Pulmonary Blood Flow

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Objectives

- The student will know the structure, function, distribution and control of pulmonary blood supply
  - Compare pulmonary and bronchial circulation
  - Compare and contrast pulmonary and systemic circulation
  - Describe and explain the effects of cardiac output and lung volume on pulmonary vascular resistance
  - Describe the effects of hypoxia on pulmonary vascular resistance
  - Describe the effects of gravity of pulmonary blood flow
  - Explain Starling's equation
  - Describe the mechanisms of pulmonary edema

Two Circulations in the Lung

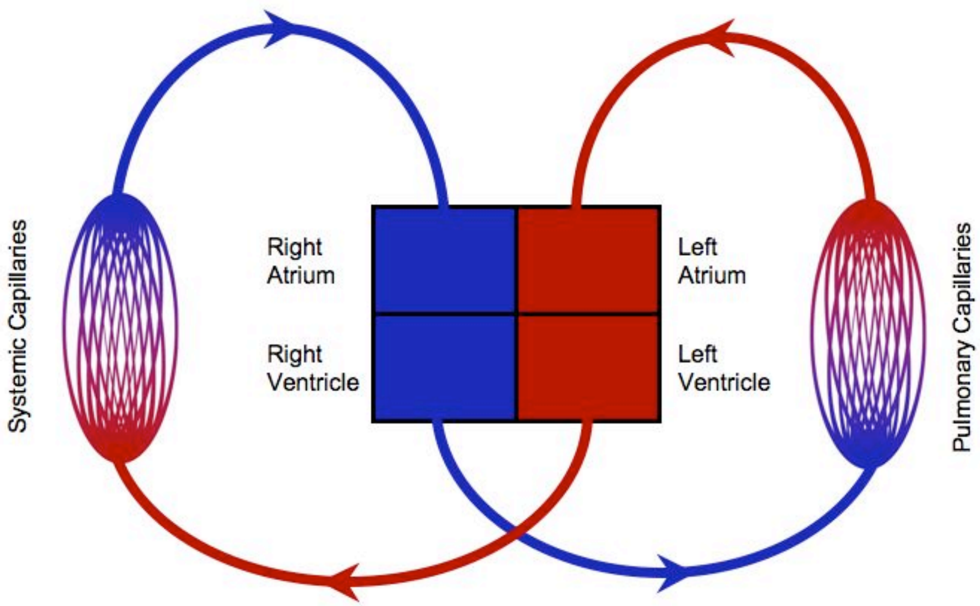
- Pulmonary Circulation.
  - Arises from Right Ventricle.
  - Receives 100% of blood flow.

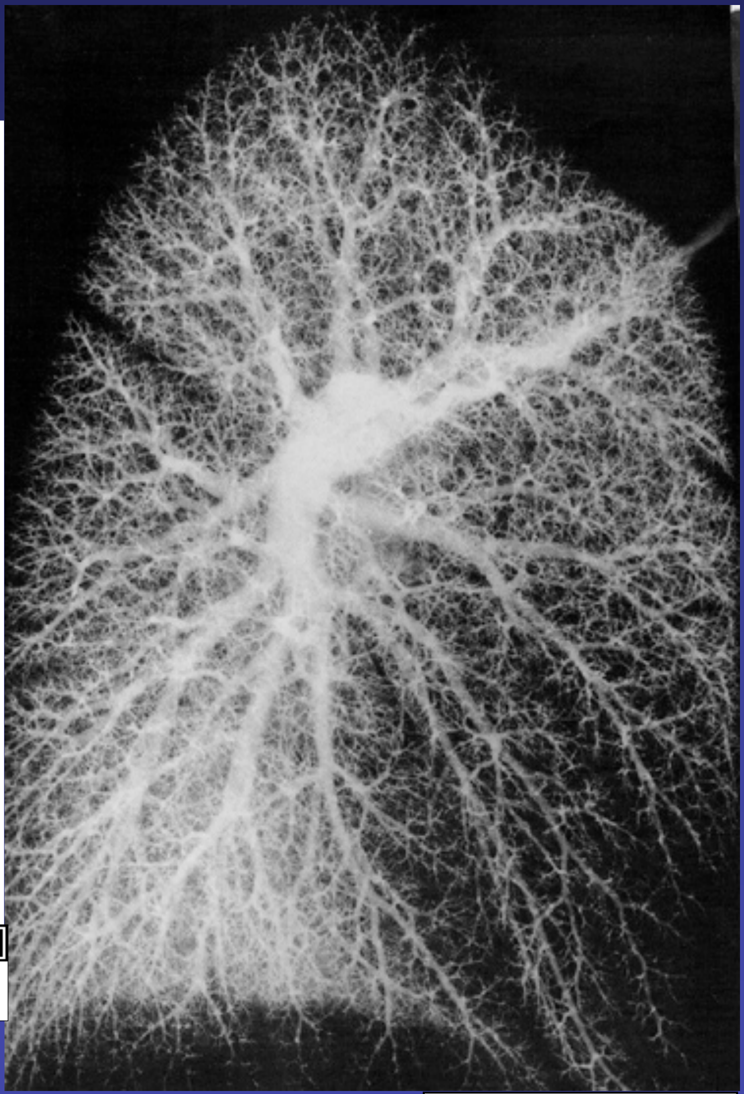
- Bronchial Circulation.
  - Arises from the aorta.
  - Part of systemic circulation.
  - Receives about 2% of left ventricular output.

Bronchial Circulation

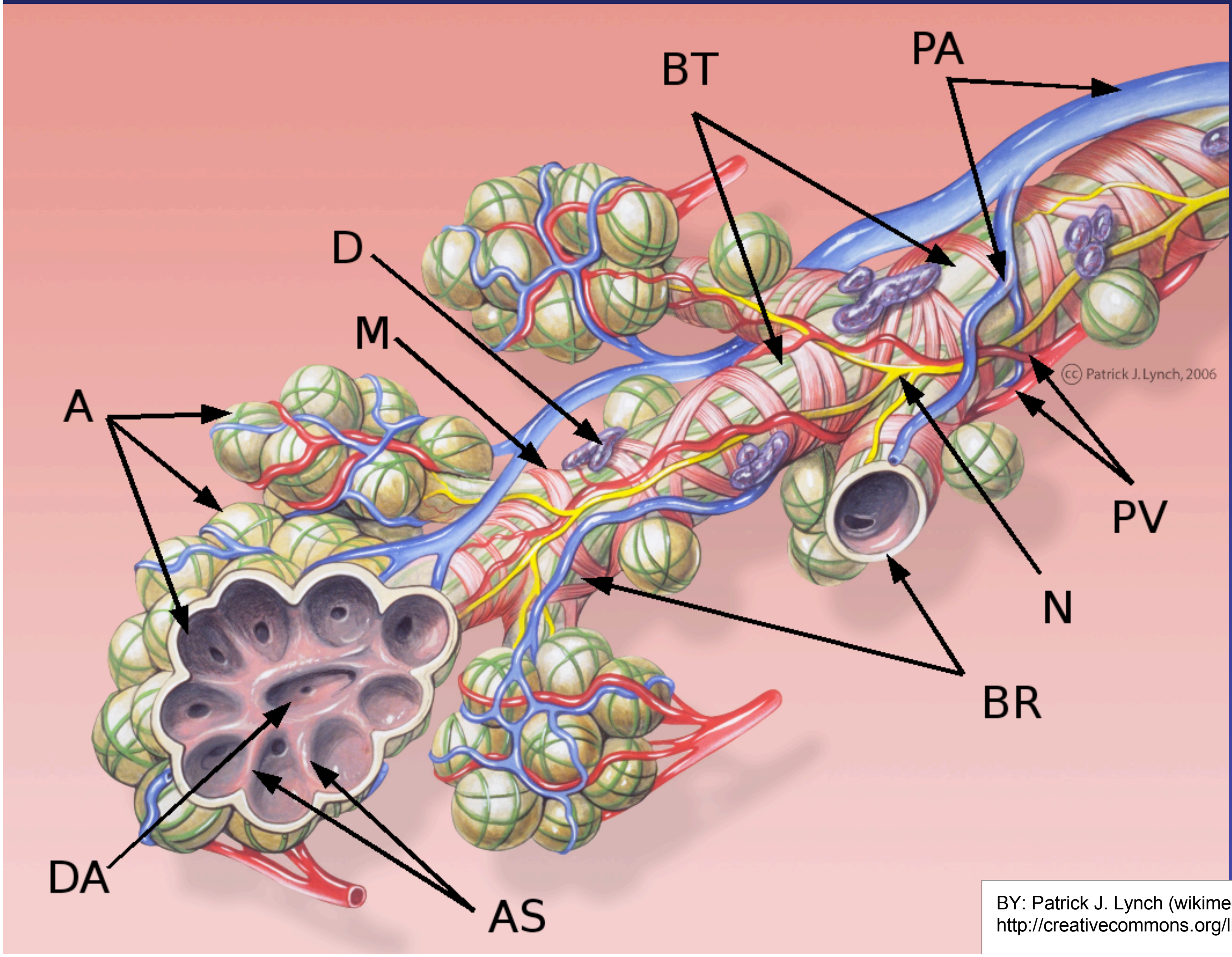
Image of bronchopulmonary
anastamosis removed

