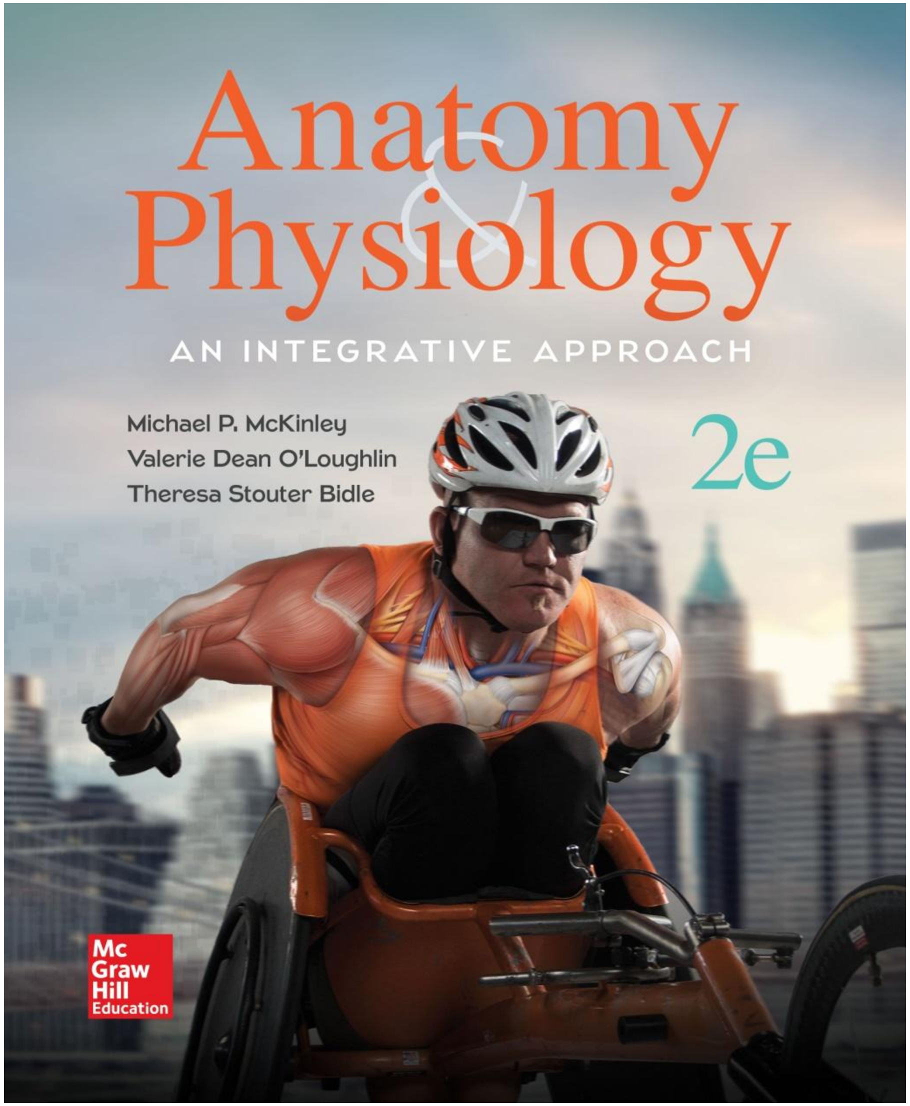
Chapter 23 - Part 2
Lecture Outline

See separate PowerPoint slides for all figures and tables pre-inserted into PowerPoint without notes.



Processes of Respiration

- Respiration (exchange of gases between atmosphere and body's cells) involves four processes
  - **Pulmonary ventilation:** movement of gases between atmosphere and alveoli
  - **Alveolar gas exchange** (*external respiration*): exchange of gases between alveoli and blood
  - **Gas transport:** transport of gases in blood between lungs and systemic cells
  - **Systemic gas exchange** (*internal respiration*): exchange of respiratory gases between the blood and the systemic cells

Processes of Respiration

- Net movement of respiratory gases
  1) Air containing O_2 is inhaled into alveoli during inspiration
  2) O_2 diffuses from alveoli into pulmonary capillaries
  3) Blood from lungs transports O_2 to systemic cells
  4) O_2 diffuses from systemic capillaries into systemic cells
  5) CO_2 diffuses from systemic cells into systemic capillaries
  6) CO_2 is transported in blood from systemic cells to lungs
  1) CO_2 diffuses from pulmonary capillaries into alveoli
  5) Air containing CO_2 is exhaled from alveoli into the atmosphere

Overview of Respiration

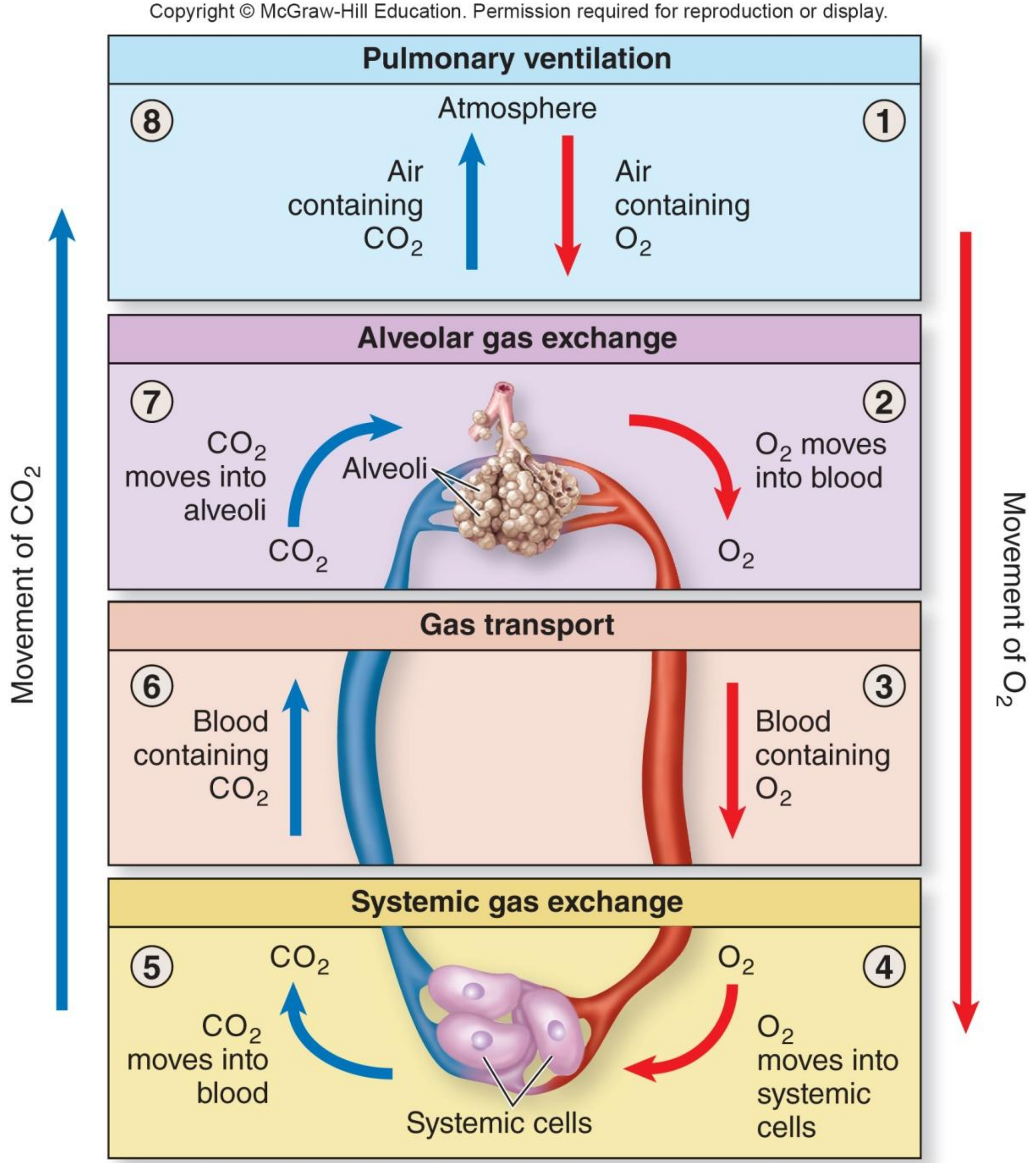


Figure 23.18

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 systemic cells of the body • Oxygen is transported from lungs to systemic 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 systemic cells (step 4) • Carbon dioxide diffuses from systemic cells into blood (step 5)	Cardiovascular

23.5 Respiration: Pulmonary Ventilation

Learning Objectives:

1. Give an overview of the process of pulmonary ventilation.
2. Explain how pressure gradients are established and result in pulmonary ventilation.
3. State the relationship between pressure and volume as described by Boyle's law.
4. Distinguish between quiet and forced breathing.
5. Describe the anatomic structures involved in regulating breathing.

23.5 Respiration: Pulmonary Ventilation (*continued*)

Learning Objectives:

6. Explain the physiologic events associated with controlling quiet breathing.
7. Explain the different reflexes that alter breathing rate and depth.
8. Distinguish between nervous system control of structures of the respiratory system and nervous system control of structures involved in breathing.
9. Define airflow.
10. Explain how pressure gradients and resistance determine airflow.

23.5 Respiration: Pulmonary Ventilation (*continued*)

Learning Objectives:

11. Distinguish between pulmonary ventilation and alveolar ventilation, and discuss the significance of each.
12. Explain the relationship between anatomic dead space and physiologic dead space.
13. Define the four different respiratory volume measurements.
14. Explain the four respiratory capacities that are calculated from the volume measurements.
15. Give the meaning of forced expiratory volume (FEV) and maximum voluntary ventilation (MVV).

23.5a Introduction to Pulmonary Ventilation

- **Pulmonary ventilation** (*breathing*): air movement
  - Consists of two cyclic phases
    - **Inspiration** brings air into the lungs (*inhalation)*
    - **Expiration** forces air out of the lungs (*exhalation*)
  - **Quiet,** rhythmic breathing occurs at rest
  - **Forced,** vigorous breathing accompanies exercise
  - Autonomic nuclei in brainstem regulate breathing activity
  - Skeletal muscles contract and relax changing thorax volume
  - Volume changes result in changes in pressure gradient between lungs and atmosphere
  - Air moves down its pressure gradient
    - Air enters lung during inspiration; exits during expiration

23.5b Mechanics of Breathing

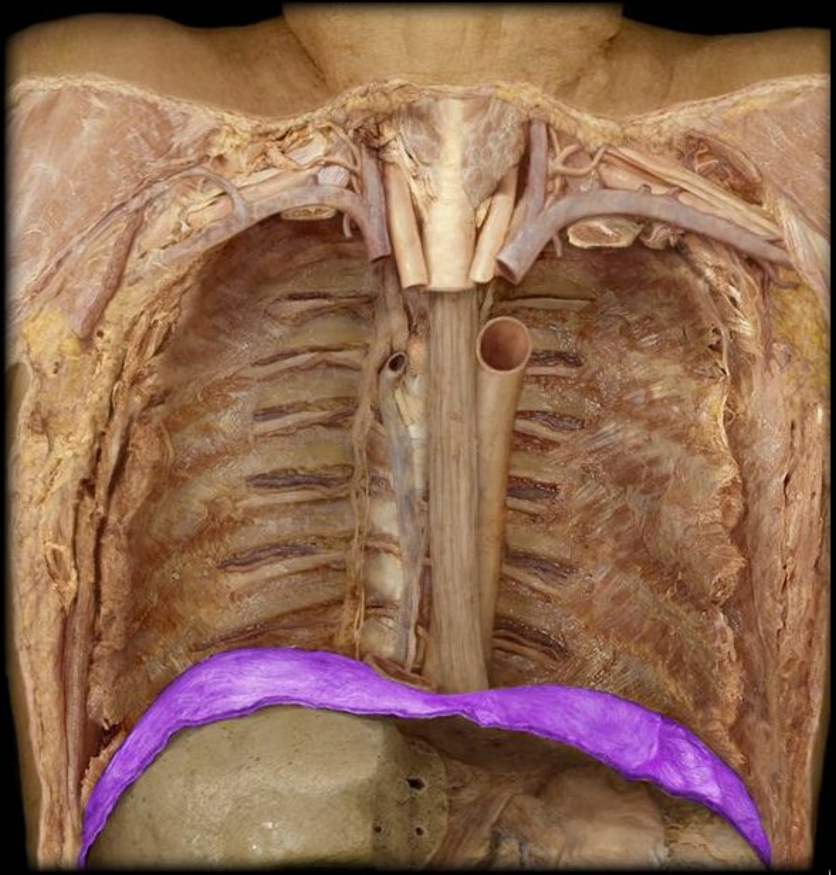
- Involve several integrated aspects
  - Specific actions of skeletal muscles of breathing
  - Dimensional changes within the thoracic cavity
  - Pressure changes resulting from volume changes
  - Pressure gradients
  - Volumes and pressures associated with breathing

