ARF, Mechaical Ventilation and PFTs: ACOI Board Review 2020

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No Disclosures

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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.

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Acute Respiratory Failure

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

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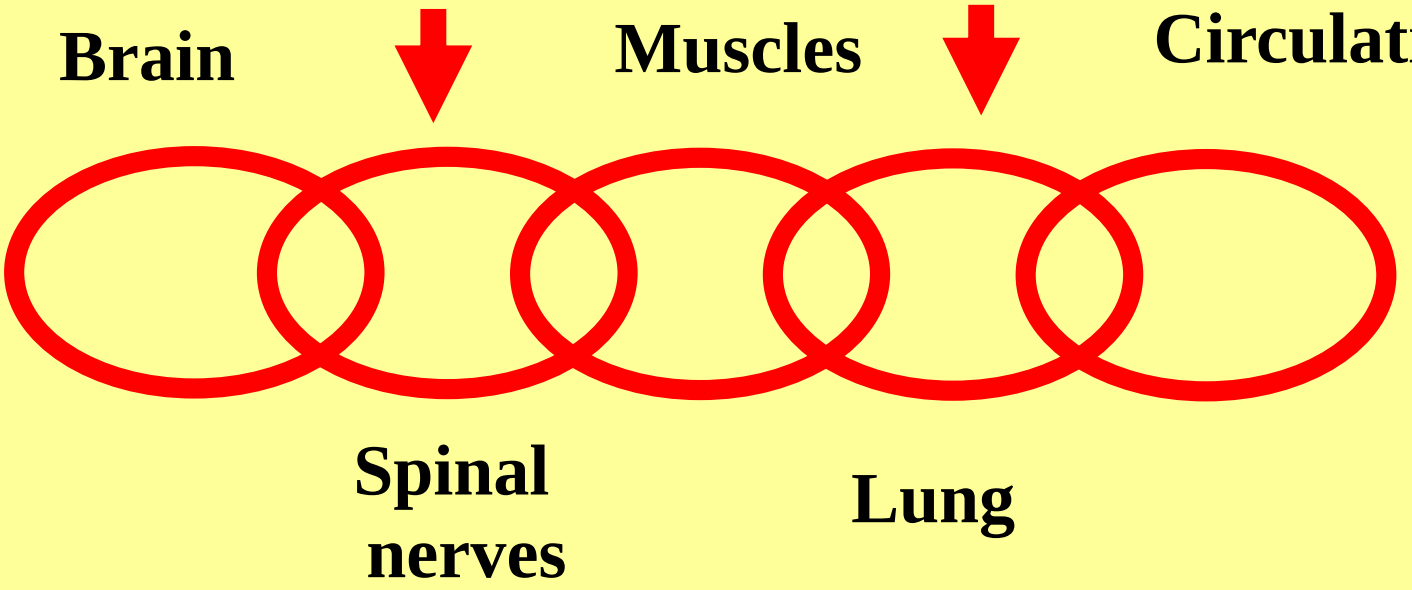
Anatomic Etiologic Classification



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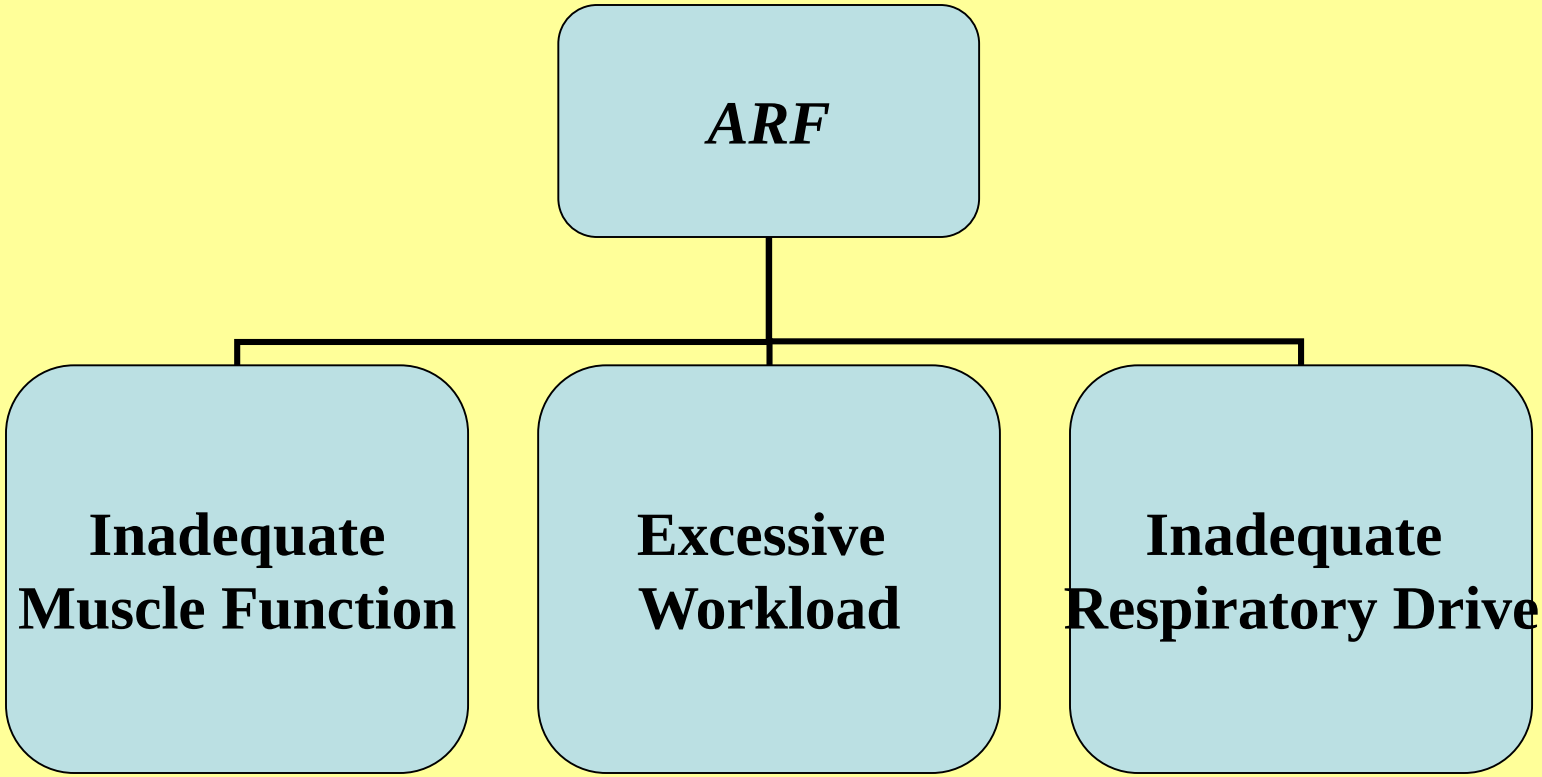
Physiologic Etiologic Classification



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Blood Gas Classification

Hypoxemic/Hypercapnic

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

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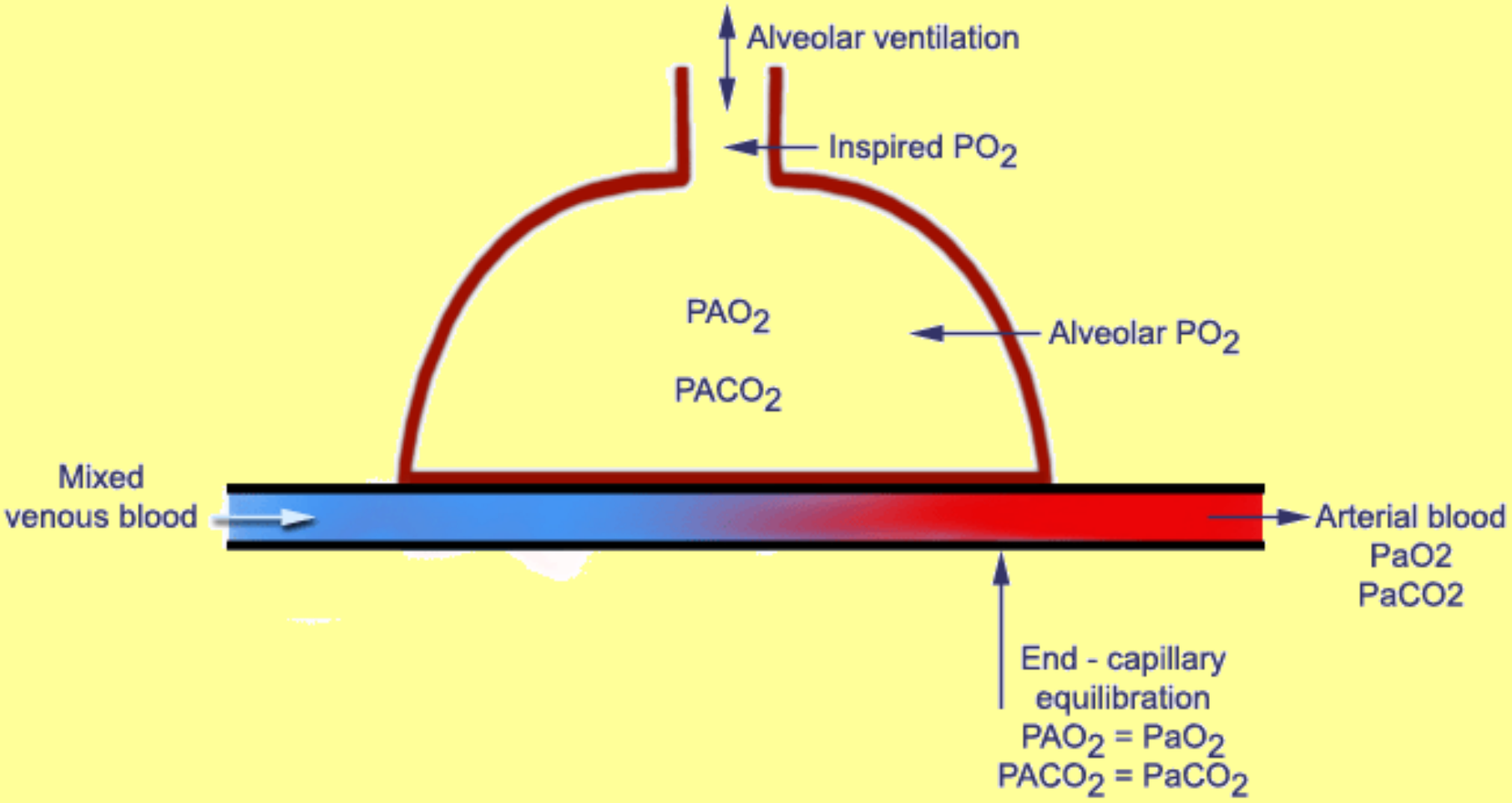

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Calculation of the A-a Gradient

$PAO_2 = FIO_2 (Pb - 47) - 1.25\ PaCO_2$

PaO_2 = measured

A-a gradient should be less than 20 mmHg breathing room air OR
Less than 100 mmHg on 100 % O_2

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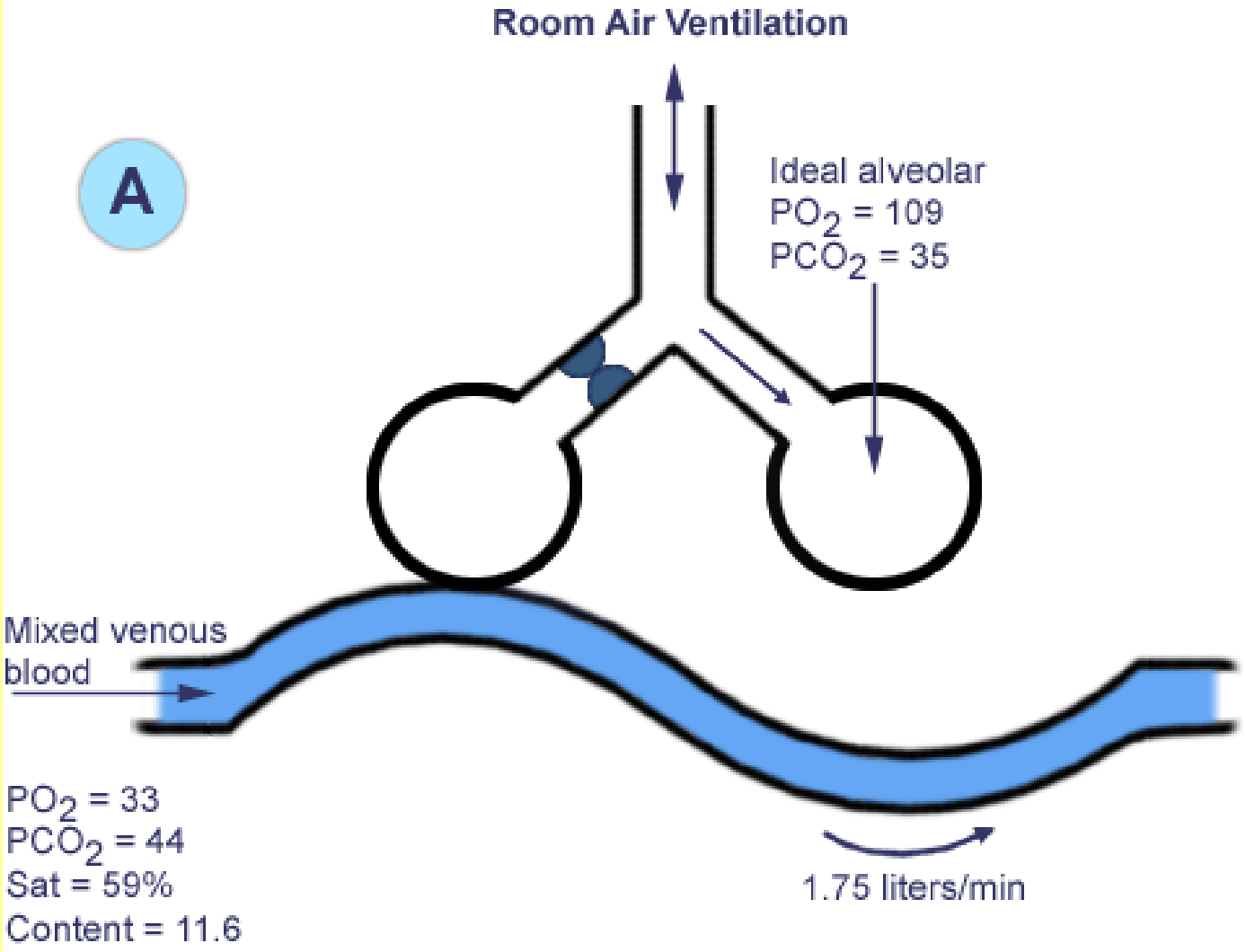
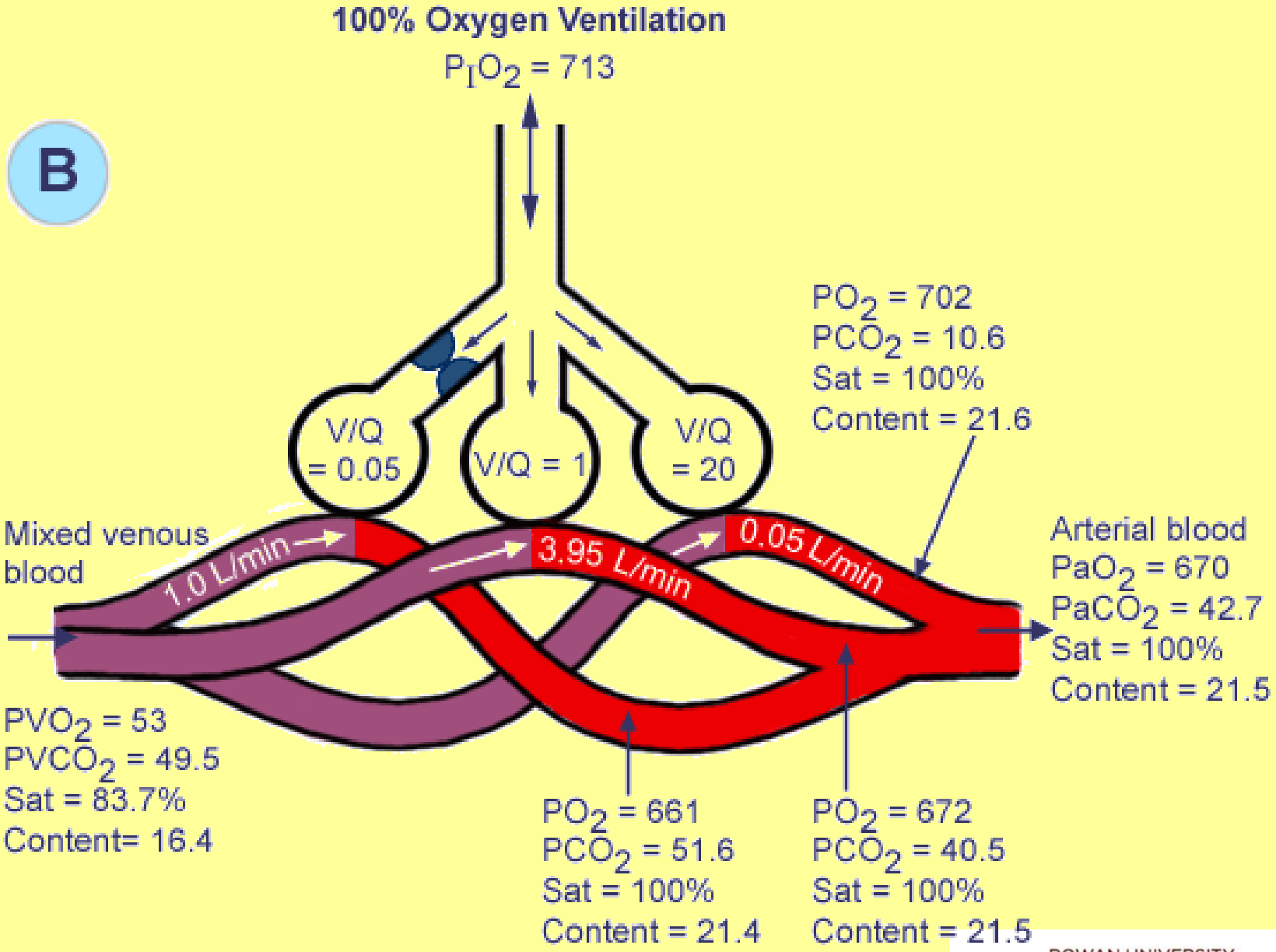
100% Oxygen and Pulmonary Shunt