Pulmonary Circulation





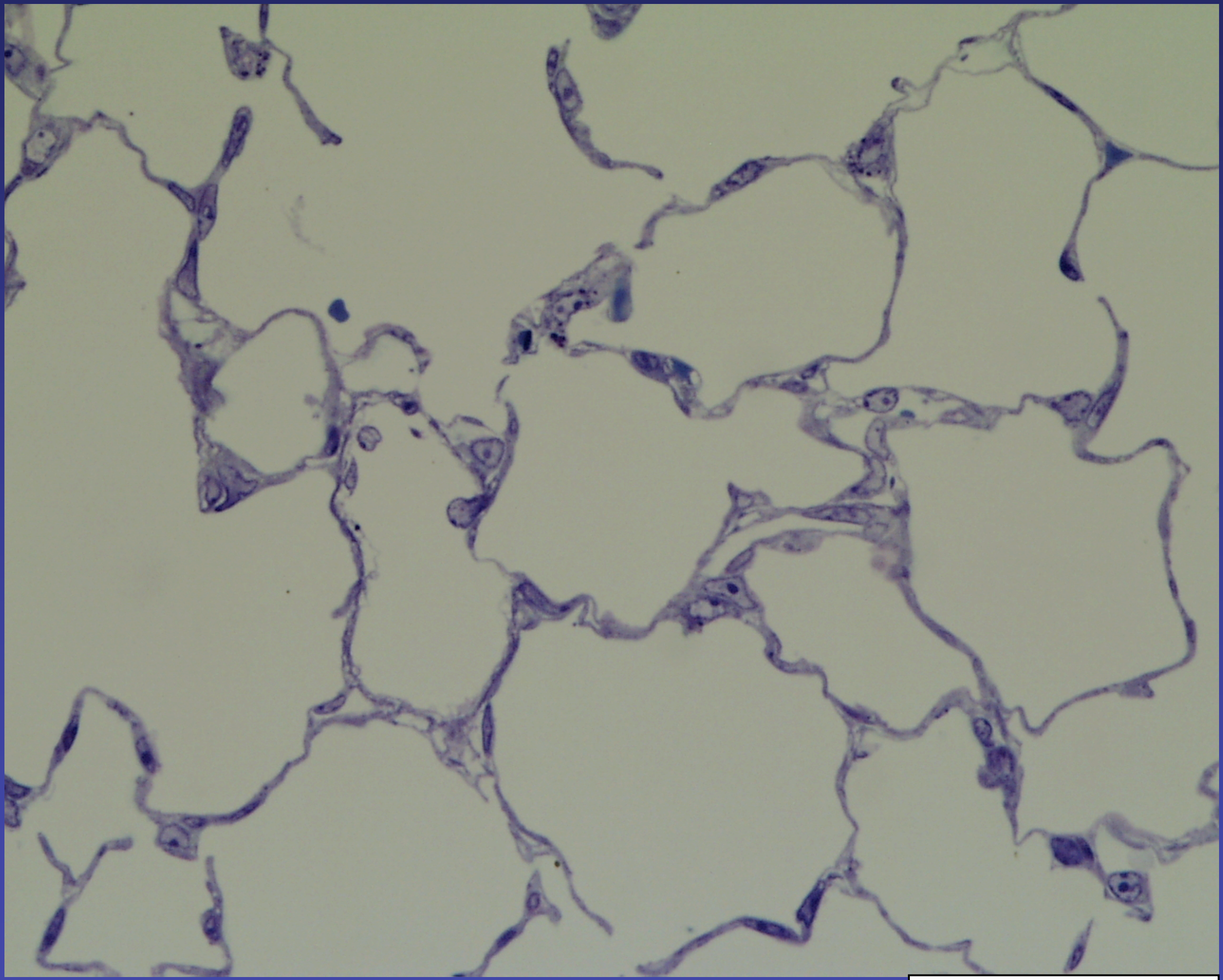
Pulmonary Circulation



Pulmonary Circulation

- In series with the systemic circulation.
- Receives 100% of cardiac output (3.5L/min/m^2).
- RBC travels through lung in 4-5 seconds.
- 280 billion capillaries, supplying 300 million alveoli.
  - Surface area for gas exchange = 50 – 100 m^2