23.5b Mechanics of Breathing

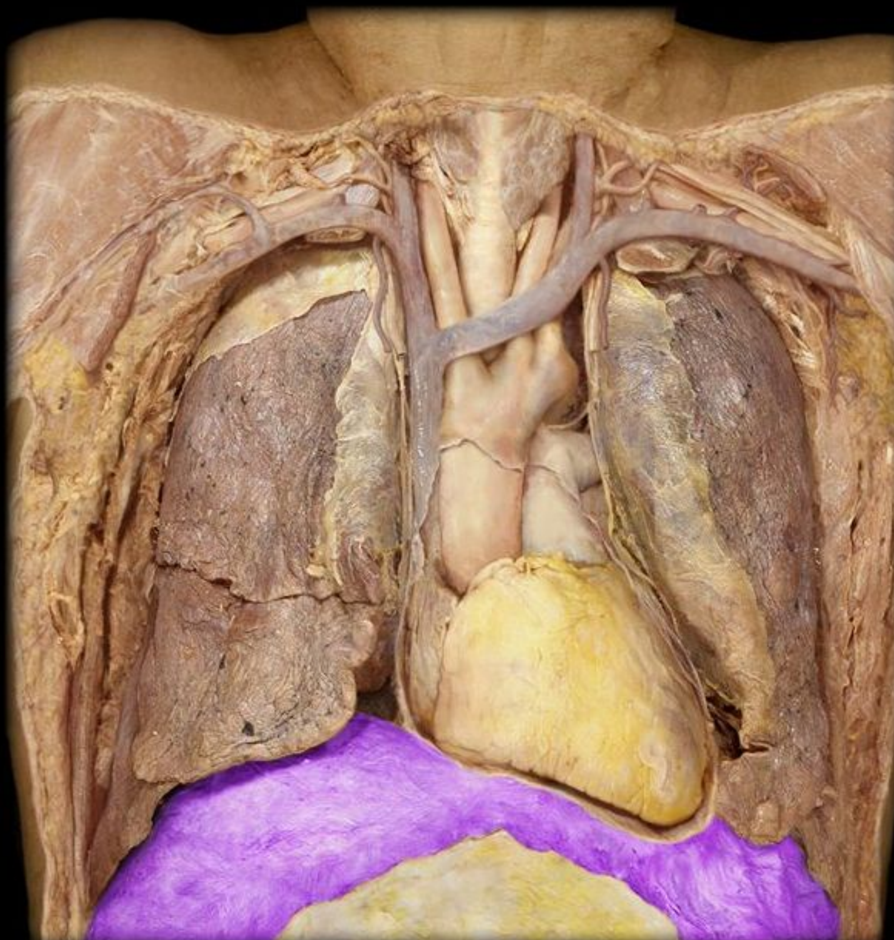
Skeletal muscles of breathing

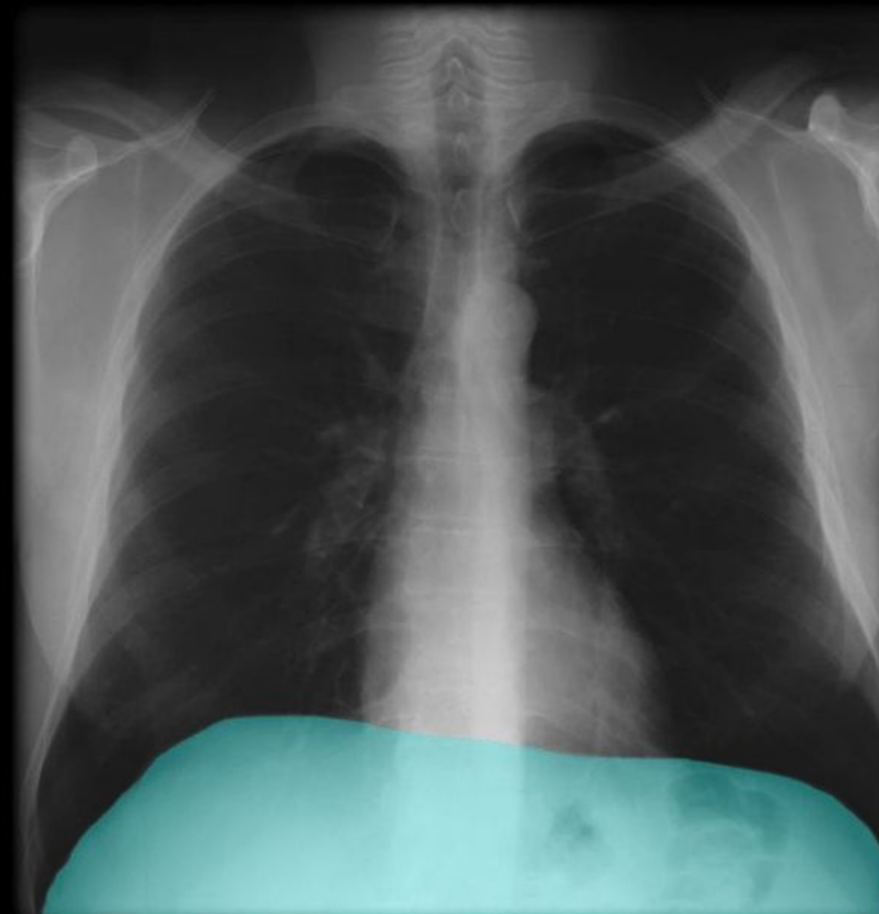
- **Muscles of quiet breathing**
  - **Diaphragm** and **external intercostals** contract for inspiration
    - Diaphragm flattens when it contracts; external intercostals elevate ribs
    - These muscles relax for expiration
- **Muscles of forced inspiration**
  - **Sternocleidomastoid, scalenes, pectoralis minor,** and **serratus posterior superior,** contract for deep inspiration
    - All are located superiorly in thorax
    - Move rib cage superiorly, laterally, and anteriorly, increasing volume
  - **Erector spinae** located along length of vertebral column
    - Contracts to help lift rib cage

Diaphragm

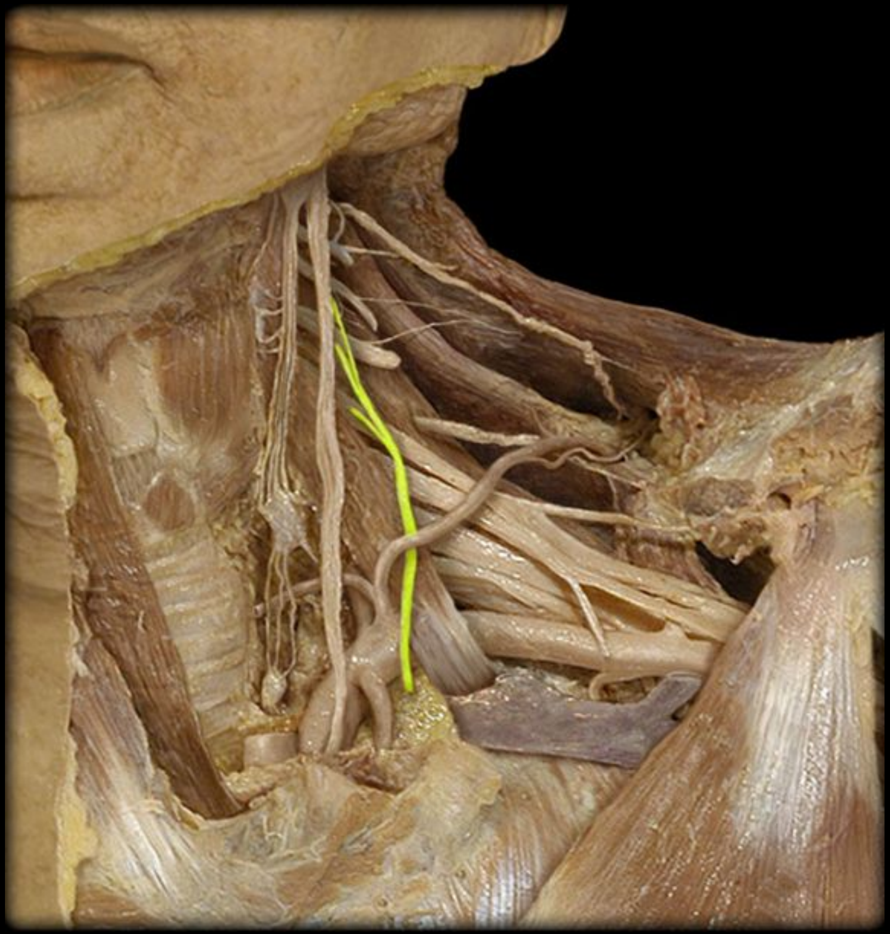


Diaphragm

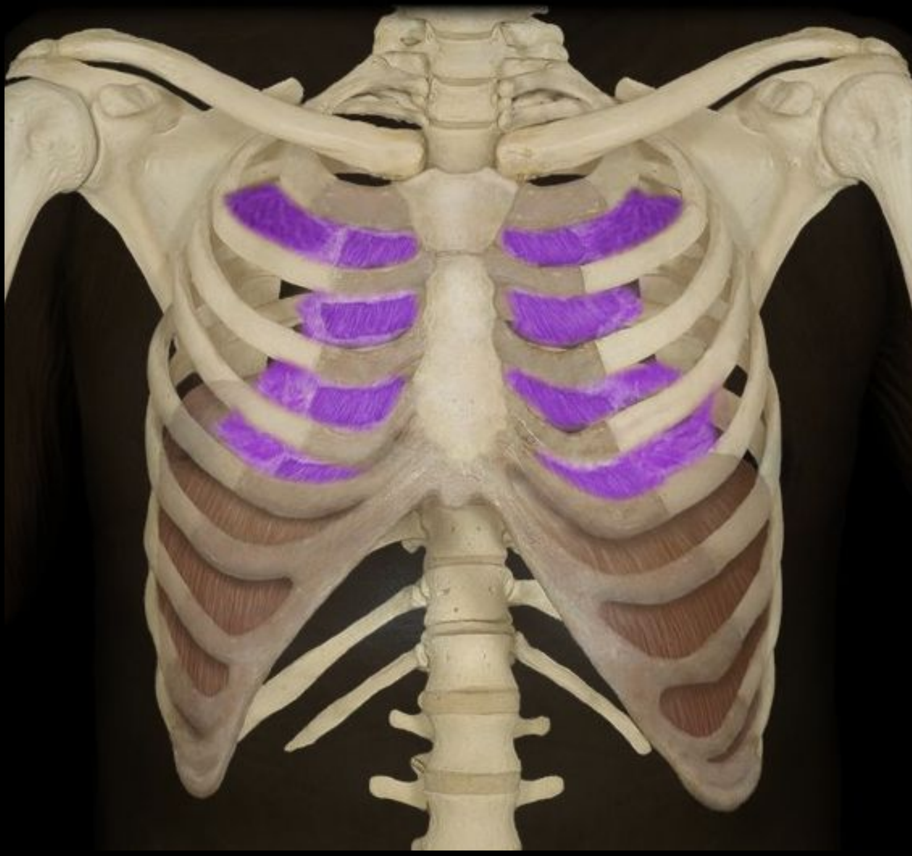




Phrenic Nerve—Diaphragm



External Intercostals



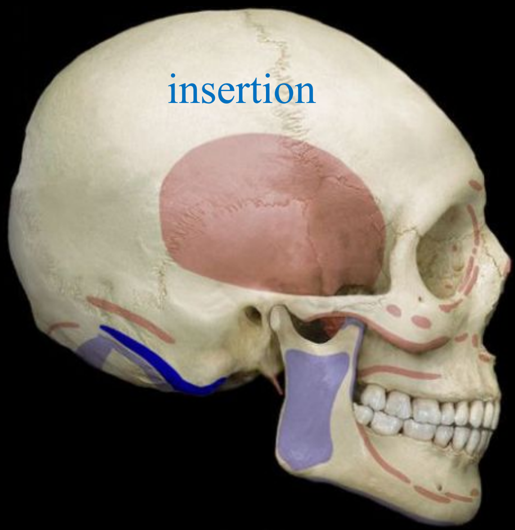
External Intercostals

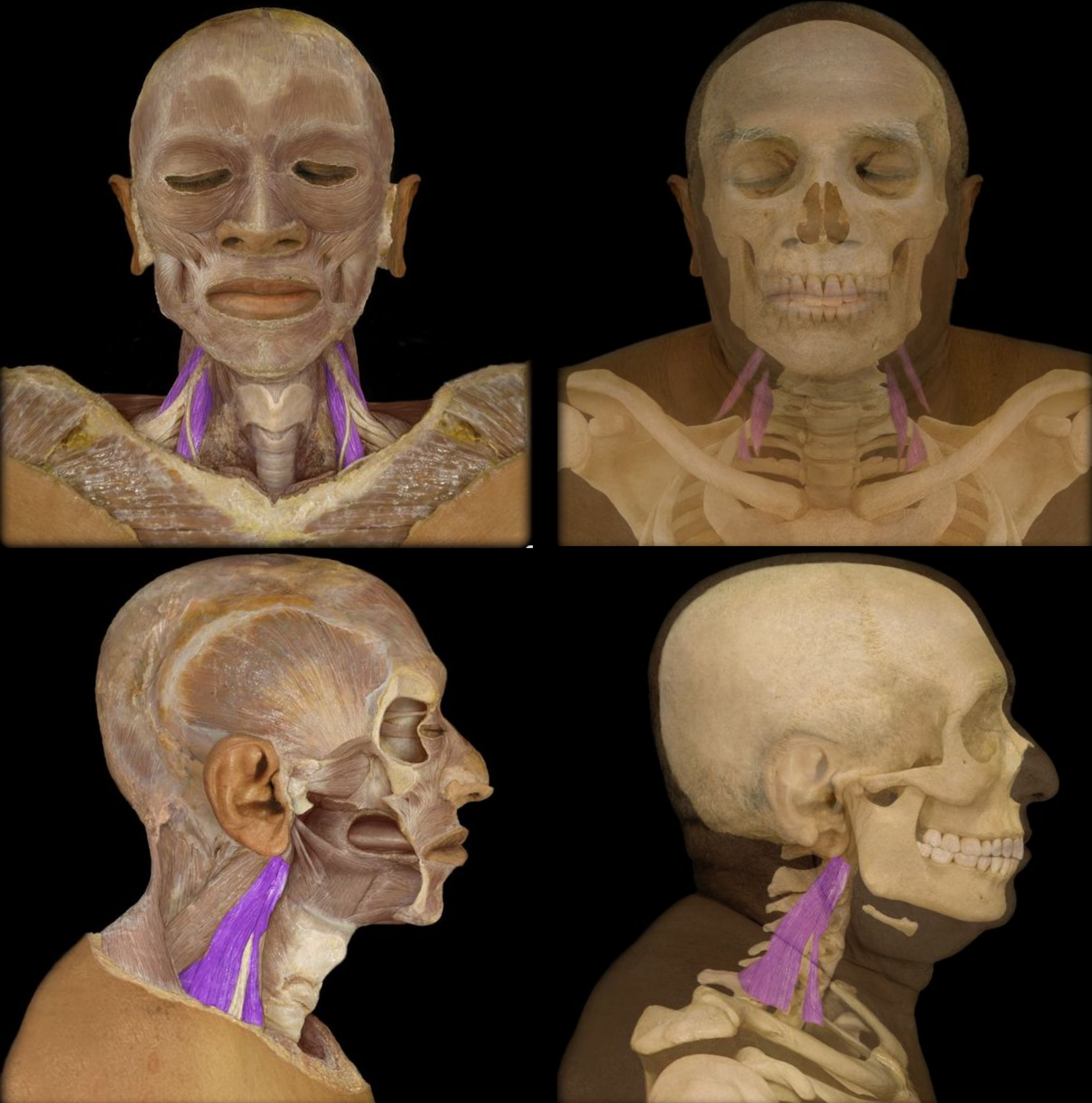
Origins

Insertions

Sternocleidomastoid

Sternocleidomastoid

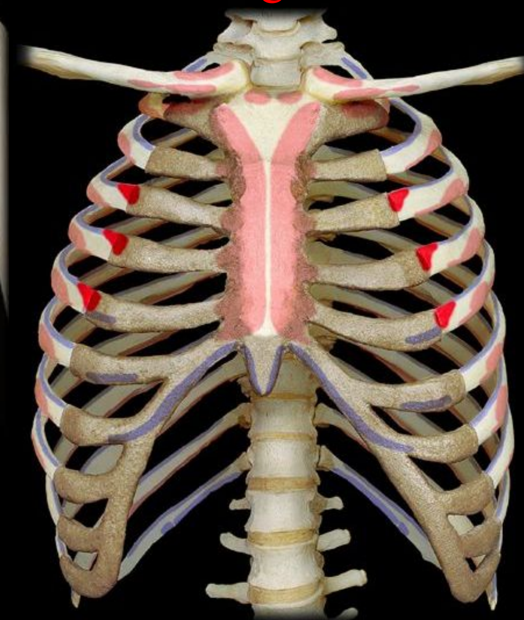




Scalenes

Pectoralis Minor

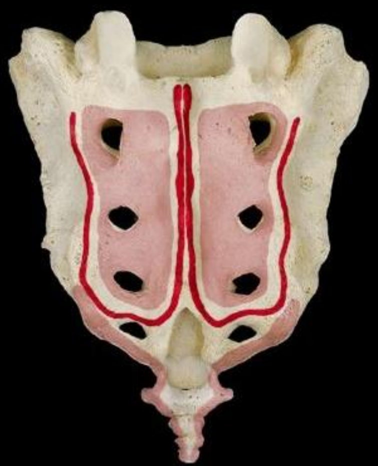
Pectoralis Minor



Serratus Posterior Superior

serrate = scalloped or zigzag

Erector Spinae



Iliocostalis of Erector Spinae

Longissimus of Erector Spinae

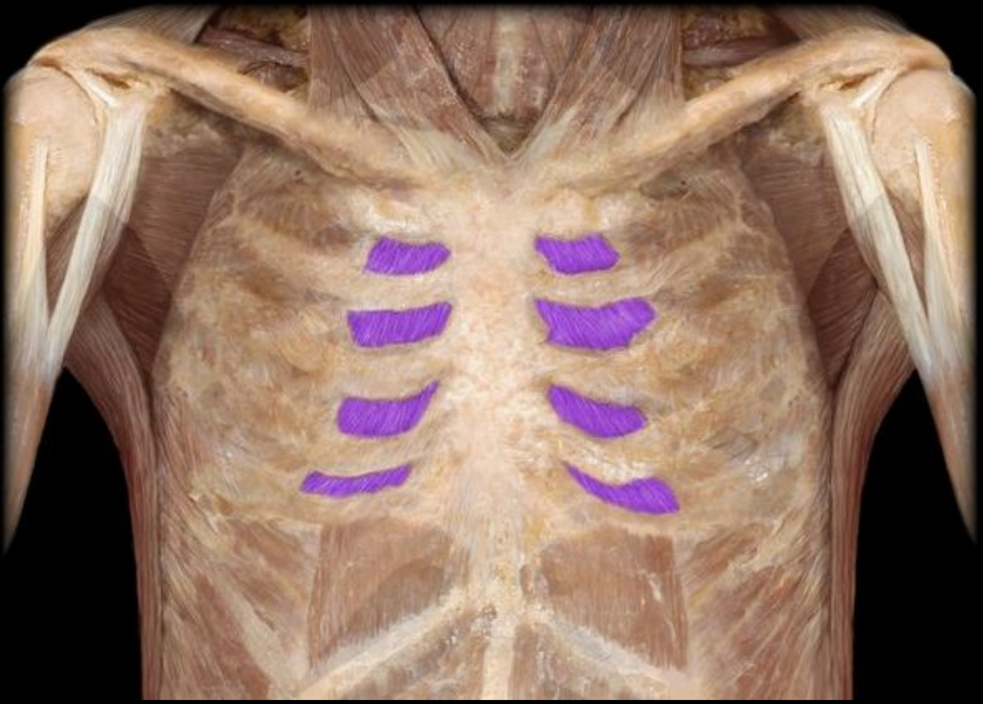
Spinalis of Erector Spinae

23.5b Mechanics of Breathing

Skeletal muscles of breathing (*continued*)

