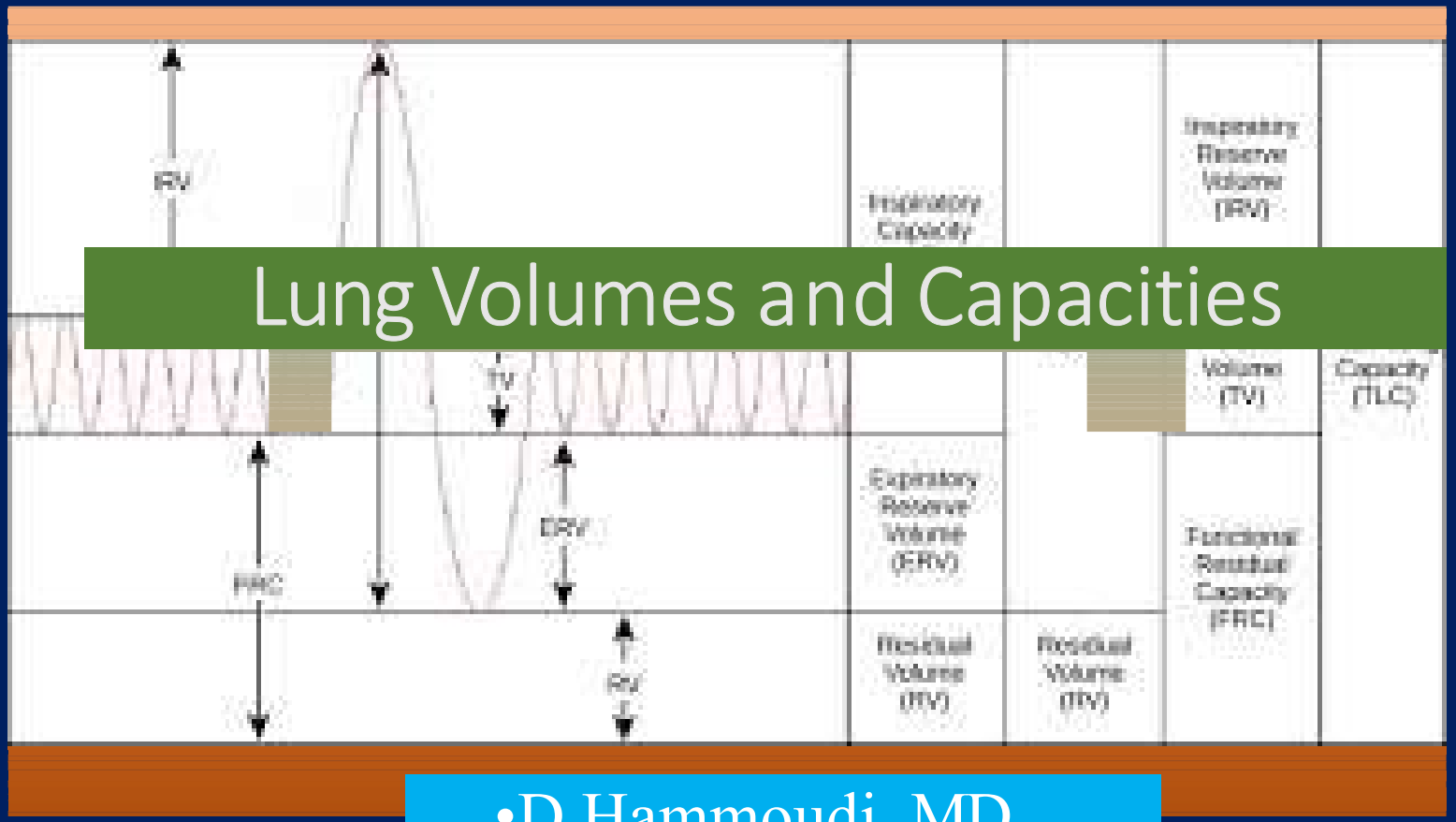
Lung Volumes and Capacities



•D.Hammoudi. MD

Respiratory System Functions

- **Gas exchange:** Oxygen enters blood and carbon dioxide leaves
- **Regulation of blood pH**: Altered by changing blood carbon dioxide levels
- **Voice production:** Movement of air past vocal folds makes sound and speech
- **Olfaction**: Smell occurs when airborne molecules drawn into nasal cavity
- **Protection** Against microorganisms by preventing entry and removing them

Major Functions of the Respiratory System

- To supply the body with oxygen and dispose of carbon dioxide
- Respiration – four distinct processes must happen
  - **Pulmonary ventilation** – moving air into and out of the lungs
  - **External respiration** – gas exchange between the lungs and the blood
  - **Transport** – transport of oxygen and carbon dioxide between the lungs and tissues
  - **Internal respiration** – gas exchange between systemic blood vessels and tissues

Function of the Nose

- **The only externally visible part of the respiratory system that functions by:**
  - Providing an airway for respiration
  - Moistening and warming the entering air
  - Filtering inspired air and cleaning it of foreign matter
  - Serving as a resonating chamber for speech
  - Housing the olfactory receptors

Age-old story: Age-related respiratory changes

Structural changes

- Nose enlargement (from continued cartilage growth)
- General atrophy of the tonsils
- Tracheal deviations (from changes in the aging spine)
- Increased anteroposterior chest diameter (resulting from altered calcium metabolism)
- Calcification of costal cartilages (resulting in reduced mobility of the chest wall)
- Kyphosis (due to osteoporosis and vertebral collapse)
- Increased lung rigidity
- Decreased number and dilation of alveoli
- Reduction in respiratory fluids by 30% (heightening the risk of pulmonary infection and mucus plugs)
- Reduction in respiratory muscle strength

Pulmonary function changes

- Diminished ventilatory capacity
- Decline in diffusing capacity
- Diminished vital capacity (due to decreased inspiratory and expiratory muscle strength)
- Decreased elastic recoil capability (resulting in an elevated residual volume)
- Decreased ventilation of basal areas (due to closing of some airways)

Patterns of Breathing

- **Eupnea**
  - **normal breathing (12-17 B/min, 500-600 ml/B)**
- **Hyperpnea**
  - **⇑ pulmonary ventilation matching ⇑ metabolic demand**
- **Hyperventilation (⇓ CO2)**
  - **⇑ pulmonary ventilation > metabolic demand**
- **Hypoventilation (⇑ CO2)**
  - **⇓ pulmonary ventilation < metabolic demand**

Patterns of breathing (cont.)

- **Tachypnea**
  - ⇑ frequency of respiratory rate
- Apnea
  - Absense of breathing. e.g. Sleep apnea
- **Dyspnea**
  - Difficult or labored breathing
- Orthopnea
  - Dyspnea when recumbent, relieved when upright. e.g. congestive heart failure, asthma, lung failure

1. Eupnea (normal)

2. Tachypnea

3. Bradypnea

4. Apnea

5. Cheyne-Stokes

6. Biot's

7. Apneustic

8. Agonal

9. Shallow

10. Hyperpnea

11. Air trapping

12. Kussmaul's

13. Sighing

SURFACTANT

- Lines the inner layer of alveolar epithelium.
- Synthesized by SER of type II pneumocytes.
- **Function –**

  1. To reduce the surface tension of alveoli mainly during expiration, thus reduces the work of lung inflation.
  2. Waterproofing.

- Surfactant synthesis starts after 26 weeks of fetal life. Therefore premature infants,with insufficient surfactant suffer from HMD.

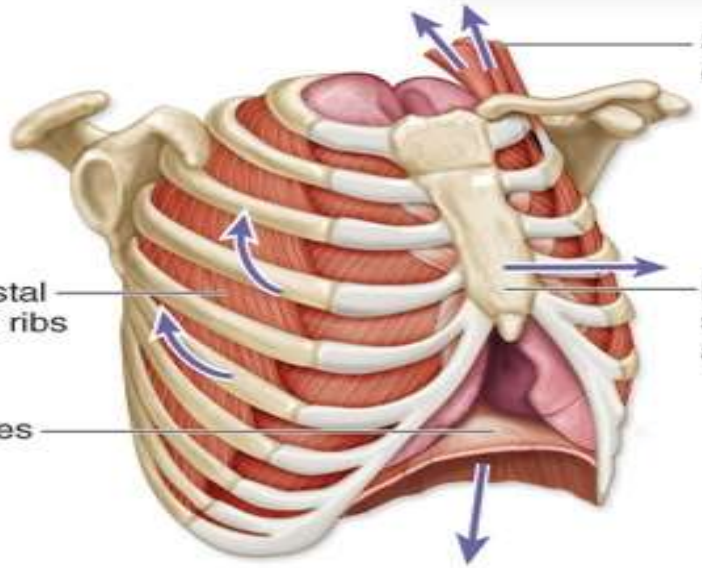
Important lung products

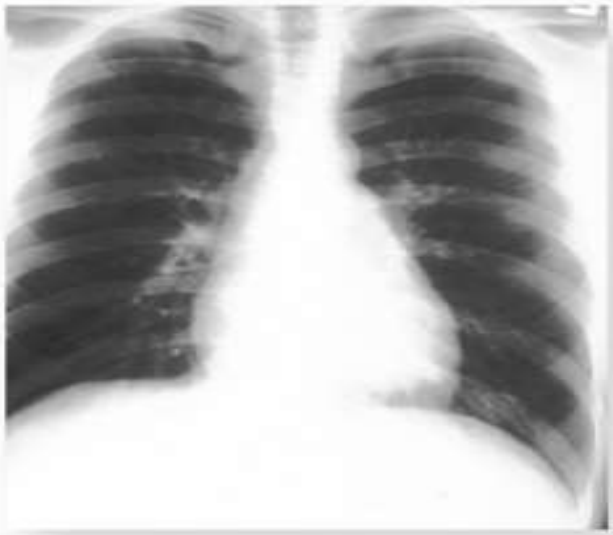
1. Surfactant—produced by type II pneumocytes, ↓ alveolar surface tension, ↑ compliance, ↓ work of inspiration
2. Prostaglandins
3. Histamine ↑ bronchoconstriction
4. Angiotensin-converting enzyme (ACE)—angiotensin I → angiotensin II; inactivates bradykinin (ACE inhibitors ↑ bradykinin and cause cough, angioedema)
5. Kallikrein—activates bradykinin

Surfactant––dipalmitoyl phosphatidylcholine (lecithin) deficient in neonatal RDS.

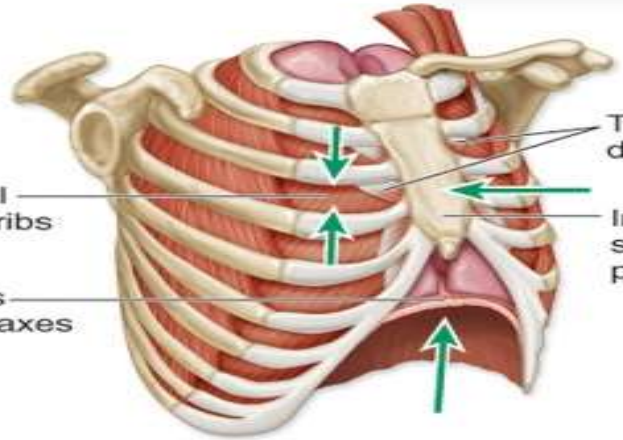
Collapsing pressure = $\frac{2\,(\text{tension})}{\text{radius}}$

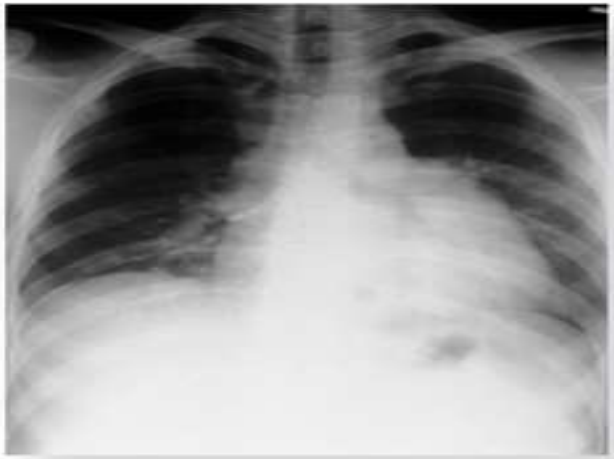
Inhalation

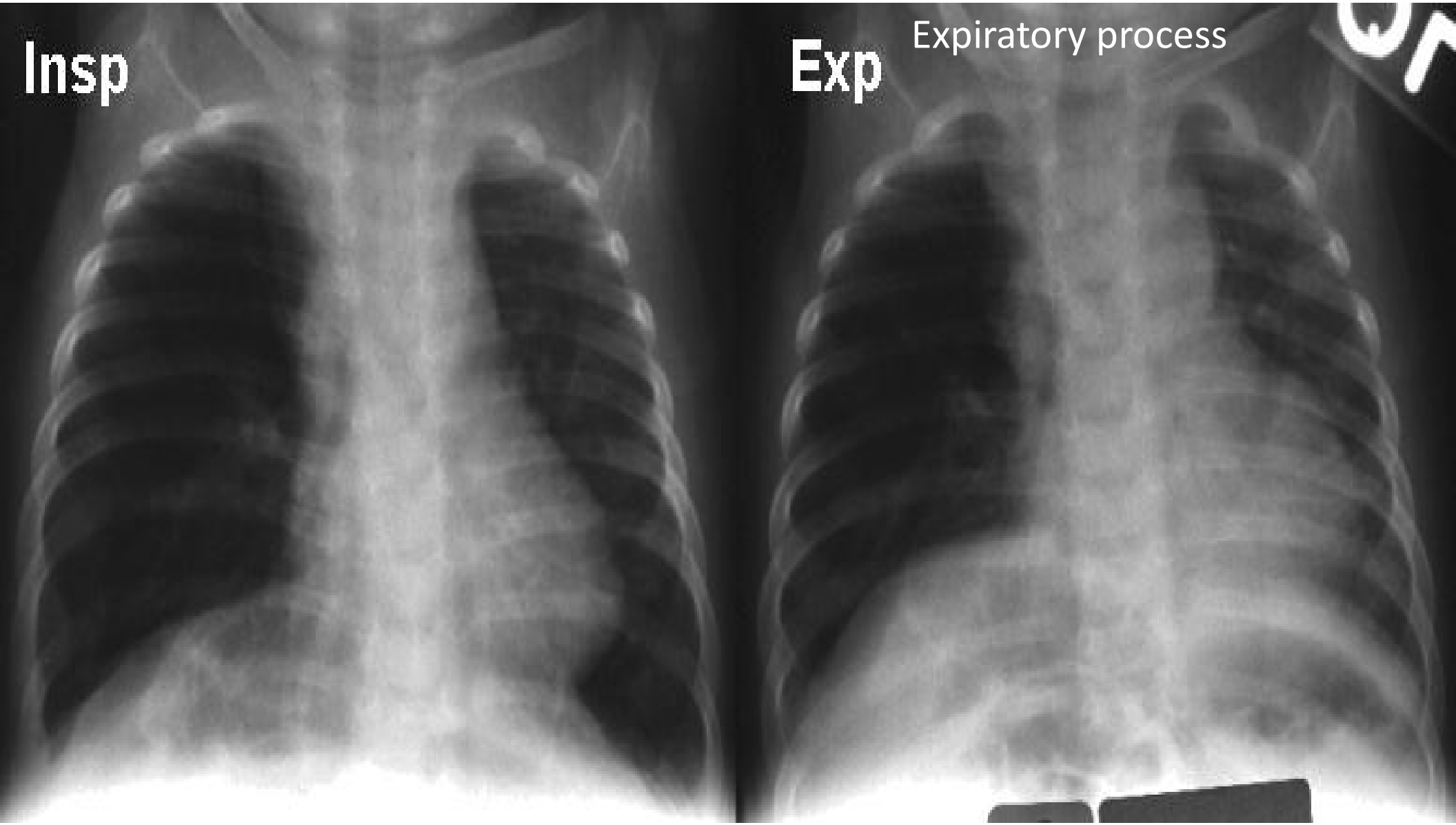




Exhalation






Insp
Exp
Expiratory process

Muscles of inspiration

Accessory

Sternocleidomastoid (elevates sternum)

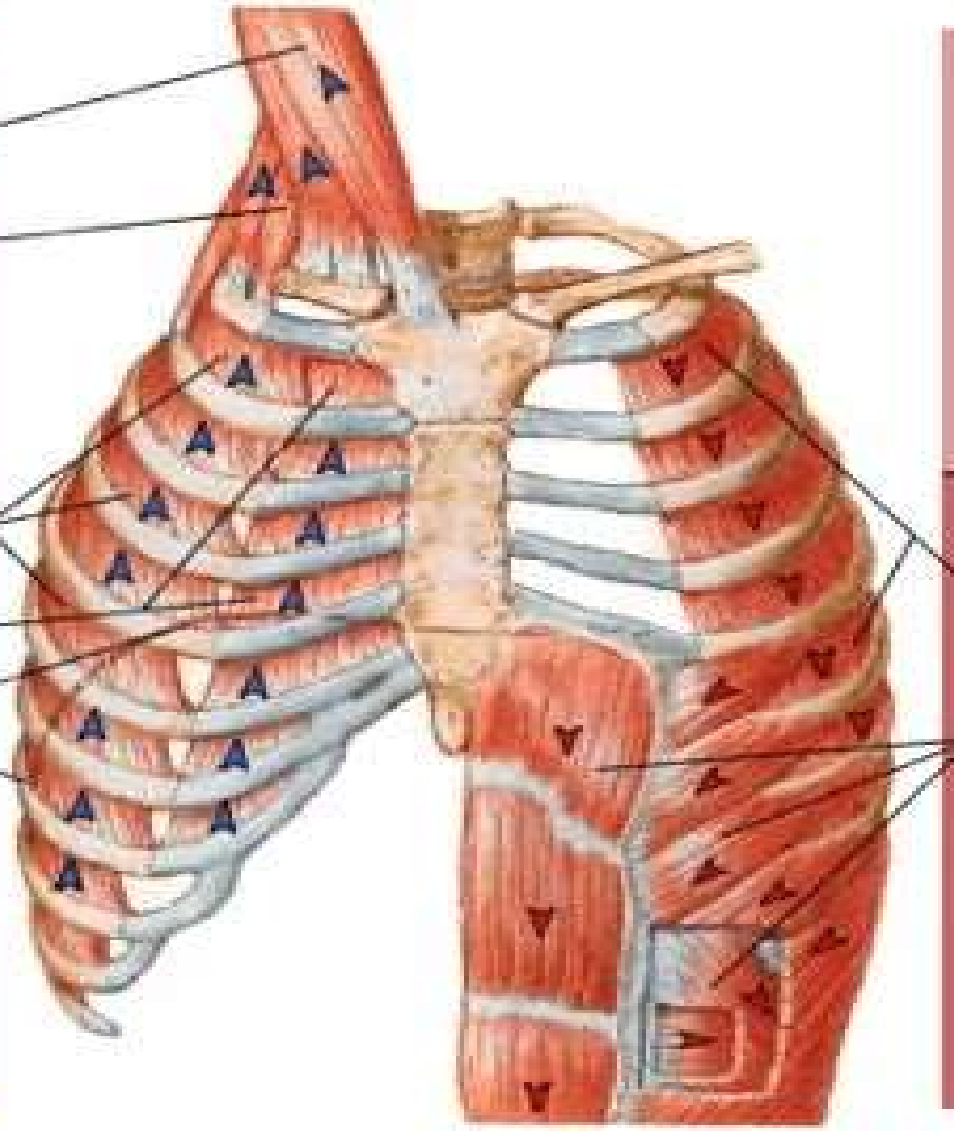
Scalenes Group (elevate upper ribs)

Not shown:
Pectoralis minor

Principal

External intercostals Interchondral part of internal intercostals (also elevates ribs)

Diaphragm (dome descends, thus increasing vertical dimension of thorac cavity; also elevates lower ribs)



Muscles of expiration

Quiet breathing

Expiration results from passive, elastic recoil of the lungs, dib cage and diaphragm

Active breathing

Internal intercostals, except interchondral part (pull ribs down)

Abdominals (pull ribs down, compress abdominal contents thus pushing diaphragm up)

Note shown:
Quadratus lumborum (pulls ribs down)

Intrapulmonary Pressures

- **Air entering the lungs during inspiration because the atmospheric pressure is greater than the intrapulmonary pressure**..
- **Usually during quiet inspiration, intrapulmonary pressure is at 3 mmHg below the pressure of the atmosphere. But it shows as -3mmHg.**
- **Expiration occurs when the intrapulmonary pressure is greater than the atmospheric pressure.**
- During quiet expiration it's shown as +3mmHg above atmospheric pressure.

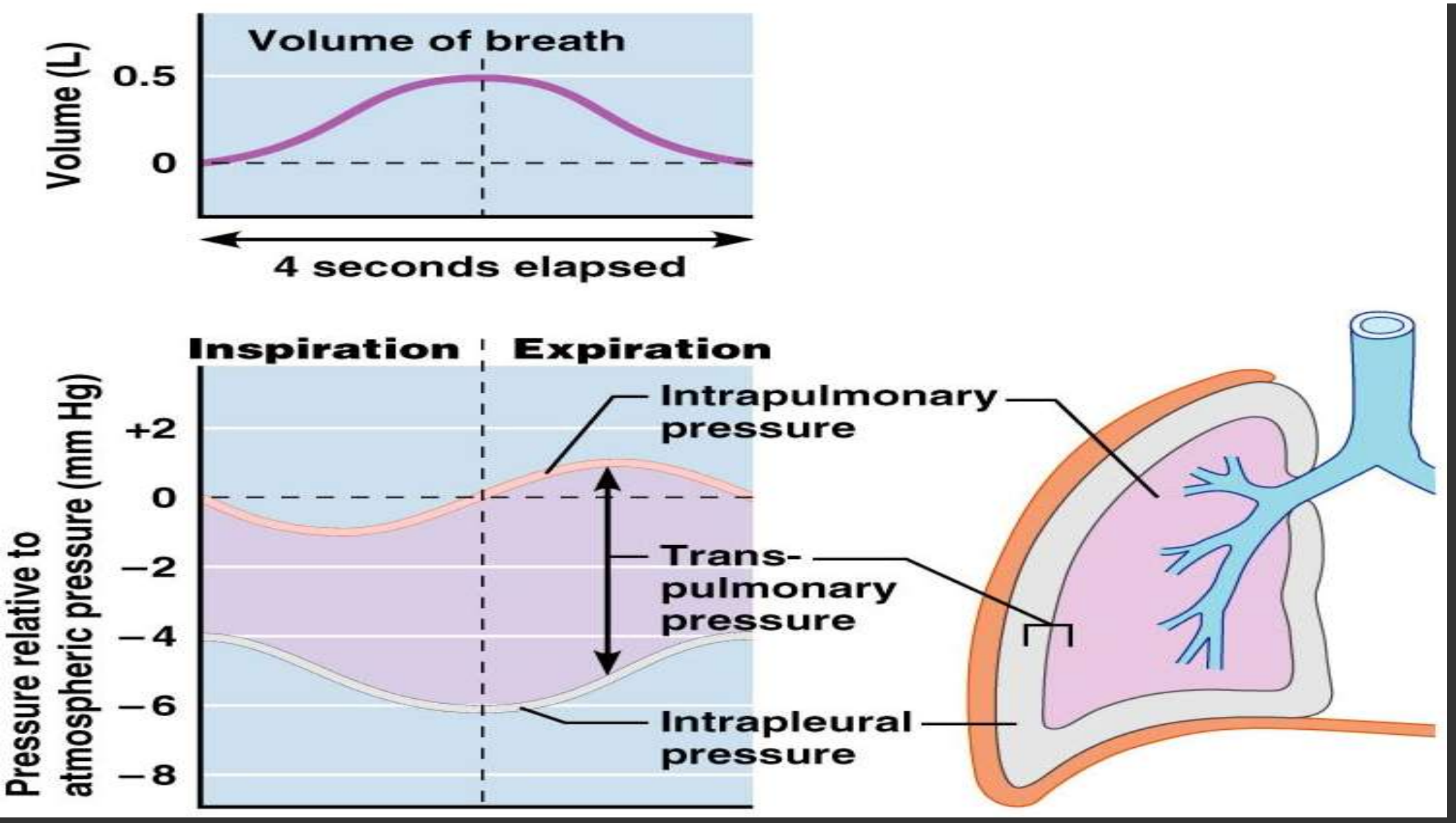
Intrapleural Pressure

The opposing elastic recoil of the lungs and the chest wall produces a subatmospheric pressure in the intrapleural space between the two structures.

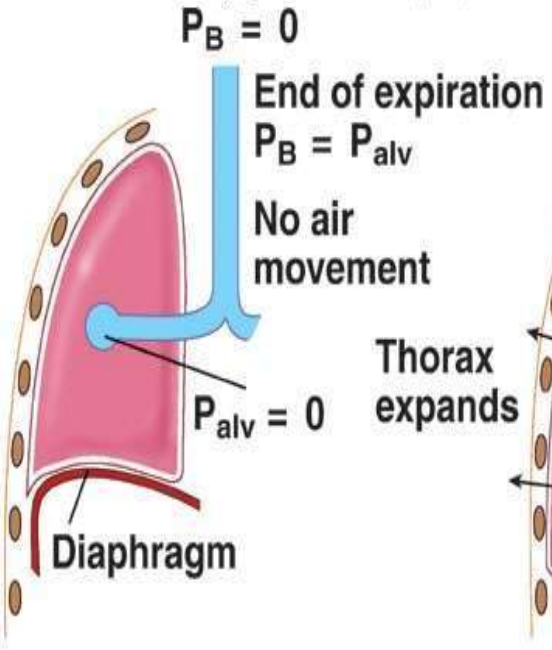
- **This intrapleural pressure is lower during inspiration because of the expansion of the thoracic cavity than it is during expiration.**
- **The intrapleural pressure is normally lower than the intraplumonary pressure during both inspiration and expiraiton.**

Transpulmonary Pressure

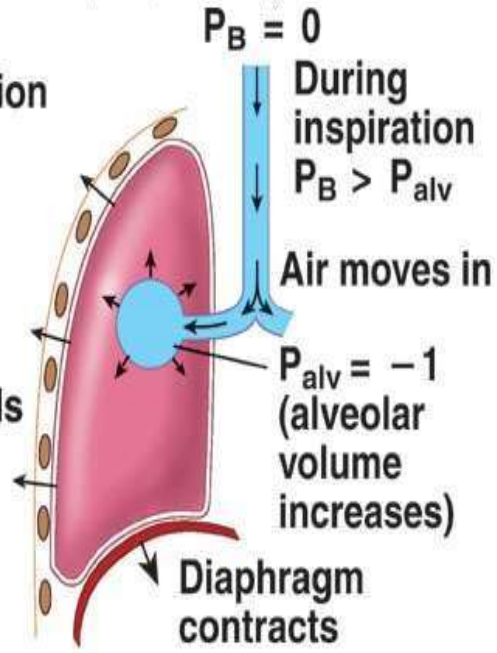
The pressure difference across the wall of the lung is transpulmonary pressure, which can also be the difference between the intrapulmonary pressure and the intrapleural pressure and keeps the lungs against the chest wall.


Volume of breath
Volume (L)
0.5
0
4 seconds elapsed
Inspiration
Expiration
Pressure relative to atmospheric pressure (mm Hg)
+2
0
−2
−4
−6
−8
Intrapulmonary pressure
Trans-pulmonary pressure
Intrapleural pressure

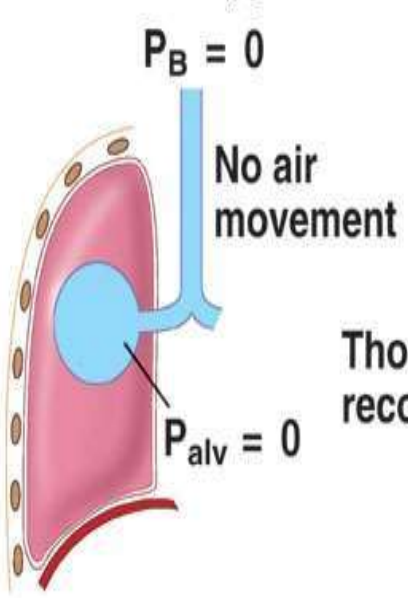
Alveolar Pressure Changes



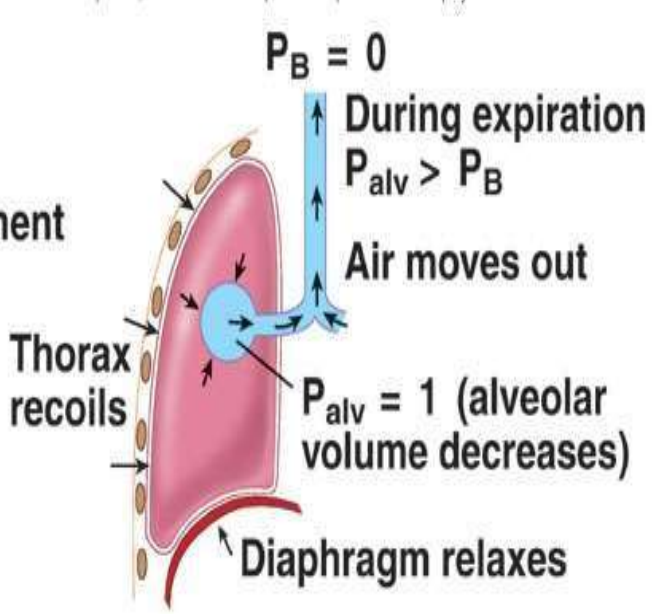
1. Barometric air pressure (P_B) is equal to alveolar pressure (P_{alv}) and there is no air movement.



