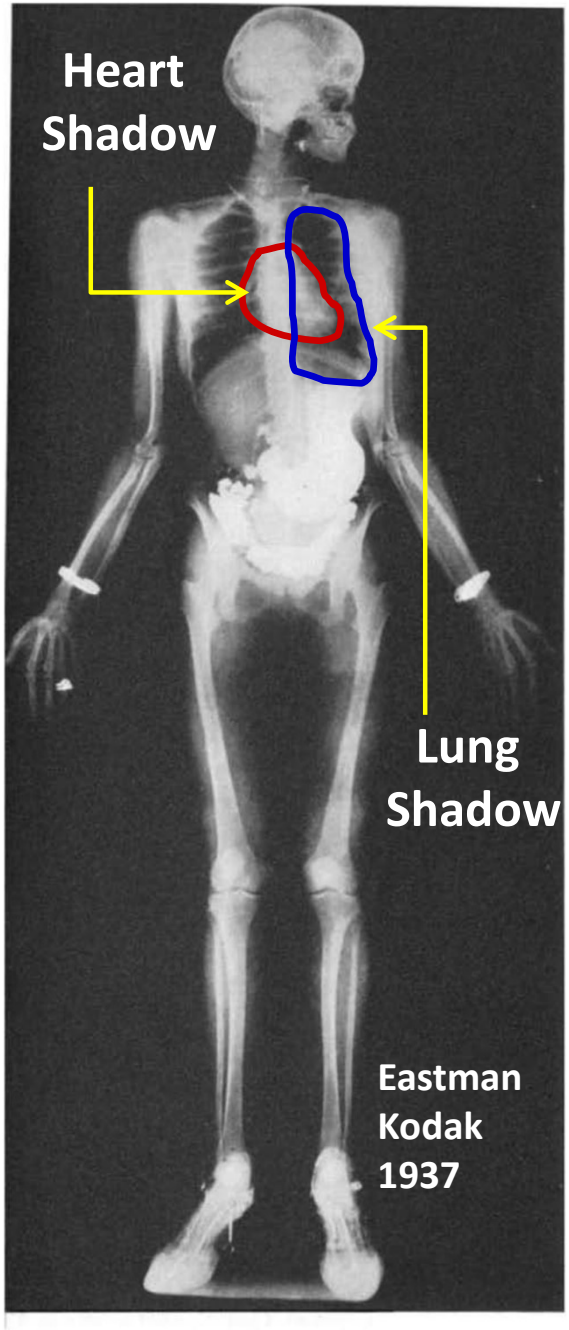
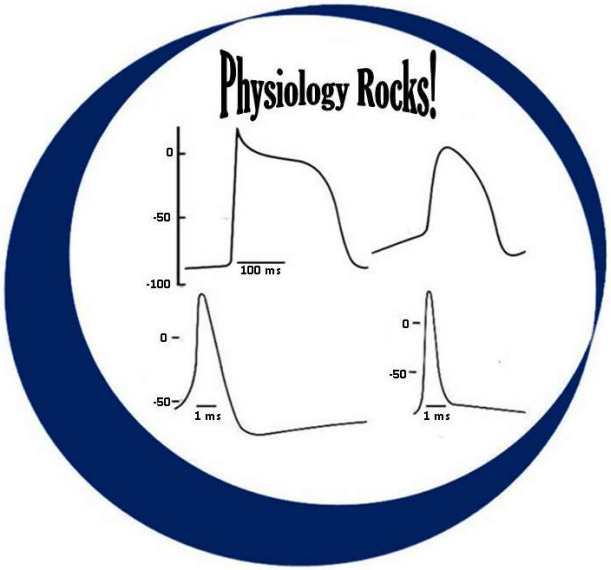



# Lecture 36
# Respiratory System Physiology

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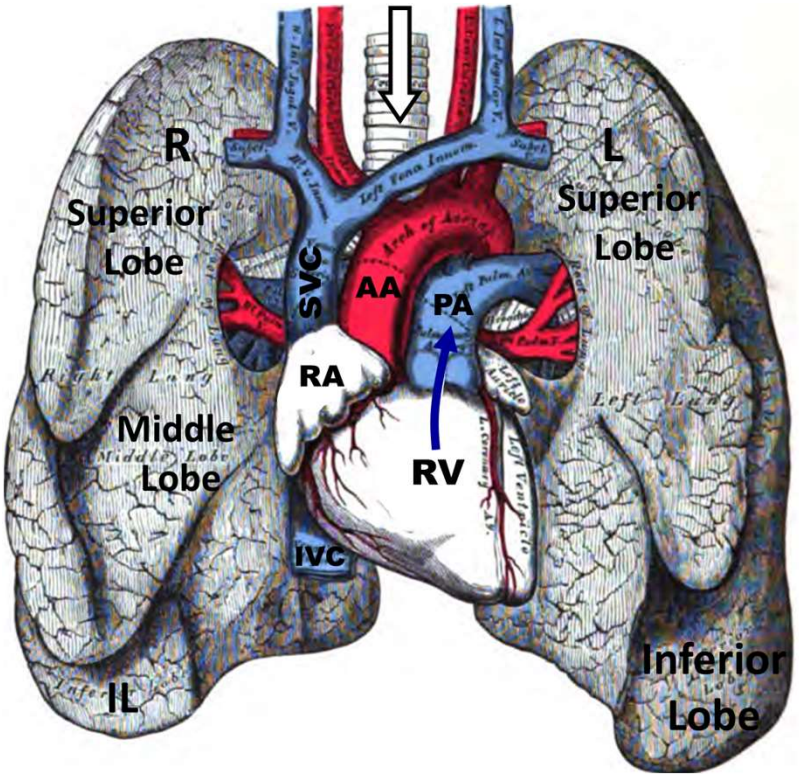
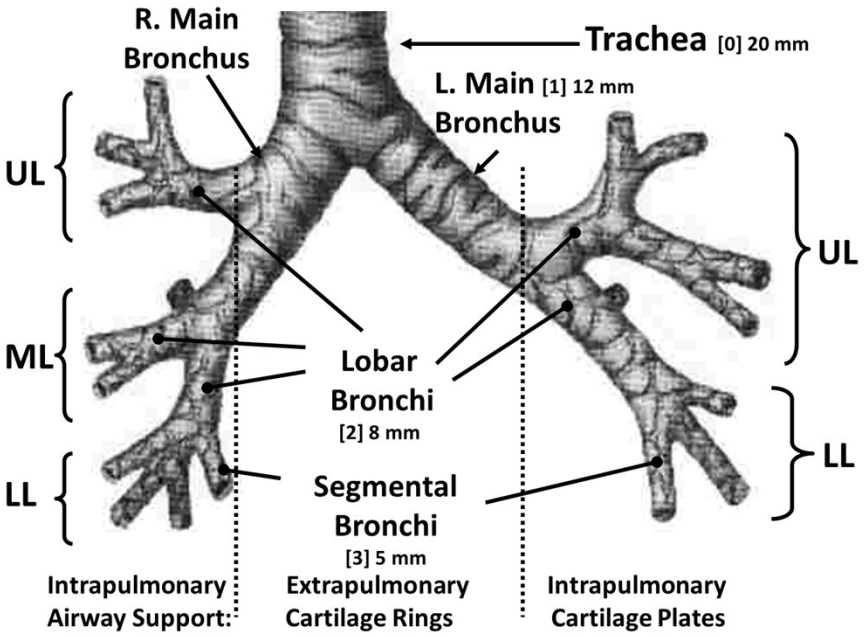
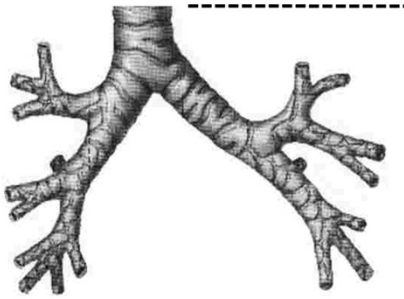
# Anatomical – Structural Considerations

# Lung Lobes and Conducting Zone Airways



- Superior and inferior lobes also called upper (UL) and lower (LL) – Lung Volume ~ 6L
- Three lobes on right – two on left
- Trachea= Main airway-branching order zero [0] Has cartilage rings with muscle providing structural support – occlude @ 50mmHg
- Near dichotomous branching with orders 1-16 part of the conducting zone
- Orders 17-23 part of the respiratory zone

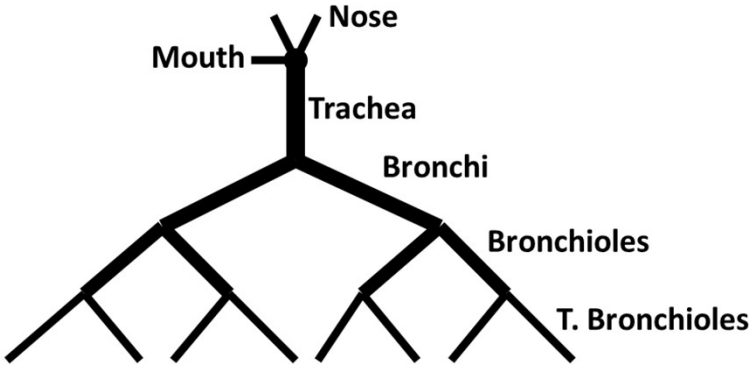
[4] 3.5 mm
Small Bronchi
[11] 1.0 mm
Bronchioles (No cartilage smooth muscle)
[16] 0.5 mm
Terminal Bronchioles (Last airway of Conducting Zone)

Conducting Zone
No Gas Exchange
Anatomical Dead space
- Air Conduction
- Humidification
- Warming

1. Airways imbedded in lung parenchyma Held open by elastic forces - lung volume dependent
2. Increased number of mucus-secreting goblet cells

