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Physical Principles of Gas Exchange-
V/Q Ratio

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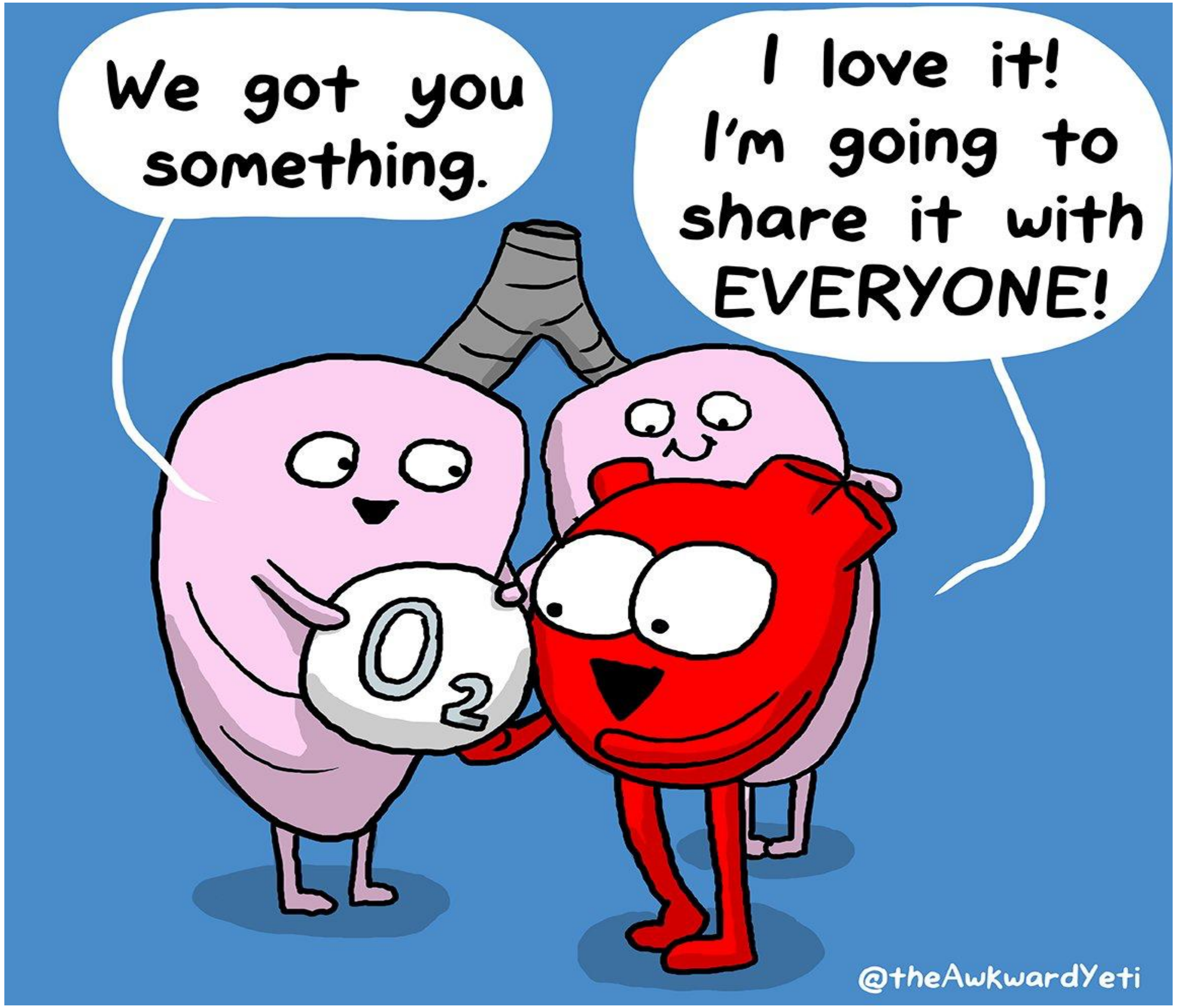
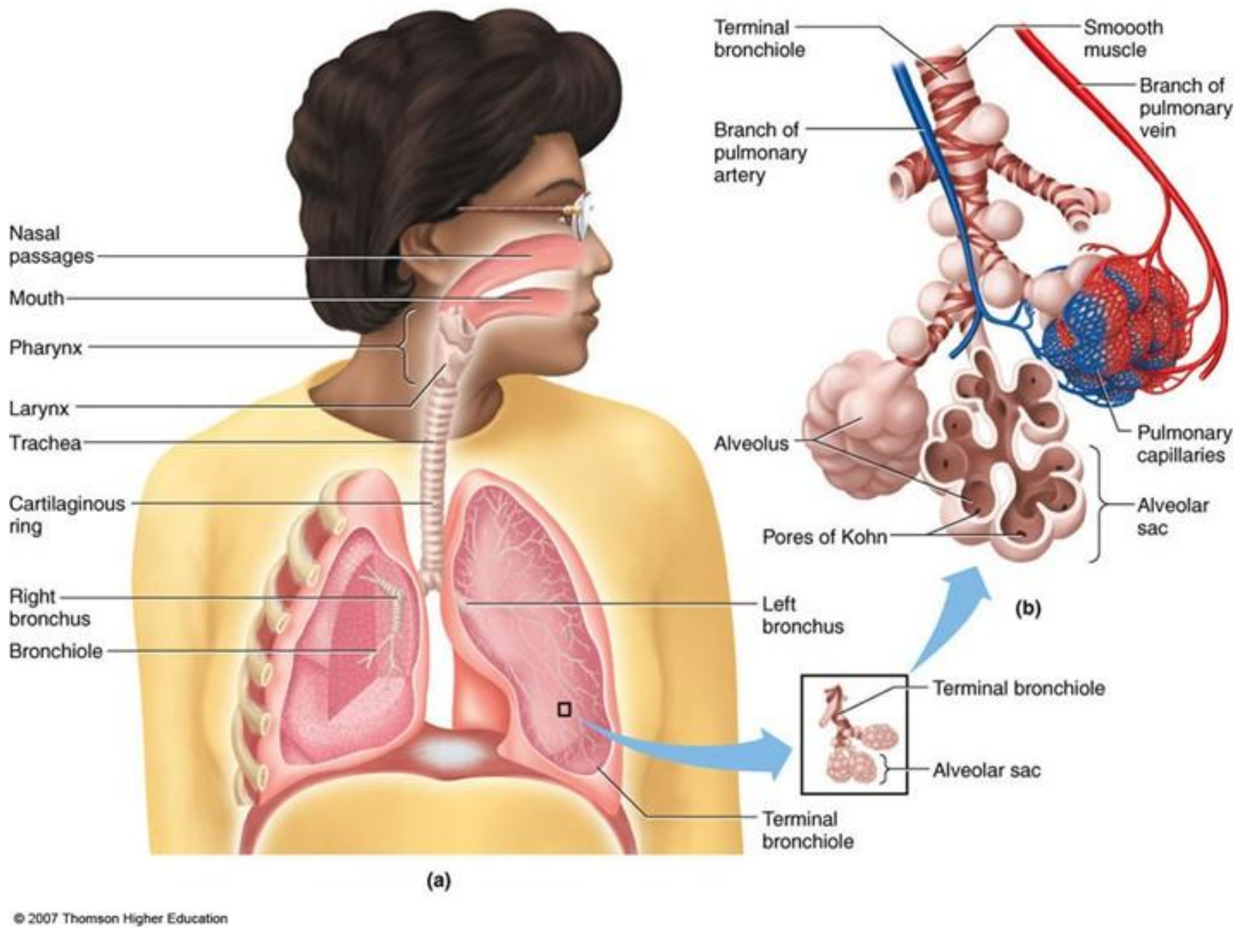
Learning Objectives

- List the factors that affect diffusive transport of a gas between alveolar gas and pulmonary capillary blood.
- Explain the concept of physiological dead space and physiological shunt
- Describe the effect of Ventilation perfusion Ratio on alveolar gas concentration
- Identify the average V/Q ratio in normal lung
- Describe the abnormalities of ventilation perfusion ratio in a normal lung and COPD

Respiration …

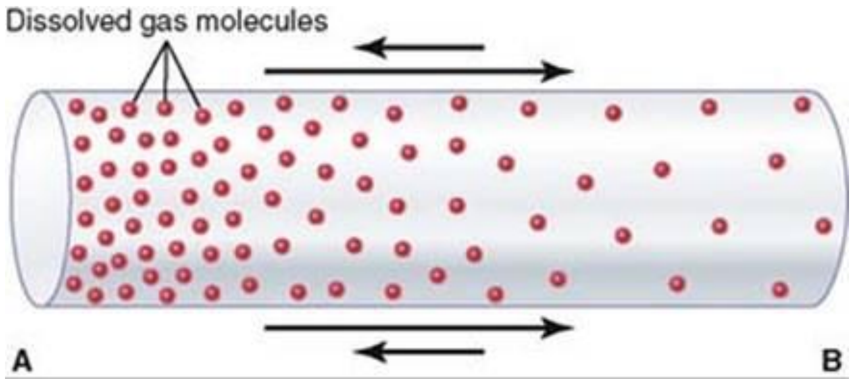


Let’s Recall our previous knowledge….



Physical Principles of Gas Exchange – key concepts

- The purpose of the respiratory system is to perform gas exchange.
- Gases tend to equalize their pressure in two regions that are connected; in such a situation, the respective pressure of each gas is known as that gas's " partial pressure."
- A gas will move from an area where its partial pressure is higher to an area where its partial pressure is lower, and the greater the difference in pressure, the more rapidly the gases will move.



Gaseous Diffusion at Alveolar level

- Alveoli are the basic units of gas exchange
- There are 300 million Alveoli. Their size is variable –