- **Muscles of forced expiration**
  - Include **internal intercostals, abdominal muscles, transversus thoracis,** and **serratus posterior inferior**
  - Contract during a hard expiration, e.g., coughing
  - Either pull the rib cage inferiorly, medially, posteriorly, or compress abdominal contents
  - Collectively termed *accessory muscles of breathing* when paired with the muscles of forced inspiration

Internal Intercostals



Lateral Abdominal Muscles

External Abdominal Oblique

Internal Abdominal Oblique

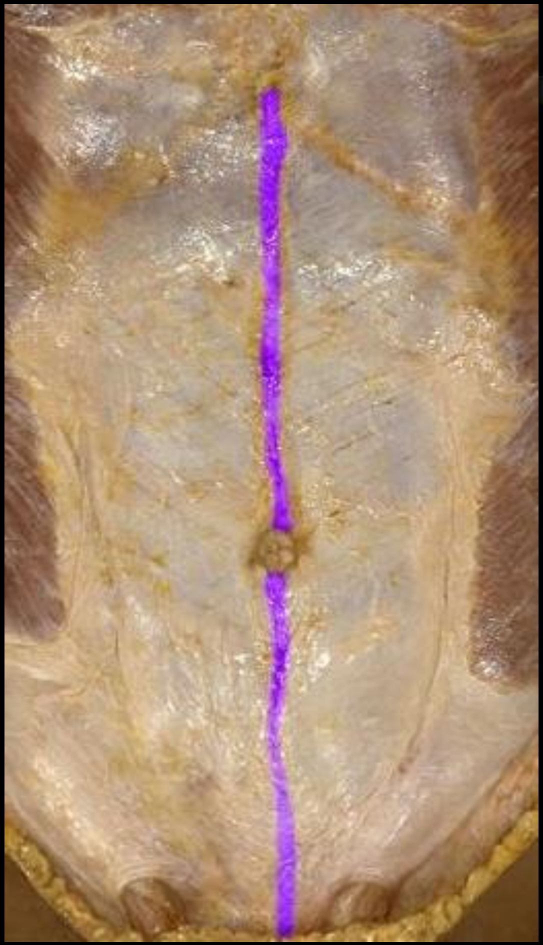
Transverse Abdominis

Internal Abdominal Oblique and Transversus Abdominis

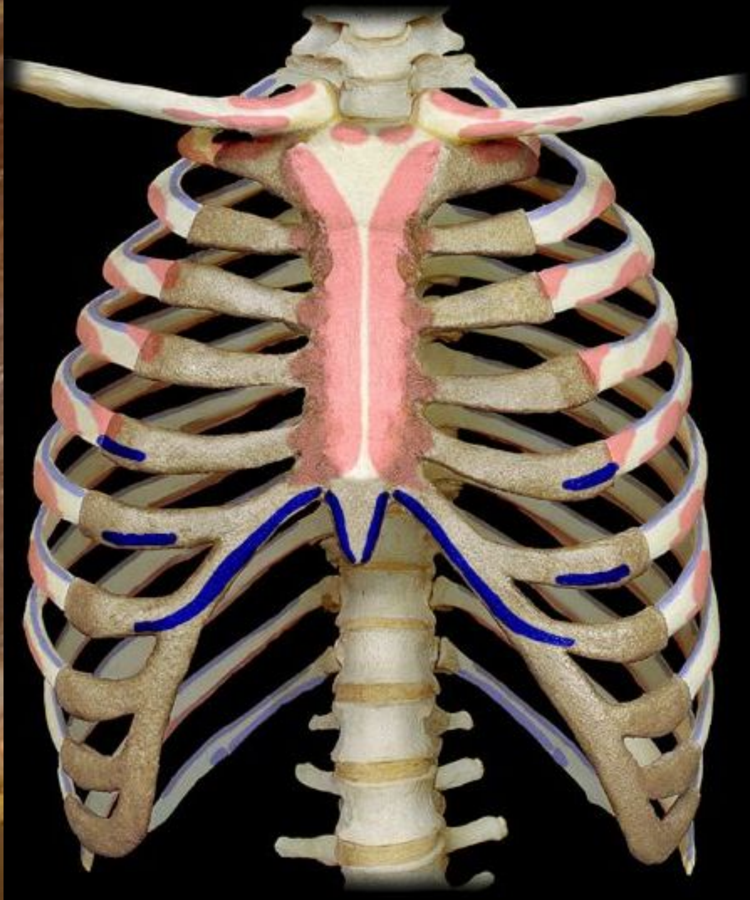
Rectus Abdominis

Tendinous intersections

Linea alba



Insertions



Rectus Abdominis

Serratus Posterior Inferior

serrate = scalloped or zigzag

Skeletal Muscles of Breathing

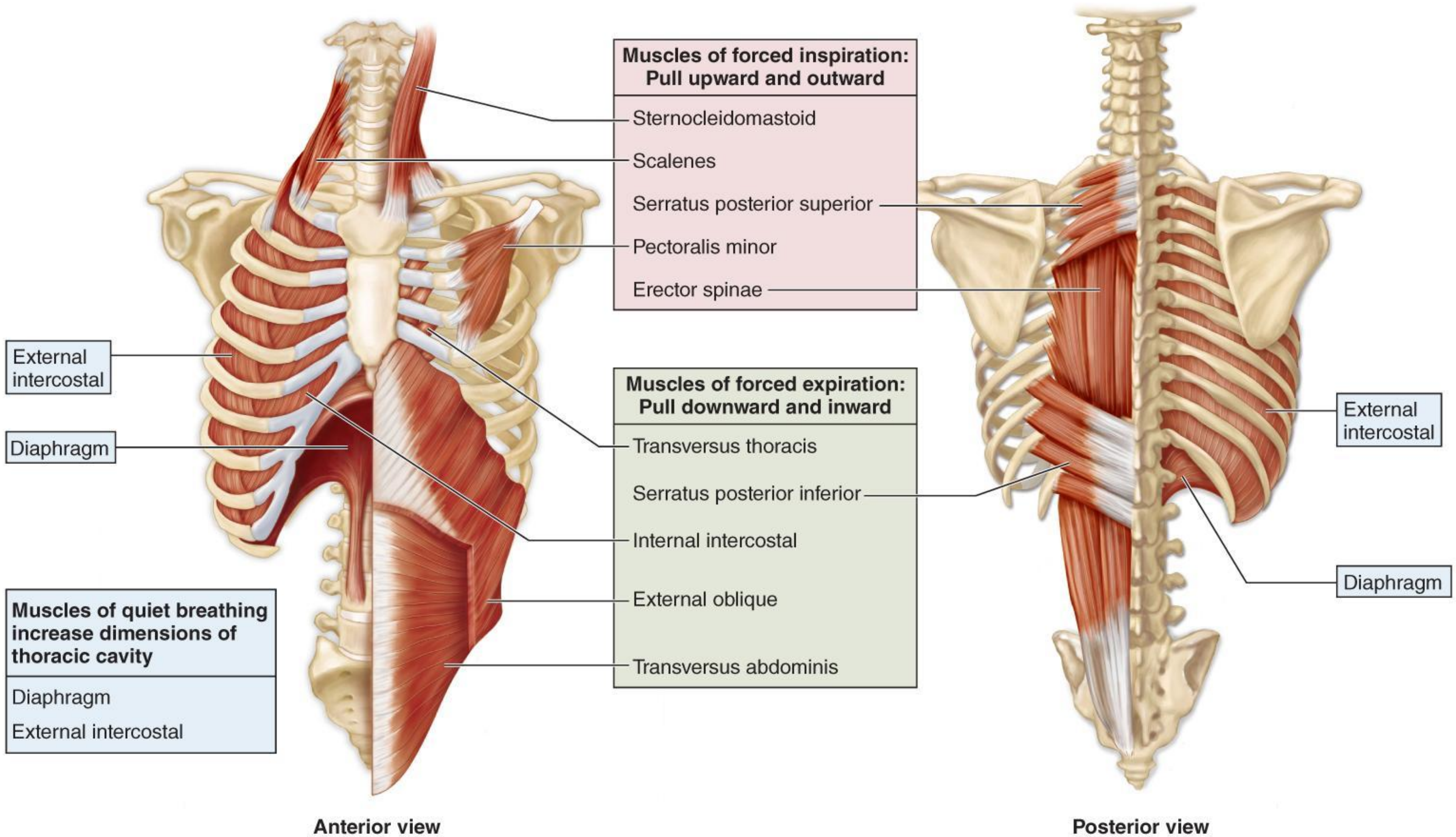


Figure 23.19-top

23.5b Mechanics of Breathing

Volume changes in the thoracic cavity

- Thoracic volume changes vertically, laterally, and anterior-posteriorly
- Vertical changes result from diaphragm movement
  - Flattens (by moving inferiorly) when contracted
  - When relaxed, returns to original position, vertical dimensions decrease
  - For relaxed breathing: only small movements required
  - For forced expiration: abdominal muscle contraction causes larger movement of diaphragm superiorly

Thoracic Cavity Dimensional Changes Associated with Breathing

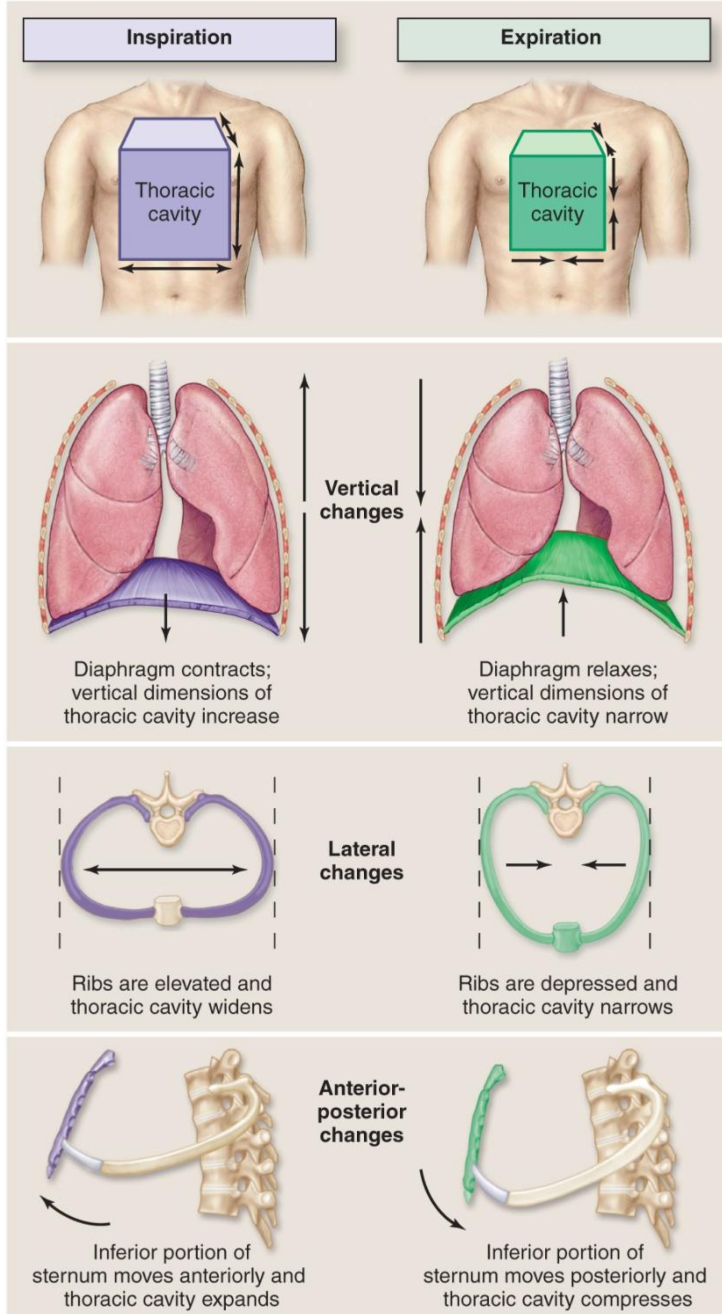


Figure 23.20

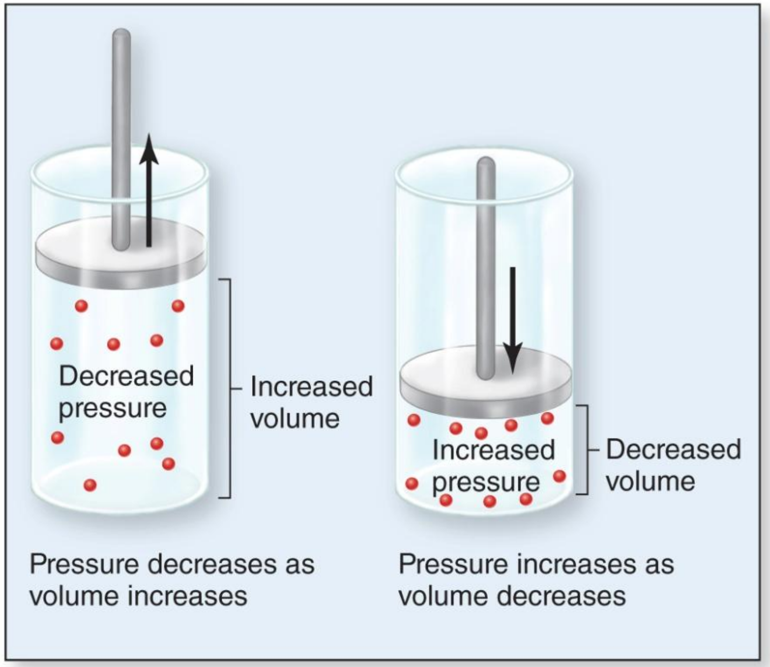
23.5b Mechanics of Breathing

Volume changes in the thoracic cavity (*continued*)