Alveolar Architecture



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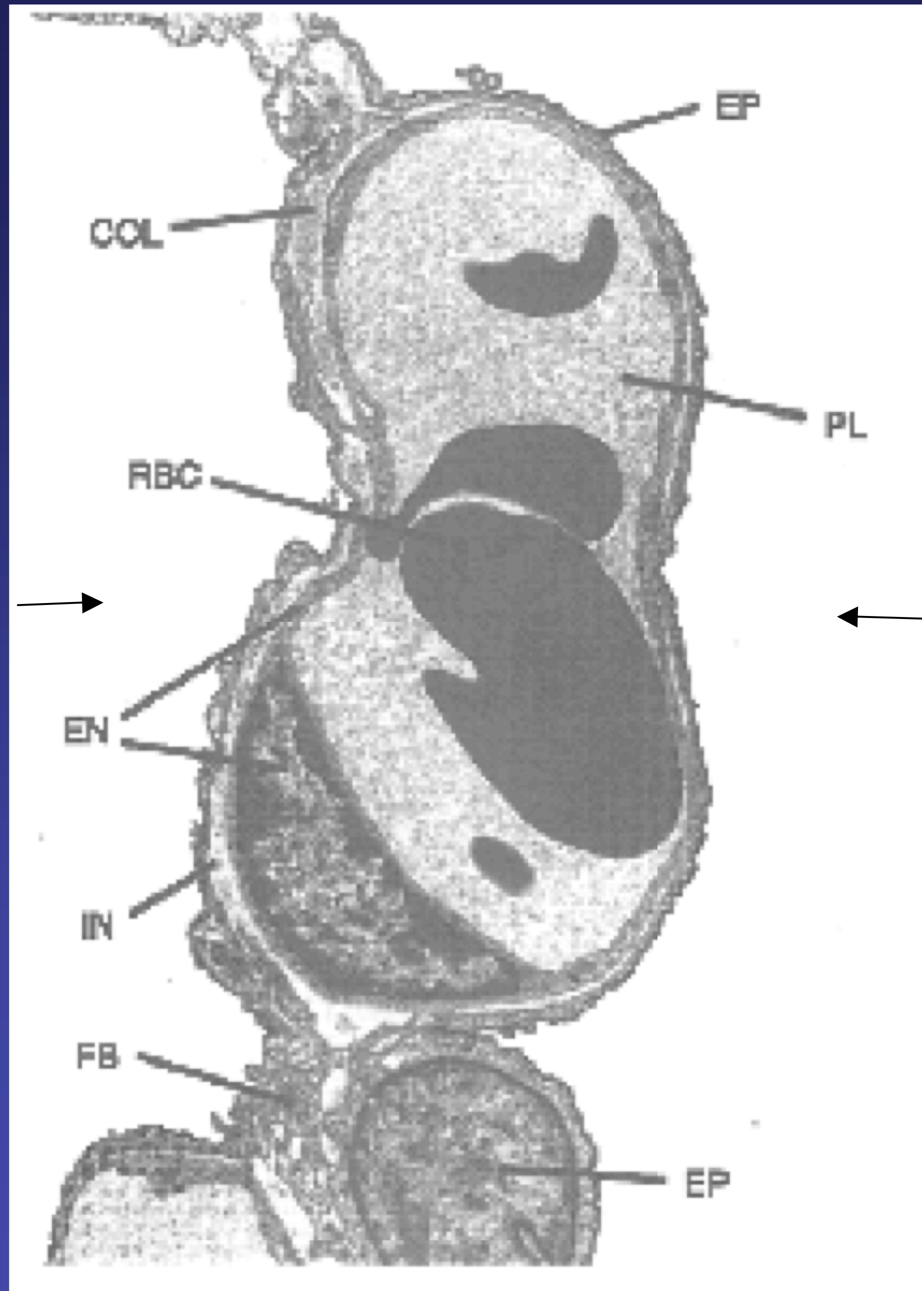

Alveolar Airspace

Alveolar Airspace

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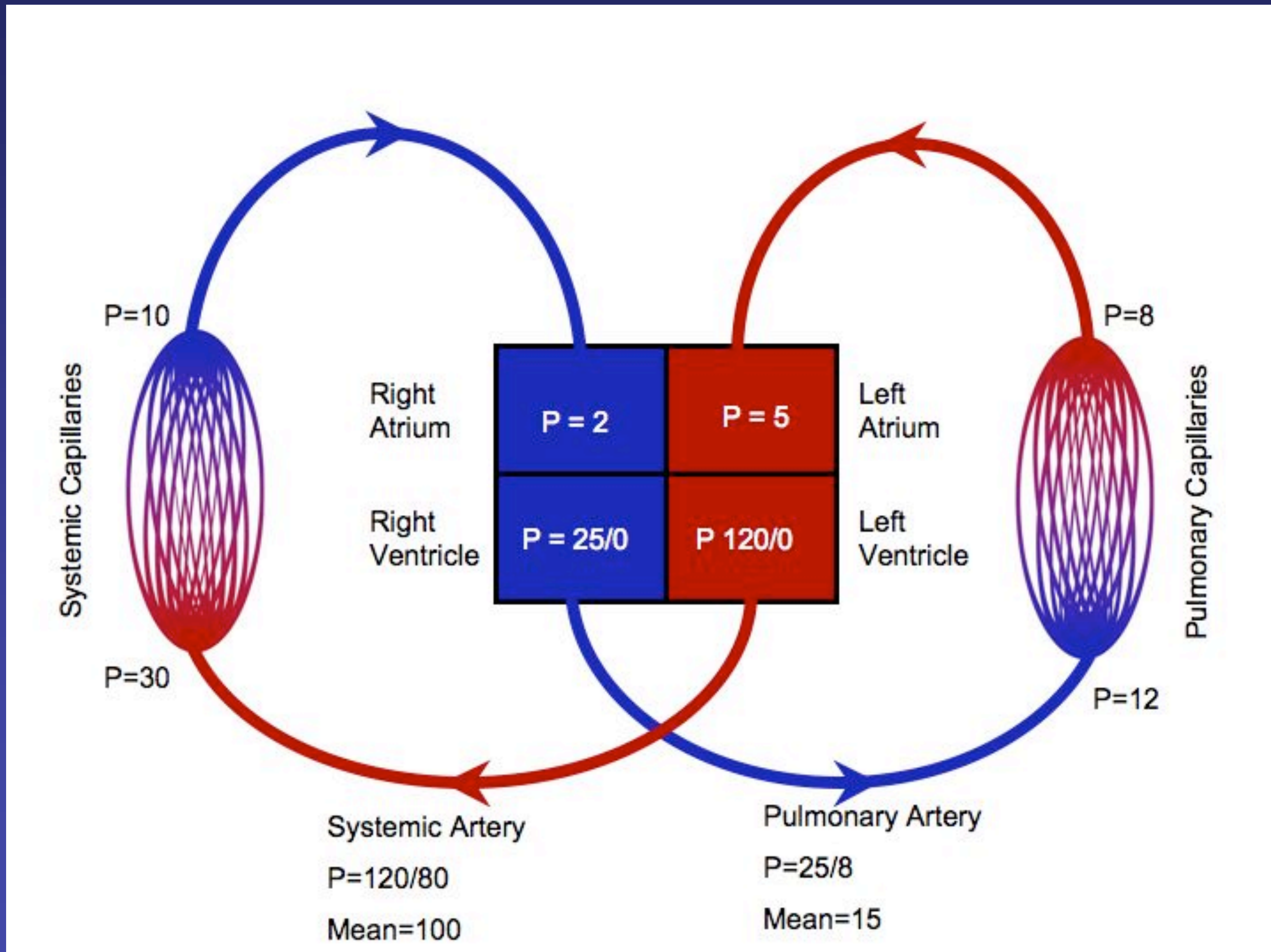
Functional Anatomy of the Pulmonary Circulation

- Thin walled vessels at all levels.
- Pulmonary arteries have far less smooth muscle in the wall than systemic arteries.
- Consequences of this anatomy- the vessels are:
  - Distensible.
  - Compressible.

Pulmonary Circulation Pressures



Pulmonary Vascular Resistance

$$\text{Vascular Resistance} = \frac{\text{input pressure - output pressure}}{\text{blood flow}}$$