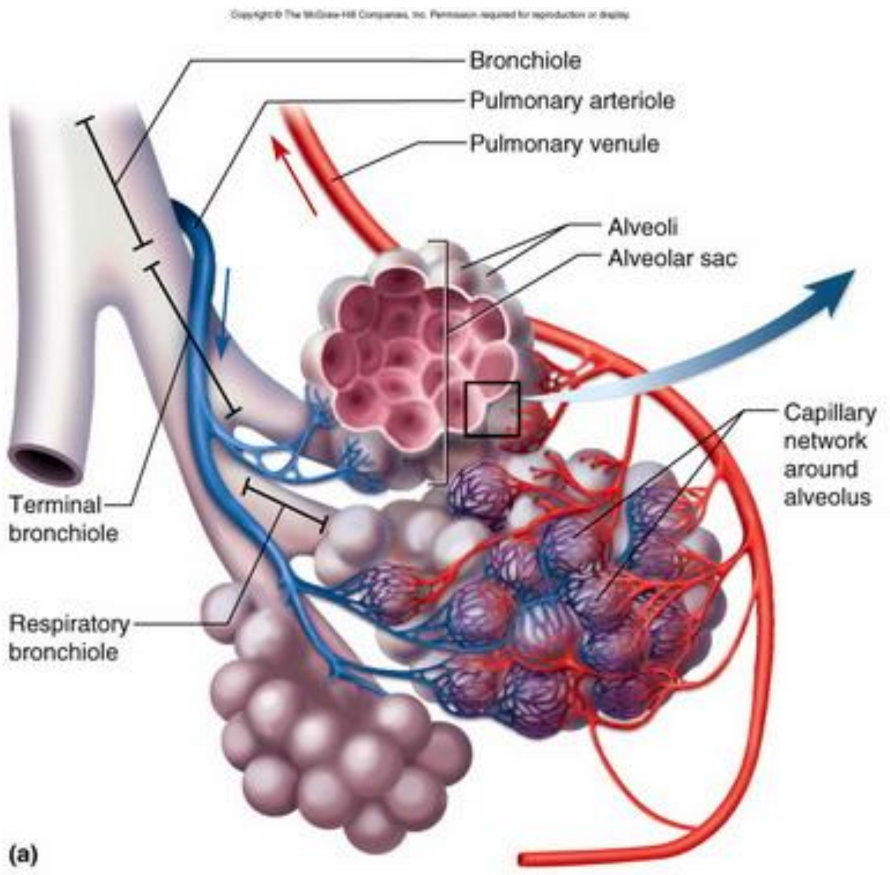
□largest in upper lung &

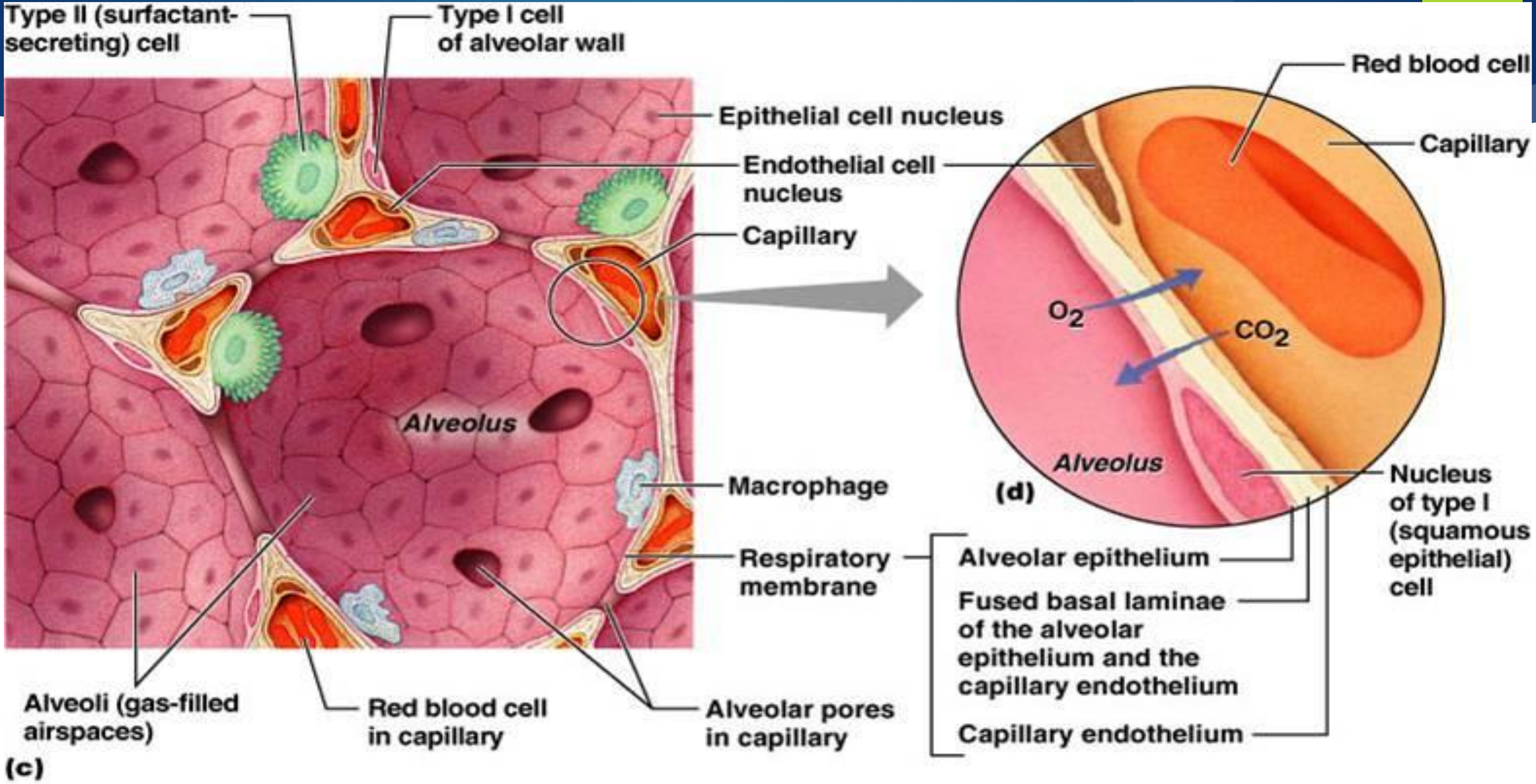
□smallest in lower lungs

- Blood gas barrier is formed by alveolar epithelium, capillary endothelium, basement membrane of each and the interstitial space in between
- Alveolar epithelium is composed of –

□mainly type I squamous epithelial cells and

□some type II alveolar cells that produce surfactant





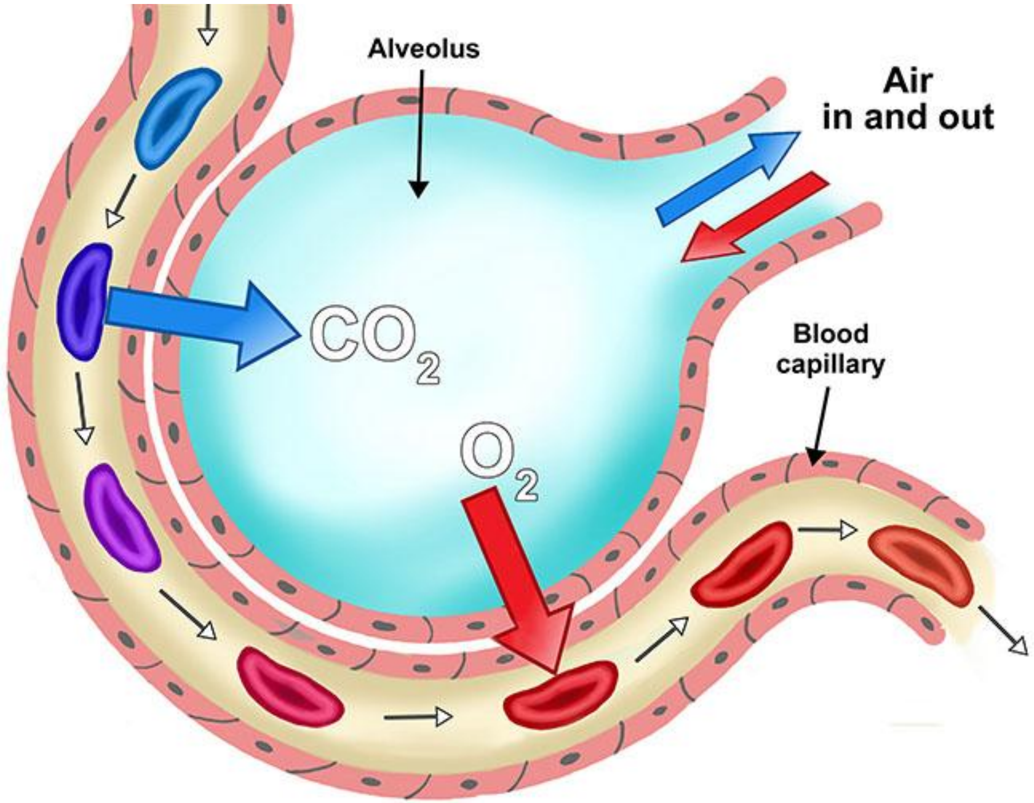
Type II (surfactant-secreting) cell
Type I cell of alveolar wall
Epithelial cell nucleus
Endothelial cell nucleus
Capillary
Alveolus
Macrophage
Respiratory membrane
Alveoli (gas-filled airspaces)
Red blood cell in capillary
Alveolar pores in capillary
(c)
Red blood cell
Capillary
O_2
CO_2
Alveolus
(d)
Nucleus of type I (squamous epithelial) cell
Alveolar epithelium
Fused basal laminae of the alveolar epithelium and the capillary endothelium
Capillary endothelium

Diffusion of gases through the respiratory membrane

Depends on membrane's thickness,

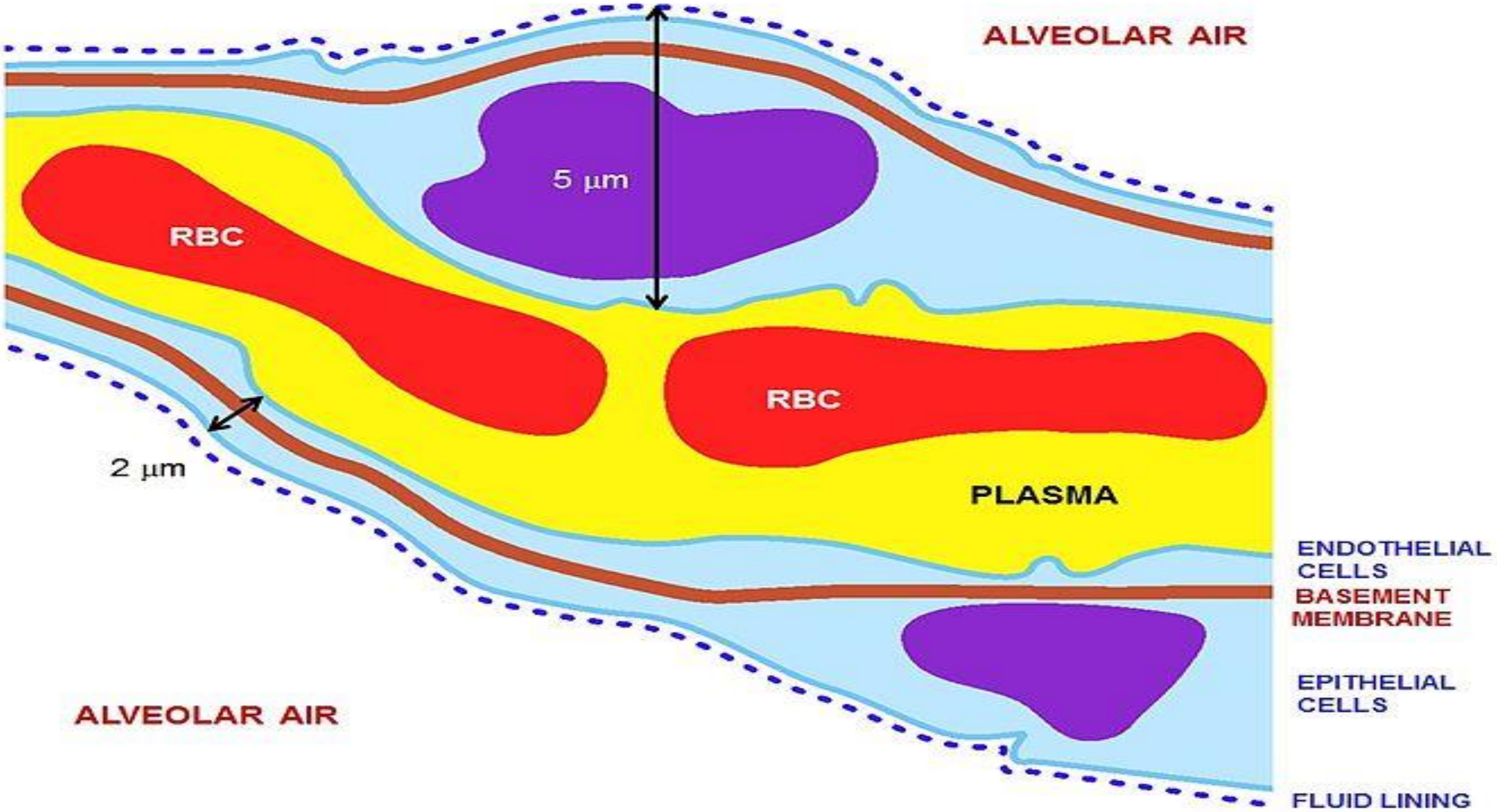
- the diffusion coefficient of gas,
- surface areas of membrane,
- partial pressure of gases in alveoli and blood

Increased ventilation or increased pulmonary capillary blood flow increases gas exchange



Diffusion barrier

- The diffusion barrier in the lungs consists of the following layers:
- Alveolar **epithelium**
- Tissue **fluid**
- Capillary **endothelium**
- **Plasma**
- **Red cell** membrane