- Lateral dimension changes
  - Rib cage elevation widens thoracic cavity in inspiration
  - Rib cage depression narrows thoracic cavity in expiration
  - Changes due to activity (or relaxation) of all breathing muscles except diaphragm
- Anterior-posterior dimension changes
  - Inferior part of sternum moves anteriorly in inspiration; posteriorly in expiration
  - According to activity level of all breathing muscles except diaphragm

23.5b Mechanics of Breathing

- Boyle's gas law: Relationship of volume and pressure
  - At constant temperature, pressure (P) of a gas decreases if volume (V) of the container increases, and vice versa
  - P_1 and V_1 represent initial conditions and P_2 and V_2 the changed conditions
  - $P_1V_1 = P_2V_2$
  - Inverse relationship between gas pressure and volume

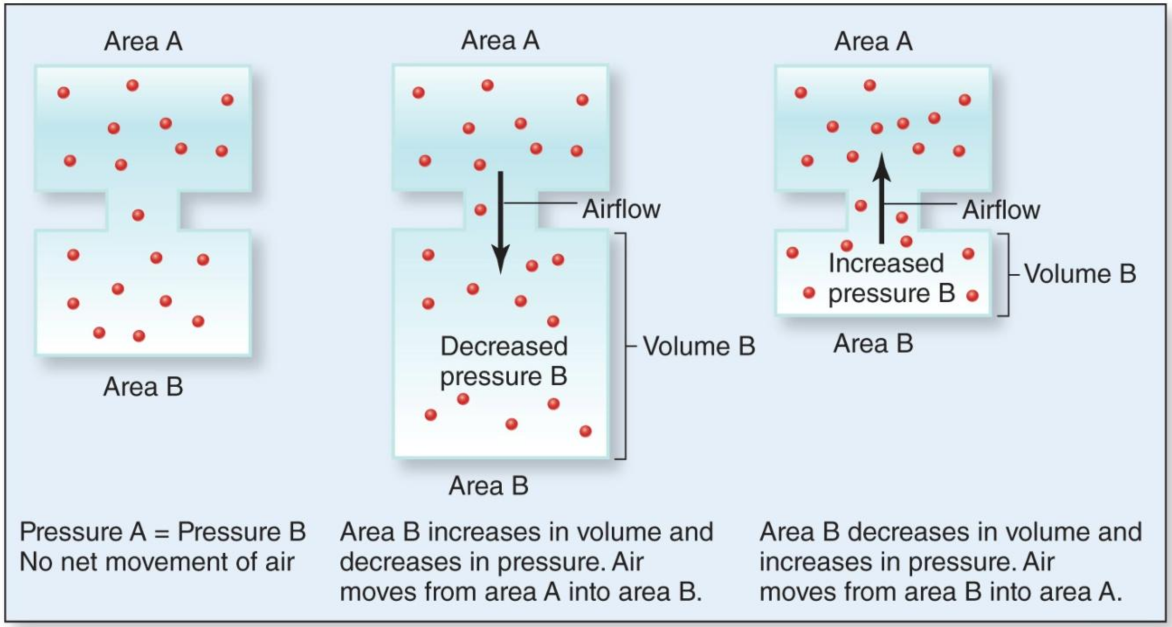


(a) Boyle's law

Figure 23.21a

23.5b Mechanics of Breathing

- An air pressure gradient exists when force per unit area is greater in one place than another
  - If the two places are interconnected, air flows from high to low pressure until pressure is equal



(b) Pressure gradients

Figure 23.21b

23.5b Mechanics of Breathing

Volumes and pressures associated with breathing

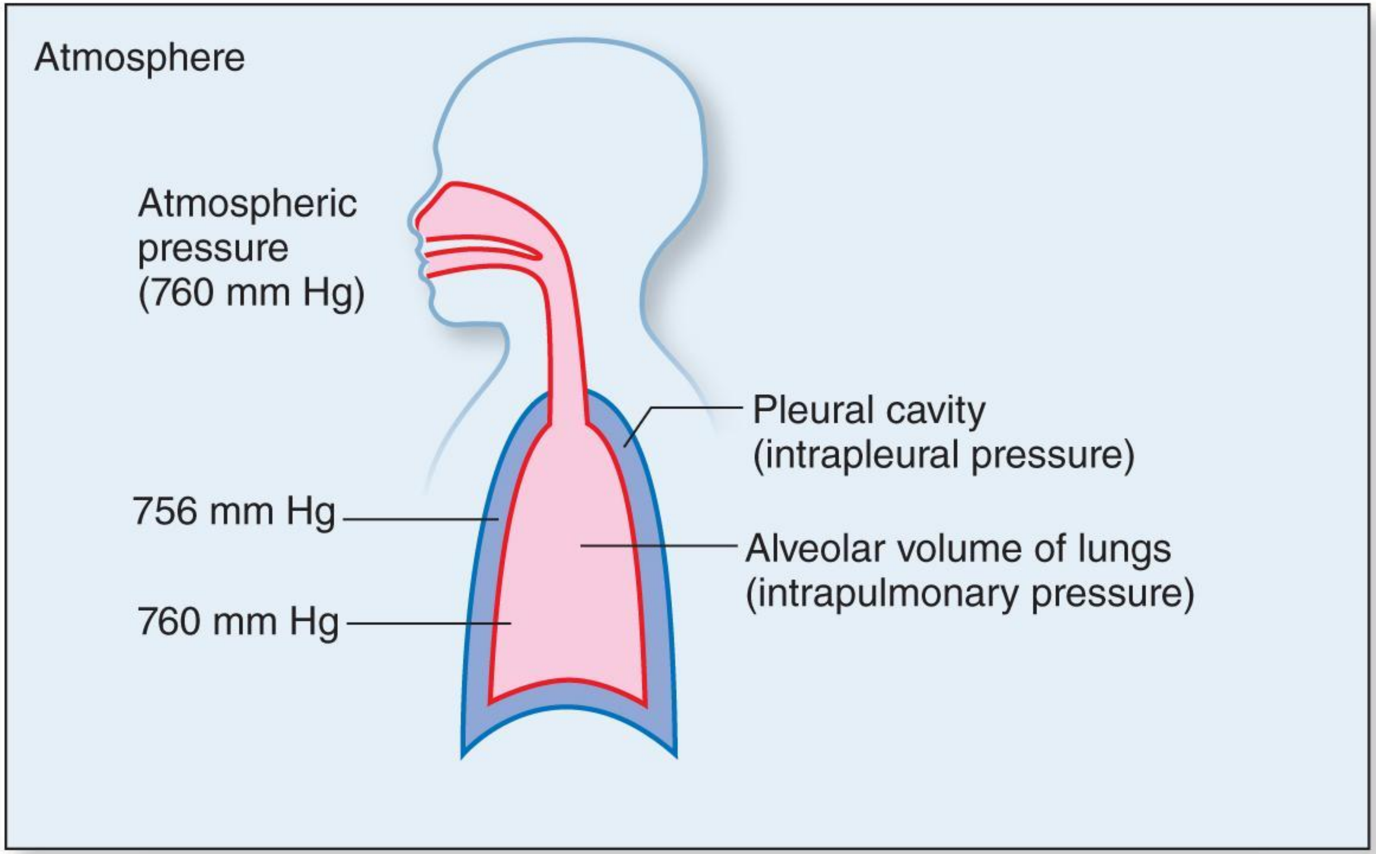
- **Atmospheric pressure:** pressure of air in environment
  - Changes with altitude
    - Increased altitude = "thinner air" = lower pressure
    - Sea level value is 760 mm Hg = 14.7 lbs per square inch = 1 atm
  - Unchanged in process of breathing
- **Alveolar volume:** collective volume of alveoli
- Intrapulmonary pressure: pressure in alveoli
  - Fluctuates with breathing
    - May be higher, lower, or equal to atmospheric pressure
    - Is equal to atmospheric pressure at end of inspiration and expiration

23.5b Mechanics of Breathing

Volumes and pressures associated with breathing (*cont'd.*)

- Intrapleural pressure: pressure in pleural cavity
  - Fluctuates with breathing
  - Is lower than intrapulmonary pressure (keeps lungs inflated)
    - About 4 mm Hg lower than intrapulmonary pressure between breaths
- Volume changes create pressure changes and air flows down its pressure gradient
  - During inspiration: thoracic volume increases, thoracic pressure decreases, so air flows in
  - During expiration: thoracic volume decreases, thoracic pressure increases, so air flows out

Pressures Associated with Breathing



(c) Volumes and pressures with breathing (at the end of an expiration)

Figure 23.21c

23.5b Mechanics of Breathing

Quiet breathing: Inspiration

1) Intrapulmonary pressure and atmospheric pressure are initially equal (760 mg Hg)
   - Intrapleural pressure is 4 mm Hg lower
2) Diaphragm and external intercostals contract increasing thoracic volume
   - Diaphragm movement accounts for 2/3 of volume change; external intercostal movement accounts for 1/3
   - Intrapleural volume increases, so intrapleural pressure decreases
   - Lungs pulled by pleurae, so lung volume increases and intrapulmonary pressure decreases
   - Because intrapulmonary pressure is less than atmospheric pressure, air flows in until these pressures are equal
     - Typically 0.5 L flows in as tidal volume

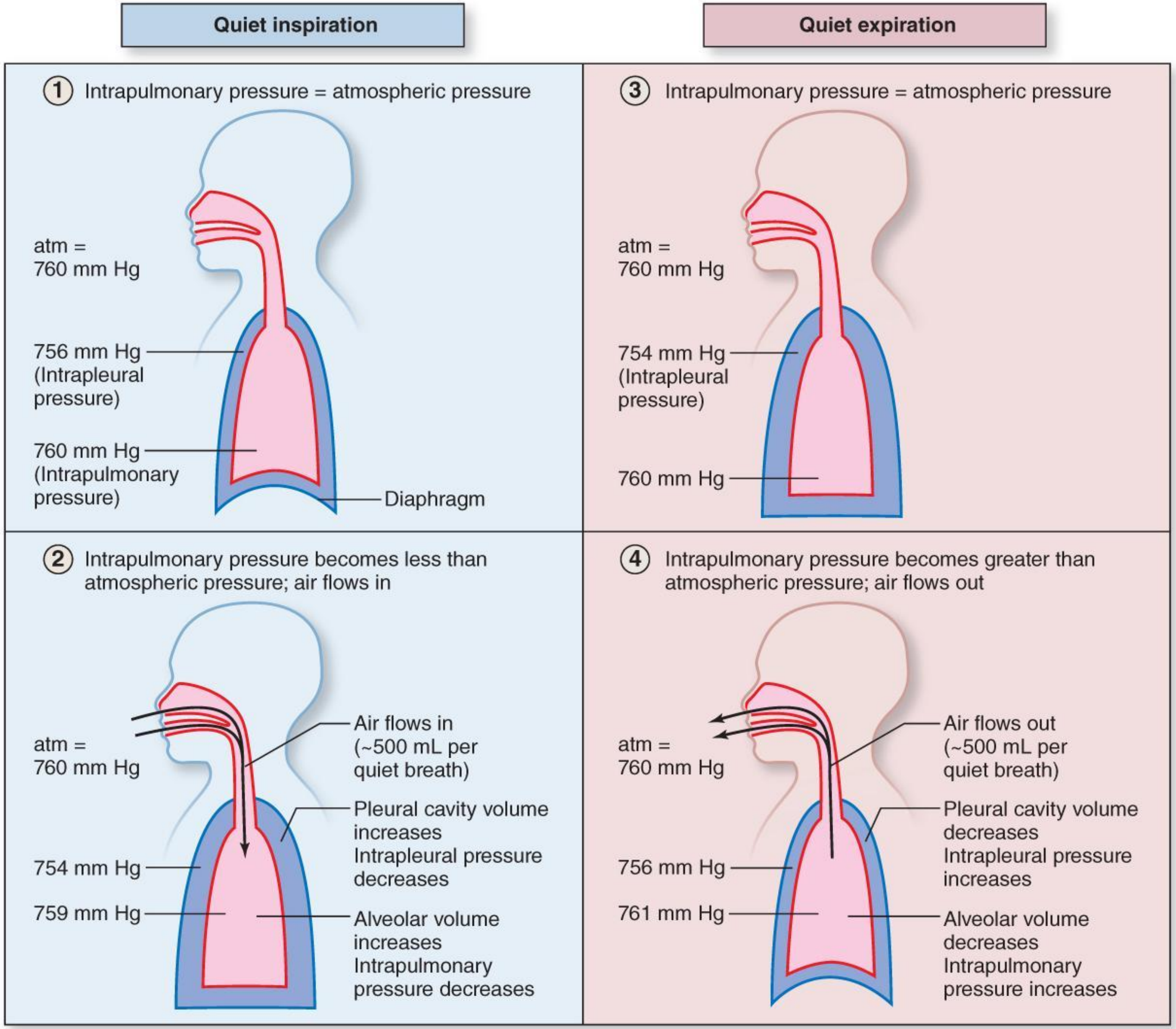
23.5b Mechanics of Breathing

Quiet breathing: Expiration

3) Initially, intrapulmonary pressure equals atmospheric pressure
   - Intrapleural pressure is about 6 mm Hg lower

5) Diaphragm and external intercostals relax decreasing thoracic volume
   - Pleural cavity volume decreases, so intrapleural pressure increases
   - Elastic recoil pulls lungs inward, so alveolar volume decreases and intrapulmonary pressure increases
   - Since intrapulmonary pressure is greater than atmospheric pressure, air flows out until these pressures are equal
     - About 0.5 L of air leaves the lung

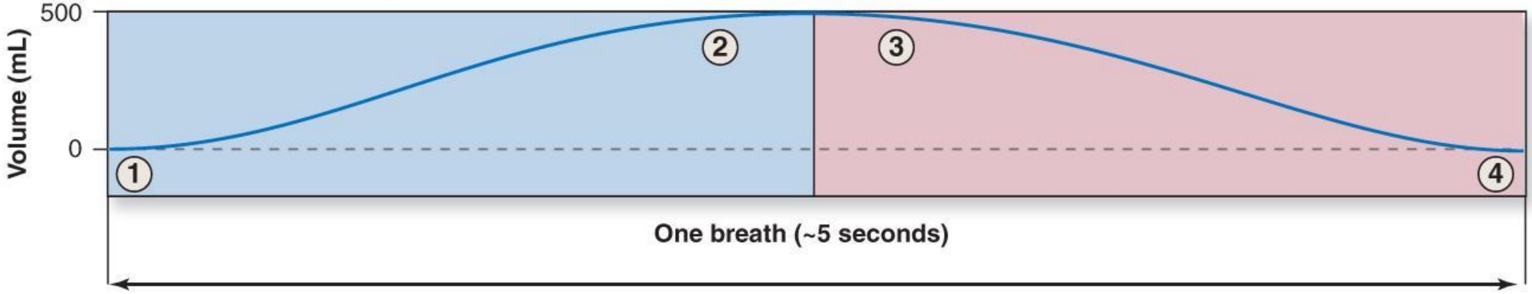
Volume and Pressure Changes During Quiet Breathing