Causes of Hypoxemia

CAUSE	A-a Gradient	PaCO2	Response to 100 % Oxygen
Low FIO2	Normal	Normal	Improved
Hypoventilation	Normal	Increased	Improved
Diffusion Impair	Increased	Normal	Improved
Low V/Q	Increased	Normal	Improved
Shunt	Increased	Normal	NOT Improved
Low PvO2	Increased	Normal	? Improved

Mechanisms of Hypercapnia

$$PaCO_2 = K \frac{VCO_2}{Va}$$

PaCO2 = arterial CO2 tension

K = proportionality constant

VCO2 = CO2 production

Va = Alveolar ventilation

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Causes of Hypercapnia

1. **Alterations in CO_2 production**
2. **Disturbances in the Gas Exchanger (the lungs)**
3. **Abnormalities in the mechanical system (the bellows)**
4. **Changes in ventilatory control**

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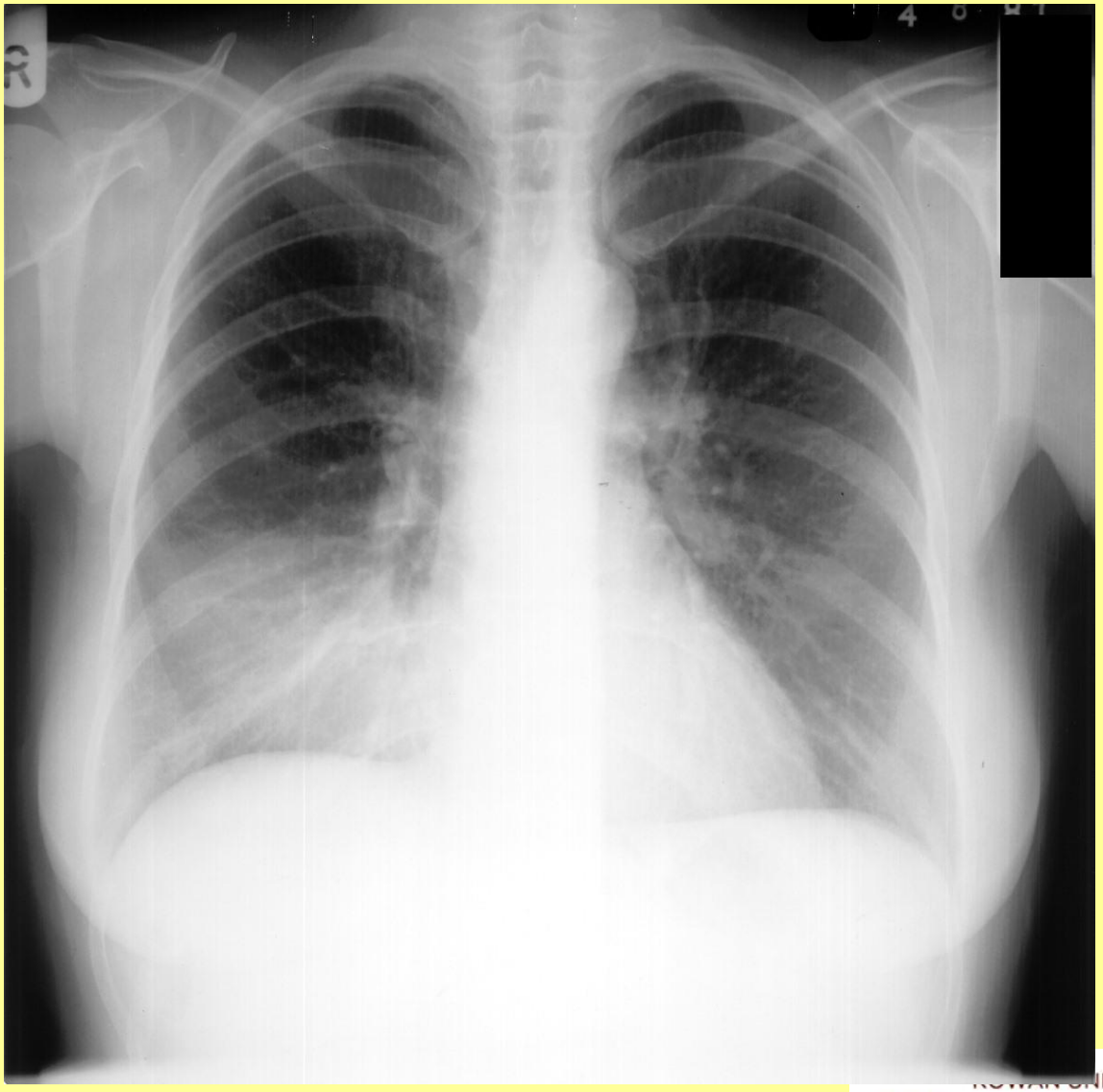
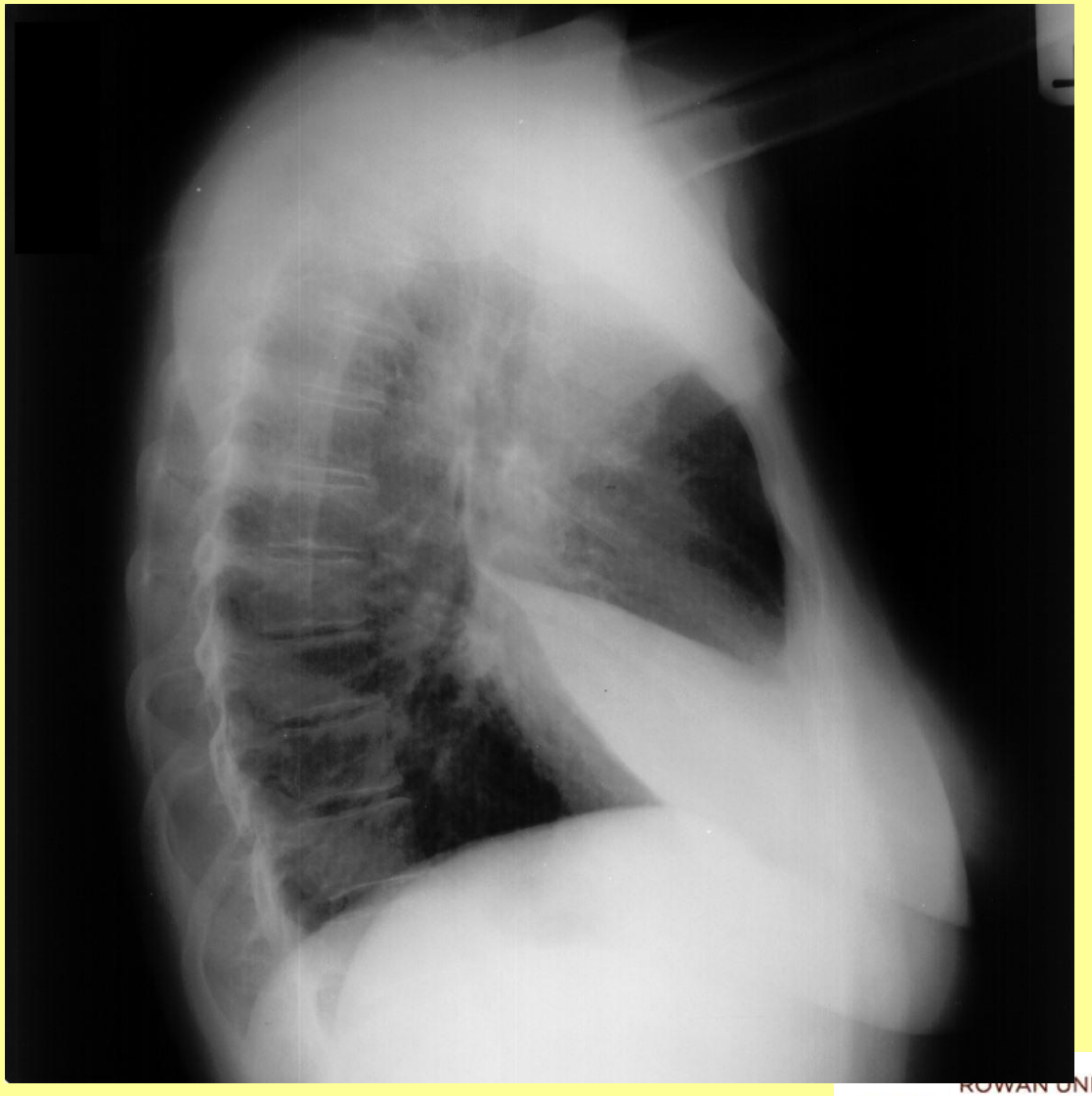
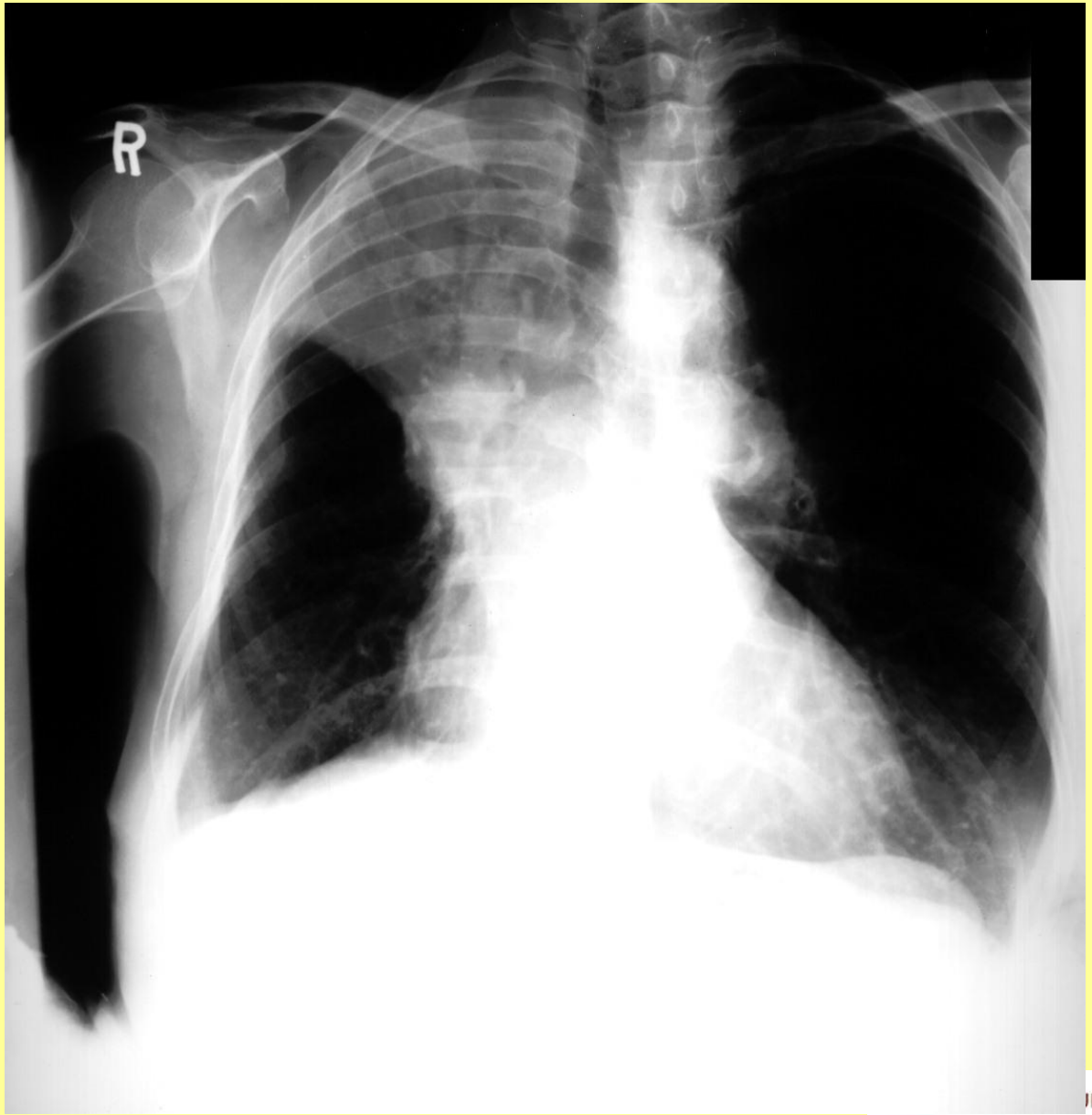
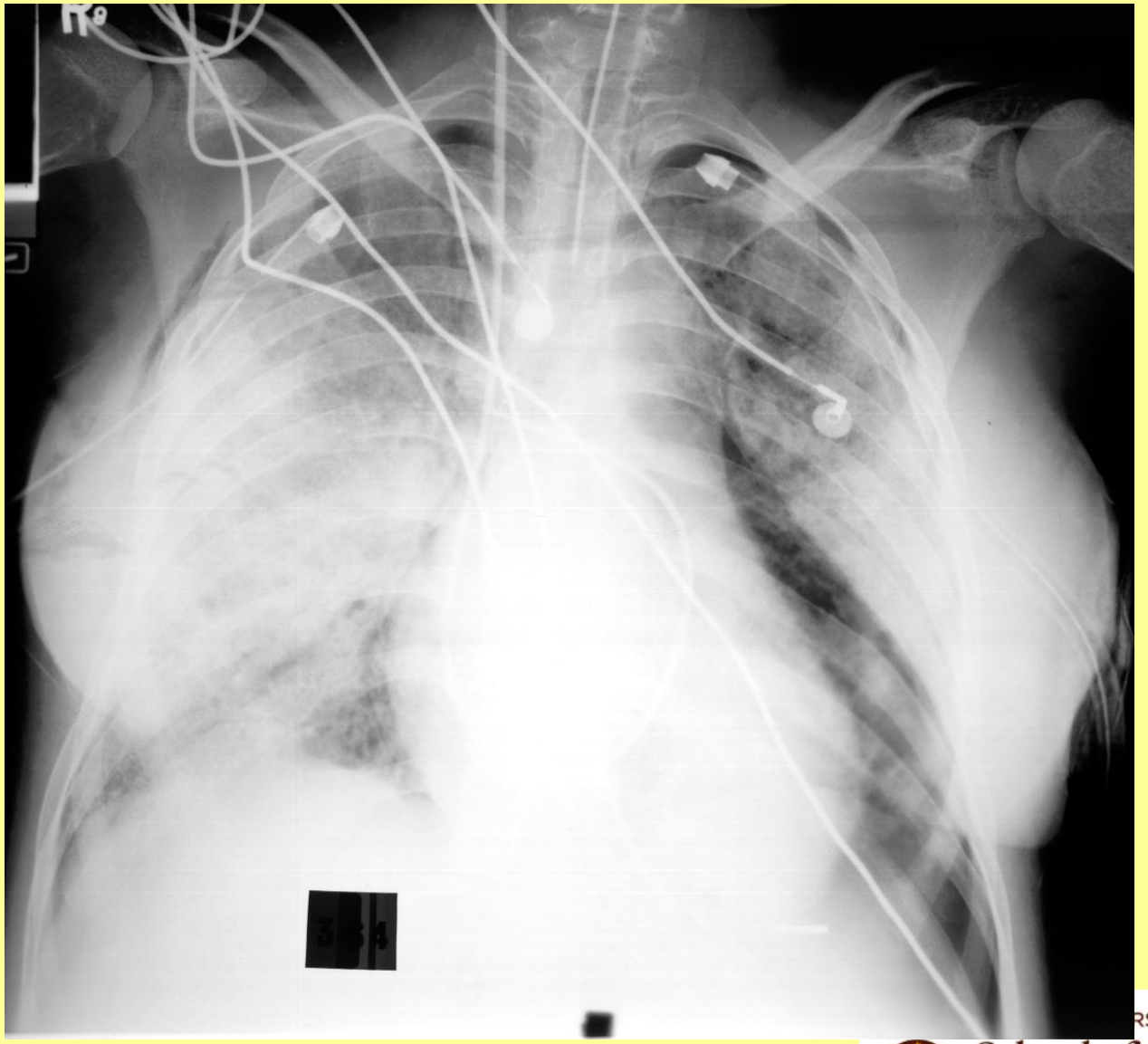
Radiographic Classification of ARF