ALVEOLAR AIR
5 µm
RBC
RBC
2 µm
PLASMA
ENDOTHELIAL CELLS
BASEMENT MEMBRANE
EPITHELIAL CELLS
ALVEOLAR AIR
FLUID LINING

Factors That Affect The Rate of Diffusion

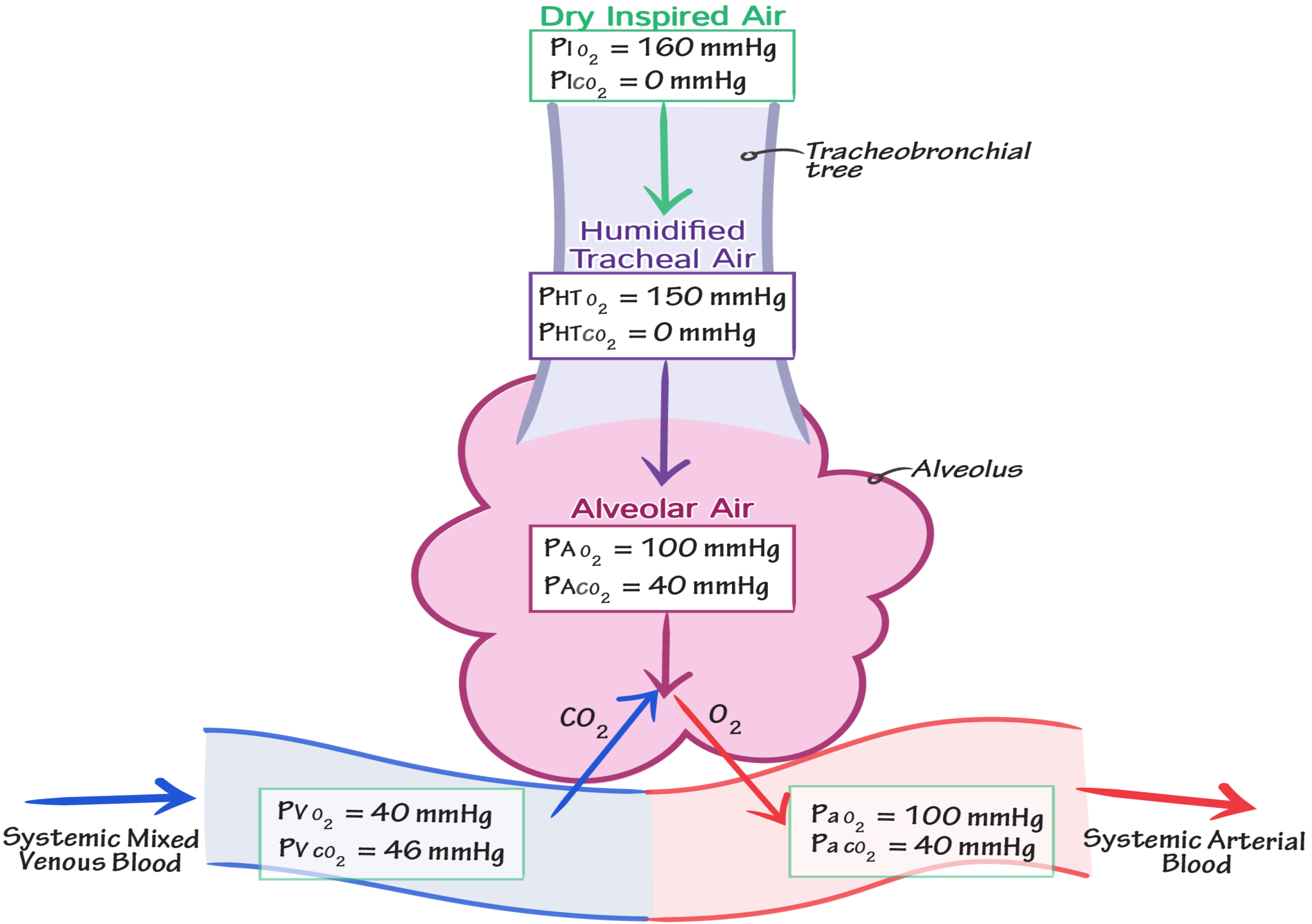
- **Membrane thickness** –

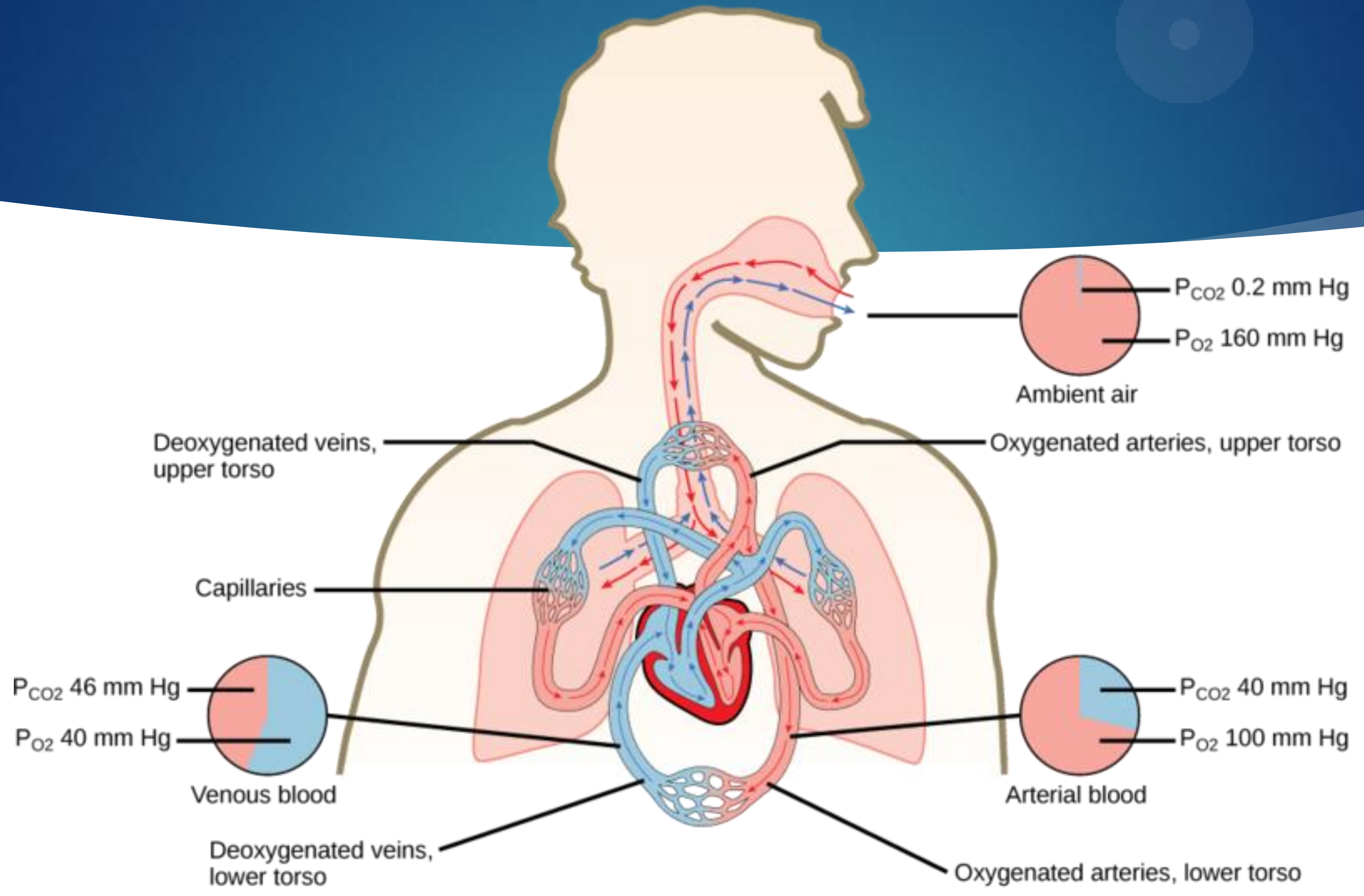
the thinner the membrane, the faster the rate of diffusion. The diffusion barrier in the lungs is extremely thin , however some conditions cause thickening of the barrier, thereby impairing diffusion. Examples include:

  - Fluid in the interstitial space (pulmonary oedema).
  - Thickening of the alveolar membrane (pulmonary fibrosis).

Factors That Affect The Rate of Diffusion

- **Membrane surface area** – the larger the surface area, the faster the rate of diffusion. The lungs normally have a very large surface area for gas exchange due to the alveoli.
  - Diseases such as **emphysema** lead to the destruction of the **alveolar architecture**, leading to the formation of large air-filled spaces known as **bullae.** This reduces the surface area available and slows the rate of gas exchange.
- **Pressure difference across the membrane**
- **Diffusion coefficient of the gas**


Dry Inspired Air
$P_{I_{O_2}}$ = 160 mmHg
$P_{I_{CO_2}}$ = 0 mmHg
Tracheobronchial tree
Humidified Tracheal Air
$P_{HT_{O_2}}$ = 150 mmHg
$P_{HT_{CO_2}}$ = 0 mmHg
Alveolus
Alveolar Air
$P_{A_{O_2}}$ = 100 mmHg
$P_{A_{CO_2}}$ = 40 mmHg
CO_2
O_2
$P_{V_{O_2}}$ = 40 mmHg
$P_{V_{CO_2}}$ = 46 mmHg
Systemic Mixed Venous Blood
$P_{a_{O_2}}$ = 100 mmHg
$P_{a_{CO_2}}$ = 40 mmHg
Systemic Arterial Blood



P_{CO2} 0.2 mm Hg
P_{O2} 160 mm Hg
Ambient air
Deoxygenated veins, upper torso
Oxygenated arteries, upper torso
Capillaries
P_{CO2} 46 mm Hg
P_{O2} 40 mm Hg
Venous blood
P_{CO2} 40 mm Hg
P_{O2} 100 mm Hg
Arterial blood
Deoxygenated veins, lower torso
Oxygenated arteries, lower torso

Partial Pressure of Gases

	Inspired air	Alveolar air
H_2O	Variable	47 mmHg
CO_2	000.3 mmHg	40 mmHg
O_2	159 mmHg	105 mmHg
N_2	601 mmHg	568 mmHg
Total pressure	760 mmHg	760 mmHg

Partial Pressures of Respiratory Gases as They Enter and Leave the Lungs (at Sea Level)

	Atmospheric Air (mm Hg)	Humidified Air (mm Hg)	Alveolar Air (mm Hg)	Expired Air (mm Hg)
N2	597.0 (78.62%	563.4 (74.09%)	569.0 (74.9%)	566.0 (74.5%)
O2	159.0 (20.84%	149.3 (19.67%)	104.0 (13.6%)	120.0 (15.7%)
CO2	0.3(0.04%	0.3 (0.04%	40.0 (5.3%)	27.0(3.6%
H2O	3.7(0.5)	47.0 (6.20%)	47.0 (6.2%)	47.0 (6.2%)
Total	760.0 (100.0%)	760.0 (100.0%)	760.0 (100.0%)	760.0 (100.0%)

Ventilation /Perfusion Ratio

ALVEOLAR VENTILATION

- **The amount of air utilized for gaseous exchange every minute**
- The air trapped in the respiratory passages does not take part in gaseous exchange
- Alveolar ventilation:

 (Tidal volume – dead space volume) x Respiratory rate

 = (500 - 150) x 12

 = 4,200 ml

 = 4.2 ml/min

VENTILATION

- The process by which fresh air enters the lungs and an equal volume of air leaves the lungs.
- It is of two types:
- Pulmonary ventilation
- Alveolar ventilation

PULMONARY VENTILATION

- **It's the volume of air coming in and out of the lungs per minute in quiet breathing**
- Also called Respiratory Minute Volume
- Pulmonary ventilation =

  Tidal volume x respiratory rate

  = 500 ml x 12/min

  = 6000 ml/ min

PERFUSION (Q)

The movement of blood into the lungs through pulmonary capillaries

(Q – from German Quellen, meaning to gush)

- Pulmonary blood flow 5 L/min
- These volume going to be spreaded all along the alveolar capillary membrane which has 50 to 100 m^2 surface area

Distribution of Pulmonary Perfusion

- Due to gravitational influence the lower dependent areas receive more blood
- Upper zone – nondependent areas are less perfused

VENTILATION- PERFUSION RATIO

- It's the ratio of alveolar ventilation to blood flow in lung.
- In normal individual at rest alveolar ventilation (V) is 4L/min and pulmonary blood flow (Q) is 5 lit/min.
- **The normal ventilation-perfusion ratio is 0.8.**
- Ensuring that the ventilation and perfusion of the lungs are **adequately matched** is vital for ensuring continuous **delivery** of oxygen and **removal** of carbon dioxide from the body.