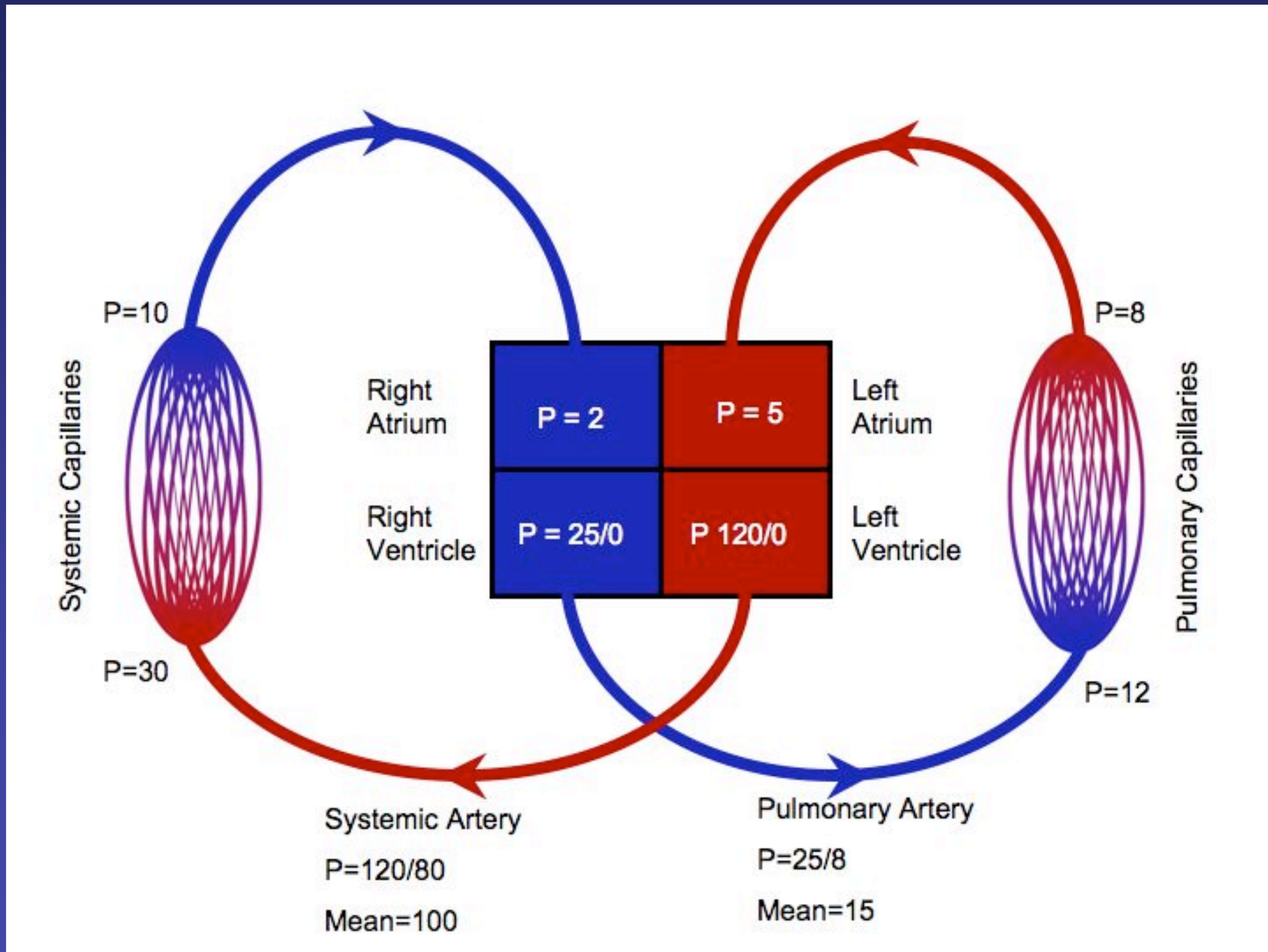
$$\text{PVR} = k \bullet \frac{\text{mean PA pressure - left atrial pressure}}{\text{cardiac output (index)}}$$

mean PA pressure - left atrial pressure = 10 mmHg

mean aorta pressure - right atrial pressure = 98 mmHg

Therefore PVR is 1/10 of SVR

Vascular Resistance is Evenly Distributed in the Pulmonary Circulation



Reasons Why Pressures Are Different in Pulmonary and Systemic Circulations?

- Gravity and Distance:
  - Distance above or below the heart adds to, or subtracts from, **both** arterial and venous pressure
  - Distance between Apex and Base

Systemic

Aorta	100 mmHg
Head	50 mmHg
Feet	180 mmHg

Pulmonary

Main PA	15 mmHg
Apex	2 mmHg
Base	25 mmHg

Reasons Why Pressures Are Different in Pulmonary and Systemic Circulations?

- Control of regional perfusion in the systemic circulation:
  – Large pressure head allows alterations in local vascular resistance to redirect blood flow to areas of increased demand (e.g. to muscles during exercise).

  – Pulmonary circulation is all performing the same job, no need to redirect flow (exception occurs during hypoxemia).

- Consequences of pressure differences:
  – Left ventricle work load is much greater than right ventricle
  – Differences in wall thickness indicates differences in work load.

Influences on Pulmonary Vascular Resistance

Pulmonary vessels have:

-Little vascular smooth muscle.

-Low intravascular pressure.

-High **distensiblility** and **compressiblility.**

Vessel diameter influenced by extravascular forces:

-Gravity

-Body position

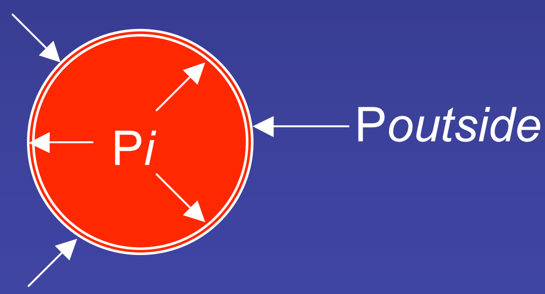
-Lung volume

-Alveolar pressures/intrapleurql pressures

-Intravascular pressures

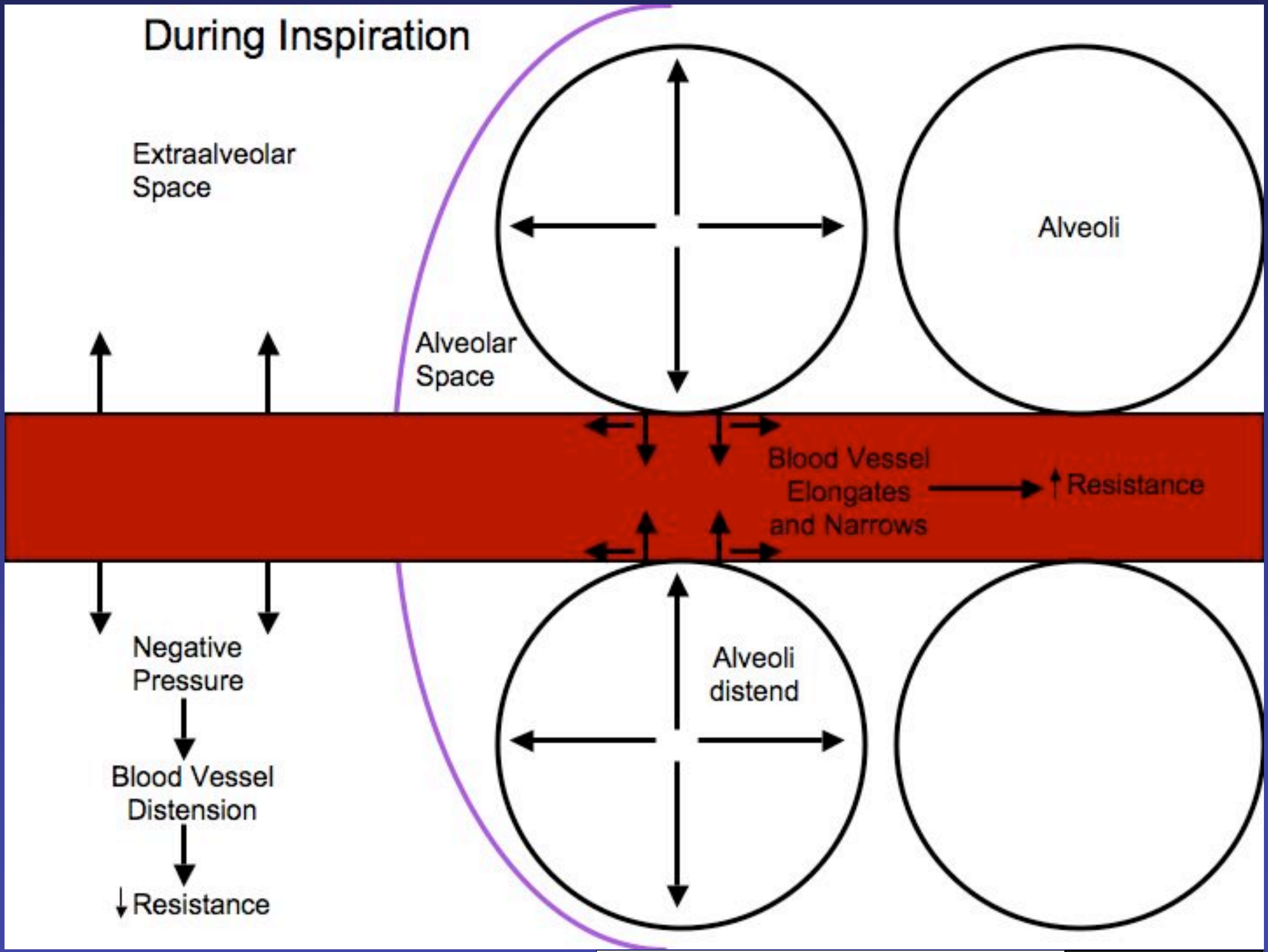
Influences of Pulmonary Vascular Resistance