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









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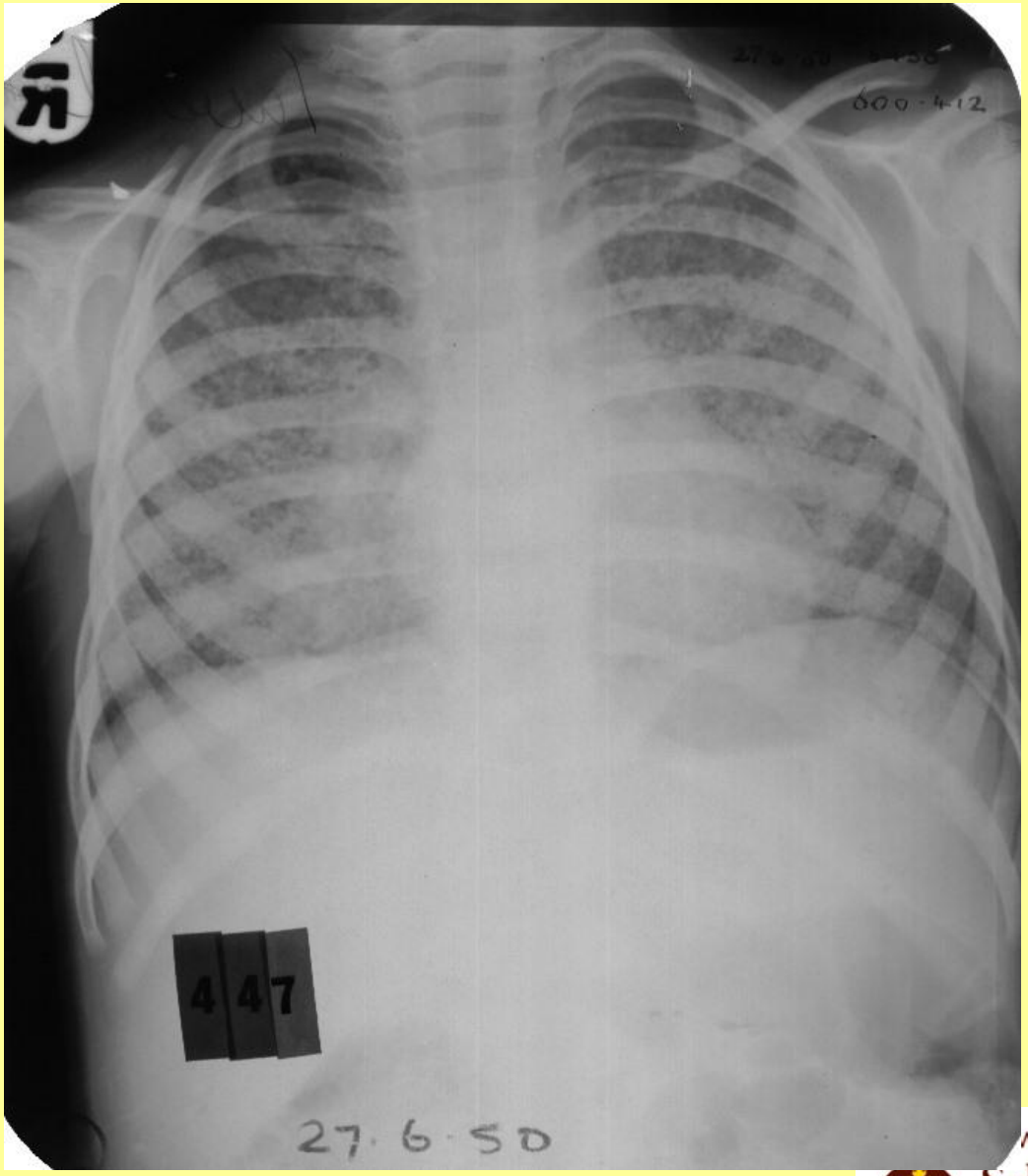
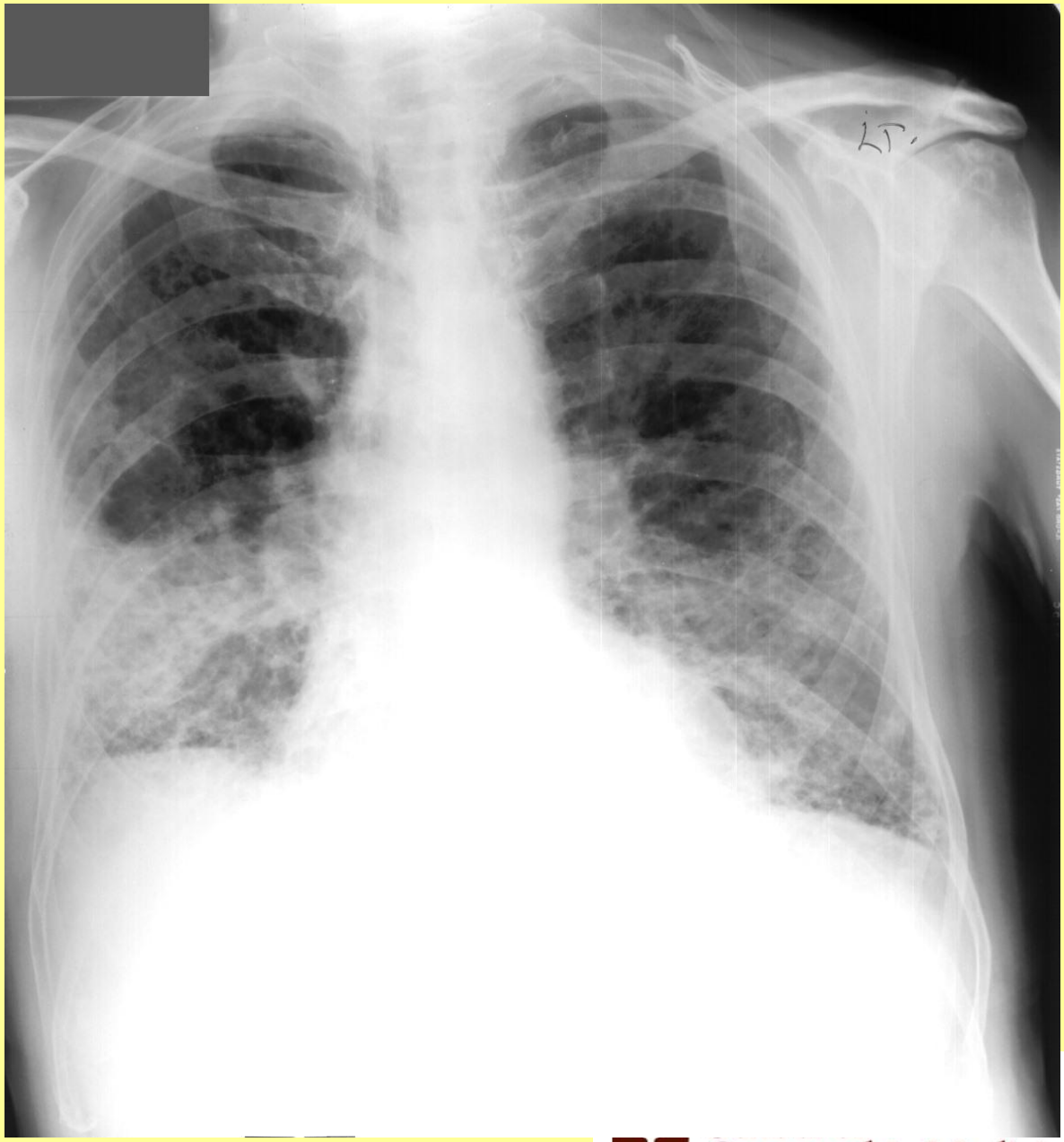


pulmonary fibrosis due to RA



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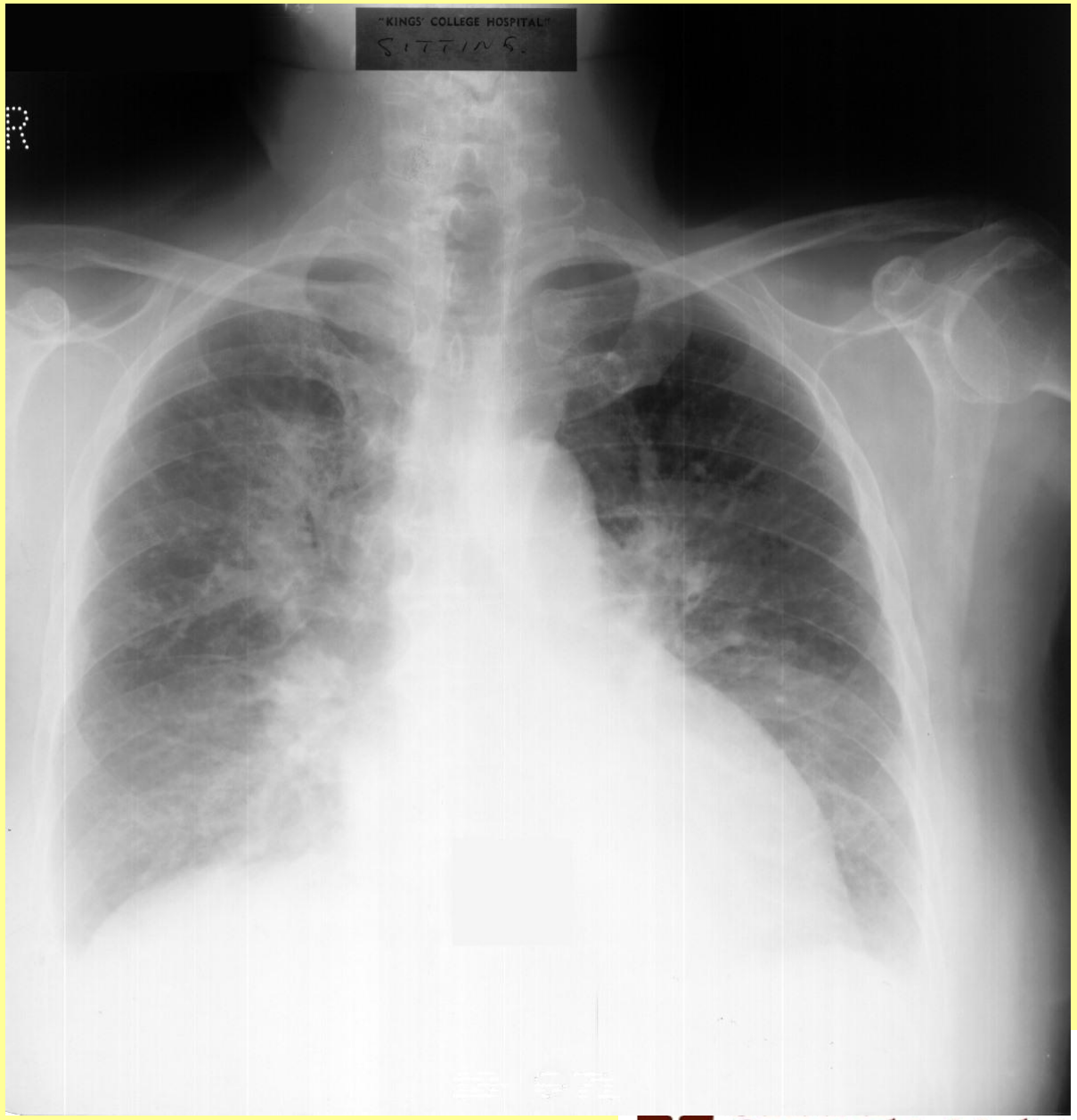
76 yo
Female

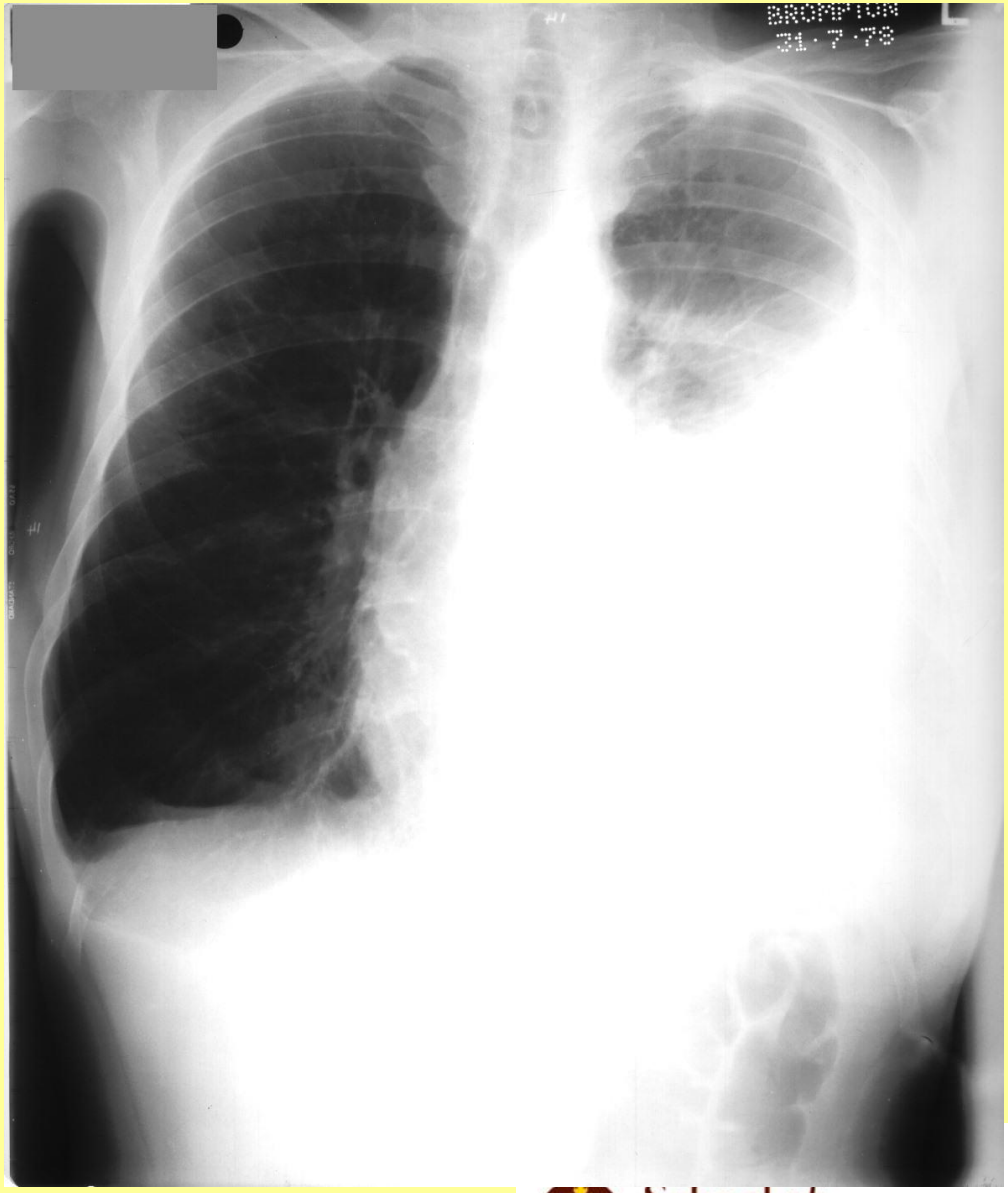
SOB
Edema
Orthopnea



Osteopathic Medicine

Male

SOB



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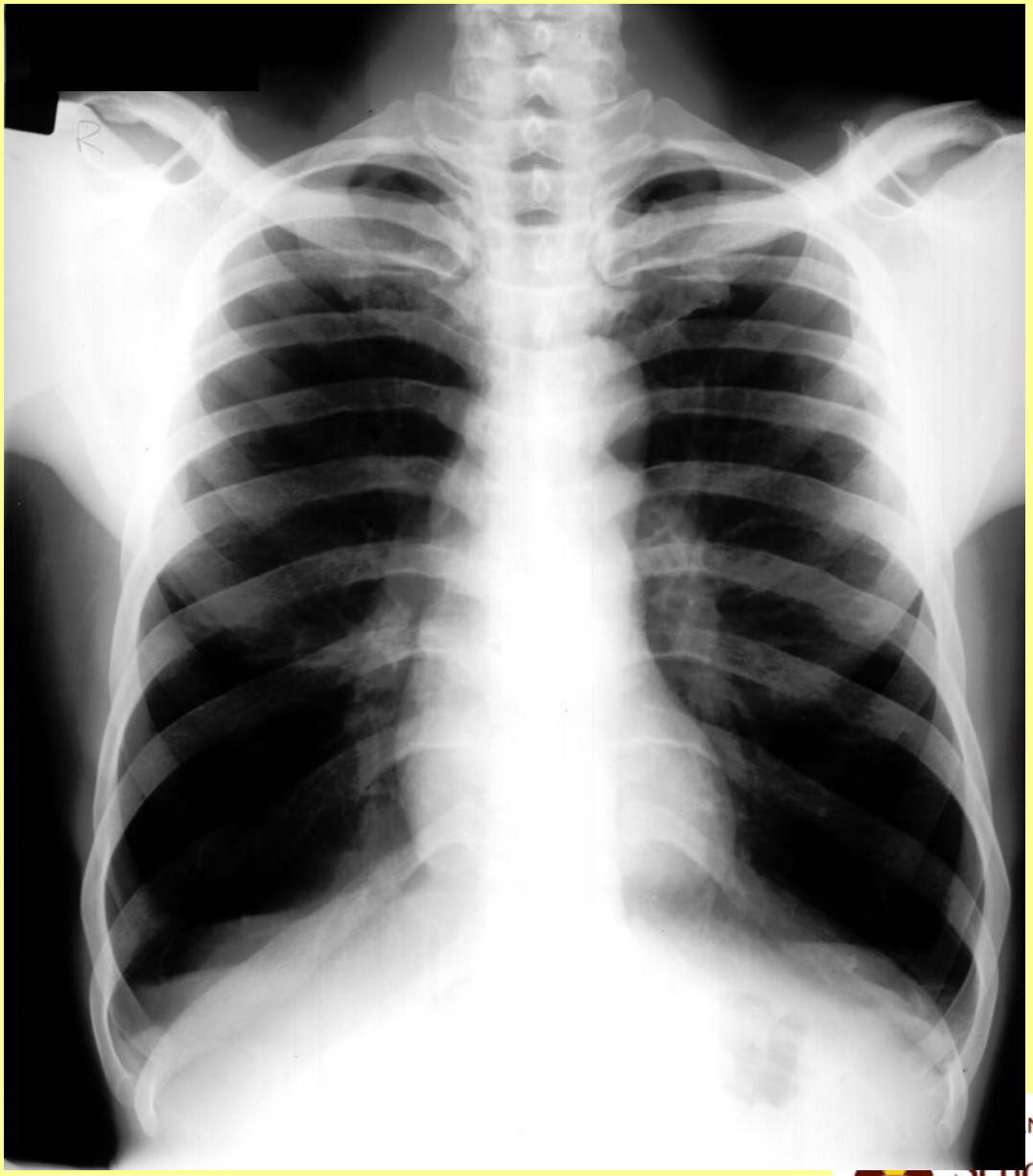
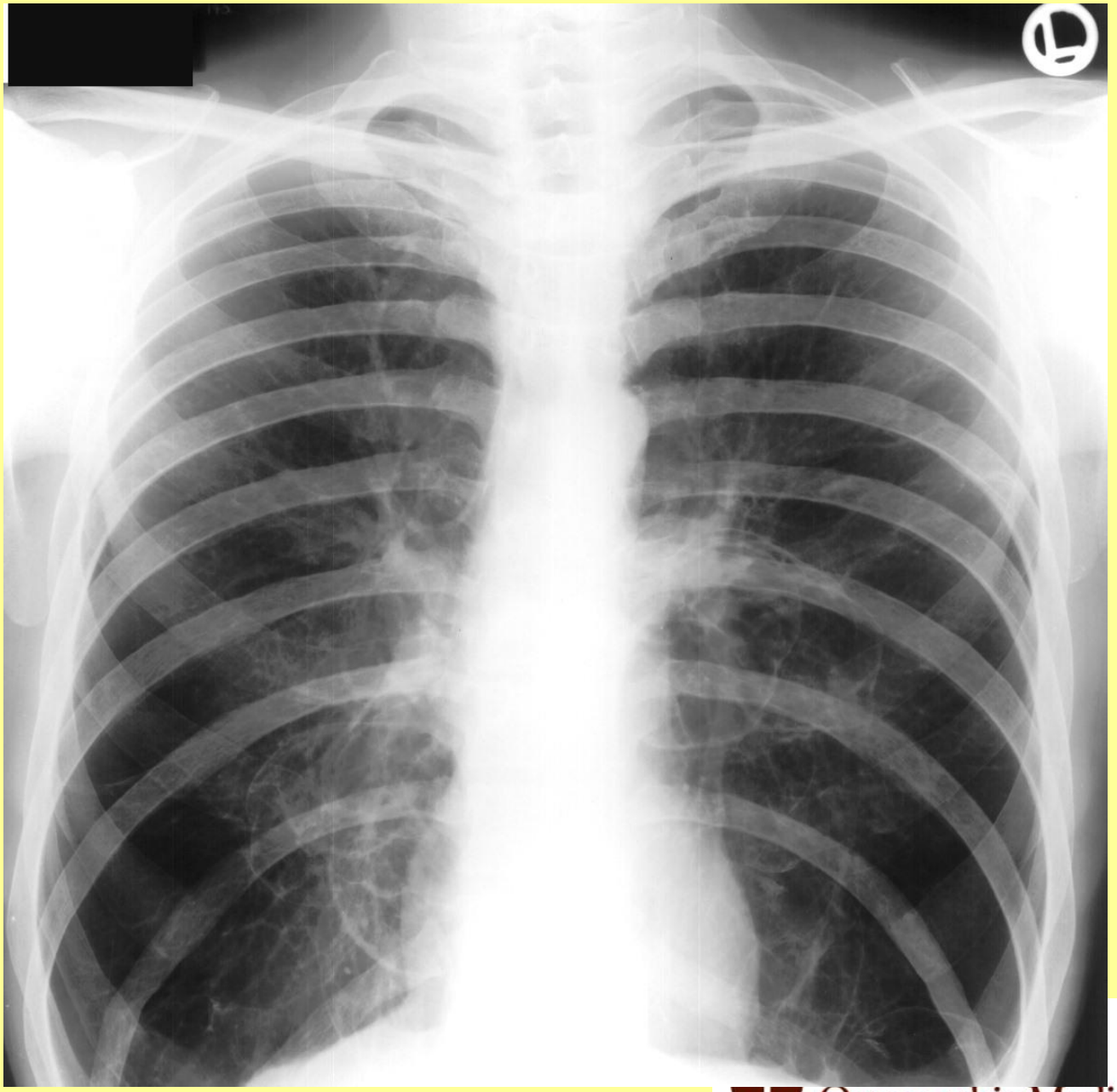