Effect of ventilation/perfusion ratio on alveolar gas concentration

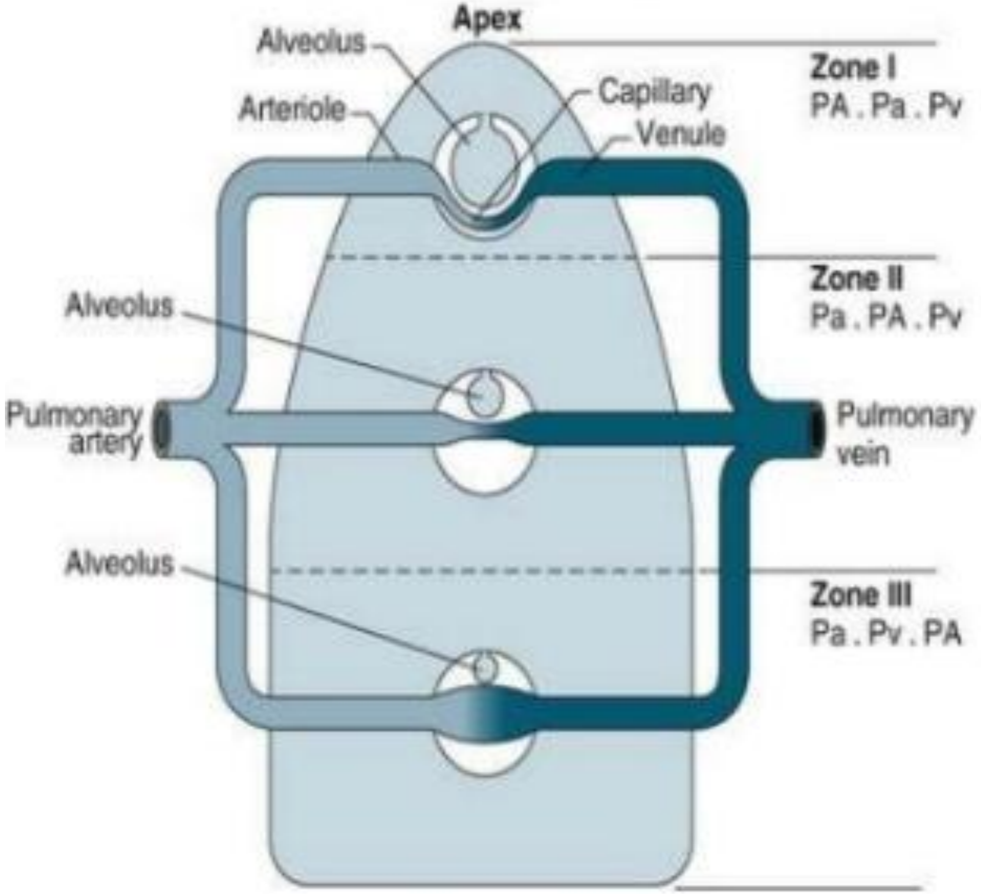
- Two factors determine the PO_2 and the PCO_2 in the alveoli:

(1) the rate of alveolar ventilation and

(2) the rate of transfer of oxygen and carbon dioxide through the respiratory membrane.

- normally to some extent, and especially in many lung diseases, some areas of the lungs are well ventilated but have almost no blood flow, whereas other areas may have excellent blood flow but little or no ventilation.

REGIONAL GAS EXCHANGE IN THE LUNG



- A normal lung has different V/Q ratios in its different regions. This may be attributed to the pull of gravity and the heart's location relative to the lung.
- Airflow and blood flow increase down the lung, but the differences in perfusion are greater than the differences in ventilation.
- Blood flow is proportionately greater than ventilation at the base- lower V/Q ratio
- Ventilation is proportionately greater than blood flow at the apex- higher V/Q ratio.

Dead Space

- **Part of respiratory tract where gaseous exchange does not take place**. The air present in dead space is known as dead space air
- Ventilation is present but no blood flow. This leads to wasted ventilation
- There are three types of dead space:
  - Anatomical
  - Alveolar
  - physiological

Anatomical Dead Space

- The dead space areas are:
- Nose,
- Pharynx
- Trachea
- Bronchi
- Bronchioles (upto terminal bronchioles)

In Conducting airways: about **150mL** in an average adult

Alveolar dead space

- This occurs when areas of the lung are being ventilated but not being perfused and this leads to what is known as V/Q mismatch.
- Large increases in alveolar dead space commonly occur in the following conditions:
- pulmonary edema,
- pulmonary embolism

Physiological Dead Space

- This is a combination of alveolar and anatomical dead space added together.
- Part of the tidal volume which does not participate in gas exchange.
- The quantity of CO_2 exhaled from the healthy alveoli will be diluted by anatomical dead space and alveoli with no perfusion.

Physiologic Dead Space

$$V_d = V_t * \frac{P_aCO_2 - P_ECO_2}{P_aCO_2}$$

V_d : dead space volume in 'mL'

V_t : tidal volume in 'mL'

P_aCO_2 : partial pressure of carbon dioxide in 'mmHg'

P_ECO_2 : partial pressure of carbon dioxide in the expired air in 'mmHg'

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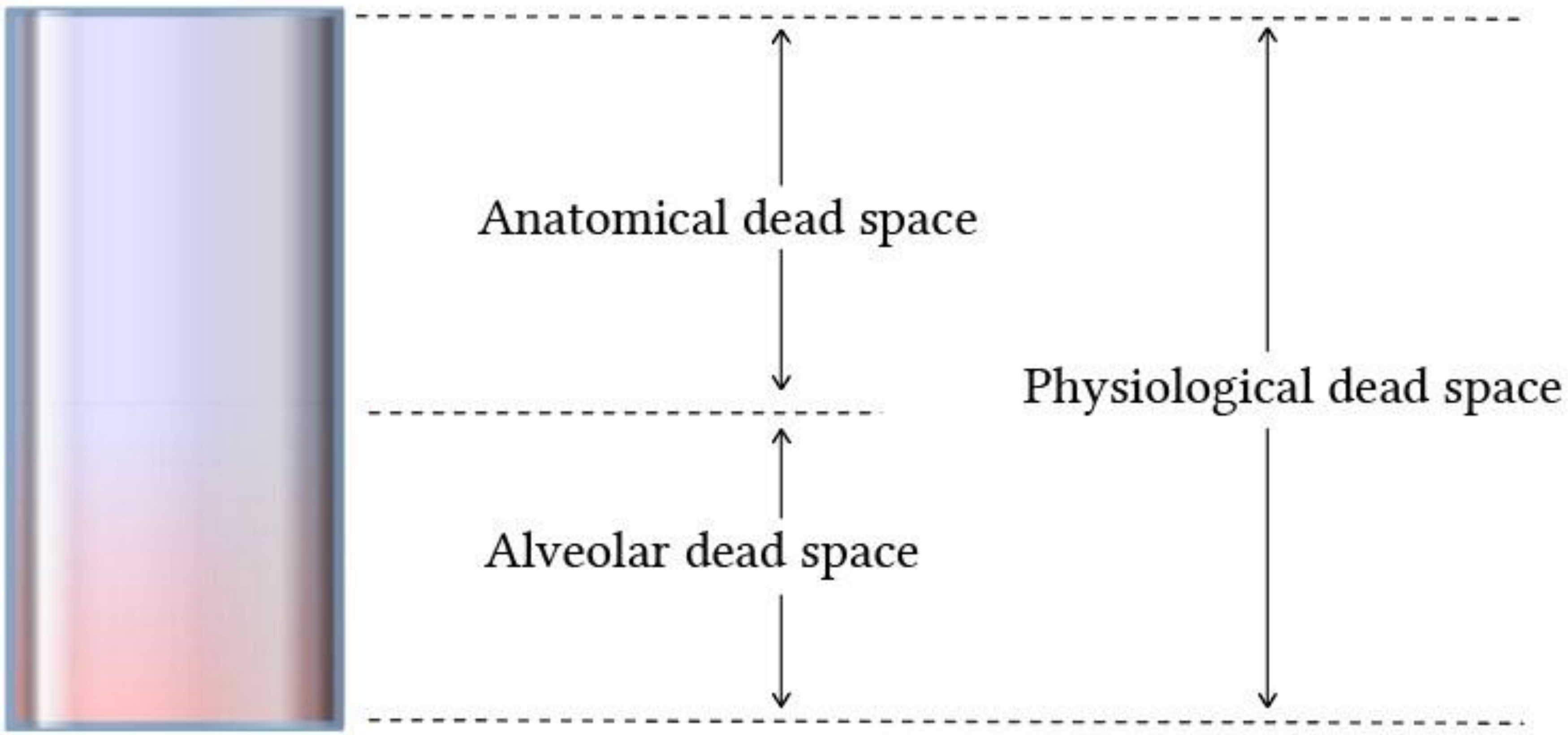
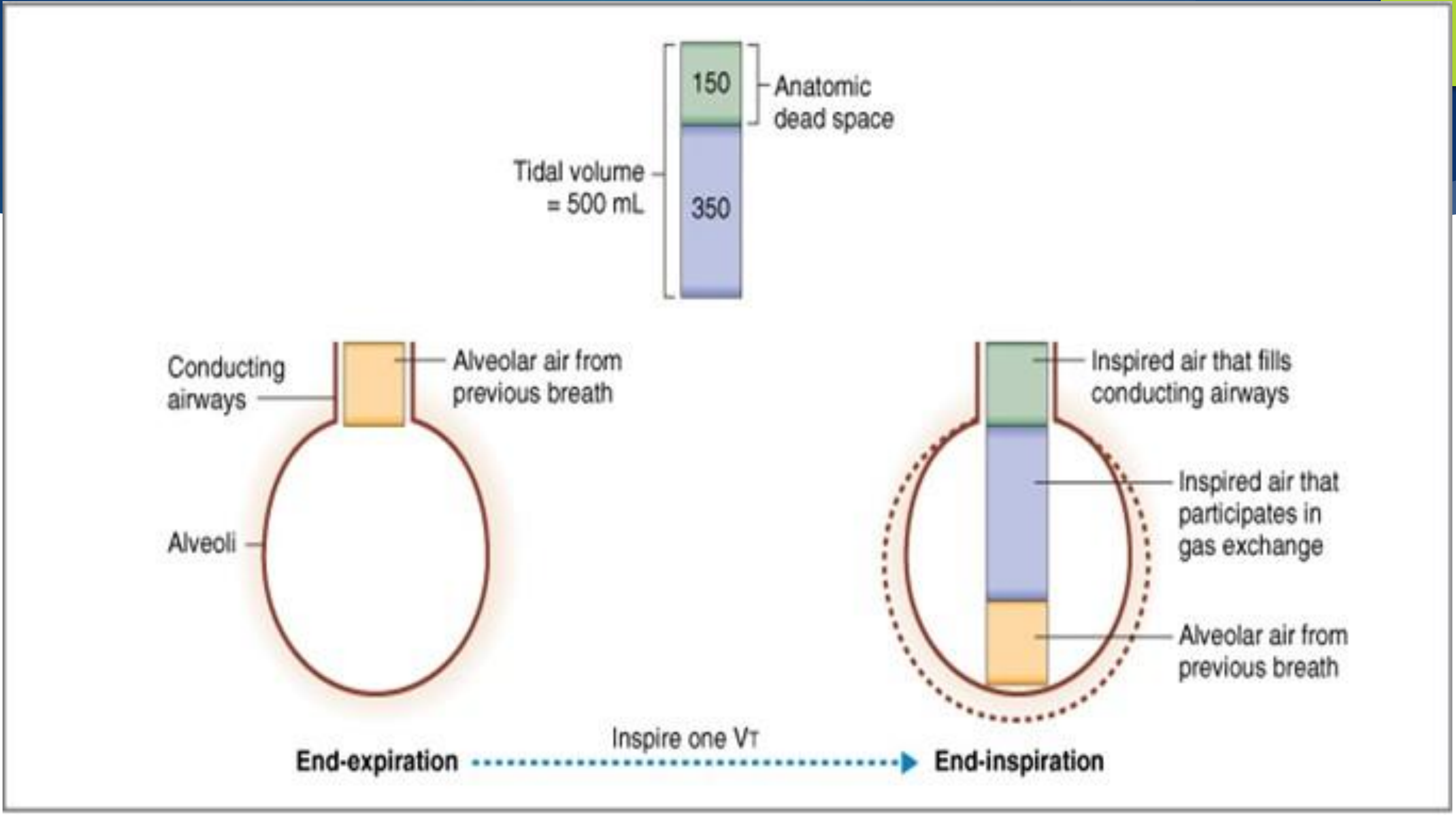
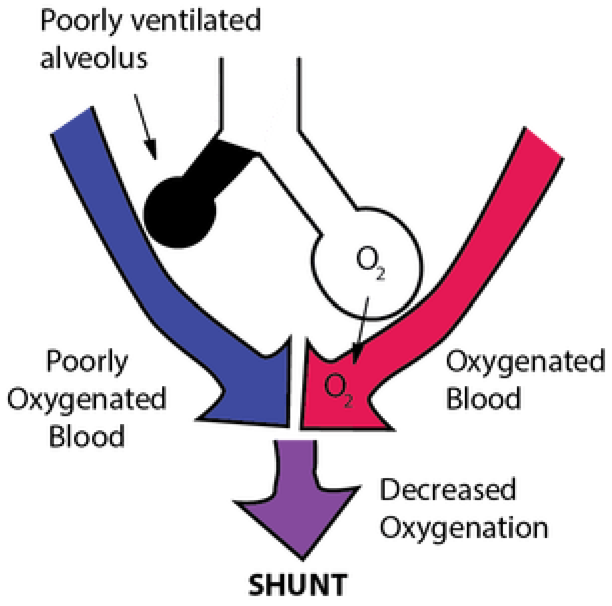

Anatomical dead space
Physiological dead space
Alveolar dead space


150
350
Anatomic dead space
Tidal volume = 500 mL
Conducting airways
Alveolar air from previous breath
Alveoli
Inspired air that fills conducting airways
Inspired air that participates in gas exchange
Alveolar air from previous breath
Inspire one VT
End-expiration
End-inspiration

Physiological Shunt

- Shunt is the opposite of dead space and consists of alveoli that are perfused, but not ventilated.
- blood flowing past poorly ventilated alveoli doesn't pick up additional oxygen.
- This poorly oxygenated blood returns to the heart and mixes with oxygenated blood coming from other areas of the lungs that are ventilated.
- The mixture lowers the total oxygen content of the arterial blood, producing hypoxemia.
- the larger the shunt, the lower the oxygen content.

