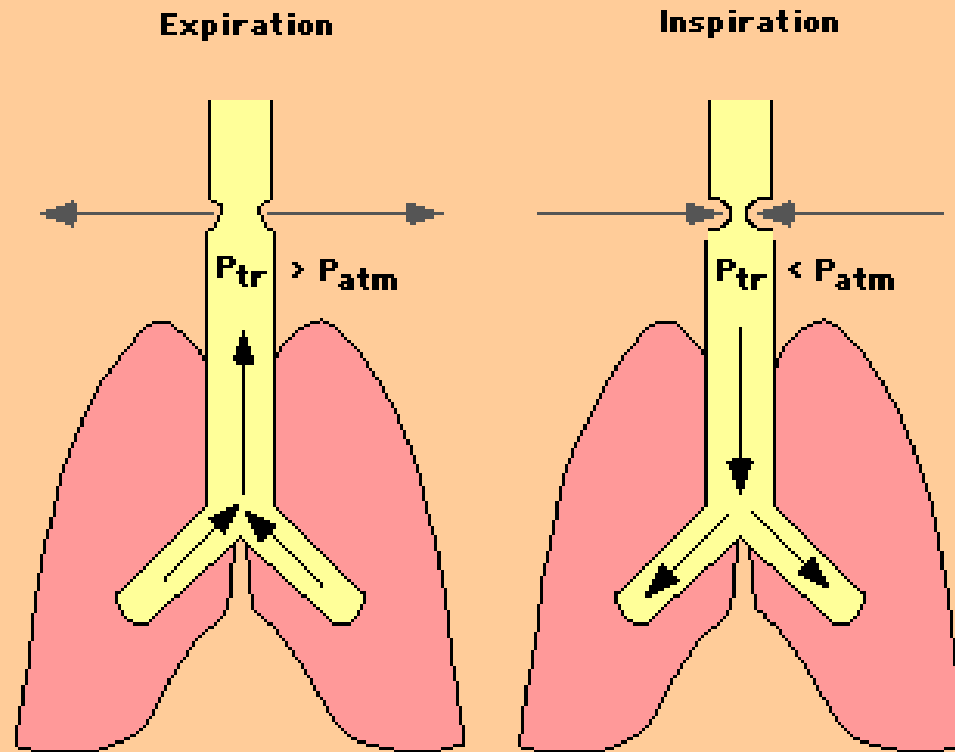
# Flow-Volume Curve

## Severe Airway Obstruction

**FLOW**

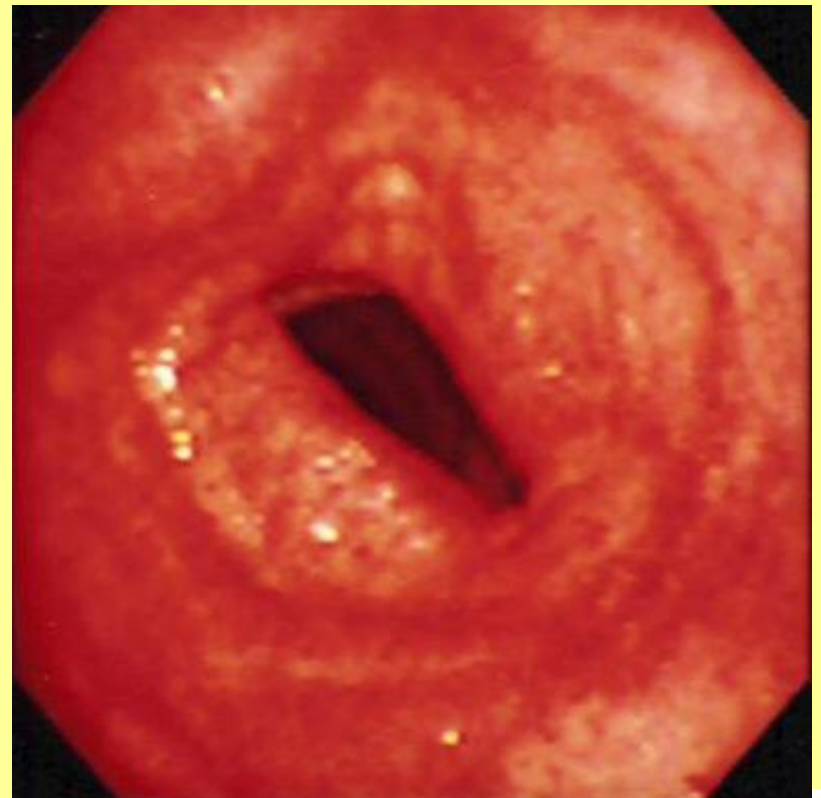






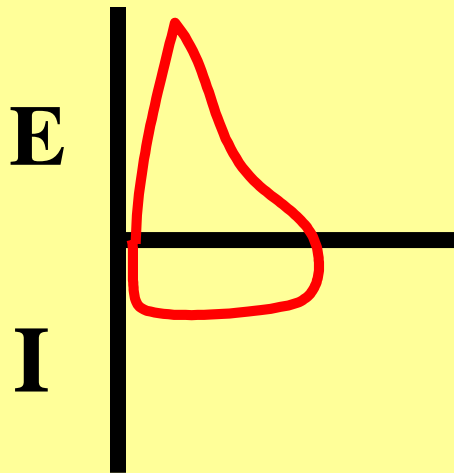
**Effect of dynamic extrathoracic airway obstruction** Effects of forced expiration and inspiration in dynamic extrathoracic airway obstruction. Left, during forced expiration, intratracheal pressure ( $P_{tr}$ ) exceeds the pressure around the airway ( $P_{atm}$ ), lessening the obstruction. Right, during forced inspiration, when intratracheal pressure falls below the atmospheric pressure, the obstruction worsens resulting in flow limitation. (Redrawn from Kryger, M, Bode, F, Antic, R, et al, Am J Med 1976; 61:85.)

# Subglottic Stenosis



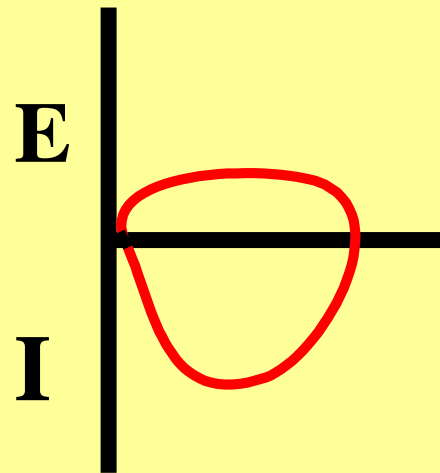
# ***Intra and Extra Thoracic Obstructions***