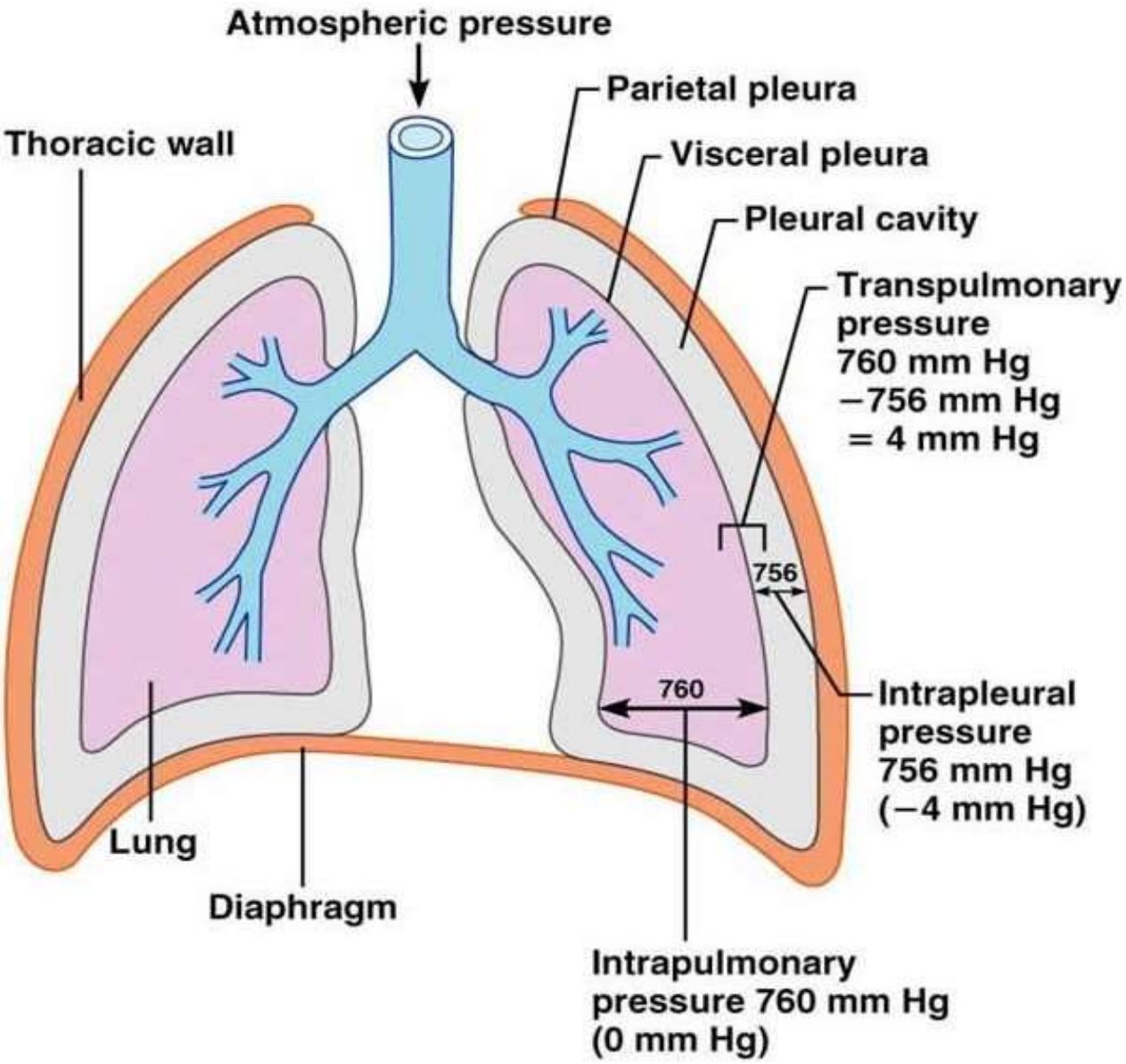
2. Increased thoracic volume results in increased alveolar volume and decreased alveolar pressure. Barometric air pressure is greater than alveolar pressure, and air moves into the lungs.



3. End of inspiration.



4. Decreased thoracic volume results in decreased alveolar volume and increased alveolar pressure. Alveolar pressure is greater than barometric air pressure, and air moves out of the lungs.

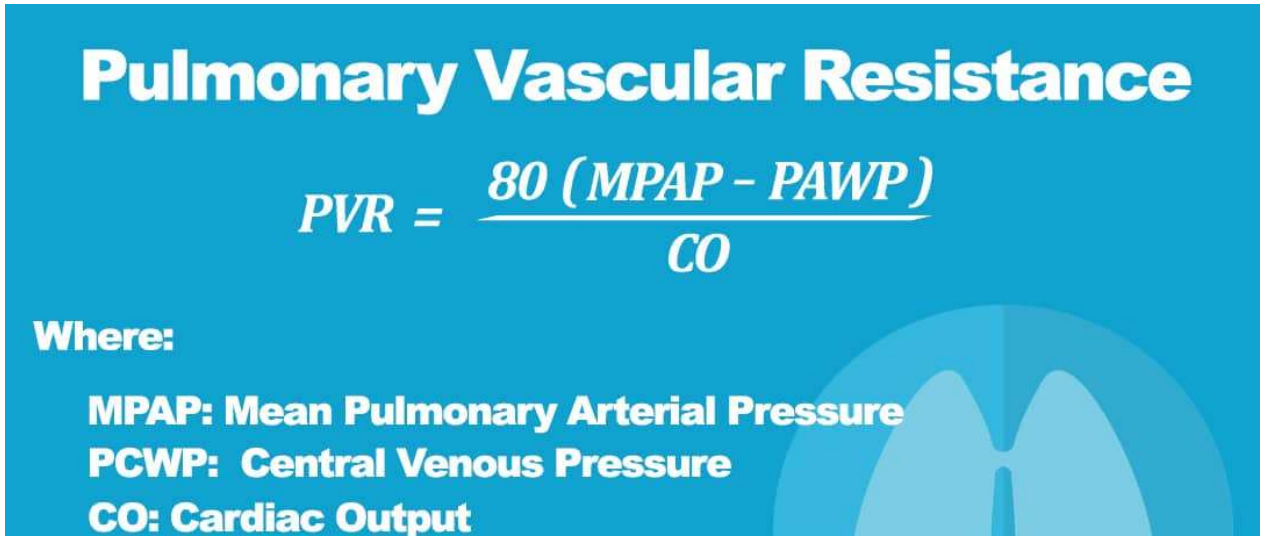


Laplace's law:
Pressure =(4 x surface tension) / radius

The surface tension contributes a large part of the static recoil force of the lung (expiration)

FRC (Functional residual capcity) is the volume in the **lungs** at the end of a natural exhalation.

- At FRC (functional residual capacity) , inward pull of lung is balanced by outward pull of chest wall, and system pressure is atmospheric.
- At FRC, airway and alveolar pressures equal atmospheric pressure (called zero), and intrapleural pressure is negative (prevents atelectasis).
- The inward pull of the lung is balanced by the outward pull of the chest wall.
- System pressure is atmospheric.
- PVR (Pulmonary vascular resistance) is at a minimum.



Compliance

- **Describes distensibility of respiratory system=** change in lung volume for a
- change in pressure
- Describes change in lung volume for a given change in pressure (C = V/P) expressed as ΔV/ΔP and is
- inversely proportional to wall stiffness
- ↑ compliance in emphysema
- ↓ compliance in pulmonary fibrosis, pulmonary edema, ARDS, chest wall disease

- Measure of the ease with which lungs and thorax expand
  - The greater the compliance, the easier it is for a change in pressure to cause expansion
  - A lower-than-normal compliance means the lungs and thorax are harder to expand
    - Conditions that decrease compliance
      - Pulmonary fibrosis
      - Pulmonary edema
      - Respiratory distress syndrome

High compliance = lung easier to fill (emphysema, normal aging),

lower compliance = lung harder to fill (pulmonary fibrosis, pneumonia, NRDS, pulmonary edema).

Surfactant increases compliance

. Compliant lungs comply (cooperate) and fill easily with air

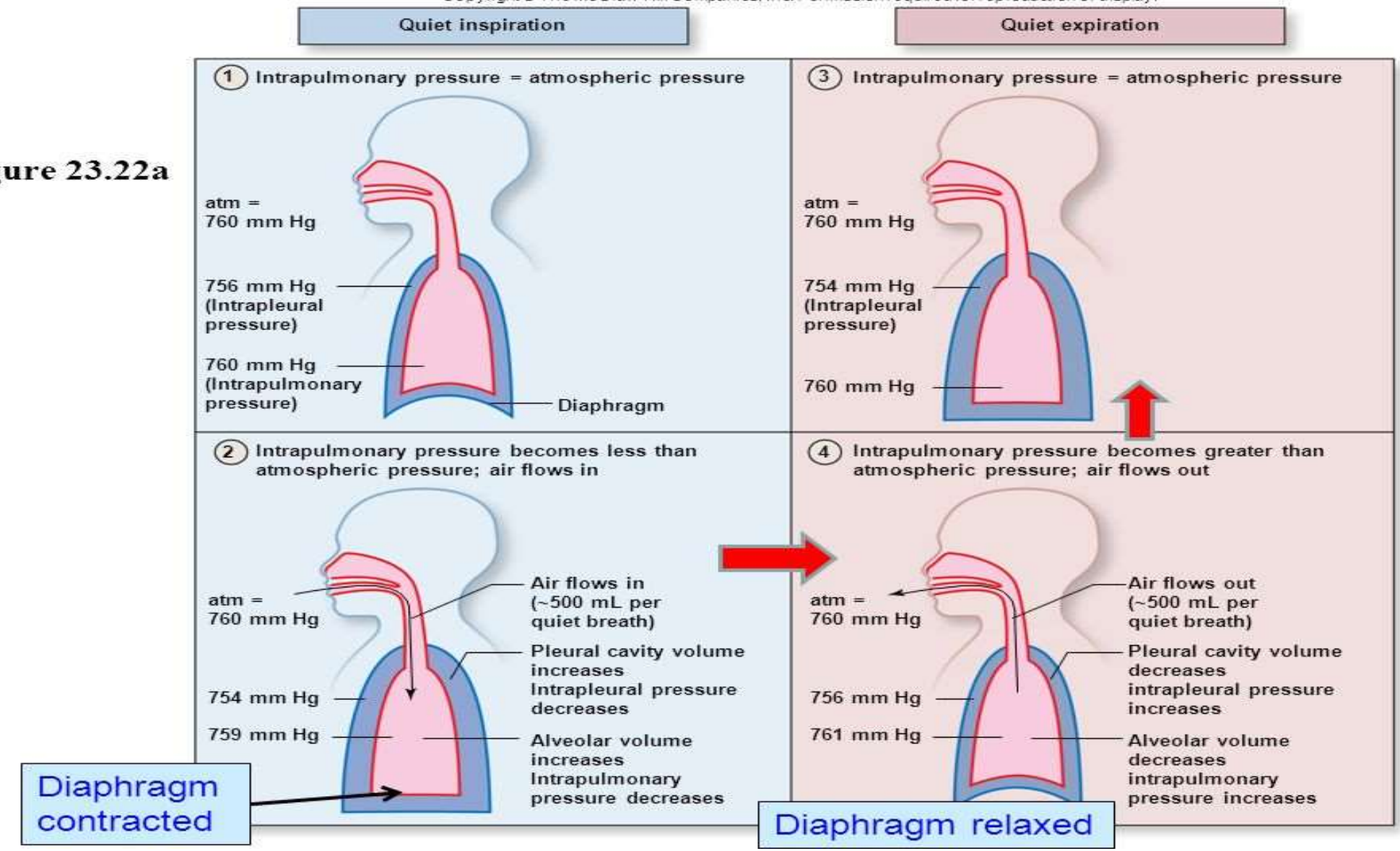
Elastance

- **Describes elastic properties (inverse of compliance, elastance = P/V)**
- Lungs tend to collapse inward
- Chest wall tends to expand outward

- **During inspiration intrapleural pressure becomes more negative**
- **Respiration stops in late expiration because of dynamic compression of airways**
- **Total lung capacity depends on compliance**
- **Nitrogen wash out method detects functional residual capacity**
- **FRC (FUNCTIONAL RESIDUAL CAPACITY) is not estimated by spirometry**
- **Slow and deep breathing are the most economical way of breathing.**

Elastic recoil—tendency for lungs to collapsinward and chest wall to spring outward.

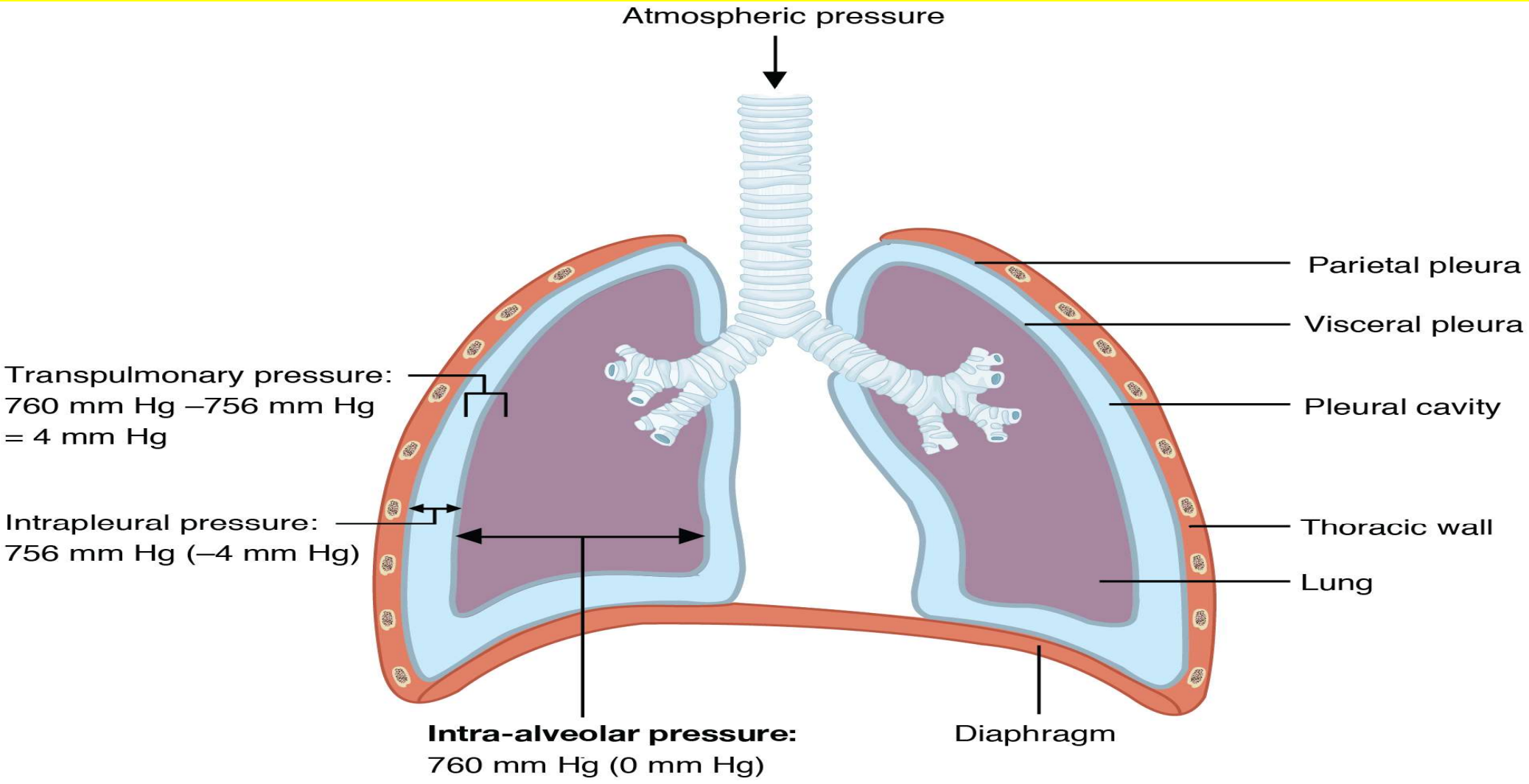
Figure 23.22a



Pressures

- **Atmospheric pressure – 760 mm Hg, 630 mm Hg here**
- **Intrapleural pressure – 756 mm Hg – pressure between pleural layers**
- **Intrapulmonary pressure – varies, pressure inside lungs**

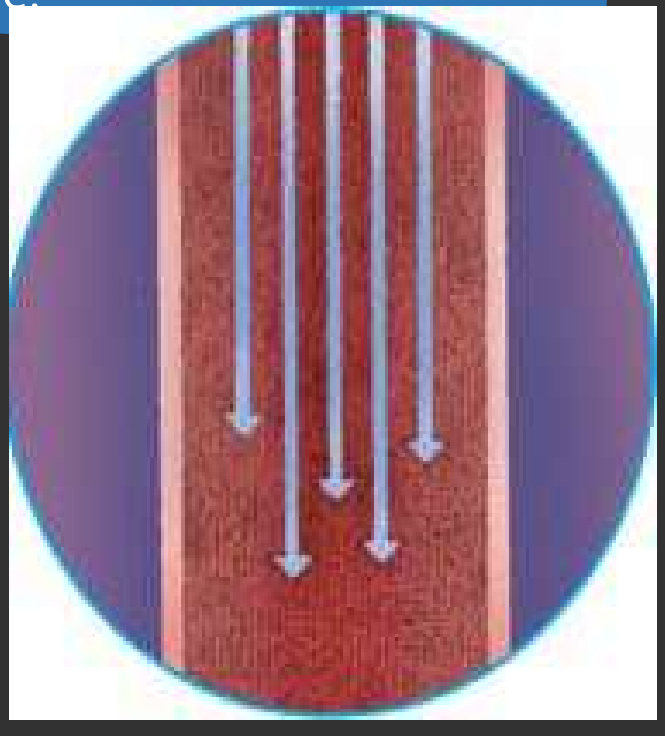
Pressure Relationships



Laminar flow

Laminar flow, a linear pattern that occurs at low flow rates, offers minimal resistance.

This flow type occurs mainly in the small peripheral airways of the bronchial tree.

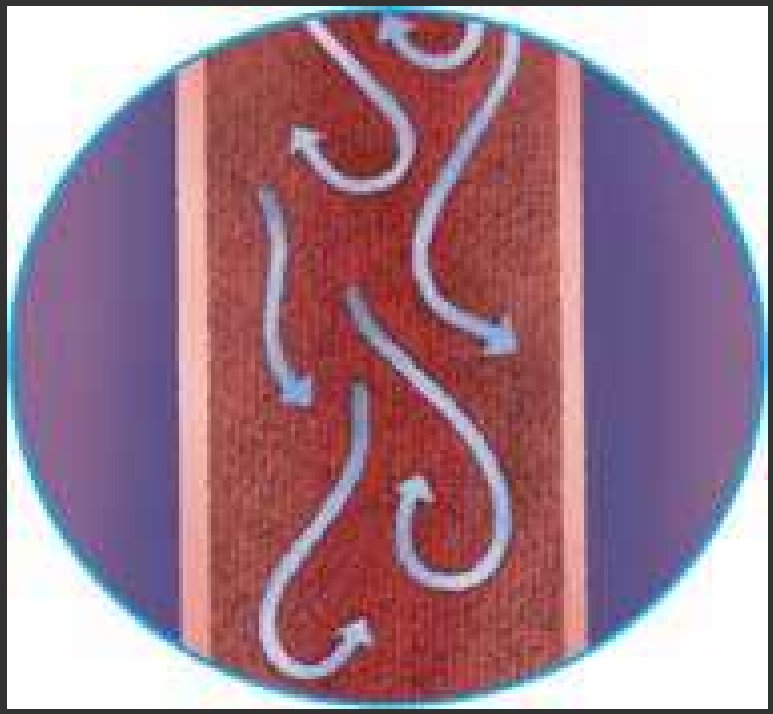


Turbulent flow

The eddying pattern of turbulent flow creates friction and increases resistance.

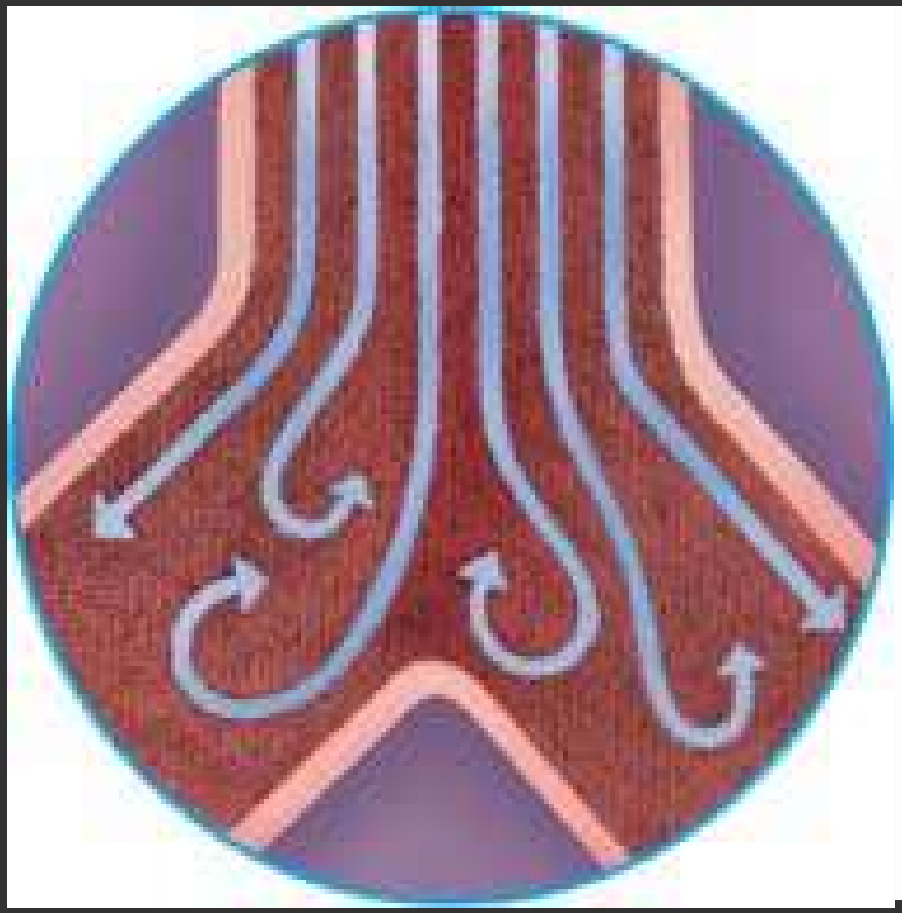
Turbulent flow is normal in the trachea and large central bronchi.

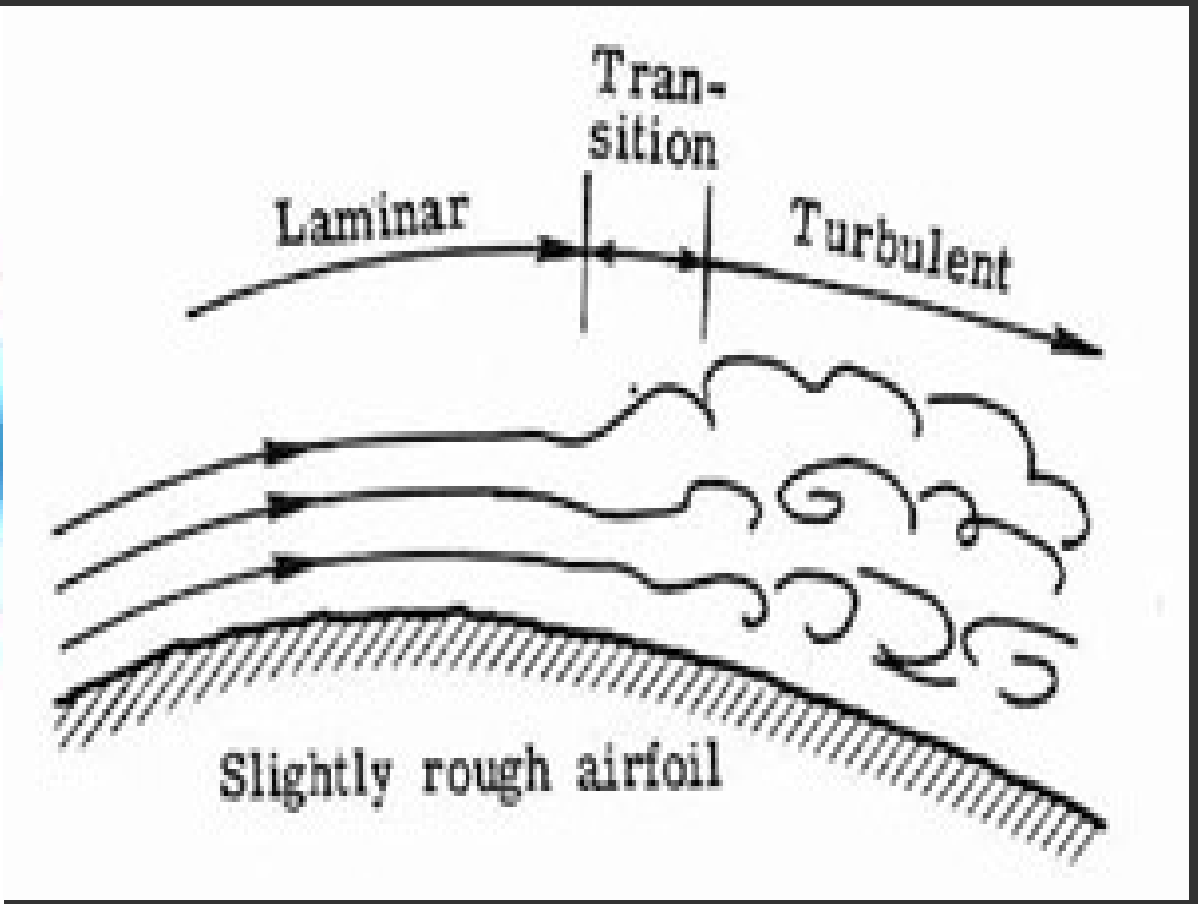
If the smaller airways become constricted or clogged with secretions, however, turbulent flow may also occur there.



Transitional flow

A mixed pattern known as transitional flow is common at lower flow rates in the larger airways, especially where the airways narrow from obstruction, meet, or branch.





PART 2

Respiration division

Respiration is divided into 4 processes:

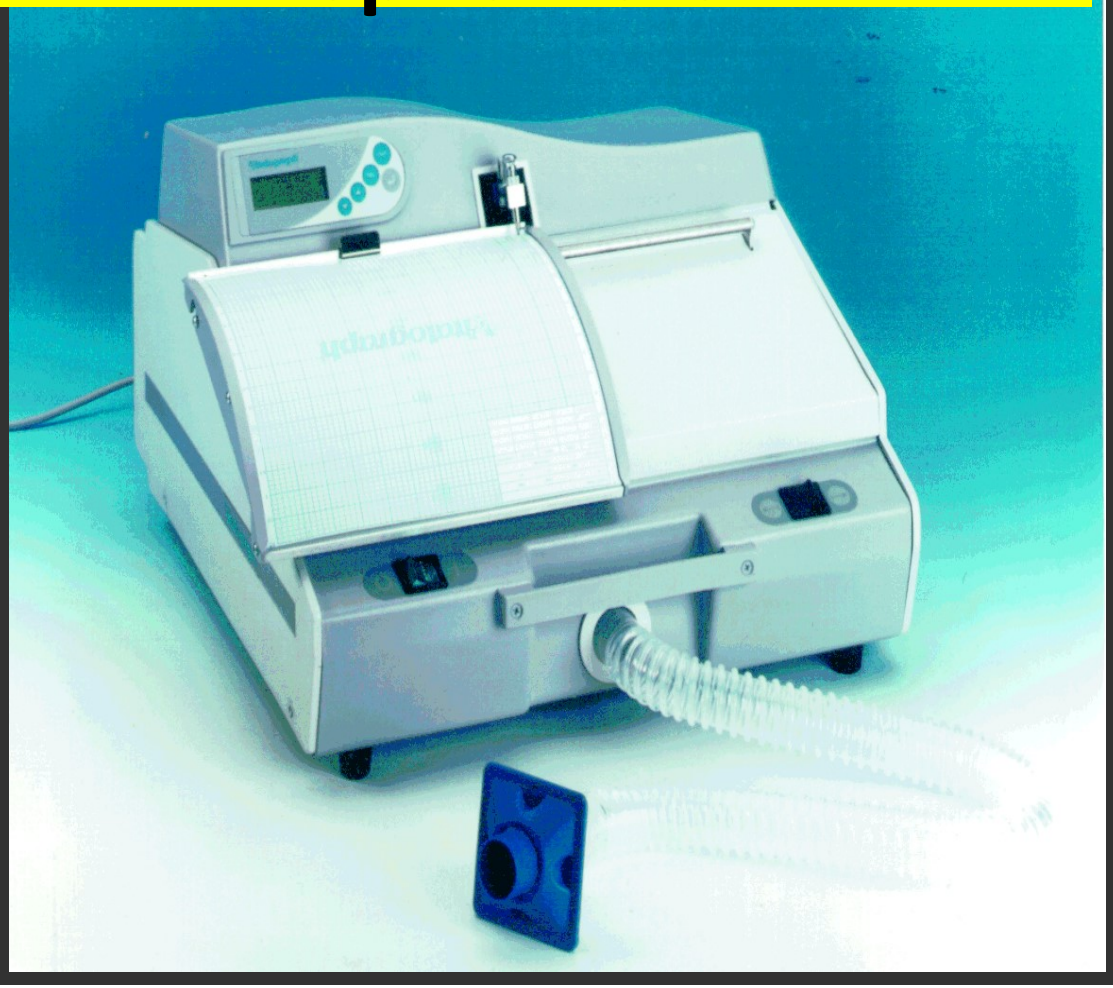
1. **Pulmonary ventilation is the movement of air into/out of the lungs**

2. **External respiration is the movement of O2 from the lungs to the blood and CO2 from the blood to the lungs.**

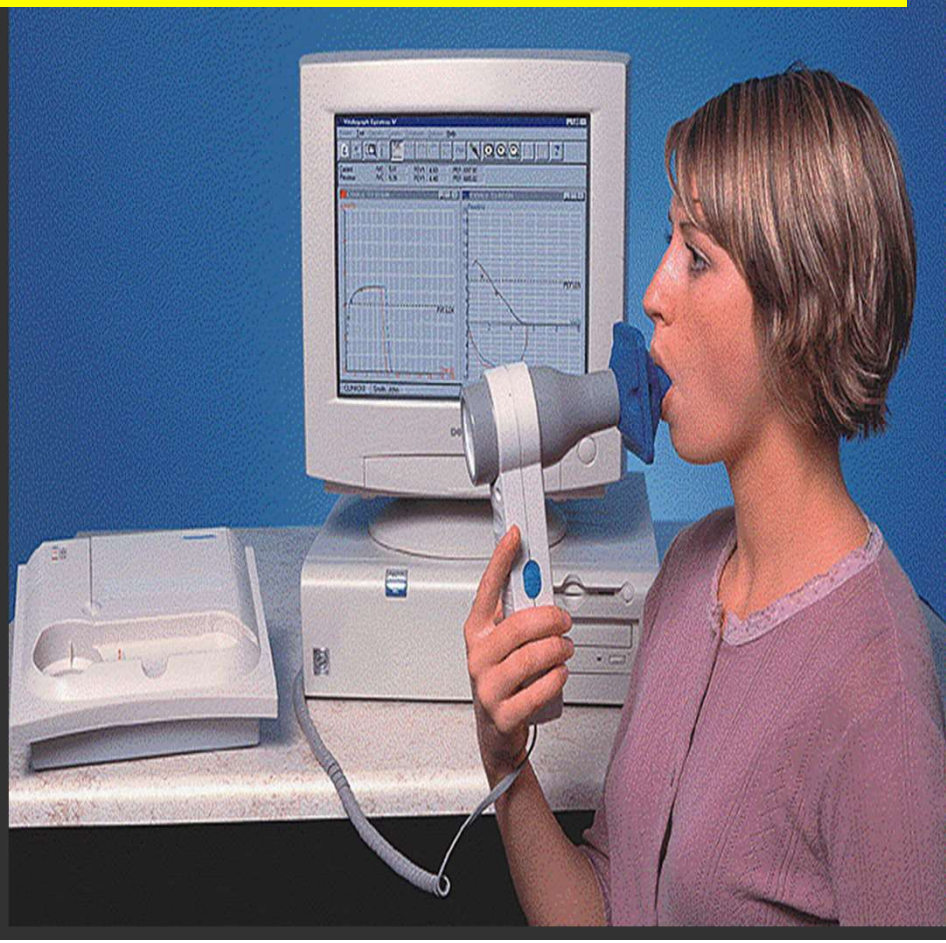
3. **Internal respiration is the movement of O2 from the blood to the cell interior and CO2 from the cell interior to the blood.**

4. **Cellular respiration is the breakdown of glucose, fatty acids and amino acids that occurs in mitochondria and results in production of ATP.**
   It requires O2 and produces CO2. (Note that this type of cellular respiration, which requires O2, is known as "aerobic metabolism," whereas breakdown of glucose that produces ATP but does not require O2 is "anaerobic metabolism.")