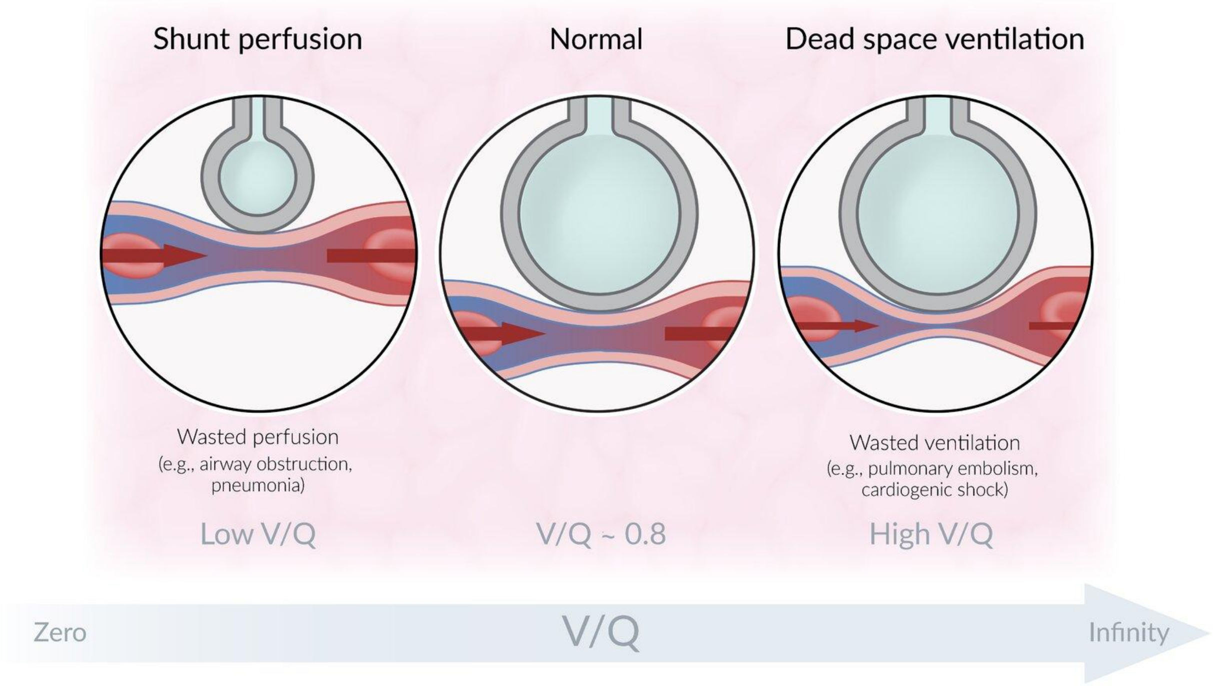


Causes of Shunt

- Common causes of shunt occur in lung tissue disease and include:
- pneumonia and pulmonary edema: some alveoli filled with fluid
- tissue trauma: alveolar wall swelling
- atelectasis: collapse of alveoli from failure to expand, or absorbtion of the air out of the alveoli without replacing it
- mucous plugging: air can't get into the alveoli
- pulmonary arteriovenous fistulas

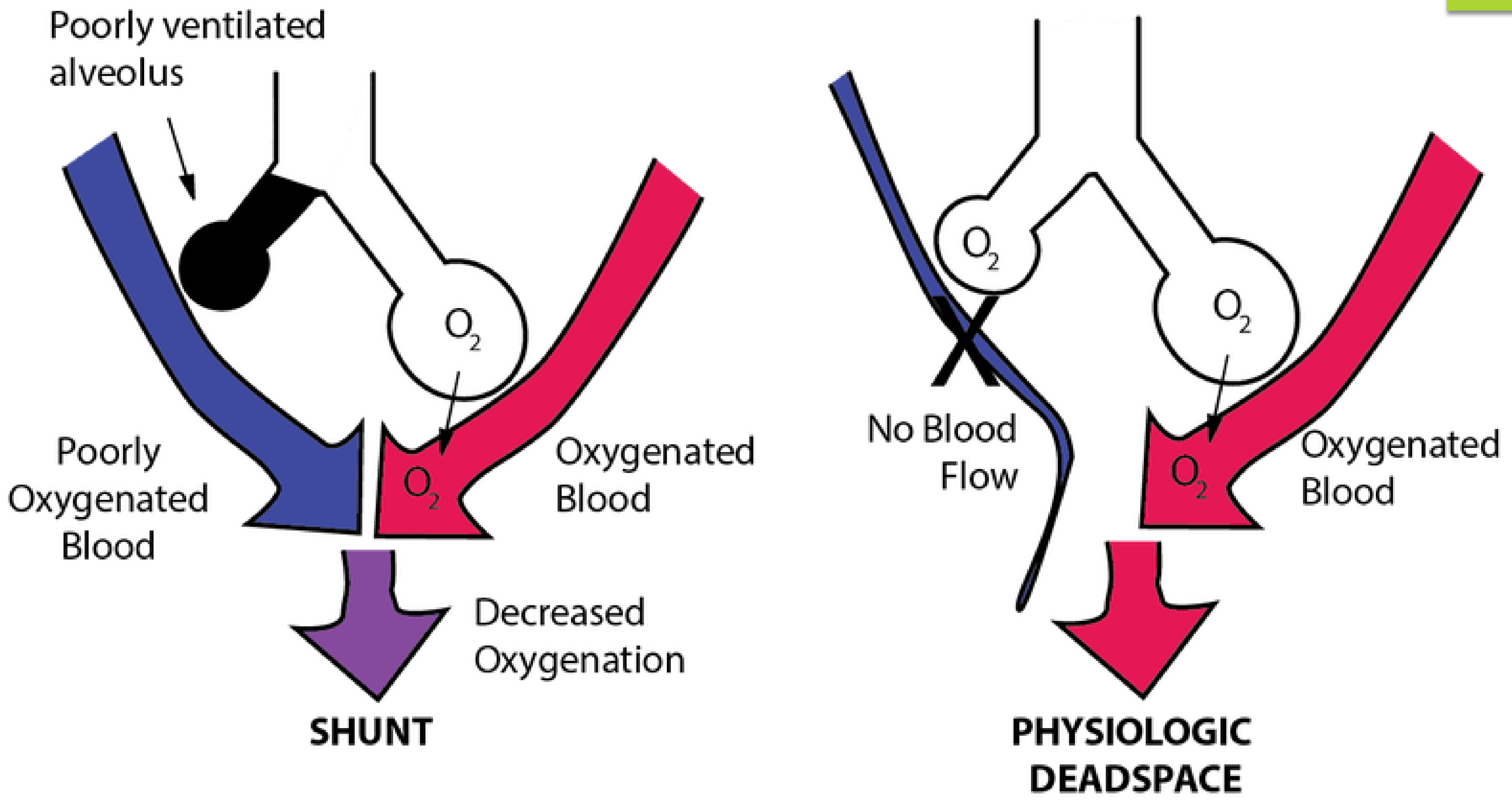
- **A woman has a respiratory rate of 18, a tidal volume of 350 mL, and a dead space of 100 mL. What is her alveolar ventilation?**
- a. 4.0 L
- b. 4.5 L
- c. 5.0 L
- d. 5.5 L
- e. 6.0 L

V/Q Mismatch

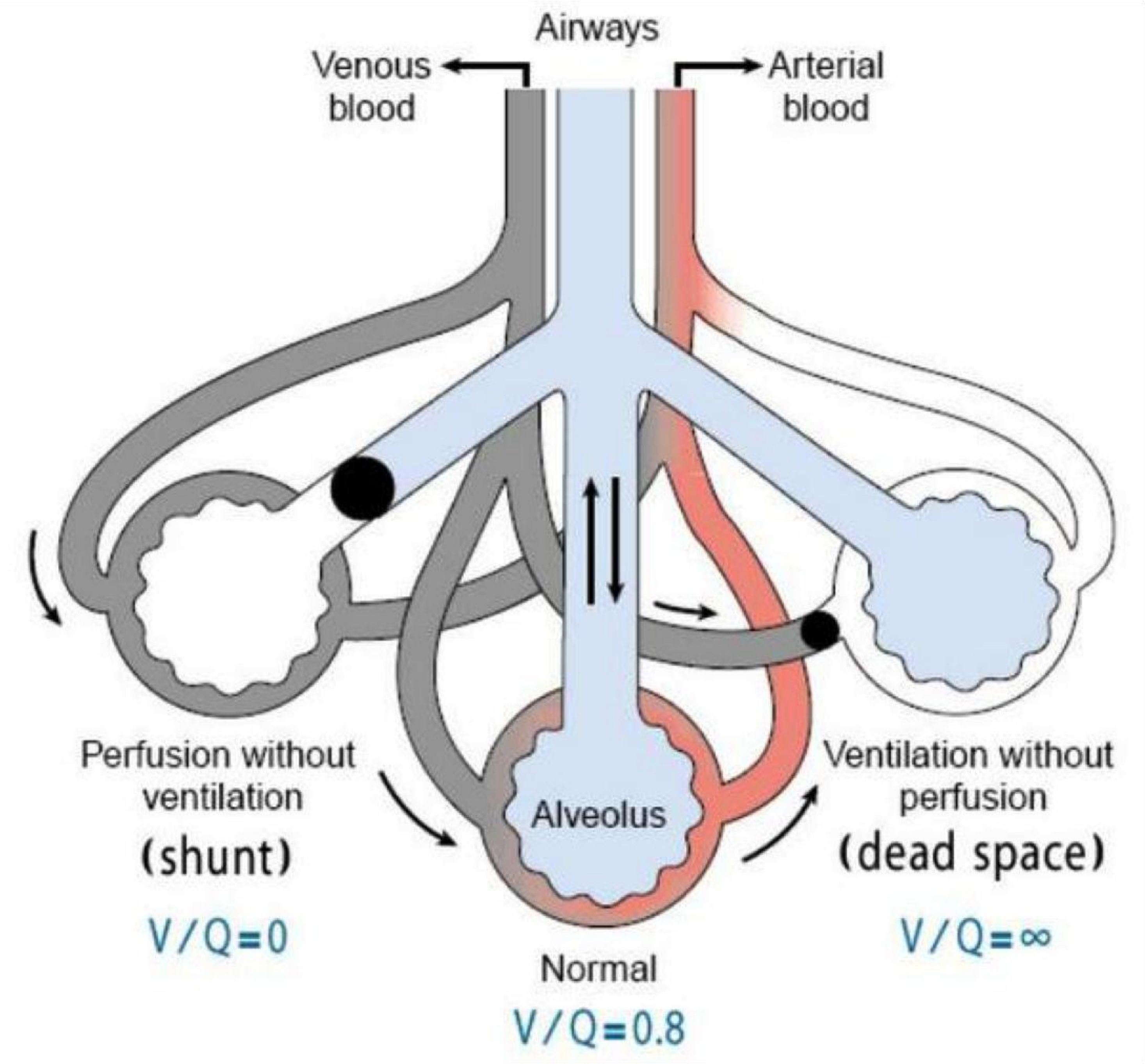

Shunt perfusion
Normal
Dead space ventilation
Wasted perfusion
(e.g., airway obstruction, pneumonia)
Low V/Q
V/Q ~ 0.8
Wasted ventilation
(e.g., pulmonary embolism, cardiogenic shock)
High V/Q
Zero
V/Q
Infinity

Extreme alterations of V/Q

- An area with perfusion but no ventilation (and thus a V/Q of zero) is termed **shunt**.
- An area with ventilation but no perfusion (and thus a V/Q undefined though approaching infinity) is termed "**dead space**".



Poorly ventilated alveolus
O_2
O_2
Poorly Oxygenated Blood
Oxygenated Blood
Decreased Oxygenation
SHUNT
O_2
O_2
No Blood Flow
O_2
Oxygenated Blood
PHYSIOLOGIC DEADSPACE


Airways
Venous blood
Arterial blood
Perfusion without ventilation
(shunt)
V/Q=0
Alveolus
Normal
V/Q=0.8
Ventilation without perfusion
(dead space)
V/Q=∞

Alveolar Oxygen and Carbon Dioxide Partial Pressures When V/Q Equals Zero

- When V/Q is equal to zero—that is, without any alveolar ventilation—the air in the alveolus comes to equilibrium with the blood oxygen and carbon dioxide because these gases diffuse between the blood and the alveolar air.
- Because the blood that perfuses the capillaries is venous blood returning to the lungs from the systemic circulation, it is the gases in this blood with which the alveolar gases equilibrate.
- the normal venous blood has a **PO_2 of 40 mm Hg and a PCO_2 of 45 mm Hg.** Therefore, these are also the normal partial pressures of these two gases in alveoli that have blood flow but no ventilation.