Male 40 yo

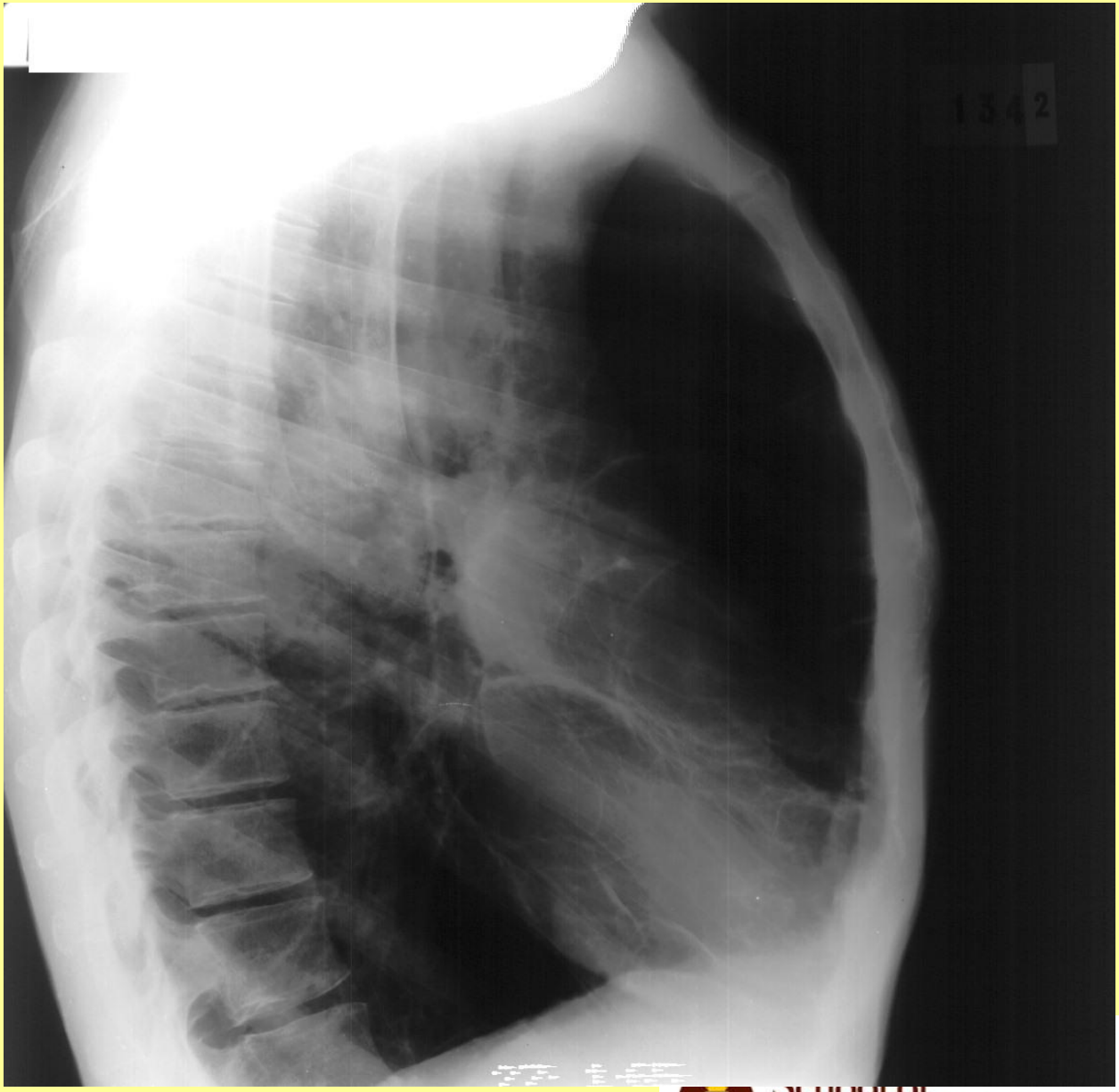
Dyspnea



Osteopathic Medicine

Male 40 yo

Dyspnea



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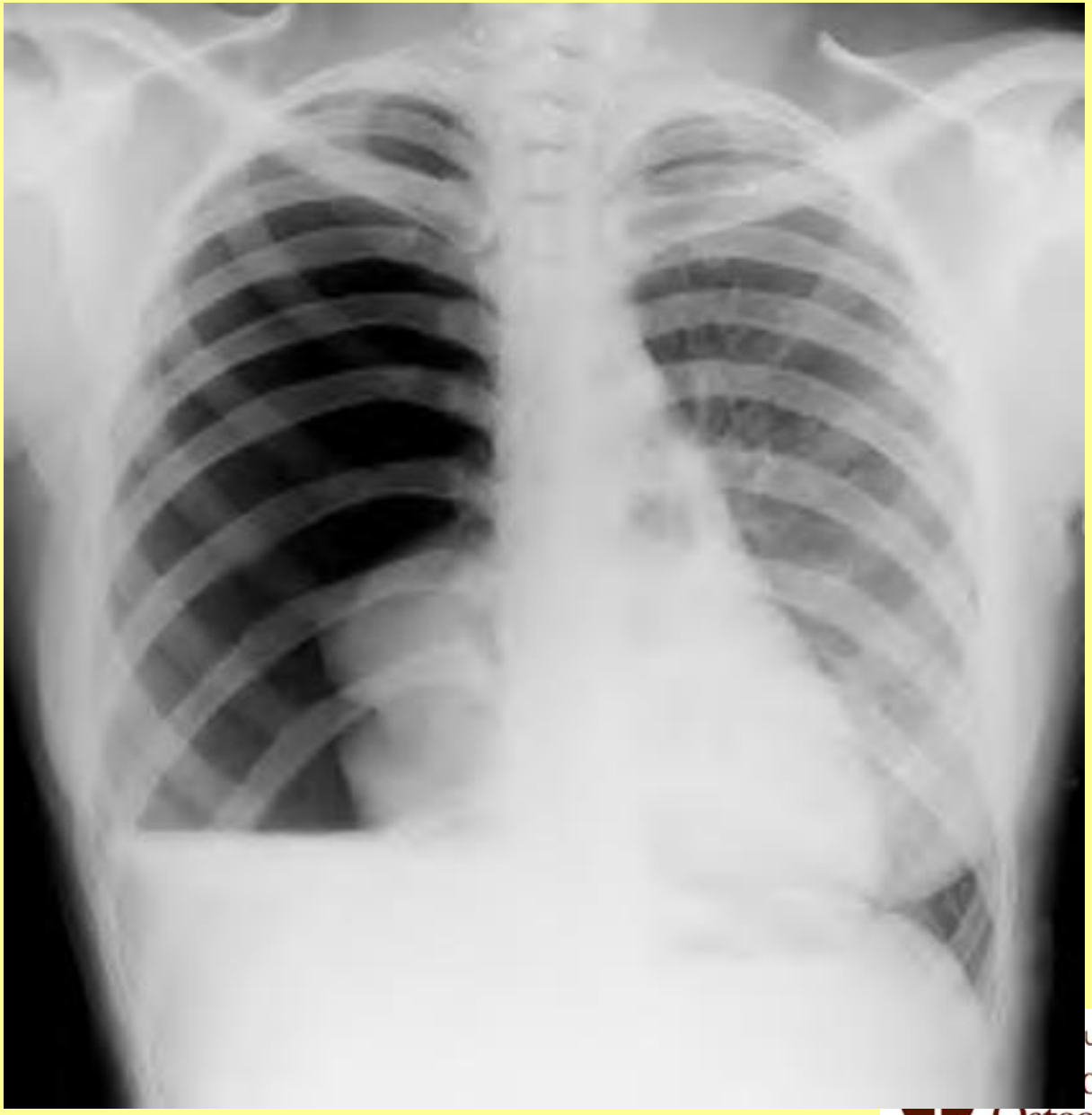

CONDITION	DEFINITION	EXAMPLE	ABNORMALITY
Ventilatory Failure	Abnormal CO2 elimination by lungs	Drug overdose Asthma	$PaCO_2$ > 50 mmHg
Failure of Arterial Oxygenation	Abnormal O_2 uptake by lung	Pneumonia, ARDS	PaO_2 < 50 mm Hg
Failure of Oxygen Delivery	Abnormal O_2 delivery to the tissues	Cardiogenic shock Anemia, CO poisoning	CvO_2< 18 cc/dl PvO_2< 30 mmHg SvO_2< 60 %
Failure of Oxygen Utilization	Failure of O_2 uptake by tissues	Cyanide poisoning septic shock	CvO_2> 18 cc/dl PvO_2> 60 mmHg SvO_2> 80 %

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Barotrauma

- In ther past it was believed that alveolar rupture was due to excessive proximal airway pressure

- If peak airway pressure exceeded 50 cm H2O then the patient was considered to be at high risk for pneumothorax

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Key Concepts in VILI

- **VILI – ventilator induced lung injury**
- **Barotrauma – It is not the pressure applied to the lung that causes lung injury**
  - Atelectrauma – ventilating at too LOW lung volumes or PEEP that is too low
  - Volutrauma – ventilating at too high lung volumes leads to alveolar over distention
  - Ventilation at too High PEEP - Volutrauma

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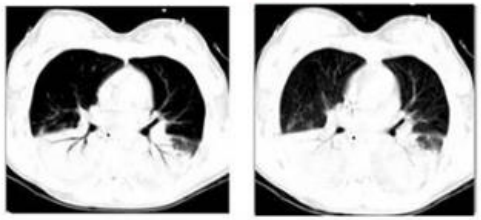
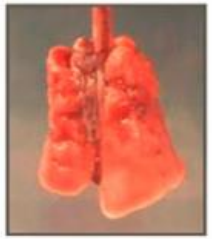
A Ventilation at low lung volume

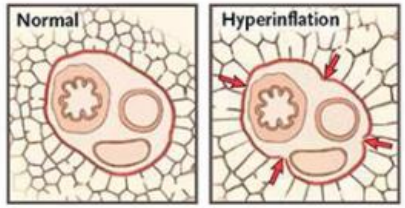


atelectotrauma



lung inhomogeneity

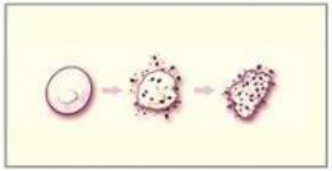
B Ventilation at high lung volume



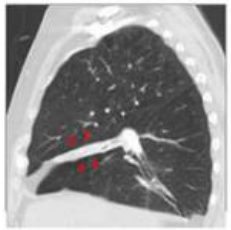
air leaks

lung overdistension

E Ventilation with high FiO_2

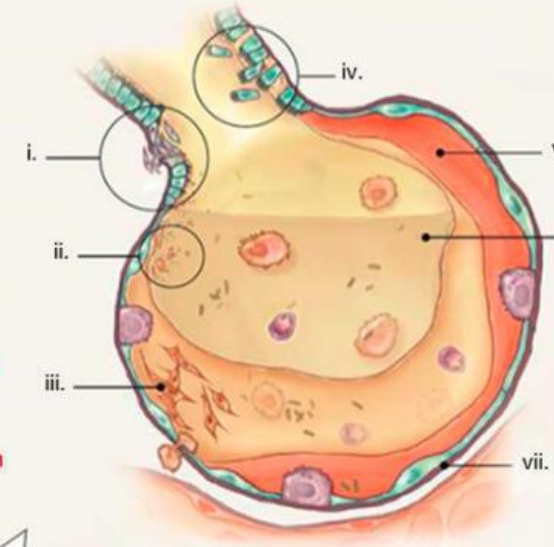


free oxygen radicals–induced cell injury



resorbtion atelectasis

F Structural consequences



i. epithelial-mesynchymal transformations
ii. surfactant dysfunction
iii. fibroproliferation

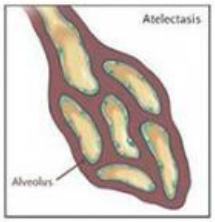
iv. sloughing of bronchial epithelium
v. hyaline membranes
vi. pulmonary edema
vii. increased alveolar-capillary permeability

G Postoperative Lung Injury

- need for postoperative O_2
- need for postoperative ventilation
- ARDS, pneumonia
- pneumothorax

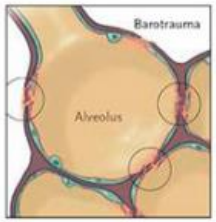
H Longer stay in hospital and increased mortality

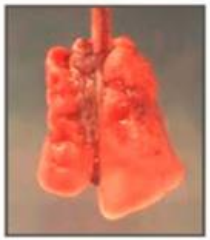
C Ventilation at low PEEP



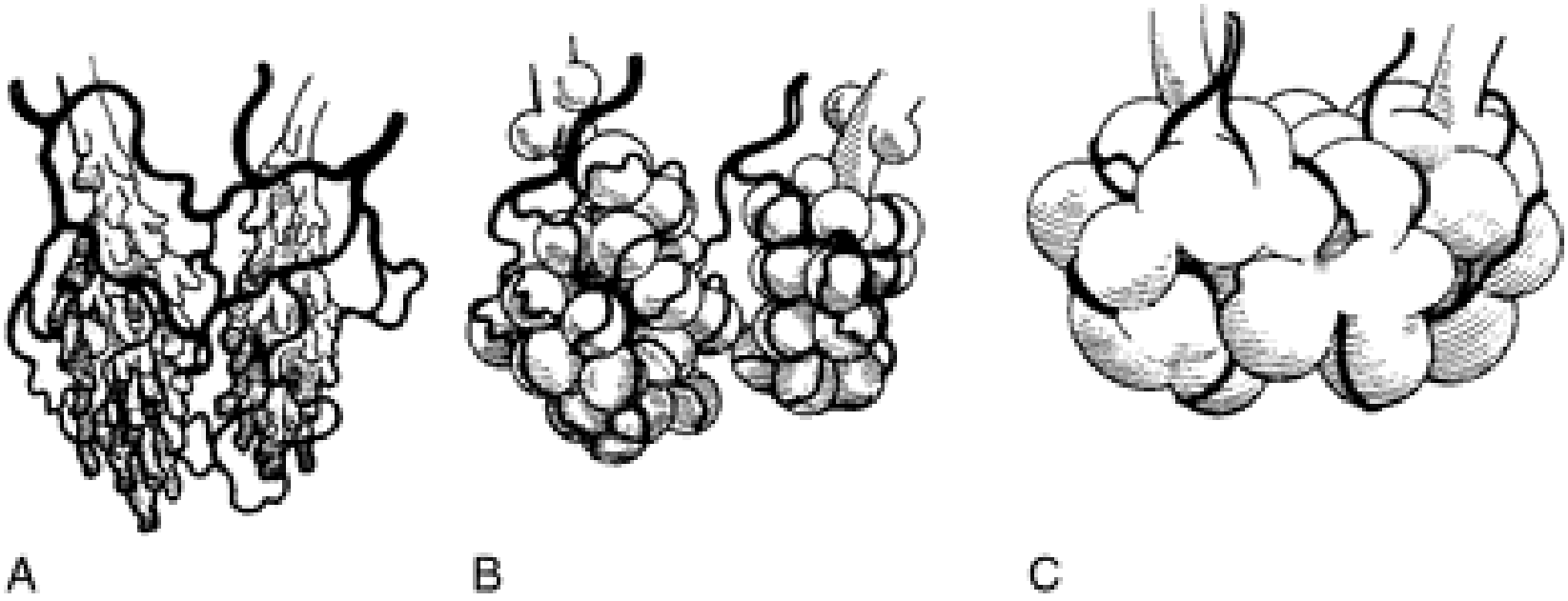
lung tissue with atelectasis

D Ventilation at high PEEP



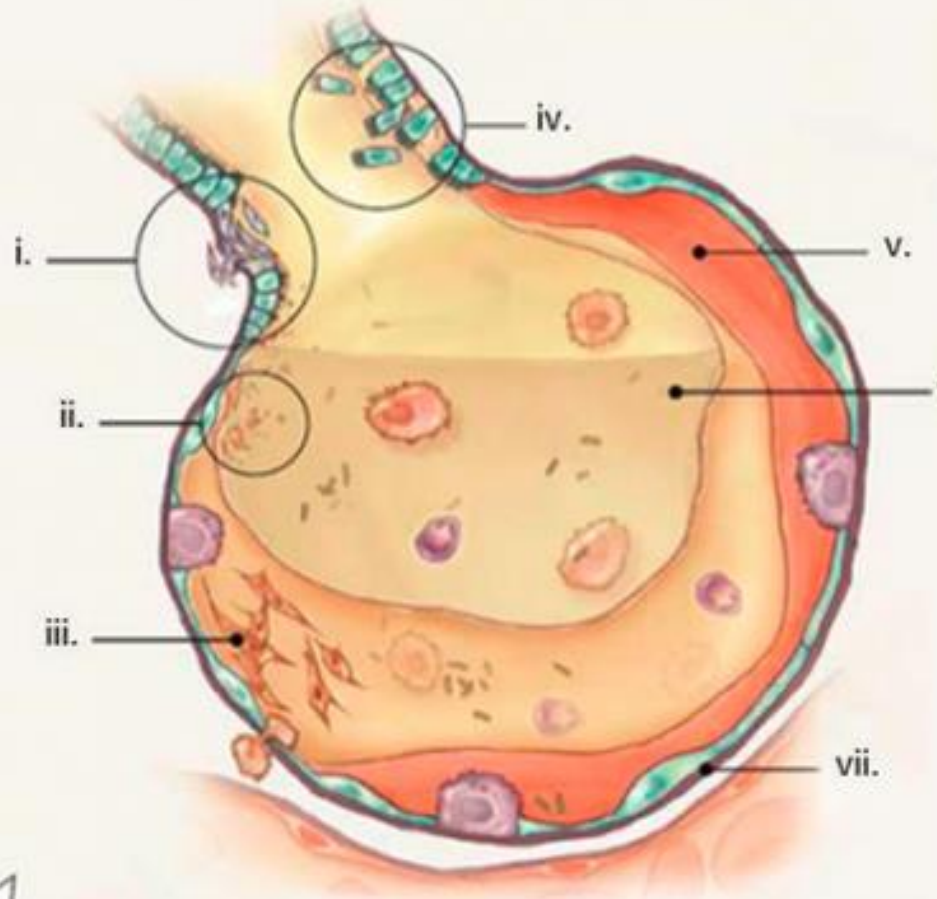


lung tissue with overdistension



Effects of the application of positive end-expiratory pressure (PEEP) on the alveoli. *A*, Atelectatic alveoli before PEEP application. *B*, Optimal PEEP application has reinflated alveoli to normal volume. *C*, Excessive PEEP application overdistends the alveoli and compresses adjacent pulmonary capillaries, creating dead space with its attendant hypercapnia. From Pierce, 1995.