Volume Measuring Spirometer



Flow Measuring Spirometer



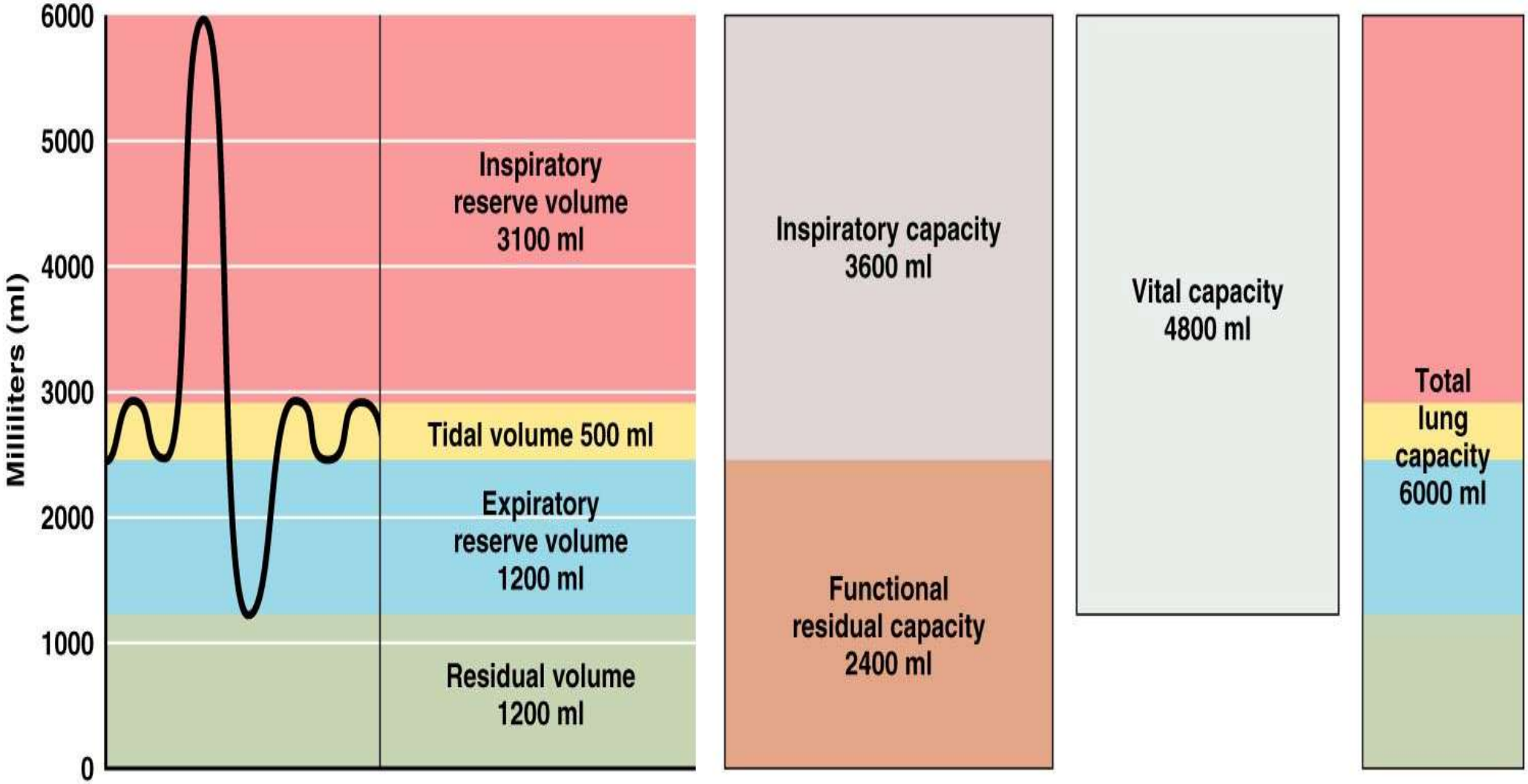
Desktop Electronic Spirometers

Small Hand-held Spirometers

Respiratory volumes

	Measurement	Adult male average value	Adult female average value	Description
Respiratory volumes	Tidal volume (TV)	500 ml	500 ml	Amount of air inhaled or exhaled with each breath under resting conditions
	Inspiratory reserve volume (IRV)	3100 ml	1900 ml	Amount of air that can be forcefully inhaled after a normal tidal volume inhalation
	Expiratory reserve volume (ERV)	1200 ml	700 ml	Amount of air that can be forcefully exhaled after a normal tidal volume exhalation
	Residual volume (RV)	1200 ml	1100 ml	Amount of air remaining in the lungs after a forced exhalation
Respiratory capacities	Total lung capacity (TLC)	6000 ml	4200 ml	Maximum amount of air contained in lungs after a maximum inspiratory effort: TLC = TV + IRV + ERV + RV
	Vital capacity (VC)	4800 ml	3100 ml	Maximum amount of air that can be expired after a maximum inspiratory effort: VC = TV + IRV + ERV (should be 80% TLC)
	Inspiratory capacity (IC)	3600 ml	2400 ml	Maximum amount of air that can be inspired after a normal expiration: IC = TV + IRV
	Functional residual capacity (FRC)	2400 ml	1800 ml	Volume of air remaining in the lungs after a normal tidal volume expiration: FRC = ERV + RV

(b) Summary of respiratory volumes and capacities for males and females



(a) Spirographic record for a male

- **The following terms describe the various lung (respiratory) volumes:**
- **The tidal volume (TV),** about 500 ml, is the ***amount of air inspired during normal, relaxed breathing***.
- **The inspiratory reserve volume (IRV),** about 3,100 ml, is the ***additional air that can be forcibly inhaled after the inspiration of a normal tidal volume***.
- **The expiratory reserve volume (ERV),** about 1,200 ml, is ***the additional air that can be forcibly exhaled after the expiration of a normal tidal volume.***
- **Residual volume (RV),** about 1,200 ml, ***is the volume of air still remaining in the lungs after the expiratory reserve volume is exhaled.***

Summing specific lung volumes produces the following lung capacities:

- **The total lung capacity (TLC),** about **6,000 ml**, **is the maximum amount of air that can fill the lungs**
  - (***TLC = TV + IRV + ERV + RV***).
- **The vital capacity (VC),** about **4,800 ml**, is **the total amount or air that can be expired after fully inhaling**
  - ***(VC = TV + IRV + ERV = approximately 80% TLC).***
- **The inspiratory capacity (IC),** about **3,600 ml**, is **the maximum amount of air that can be inspired**
  - ***(IC = TV + IRV).***
- **The functional residual capacity (FRC),** about **2,400 ml**, **is the amount of air remaining in the lungs after a normal expiration**
  - (FRC = RV + ERV).
- Some of the air in the lungs does not participate in gas exchange. Such air is located in the anatomical dead space within bronchi and bronchioles—that is, outside the alveoli.

Alveolar Ventilation

- **Alveolar ventilation rate (AVR)**

  Alveolar ventilation = Volume of gas that reaches alveoli each minute

- Slow, deep breathing increases AVR and rapid, shallow breathing decreases AVR

AVR	=	frequency	X	(TV – dead space)
(ml/min)		(breaths/min)		(ml/breath)

- Total volume of gas entering lungs per minute= VE = VT× RR = **Minute ventilation**

Normal values:
Respiratory rate (RR) = 12–20 breaths/min
VT= 500 mL/breath = TIDAL VOLUME
VD= 150 mL/breath

Dead Space

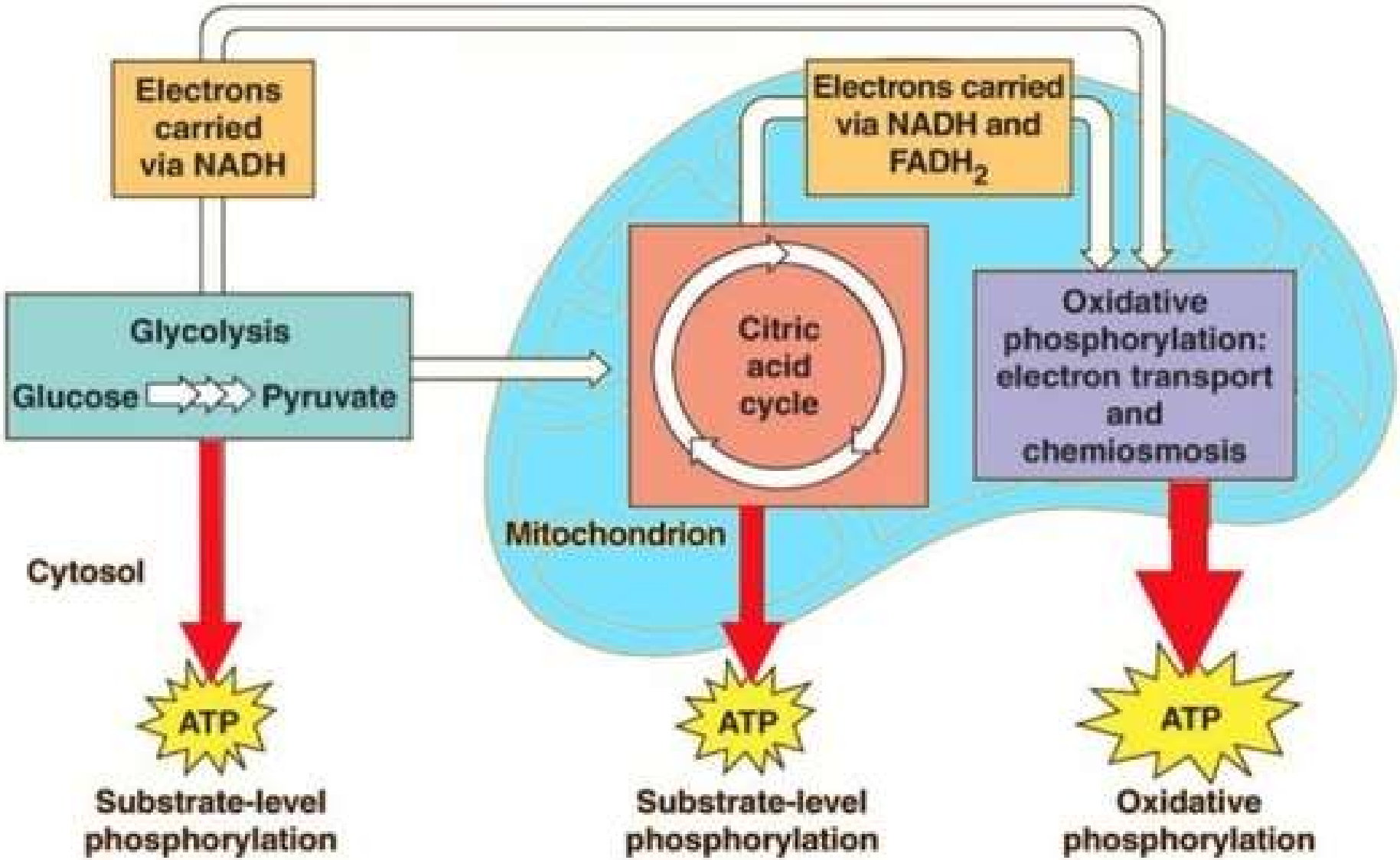
- **Anatomical dead space** – volume of the conducting respiratory passages (150 ml)
- **Alveolar dead space** – alveoli that cease to act in gas exchange due to collapse or obstruction
- **Total dead space** – sum of alveolar and anatomical dead spaces

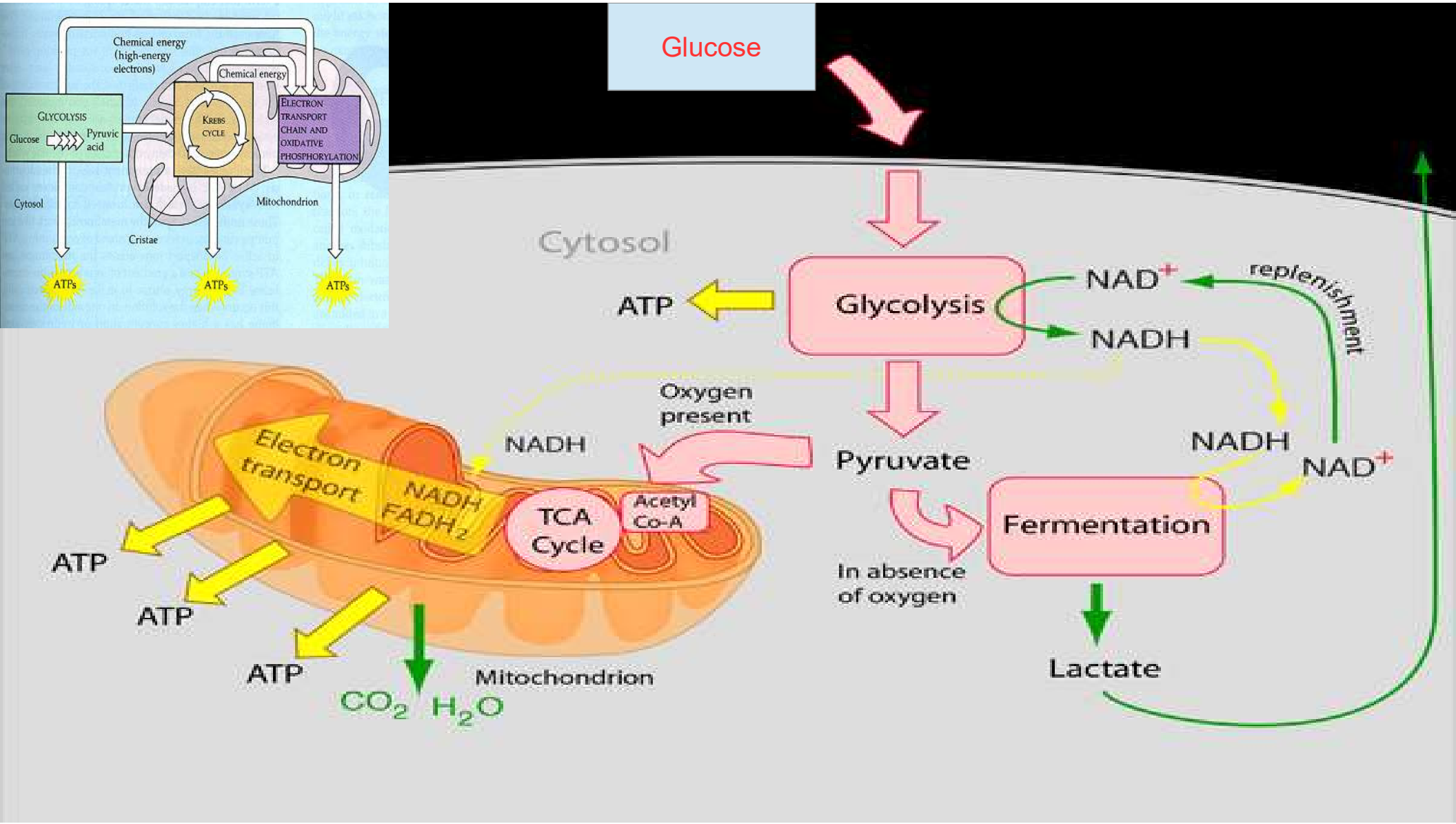
Determination of physiologic dead space

$$VD = VT \times \frac{Paco2 - Peco2}{Paco2}$$

VD= physiologic dead space = anatomic dead space of conducting airways plus alveolar dead space; apex of healthy lung is largest contributor of alveolar dead space.
Volume of inspired air that does not take part in gas exchange.
VT= tidal volume.
Paco 2 = arterial Pco2
.**Peco2**= expired air Pco2
.

Physiologic dead space—approximately equivalent to anatomic dead space in normal lungs.
May be greater than anatomic dead space in lung diseases with V/Q DEFECT(VENTILATION PERFUSION)

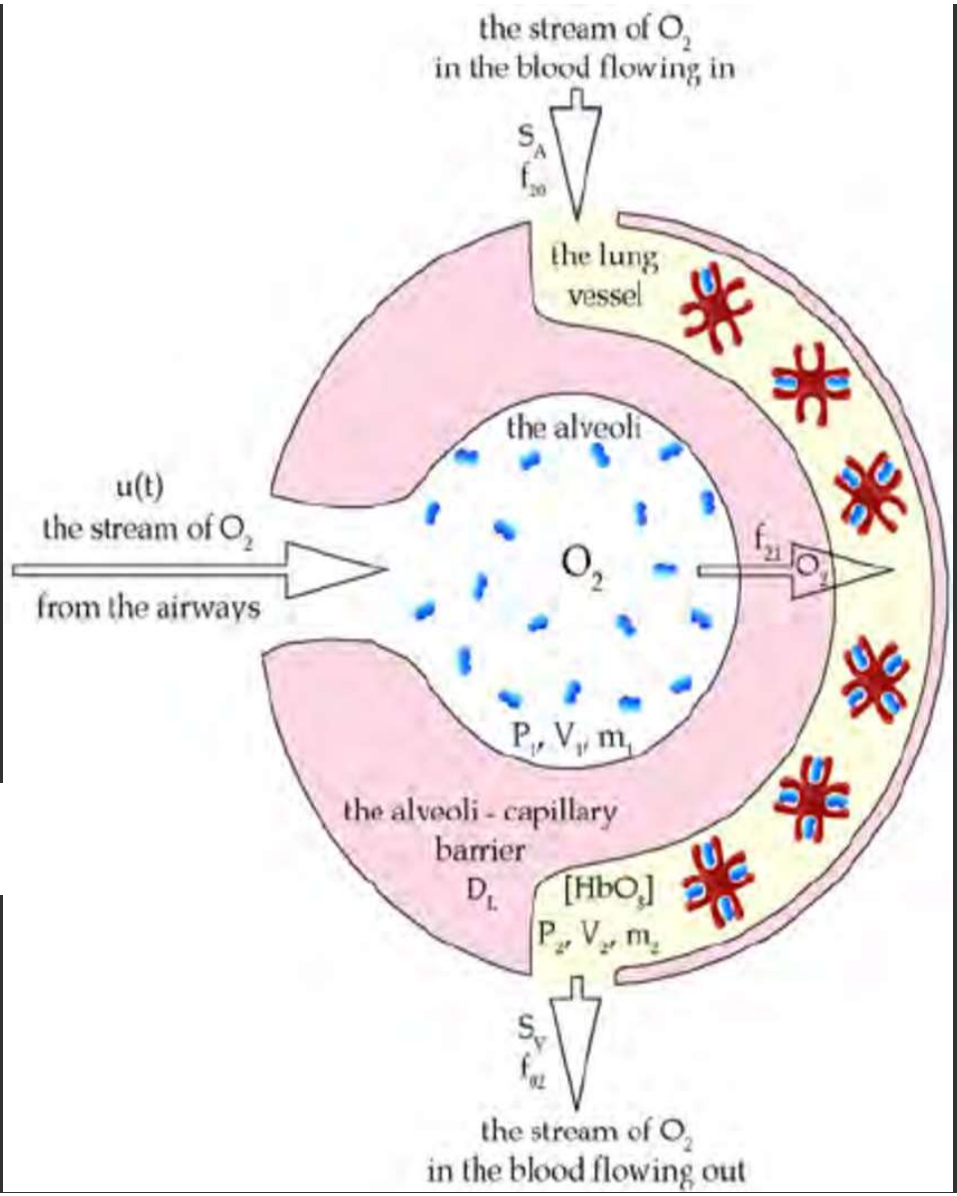

Electrons carried via NADH
Electrons carried via NADH and $FADH_2$
Glycolysis
Glucose
Pyruvate
Citric acid cycle
Oxidative phosphorylation: electron transport and chemiosmosis
Mitochondrion
Cytosol
ATP
ATP
ATP
Substrate-level phosphorylation
Substrate-level phosphorylation
Oxidative phosphorylation


Chemical energy (high-energy electrons)
Chemical energy
GLYCOLYSIS
Glucose
Pyruvic acid
KREBS CYCLE
ELECTRON TRANSPORT CHAIN AND OXIDATIVE PHOSPHORYLATION
Cytosol
Mitochondrion
Cristae
ATPs
ATPs
ATPs
Glucose
Cytosol
ATP
Glycolysis
NAD+
NADH
replenishment
Oxygen present
NADH
Pyruvate
NADH
NAD+
Electron transport
NADH
FADH2
TCA Cycle
Acetyl Co-A
Fermentation
ATP
ATP
ATP
In absence of oxygen
Lactate
Mitochondrion
CO2
H2O

4 rules for diffusion of gas

- Surface area
- Thickness
- Concentration
- Distance

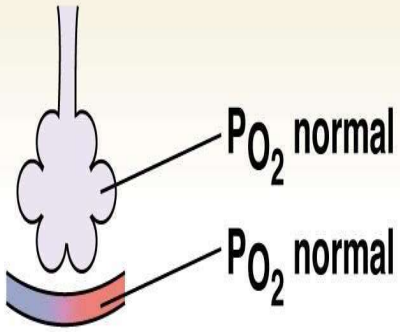
Laplace's law:
Pressure =(4 x surface tension) / radius



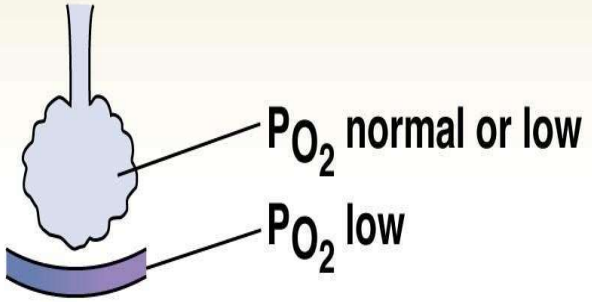
$$\text{collapsing pressure (P)} = \frac{2\ (\text{surface tension})}{\text{radius}}$$

- **Alveoli have increased tendency to collapse on expiration as radius decreased (law of Laplace).**
- **Pulmonary surfactant is a complex mix of lecithins, the most important of which is dipalmitoylphosphatidylcholine (DPPC).**
- **Surfactant synthesis begins around week 26 of gestation, but mature levels are not achieved until around week 35.**

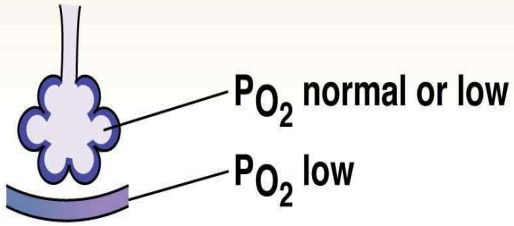
(a) Normal lung



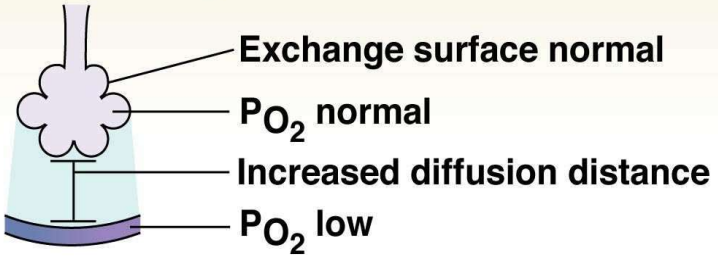
(b) **Emphysema:** destruction of alveoli reduces surface area for gas exchange.



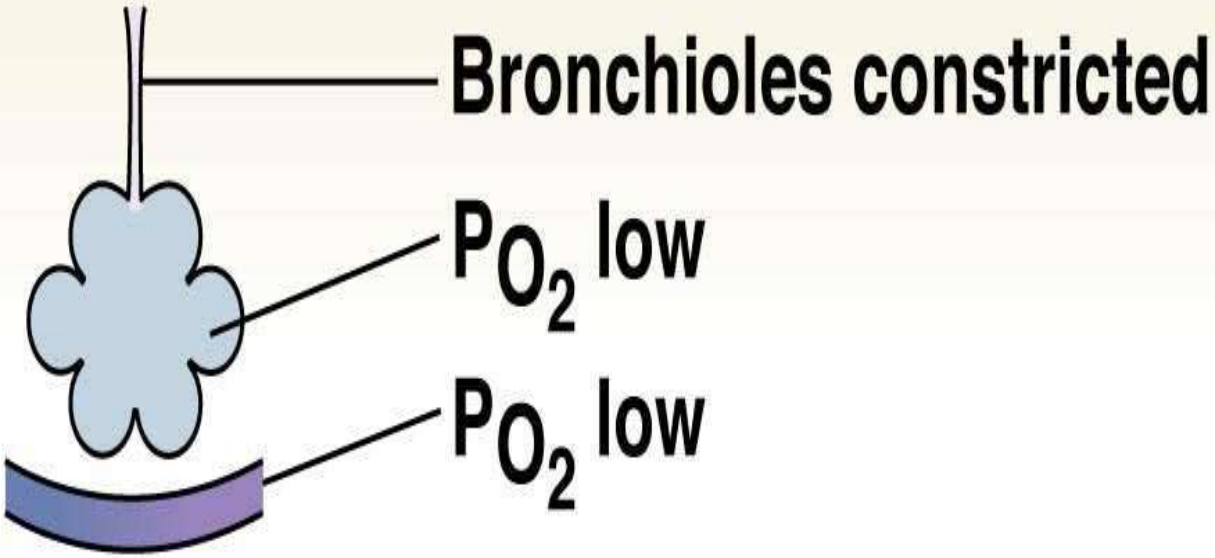
(c) **Fibrotic lung disease:** thickened alveolar membrane slows gas exchange. Loss of lung compliance may decrease alveolar ventilation.



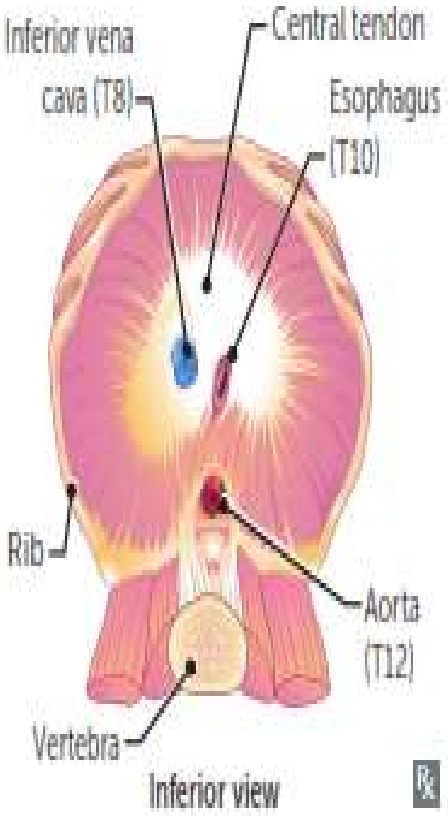
(d) **Pulmonary edema:** fluid in interstitial space increases diffusion distance. Arterial P_{CO_2} may be normal due to higher CO_2 solubility in water.



(e) Asthma: increased airway resistance decreases airway ventilation.



Diaphragm structures



Structures perforating diaphragm:

- At T8: IVC, right phrenic nerve
- At T10: esophagus, vagus (CN 10; 2 trunks)
- At T12: aorta (red), thoracic duct (white), azygos vein (blue) ("At T-1-2 it's the red, white, and blue")

Diaphragm is innervated by C3, 4, and 5 (phrenic nerve). Pain from diaphragm irritation (eg, air, blood, or pus in peritoneal cavity) can be referred to shoulder (C5) and trapezius ridge (C3, 4).

Number of letters = T level:

T8: vena cava

T10: "oesophagus"

T12: aortic hiatus

I (IVC) ate (8) ten (10) eggs (esophagus) at (aorta) twelve (12).

C3, 4, 5 keeps the diaphragm alive.

Other bifurcations:

- The common carotid bifourcates at C4.
- The trachea bifourcates at T4.
- The abdominal aorta bifourcates at L4.

Blood air barrier:

Wall through which gas exchange occur. It is present in() blood in the capillaries & air within lung alveoli.