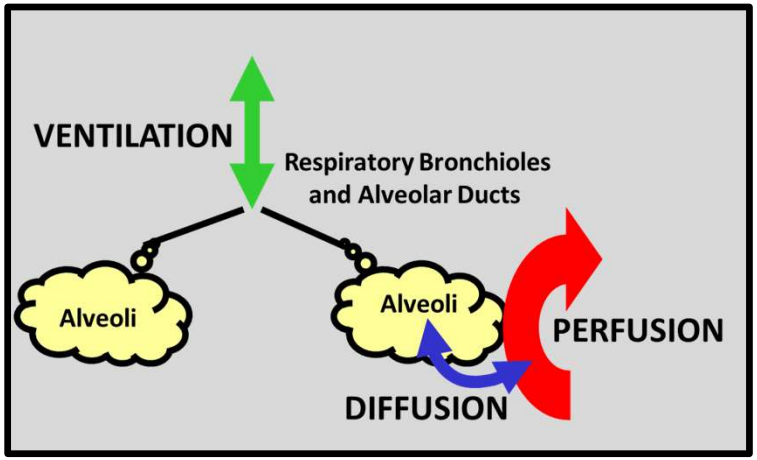
# Schematic Overview

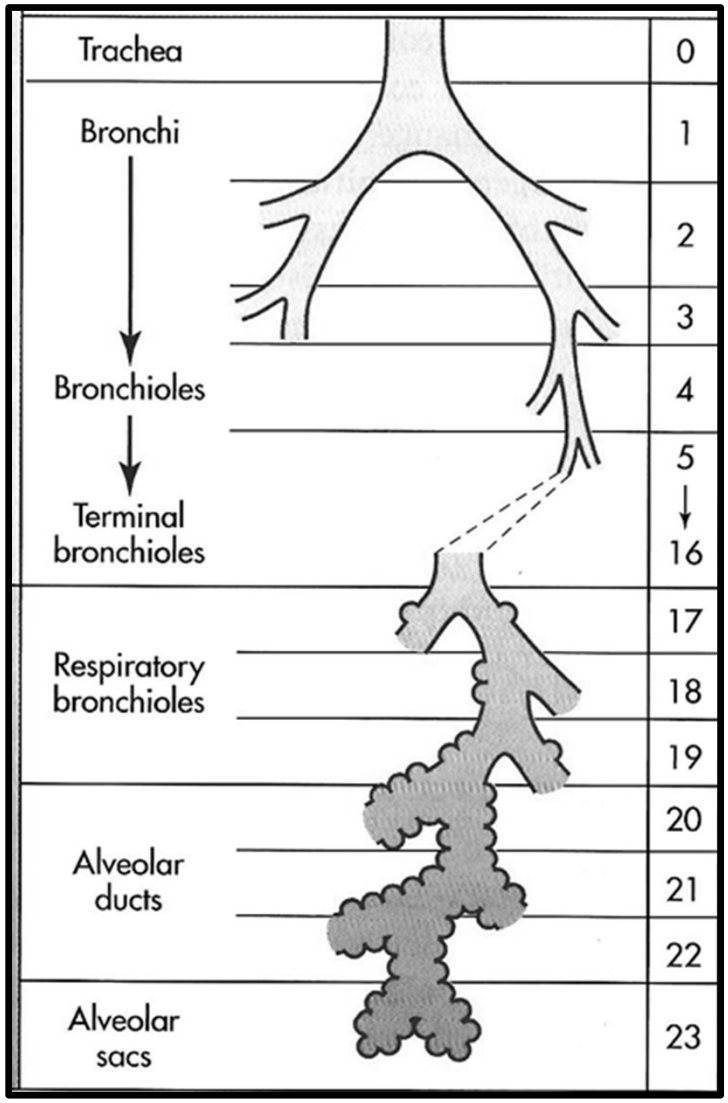
***Conducting Zone***
**No Gas Exchange**
***Dead Space***