F Structural consequences

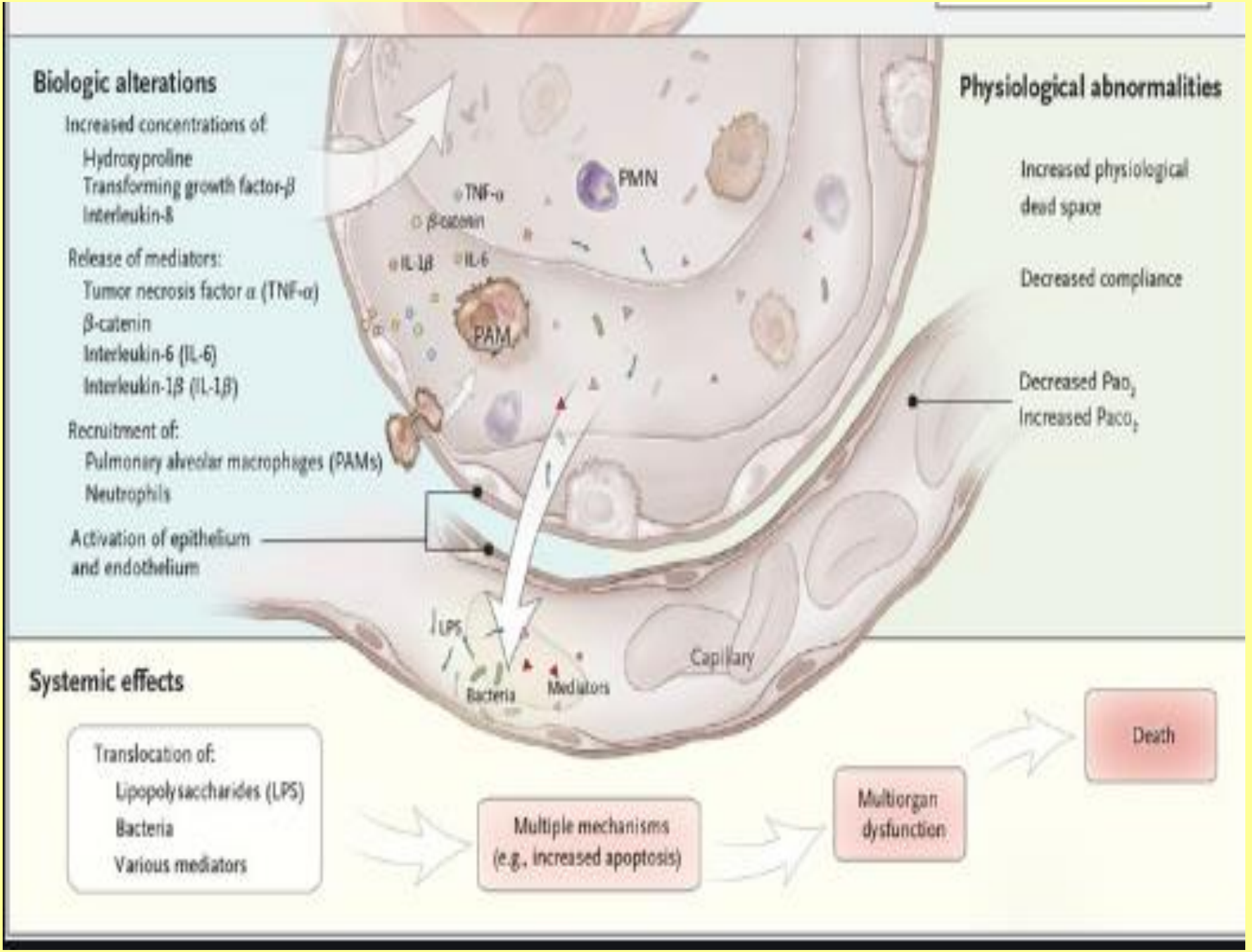


i. epithelial-mesynchymal transformations
ii. surfactant dysfunction
iii. fibroproliferation

iv. sloughing of bronchial epithelium
v. hyaline membranes
vi. pulmonary edema
vii. increased alveolar-capillary permeability

Biotrauma

- **The combination of Atectrauma and Volutrauma lead to the damage to the vascular endothelium, capillary epithelium and even some airway damage that leads to the recruitment of effector cells into the alveolus that enhance further lung damage.**


Biologic alterations
Increased concentrations of
Hydroxyproline
Transforming growth factor-β
Interleukin-8
Release of mediators:
Tumor necrosis factor α (TNF-α)
β-catenin
Interleukin-6 (IL-6)
Interleukin-1β (IL-1β)
Recruitment of:
Pulmonary alveolar macrophages (PAMs)
Neutrophils
Activation of epithelium and endothelium
TNF-α
β-catenin
IL-1β
IL-6
PMN
PAM
LPS
Bacteria
Mediators
Capillary
Physiological abnormalities
Increased physiological dead space
Decreased compliance
Decreased Pao_2
Increased $Paco_2$
Systemic effects
Translocation of:
Lipopolysaccharides (LPS)
Bacteria
Various mediators
Multiple mechanisms (e.g., increased apoptosis)
Multiorgan dysfunction
Death

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.

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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 Ve above 20 L/min (Brown. Respir Care 1986; 31:1069-74)

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AutoPEEP (AP)

Causes

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

AutoPEEP
Methods for Detection

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

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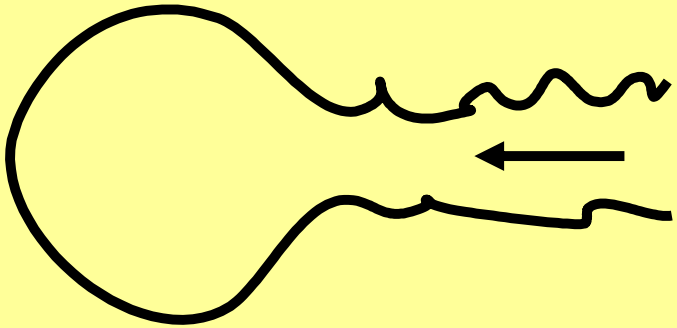
- **If inspiratory resistance is HIGH DISTAL ALVEOLAR PRESSURE may be LOWER than PEAK AIRWAY PRESSURE !**

Alveolar Pres = 20 cm H20



PAP = 50 cm H2O

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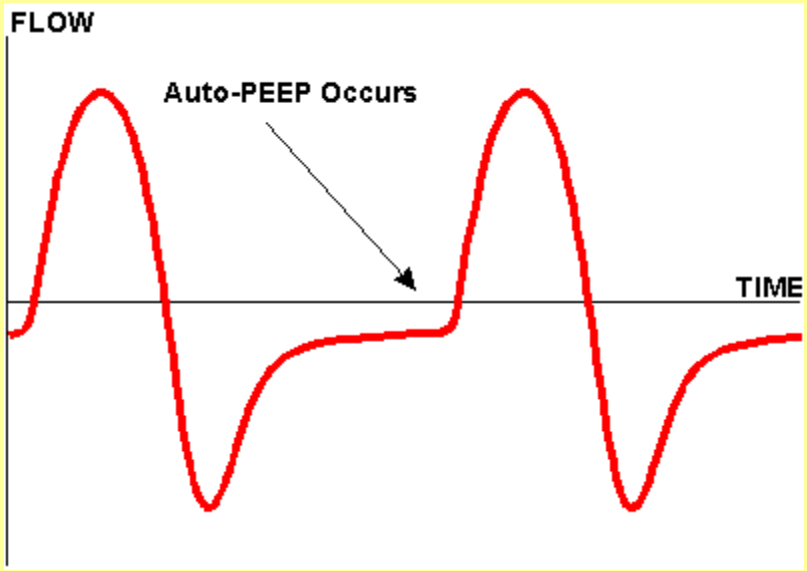
Auto PEEP detection



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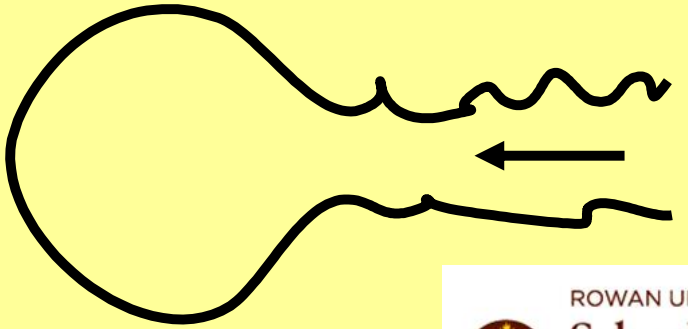
AutoPEEP

- 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



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How do we measure AutoPEEP

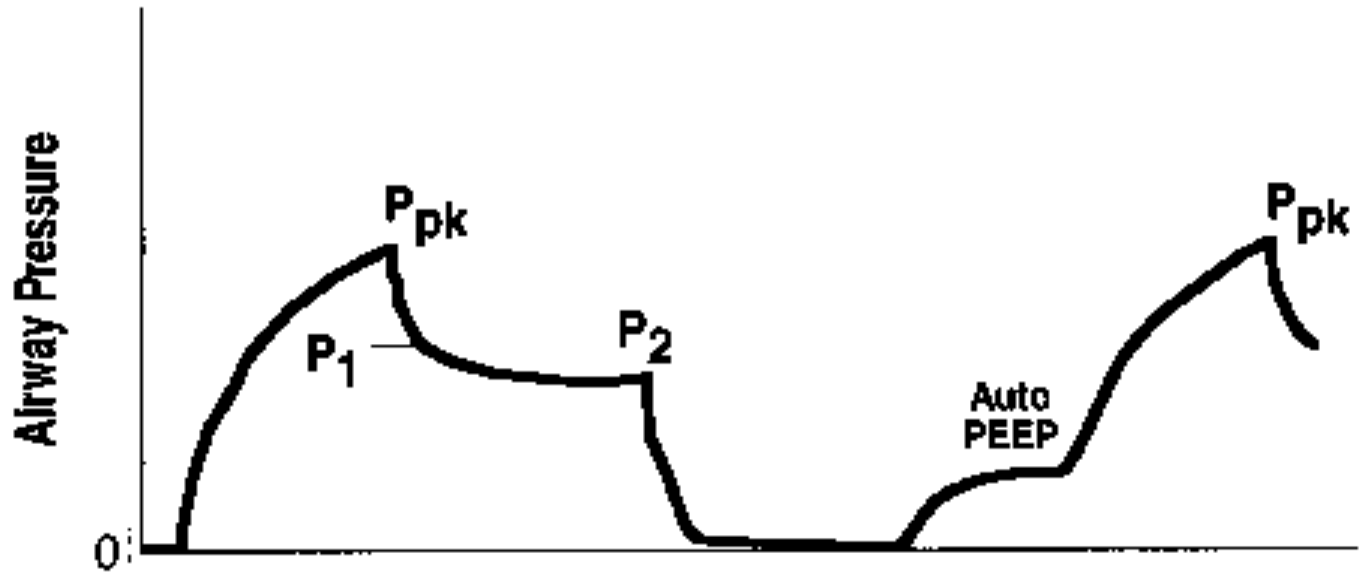

Figure 1. Proximal airway pressure recording during an end-inspiratory airway occlusion and during an end-expiratory occlusion.

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AutoPEEP

Adverse Effects

Effect	*Mechanism*	*Treatment*
"Routine"	*↑ PVR, ↓CO* *↑ Vd/Vt*	*Decrease RR* *Increase Vt/Ti* *Decrease Vt*
Triggering	*Patient has to create a - pressure greater than AP to trigger a MV breath*	*Extrinsic PEEP to = AP*

AutoPEEP
Methods to Reduce

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

"Berlin definition" ARDS

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

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The Berlin ARDS Definition

ARDS Severity	PaO2/FiO2*	Mortality**
Mild	**200 – 300**	**27%**
Moderate	**100 – 200**	**32%**
Severe	**< 100**	**45%**
***on PEEP 5+; **observed in cohort**		

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"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.**

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Pulmonary Function Tests

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

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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

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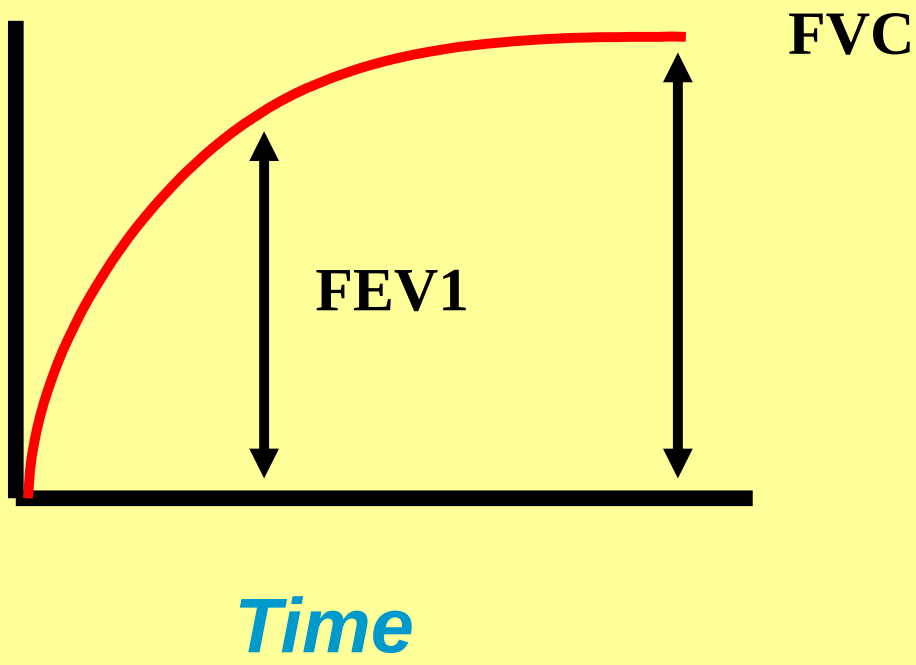
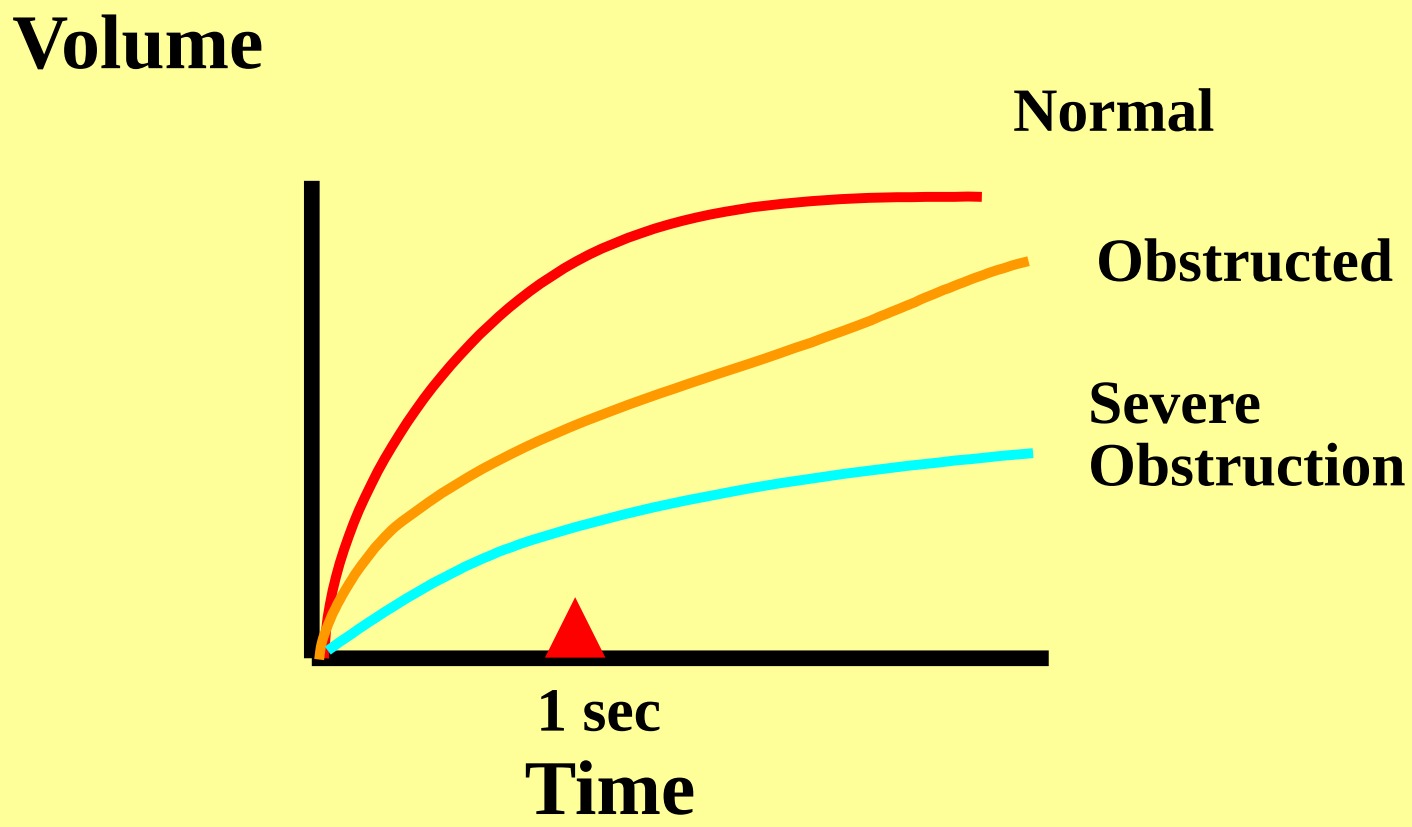
Volume/Time Curves