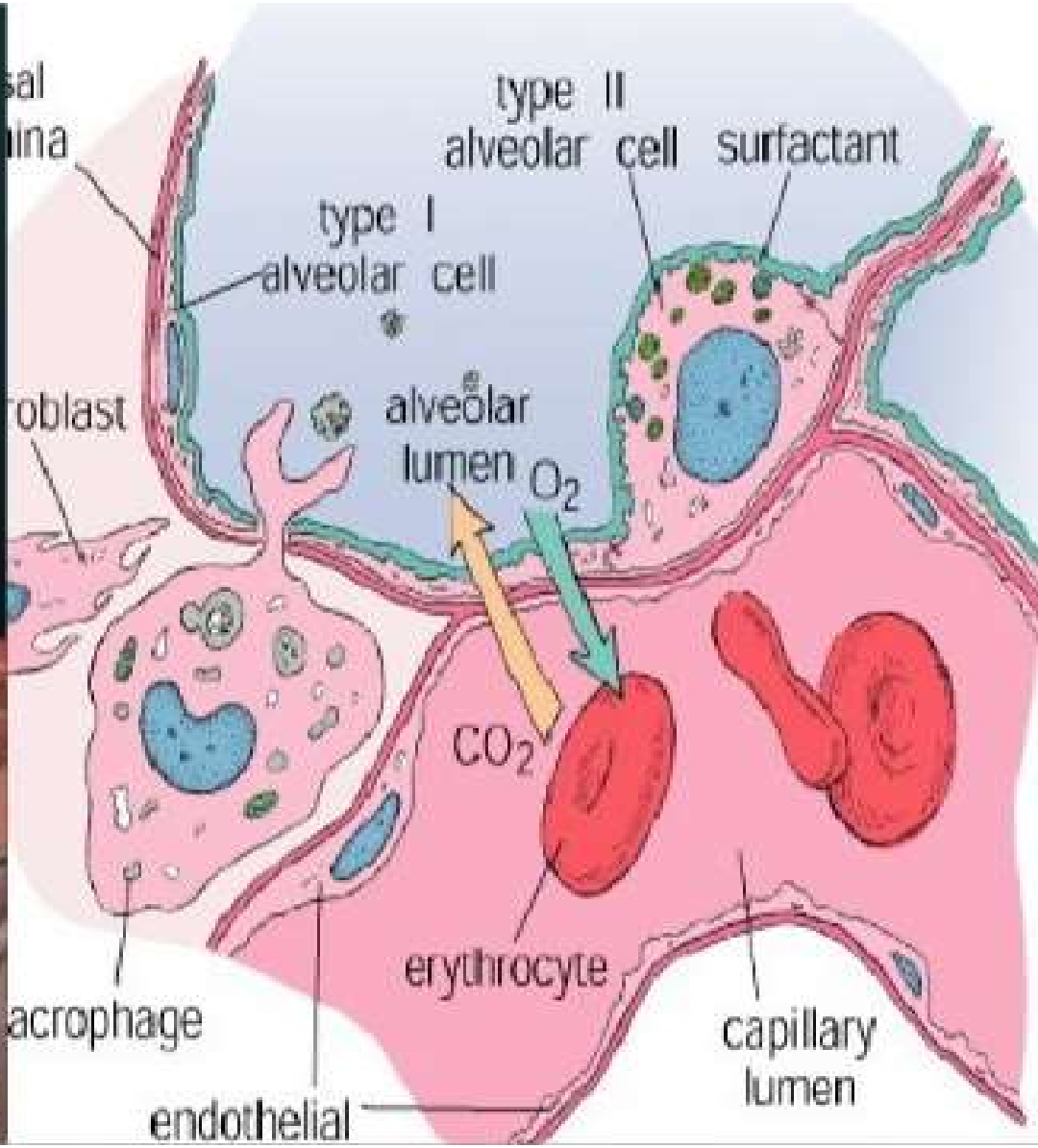
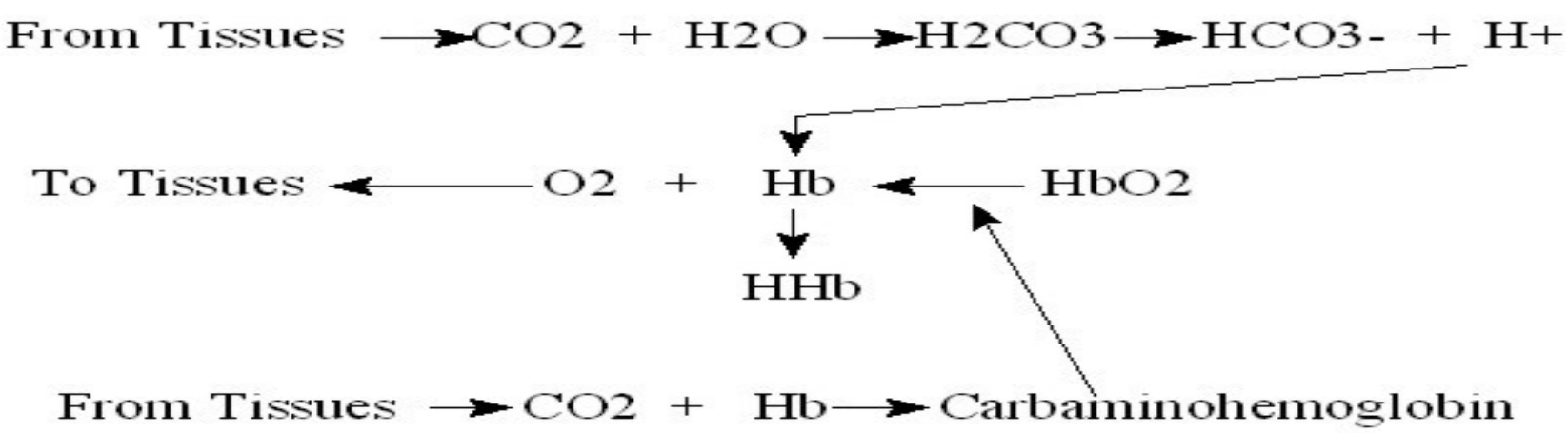


Table 23.2 Respiration Processes

Process	Description	Body Systems
Pulmonary ventilation	Movement of air between atmosphere and the alveoli • Net movement of oxygen from atmosphere to alveoli during inspiration (step 1) • Net movement of carbon dioxide from alveoli to atmosphere during expiration (step 8)	Respiratory, skeletal, muscular, and nervous
Alveolar gas exchange	Exchange of respiratory gases between alveoli of the lungs and the blood • Oxygen diffuses from alveoli into blood (step 2) • Carbon dioxide diffuses from blood into alveoli (step 7)	Respiratory and cardiovascular
Gas transport	Blood transport of respiratory gases between lungs and tissue cells of the body • Oxygen is transported from lungs to tissue cells (step 3) • Carbon dioxide is transported from systemic cells to lungs (step 6)	Cardiovascular
Systemic gas exchange	Exchange of respiratory gases between blood and systemic cells • Oxygen diffuses from blood into tissue cells (step 4) • Carbon dioxide diffuses from systemic cells into blood (step 5)	Cardiovascular

The Bohr Effect Occurs in the Systemic Capillaries



The **Bohr Effect** describes the result of increasing CO2 in causing more oxygen unloading from hemoglobin.

It results from two circumstances:

the effect of lowering pH as described above,

2) the effect of carbaminohemoglobin in stimulating oxygen unloading

Main Gases of the Atmosphere

Gas	Symbol	Approximate %
Nitrogen	N_2	78.6
Oxygen	O_2	20.9
Carbon Dioxide	CO_2	0.04
Water Vapor	H_2O	0.46

Gas Exchange

- Partial Pressure
  - Each gas in atmosphere contributes to the entire atmospheric pressure, denoted as P
- Gases in liquid
  - Gas enters liquid and dissolves in proportion to its partial pressure
- O_2 and CO_2 Exchange by DIFFUSION
  - PO_2 is 105 mmHg in alveoli and 40 in alveolar capillaries
  - PCO_2 is 45 in alveolar capillaries and 40 in alveoli

Partial Pressures

- Oxygen is 21% of atmosphere
- 760 mmHg x .21 = 160 mmHg PO2
- This mixes with “old” air already in alveolus to arrive at PO2 of 105 mmHg

Partial Pressures

- Carbon dioxide is .04% of atmosphere
- 760 mmHg x .0004 = .3 mm Hg PCO_2
- This mixes with high CO_2 levels from residual volume in the alveoli to arrive at PCO_2 of 40 mmHg

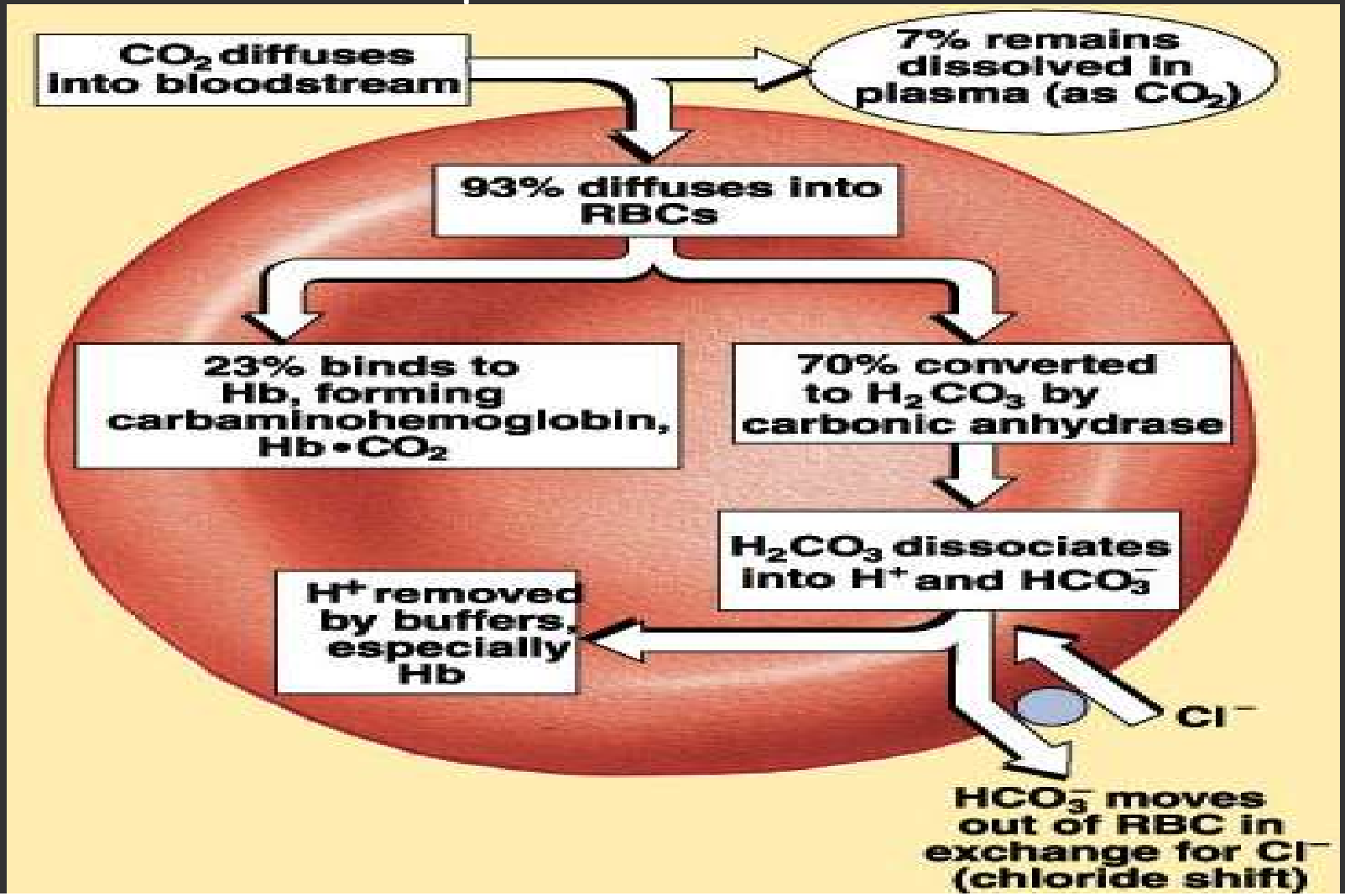
Carbon Dioxide Transport

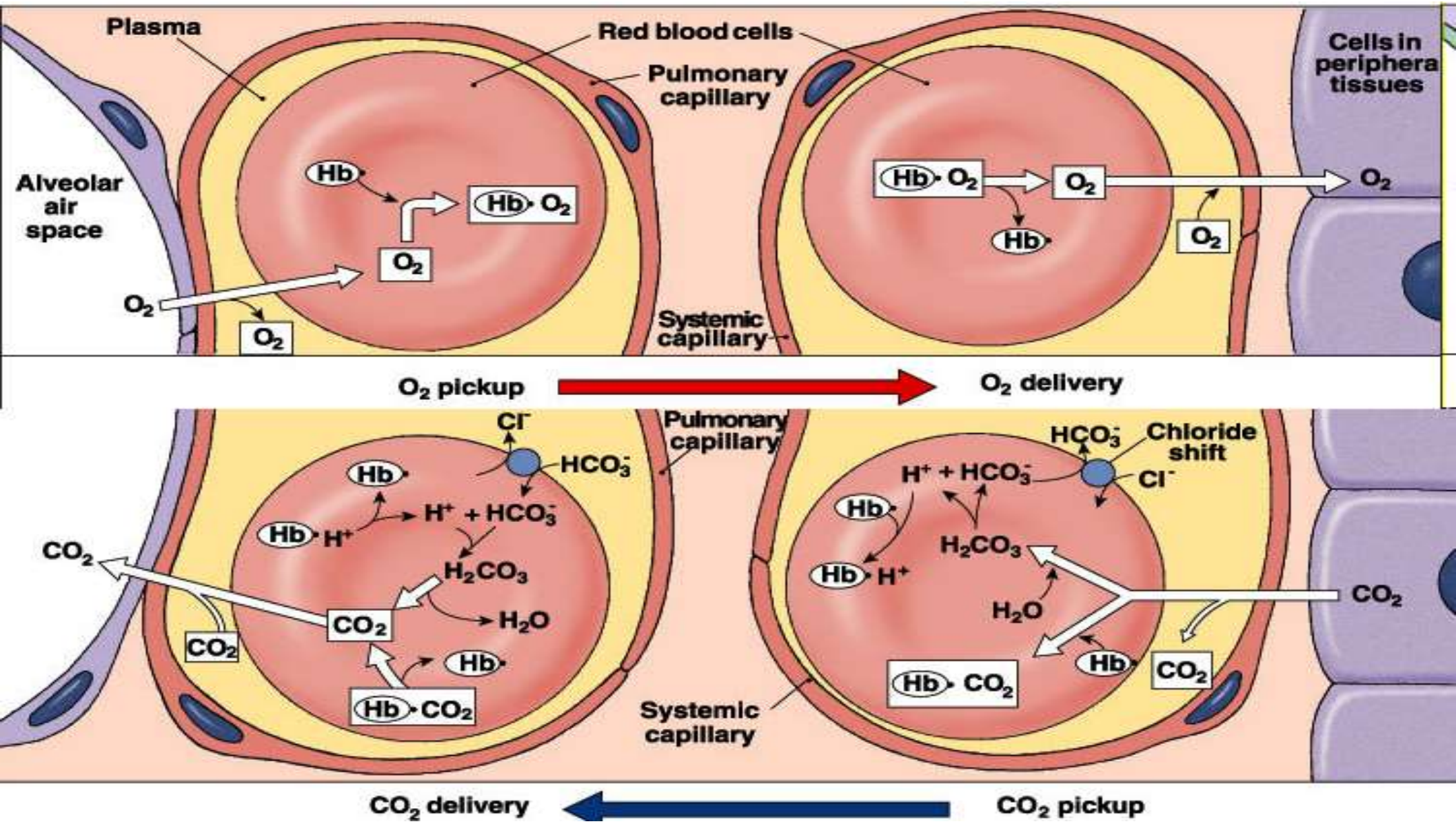
• **Method**	**Percentage**
•Dissolved in Plasma	7 - 10 %
•Chemically Bound to •Hemoglobin in RBC's	20 - 30 %
•As Bicarbonate Ion in •Plasma	60 -70 %

Oxygen Transport

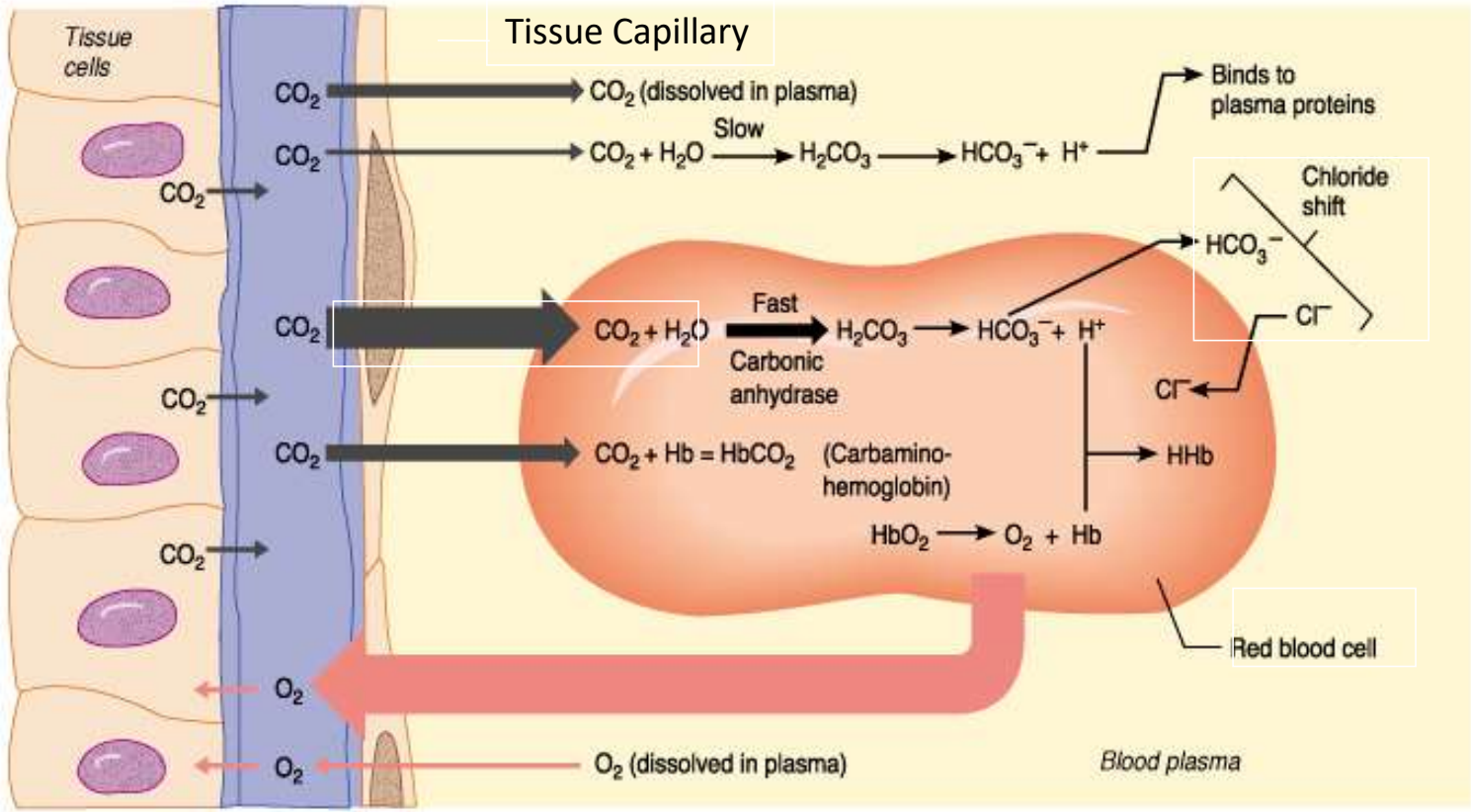
Method	**Percentage**
Dissolved in Plasma	1.5 %
Combined with Hemoglobin	98.5 %

CO2 Transport and Cl- Movement



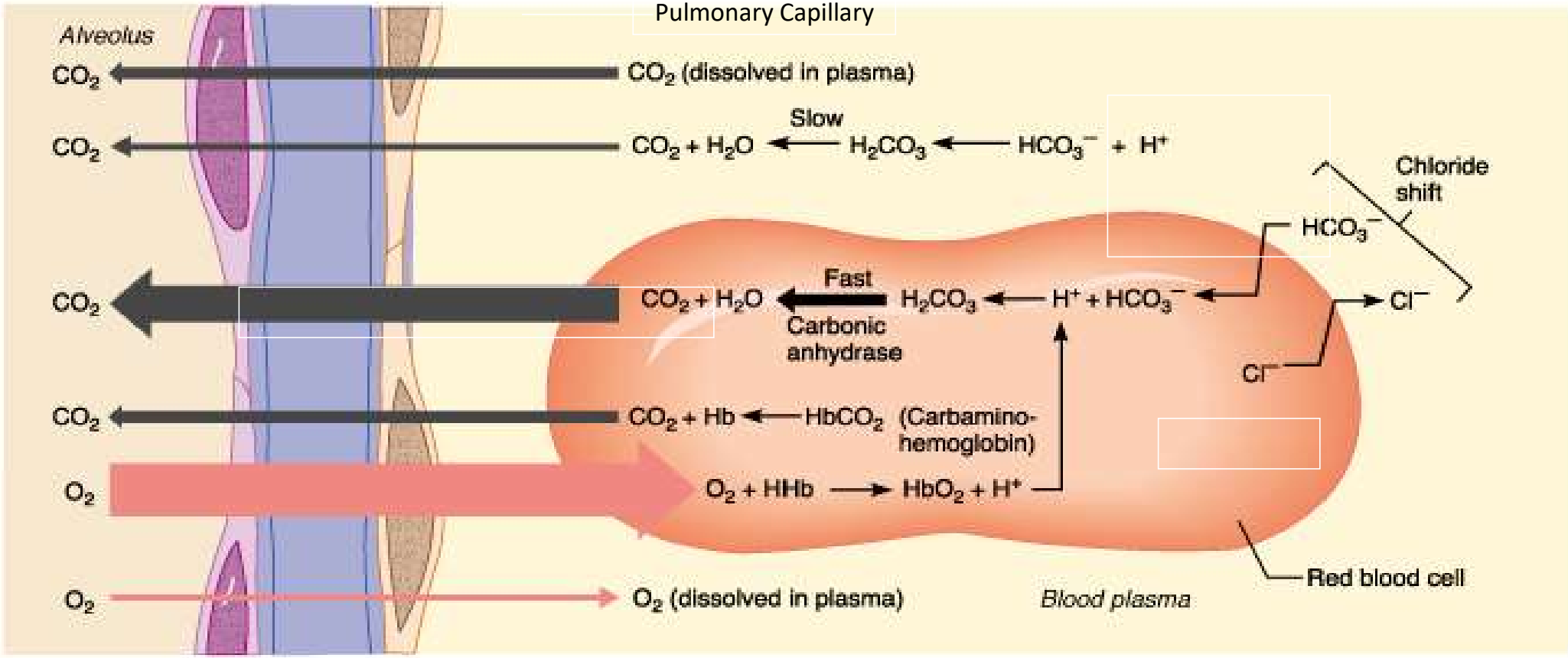

Plasma
Red blood cells
Pulmonary capillary
Cells in peripheral tissues
Alveolar air space
Hb
Hb·O2
O2
O2
O2
Hb·O2
O2
Hb
O2
O2
Systemic capillary
O2 pickup
O2 delivery
Cl-
HCO3-
Pulmonary capillary
HCO3-
Chloride shift
Hb
H+ + HCO3-
Cl-
Hb·H+
H+ + HCO3-
CO2
H2CO3
Hb
H2CO3
H2O
Hb·H+
CO2
CO2
H2O
CO2
Hb
Hb
Hb·CO2
Hb·CO2
CO2
Systemic capillary
CO2 delivery
CO2 pickup

Chloride Shift in Tissue Capillaries

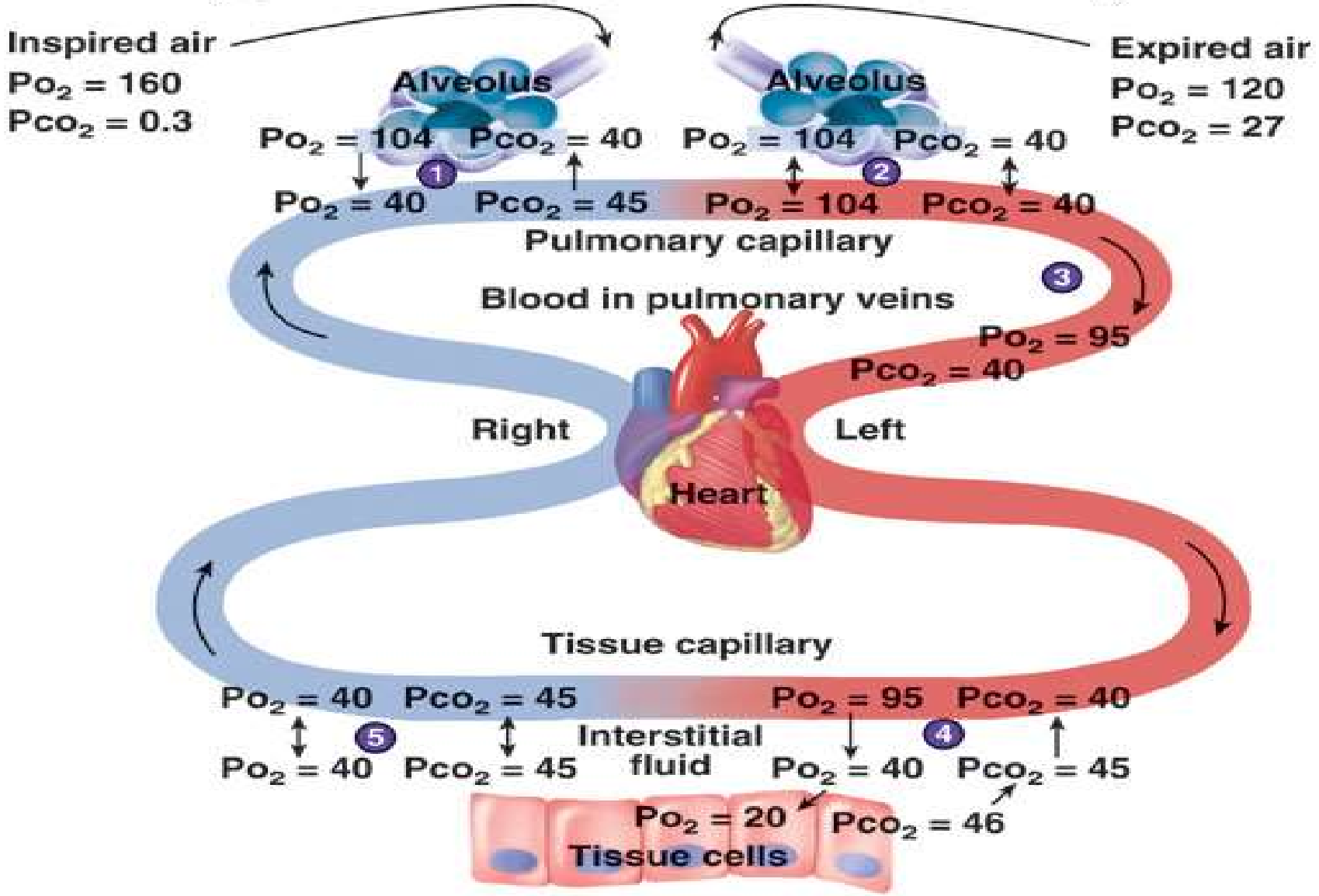


(a) Oxygen release and carbon dioxide pickup at the tissues

Chloride Shift in Pulmonary Capillaries



(b) Oxygen pickup and carbon dioxide release in the lungs



Pulmonary vascular resistance

$$PVR = \frac{P_{\text{pulm artery}} - P_{\text{L atrium}}}{\text{cardiac output}}$$

Remember: $\Delta P = Q \times R$, so $R = \Delta P / Q$

$R = 8\eta l / \pi r^4$

$P_{\text{pulm artery}}$ = pressure in pulmonary artery
$P_{\text{L atrium}} \approx$ pulmonary capillary wedge pressure
Q = cardiac output (flow)
R = resistance
η = viscosity of blood
l = vessel length
r = vessel radius

Alveolar gas equation

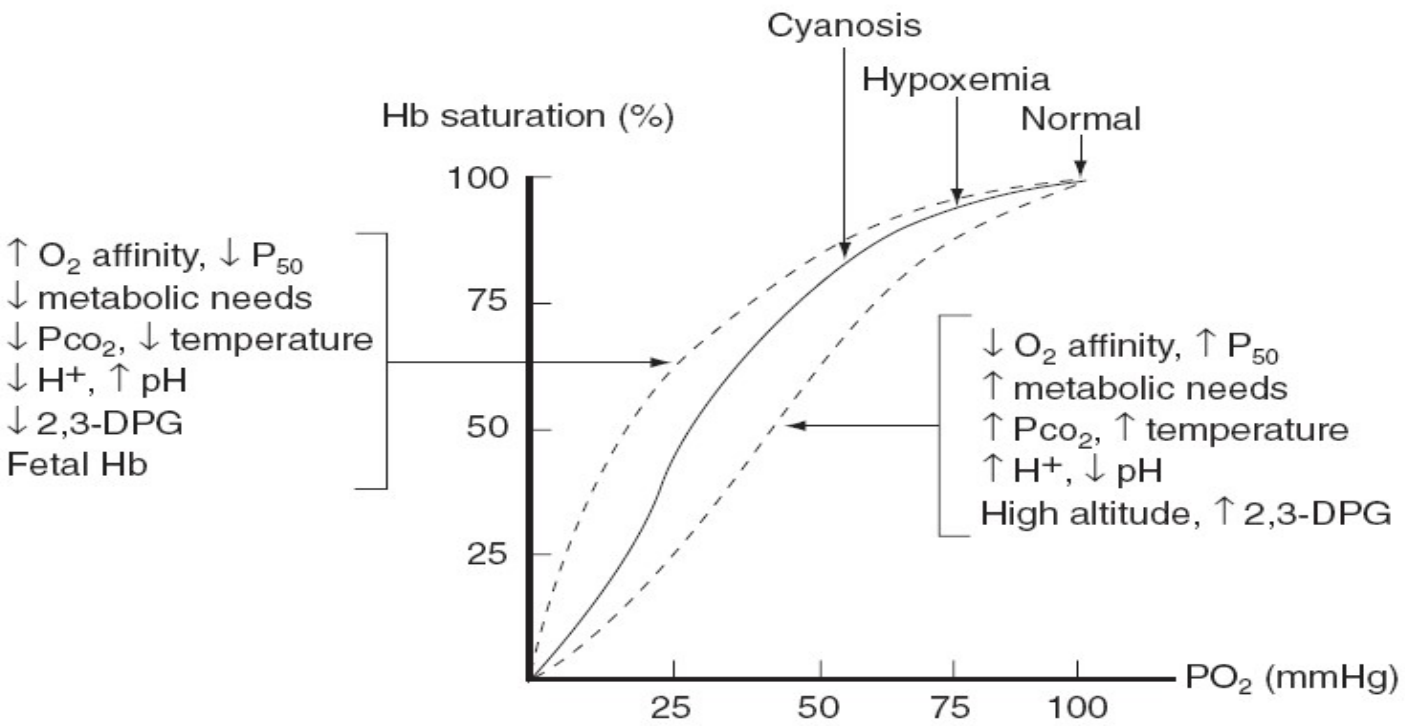
$$P_{A}O_2 = PIO_2 - \frac{PaCO_2}{R}$$

$$\approx 150 \text{ mm Hg}^{a} - \frac{PaCO_2}{0.8}$$

[a]At sea level breathing room air

$P_{A}O_2$ = alveolar P_{O_2} (mm Hg)
PIO_2 = P_{O_2} in inspired air (mm Hg)
$PaCO_2$ = arterial P_{CO_2} (mm Hg)
R = respiratory quotient = CO_2 produced/O_2 consumed
A-a gradient = $P_{A}O_2 - PaO_2$. Normal range = 10–15 mm Hg
↑ A-a gradient may occur in hypoxemia; causes include shunting, $\dot{V}/\dot{Q}$ mismatch, fibrosis (impairs diffusion)

Oxygen-hemoglobin dissociation curve



Sigmoidal shape due to positive cooperativity, i.e., hemoglobin can bind 4 oxygen molecules and has higher affinity for each subsequent oxygen molecule bound.