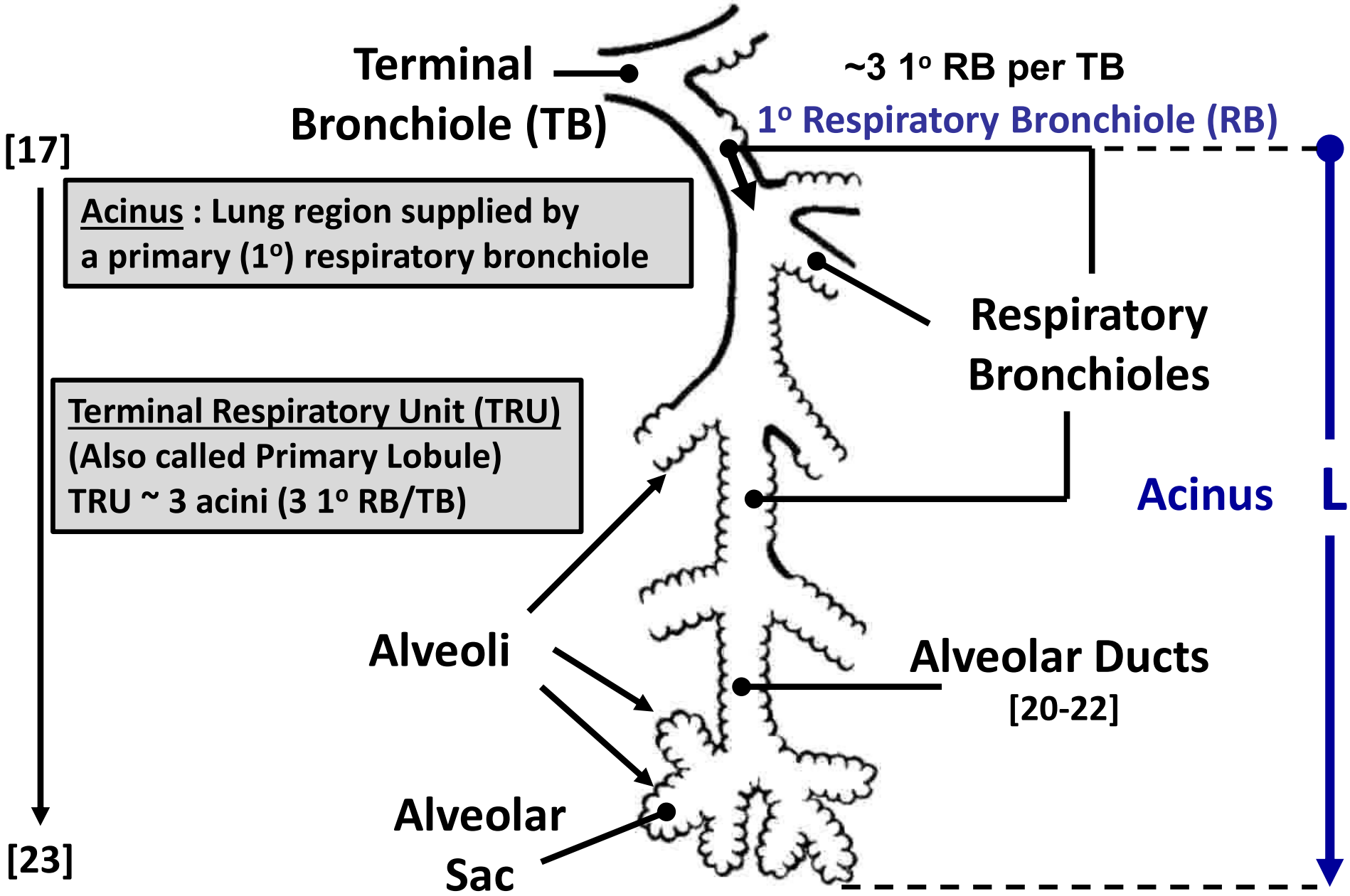

***Transition Zone***

***Respiratory Zone***
**Gas Exchange**

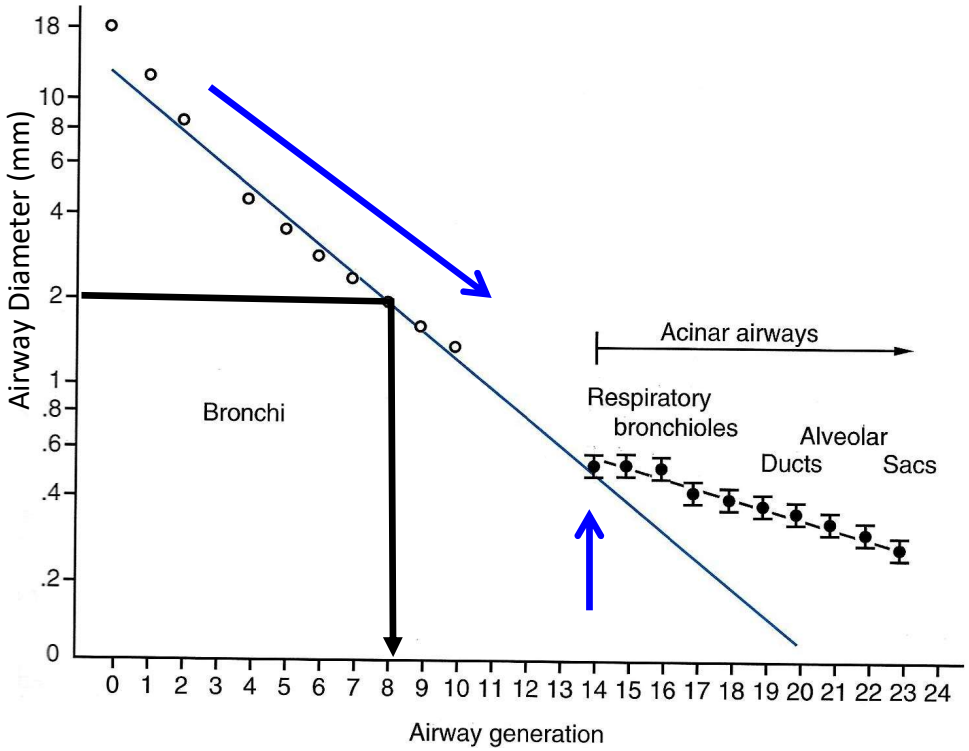


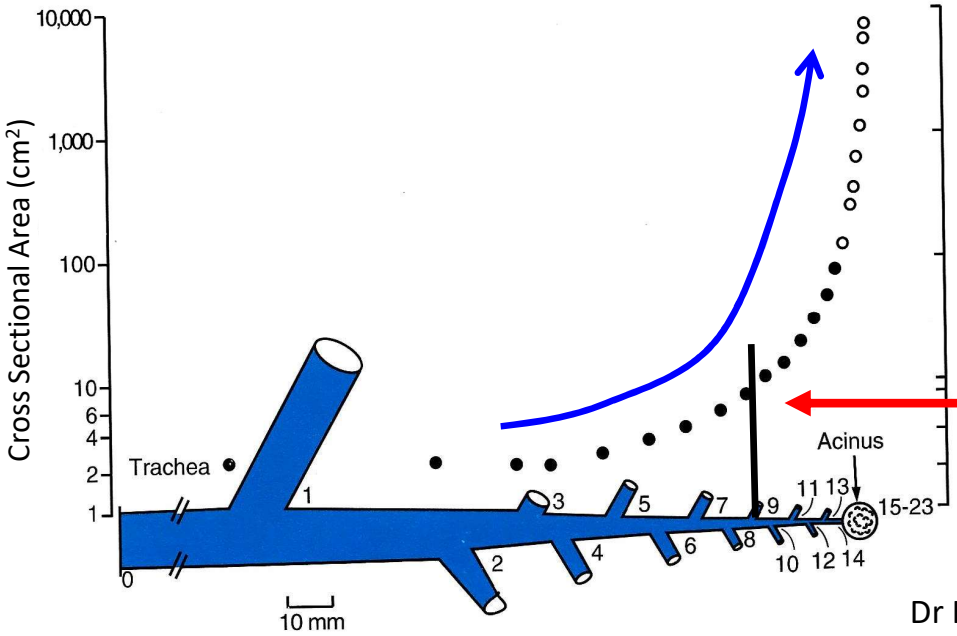



# The Respiratory Zone



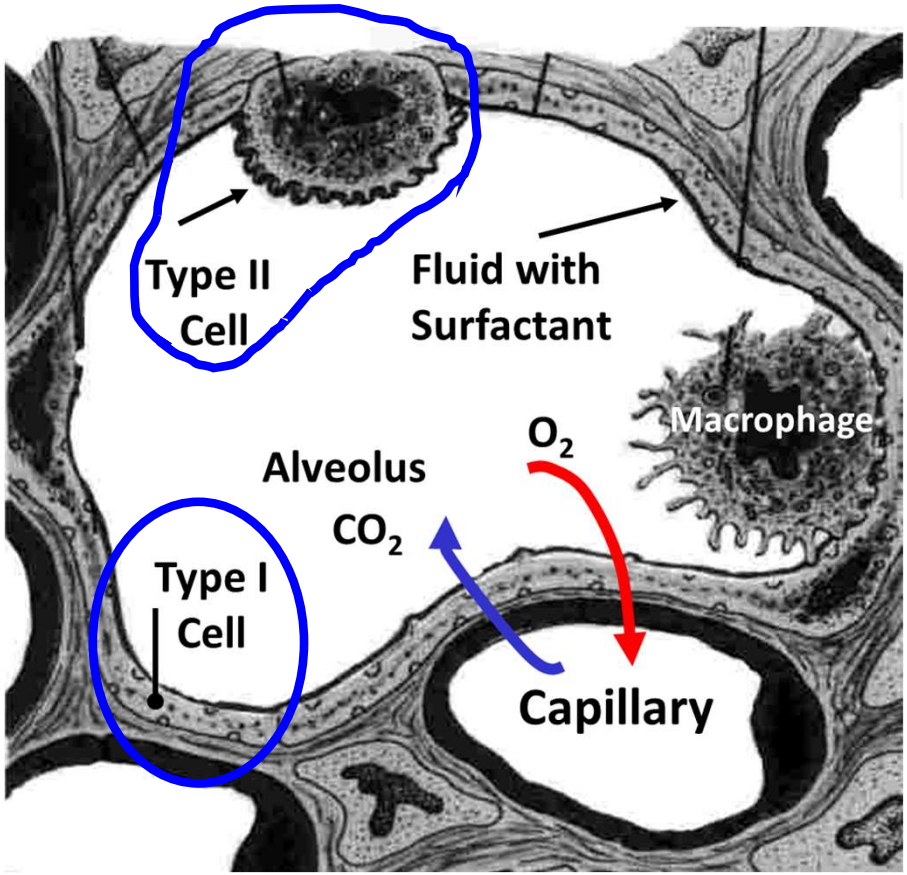
# Airway Metrics: Summary



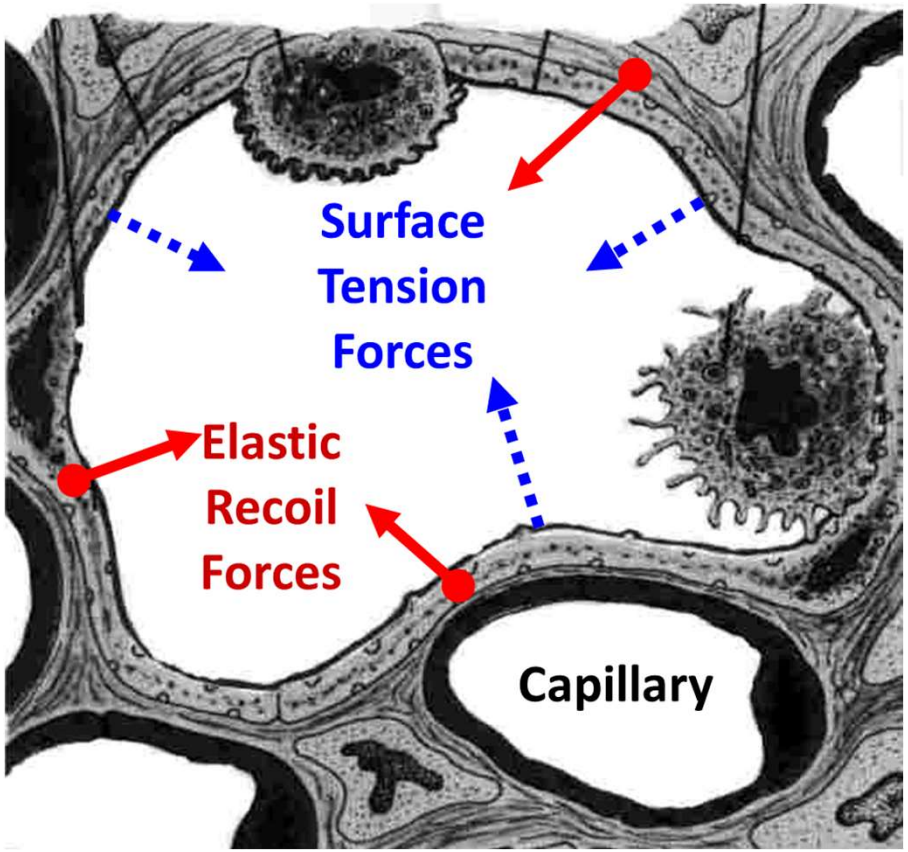

- **Diameters decrease with increasing generation**
- **Rate of decrease transitions at start of respiratory bronchioles with much less change**
- **Despite decrease in diameter the effective cross sectional area increases dramatically toward the respiratory zone**
- **At terminal bronchiole level cross-sectional area is much greater than in large bronchi**
- **As a consequence, resistance to air flow in small airways (< 2 mm) is ≈ 10% of total**

# Alveoli




- Number of alveoli varies by person's height with a range of ~ 200-600 million (Avg=300 million)
- Size (volume) is ~ to lung volume
- Volume less toward base compared to apex in the dependent lung (gravity effect)
- Actual effective diameter at functional residual capacity (FRC) is ≈ 0.2 mm
- Type I alveolar epithelial cells → tight junctions prevents protein (e.g. albumin) from entering alveoli but macrophages and granulocytes pass via chemotactic stimuli
- Type I cells are particularly sensitive to injury from high levels of $O_2$
- Type II cells are progenitors of Type I cells and if Type I are destroyed can replace
- Type II cells contain a special lung surfactant that is released and distributed at the alveolar-tissue interface

# Alveolar Inward Forces

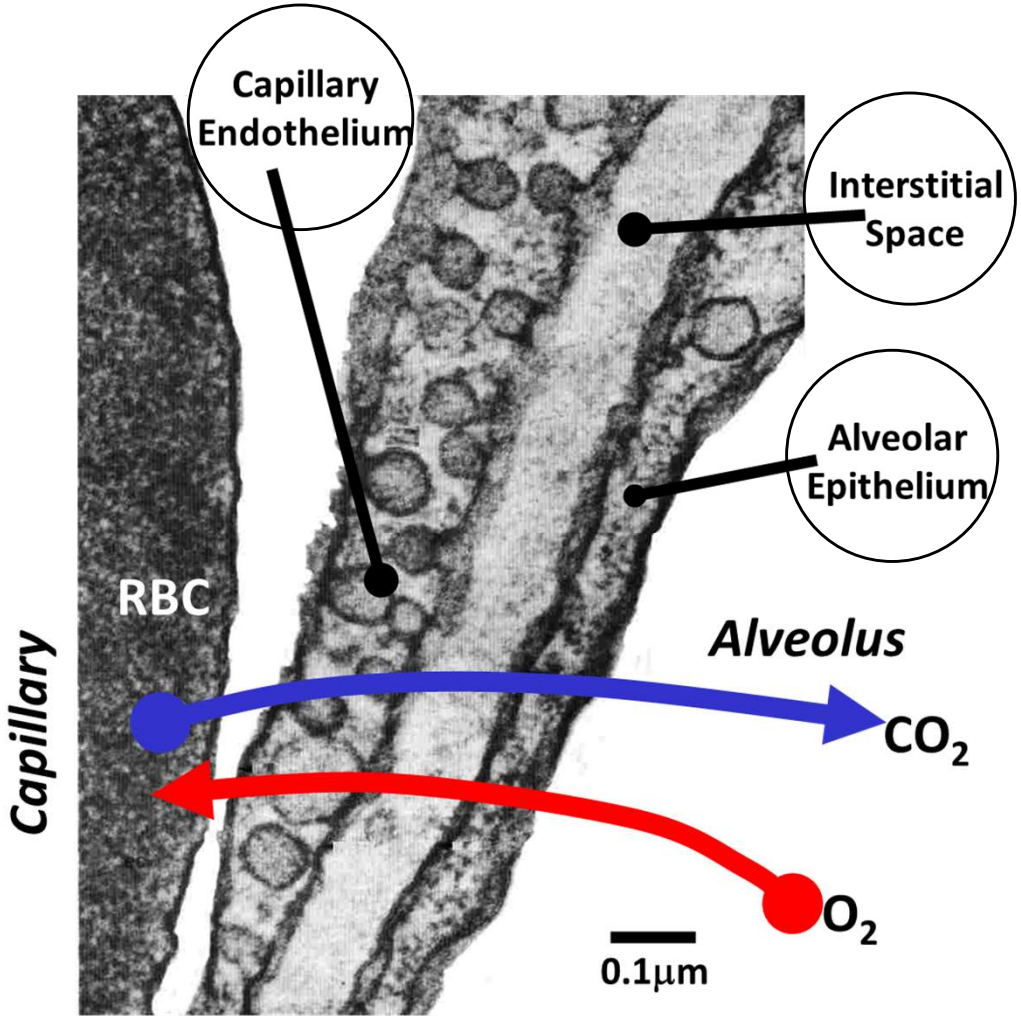



- There are two inwardly directed forces
- An elastic recoil force due to alveoli stretch

  Similar to a stretched rubber band or spring
- A surface tension force at the interface

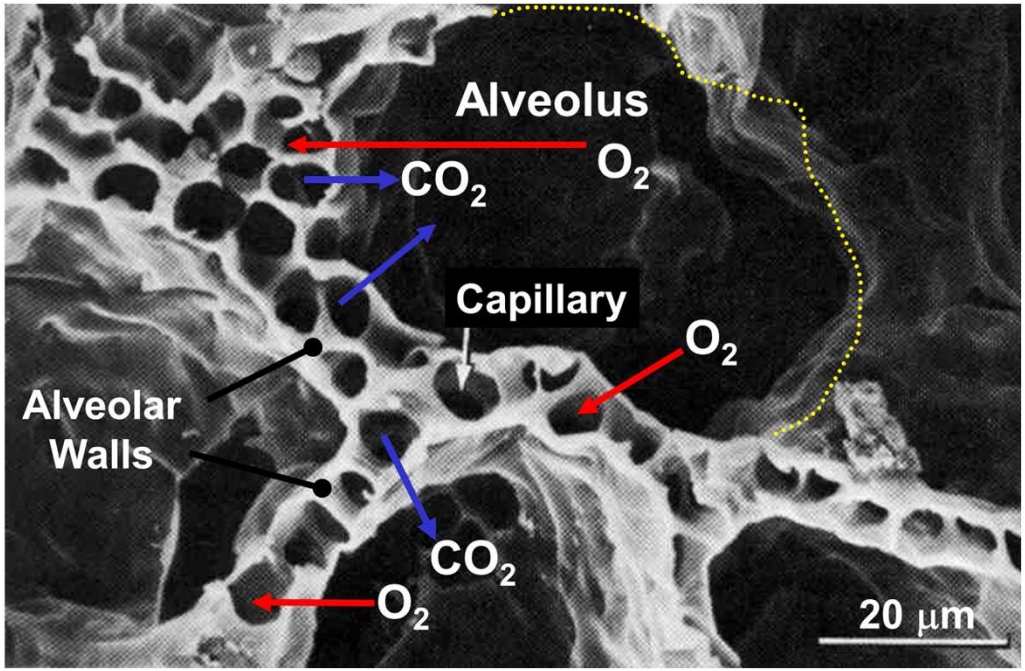
  between moist tissue and gas in the alveolus
- Both forces tend to close alveoli (atelectasis)