**VARIABLE**



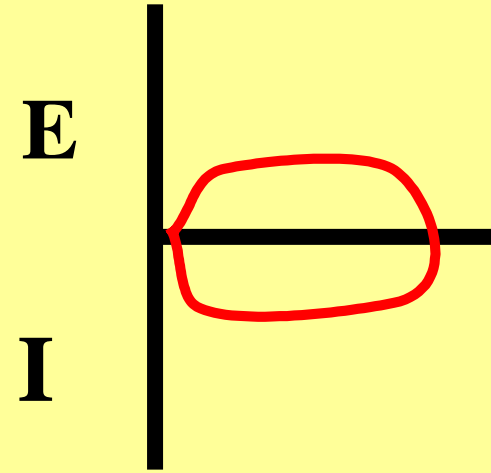
**Extrathoracic**

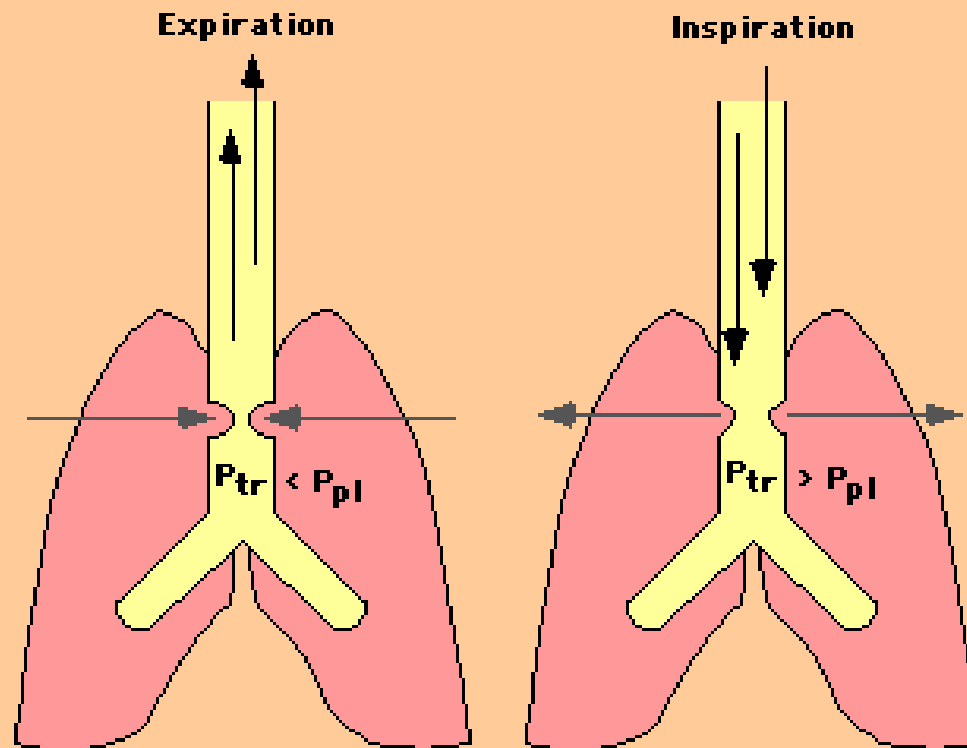
**VARIABLE**



**Intrathoracic**

**FIXED**





**Effects of dynamic intrathoracic airway obstruction** Left panel, during forced expiration, the intrathoracic intratracheal pressure ( $P_{tr}$ ) is less than the pressure in the pleural pressure ( $P_{pl}$ ), worsening the obstruction. Right, during forced inspiration, intratracheal pressure exceeds the pleural pressure, lessening the degree of obstruction. (Redrawn from Kryger, M, Bode, F, Antic, R, et al, Am J Med 1976; 61:85.)



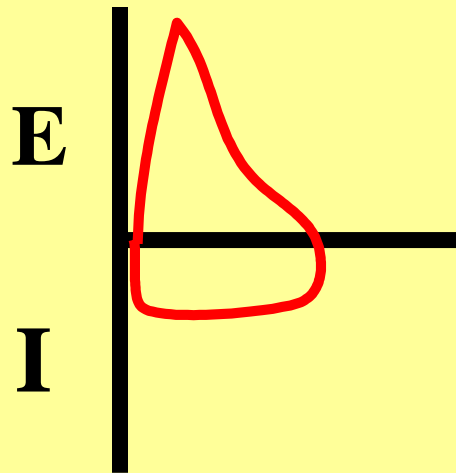
***Intrathoracic***

***Tracheal  
Compression***



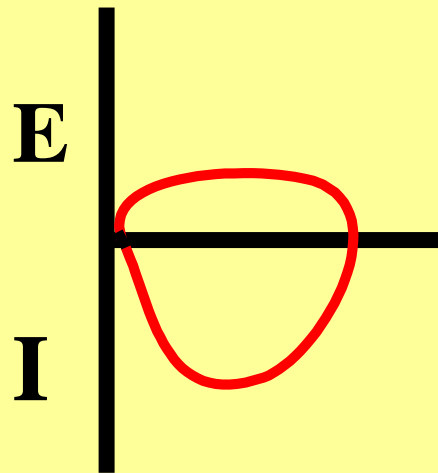
# ***Intra and Extra Thoracic Obstructions***