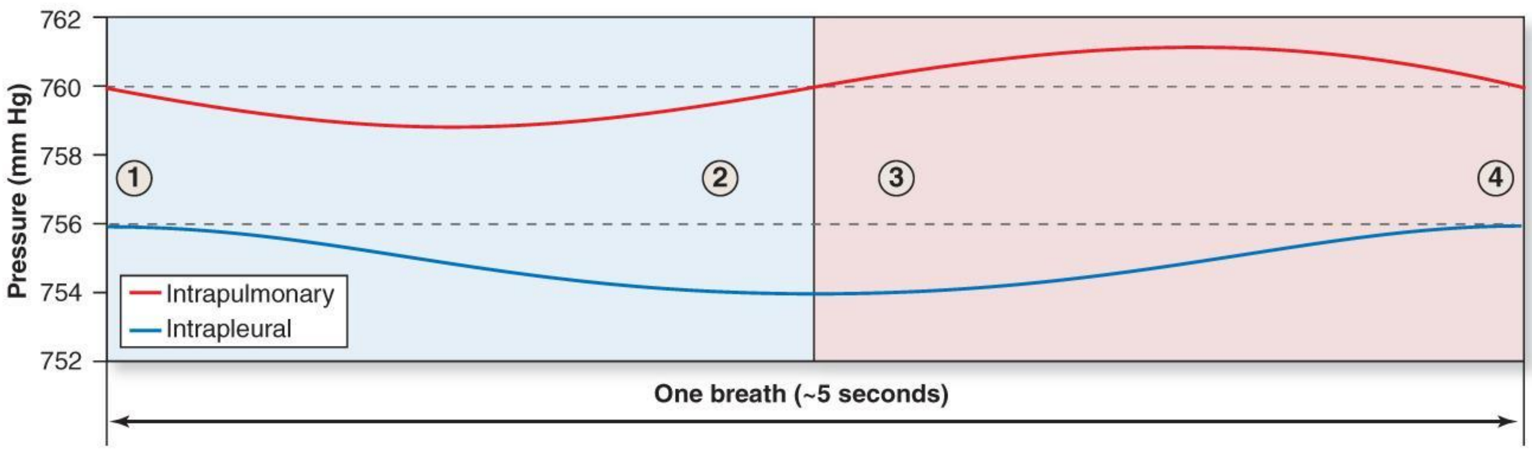
(a) Mechanical events of quiet inspiration and quiet expiration

Figure 23.22a

Volume and Pressure Changes During Quiet Breathing



(b) Volume changes within lungs during one breath



(c) Pressure changes within lungs and pleural cavity

Figure 23.22b-c

Table 23.3 Changes Associated with Quiet Breathing

Variable	Inspiration	Expiration
Diaphragm and external intercostals	Contracting (active)	Relaxing (passive)
Pleural cavity	Volume increases; pressure decreases	Volume decreases; pressure increases
Lungs	Volume increases; pressure decreases	Volume decreases; pressure increases
Air movement	Into lungs	Out of lungs

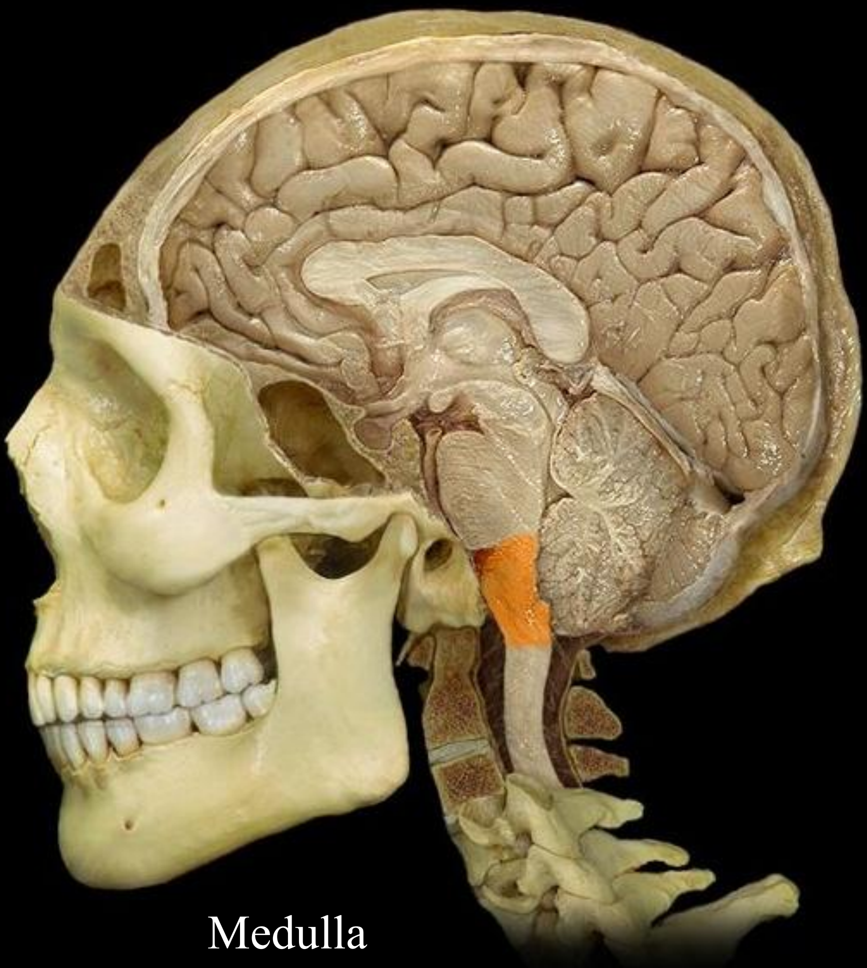
23.5b Mechanics of Breathing

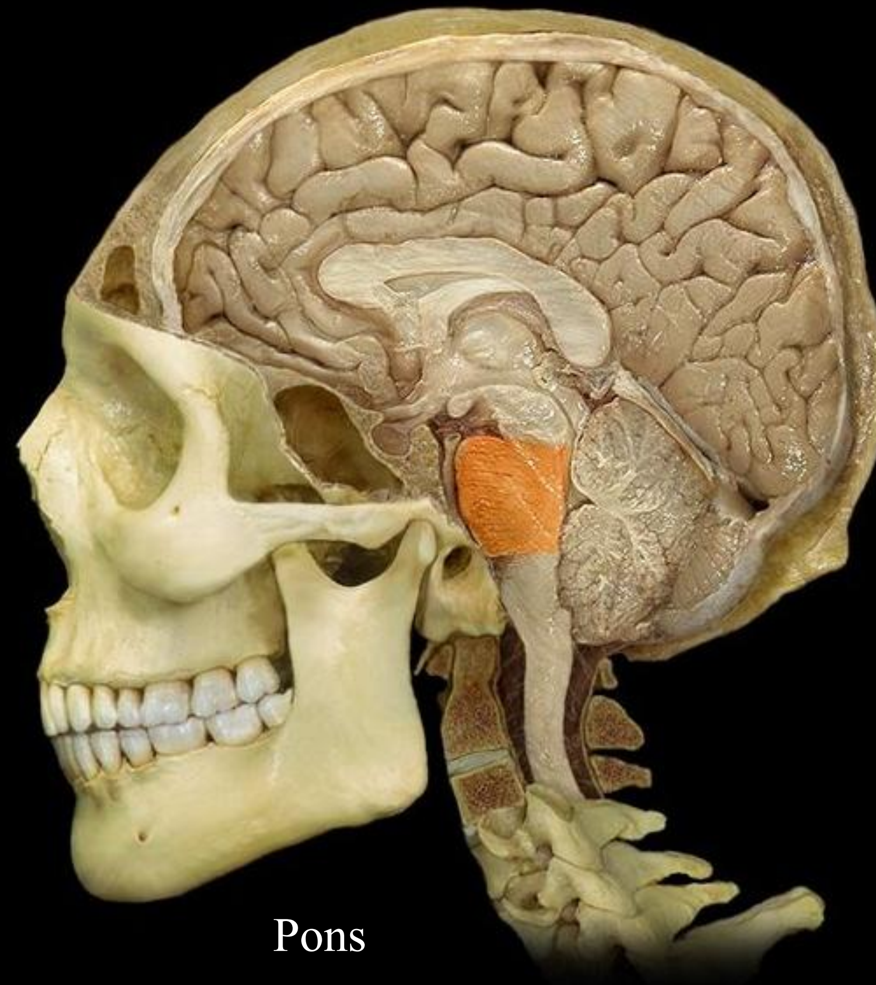
- **Forced breathing**
  - Involves steps similar to quiet breathing
  - Requires contraction of additional muscles
  - Causes greater changes in thoracic cavity volume and intrapulmonary pressure
  - More air moves into and out of lungs
  - Significant chest volume changes are apparent

23.5c Nervous Control of Breathing

- Autonomic nuclei within the brain coordinate breathing
  - **Respiratory center** of the brainstem
    - **Medullary respiratory center** contains two groups
      - **Ventral respiratory group (VRG)** in anterior medulla
      - **Dorsal respiratory group (DRG)** in posterior medulla
    - **Pontine respiratory center** in pons also known as *pneumotaxic center*
- Brainstem neurons influence respiratory muscles
  - VRG neurons synapse with lower motor neurons of skeletal muscles in spinal cord
  - Lower motor neuron axons project to respiratory muscles
    - Axons innervating diaphragm travel in phrenic nerves
    - Axons innervating intercostal muscles travel in intercostal nerves

Neural Control of Breathing





23.5c Nervous Control of Breathing

- Chemoreceptors monitor changes in concentrations of H^+, P_{CO_2} and P_{O_2}
  - **Central chemoreceptors** in medulla monitor pH of CSF
    - CSF pH changes are caused by changes in blood P_{CO_2}
      - CO_2 diffuses from blood to CSF where carbonic anhydrase is
      - Carbonic anhydrase builds carbonic acid from CO_2 and water
  - **Peripheral chemoreceptors** are in aortic and carotid bodies
    - Stimulated by changes in H^+ or respiratory gases in blood
      - Respond to H^+ produced independently of CO_2
        - » E.g., H^+ from ketoacidosis (from fatty acid metabolism)
    - Carotid chemoreceptors send signals to respiratory center via glossopharyngeal nerve
    - Aortic chemoreceptors send signals to respiratory center via vagus nerve

23.5c Nervous Control of Breathing

- Other receptors also influence respiration
  - **Proprioceptors** of muscles and joints are stimulated by body movements
  - **Baroreceptors** in pleurae and bronchioles respond to stretch
  - **Irritant receptors** in air passageways stimulated by particulate matter

Respiratory Center

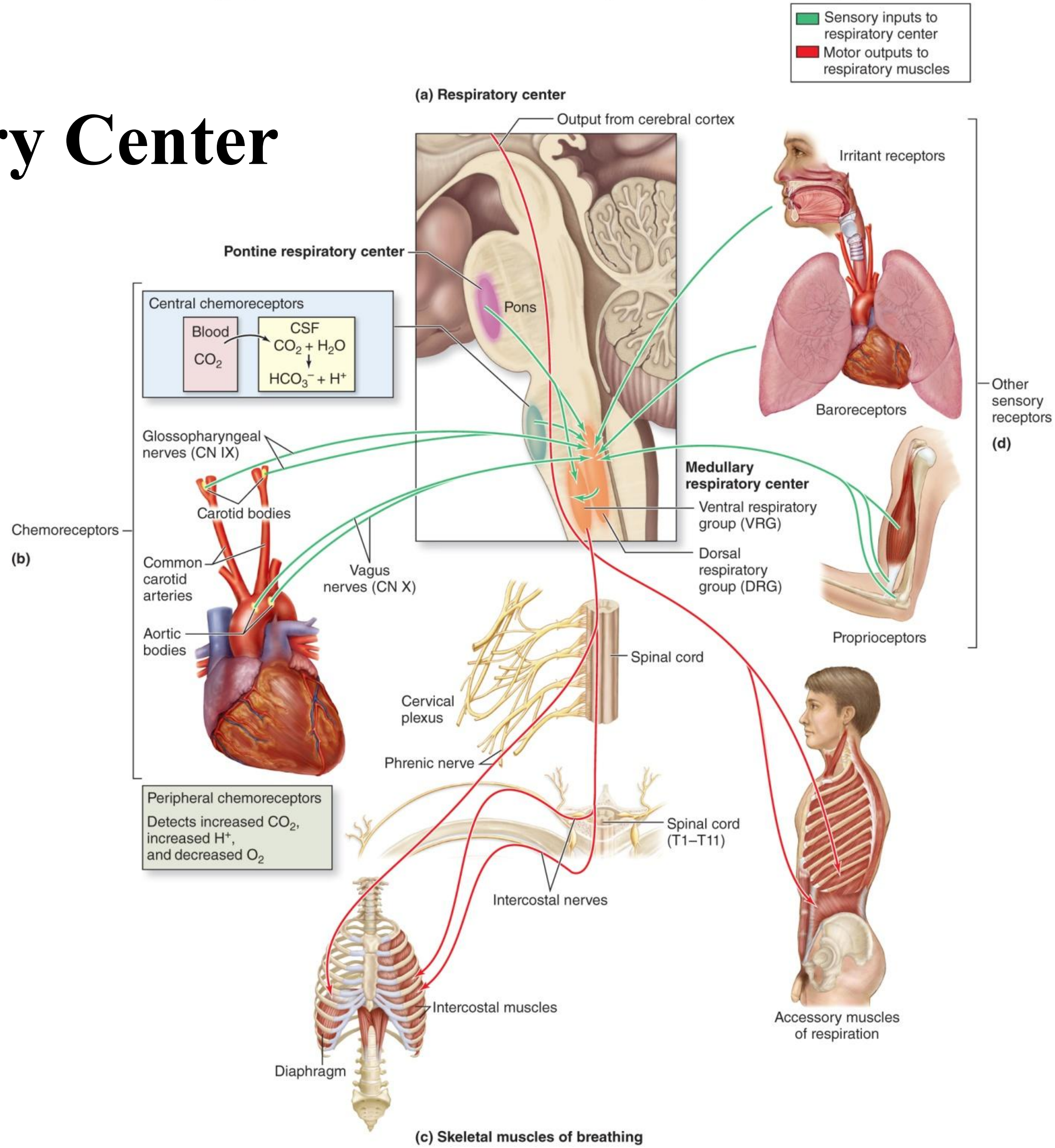


Figure 23.23

23.5c Nervous Control of Breathing

- Physiology of quiet breathing
  - Inspiration begins when VRG inspiratory neurons fire spontaneously
  - Signals are sent from VRG to nerve pathways exciting skeletal muscles for about 2 seconds
    - Diaphragm and external intercostals contract causing air to flow in
  - *Quiet expiration* occurs when VRG is inhibited
    - Signals from inspiratory neurons are relayed to VRG expiratory neurons
    - Expiratory neurons send inhibitory signals back (negative feedback)
  - Signals no longer sent to inspiratory muscles (for about 3 sec)
    - Diaphragm and external intercostals relax causing air to flow out

23.5c Nervous Control of Breathing

- Physiology of quiet breathing (*continued*)
  - Respiration rate for normal, quiet breathing is **eupnea**
    - Average of 12–15 breaths per minute
  - Pontine respiratory center facilitates smooth transitions between inspiration and expiration
    - Sends signals to medullary respiratory center
    - Damage to pons causes erratic breathing

Clinical View: Apnea

- **Apnea =** absence of breathing
  - Can occur voluntarily
    - Swallowing or holding your breath
  - Can be drug-induced
  - Can result from neurological disease or trauma
- **Sleep apnea =** temporary cessation of breathing during sleep

23.5c Nervous Control of Breathing

- Reflexes that alter breathing rate and depth
  - Chemoreceptors alter breathing by sending signals to DRG, which are then relayed to VRG
    - VRG triggers changes in rhythm and force of breathing
      - Rate changes by altering amount of time in inspiration and expiration
      - Depth changes by stimulation of accessory muscles
  - Ventilation increases in response to
    - Central chemoreceptors detecting increase in H^+ concentration of CSF
    - Peripheral chemoreceptors detecting increase in blood H^+ or P_{CO_2}
  - Increased ventilation expels more CO_2 returning conditions to normal
  - Ventilation decreases if chemoreceptors detect decreases in H^+ or P_{CO_2}