# Gas Exchange "Sandwich"

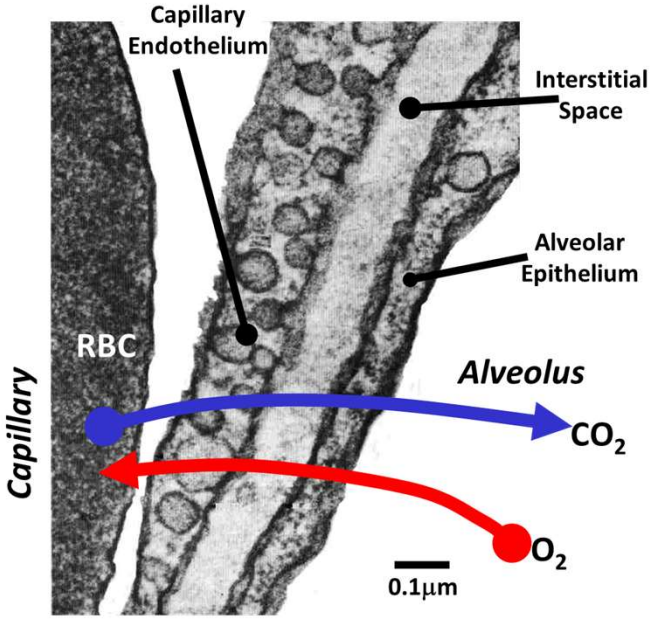



- $O_2$ and $CO_2$ pass through a thin membrane "sandwich"
- Increased pathlengths reduce the ability of gasses to properly diffuse to their targets
- Structural and compositional changes also effect also effect the diffusion properties of the gasses toward their intended targets

# Gas Exchange Interface




- Multiple capillaries surround each alveoli and are imbedded within the supporting structures
- Not all capillaries experience active blood flow at a given time and can be recruited as needed
- Distance traversed by $O_2$ and $CO_2$ is the thickness of "sandwich"
- Normally this distance is ≈ < 0.5μm



# Functional Aspects

# Respiratory Processes: Ventilation-Perfusion-Diffusion

[O2] = 0.21 → $FIO_2$
[CO2] = 0%
[N2] = 0.79
Gas fractions at sea level

$P_{ATM}$=760 mmHg

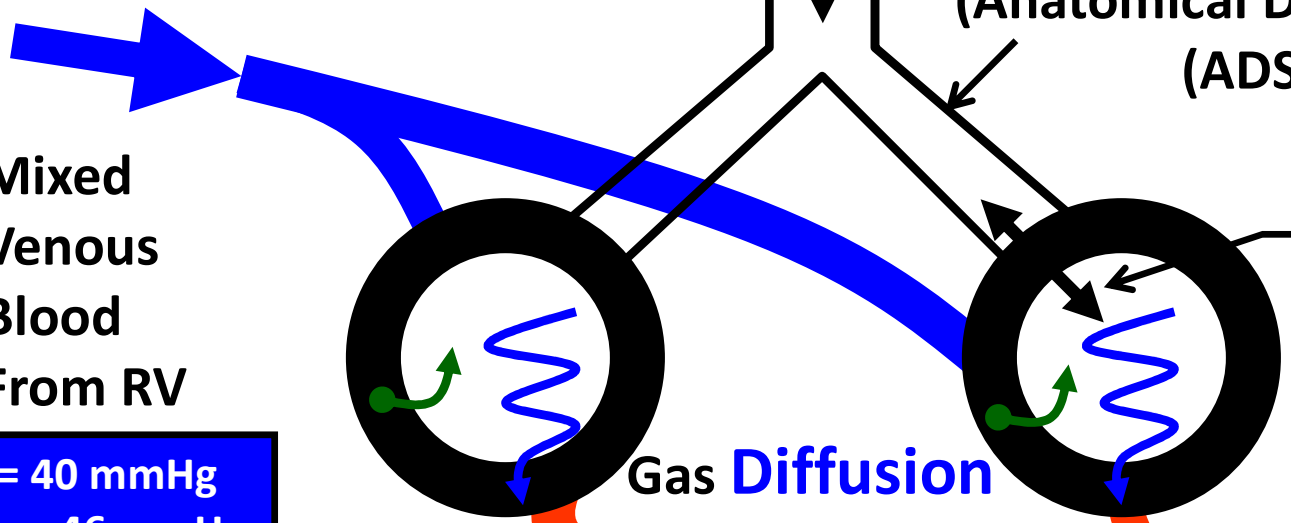
$PO_2 = FIO_2 \times P_{ATM} = 0.21 \times 760 = 159.6 \rightarrow$ 160 mmHg

$V'_T$ = Tidal Volume x Respiratory Rate
$V'_T = TV \times RR$

Total Ventilation ($V'_T$)

$PO_2 = (P_{ATM} - 47)FIO_2$
149.7 → 150 mmHg

Upper Airways (Conducting zone) (Anatomical Dead Space (ADS)



Mixed Venous Blood From RV

$PO_2$ = 40 mmHg
$PCO_2$ = 46 mmHg
$SO_2$ = 75%

Respiratory zone
Alveolar Ventilation
$V'_A = (TV - ADS) \times RR$

Gas Diffusion

Blood Perfusion

$PO_2$ = 100 mmHg
$PCO_2$ = 40 mmHg
$SO_2$ = 97.4%

Arterialized Blood to LA

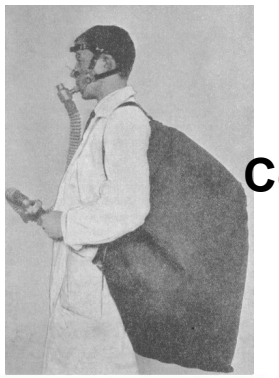
# Pulmonary Blood Flow

# Determining Pulmonary Blood Flow = CO

Air
FIO2
(0.21)

FEO2
(0.16)

$\Delta$ = - 0.05

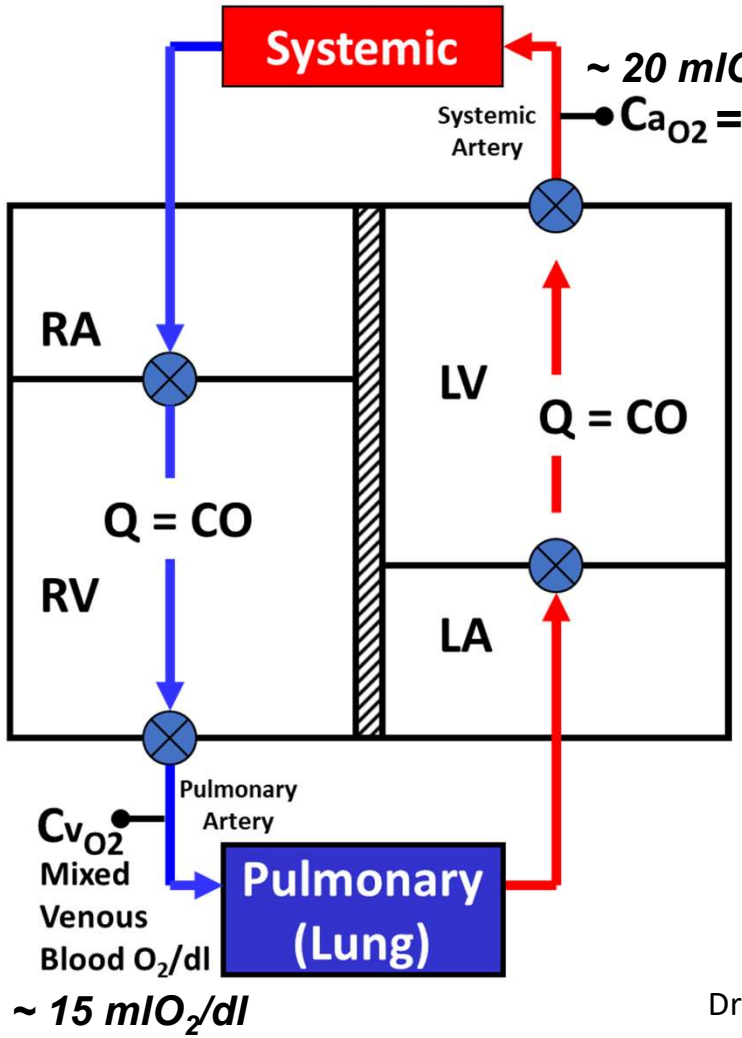


Collect for
5 min

Measure
Total
Volume ($V_T$)

Minute
Ventilation
$\dot{V} = V_T/5$

$\dot{V}_{O_2} = \dot{V} \times 0.05$ $\Delta$




$$CO = \frac{\dot{V}_{O_2}}{Ca_{O_2} - Cv_{O_2}}$$ Fick's Equation

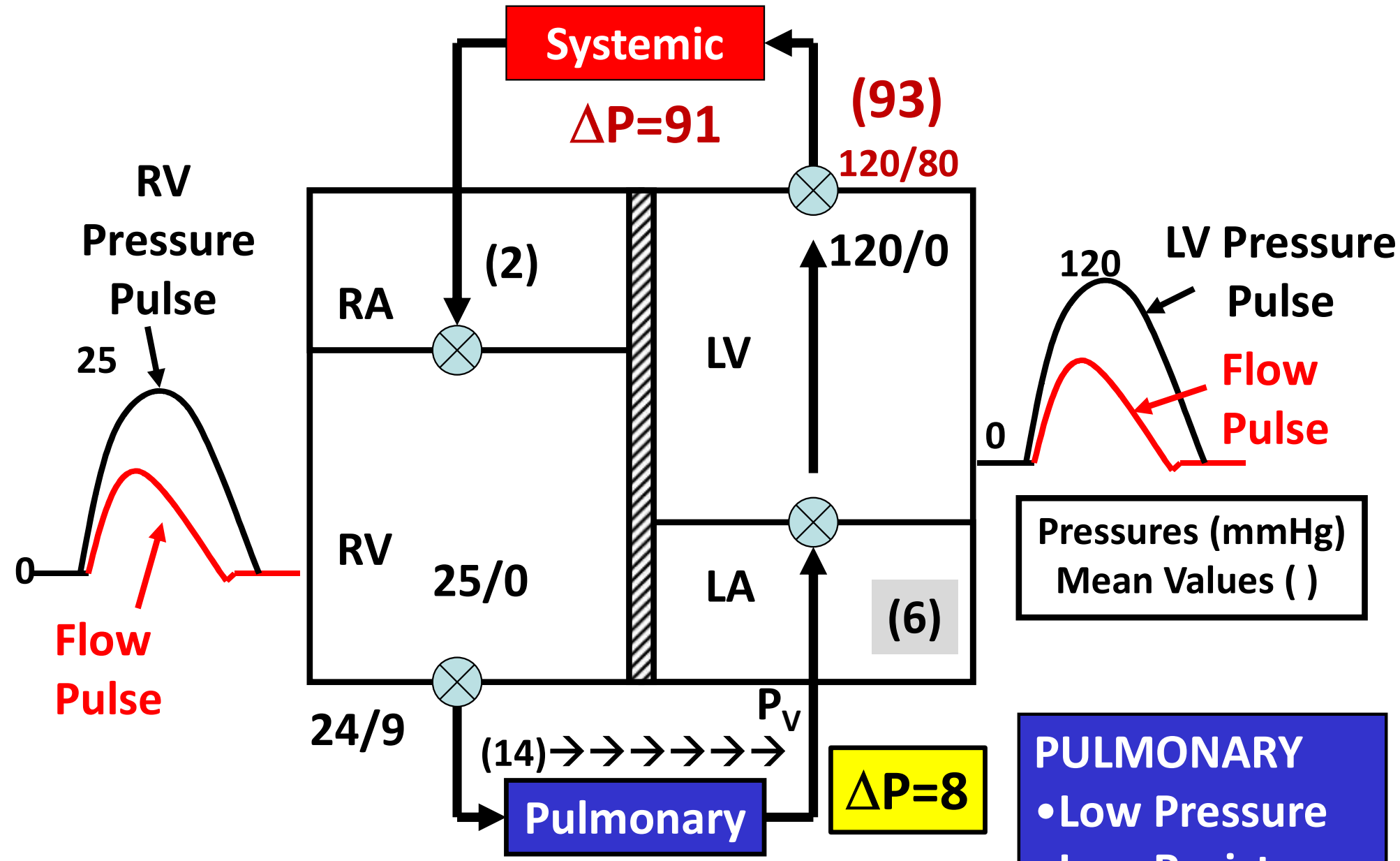
$\dot{V}_{O_2}$ = Oxygen Utilization

If V' = 5000 ml/min then $V'_{O_2}$ = 250 ml/min

$$CO = \frac{250\ ml_{O_2}/min}{\sim(20 - 15)\ ml_{O_2}/100ml = 0.05\ ml_{O_2}/ml\ blood}$$