- Transmural pressure = Pressure Inside – Pressure Outside.
  - Increased transmural pressure-increases vessel diameter.
  - Decreased transmural pressure-decreased vessel diameter (increase in PVR).
  - Negative transmural pressure-vessel collapse.



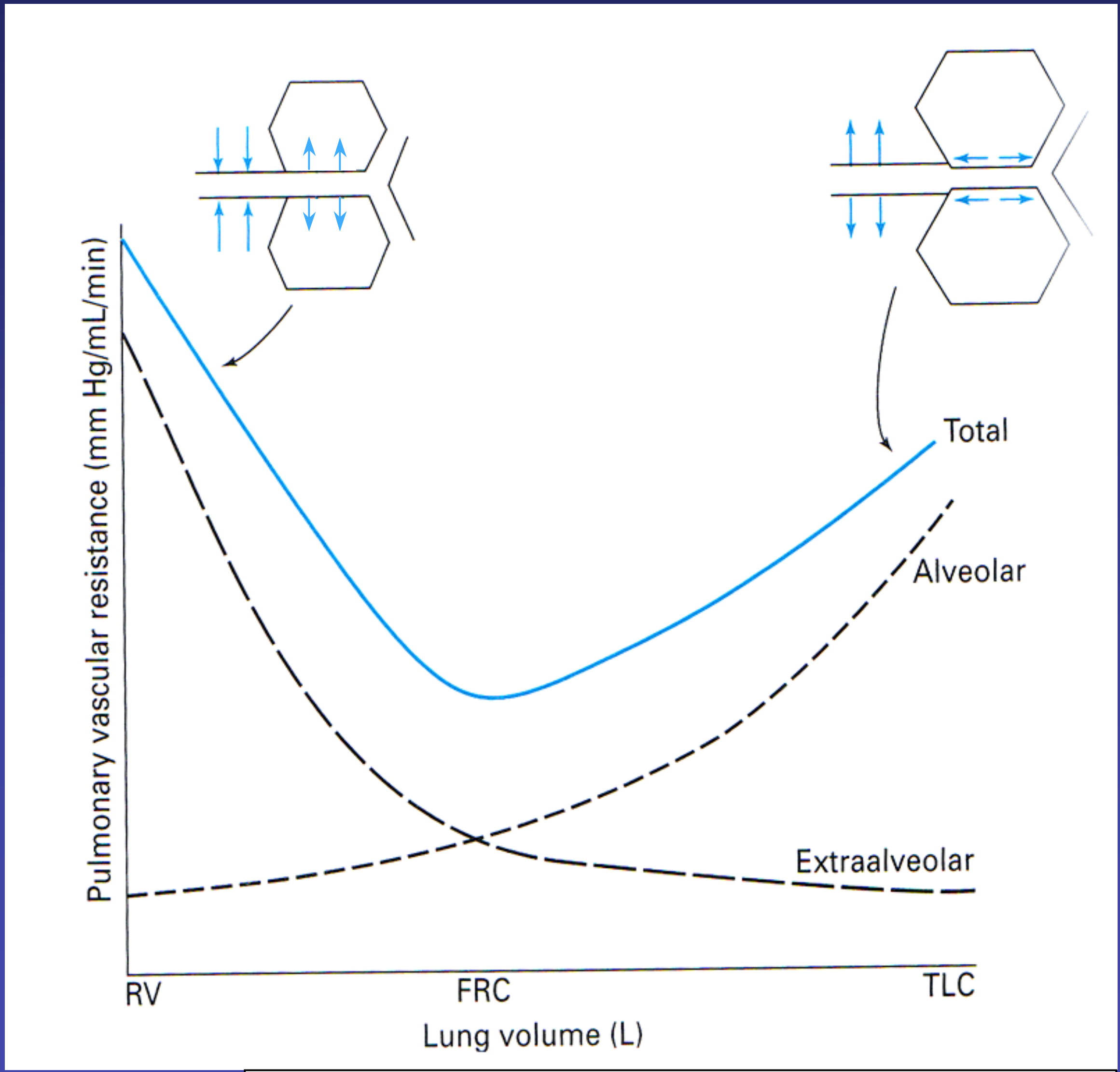
- Different effects of lung volume on <u>alveolar</u> and <u>extraalveolar</u> vessels.

Effect of Transmural Pressure on Pulmonary Vessels During Inspiration



Resistance ∝ Length and Resistance ∝ 1/(Radius)^4

Effect of Lung Volume on PVR



Source: *Pulmonary Physiology*, The McGraw-Hill Companies, Inc., 2007

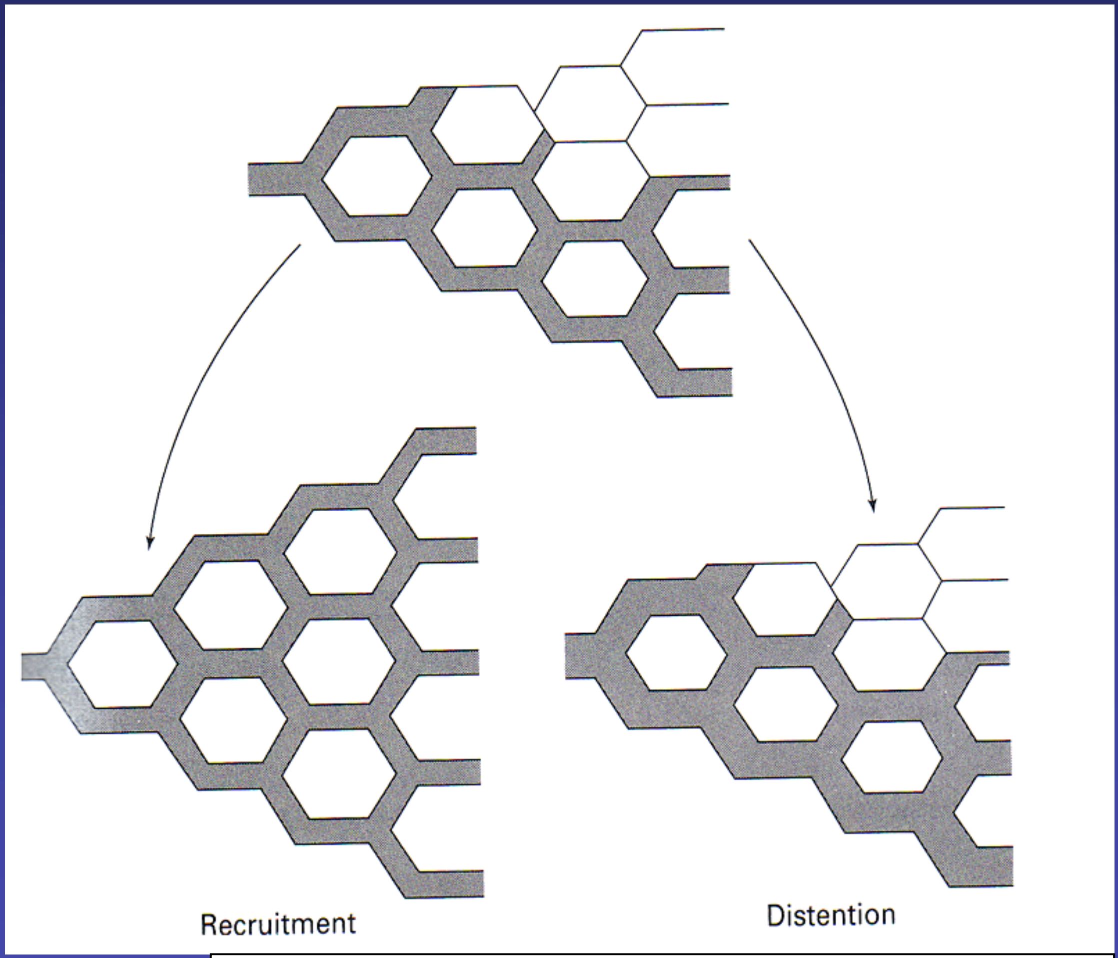
Pulmonary Vascular Resistance During Exercise