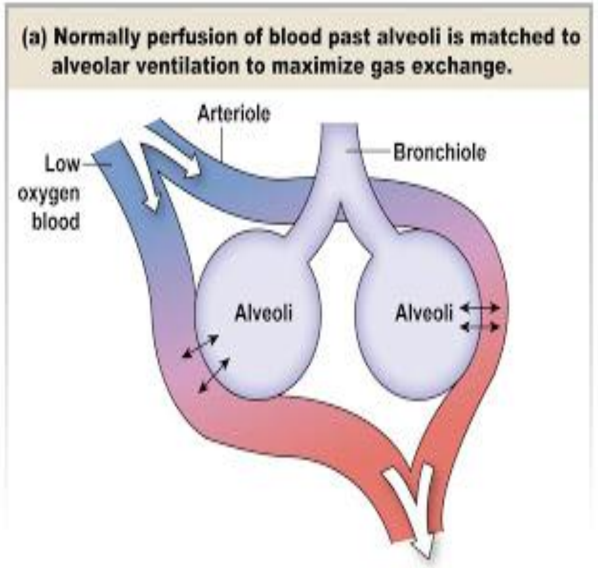
Alveolar Oxygen and Carbon Dioxide Partial Pressures When V/Q Equals Infinity

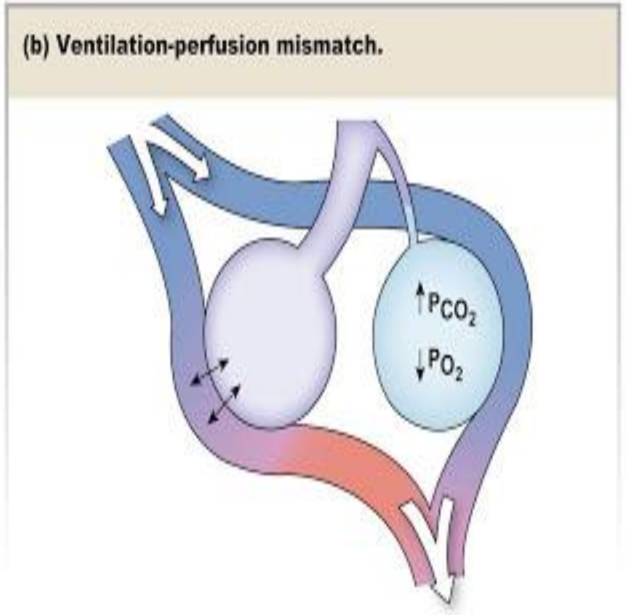
- There is no capillary blood flow to carry oxygen away or to bring carbon dioxide to the alveoli.
- instead of the alveolar gases coming to equilibrium with the venous blood, the alveolar air becomes equal to the humidified inspired air.
- So the air that is inspired loses no oxygen to the blood and gains no carbon dioxide from the blood.
- And because normal inspired and humidified air has a **PO_2 of 149 mm Hg and a PCO_2 of 0 mm Hg**, these will be the partial pressures of these two gases in the alveoli.

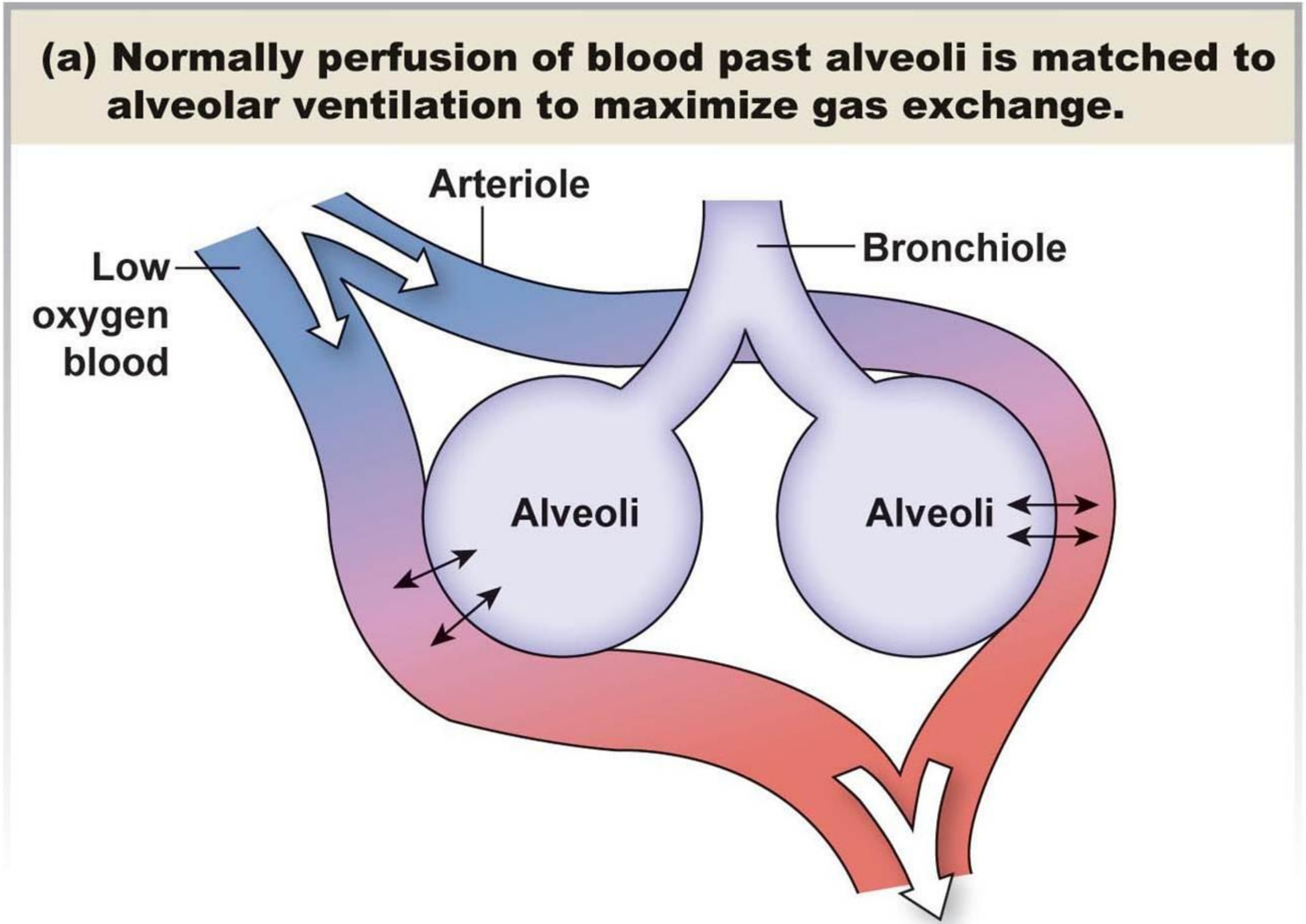
V and Q Matching

V and Q Matching

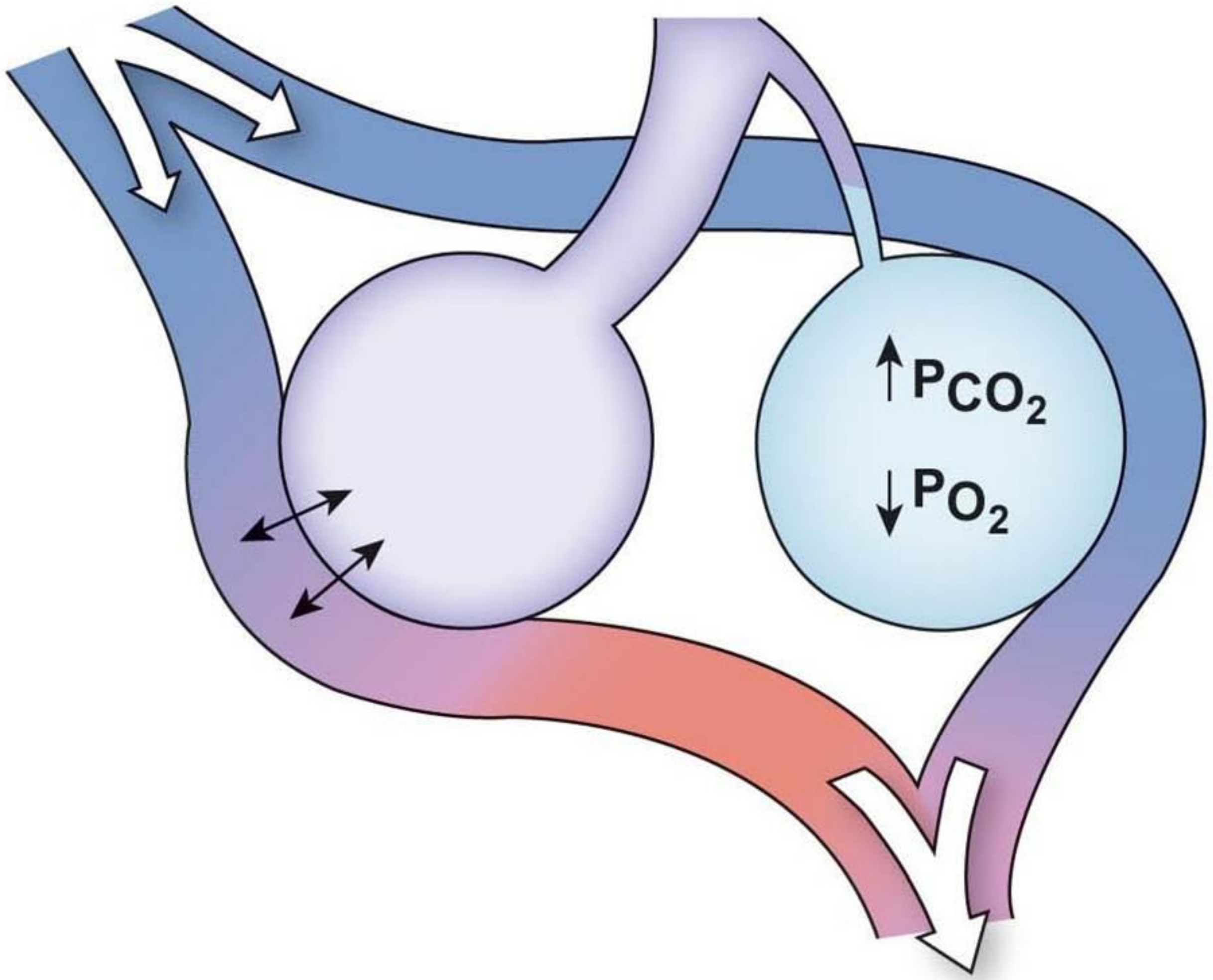
Ensuring that the ventilation and perfusion of the lungs are **adequately matched** is vital for ensuring continuous **delivery** of oxygen and **removal** of carbon dioxide from the body.