23.5c Nervous Control of Breathing

- Reflexes that alter breathing rate and depth (*cont'd.*)
  - Blood P_{CO_2} is most important stimulus affecting breathing
    - Raising blood P_{CO_2} by 5 mm Hg causes doubling of breathing rate
    - CO_2 fluctuations influence sensitive central chemoreceptors
      - CO_2 combines with water to form carbonic acid in CSF
      - CSF lacks protein buffers and so its pH change triggers reflexes
    - Blood P_{O_2} is not a sensitive regulator of breathing
      - Arterial oxygen must decrease from 95 to 60 mm Hg to have major effect independent of P_{CO_2}
      - When P_{O_2} drops it causes peripheral chemoreceptors to be more sensitive to blood P_{CO_2}

Clinical View: Hypoxic Drive

- Normally the most important stimulus affecting breathing rate and depth is blood P_{CO_2}
- **Hypoxic drive =** P_{O_2} levels become stimulus for breathing
  - Occurs in some respiratory disorders such as emphysema with decreased ability to exhale carbon dioxide
  - Carbon dioxide levels in the blood remain elevated for a long period
  - Chemoreceptors become less sensitive to P_{CO_2}
  - By default, decreased P_{O_2} stimulates them
  - Administering oxygen can elevate P_{O_2} and interfere with the person's ability to breathe on his own

23.5c Nervous Control of Breathing

- Reflexes that alter breathing rate and depth (*cont'd.*)
  - Altering breathing through other receptors
    - Joint and muscle proprioceptors are stimulated by body movement
      - Signal respiratory center to increase breathing depth
    - Baroreceptors within visceral pleura and bronchiole smooth muscle
      - Send signals to respiratory center when overstretched
      - Initiate **inhalation reflex** (*Hering-Breuer reflex*) to shut off inspiration and protect against overinflation
    - Irritant receptors initiate sneezing and coughing
      - Exaggerated intake of breath followed by closure of larynx
      - Contraction of abdominal muscles
      - Abrupt opening of vocal cords and explosive blast of exhaled air

23.5c Nervous Control of Breathing

- Reflexes that alter breathing rate and depth (*cont'd.*)
  - Action of higher brain centers
    - Hypothalamus increases breathing rate if body is warm
      - Works through respiratory center
    - Limbic system alters breathing rate in response to emotions
      - Works through respiratory center
    - Frontal lobe of cerebral cortex controls voluntary changes in breathing patterns
      - Bypasses respiratory center stimulating lower motor neurons directly

23.5c Nervous Control of Breathing

Nervous control of respiratory system structures and breathing structures

- Respiratory system includes smooth muscles and glands
  - Innervated by axons of lower motor neurons of autonomic nervous system
  - Controlled by autonomic brainstem nuclei
- Breathing muscles are skeletal muscles
  - Innervated by lower motor neurons of somatic nervous system
  - Controlled by brainstem autonomic nuclei, cerebral cortex, and somatic nervous system
- Thus, there are both reflexive and conscious controls of breathing

23.5d Airflow, Pressure Gradients, and Resistance

- **Airflow:** amount of air moving in and out of lungs with each breath
  - Depends on
    1) The *pressure gradient* established between atmospheric pressure and intrapulmonary pressure
    2) The *resistance* that occurs due to conditions within the airways, lungs, and chest wall

23.5d Airflow, Pressure Gradients, and Resistance

- $F = \Delta P/R$
  - F = flow
  - ΔP = difference in pressure between atmosphere and intrapulmonary pressure = **pressure gradient =** $P_{atm} - P_{alv}$
  - R = resistance
  - Flow directly related to pressure gradient and inversely related to resistance
  - If pressure gradient increases, airflow to lungs increases
  - If resistance increases, airflow lessens

23.5d Airflow, Pressure Gradients, and Resistance

- Pressure gradient
  - Can be changed by altering volume of thoracic cavity
    - Small volume changes of quiet respiration allow 500 mL of air to enter
    - If accessory muscles of inspiration are used, volume increases more
      - Airflow increases due to greater pressure gradient
- **Resistance:** greater difficulty moving air
  - May be altered by
    1) Change in elasticity of chest wall and lungs
    2) Change in bronchiole diameter (size of air passageway)
    3) Collapse of alveoli

23.5d Airflow, Pressure Gradients, and Resistance

- Resistance (*continued*)
  - Decreases in chest wall elasticity increase resistance
    - Chest wall elasticity decreases with aging and disease
      - Vertebral malformations (scoliosis) can decrease elasticity
      - Arthritis in thoracic cage
      - Replacement of elastic tissue with scar tissue (pulmonary fibrosis)
  - Bronchiole diameter varies inversely with resistance
    - Bronchoconstriction or occlusion increase resistance
      - Constriction caused by parasympathetic activity, histamine, or cold
      - Occlusion by excess mucus or inflammation
    - Bronchodilation decreases resistance
      - Caused by sympathetic stimulation, epinephrine

23.5d Airflow, Pressure Gradients, and Resistance

- Resistance (*continued*)
  - Collapsed alveoli increase resistance
    - Can occur if alveolar type II cells are not producing surfactant (high surface tension of alveoli is not overcome)
      - An important factor for premature infants
      - Alveoli collapse with expiration increasing resistance
      - Condition referred to as **acute respiratory distress syndrome (ARDS)**
- **Compliance**
  - Ease with which lungs and chest wall expand
  - Determined by surface tension and elasticity of chest and lung
  - The easier the lung expands, the greater the compliance

23.5d Airflow, Pressure Gradients, and Resistance

- Several conditions can increase resistance to airflow
  - Decreases in size of bronchiole lumen (asthma)
  - Decrease in compliance (pulmonary fibrosis)
  - The result is a need for more forceful inspirations
  - More forceful inspirations of respiratory disorders require high amount of energy
    - Can cause four-fold to six-fold increase in energy need
      - From 5% to 25% of body's total energy expenditure
    - Individuals with these conditions can become exhausted

23.5e Pulmonary and Alveolar Ventilation

- **Pulmonary ventilation**
  - Process of moving air into and out of the lungs
  - Amount of air moved between atmosphere and alveoli in 1 minute
- Tidal volume = amount of air per breath
- Respiration rate = number of breaths per minute
- Tidal volume × Respiration rate = Pulmonary ventilation

  500 mL × 12 breaths/min = 6 L/ minute (typical amount)

23.5e Pulmonary and Alveolar Ventilation

- **Anatomic dead space:** conducting zone space
  - No exchange of respiratory gases here
  - About 150 mL
- **Alveolar ventilation**
  - Amount of air reaching alveoli per minute
  - (Tidal volume – anatomic dead space) × Respiration rate = Alveolar ventilation

    (500 mL – 150 mL) × 12 = 4.2 L/min
  - Deep breathing maximizes alveolar ventilation

23.5e Pulmonary and Alveolar Ventilation

- **Physiologic dead space**
  - Normal anatomic dead space + any loss of alveoli
  - Some disorders decrease number of alveoli participating in gas exchange
    - Due to damage to alveoli or changes in respiratory membrane (e.g., pneumonia)

23.5f Volume and Capacity

- **Spirometer** measures respiratory volume
  - Can be used to assess respiratory health
    - Standard values are available (e.g., for people of different ages)
  - Four volumes measured by spirometry
    - **Tidal volume:** amount of air inhaled or exhaled per breath during quiet breathing
    - **Inspiratory reserve volume (IRV):** amount of air that can be forcibly inhaled beyond the tidal volume
      - Measure of compliance
    - **Expiratory reserve volume (ERV):** amount that can be forcibly exhaled beyond tidal volume
      - Measure of elasticity
    - **Residual volume:** amount of air left in the lungs after the most forceful expiration

23.5f Volume and Capacity

- Four capacities calculated from respiratory volumes
  - **Inspiratory capacity (IC)**
    - Tidal volume + inspiratory reserve volume
  - **Functional residual capacity (FRC)**
    - Expiratory reserve volume + residual volume
    - Volume left in the lungs after a quiet expiration
  - **Vital capacity**
    - Tidal volume + inspiratory and expiratory reserve volumes
    - Total amount of air a person can exchange through forced breathing
  - **Total lung capacity (TLC)**
    - Sum of all volumes, including residual volume
    - Maximum volume of air that the lungs can hold

23.5f Volume and Capacity

- Additional respiratory measurements—*rates* of air movement
  - **Forced expiratory volume (FEV)**
    - Percent of vital capacity that can be expelled in a set period of time
    - FEV_1 = percentage expelled in one second
    - 75–85% of vital capacity in a healthy person
      - Less in emphysema patients and others with poor expiration
  - **Maximum voluntary ventilation (MVV)**
    - Greatest amount of air that can be taken in and then expelled from the lungs in 1 minute
    - Breathing as quickly and as deeply as possible
    - Can be as high as 30 L/min (compared to 6 L/min at rest)
    - All respiratory disorders impair this

Respiratory Volumes and Capacities

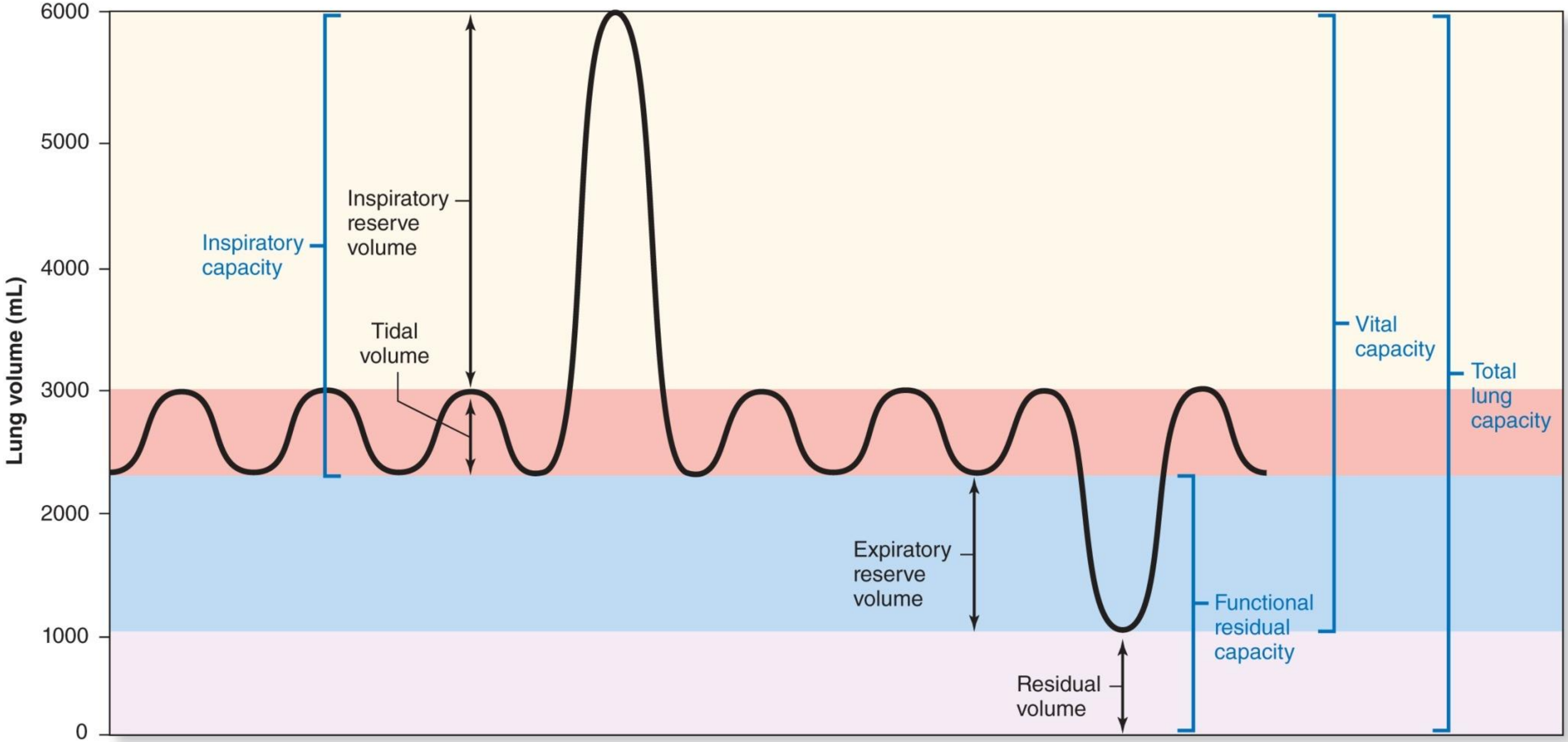


Figure 23.24

Table 23.4 Respiratory Volumes and Capacities

VOLUMES

Volume	Definition	Normal Values (Male)	Normal Values (Female)
Tidal volume (TV)	The amount of air taken into or expelled out of lungs during a quiet breath	500 mL	500 mL
Inspiratory reserve volume (IRV)	The amount of air taken into the lungs during a forced inspiration, following a quiet inspiration; IRV is a measure of lung compliance	3100 mL	1900 mL
Expiratory reserve volume (ERV)	The amount of air expelled from the lungs during a forced expiration, following a quiet expiration; ERV is a measure of lung and chest wall elasticity	1200 mL	700 mL
Residual volume (RV)	The amount of air left (residual) in lungs following a forced expiration	1200 mL	1100 mL

CAPACITIES

Capacity	Formula	Definition	Normal Values (Male)	Normal Values (Female)
Inspiratory capacity	TV + IRV	Total ability to inspire	3600 mL	2400 mL
Functional residual capacity	ERV + RV	Amount of air normally left (residual) in lungs after you expire quietly	2400 mL	1800 mL
Vital capacity	TV + IRV + ERV	Measure of the amount of air the lungs are capable of holding	4800 mL	3100 mL
Total lung capacity	TV + IRV + ERV + RV	Total amount of air that can be in lungs	6000 mL	4200 mL

What did you learn?

- What is Boyle's law and how does it relate to respiration?
- Which muscles are involved in quiet respiration, and what nerves control them?
- To what chemical signal is the body most sensitive with regard to respiratory control?
- What parts of the brain control respiration
- What is vital capacity?

23.6 Respiration: Alveolar and Systemic Gas Exchange

Learning Objectives:

1. Define partial pressure and the movement of gases relative to a partial pressure gradient.
2. Describe the partial pressures that are relevant to gas exchange.
3. Explain the laws that govern gas solubility.
4. Describe alveolar gas exchange and the partial pressure gradients responsible.

23.6 Respiration: Alveolar and Systemic Gas Exchange (*continued*)

Learning Objectives:

5. Name the two anatomic features of the respiratory membrane that contribute to efficient alveolar gas exchange.
6. Explain ventilation-perfusion coupling and how it maximizes alveolar gas exchange.
7. Explain the partial pressure gradients between the systemic cells and the blood in capillaries.
8. Differentiate between alveolar and systemic gas exchange.