Definitions



Volume/Time Curves

Obstruction



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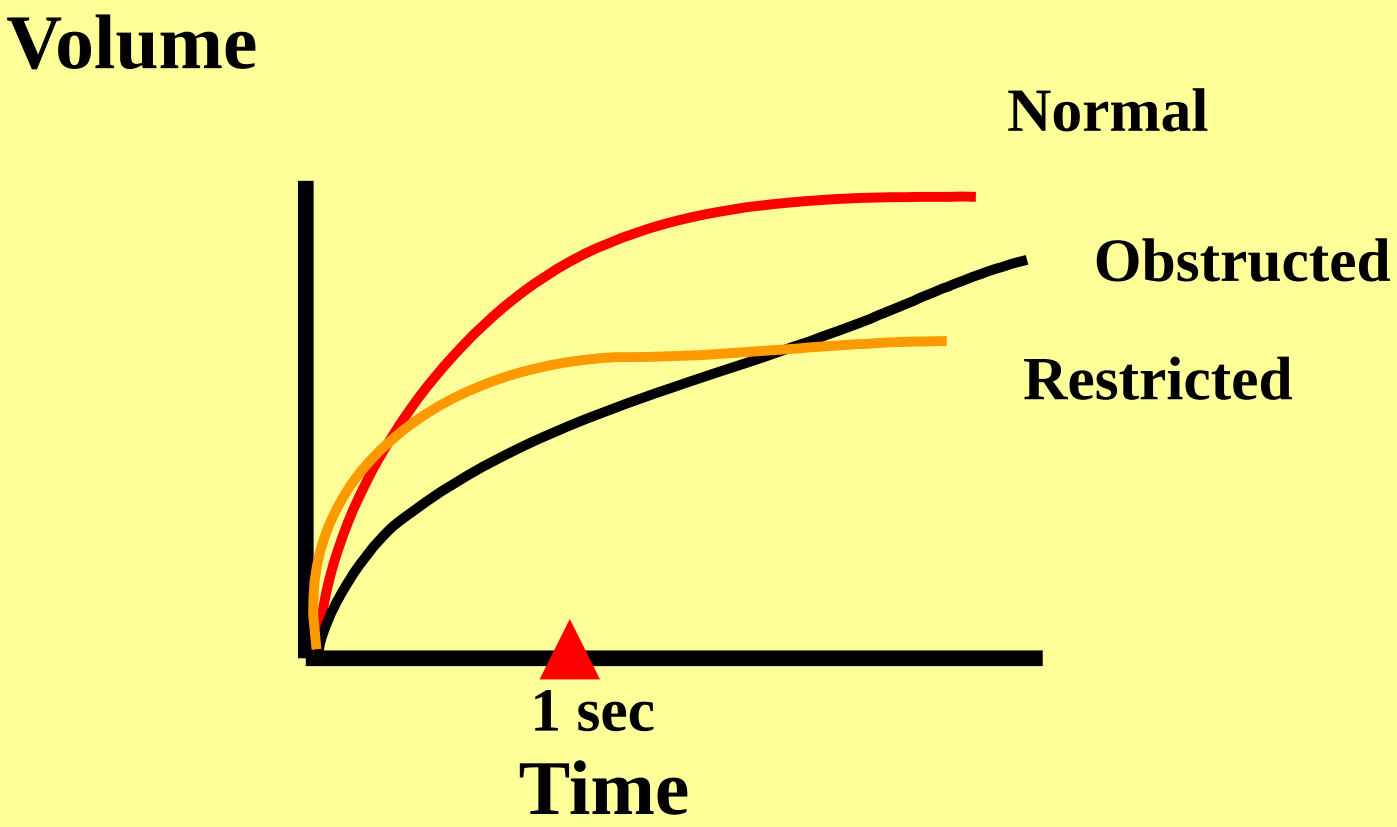
Volume/Time Curves

Obstruction versus Restriction



FEV1 can be reduced by Obst or Rest disease

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Differentiation of Obstruction from Restriction

VARIABLE	*RESTRICTION*	*OBSTRUCTION*
FVC	*Reduced*	*N or Reduced*
FEV1	*Reduced*	*Reduced*
FEV1/FVC	*Normal*	*Reduced*
TLC/RV/FRC	*Reduced*	*N or Increased*

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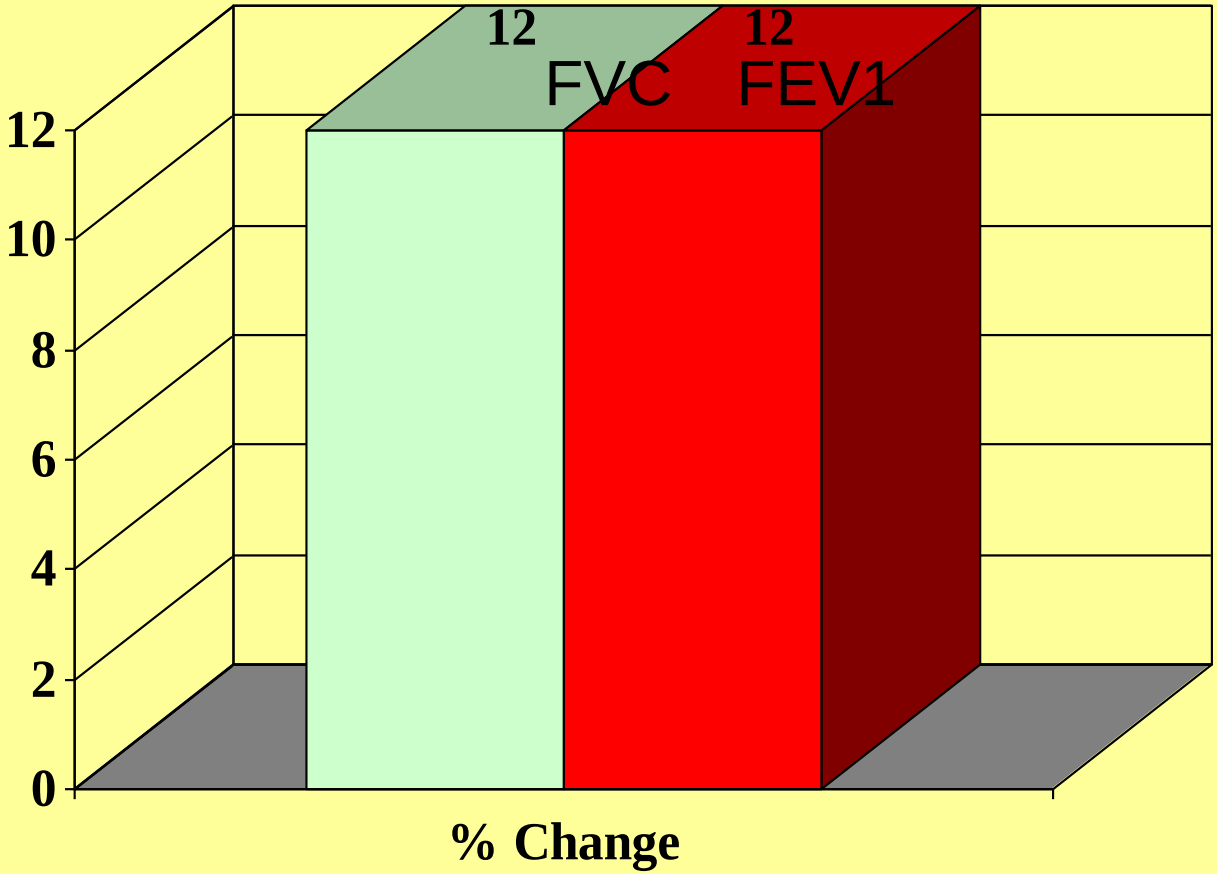
Response to Bronchodilator



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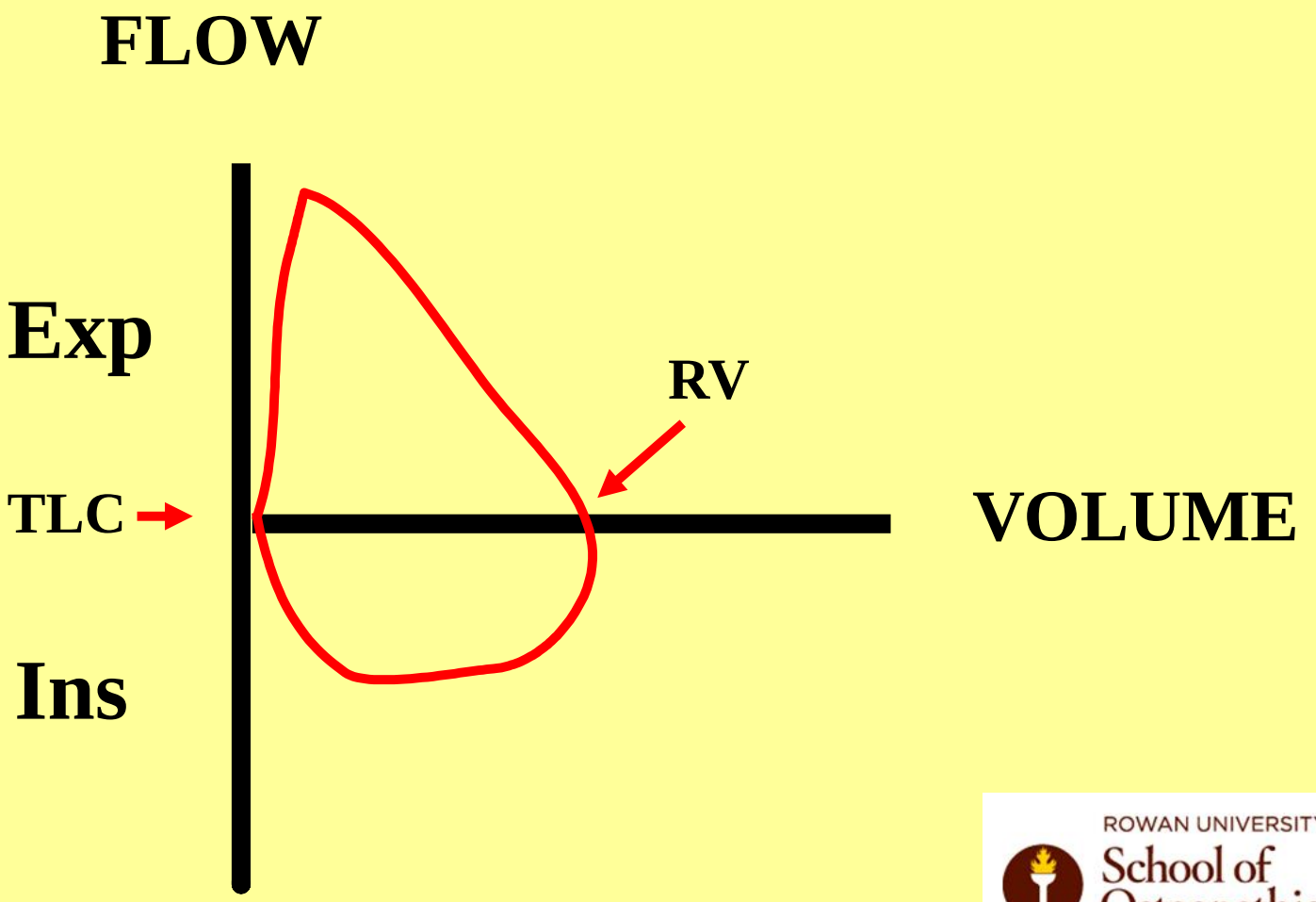
Flow-Volume Curve

Definitions



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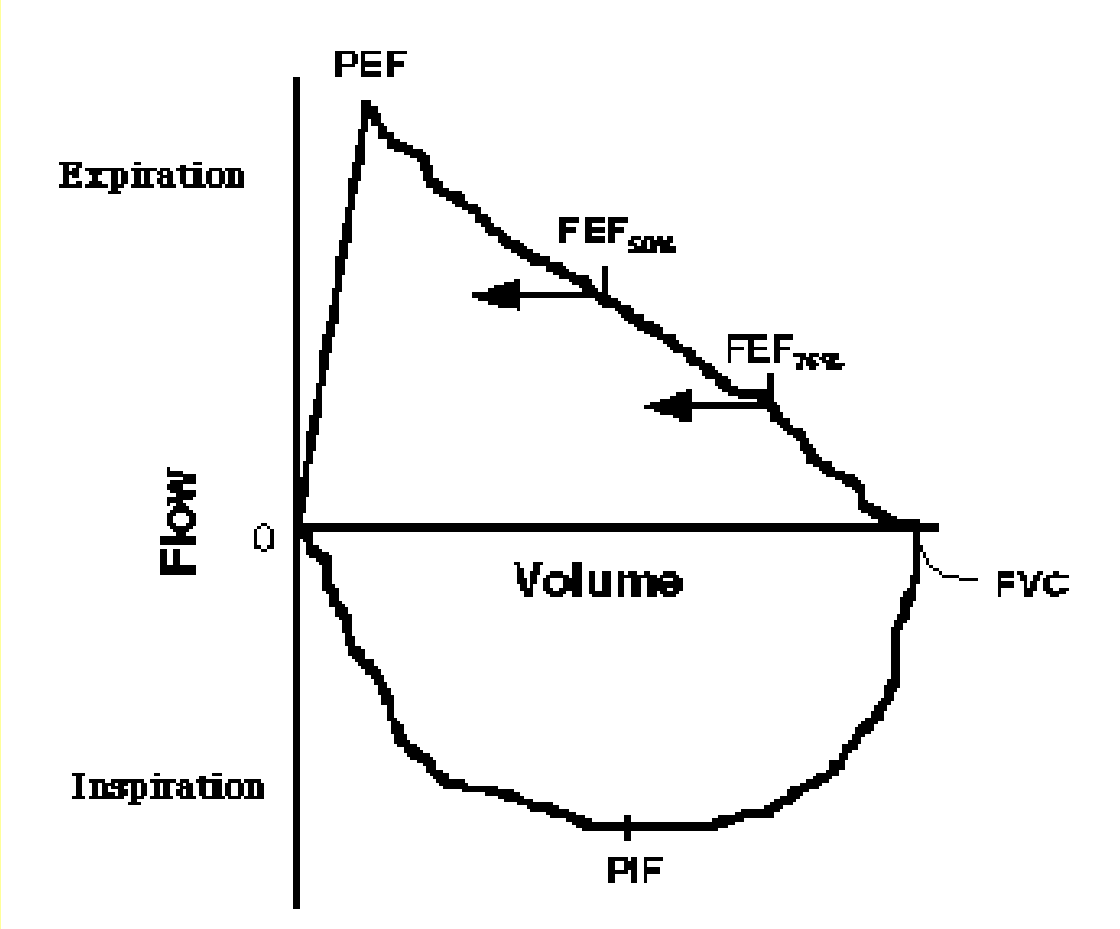
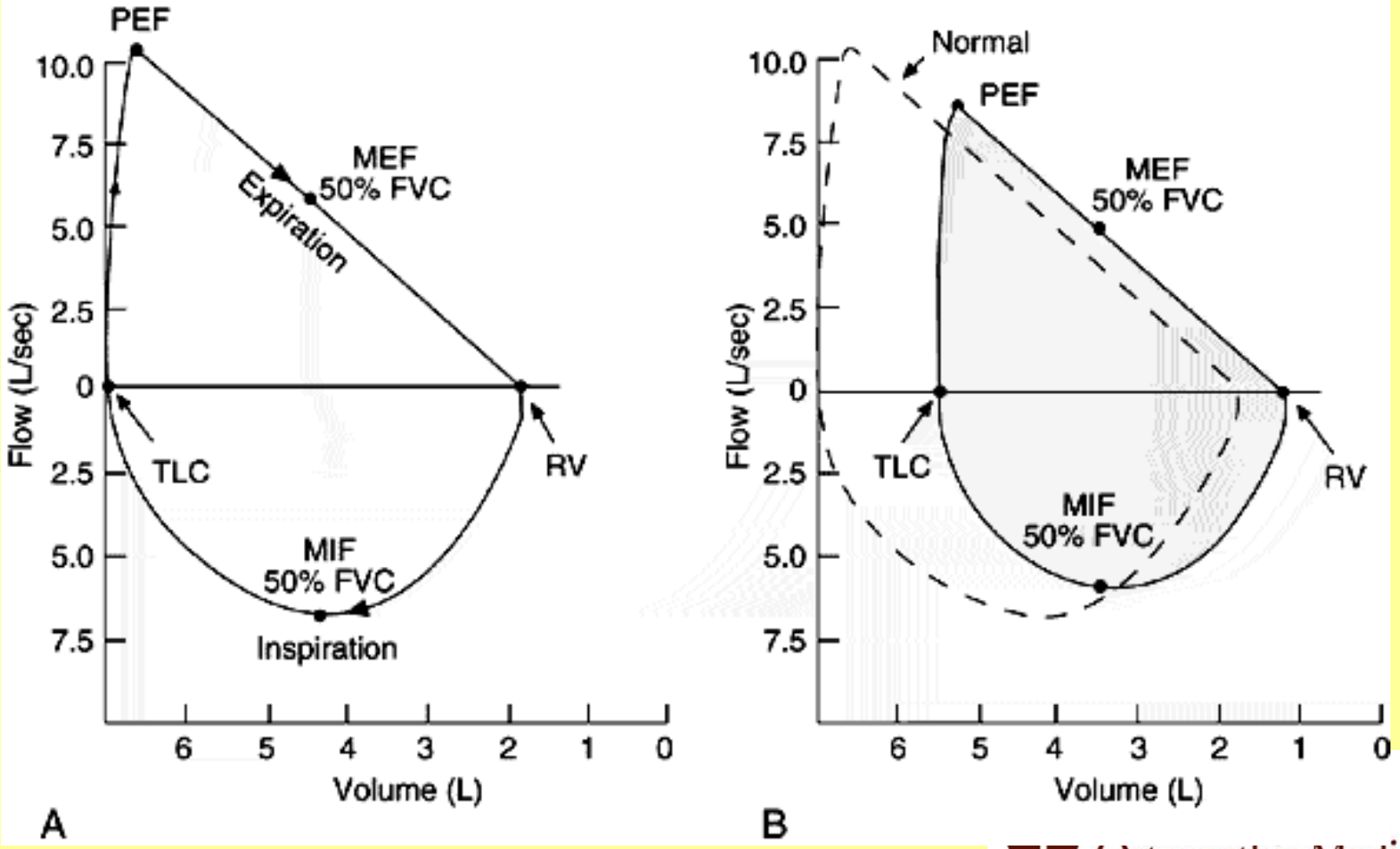
Flow-Volume Loop