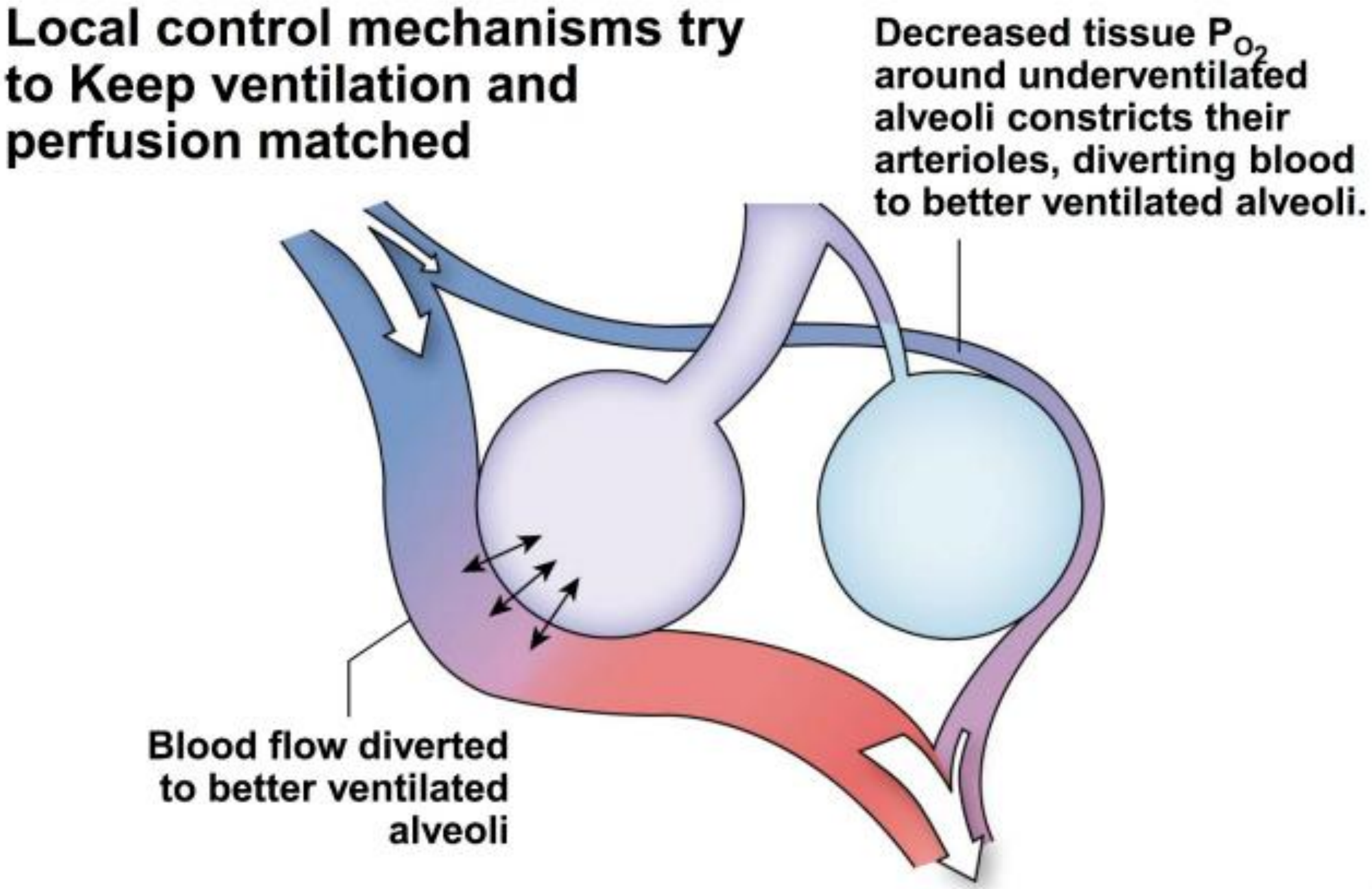



(a) Normally perfusion of blood past alveoli is matched to alveolar ventilation to maximize gas exchange.
Arteriole
Bronchiole
Low oxygen blood
Alveoli
Alveoli

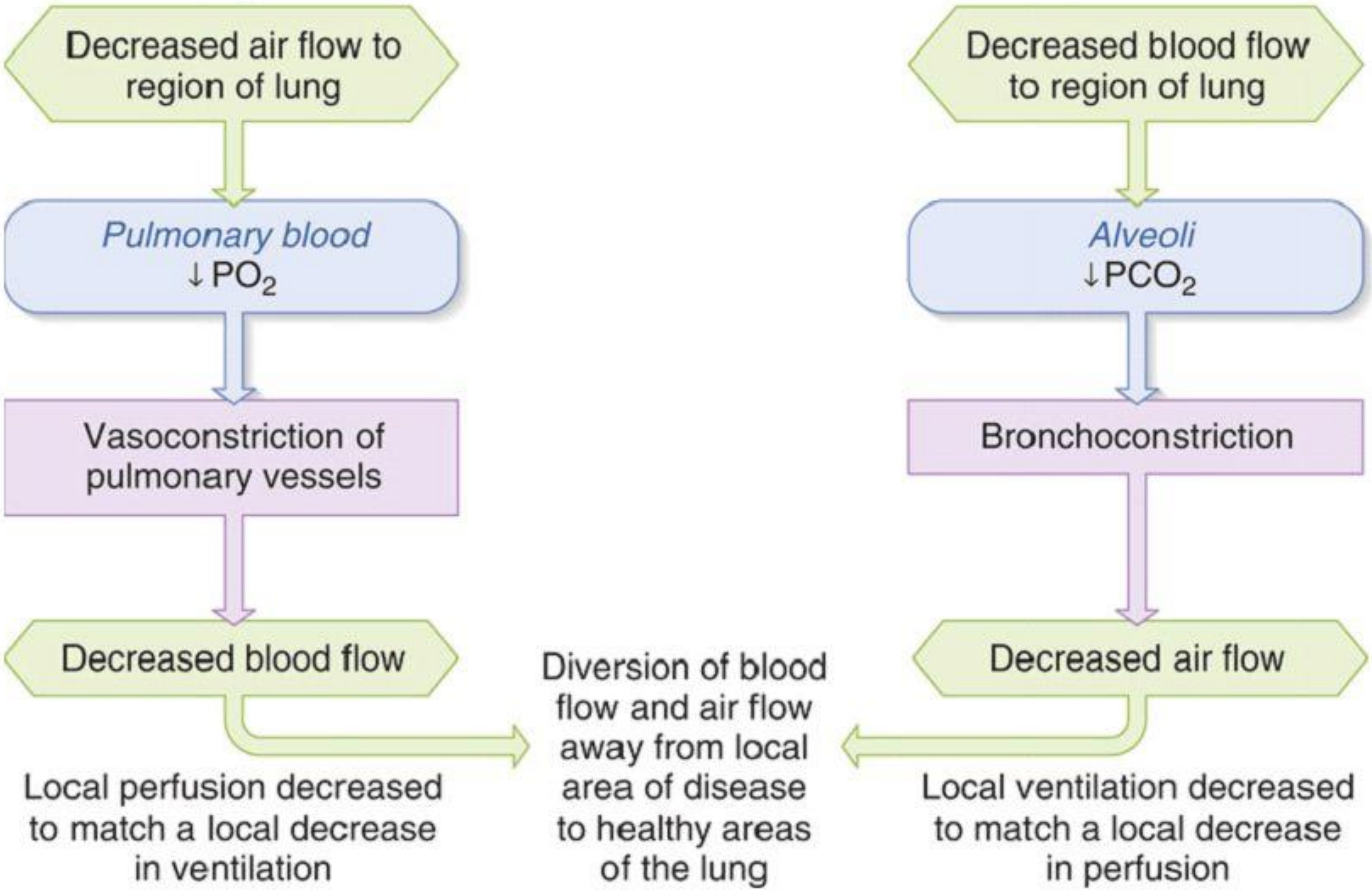
(b) Ventilation-perfusion mismatch.



Hypoxic Vasoconstriction



Local control of ventilation-perfusion matching



Gas	Bronchiole	Pulm. arteriole	Systemic arteriole
↑CO_2	dilate	(constrict)	dilate
↓CO_2	constrict	(dilate)	constrict
↑O_2	(constrict)	dilate	constrict
↓O_2	(dilate)	constrict	dilate

TABLE 17.4 **Local Controls of the Radius of Bronchioles and Pulmonary Arterioles**

Change in gas composition in lungs	Response of bronchioles	Response of pulmonary arterioles
Increased P_{CO_2}	Dilation (increased $\dot{V}_A$*)	Weak constriction (decreased $\dot{Q}$†)
Decreased P_{CO_2}	Constriction (decreased $\dot{V}_A$)	Weak dilation (increased $\dot{Q}$)
Increased P_{O_2}	Weak constriction (decreased $\dot{V}_A$)	Dilation (increased $\dot{Q}$)
Decreased P_{O_2}	Weak dilation (increased $\dot{V}_A$)	Constriction (decreased $\dot{Q}$)

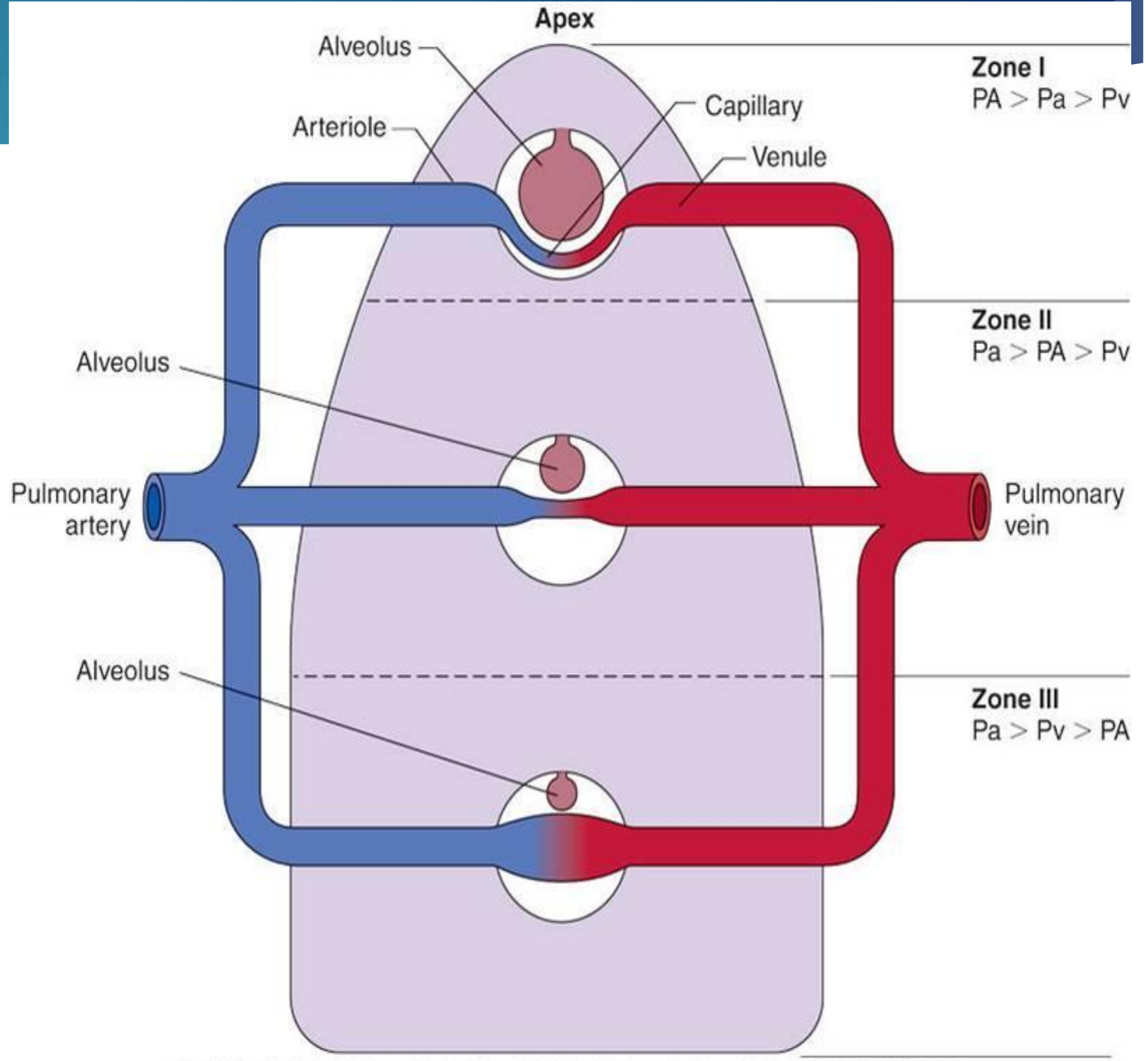
*$\dot{V}_A$ = ventilation
†$\dot{Q}$ = perfusion

Abnormalities of Ventilation-Perfusion Ratio

Abnormalities of Ventilation-Perfusion Ratio

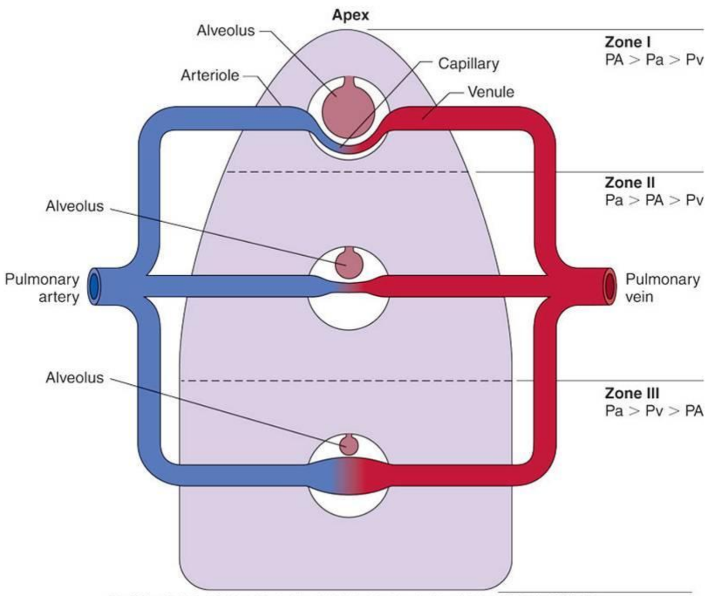
Abnormal V/Q in the Upper and Lower Normal Lung

- In a normal person in the upright position, both pulmonary capillary blood flow and alveolar ventilation are considerably less in the upper part of the lung than in the lower part; however, blood flow is decreased considerably more than ventilation is.