CO = 5000 ml/min

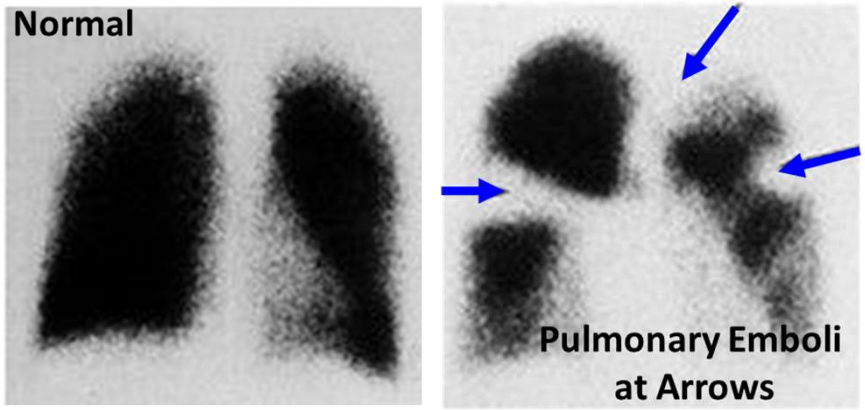
# Pulmonary Pressure and Flow Features

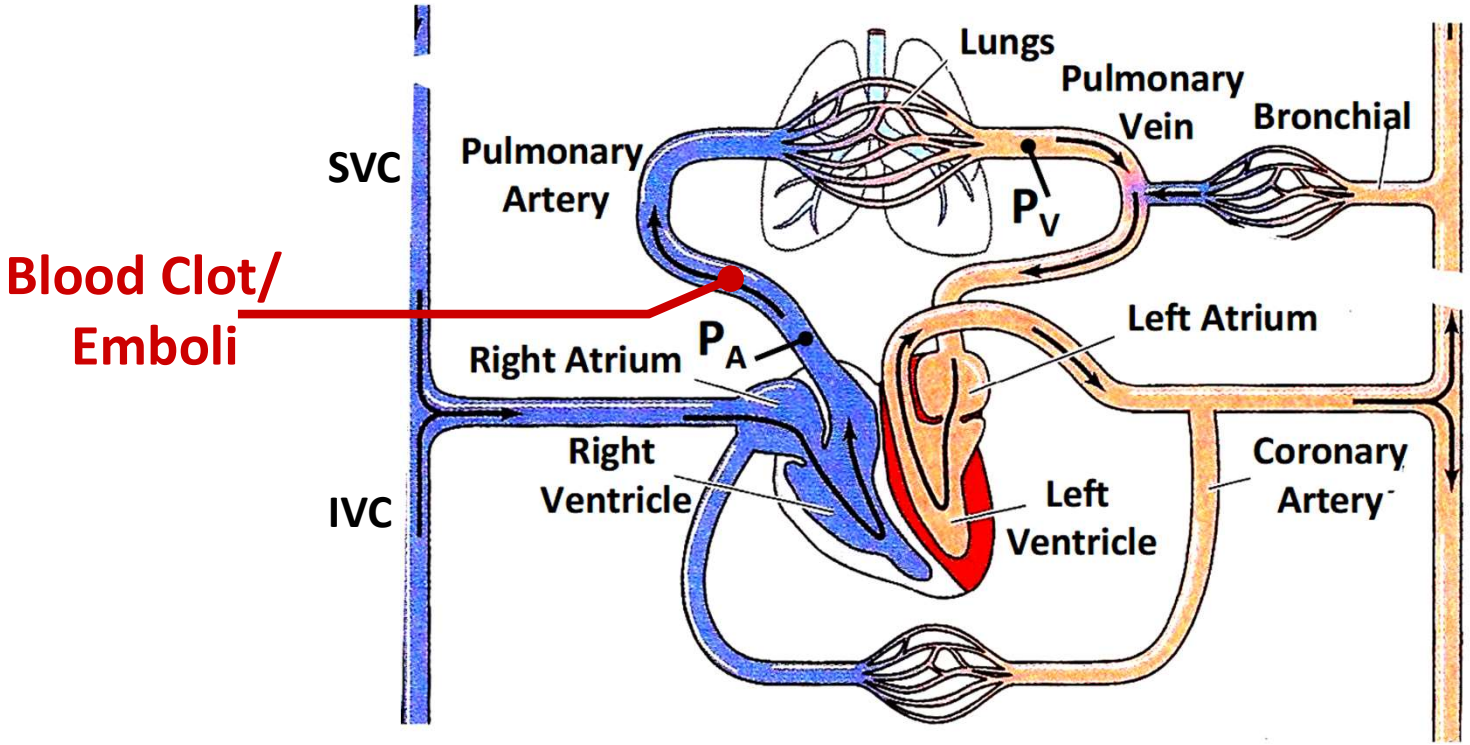


# Clinical Correlations

# Clinical Correlation: Pulmonary Embolism

- Inject radiolabeled albumin ($^{99m}$Tc-labeled macroaggregated albumin)
- Detect distribution of radiation (Gamma-camera)



# Clinical Correlation: Pulmonary Artery Hypertension

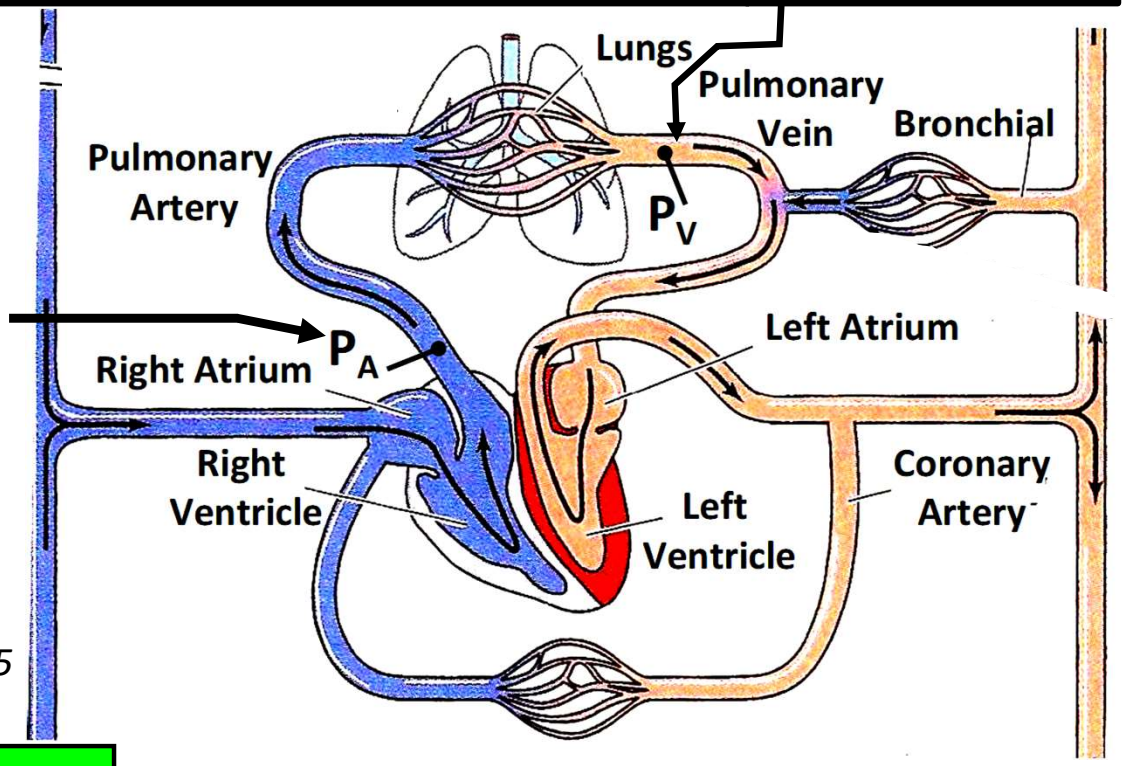
**ENDOTHELIAL CELL VASOACTIVE COMPOUNDS**