Normal and Restrictive FVL



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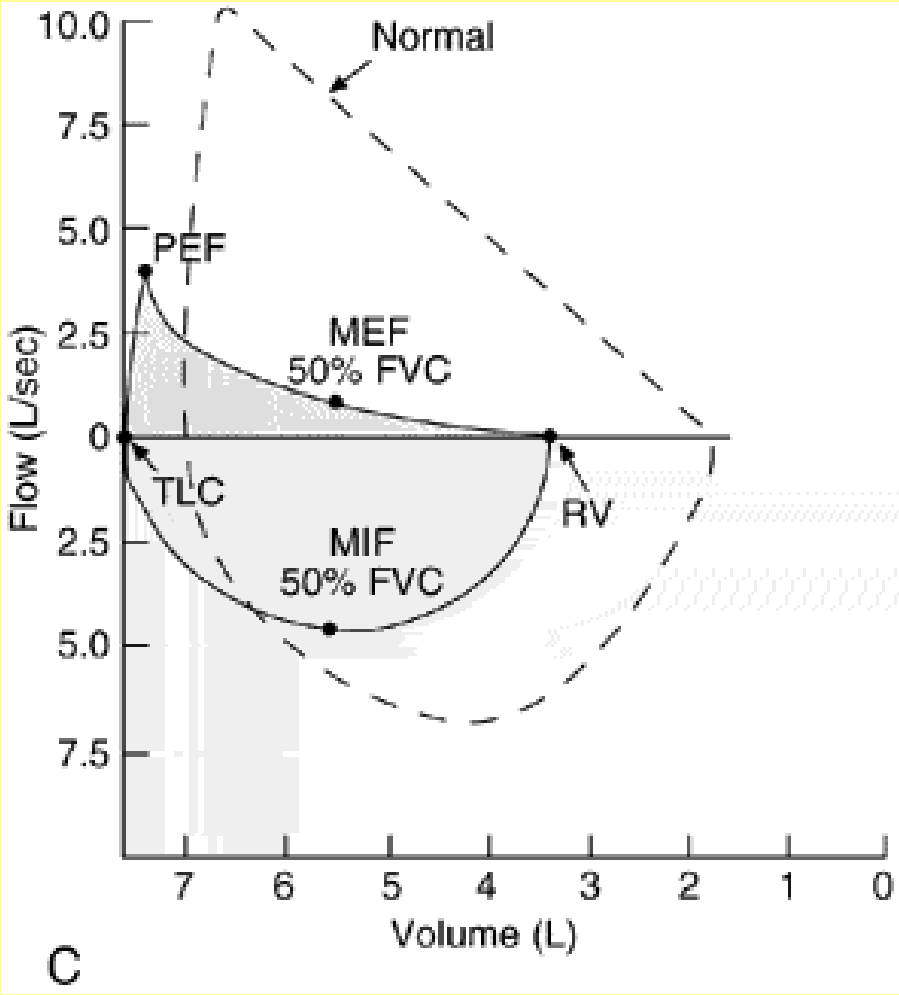
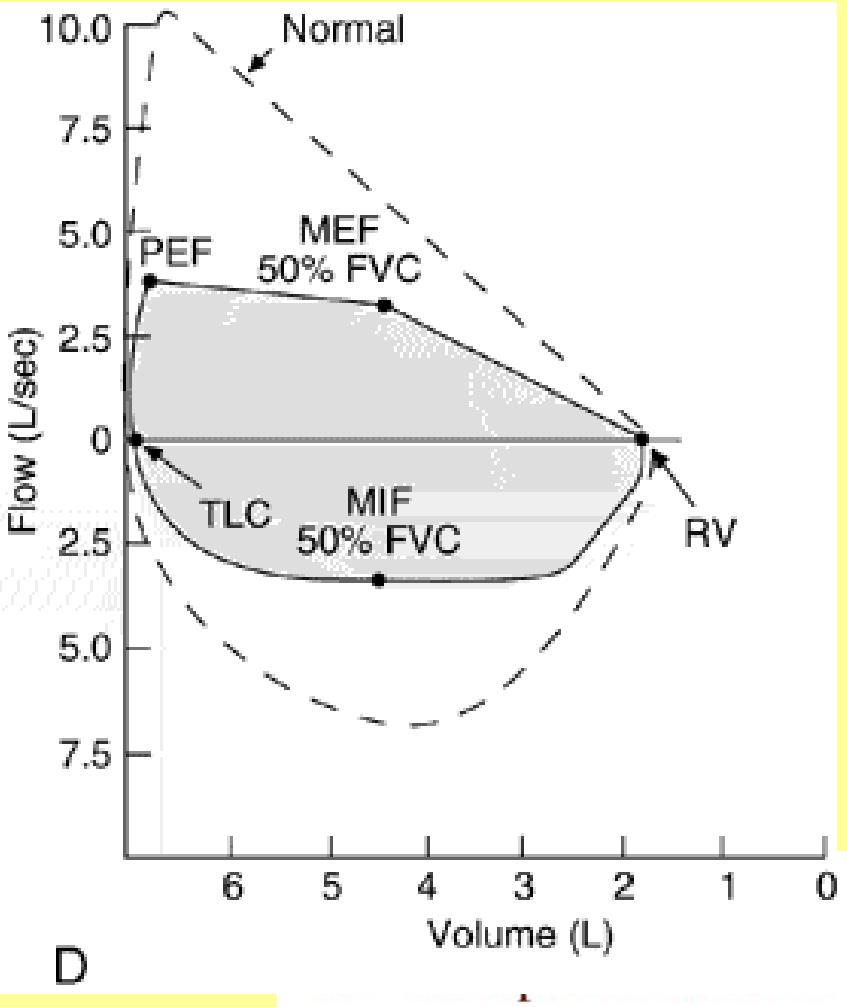
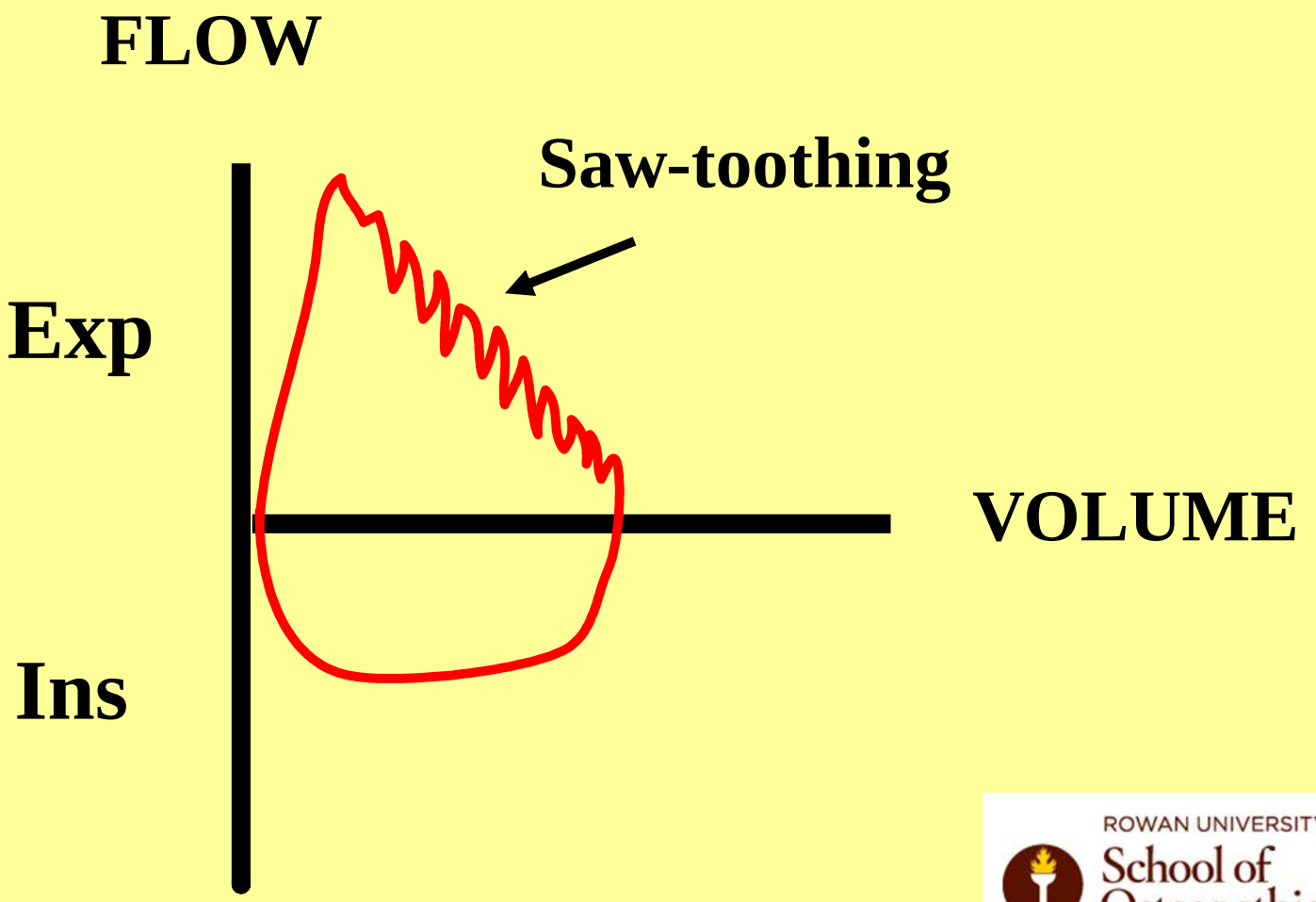
Obstructive FVL





Flow-Volume Curve

Sleep Apnea/ OHS



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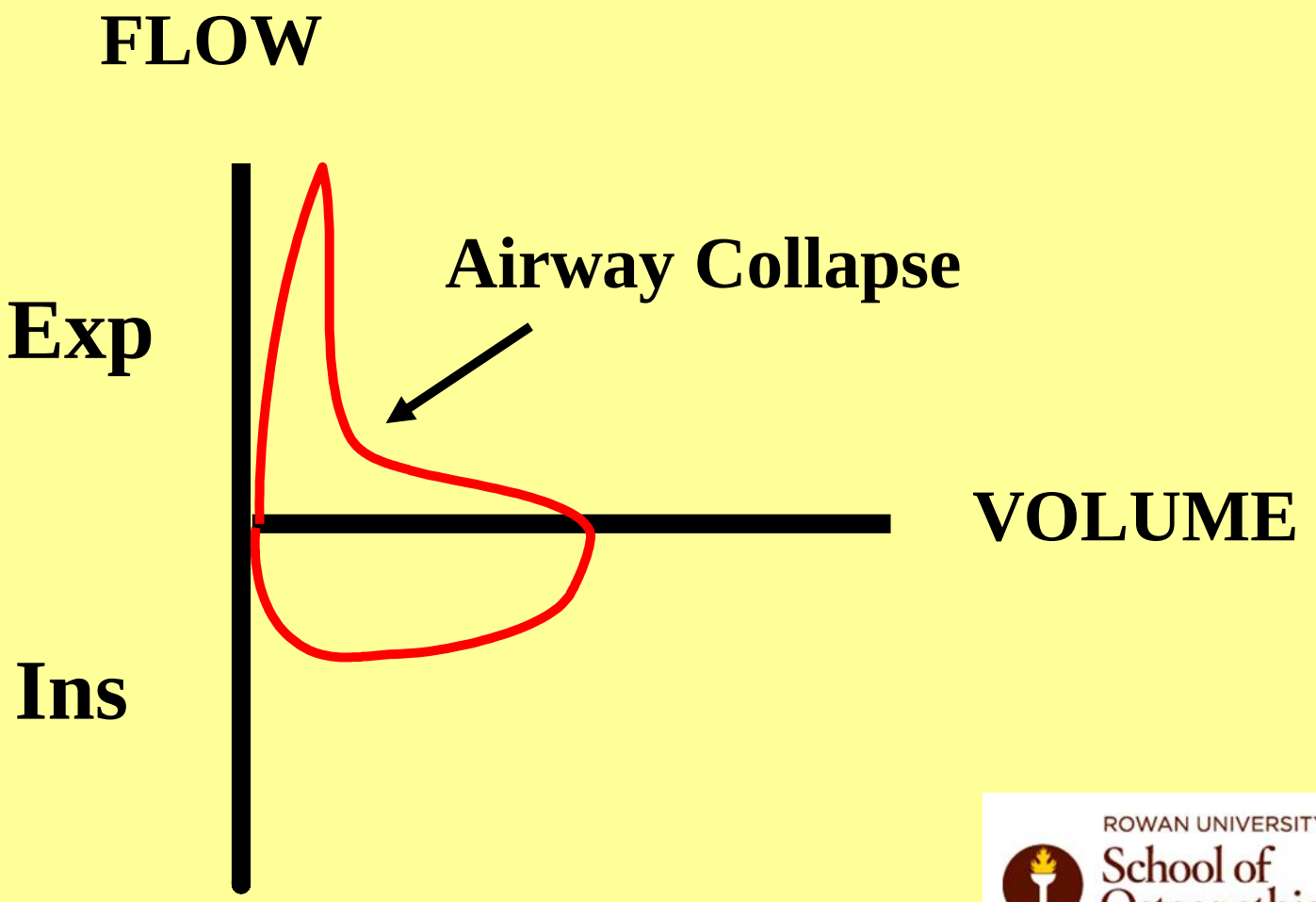
Flow-Volume Curve

Severe Airway Obstruction



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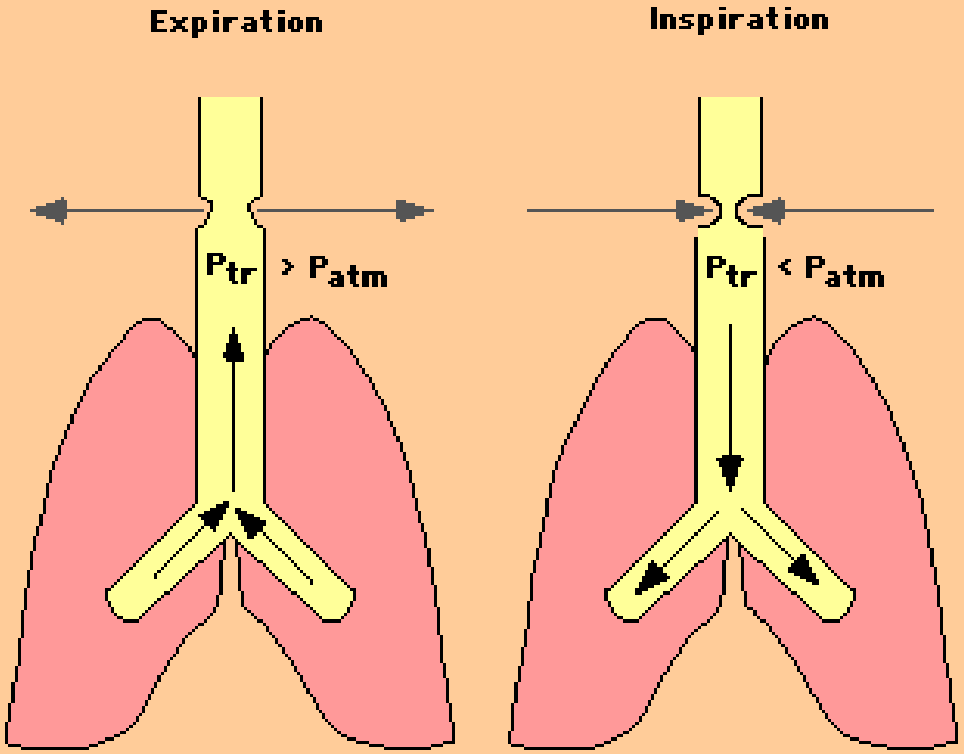
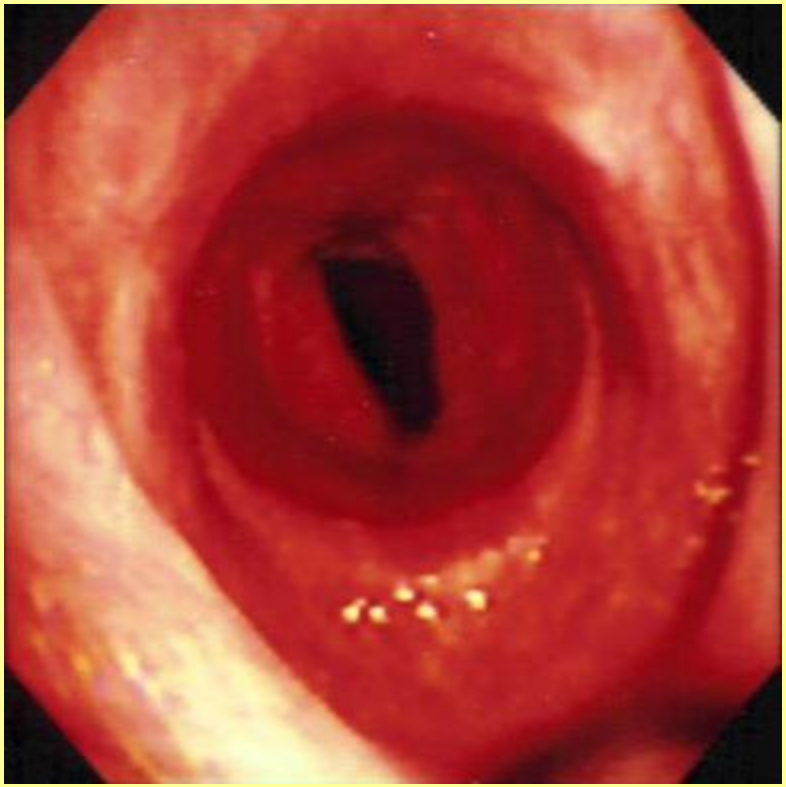
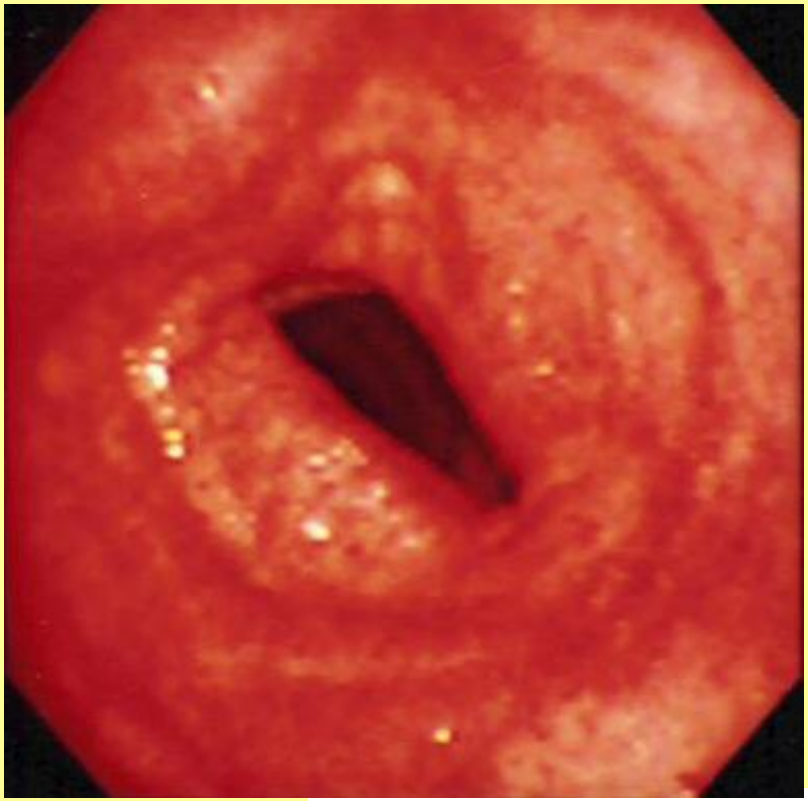