When curve shifts to the right, ↓ affinity of hemoglobin for O_2 (facilitates unloading of O_2 to tissue).

An ↑ in all factors (except pH) causes a shift of the curve to the right.

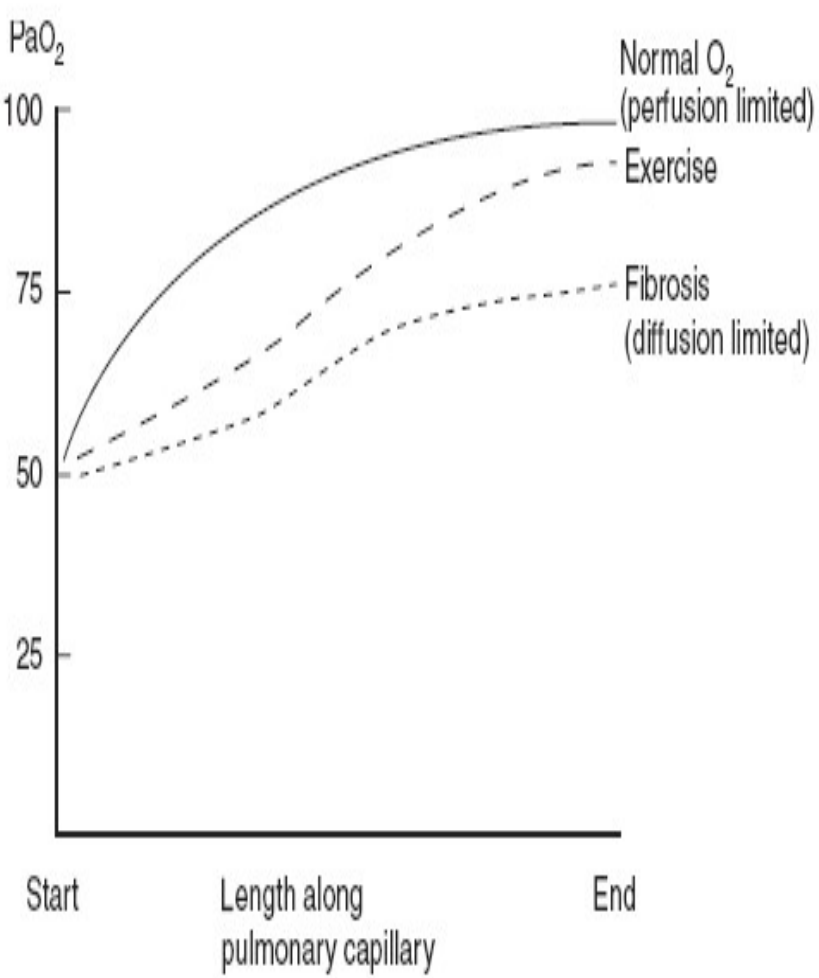
A ↓ in all factors (except pH) causes a shift of the curve to the left.

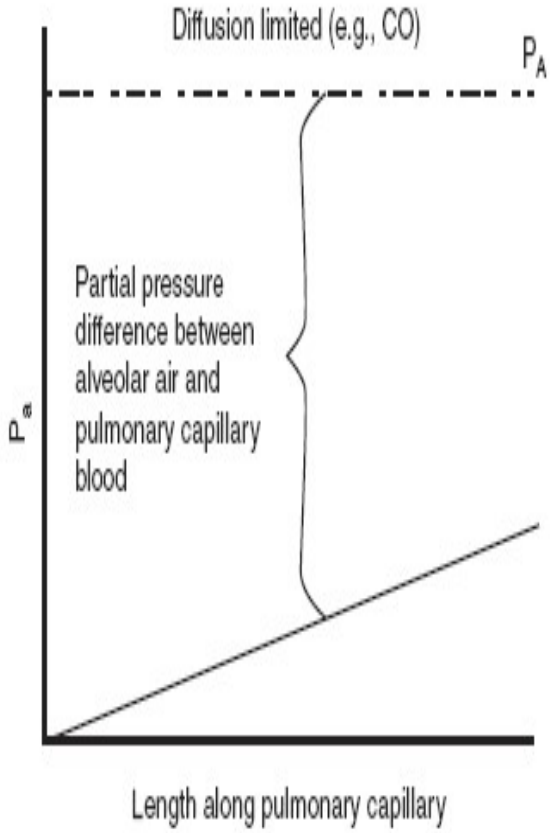
Fetal Hb has a higher affinity for oxygen than adult Hb, so its dissociation curve is shifted left.

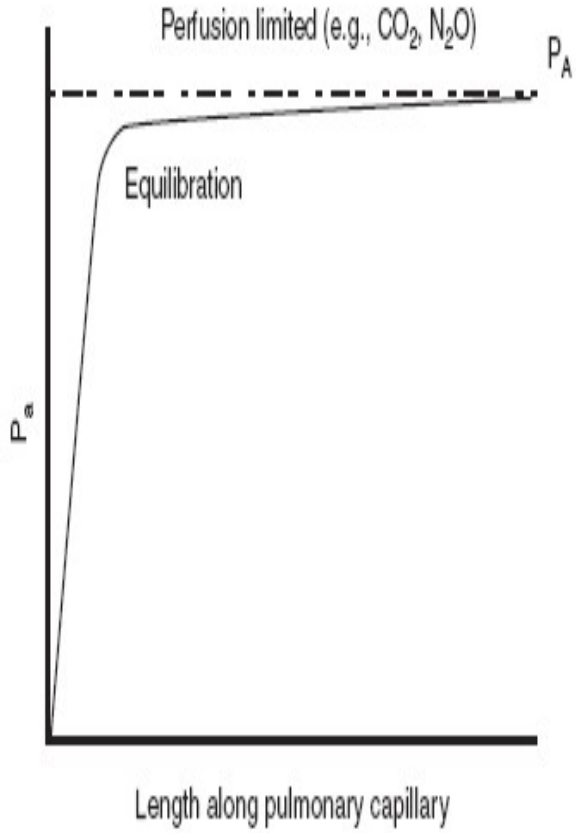
Right shift—**CADET** face **right**:

C CO_2
A Acid/Altitude
D DPG (2,3-DPG)
E Exercise
T Temperature

Pulmonary circulation	Normally a low-resistance, high-compliance system. P_{O_2} and P_{CO_2} exert opposite effects on pulmonary and systemic circulation. A ↓ in Pa_{O_2} causes a hypoxic vasoconstriction that shifts blood away from poorly ventilated regions of lung to well-ventilated regions of lung. 1. Perfusion limited—O_2 (normal health), CO_2, N_2O. Gas equilibrates early along the length of the capillary. Diffusion can be ↑ only if blood flow ↑. 2. Diffusion limited—O_2 (emphysema, fibrosis), CO. Gas does not equilibrate by the time blood reaches the end of the capillary.	A consequence of pulmonary hypertension is cor pulmonale and subsequent right ventricular failure (jugular venous distention, edema, hepatomegaly).







P_a = partial pressure of gas in pulmonary capillary blood
P_A = partial pressure of gas in alveolar air

Pulmonary vascular resistance (PVR)

$$PVR = \frac{P_{pulm\ artery} - P_{L\ atrium}}{\text{Cardiac output}}$$

Remember: $\Delta P = Q \times R$, so $R = \Delta P / Q$.

$R = 8\eta l / \pi r^4$

$P_{pulm\ artery}$ = pressure in pulmonary artery.

$P_{L\ atrium}$ = pulmonary wedge pressure.

η = the viscosity of inspired air;

l = airway length;

r = airway radius.

Oxygen content of blood

O_2 content = (O_2 binding capacity × % saturation) + dissolved O_2.

Normally 1 g Hb can bind 1.34 mL O_2; normal Hb amount in blood is 15 g/dL.

Cyanosis results when Hb is < 5 g/dL.

O_2 binding capacity ≈ 20.1 mL O_2 / dL.

O_2 content of arterial blood ↓ as Hb falls, but O_2 saturation and arterial P_{O_2} do not.

Arterial P_{O_2} ↓ with chronic lung disease because physiologic shunt ↓ O_2 extraction ratio.

Oxygen delivery to tissues = cardiac output × oxygen content of blood.

Alveolar gas equation

$$PAO_2 = PIO_2 - \frac{PACO_2}{R}$$

Can normally be approximated:

$$PAO_2 = 150 - PaCO_2 / 0.8$$

PAO_2 = alveolar PO_2 (mmHg).

PIO_2 = PO_2 in inspired air (mmHg).

$PACO_2$ = alveolar PCO_2 (mmHg).

R = respiratory quotient.

A-a gradient = $PAO_2 - PaO_2$ = 10–15 mmHg.

↑ A-a gradient may occur in hypoxemia; causes include shunting, V/Q mismatch, fibrosis (diffusion block).

	Hb CONCENTRATION	% O_2 SAT OF Hb	DISSOLVED O_2 (Pao_2)	TOTAL O_2 CONTENT
CO poisoning	Normal	↓ (CO competes with O_2)	Normal	↓
Anemia	↓	Normal	Normal	↓
Polycythemia	↑	Normal	Normal	↑

V/Q mismatch

Ideally, ventilation is matched to perfusion (i.e., V/Q = 1) in order for adequate gas exchange to occur.

Lung zones:

1. Apex of the lung—V/Q = 3 (wasted ventilation)
2. Base of the lung—V/Q = 0.6 (wasted perfusion)

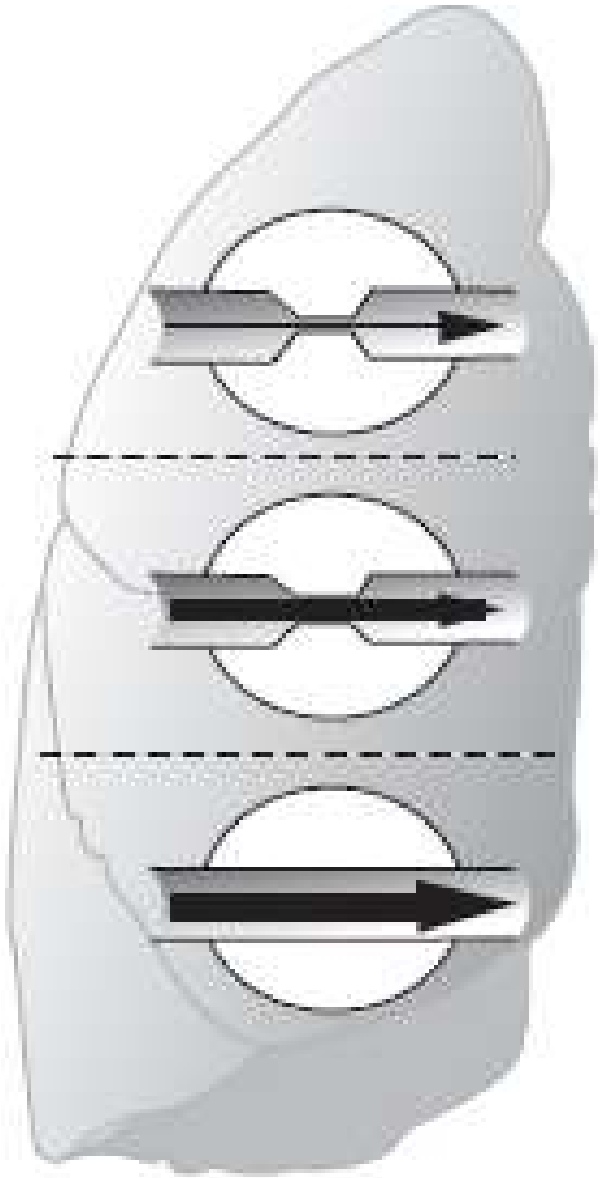
Both ventilation and perfusion are greater at the base of the lung than at the apex of the lung.

With exercise (↑ cardiac output), there is vasodilation of apical capillaries, resulting in a V/Q ratio that approaches 1.

Certain organisms that thrive in high O_2 (e.g., TB) flourish in the apex.

V/Q → 0 = airway obstruction (shunt). In shunt, 100% O_2 does not improve P_{O_2}.

V/Q → ∞ = blood flow obstruction (physiologic dead space). Assuming < 100% dead space, 100% O_2 improves P_{O_2}.

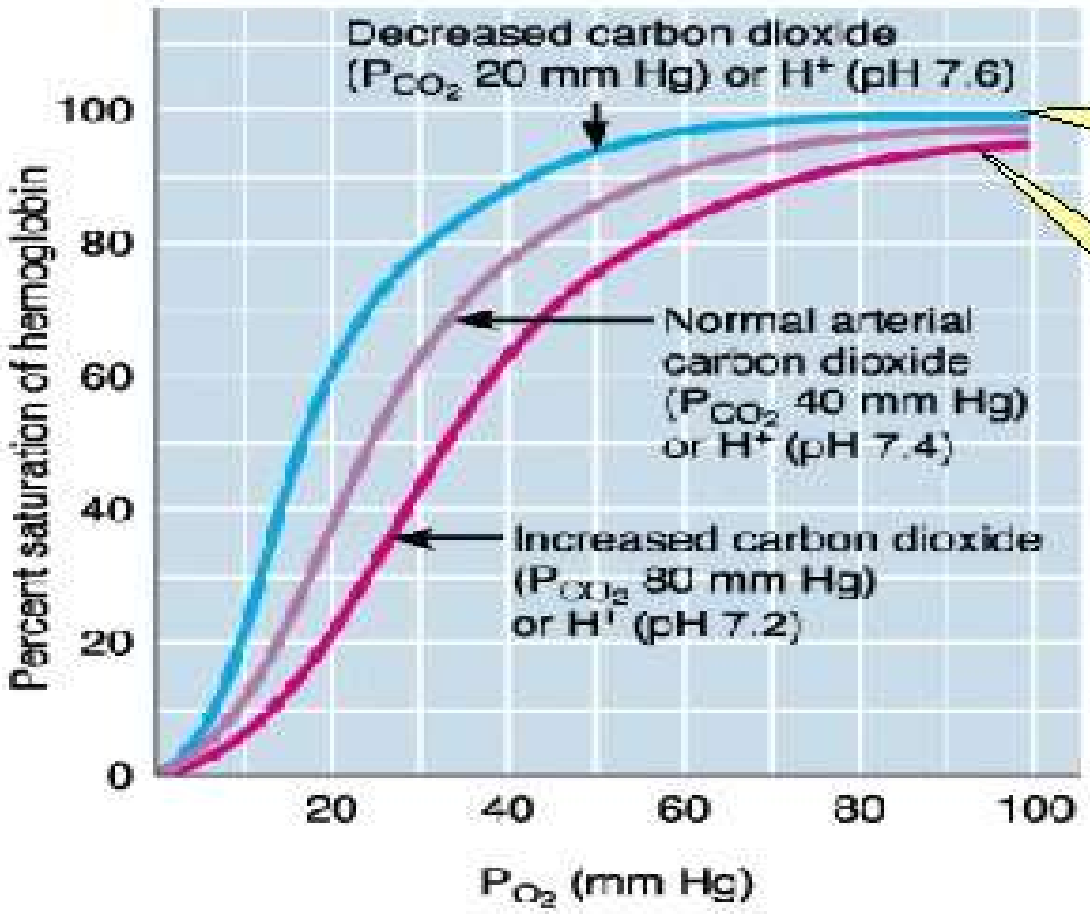


Apex: $P_A > P_a > P_v \rightarrow V/Q = 3$
(wasted ventilation)

$P_a > P_A > P_v$

Base: $P_a > P_v > P_A \rightarrow V/Q = 0.6$
(wasted perfusion); NOTE: both ventilation and perfusion are greater at the base of the lung than at the apex

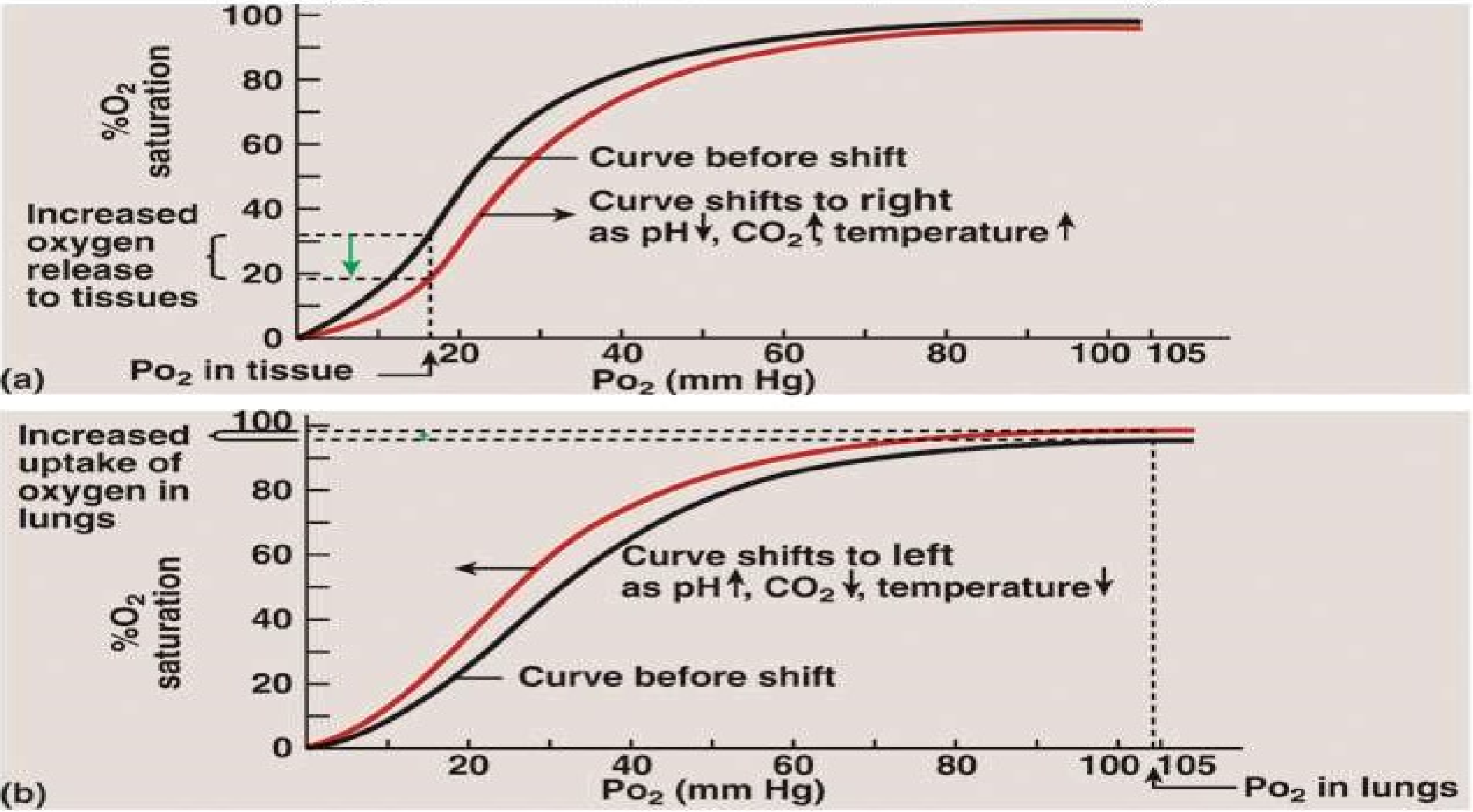
Effect of pH on Respiration

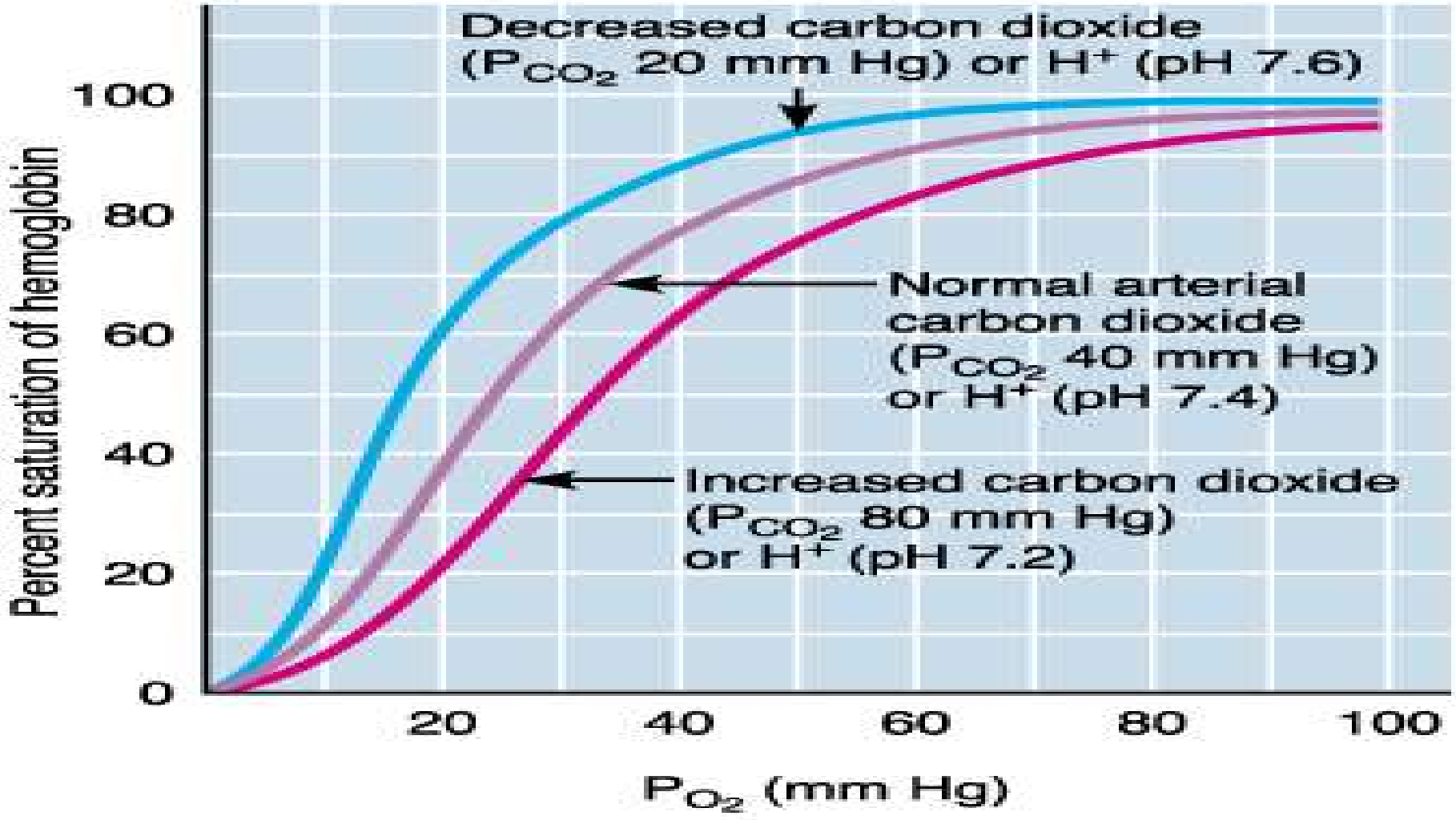


(b)

↑ pH resulting from ↓CO2 or H+ increases the association of oxygen with hemoglobin.

↓ pH resulting from ↑CO2 or H+ decreases the association of oxygen with hemoglobin.





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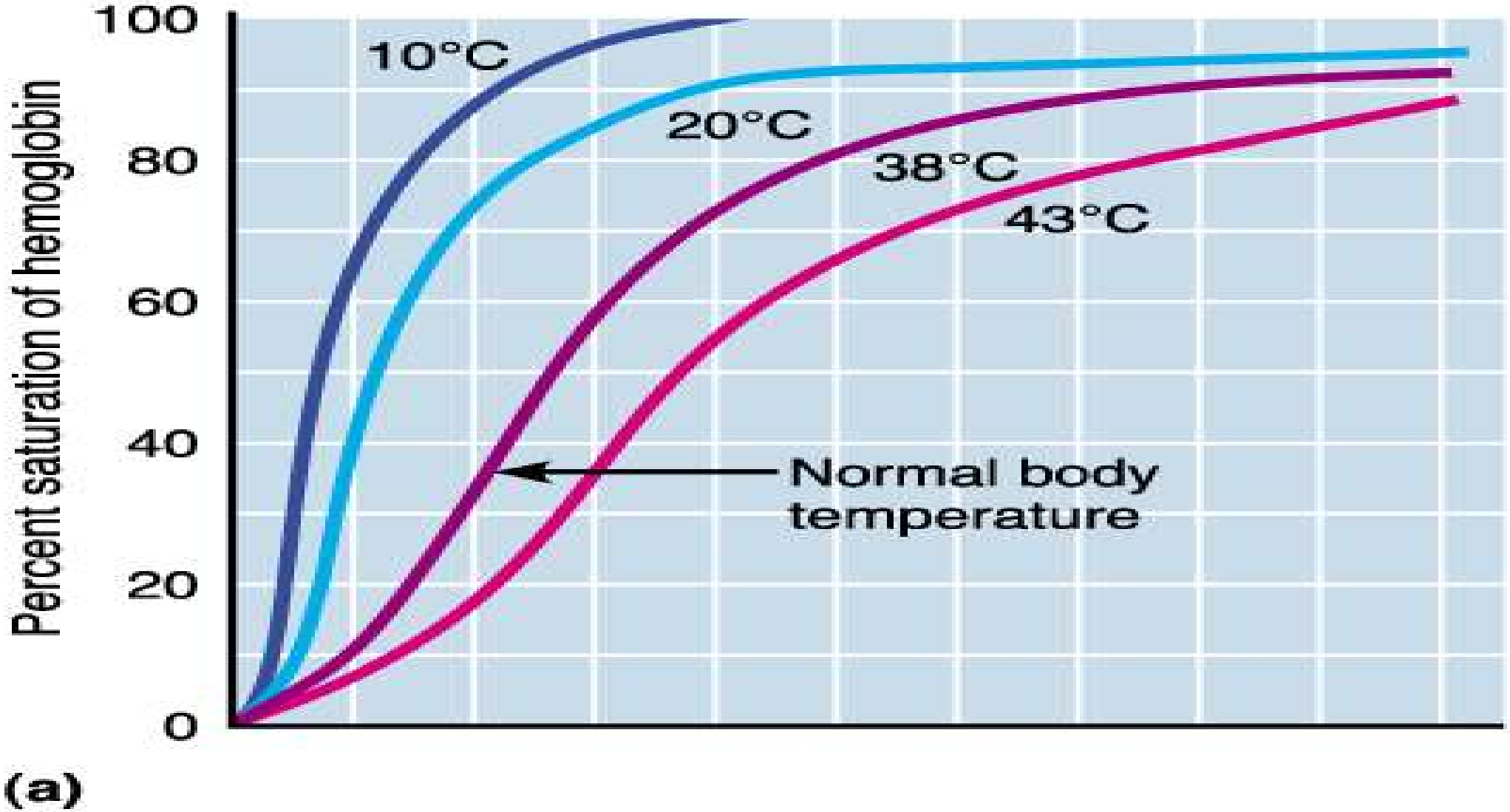
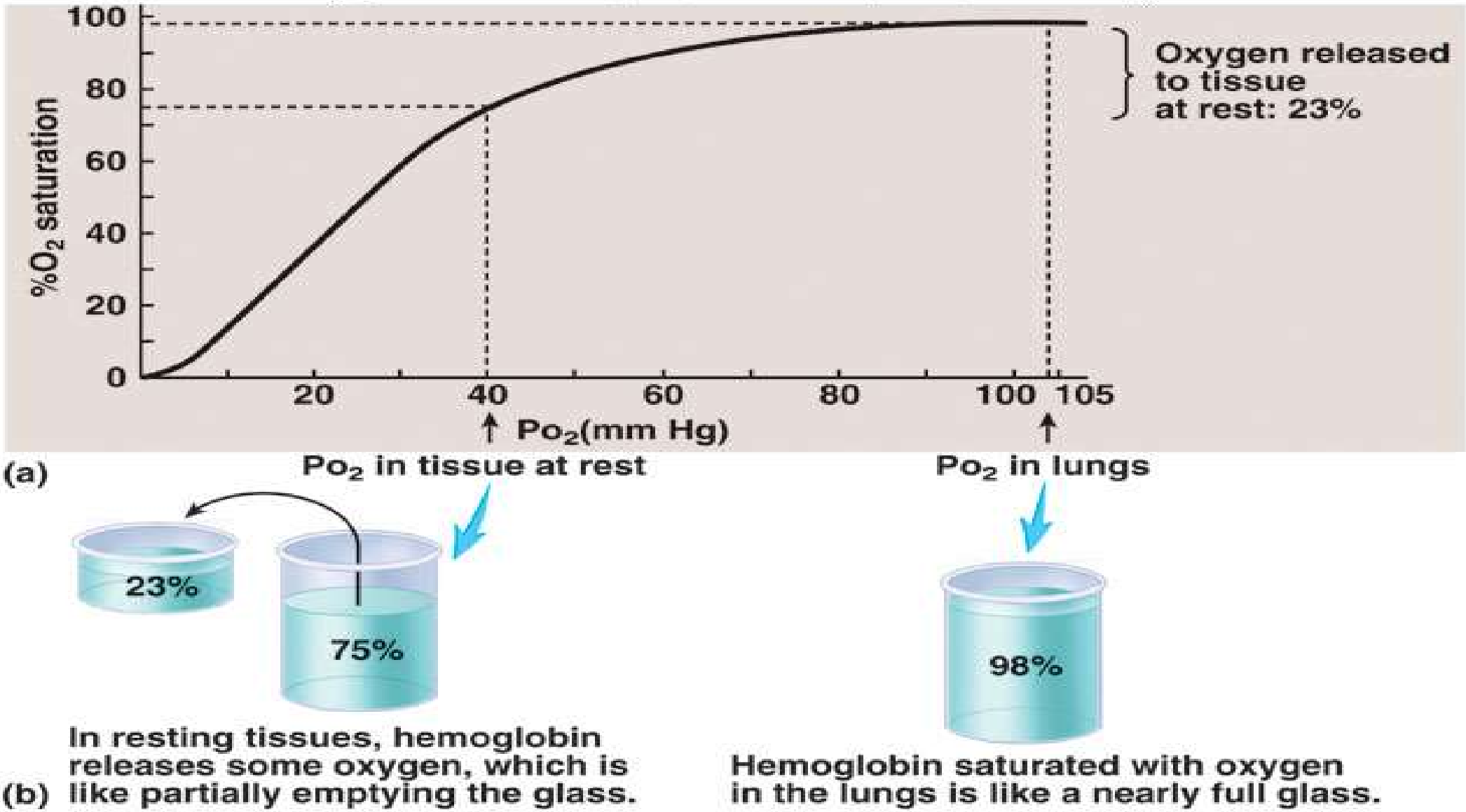
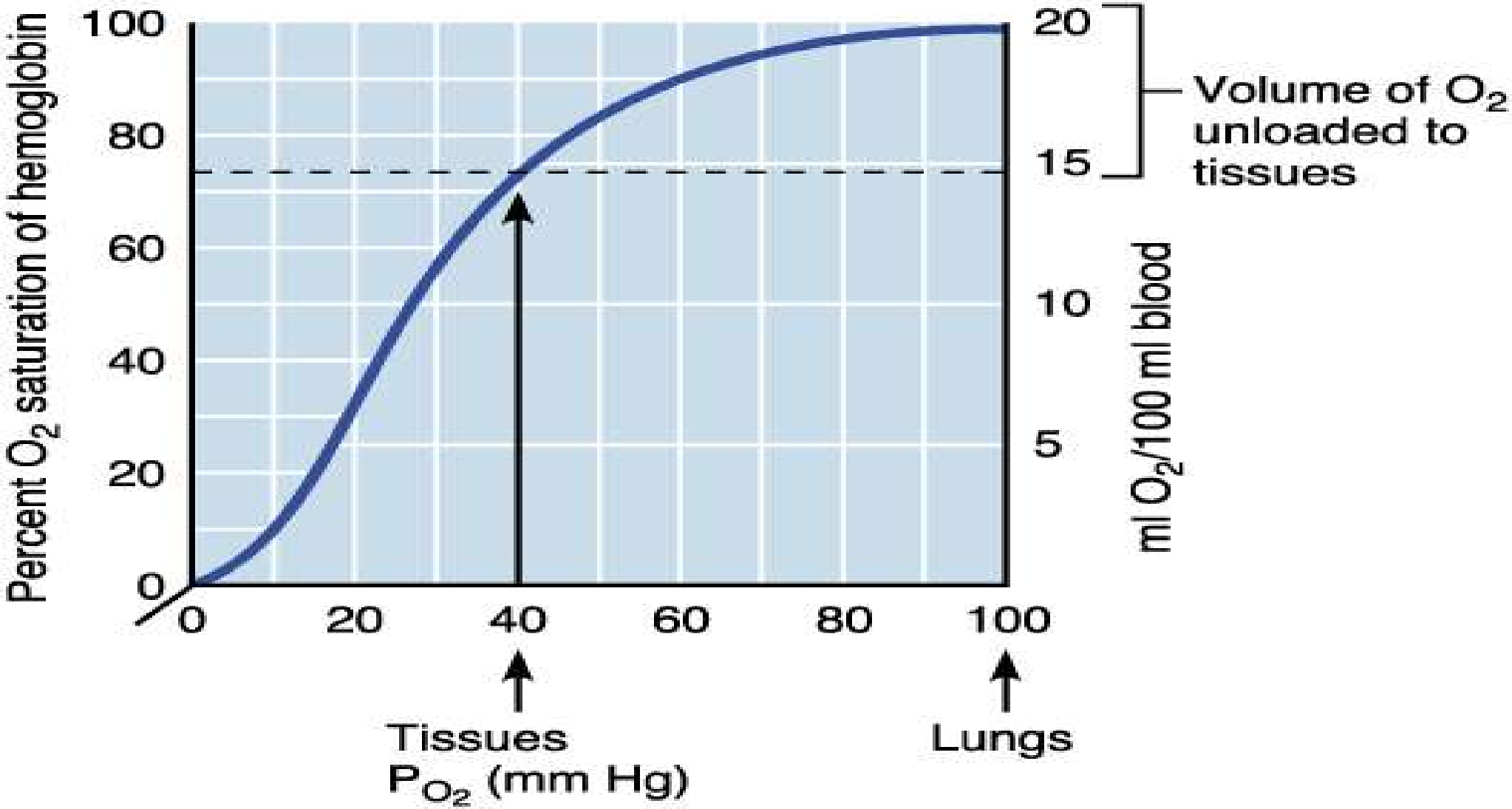