- During exercise cardiac output increases (e.g. 5-fold), but with little change in mean pulmonary artery pressure
  - How is this possible?

$$\text{Vascular Resistance} = \frac{\text{input pressure - output pressure}}{\text{blood flow}}$$

- ΔPressure= Flow x Resistance
- If pressure does not change, then PVR must decrease with increased blood flow
    - Passive effect (seen in isolated lung prep)
  - Recruitment: Opening of previously collapsed capillaries
  - Distensibility: Increase in diameter of open capillaries.

Recruitment and Distention in Response to Increased Pulmonary Artery Pressure



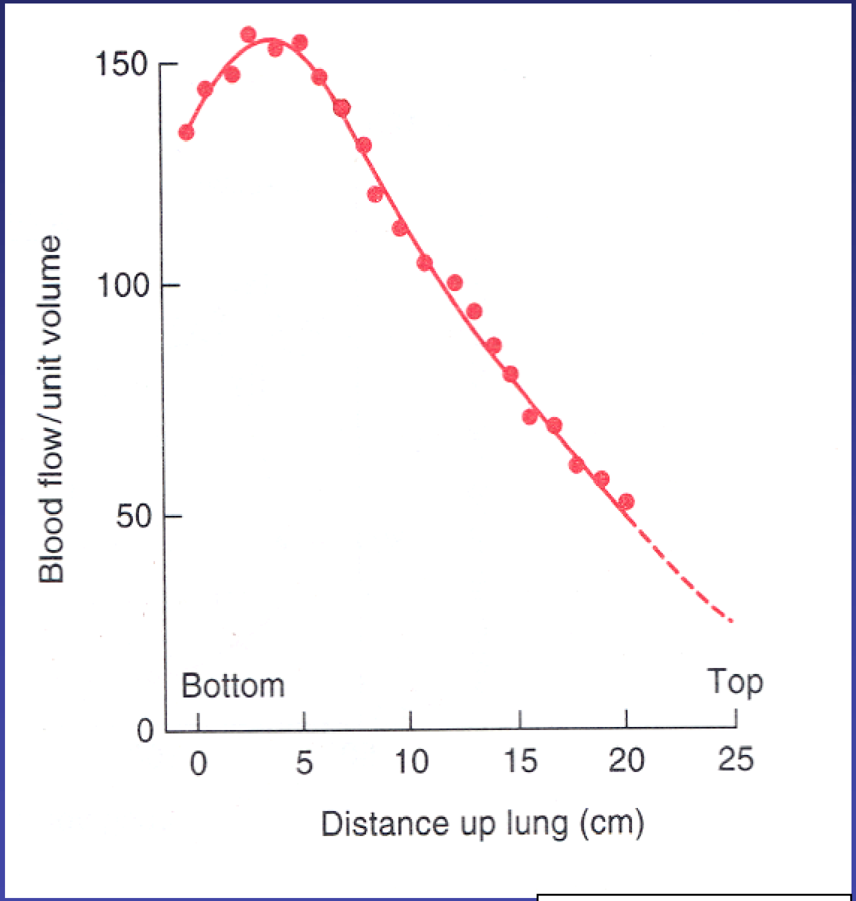
Source: *Pulmonary Physiology*, The McGraw-Hill Companies, Inc., 2007

Control of Pulmonary Vascular Resistance

- Passive Influences on PVR:

Influence	Effect on PVR	Mechanism
↑ Lung Volume (above FRC)	Increase	Lengthening and Compression
↓ Lung Volume (below FRC)	Increase	Compression of Extraalveolar Vessels
↑ Flow, ↑Pressure	Decrease	Recruitment and Distension
Gravity	Decrease in Dependent Regions	Recruitment and Distension
↑ Interstitial Pressure	Increase	Compression
Positive Pressure Ventilation	Increase	Compression and Derecruitment

Regional Pulmonary Blood Flow Depends Upon Position Relative to the Heart



Source: Undetermined

Main PA	15 mmHg
Apex	2 mmHg
Base	25 mmHg

Gravity, Alveolar Pressure and Blood Flow

- Pressure in the pulmonary arterioles depends on both mean pulmonary artery pressure and the vertical position of the vessel in the chest, relative to the heart.

- Driving pressure (gradient) for perfusion is different in the 3 lung zones:

  - Flow in zone may be absent because there is inadequate pressure to overcome alveolar pressure.