**Nitric Oxide**
**Prostacyclin**

**Thromboxane**
**Endothelin**

**Vasoconstriction & Wall Hypertrophy**
**"Precapillary"**

**CHF**
**"Postcapillary"**




**Resting Mean $P_A$ ≥25 mmHg**

*Hoeper, MM*
*Eur Respir J*
*2009;34:790-791*
*Latest guideline 2015*

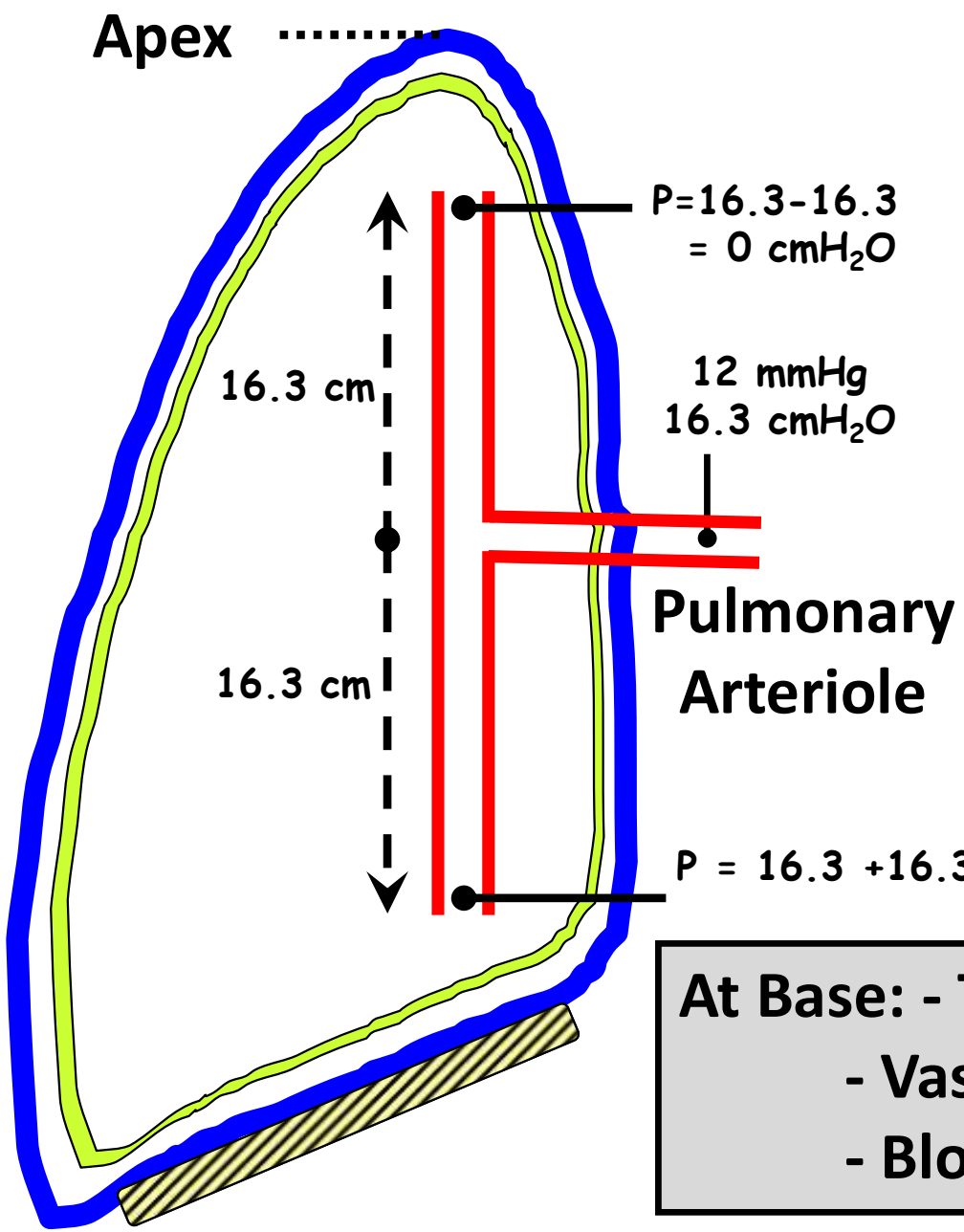
**Normal $P_A$**
**14.3 ± 3.3 mmHg**

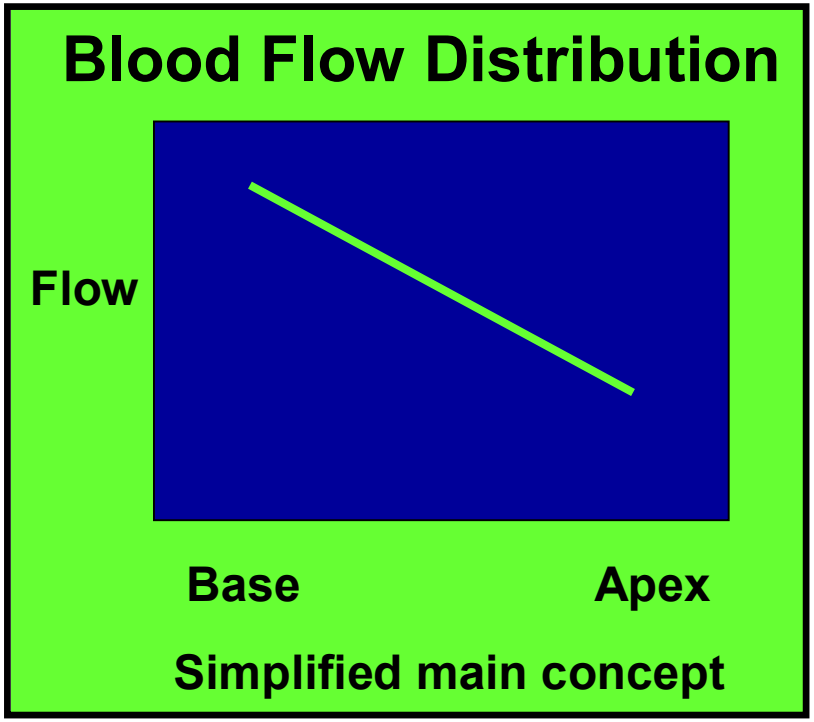
**Perfusion Lung Scans**


*After: Fukuchi et al.*
*J Nuclear Med*
*2002;43(6)757-761*

**Regional or local blood flow deficits**

# Gravity Affects Vascular Resistance



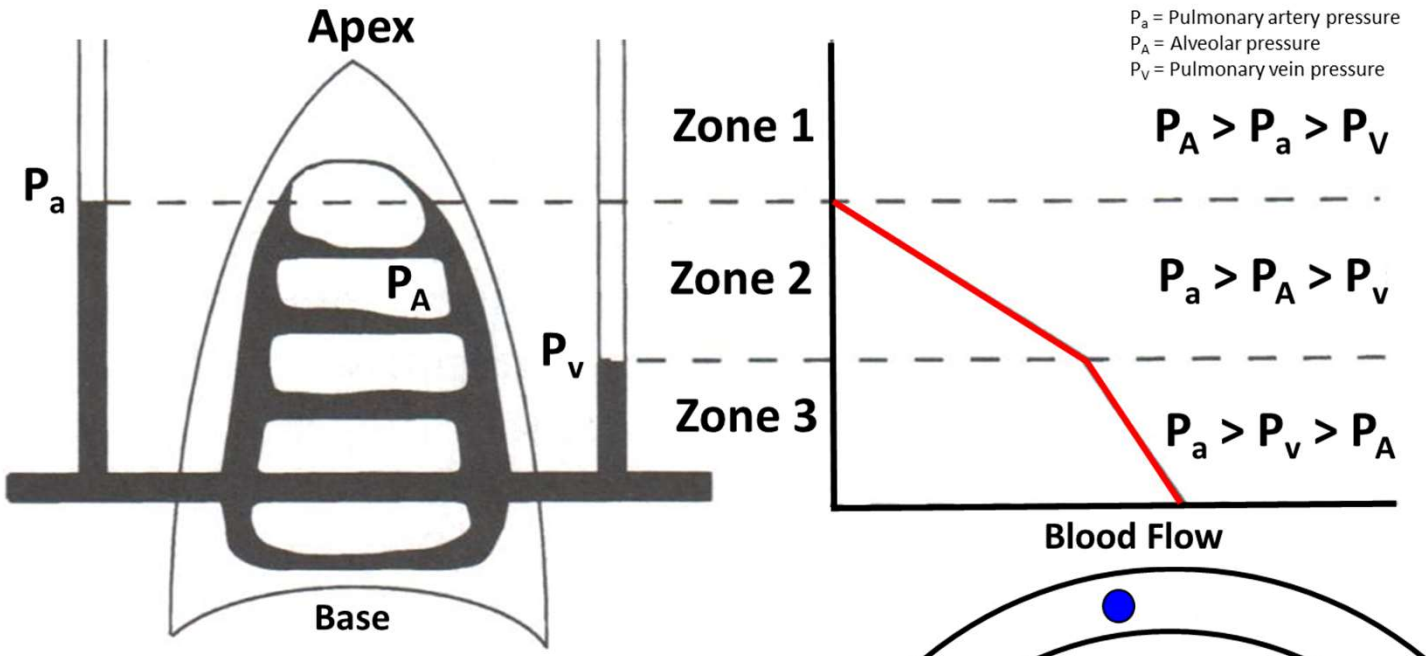

**At Base: - TMP Greater**
**- Vascular Resistance Less**
**- Blood Flow is Greater**

1 cmH2O = 0.736 mmHg

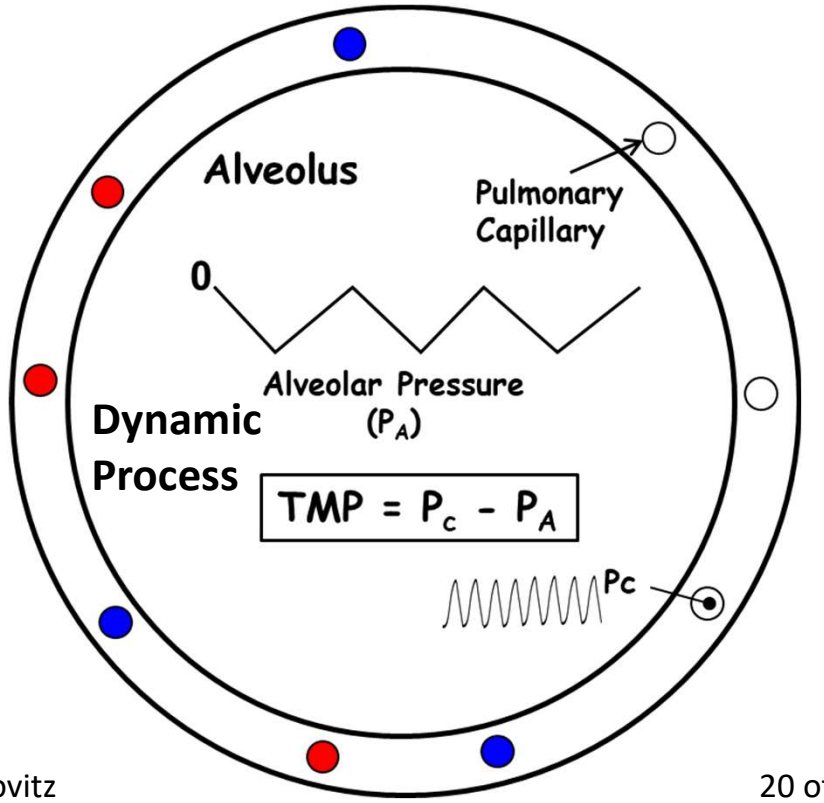