(a)





Response to High Altitude

- increased Ventilation (EARLIEST CHANGE)
- increased Sensitivity of central receptors
- increased Response of carotid bodies
- increased Erythropoietin
- increased 2 3 DPG
- increased Mitochondria
- increased Renal excretion of Bicarbonate

•Respiratory alkalosis

•Pulmonary edema when occurs is due to increased pulmonary capillary pressure

•High altitude → ↓ atmospheric pressure (Patm) and ↓ alveolar PO_2

•Ventilation

- •↓ alveolar PO_2 → ↑ respiratory rate (hyperventilation)
- ↓ alveolar PO_2 stimulates peripheral chemoreceptors in aortic bodies and carotid bodies to instruct medullary inspiration center to increase respiratory rate

•Arterial blood

•↑ ventilation rate → ↑ PaO_2 and ↓ $PaCO_2$ → respiratory alkalosis

•A number of physiologic changes occur in a person living at high altitude.

- The diminished barometric pressure at high altitude
- causes alveolar hypoxia and arterial hypoxia.
- Pulmonary vasoconstriction occurs in response to alveolar hypoxia; therefore, the diameter of the pulmonary vessels would be greater in the brother living at sea level.
- Increased erythropoietin production , caused by arterial hypoxia, leads to increases in hematocrit in people living at high altitude

•Mitochondrial density increases in people chronically exposed to the hypoxemia caused by living at high altitude

•At high altitudes, the ventilation rate increases, causing a respiratory alkalosis.

The kidney then compensates by increasing the excretion of HCO3

•Increasing the rate of respiration is a very useful adaptation to the hypoxic conditions of high altitude. The primary stimulus is the hypoxic stimulation of peripheral chemoreceptors.

Oxygen deprivation

Hypoxia (↓ O_2 delivery to tissue)	Hypoxemia (↓ Pao_2)	Ischemia (loss of blood flow)
↓ cardiac output	Normal A-a gradient	Impeded arterial flow
Hypoxemia	▪ High altitude	↓ venous drainage
Anemia	▪ Hypoventilation (eg, opioid use)	
CO poisoning	↑ A-a gradient	
	▪ $\dot{V}/\dot{Q}$ mismatch	
	▪ Diffusion limitation (eg, fibrosis)	
	▪ Right-to-left shunt	

Response to high altitude

↓ atmospheric oxygen (PO_2) → ↓ Pa_{O_2} → ↑ ventilation → ↓ Pa_{CO_2} → respiratory alkalosis → altitude sickness.

Chronic ↑ in ventilation.

↑ erythropoietin → ↑ Hct and Hb (due to chronic hypoxia).

↑ 2,3-BPG (binds to Hb causing left shift so that Hb releases more O_2).

Cellular changes (↑ mitochondria).

↑ renal excretion of HCO_3^- to compensate for respiratory alkalosis (can augment with acetazolamide).

Chronic hypoxic pulmonary vasoconstriction results in pulmonary hypertension and RVH.

Response to exercise

↑ CO_2 production.

↑ O_2 consumption.

↑ ventilation rate to meet O_2 demand.

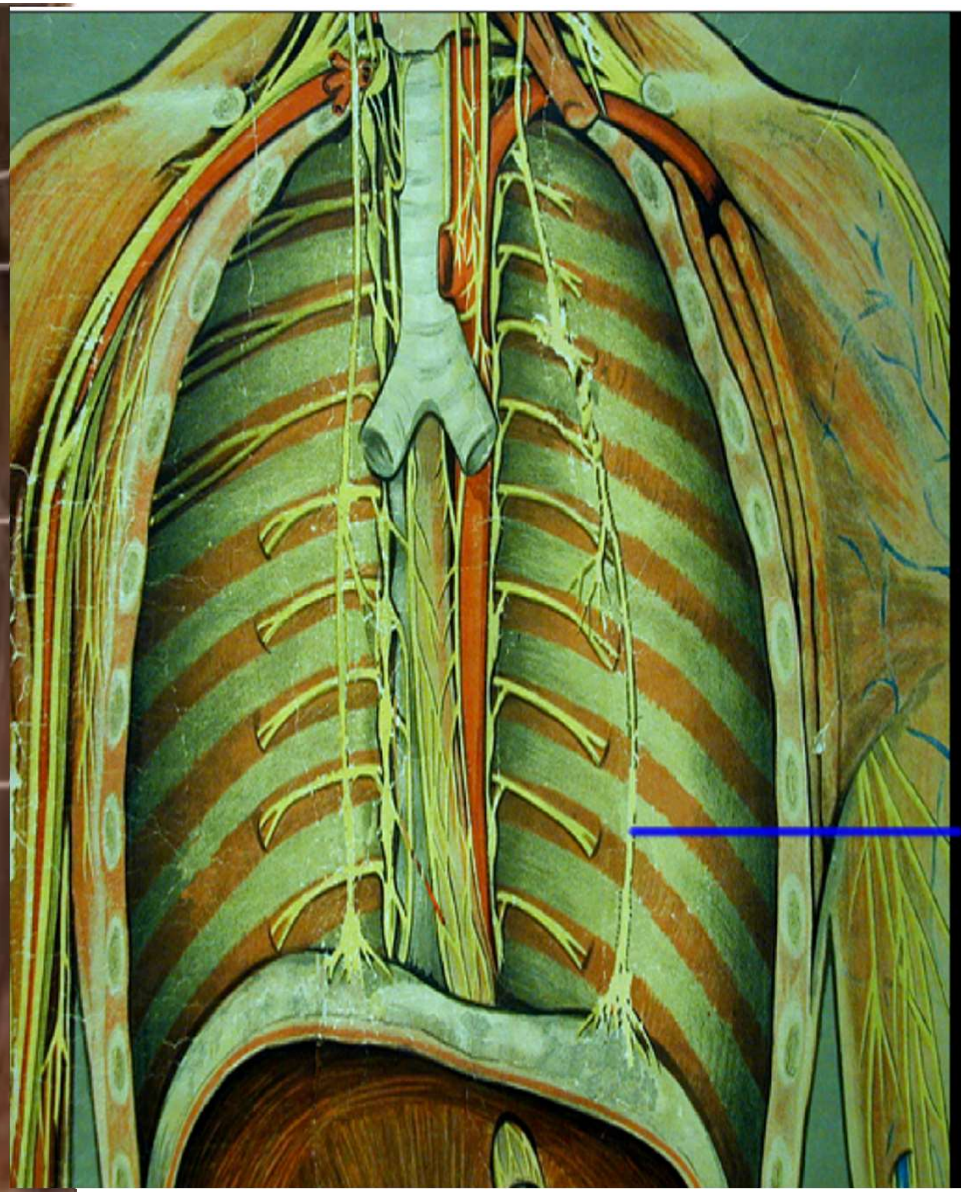
$\dot{V}/\dot{Q}$ ratio from apex to base becomes more uniform.

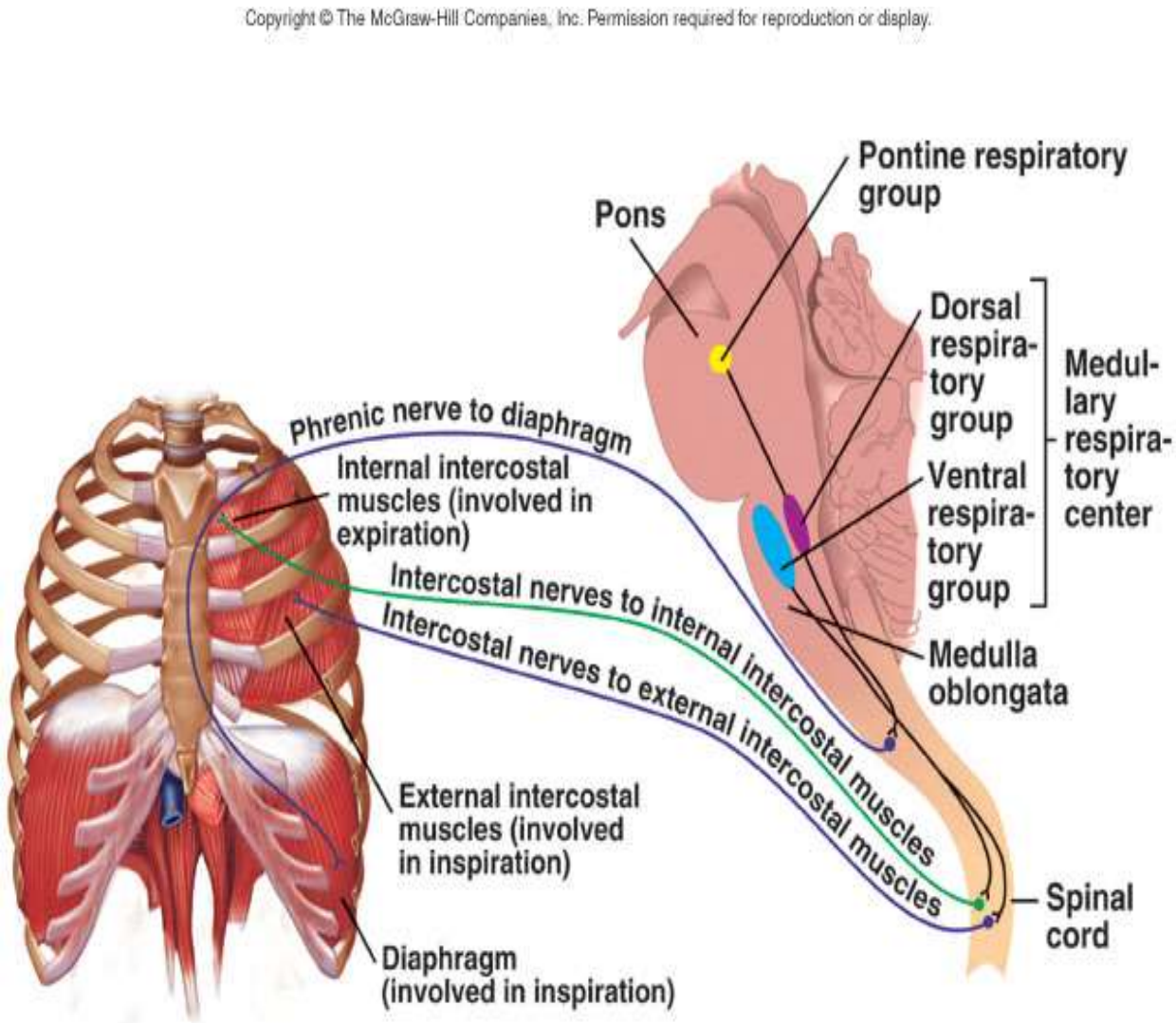
↑ pulmonary blood flow due to ↑ cardiac output.

↓ pH during strenuous exercise (2° to lactic acidosis).

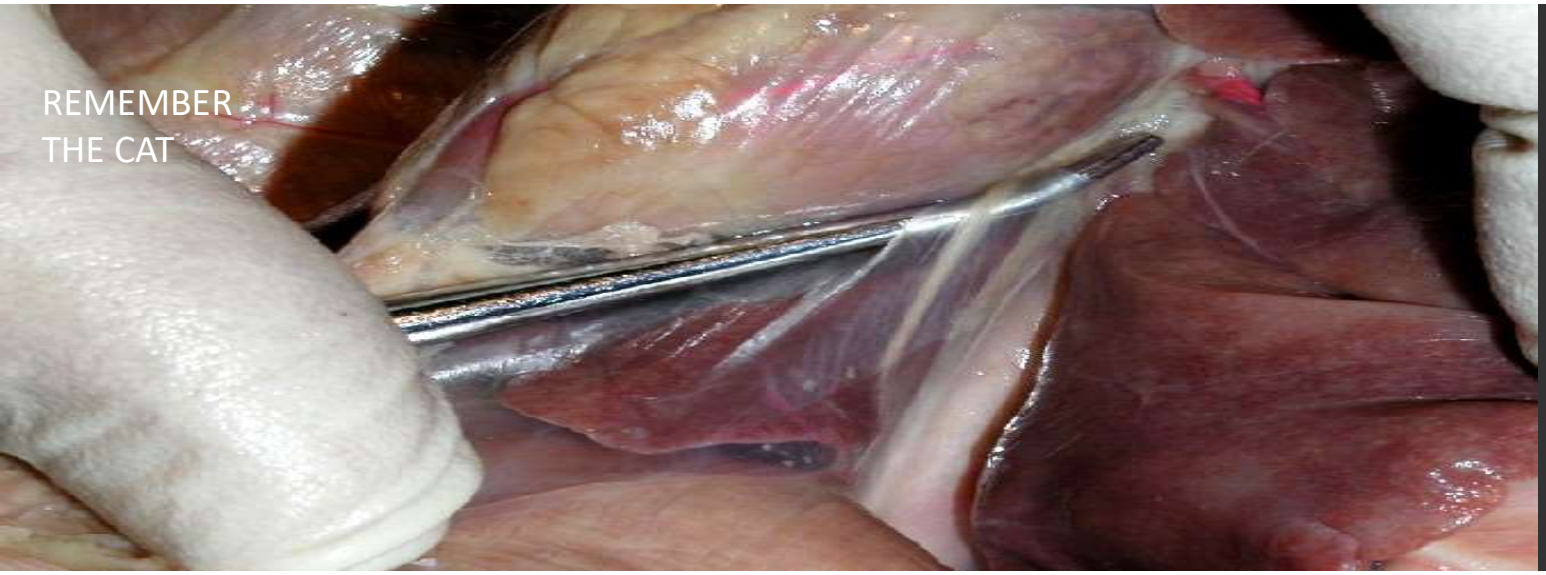
No change in Pa_{O_2} and Pa_{CO_2}, but ↑ in venous CO_2 content and ↓ in venous O_2 content.

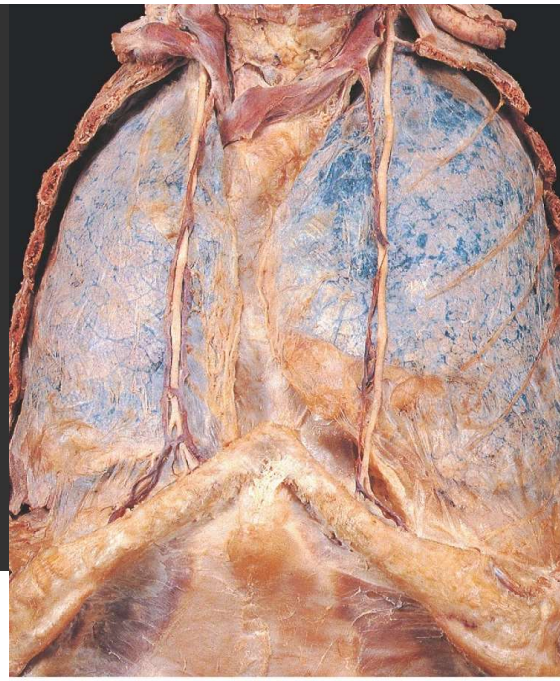
Part 3

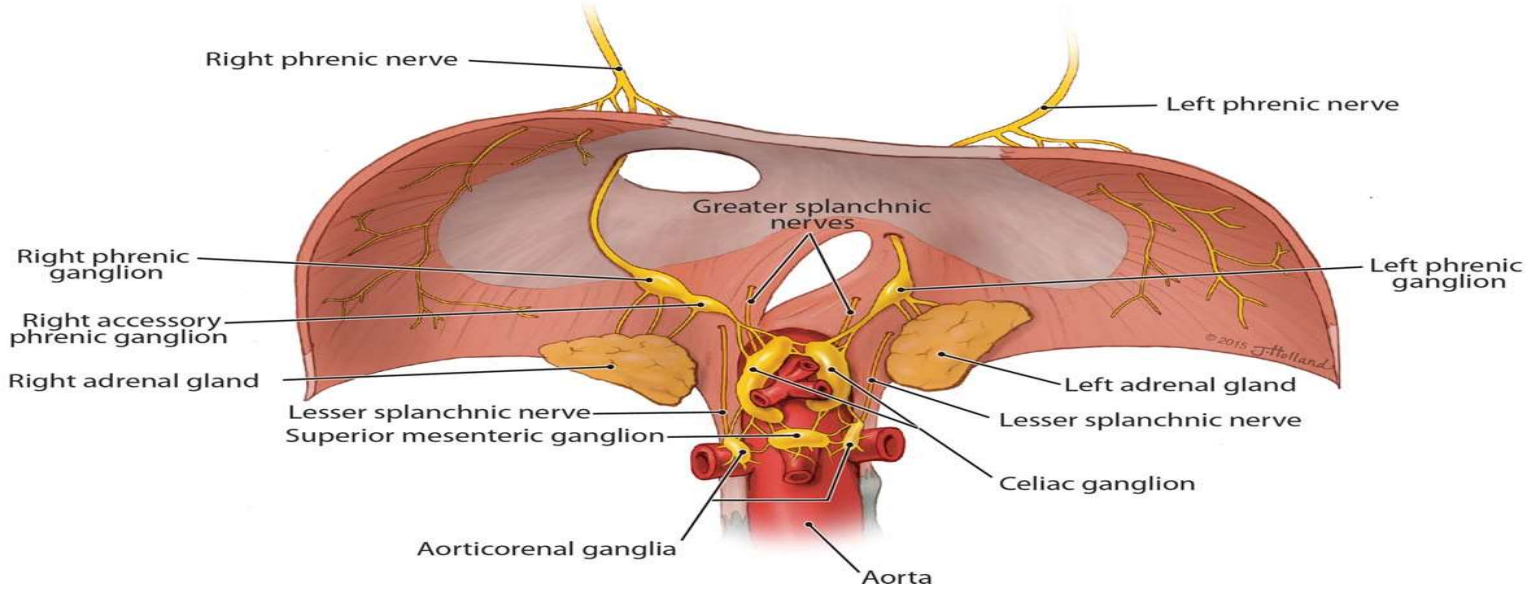

Phrenic Nerve

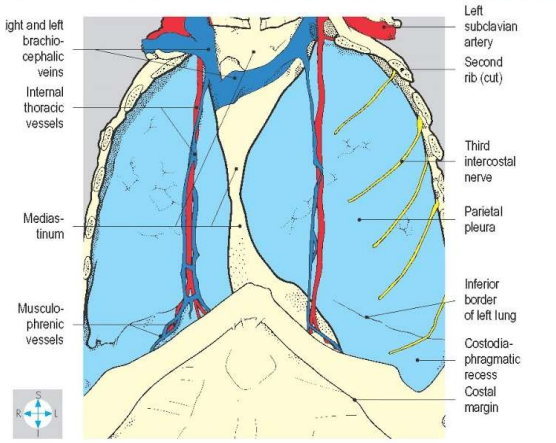


REMEMBER
THE CAT









ig. 2.17 Removal of the anterior chest wall has exposed the internal thoracic vessels and costal pa
f the parietal pleura, through which the lungs are visible.

- **Respiratory muscles** – diaphragm and other muscles that promote ventilation

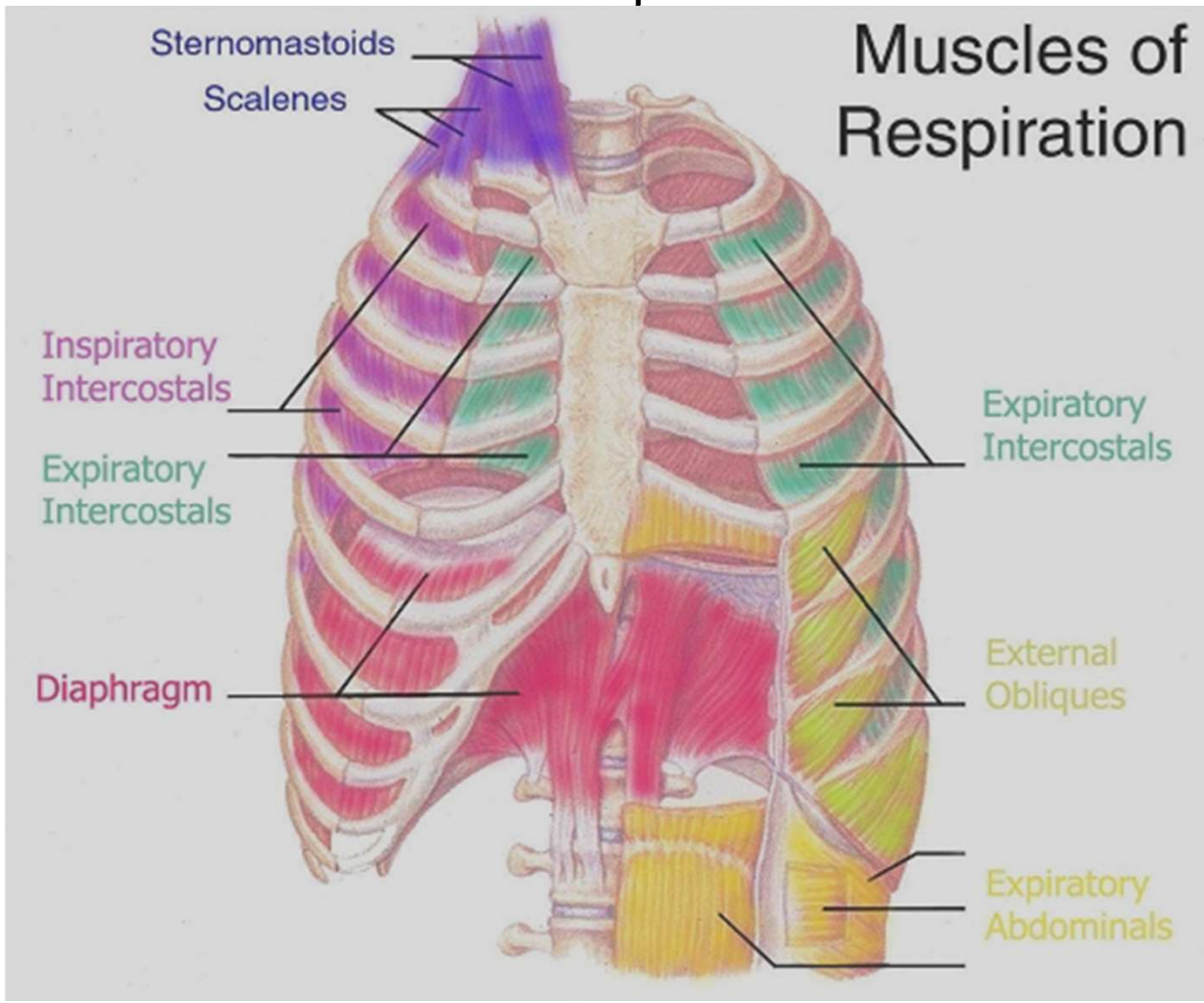
Contraction of external intercostal muscles

- > elevation of ribs & sternum
- > increased front- to-back dimension of thoracic cavity
- > lowers air pressure in lungs
- > air moves into lungs

Contraction of diaphragm

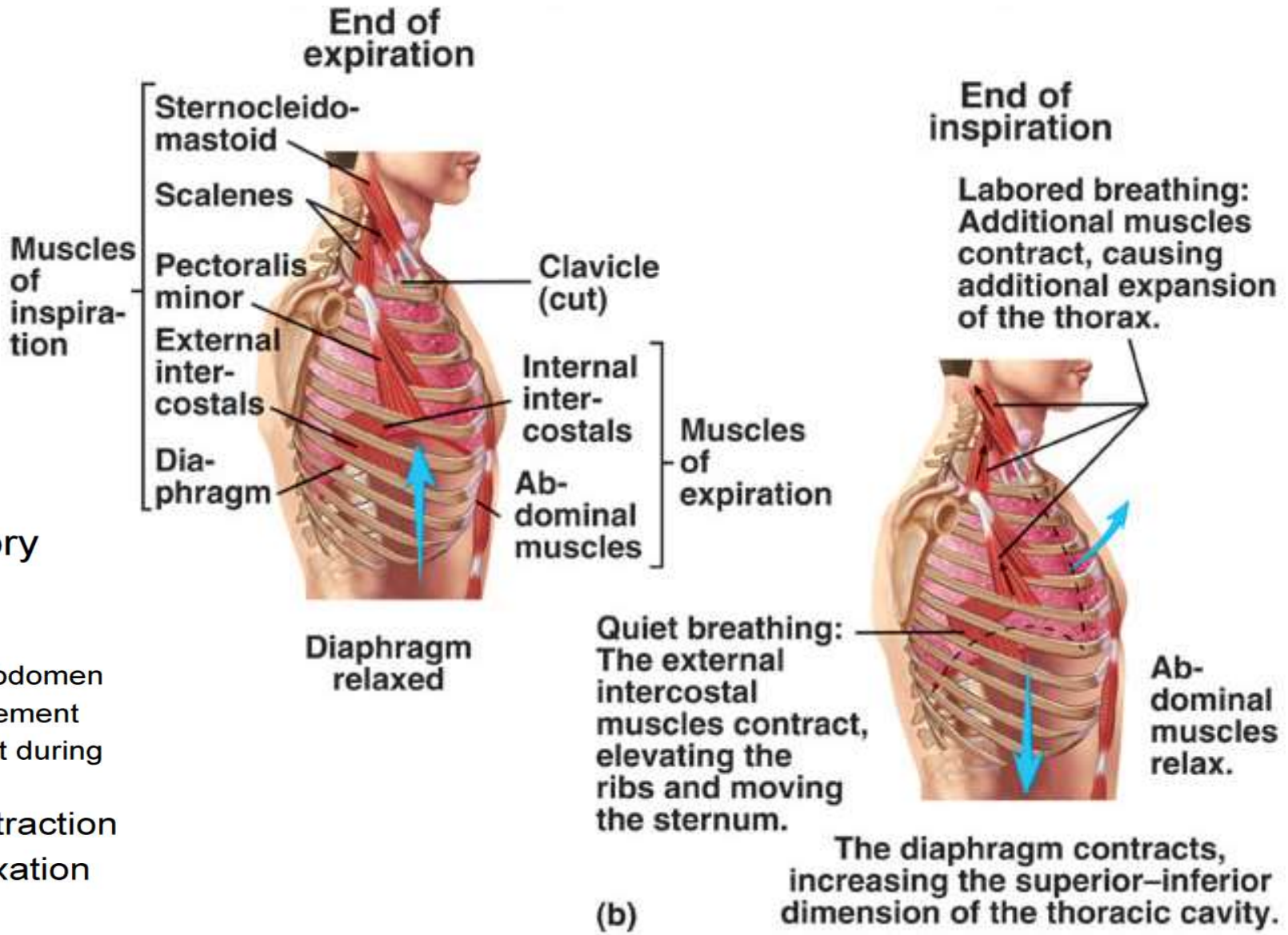
> diaphragm moves downward

- ➢ increases vertical dimension of thoracic cavity
- ➢ > lowers air pressure in lungs
- ➢ > air moves into lungs:



Thoracic Walls Muscles of Respiration

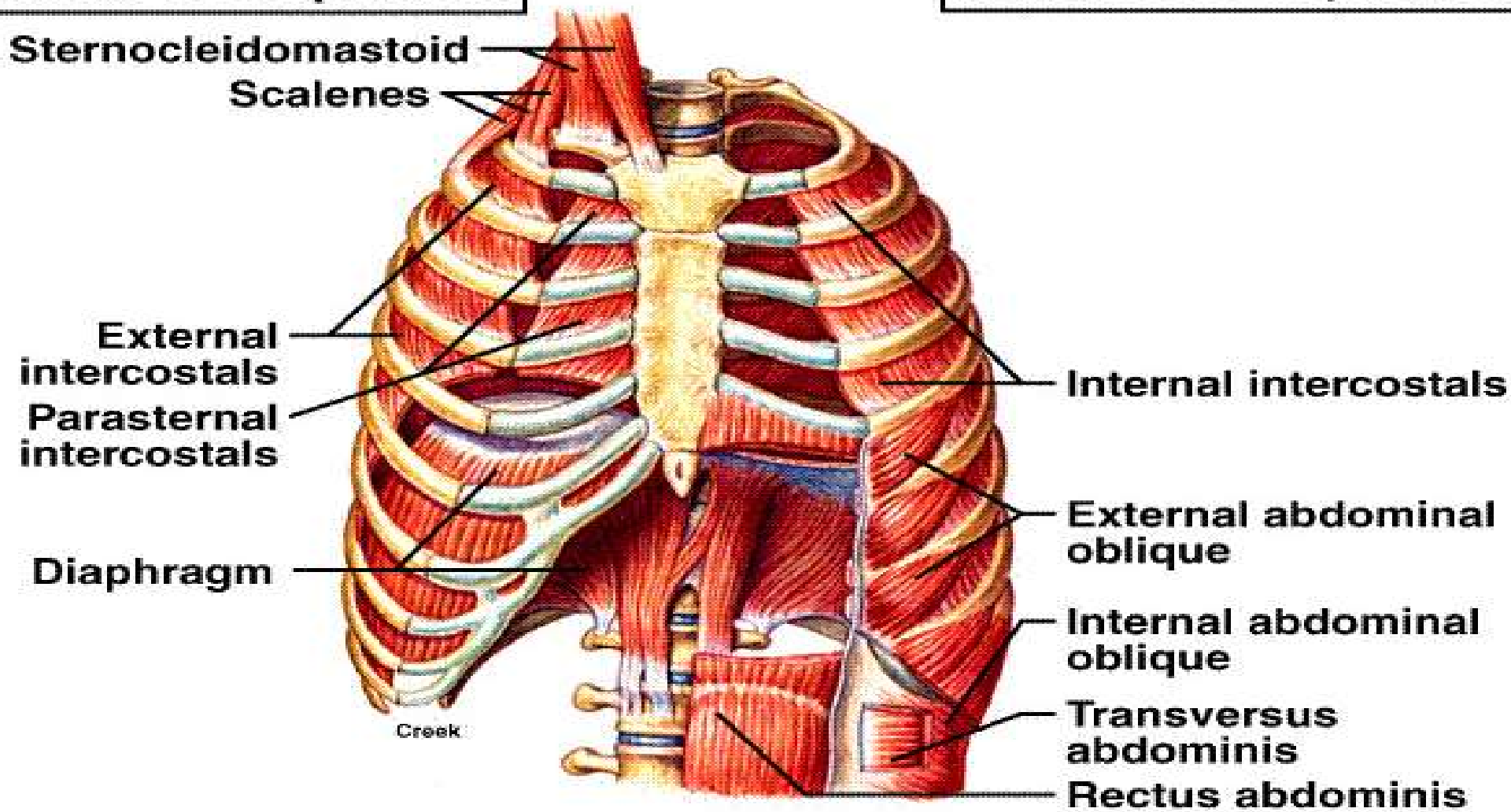
- Primary Ventilatory Muscles
  - Diaphragm
    - Divides Chest/Abdomen
    - 75% of gas movement
    - 1.5cm movement during quiet breathing
  - Inspiration – contraction
  - Expiration – relaxation
    - Elastic Recoil



Muscles Involved in Breathing

Muscles of inspiration

Muscles of expiration



Muscles of respiration

Quiet breathing:

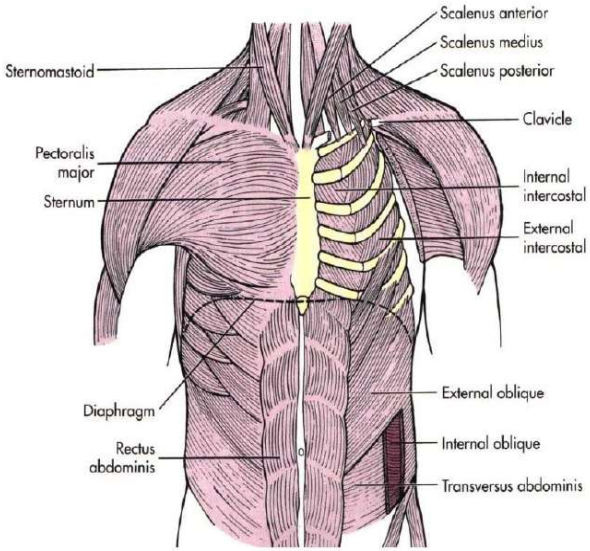
Inspiration––diaphragm.

Expiration––passive.

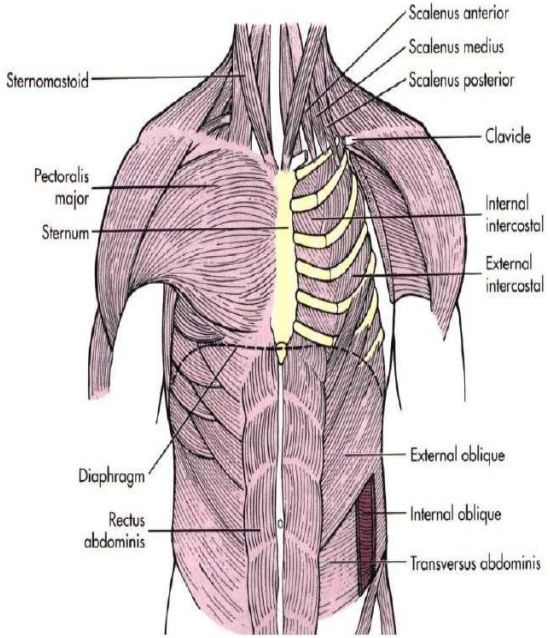
Exercise:

Inspiration––external intercostals, scalene muscles, sternomastoids.

Expiration––rectus abdominis, internal and external obliques, transversus abdominis, internal intercostals.



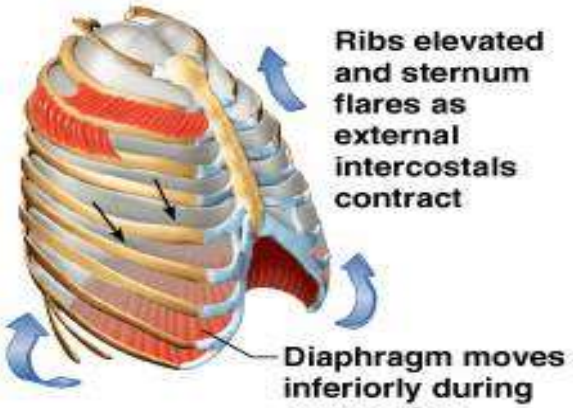
- Scalene Muscles
  - Neck muscles
  - Attach to 1st /2nd rib
  - Assist ventilatory demands
- Sternomastoid
  - Manubrium / clavicle
- Pectoralis Major
  - Clavicle / sternum

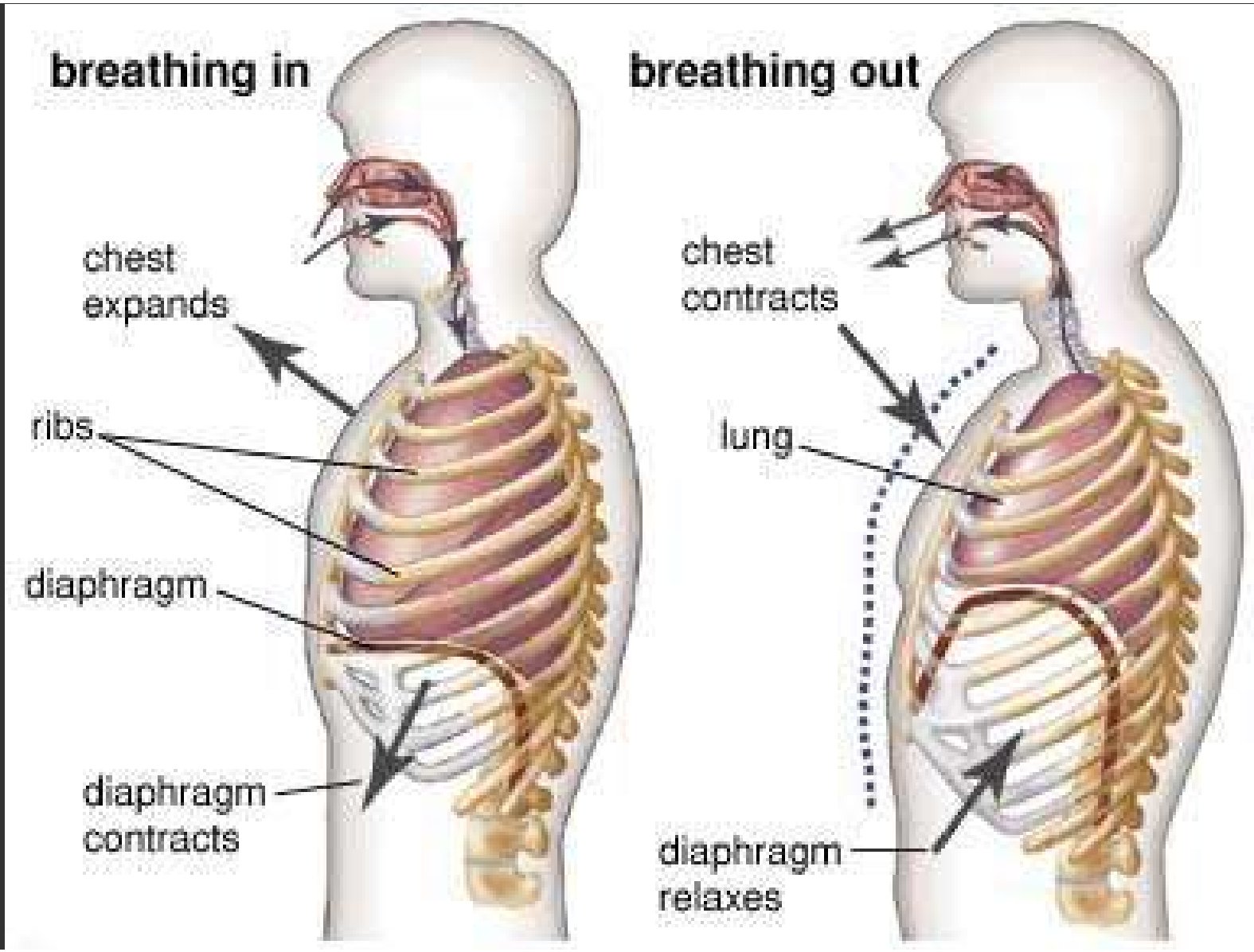


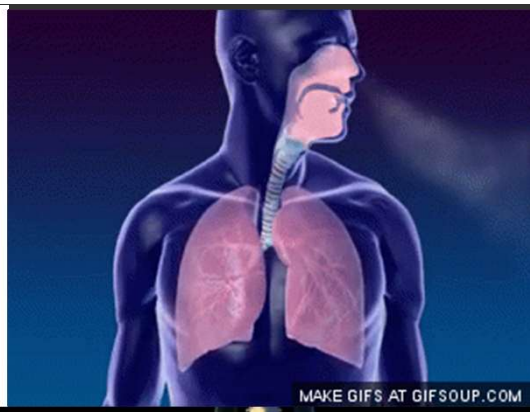
- Abdominal Muscles
  - External oblique
  - Internal oblique
  - Transverse abdominus
  - Rectus abdominus
- Inactive during quiet breathing
- Active > 40L/min

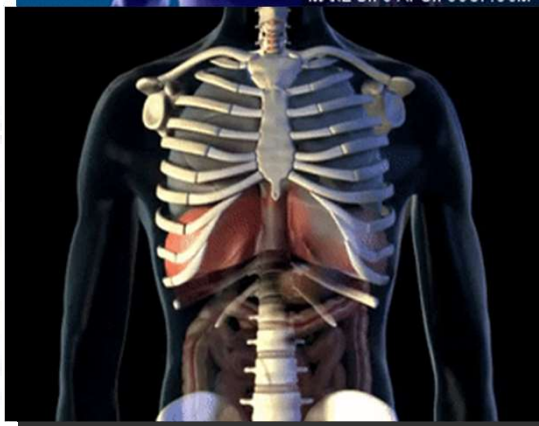
Expiration

① Inspiratory muscles relax (diaphragm rises; rib cage descends due to recoil of costal cartilages)
↓
② Thoracic cavity volume decreases
↓
③ Elastic lungs recoil passively; intrapulmonary volume decreases
↓
④ Intrapulmonary pressure rises (to +1 mm Hg)
↓
⑤ Air (gases) flows out of lungs down its pressure gradient until intrapulmonary pressure is 0

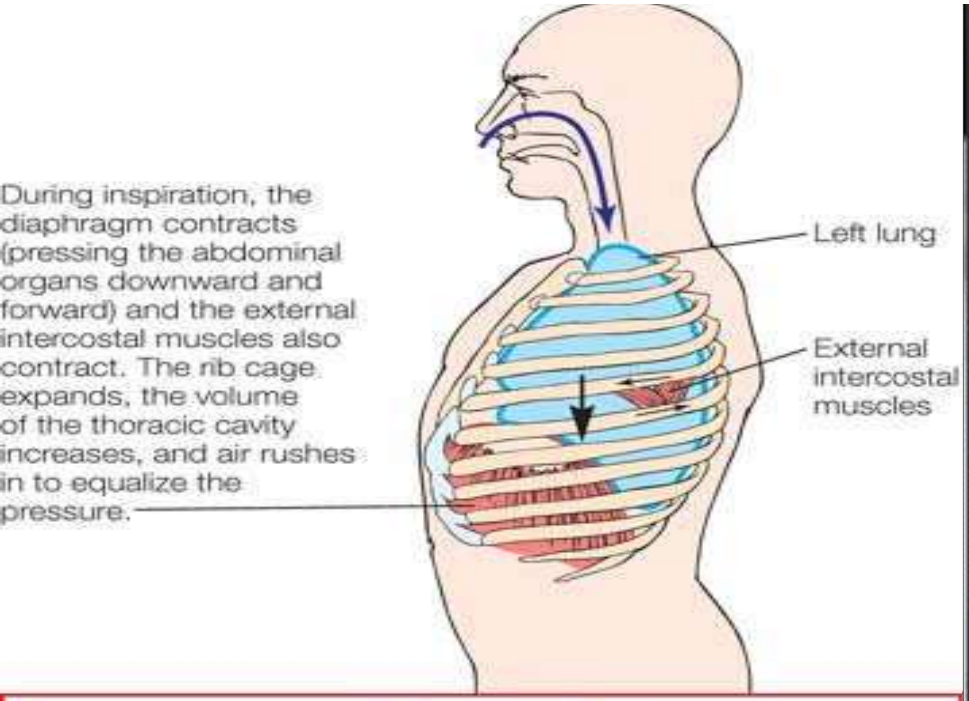
	Sequence of events	Changes in anterior-posterior and superior-inferior dimensions	Changes in lateral dimensions
Inspiration	① Inspiratory muscles contract (diaphragm descends; rib cage rises) ↓ ② Thoracic cavity volume increases ↓ ③ Lungs stretched; intrapulmonary volume increases ↓ ④ Intrapulmonary pressure drops (to −1 mm Hg) ↓ ⑤ Air (gases) flows into lungs down its pressure gradient until intrapulmonary pressure is 0 (equal to atmospheric pressure)		

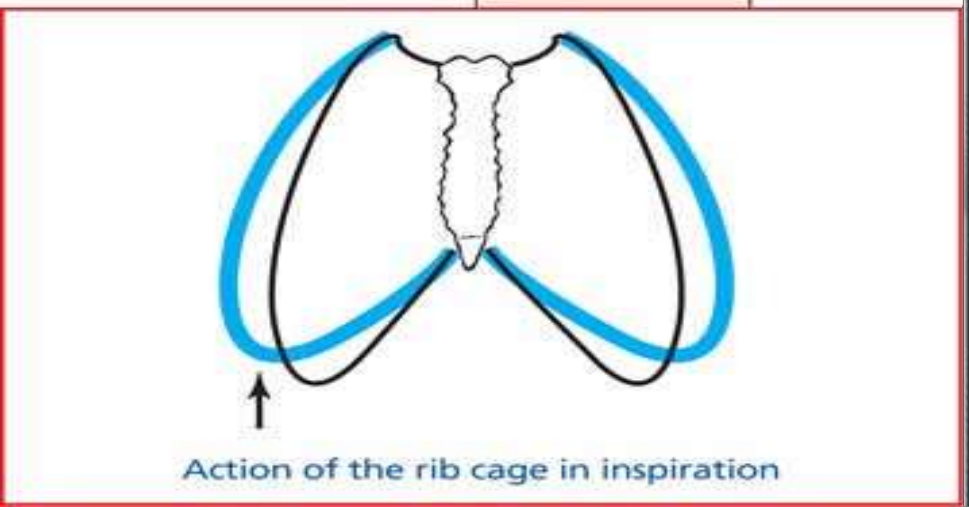

breathing in
chest expands
ribs
diaphragm
diaphragm contracts
breathing out
chest contracts
lung
diaphragm relaxes


MAKE GIFS AT GIFSOUP.COM

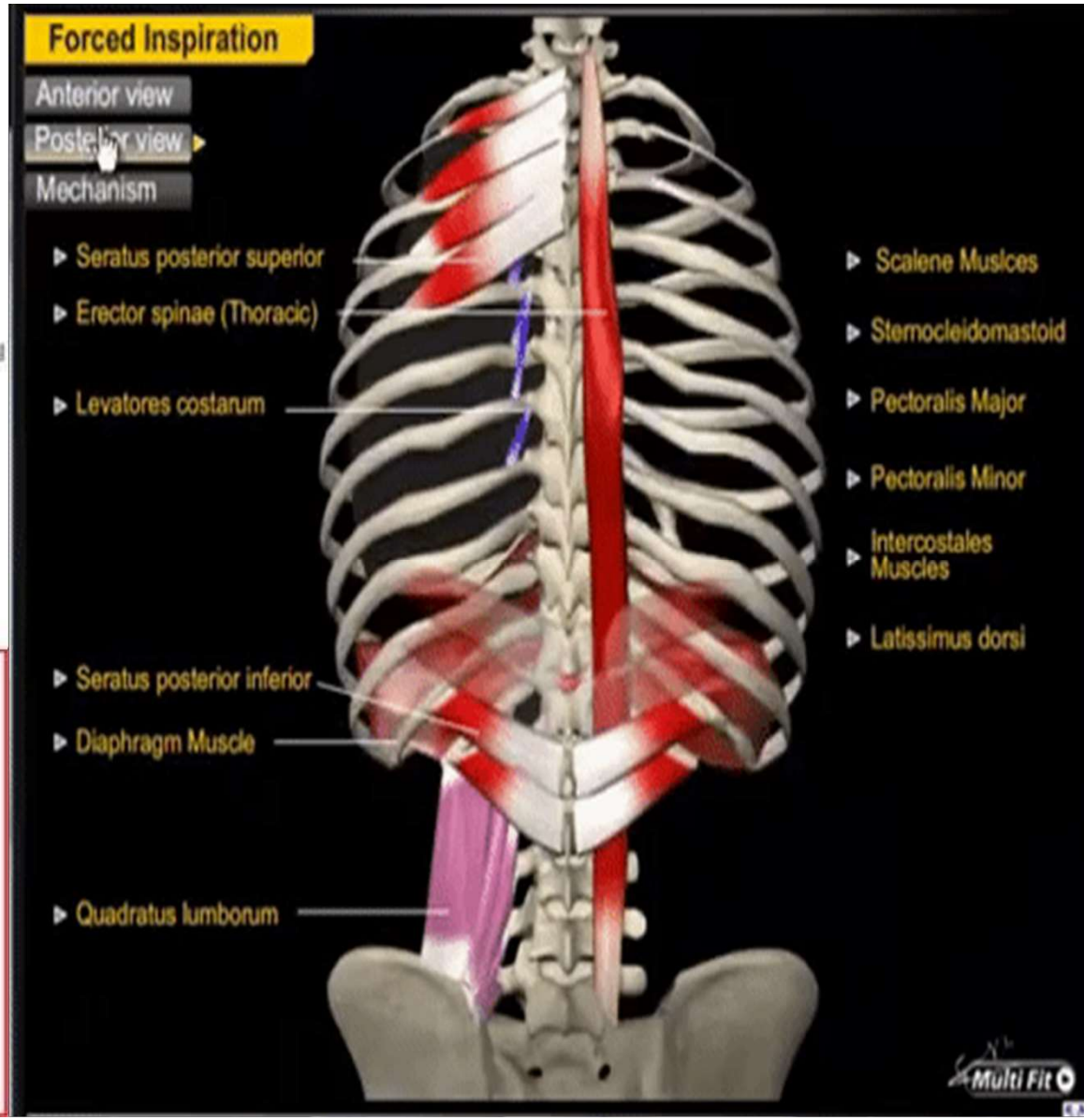


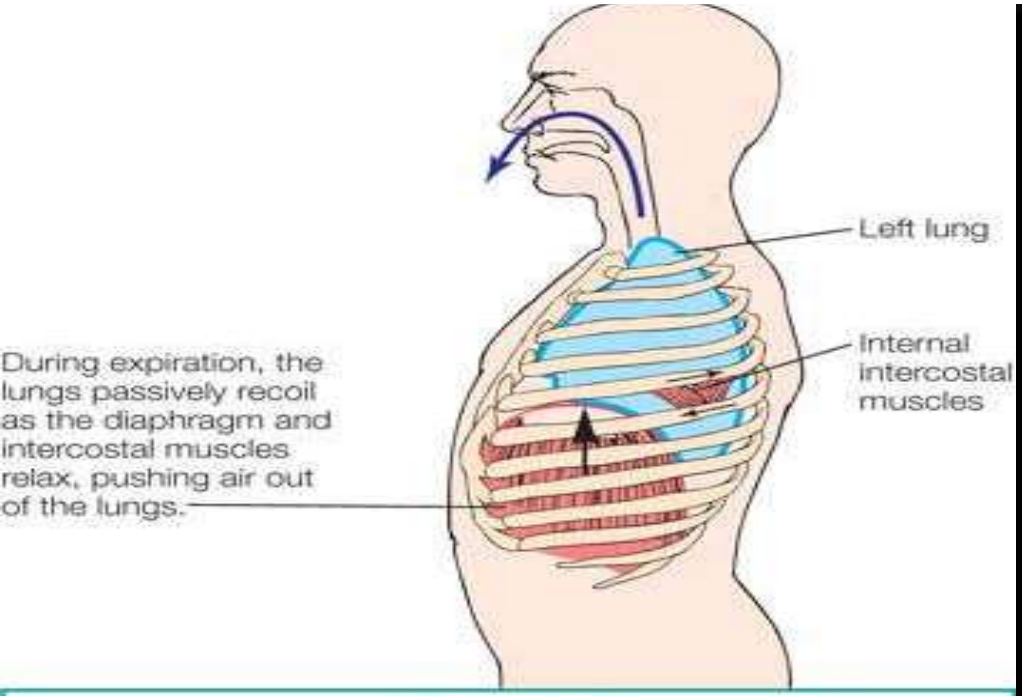
During inspiration, the diaphragm contracts (pressing the abdominal organs downward and forward) and the external intercostal muscles also contract. The rib cage expands, the volume of the thoracic cavity increases, and air rushes in to equalize the pressure.