**VARIABLE**



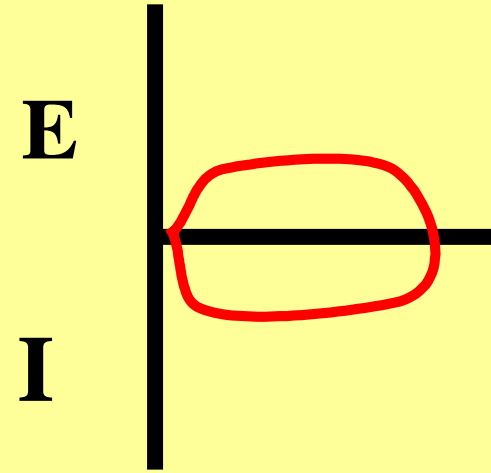
**Extrathoracic**

**VARIABLE**



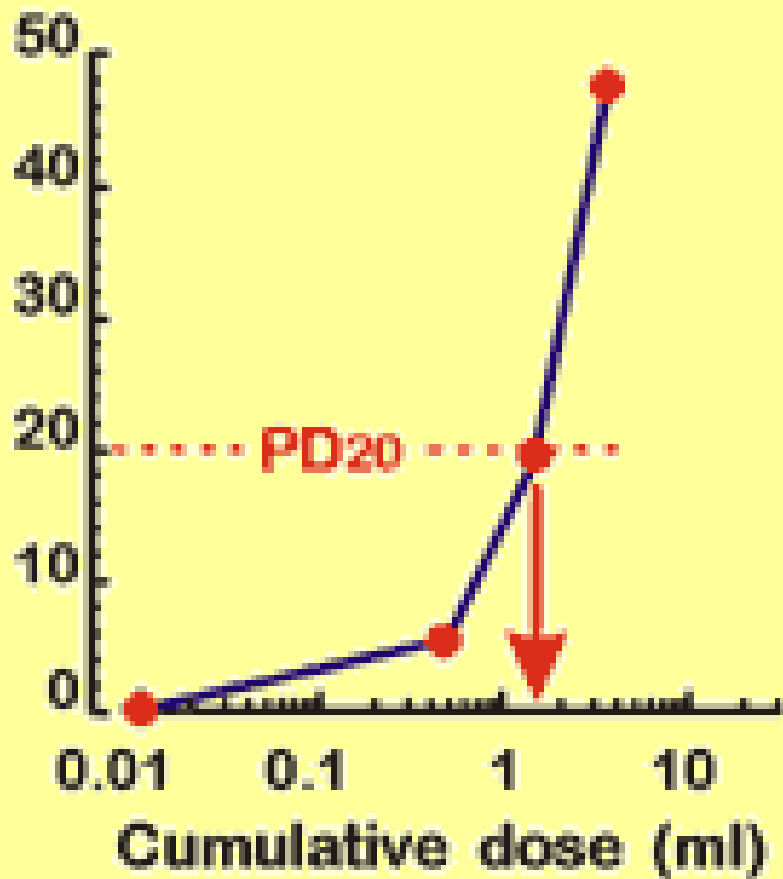
**Intrathoracic**

**FIXED**

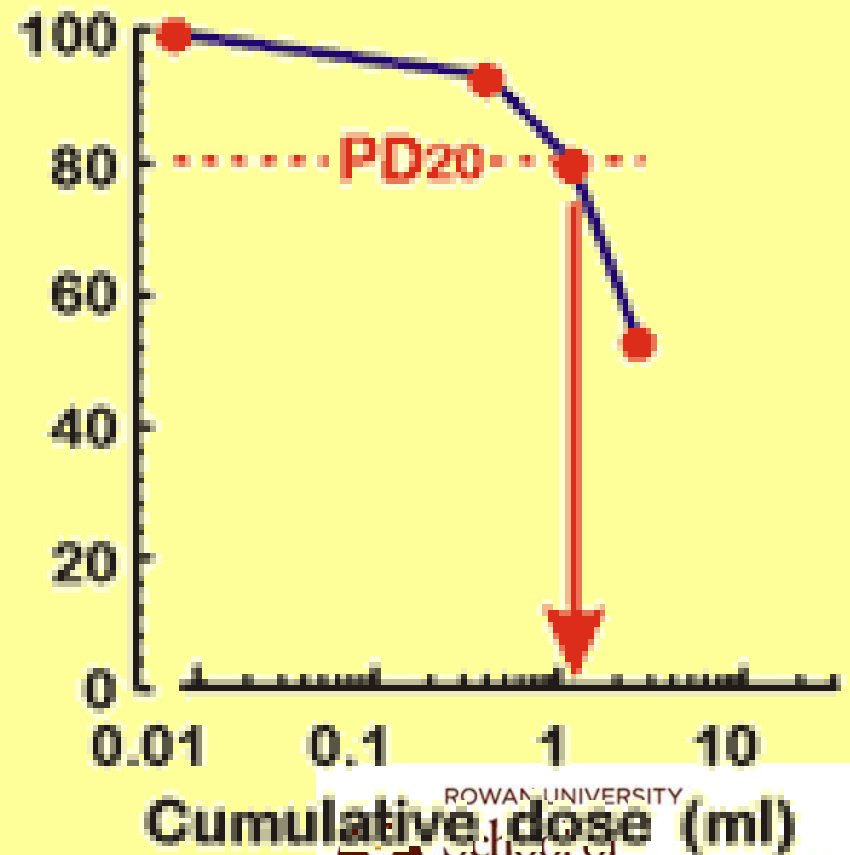


# Bronchial Provocation Testing

% Fall FEV<sub>1</sub>



% Predicted FEV<sub>1</sub>



# ***Diseases associated with Nonspecific Bronchial Hyperresponsiveness***

**Asthma**

**COPD**

**Bronchiolitis**

**Viral URI**

**Hay Fever**

**Cystic Fibrosis**

**Foreign body aspiration**

**Near drowning**

**Smoke inhalation**

**Sarcoidosis**

**Post ARDS**