## Slide 19

**HM1** Harvey Mayrovitz, 4/20/2022

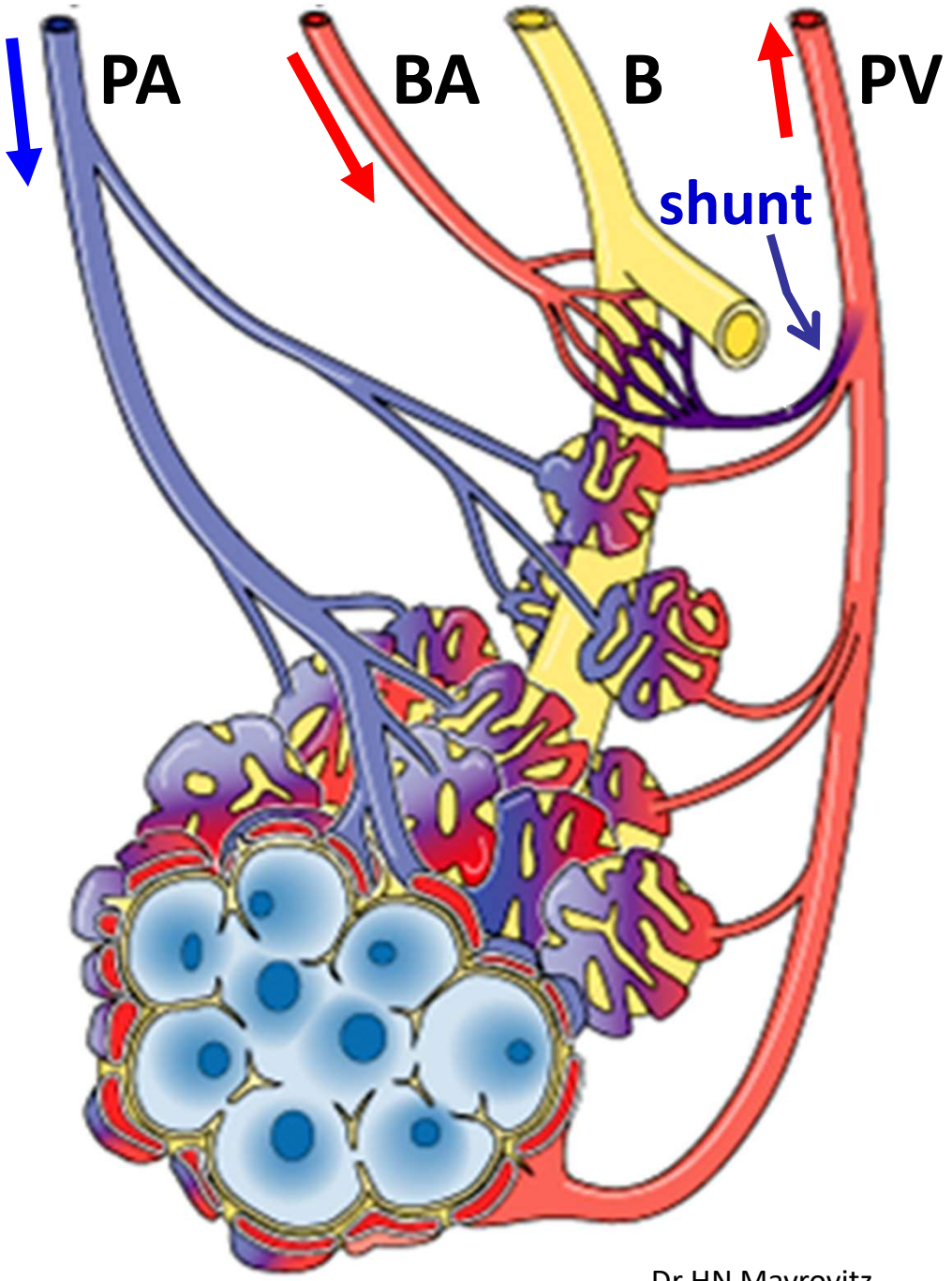
# Gravity: 3-Zone Dependent Lung Model




- Thin-walled vessels can collapse
- If surround pressure ($P_A$) > $P_a$ then Q =0 (Zone 1)
- Not normally occurring but may occur with
  → low ABP e.g. Hemorrhage
  → positive pressure ventilation
- If collapsible state (Zone 2) Q ~ $P_a - P_A$
  → Could be intermittent pulses of flow
- If non-collapsible state (Zone 3) Q ~ $P_a - P_v$



# Bronchial Circulation and Flow Features

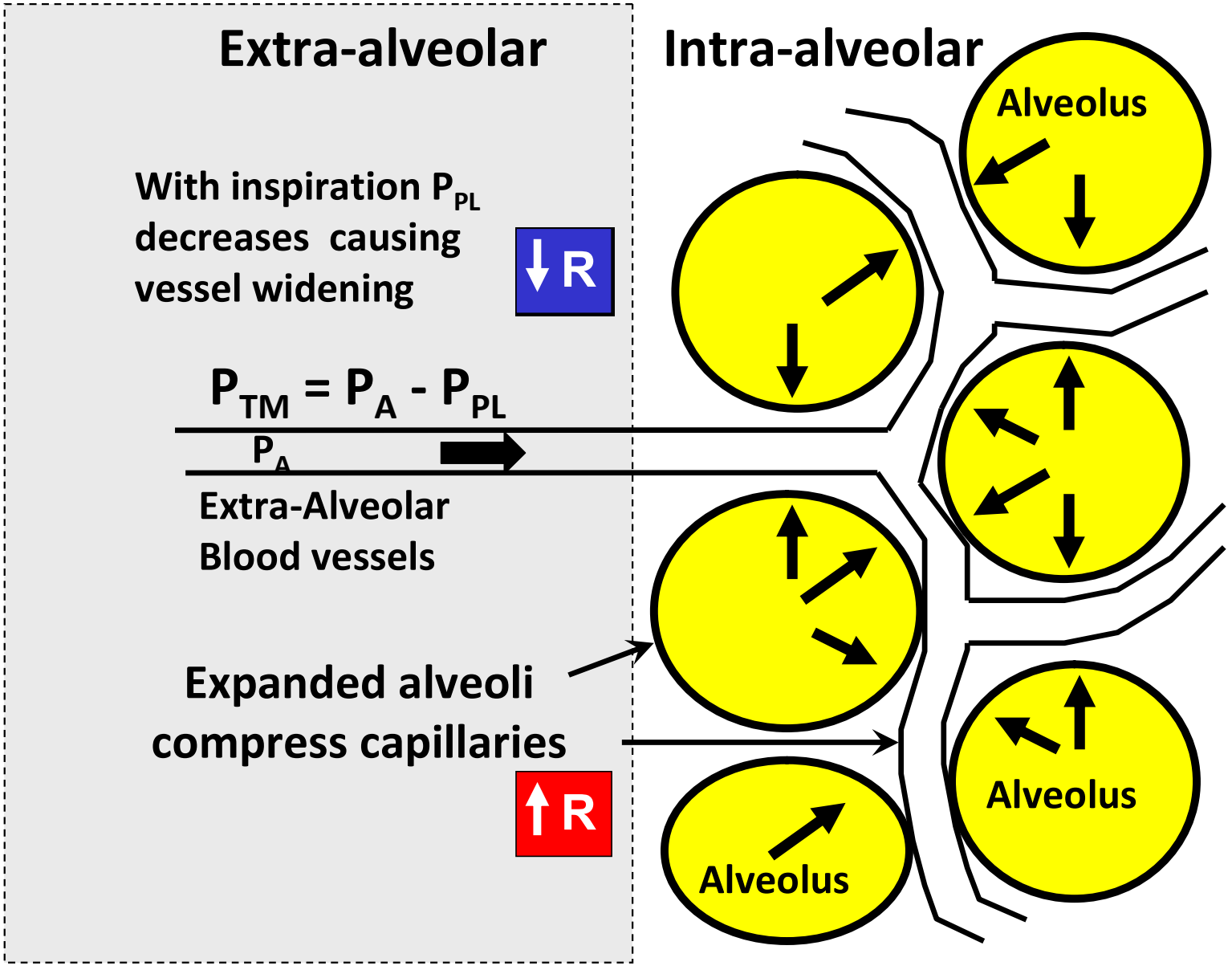



***Bronchial Circulation***

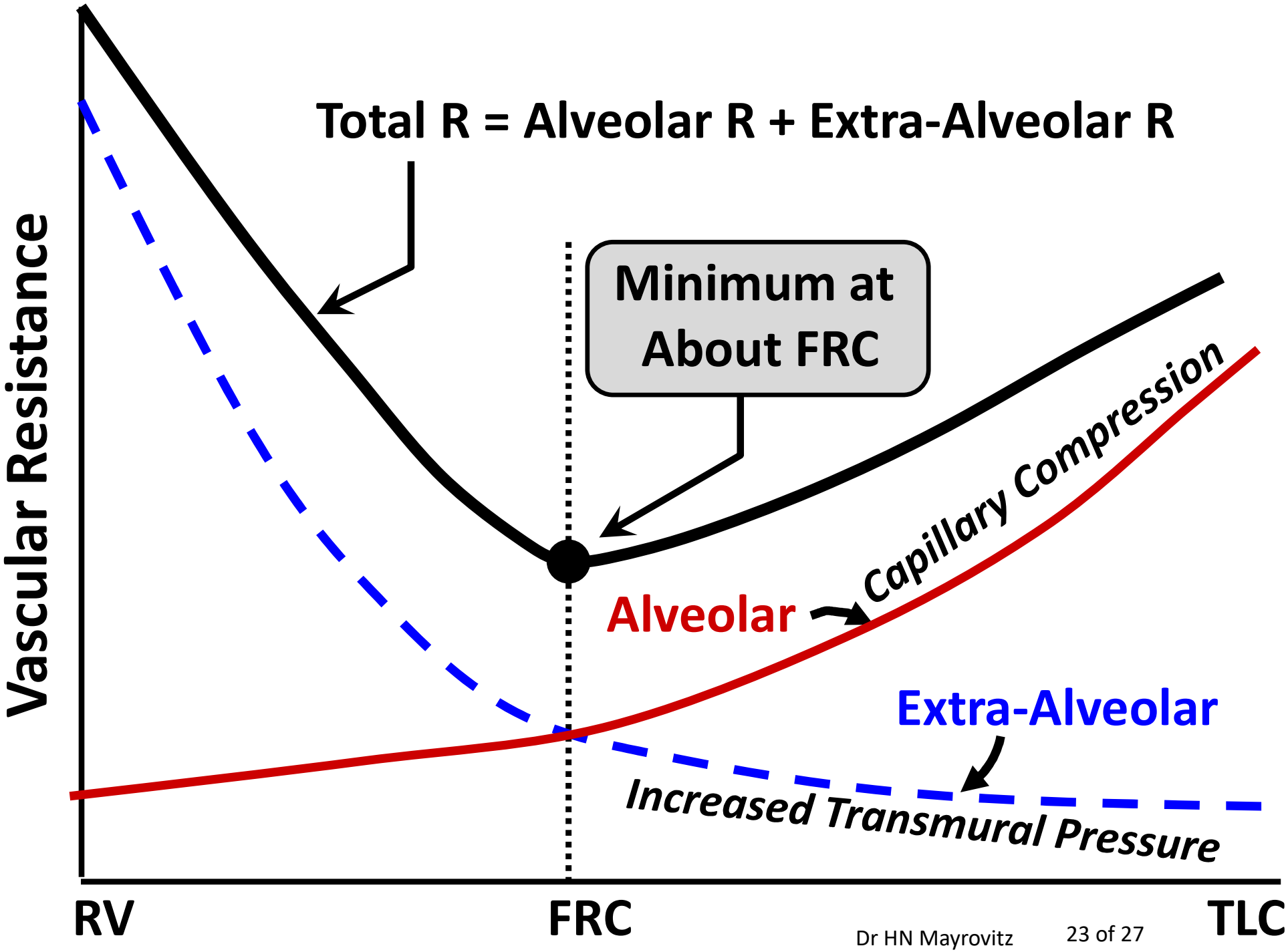
**Low $O_2$ of Bronchial mixes with high $O_2$ of Pulmonary Anatomical Shunt**