Effect of dynamic extrathoracic airway obstruction Effects of forced expiration and inspiration in dynamic extrathoracic airway obstruction. Left, during forced expiration, intratracheal pressure (Ptr) exceeds the pressure around the airway (Patm), lessening the obstruction. Right, during forced inspiration, 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





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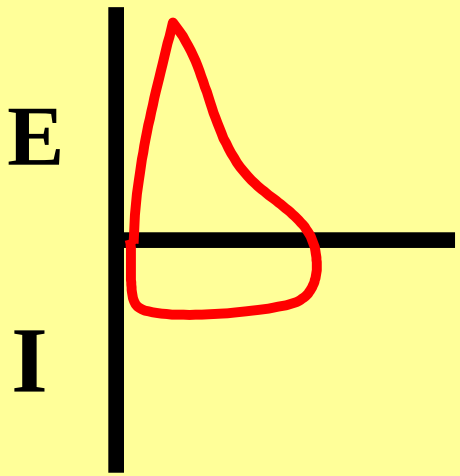
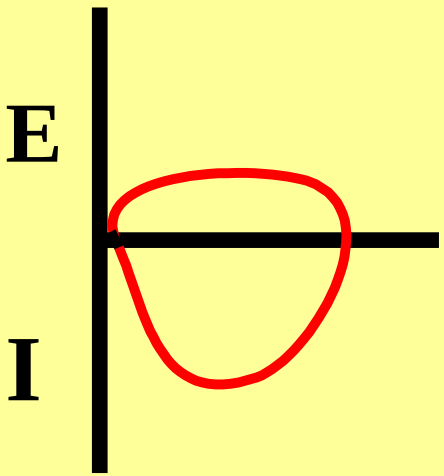
Intra and Extra Thoracic Obstructions

VARIABLE



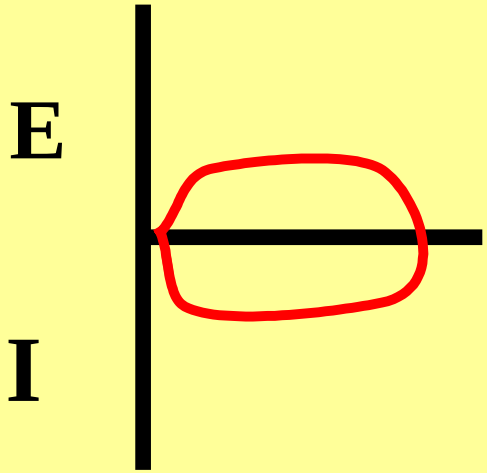
Extrathoracic

VARIABLE



Intrathoracic

FIXED



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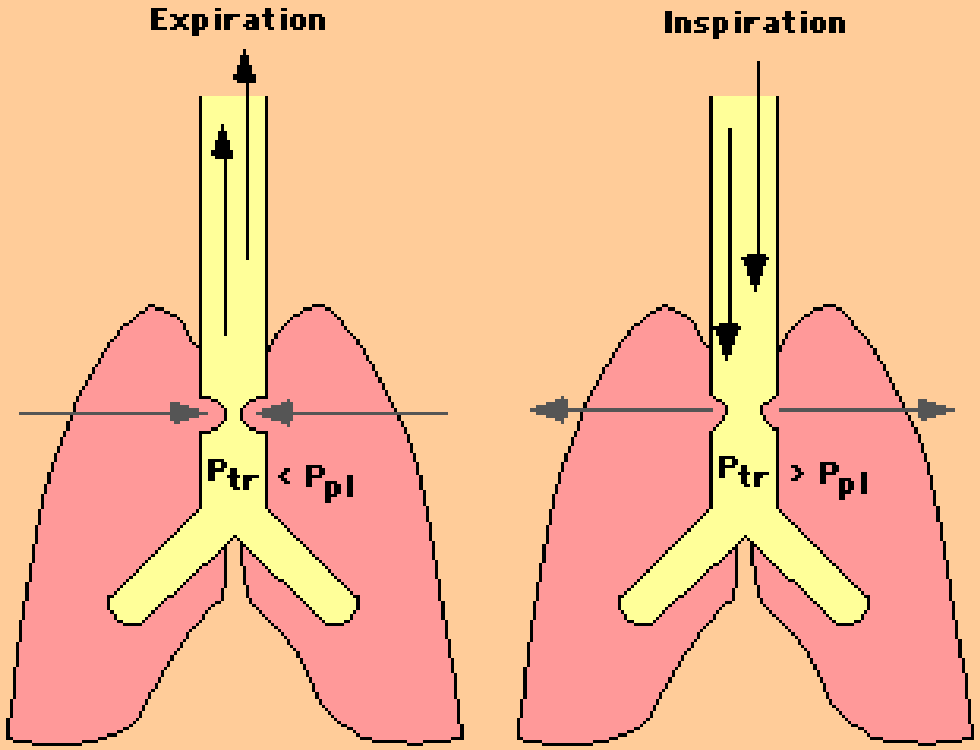
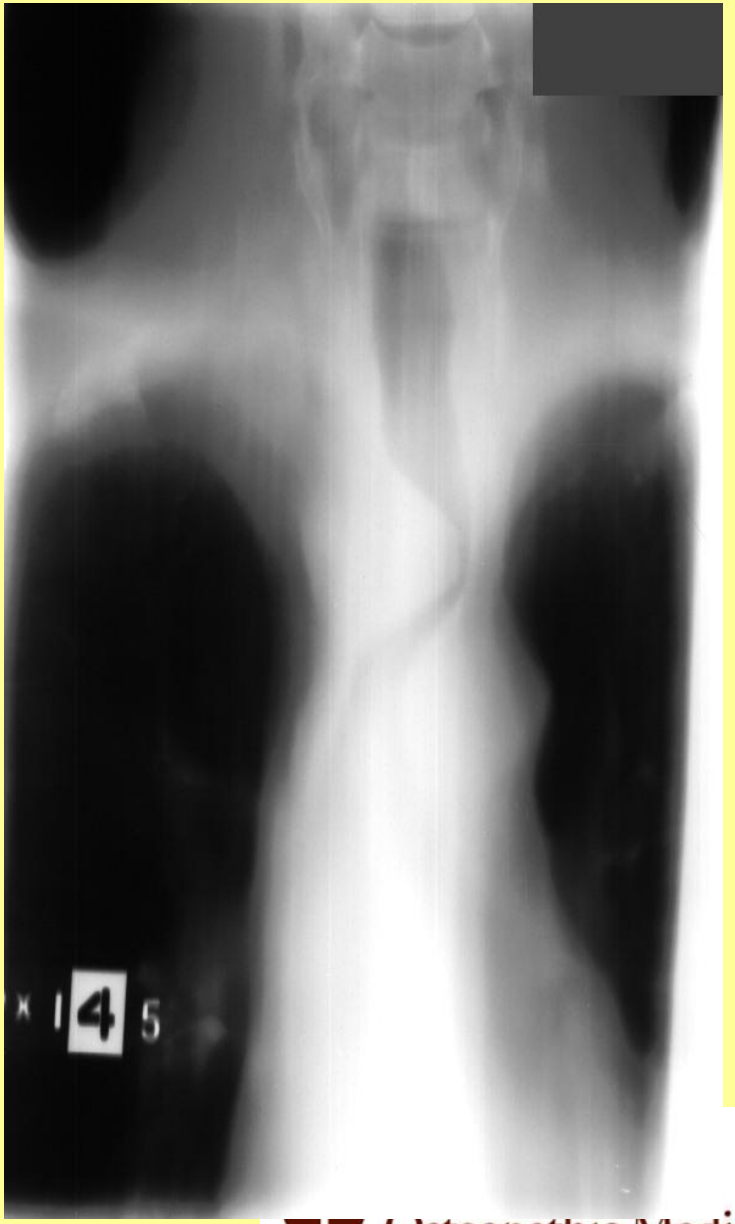


Effects of dynamic intrathoracic airway obstruction Left panel, during forced expiration, the intrathoracic intratracheal pressure (Ptr) is less than the pressure in the pleural pressure (Ppl), worsening the obstruction. Right, during forced inspiration, intratracheal pressure exceeds the pleural pressure,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

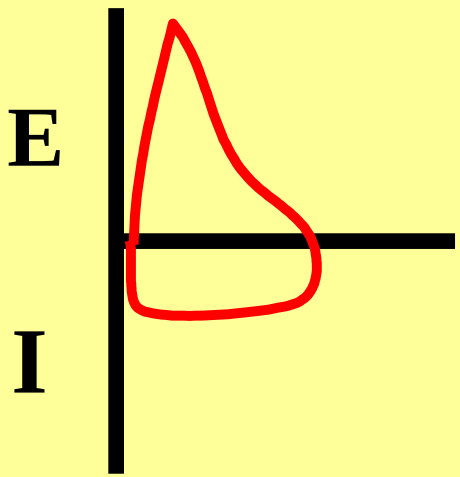
Intrathoracic

Tracheal Compression



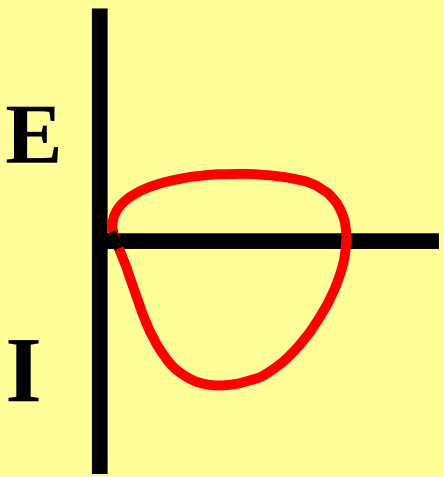
Intra and Extra Thoracic Obstructions

VARIABLE



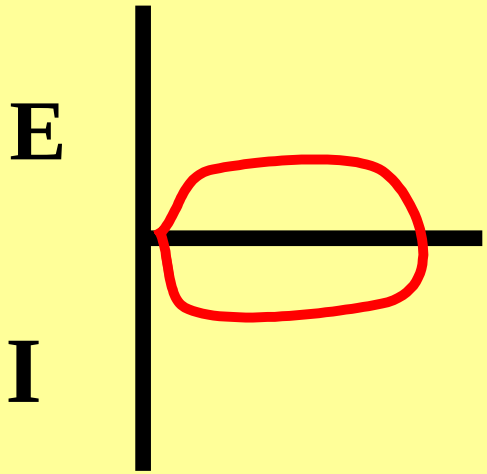
Extrathoracic

VARIABLE



Intrathoracic

FIXED



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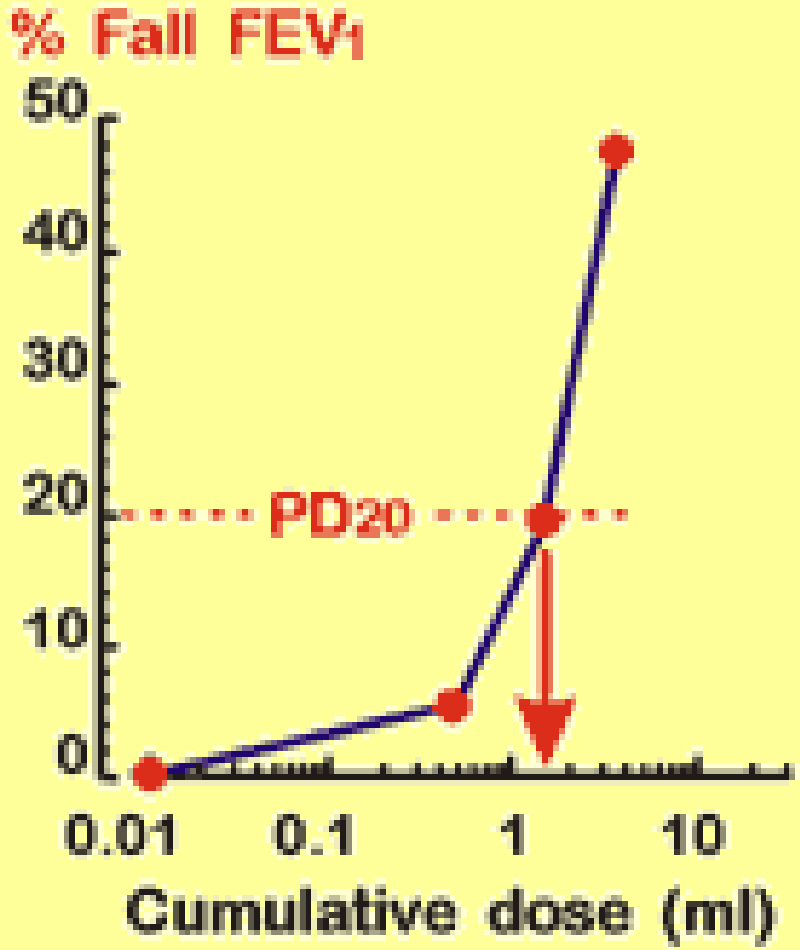
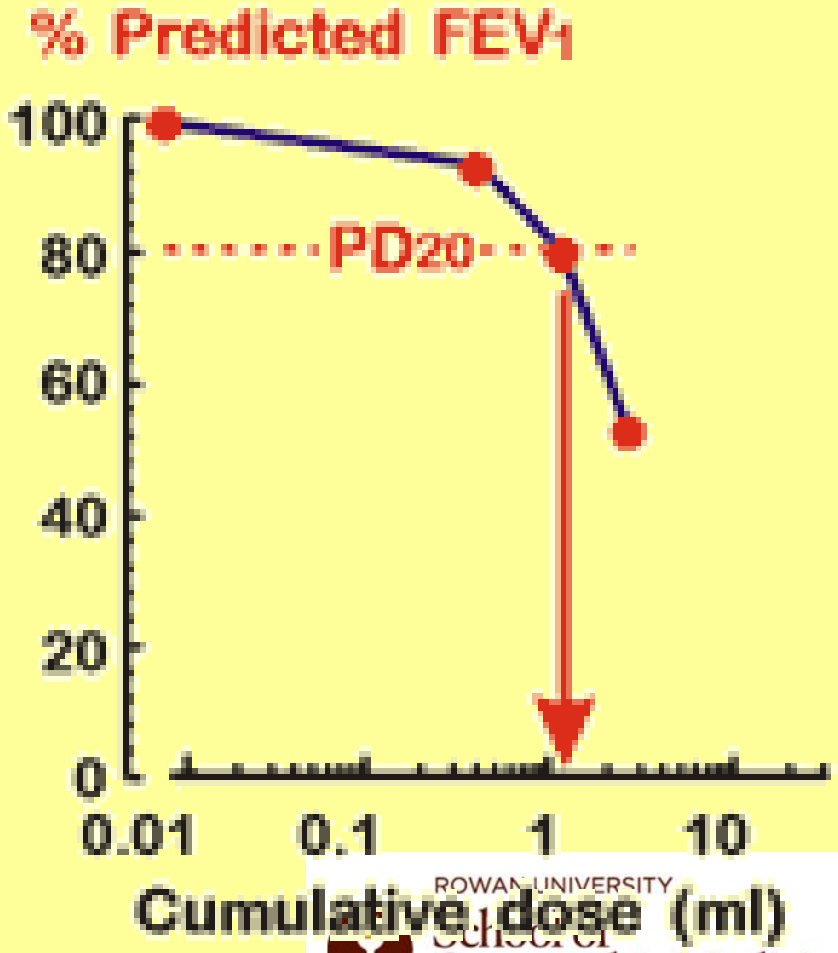
Bronchial Provocation Testing





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

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