(From McCance KL, Huether SE, editors: *Pathophysiology: the biologic basis for disease in adults and children*, ed 4, St Louis, 2002, Mosby.)

- Therefore, at the top of the lung, V/Q is as much as 2.5 times as great as the ideal value, which causes a moderate degree of *physiologic dead space* in this area of the lung.
- At the other extreme, in the bottom of the lung, there is slightly too little ventilation in relation to blood flow, with V/Q as low as 0.6 times the ideal value. In this area, a small fraction of the blood fails to become normally oxygenated, and this represents a *physiologic shunt*.



(From McCance KL, Huether SE, editors: *Pathophysiology: the biologic basis for disease in adults and children*, ed 4, St Louis, 2002, Mosby.)

Lower V/Q Ratio

- A lower V/Q ratio (with respect to the expected value for a particular lung area in a defined position) impairs pulmonary gas exchange and is a cause of low arterial partial pressure of oxygen (pO_2).

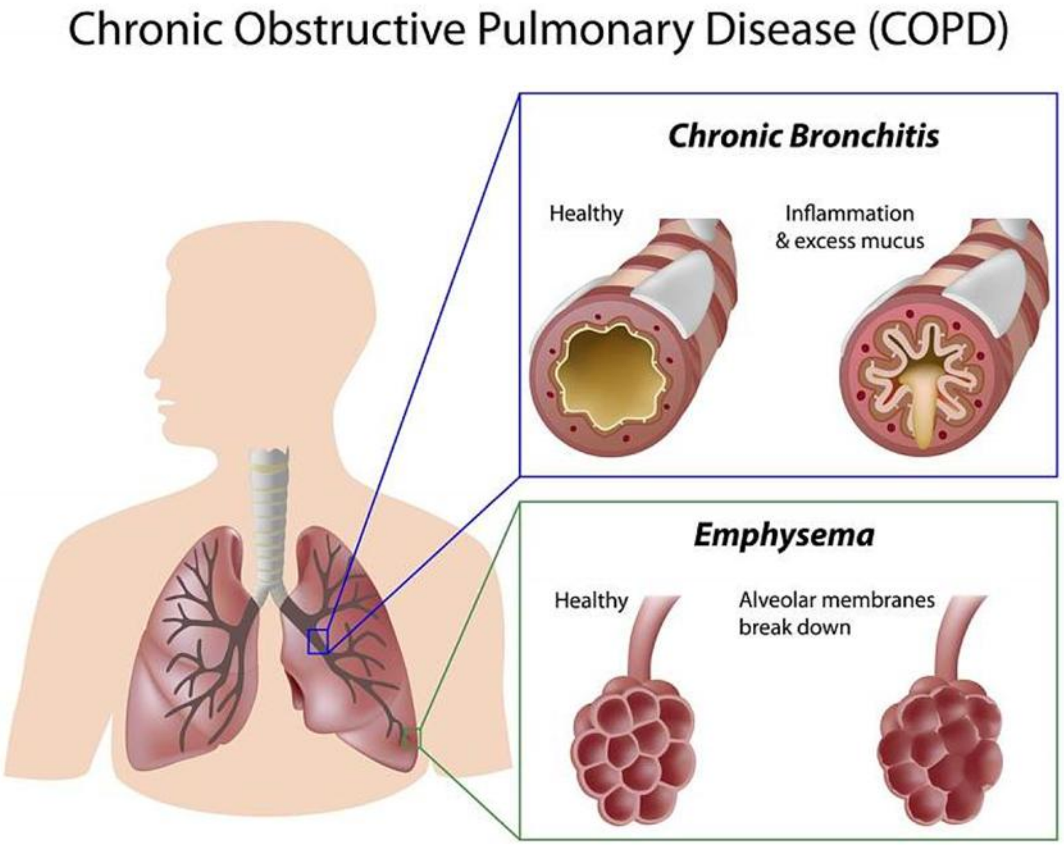
- These abnormal phenomena are usually seen in:
- chronic bronchitis
- asthma
- hepatopulmonary syndrome
- acute pulmonary edema

A high V/Q Ratio

- A high V/Q ratio decreases pCO_2 and increases pO_2 in alveoli.
- Because of the increased dead space ventilation, the pO_2 is reduced and thus also the peripheral oxygen saturation is lower than normal, leading to tachypnea and dyspnea.
- This finding is typically associated with pulmonary embolism (where blood circulation is impaired by an embolus). Ventilation is wasted, as it fails to oxygenate any blood.
- A high V/Q can also be observed in emphysema as a maladaptive ventilatory overwork of the undamaged lung parenchyma.
- Because of the loss of alveolar surface area, there is proportionally more ventilation per available perfusion area.

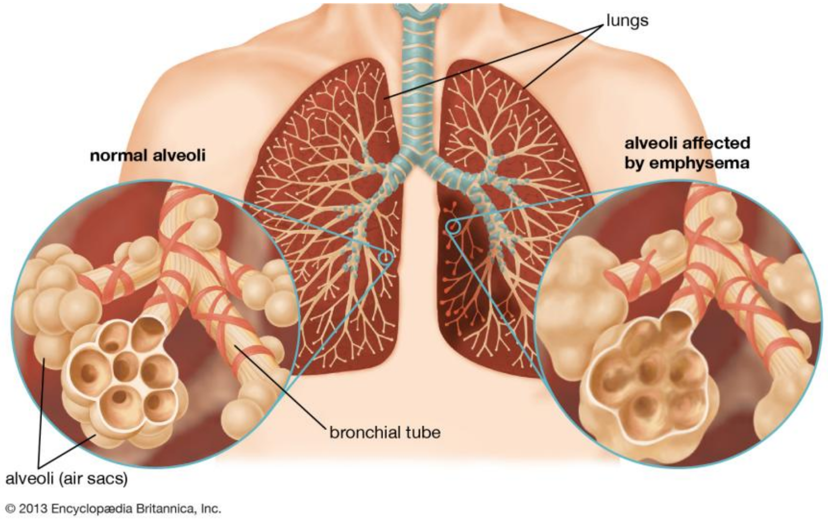
Abnormal V/Q in Chronic Obstructive Lung Disease

- Most people who smoke for many years develop various degrees of bronchial obstruction;
- this condition eventually becomes so severe that they develop serious alveolar air trapping and resultant emphysema.
- The emphysema in turn causes many of the alveolar walls to be destroyed.



Two abnormalities occur in smokers to cause abnormal V/Q

1. because many of the small bronchioles are obstructed, the alveoli beyond the obstructions are unventilated, causing a V/Q that approaches zero.
2. in those areas of the lung where the alveolar walls have been mainly destroyed but there is still alveolar ventilation, most of the ventilation is wasted because of inadequate blood flow to transport the blood gases.



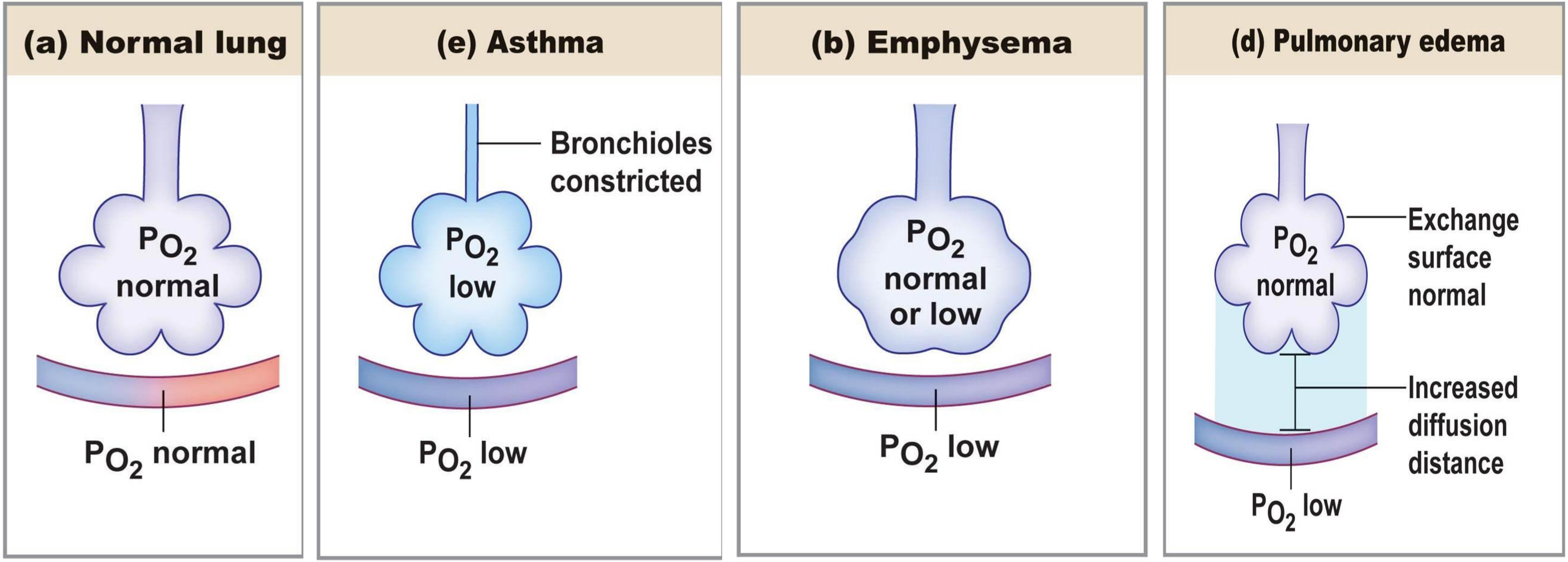
Abnormal V/Q in Chronic Obstructive Lung Disease

In chronic obstructive lung disease:

- some areas of the lung exhibit *serious physiologic shunt*,
- and other areas exhibit *serious physiologic dead space*.
- Both conditions tremendously decrease the effectiveness of the lungs as gas exchange organs, sometimes reducing their effectiveness to as little as one-tenth normal.
- In fact, this is the most prevalent cause of pulmonary disability today.

Abnormalities of V/Q ratio

- Abnormal V/Q in **COPD**
  - various degrees of bronchial obstruction occurs
  - Emphysema occurs resulting in 2 scenarios:
    - Small bronchioles obstructed – alveoli unventilated – V/Q = 0
    - Other areas – wall destruction causes loss of blood vessels – ventilation wasted – V/Q = infinity


(a) Normal lung
P_{O_2} normal
P_{O_2} normal
(e) Asthma
Bronchioles constricted
P_{O_2} low
P_{O_2} low
(b) Emphysema
P_{O_2} normal or low
P_{O_2} low
(d) Pulmonary edema
Exchange surface normal
P_{O_2} normal
Increased diffusion distance
P_{O_2} low

Hypoxemia

Hypoxemia

Hypoxemia is an abnormally low level of oxygen in the blood

Classifications	PaO_2 (rule of thumb)
Normal	80-100 mm Hg
Mild hypoxemia	60-80 mm Hg
Moderate hypoxemia	40-60 mm Hg
Severe hypoxemia	<40 mm Hg

Causes of Hypoxemia	Examples
VQ mismatch	Pneumonia ARDS Pulmonary embolism Cardiogenic pulmonary edema
Shunt physiology	Severe ARDS Hepatopulmonary syndrome Arteriovenous malformation Intracardiac right-to-left shunt
Low available inspired oxygen	High altitude Scuba-diving mishap Combustion within a closed space
Hypoventilation	Opiate overdose COPD Neuromuscular disease Chest wall rigidity Upper airway obstruction
Diffusion defect	Interstitial lung disease
Low mixed venous oxygen	Severe shock

Recommended books

- Text book of Medical Physiology-Guyton and Hall
- Principles of Human Physiology- Lauralee Sherwood
- Sembulingam- Essentials of Medical Physiology
- https://www.youtube.com/watch?v=-mL_NQ3pKnA

For Feedback

If you have any questions or confusion regarding this topic plz contact me at:

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That's all Folks!
SUPERB wallpapers

List the normal airway, alveolar, arterial, and mixed venous PO2 and PCO2 values. 93 List the normal arterial and mixed venous values for O2 saturation, [HCO3-] 94