23.6a Chemical Principles of Gas Exchange

Partial pressure and Dalton's law

- **Partial pressure:** pressure exerted by each gas within a mixture of gases, measured in mm Hg
  - Written with P followed by gas symbol (i.e., PO_2)
  - Each gas moves independently down its partial pressure gradient during gas exchange
- Atmospheric pressure = 760 mm Hg at sea level
  - Total pressure all gases collectively exert in the environment
  - Includes N_2, O_2, CO_2, H_2O, and other minor gases

23.6a Chemical Principles of Gas Exchange

Partial pressure and Dalton's law (*continued*)

- Total pressure × % of gas = Partial pressure of that gas
  - Nitrogen is 78.6% of the gas in air
  - 760 mm HG × 78.6% = 597 mm Hg = partial pressure of nitrogen
  - Partial pressures added together equal the total atmospheric pressure
- **Dalton's law**
  - The total pressure in a mixture of gases is equal to the sum of the individual partial pressures

23.6a Chemical Principles of Gas Exchange

Partial pressure gradients

- Gradient exists when partial pressure for a gas is higher in one region of the respiratory system than another
- Gas moves from region of higher partial pressure to region of lower partial pressure until pressures become equal
- Both types of gas exchange depend on gradients
  - Alveolar gas exchange: between blood in pulmonary capillaries and alveoli
  - Systemic gas exchange: between blood in systemic capillaries and systemic cells

23.6a Chemical Principles of Gas Exchange

Relevant partial pressures in the body

- Reasons partial pressures in alveoli differ from atmospheric partial pressures
  - Air from environment mixes with air remaining in anatomic dead space
  - Oxygen diffuses out of alveoli into the blood; carbon dioxide diffuses from blood into alveoli
  - More water vapor is present in alveoli than in atmosphere
- Within alveoli, the…
  - percentage and partial pressure of O_2 are lower than in atmosphere
  - percentage and partial pressure of CO_2 are higher than in atmosphere
  - partial pressures of respiratory gases normally stay constant

23.6a Chemical Principles of Gas Exchange

Relevant partial pressures in the body (*continued*)

- In systemic cells, partial pressures of gases reflect cellular respiration (use of O_2, production of CO_2)
  - The percentage of O_2 lower and CO_2 higher than in alveoli
  - Under resting, normal conditions the partial pressures remain constant
- In circulating blood, gas partial pressures are not constant
  - O_2 enters blood in pulmonary capillaries; CO_2 leaves
  - O_2 leaves blood in systemic capillaries; CO_2 enters

Alveolar and Systemic Gas Exchange

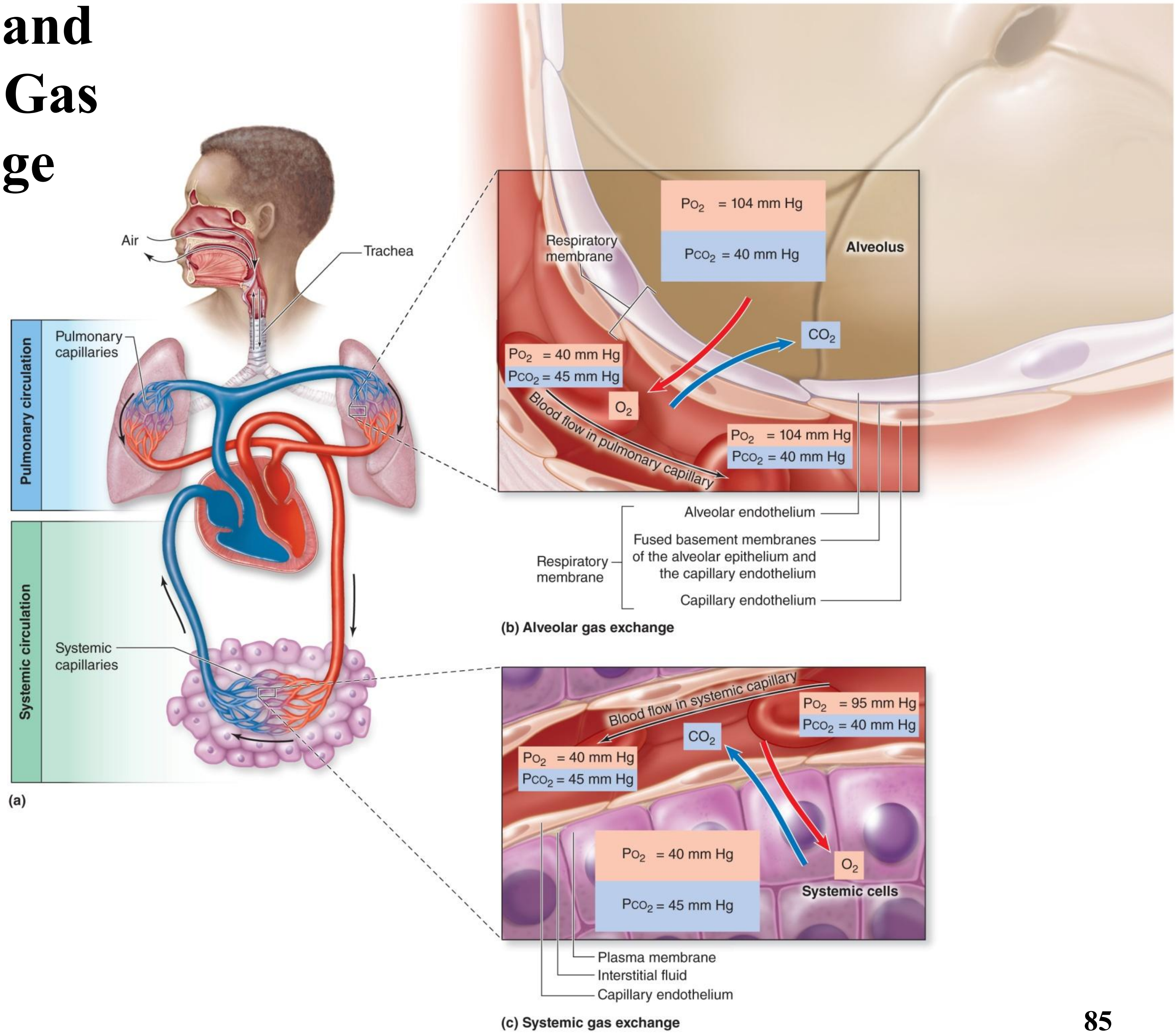


Figure 23.25

23.6a Chemical Principles of Gas Exchange

Gas solubility and Henry's law

- **Henry's law:** at a given temperature, the solubility of a gas in liquid is dependent upon the
  - Partial pressure of the gas in the air
  - Solubility coefficient of the gas in the liquid
- **Partial pressure:** driving force moving gas into liquid
  - Determined by total pressure and percentage of gas in the mixture
  - E.g., CO_2 is forced into soft drinks under high pressure
- **Solubility coefficient:** volume of gas that dissolves in a specified volume of liquid at a given temperature and pressure
  - A constant that depends upon interactions between molecules of the gas and liquid

23.6a Chemical Principles of Gas Exchange

Gas solubility and Henry's law (*continued*)

- Gases vary in their solubility in water
  - Carbon dioxide about 24 times as soluble as oxygen
  - Nitrogen about half as soluble as oxygen
    - It does not normally dissolve in blood in significant amounts
  - Gases with low solubility require larger pressure gradients to "push" the gas into the liquid

Clinical View: Decompression Sickness and Hyperbaric Oxygen Chambers

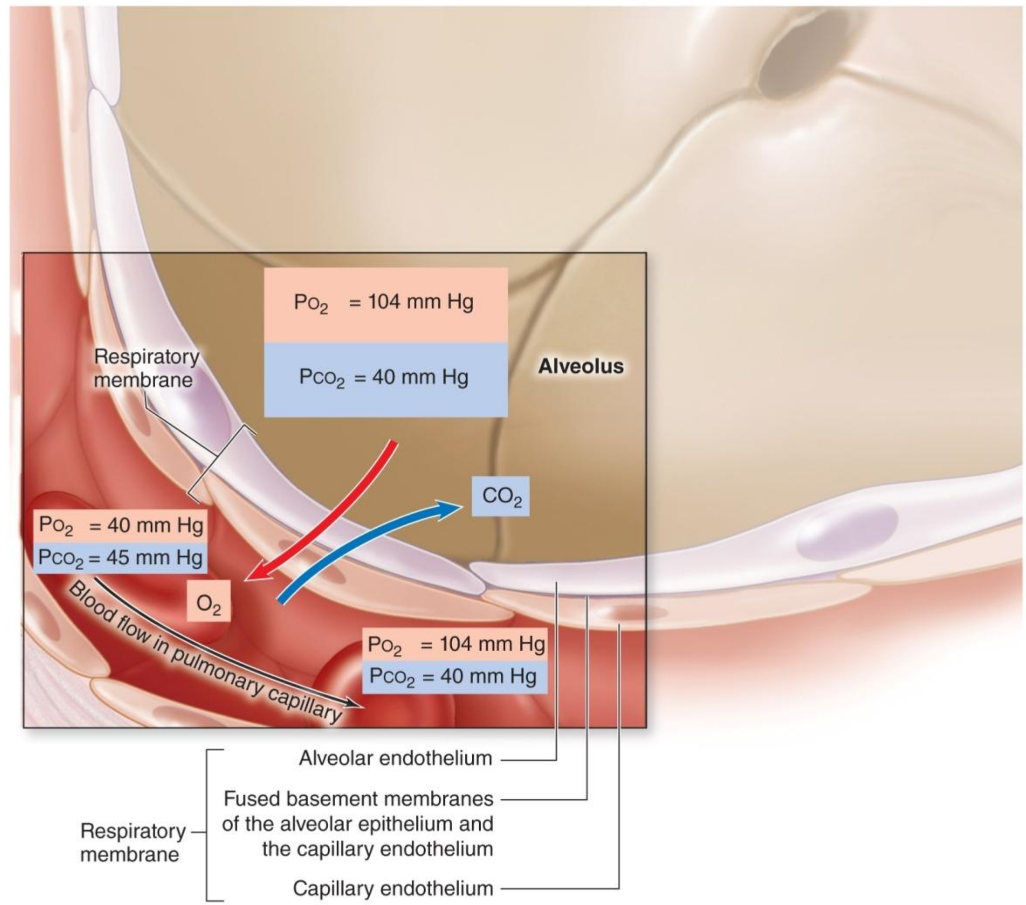
- **Decompression sickness** (*the bends*)
  - Occurs when a diver is submerged in water beyond a certain depth and returns too quickly to the surface
  - Nitrogen forced into the blood due to the higher pressure
  - Dissolved nitrogen bubbles out of solution while still in blood and tissues
  - Treated with **hyperbaric oxygen chambers**
    - Partial pressure gradient for oxygen increased
    - Additional oxygen can dissolve in blood plasma
    - Can be used for other disorders such as carbon monoxide poisoning

23.6b Alveolar Gas Exchange (External Respiration)

- Oxygen
  - PO_2 in alveoli is 104 mm Hg
  - PO_2 of blood entering pulmonary capillaries is 40 mm Hg
  - Oxygen diffuses across respiratory membrane from alveoli into the capillaries
  - Continues until blood PO_2 is equal to that of alveoli
  - Levels in alveoli remain constant as fresh air continuously enters

23.6b Alveolar Gas Exchange (External Respiration)

- Carbon dioxide
  - P_{CO_2} in alveoli 40 mm Hg
  - P_{CO_2} in blood of pulmonary capillaries 45 mm Hg
  - Carbon dioxide diffuses from blood to alveoli
  - Continues until blood levels equal alveoli levels
  - Levels in alveoli remain constant



(b) Alveolar gas exchange

Figure 23.25b

Clinical View: Emphysema

- Emphysema causes
  - Irreversible loss of pulmonary gas exchange surface area
  - Inflammation of air passageways distal to terminal bronchioles
  - Widespread destruction of pulmonary elastic connective tissue
  - Dilation and decreased total number of alveoli
  - Inability to expire effectively
- Emphysema is caused by
  - In most cases, smoking
  - Rarely from an alpha-1 antitrypsin deficiency

23.6b Alveolar Gas Exchange (External Respiration)

Efficiency of gas exchange at respiratory membrane

- Anatomical features of membrane contributing to efficiency
  - Large surface area (70 square meters)
  - Minimal thickness (0.5 micrometers)
- Physiologic adjustments: **ventilation-perfusion coupling**
  - Ability of bronchioles to regulate airflow and arterioles to regulate blood flow
    - Ventilation changes by bronchodilation or bronchoconstriction
      - E.g., dilation in response to increased P_{CO_2} in air in bronchiole
    - **Perfusion** changes by pulmonary arteriole dilation or constriction
      - E.g., dilation in response to either decreased P_{CO_2} or increased P_{O_2} in blood

Ventilation-Perfusion Coupling

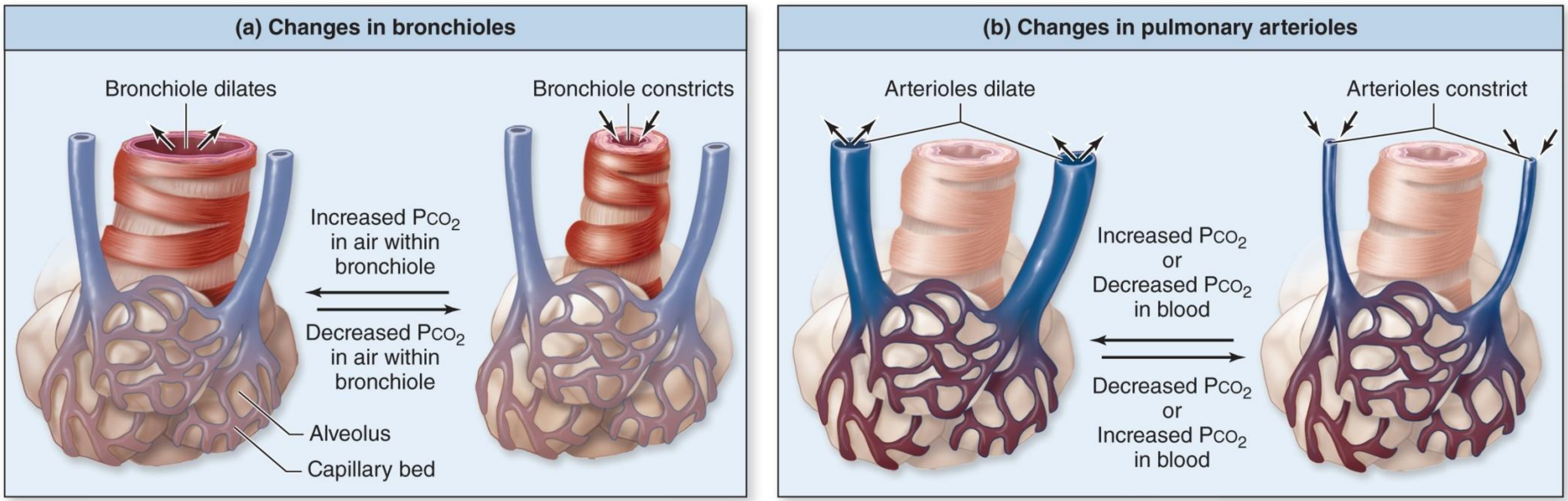


Figure 23.26

Clinical View: Respiratory Diseases and Efficiency of Alveolar Gas Exchange

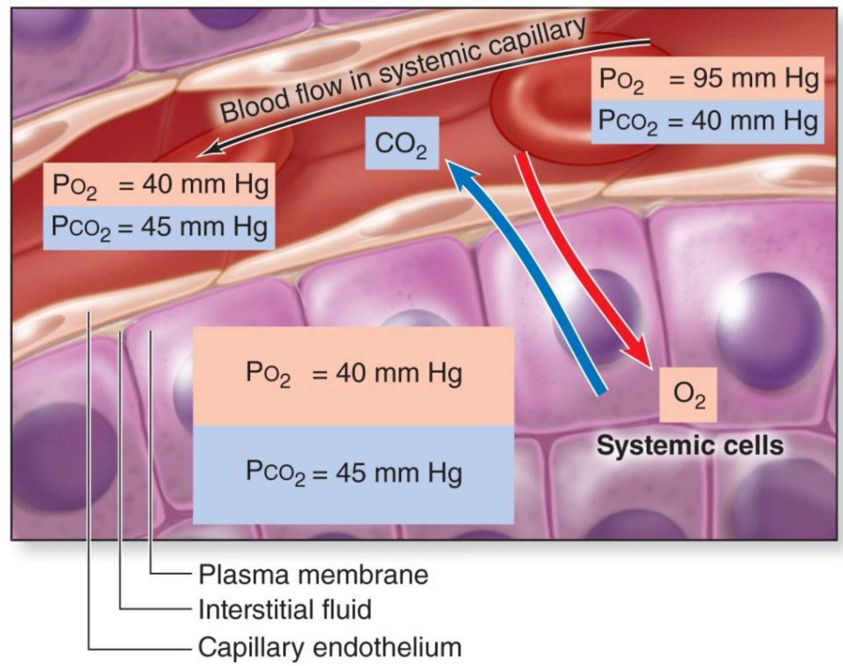
- Certain diseases decrease the efficiency of oxygen and carbon dioxide exchange
  – Due to decreased number of alveoli (e.g., lung cancer)
  – Due to thickened respiratory membrane (e.g., congestive heart failure)
  – Due to changes in ventilation-perfusion coupling (e.g., asthma or pulmonary embolism)
- Diseases result in decreased blood P_{O_2} and increased blood C_{O_2}