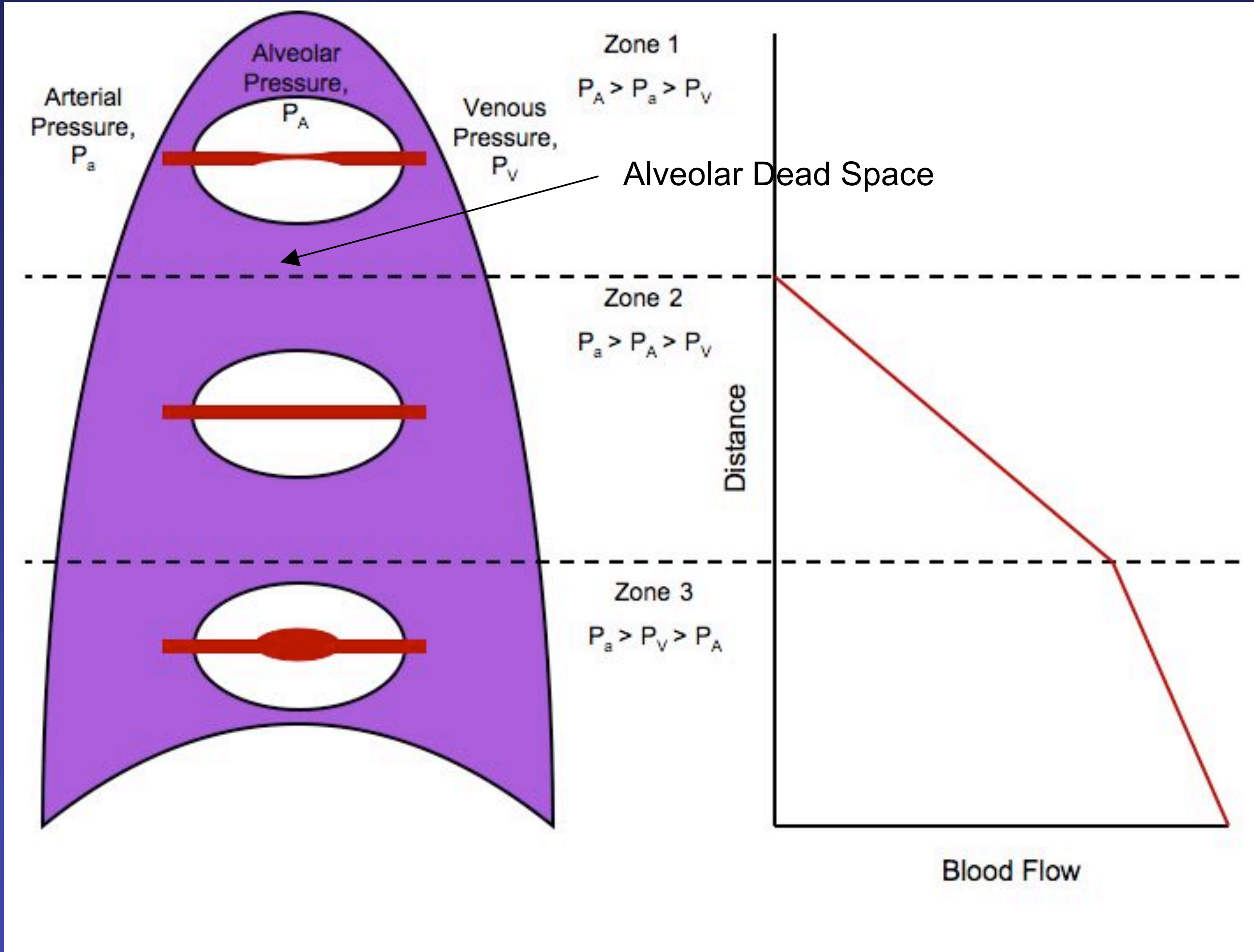
  - Flow in zone 3 is continuous and driven by the pressure in the pulmonary arteriole – pulmonary venous pressure.

  - Flow in zone 2 may be pulsatile and driven by the pressure in the pulmonary arteriole – alveolar pressure (collapsing the capillaries).

Gravity, Alveolar Pressure, and Blood Flow

Typically no zone 1 in normal healthy person

Large zone 1 in positive pressure ventilation + PEEP



Gravity Influences Pressure

Control of Pulmonary Vascular Resistance

- **Active Influences on PVR:**

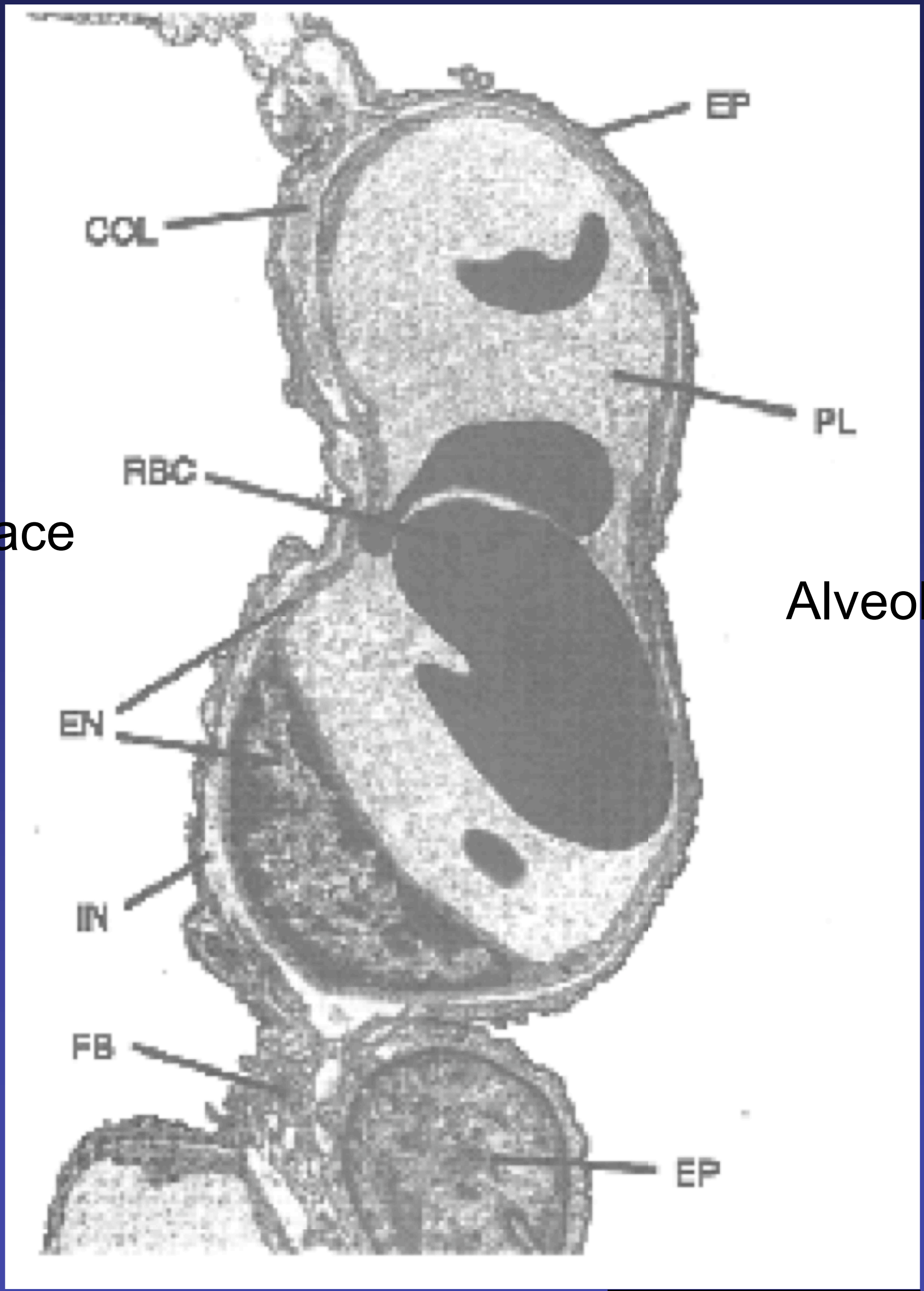
Increase	Decrease
Sympathetic Innervation	Parasympathetic Innervation
α-Adrenergic agonists	Acetylcholine
Thromboxane/PGE2	β-Adrenergic Agents
Endothelin	PGE1
Angiotensin	Prostacycline
Histamine	Nitric oxide
Alveolar Hypoxemia	Bradykinin

Hypoxic Pulmonary Vasoconstriction

- Alveolar hypoxia causes active vasoconstriction at level of pre-capillary arteriole.
- Mechanism is not completely understood:
  - Response occurs locally and does not require innervation.
  - Mediators have not been identified.
  - Graded response between pO2 levels of 100 down to 20 mmHg.
- Functions to reduce the mismatching of ventilation and perfusion.
- Not a strong response due to limited muscle in pulmonary vasculature.
- General hypoxemia (high altitude or hypoventilation) can cause extensive pulmonary artery vasoconstriction.

Barrier Function of Alveolar Wall

- Capillary endothelial cells:
  - permeable to water, small molecules, ions.
  - barrier to proteins.
- Alveolar epithelial cells:
  - more effective barrier than the endothelial cells.
  - recently found to pump both salt and water from the alveolar space.