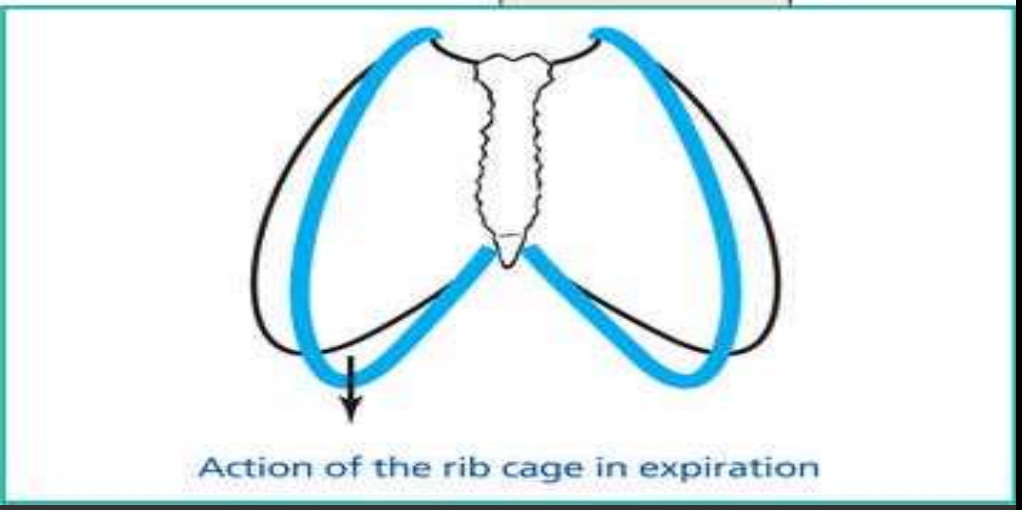


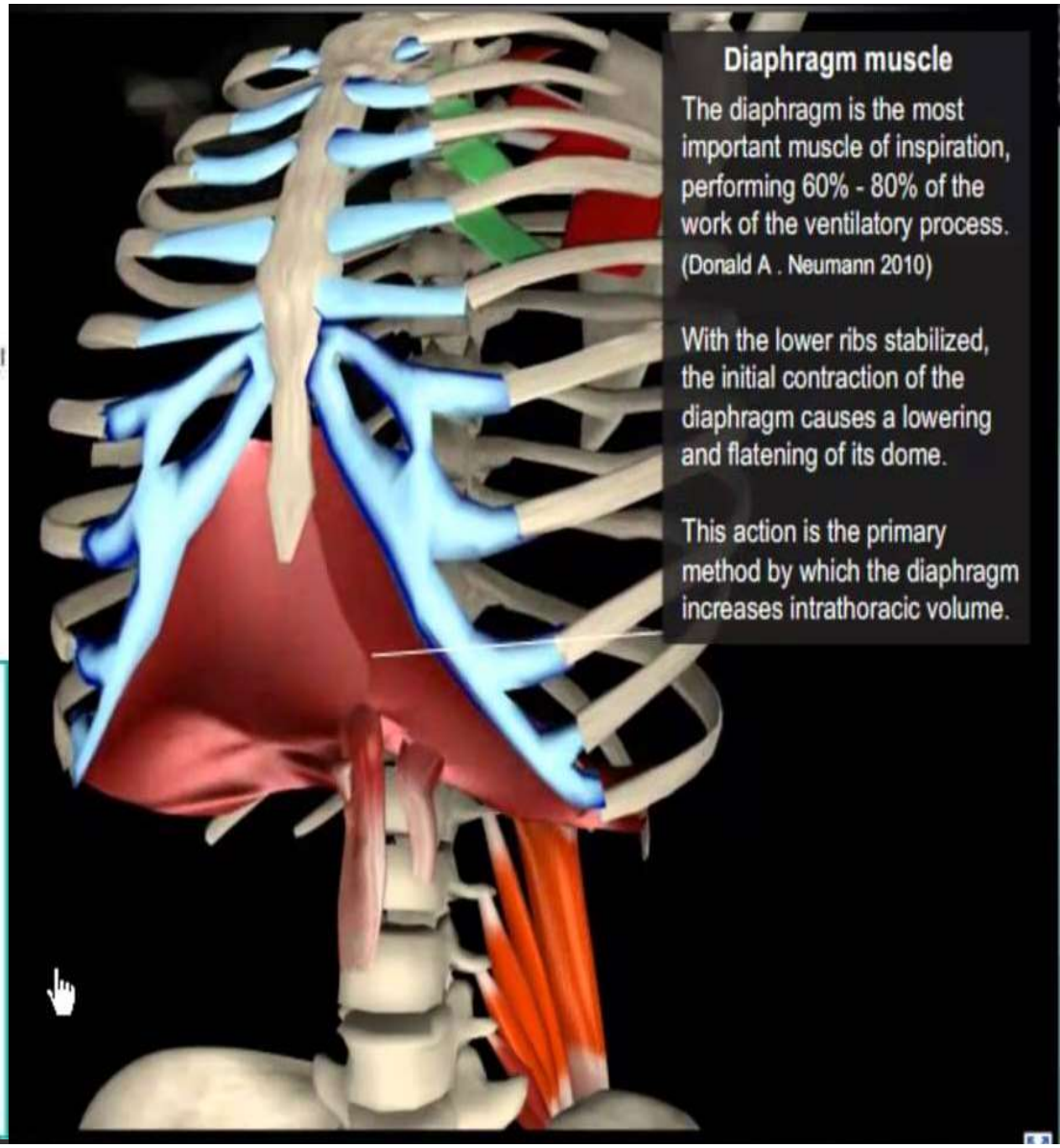


Action of the rib cage in inspiration



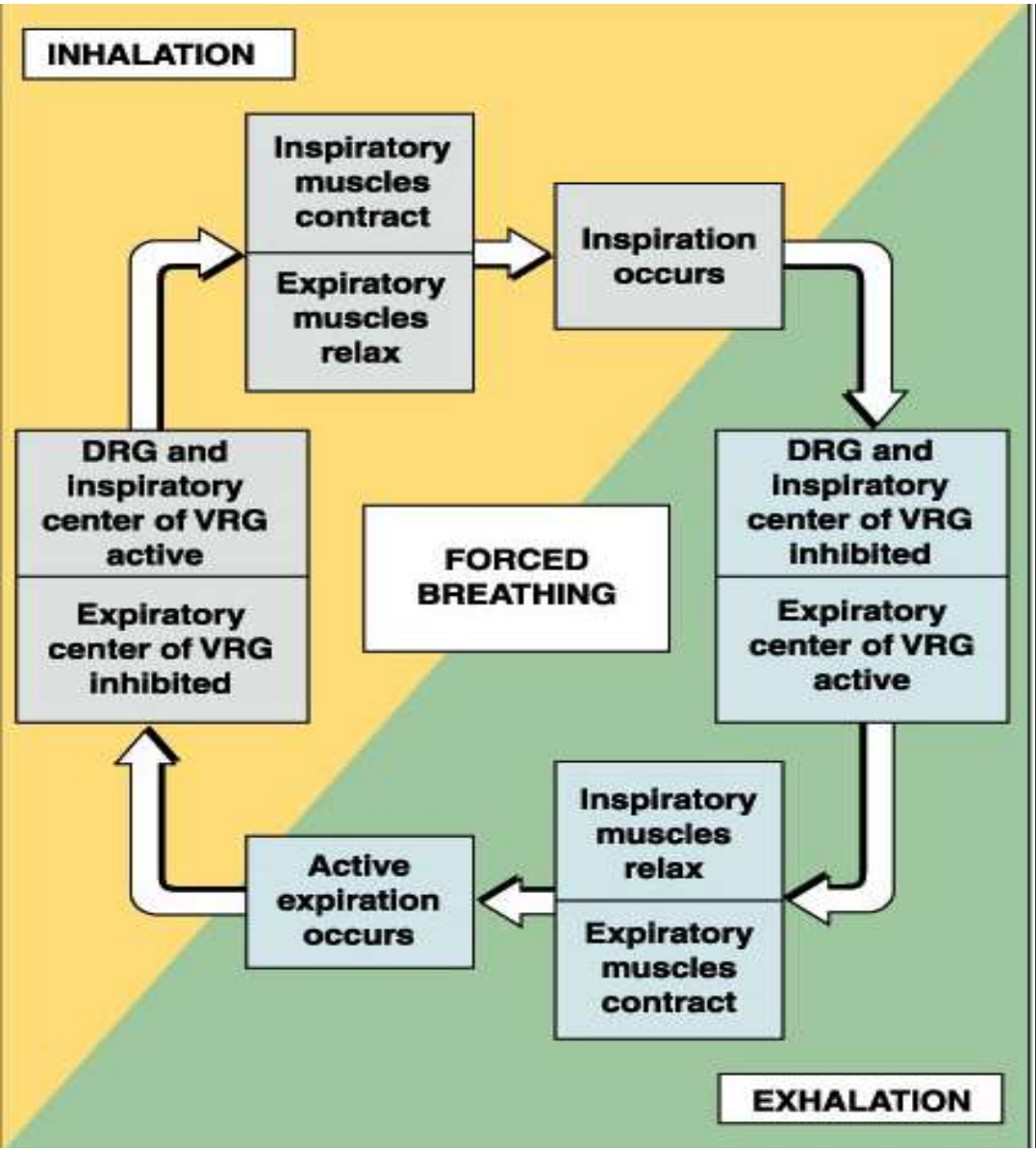

Left lung
Internal intercostal muscles
During expiration, the lungs passively recoil as the diaphragm and intercostal muscles relax, pushing air out of the lungs.

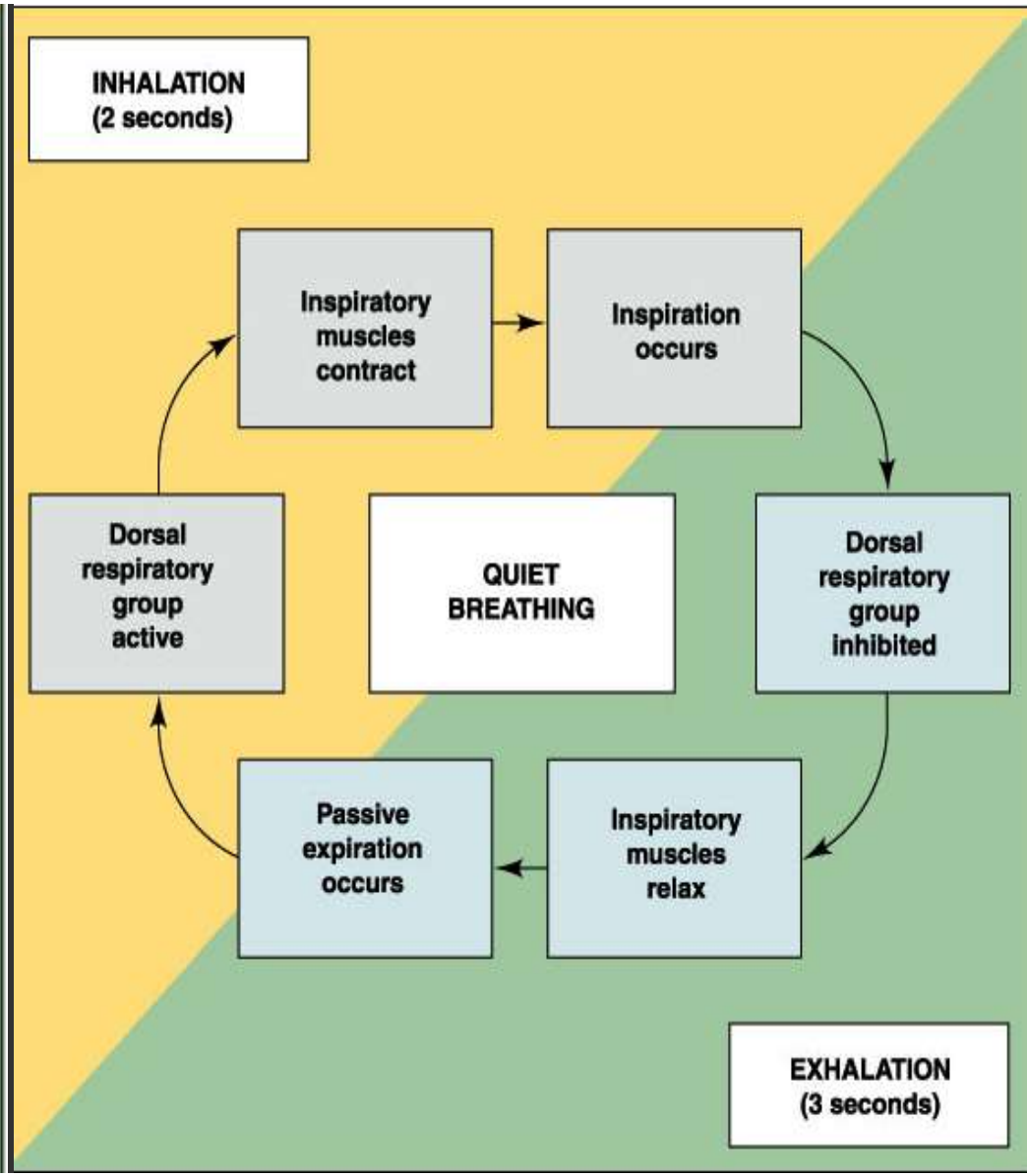

Action of the rib cage in expiration


Diaphragm muscle
The diaphragm is the most important muscle of inspiration, performing 60% - 80% of the work of the ventilatory process.
(Donald A . Neumann 2010)
With the lower ribs stabilized, the initial contraction of the diaphragm causes a lowering and flatening of its dome.
This action is the primary method by which the diaphragm increases intrathoracic volume.

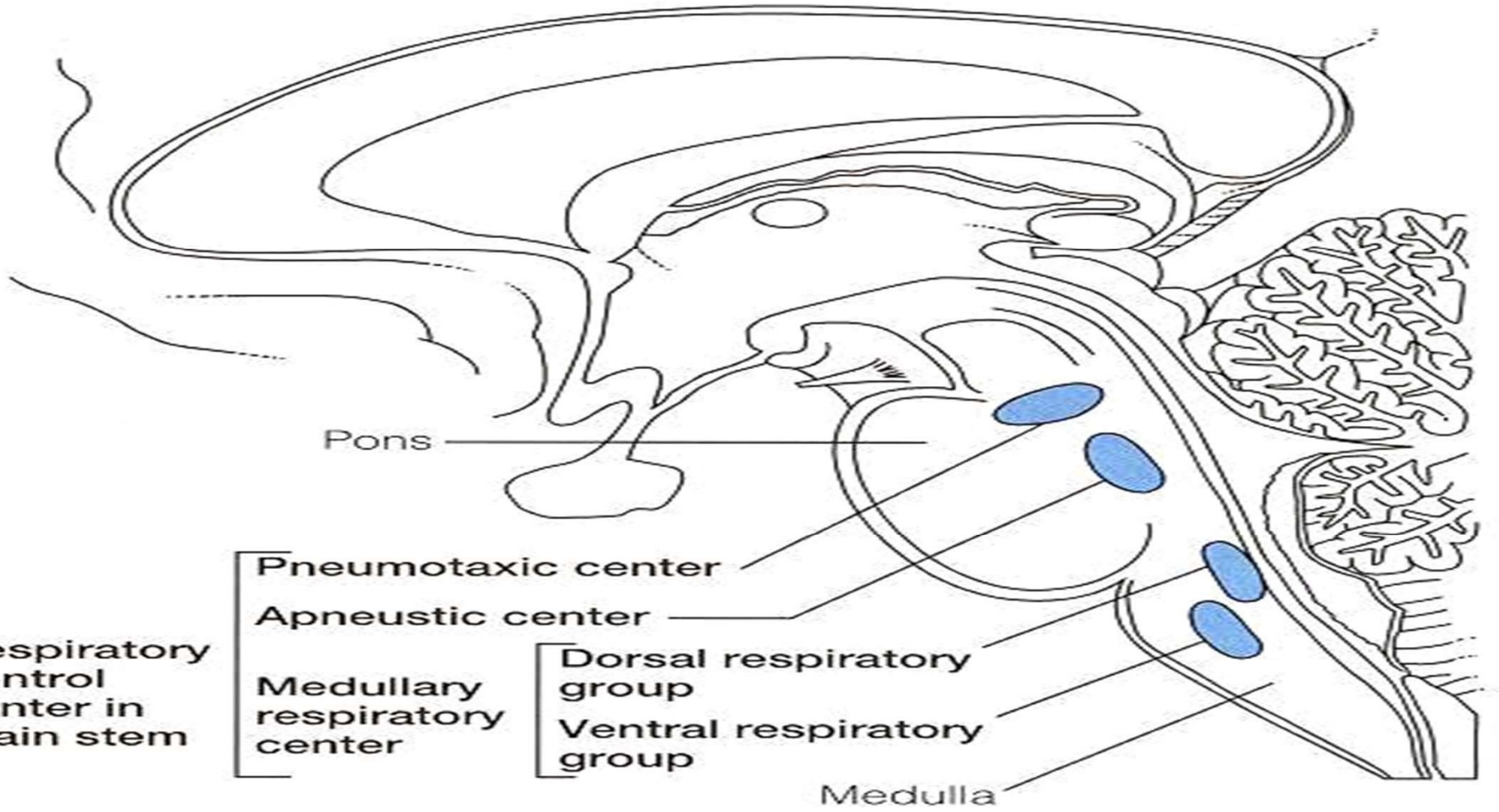
Deep Forceful Breathing

- ***Deep Inhalation***
  - During deep forceful inhalation accessory muscles of inhalation participate to increase size of thoracic cavity
    - Sternocleidomastoid – elevate sternum
    - Scalenes – elevate first two ribs
    - Pectoralis minor – elevate 3rd–5th ribs
- ***Deep Exhalation***
  - Exhalation during forceful breathing is active process
    - **Muscles of exhalation increase pressure in abdomen and thorax**
      - Abdominals
      - Internal intercostals

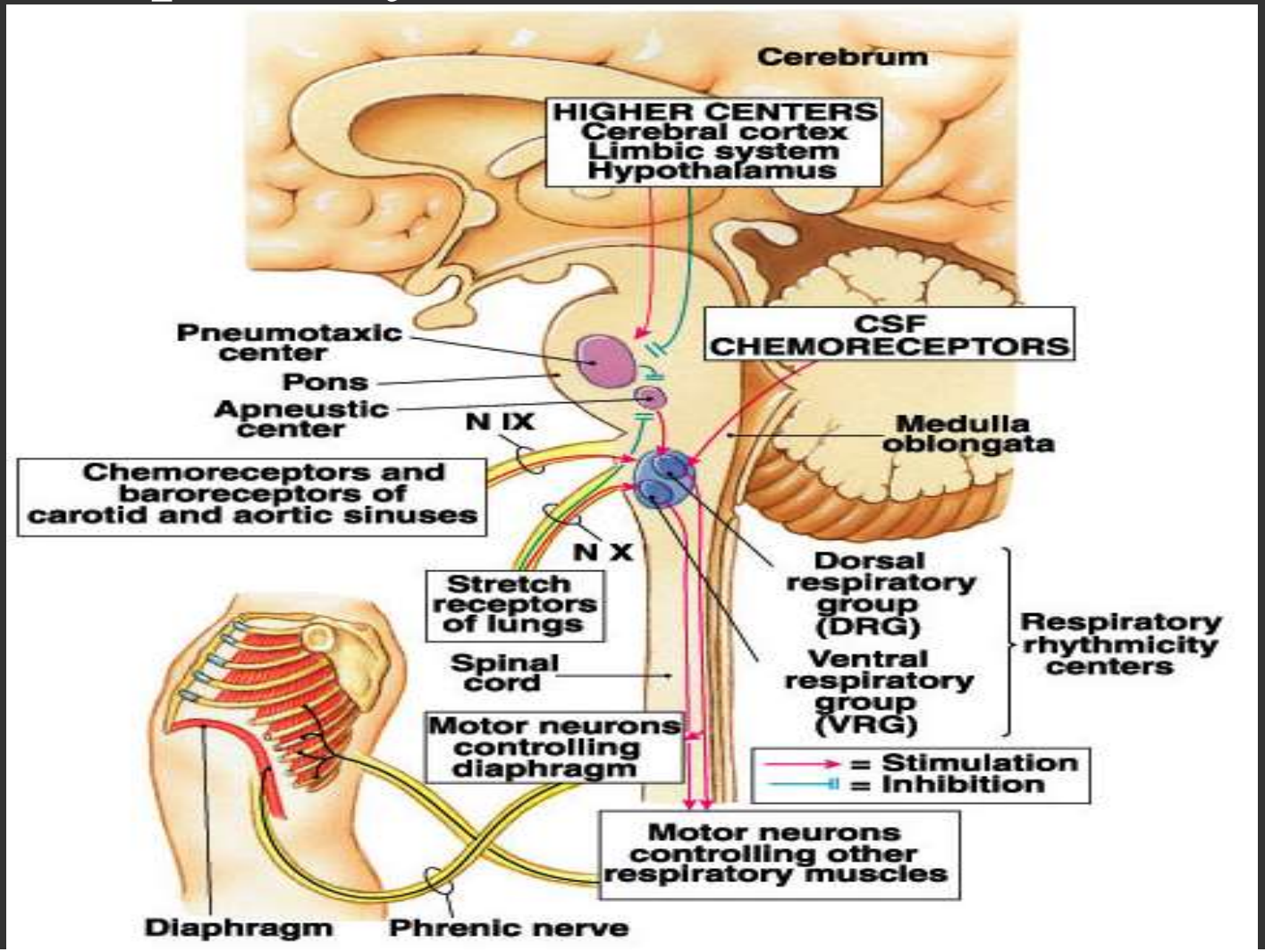

INHALATION
Inspiratory muscles contract
Expiratory muscles relax
Inspiration occurs
DRG and inspiratory center of VRG active
Expiratory center of VRG inhibited
FORCED BREATHING
DRG and inspiratory center of VRG inhibited
Expiratory center of VRG active
Inspiratory muscles relax
Expiratory muscles contract
Active expiration occurs
EXHALATION


INHALATION (2 seconds)
Inspiratory muscles contract
Inspiration occurs
Dorsal respiratory group active
QUIET BREATHING
Dorsal respiratory group inhibited
Passive expiration occurs
Inspiratory muscles relax
EXHALATION (3 seconds)

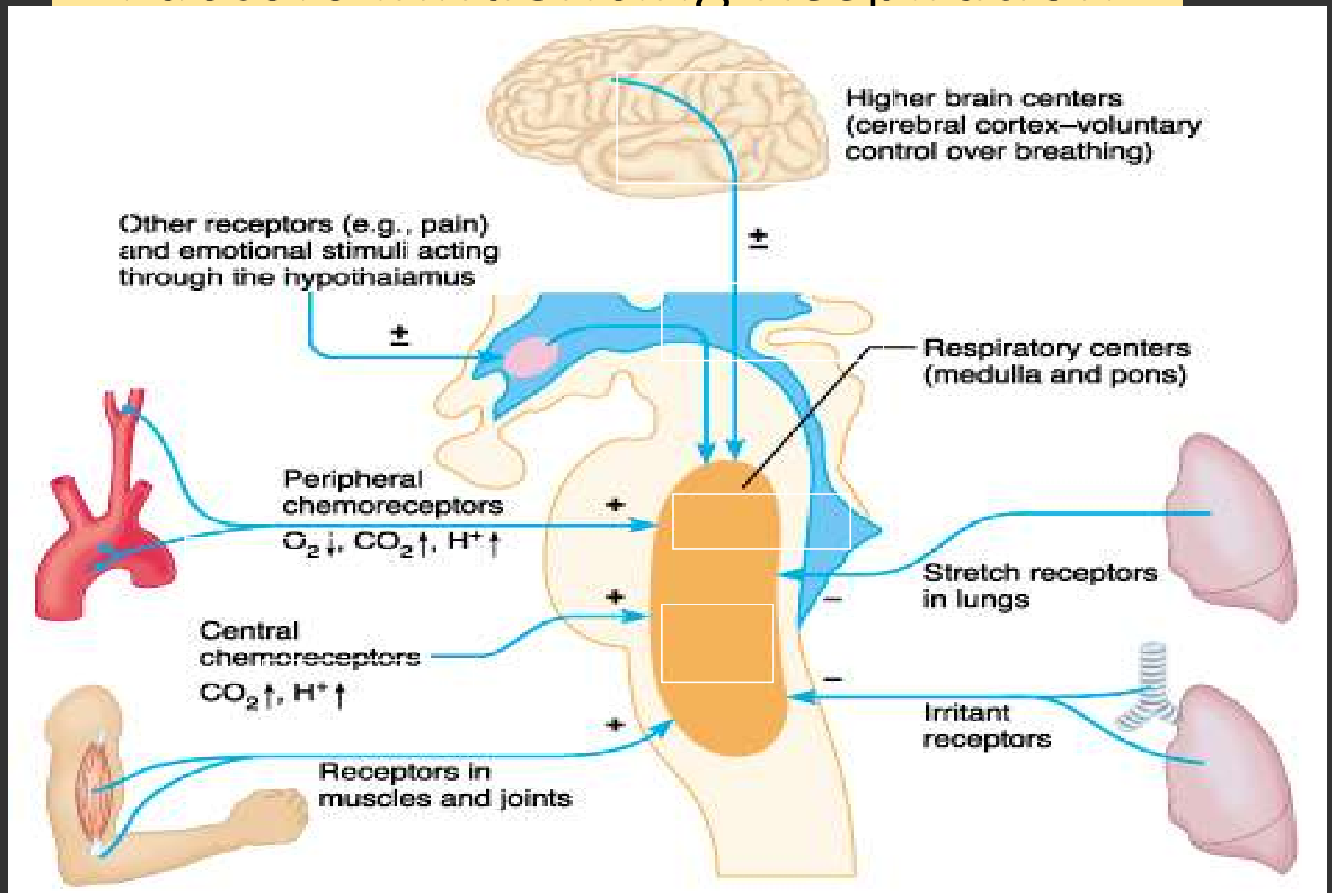
Respiratory Centers



Respiratory Structures in Brainstem



Factors Influencing Respiration



Two Sets of Chemoreceptors Exist

Central Chemoreceptors

Responsive to increased arterial PCO2

Act by way of CSF [H+] .

Peripheral Chemoreceptors

Responsive to decreased arterial PO2

Responsive to increased arterial PCO2

Responsive to increased H+ ion concentration.

Peripheral Chemoreceptors

Carotid bodies

Sensitive to: PaO_2, $PaCO_2$, and pH

Afferents in glossopharyngeal nerve.

Aortic bodies

Sensitive to: PaO_2, $PaCO_2$, but not pH

Afferents in vagus

Significance of Hering-Breuer

Limits the degree of inspiration and prevents overinflation of the lungs

Normal adults. Receptors are not activated at end normal tidal volumes.

- Become Important during exercise when tidal volume is increased.
- Become Important in Chronic obstructive lung diseases when lungs are more distended.

Infants. Probably help terminate normal inspiration.

Hering-Breuer Reflex or Pulmonary Stretch Reflex

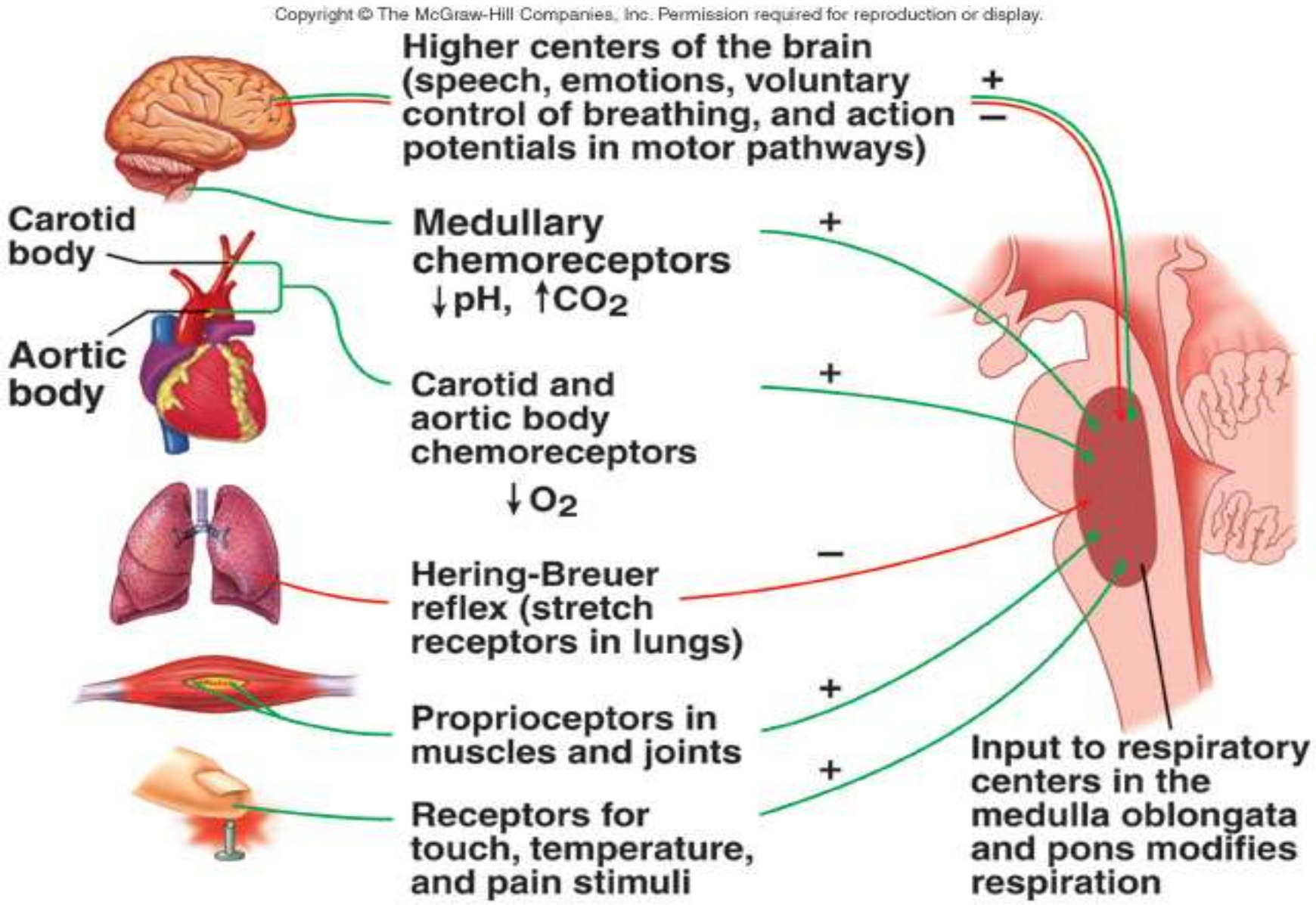
Including pulmonary inflation reflex and pulmonary deflation reflex

Receptor: Slowly adapting stretch receptors (SARs) in bronchial airways.

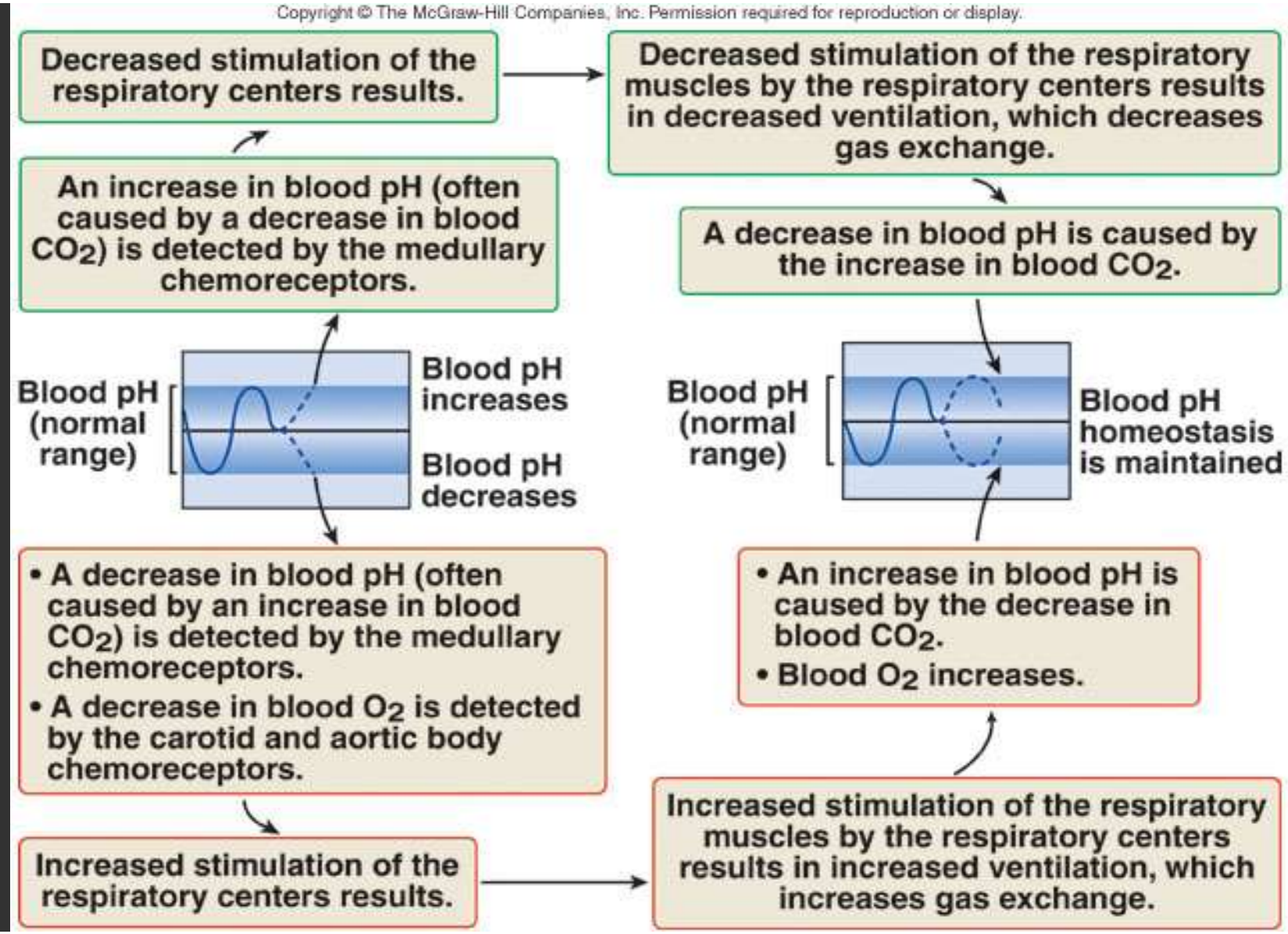
Afferent: vagus nerve

Pulmonary inflation reflex:

- Terminate inspiration.
- By speeding inspiratory termination they increase respiratory frequency.
- **Sustained stimulation of SARs:** causes activation of expiratory neurons



Modifying Respiration



Regulation of Blood pH and Gases

Flow-volume loops

FLOW-VOLUME PARAMETER	Obstructive lung disease	Restrictive lung disease
RV	↑	↓
FRC	↑	↓
TLC	↑	↓
FEV_1	↓↓	↓
FVC	↓	↓
FEV_1/FVC	↓ FEV_1 decreased more than FVC	Normal or ↑ FEV_1 decreased proportionately to FVC