# Lung Volume Affects Vascular Resistance

## Opposite effects on intra and extra alveolar vessels

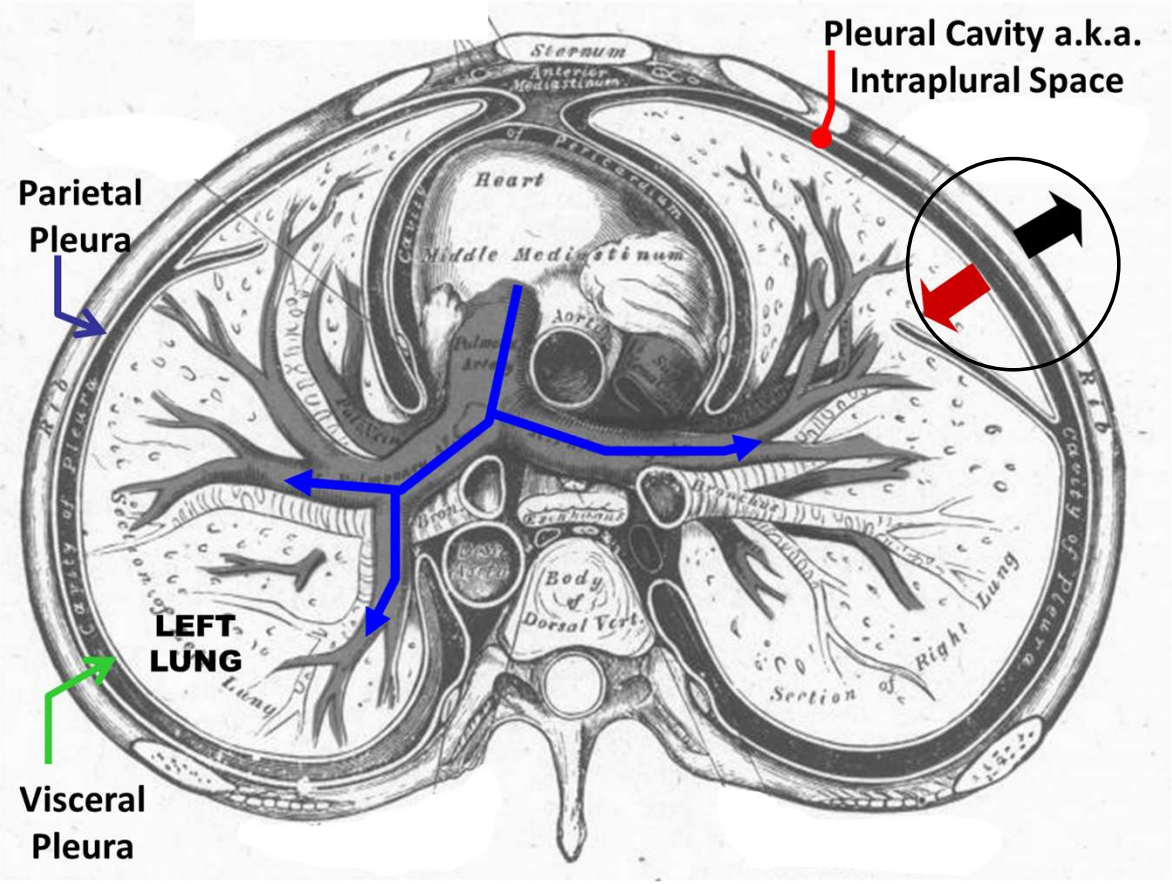


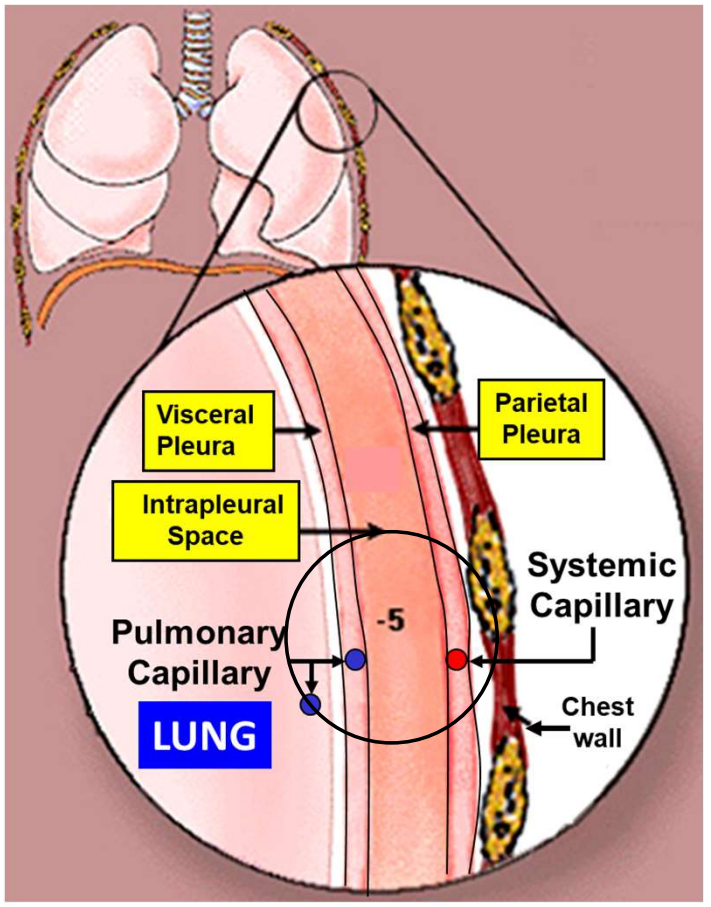
# Total Vascular Resistance: Opposite Tendencies



# Fluid Balance

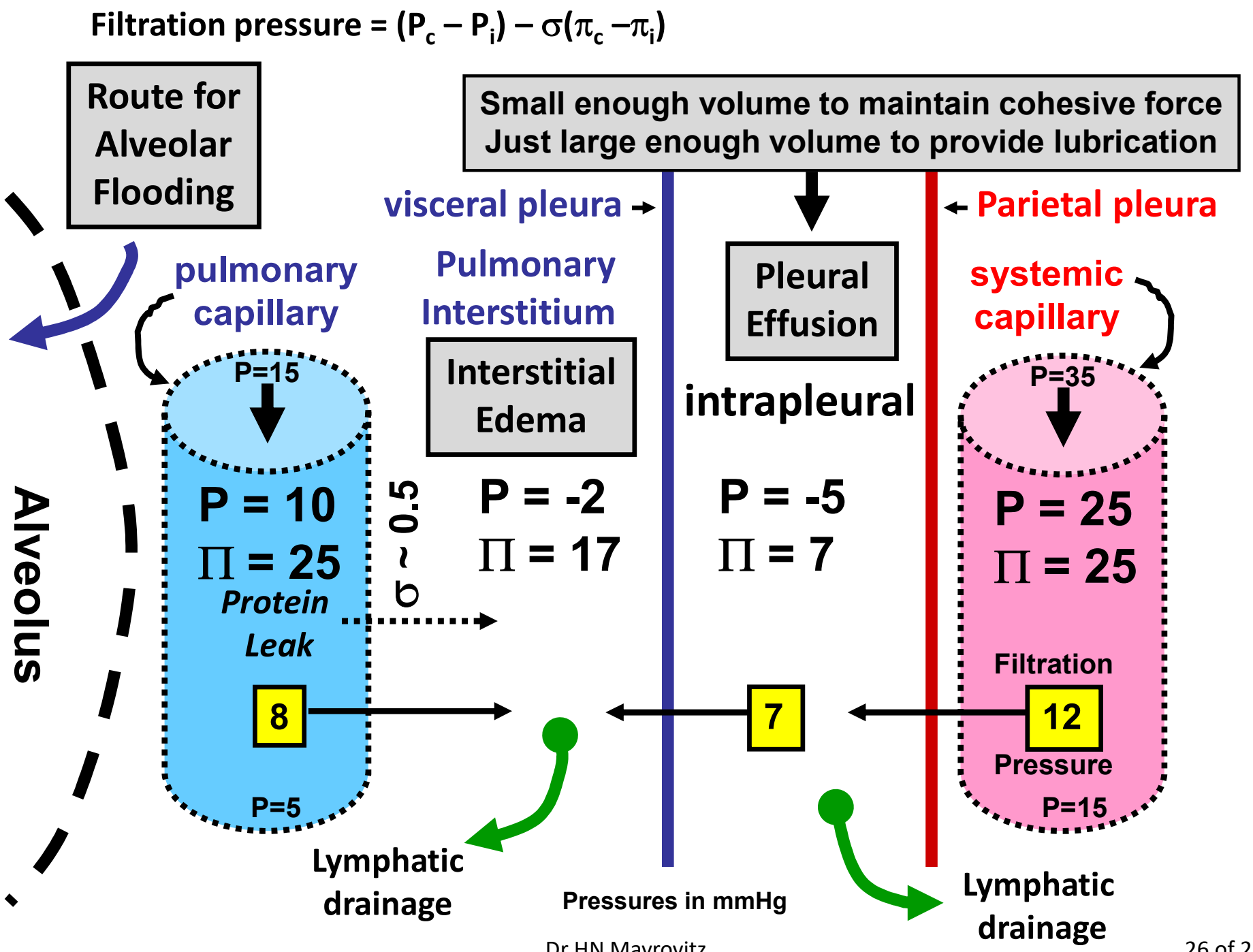
# Intrapleural Space: Couples Lung to Chest




- Oppositely directed forces act on the intrapleural space
- One due to the lung inward directed force (red arrow) and one outwardly directed force (black arrow) due to recoil of the chest wall towards its zeros stress state
- This results in a sub atmospheric pressure within the intrapleural space

# Pulmonary Fluid Balance



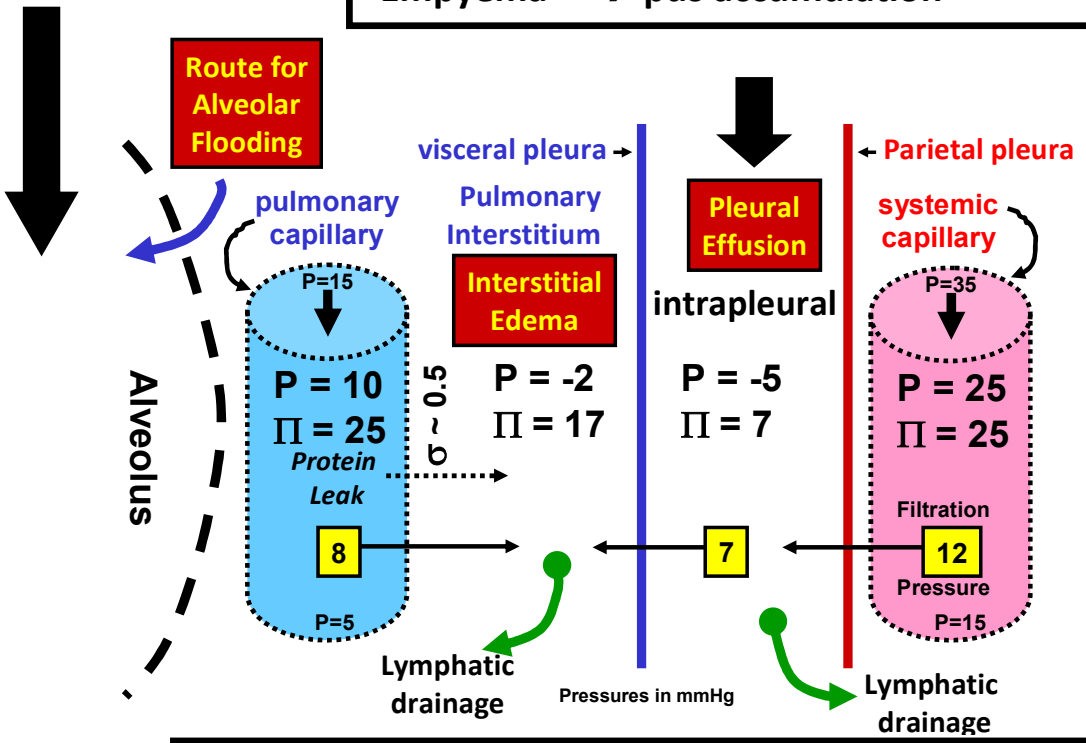
# "When things go Wrong": Definitions & Processes

**Alveolar Flooding**

- Threshold Edema
- Epithelial Cell Injury

**Pleural Effusion → Fluid Accumulating in pleural space**

Transudative → P/Π imbalance → mostly $H_2O$
Exudative → Inflammation/Infection/Cancer
+WBC → fluid secretion
+Ca cells → fluid secretion
+ Mesothelial cell secretion
Hemothorax → Blood accumulation
Empyema → pus accumulation




**Interstitial Edema → Interstitial excess fluid**

Lung → P/Π imbalance e.g.
Pulmonary HTN/+ permeability/lymph deficit

# End Respiratory Physiology
# Lecture 36