Alveolar airspace

Alveolar airspace

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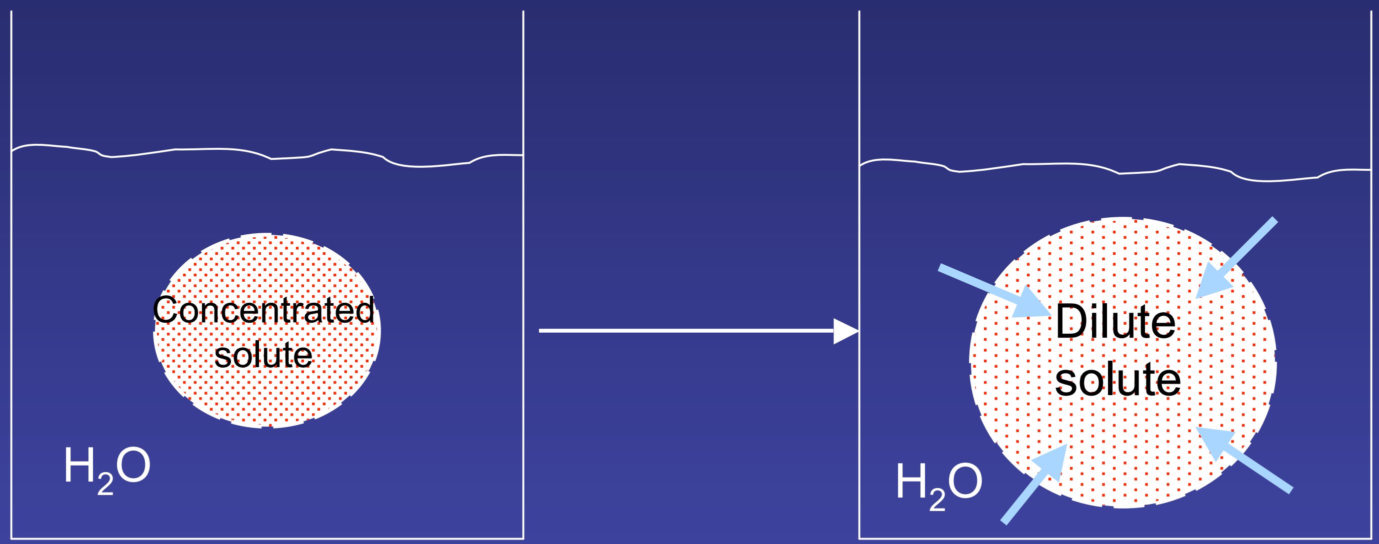
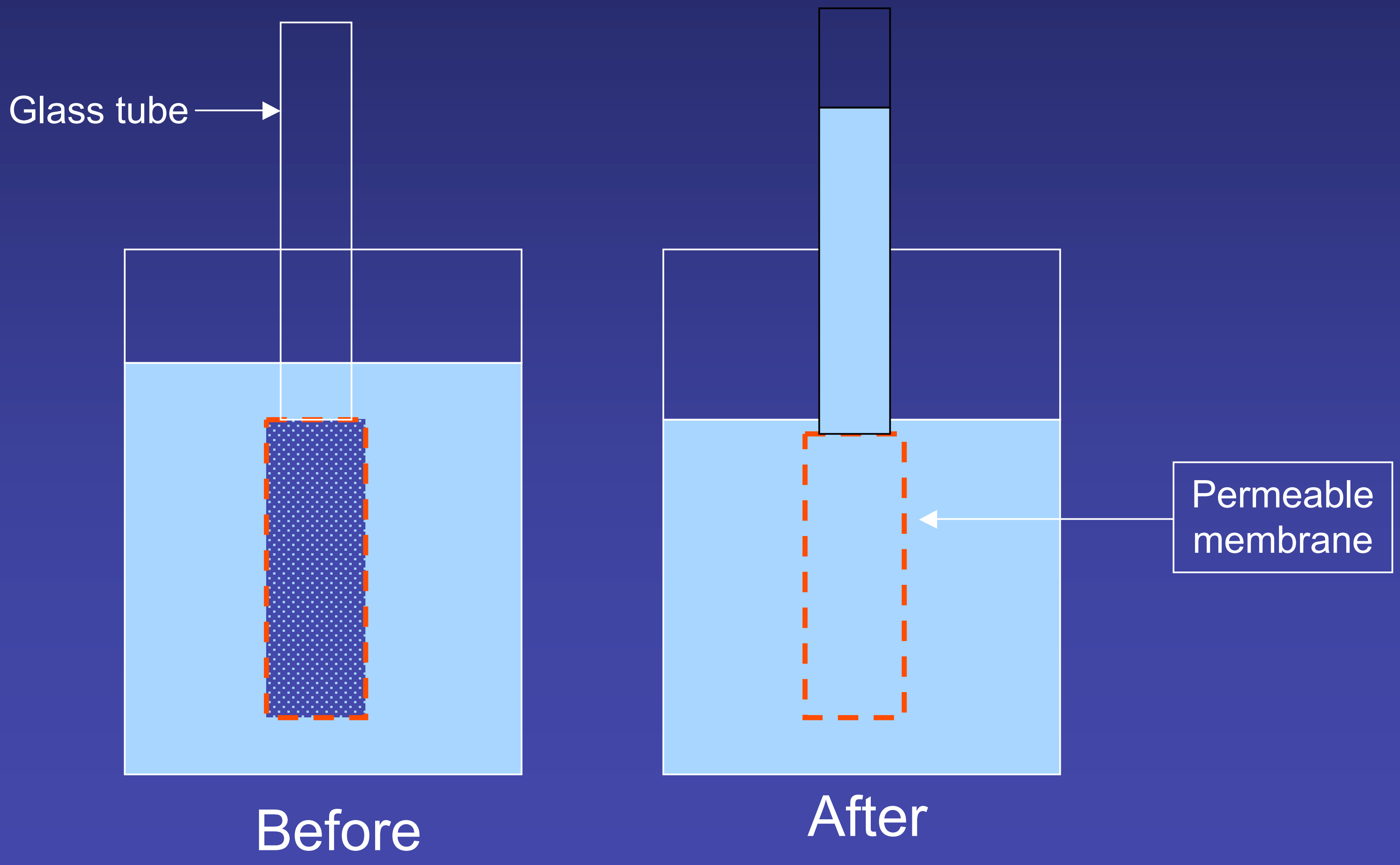
Fluid Movement Due to Osmotic Pressure



Water moves through the semi-permeable membrane down a concentration gradient to dilute the solute.

Osmotic Pressure Gradient Can Move Fluid Against Hydrostatic Pressure



Osmotic Gradient Counteracts Hydrostatic Gradient

- Hydrostatic pressure in the pulmonary capillary bed > hydrostatic pressure in the interstitium
  - hydrostatic pressure drives fluid from the capillaries into the pulmonary interstitium

- Osmotic pressure in the plasma > osmotic pressure in the interstitium
  - osmotic pressure normally would draw fluid from the interstitial space into the capillaries

Starling's Equation

$$Q=K[(Pc-Pi) - \sigma(\pi c-\pi i)]$$

Q = flux out of the capillary
K = filtration coefficient
Pc and Pi = capillary and interstitial hydrostatic pressures
πc and πi = capillary and interstitial osmotic pressures
σ = reflection (sieving) coefficient

Normally Starling's Forces Provide Efficient Protection

- Normal fluid flux from the pulmonary capillary bed is approximately 20 ml/hr.
  - recall that cardiac output through the pulmonary capillaries at rest is ~5 l/min.
  - < 0.0066% leak.

- Abnormal increase in fluid flux can result from:
  - Increased hydrostatic pressure gradient (cardiogenic pulmonary edema).
  - Decreased osmotic pressure gradient (cirrhosis, nephrotic syndrome).
  - Increased protein permeability of the capillary wall (ARDS).