23.6c Systemic Gas Exchange (Internal Respiration)

- Oxygen diffuses out of systemic capillaries to enter systemic cells
  - Partial pressure gradient drives the process
    - PO_2 in systemic cells 40 mm Hg
    - PO_2 in systemic capillaries is 95 mm Hg
  - Continues until blood PO_2 is 40 mm Hg
  - Systemic cell PO_2 stays fairly constant
    - Oxygen delivered at same rate it is used unless engaging in strenuous activity

23.6c Systemic Gas Exchange (Internal Respiration)

- Carbon dioxide
  - Diffuses from systemic cells to blood
  - Partial pressure gradient driving process
    - P_{CO_2} in systemic cells 45mm Hg
    - P_{CO_2} in systemic capillaries 40 mm Hg
  - Diffusion continuing until blood P_{CO_2} is 45 mm Hg



(c) Systemic gas exchange

Figure 23.25c

Integration of Gas Exchange

- Alveolar gas exchange decreases blood PCO_2, whereas systemic gas exchange increases it
- Alveolar gas exchange increases blood PO_2, whereas systemic gas exchange decreases it
  - As blood leaves pulmonary capillaries PO_2 is 104 mm Hg
  - Mingling with deoxygenated blood from bronchial veins (in pulmonary veins) results in a PO_2 of 95 mm Hg in left heart

Table 23.5 Gas Exchange[1]

Characteristic	Alveolar Gas Exchange	Systemic Gas Exchange
Definition	Exchange of respiratory gases between alveoli in lungs and blood in pulmonary capillaries	Exchange of respiratory gases between systemic cells and blood in systemic capillaries
Changes in Blood PO_2	Blood PO_2 increases from 40 to 104 mm Hg	Blood Po2 decreases from 95 to 40 mm Hg
Changes in Blood PCO_2	Blood PCO_2 decreases from 45 to 40 mm Hg	Blood PCO_2 increases from 40 to 45 mm Hg

1. General conditions: Location is at sea level and individual is at rest.

What did you learn?

- What is a partial pressure?
- How does Henry's law relate to human respiration?
- What are the partial pressure gradients for the respiratory gases in the alveoli?
- What anatomical features of the respiratory membrane foster gas exchange?

23.7 Respiration: Gas Transport

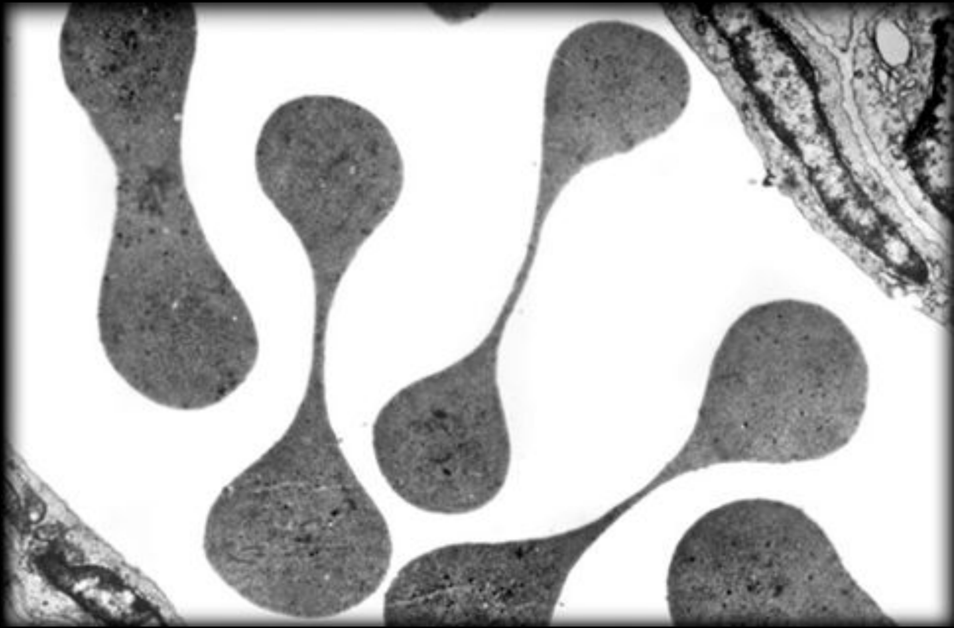
Learning Objectives:

1. Explain why hemoglobin is essential to oxygen transport.
2. Describe the three ways carbon dioxide is transported in the blood.
3. Explain the conversion of CO_2 to and from HCO_3^- within erythrocytes.
4. Name the three substances carried by hemoglobin.
5. Explain the significance of the oxygen-hemoglobin saturation curve for both alveolar and systemic gas exchange.

23.7a Oxygen Transport

- Blood's ability to transport oxygen depends on
  - Solubility coefficient of oxygen
    - This is very low, and so very little oxygen dissolves in plasma
  - Presence of hemoglobin
    - The iron of hemoglobin attaches oxygen
    - About 98% of O_2 in blood is bound to hemoglobin
      - HbO_2 is **oxyhemoglobin** (with oxygen bound)
      - HHb is **deoxyhemoglobin** (without bound oxygen)

Erythrocytes



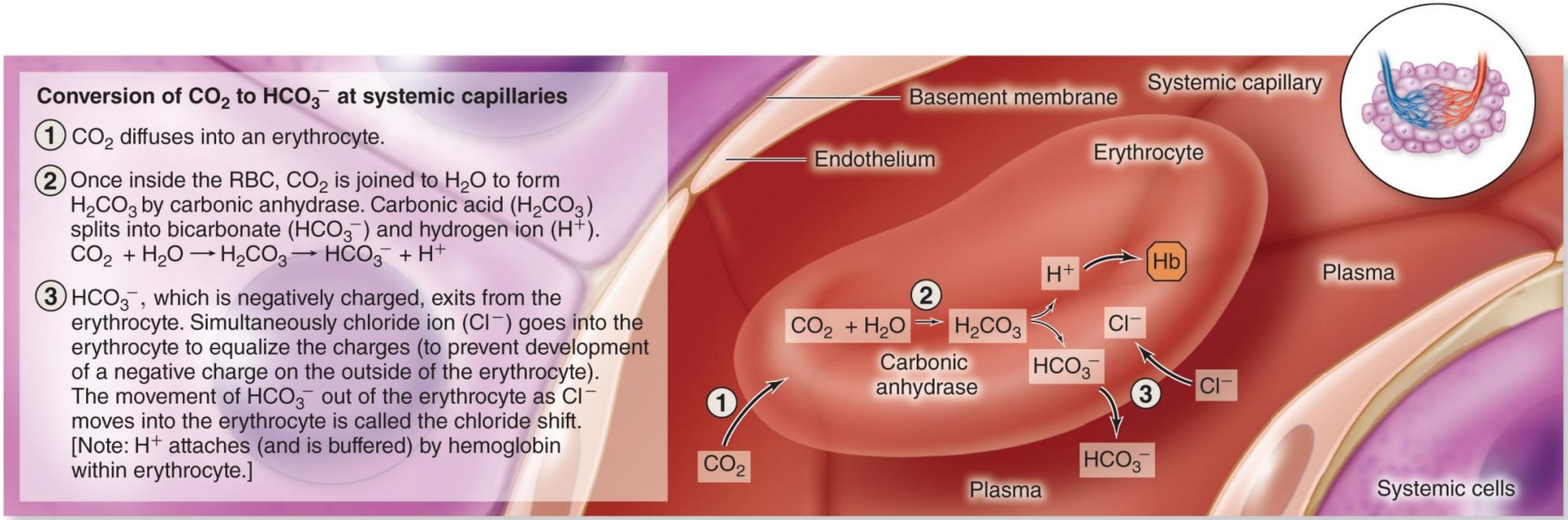
Clinical View: Measuring Blood Oxygen Levels with a Pulse Oximeter

- Noninvasive and indirect way to measure oxygen
  - Applied to finger or earlobe
- Measure hemoglobin saturation by determining the ratio of oxyhemoglobin to deoxyhemoglobin
- Normal reading hemoglobin saturation >95%

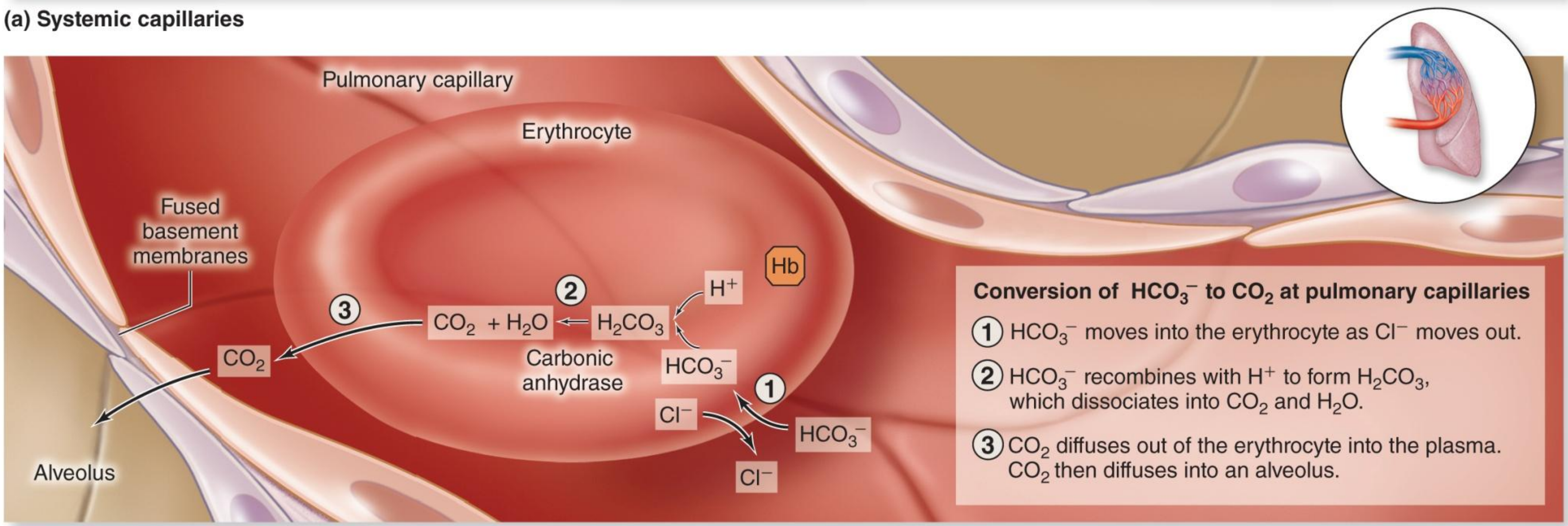
23.7b Carbon Dioxide Transport

- Carbon dioxide has three means of transport
  - As CO_2 dissolved in plasma (7%)
  - As CO_2 attached to amine group of globin portion of hemoglobin (23%)
    - $HbCO_2$ is carbaminohemoglobin
  - As bicarbonate dissolved in plasma (70%)
    - CO_2 diffuses into erythrocytes and combines with water to form bicarbonate and hydrogen ion
    - Bicarbonate diffuses into plasma
    - CO_2 is regenerated when blood moves through pulmonary capillaries and the process is reversed

Conversion of CO_2 to Bicarbonate (Figure 23.27)



(a) Systemic capillaries



(b) Pulmonary capillaries

23.7c Hemoglobin as a Transport Molecule

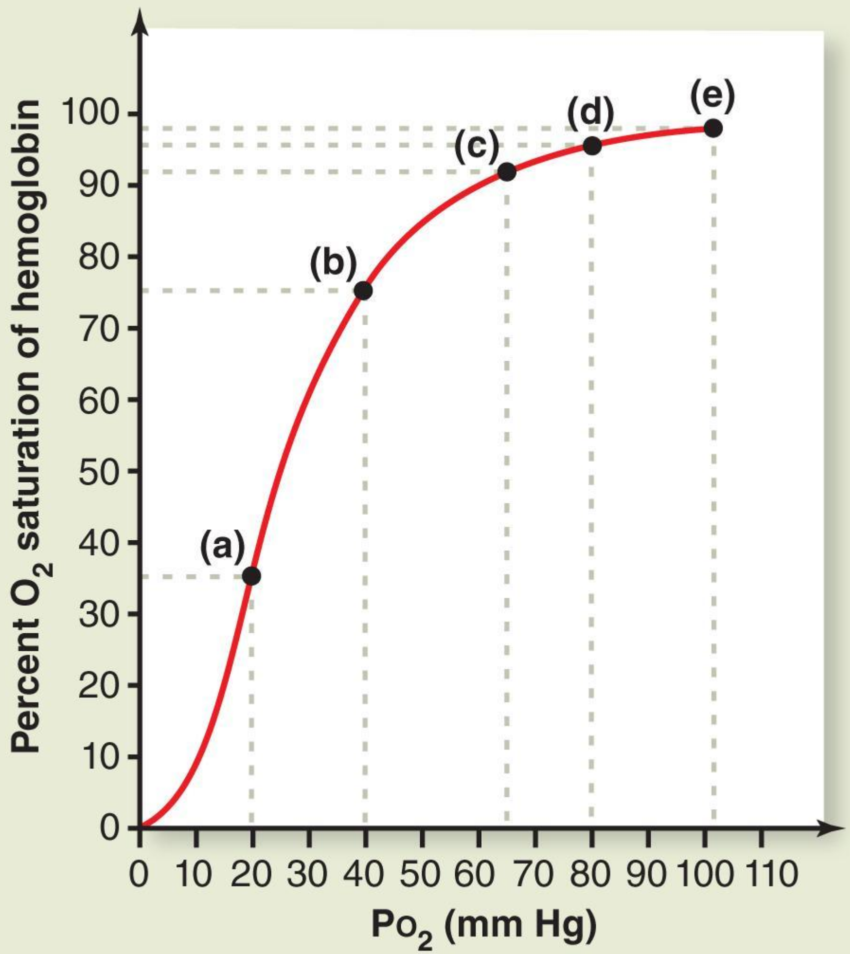
- Hemoglobin transports
  - Oxygen attached to iron
  - Carbon dioxide bound to globin part
  - Hydrogen ions bound to globin part
- Binding of one substance causes a change in shape of the hemoglobin molecule
  - Influences the ability of hemoglobin to bind or release the other two substances

23.7c Hemoglobin as a Transport Molecule

Oxygen-hemoglobin saturation curve

- Each hemoglobin can bind up to four O_2 molecules
  - One on each iron atom in the hemoglobin molecule
- Percent O_2 saturation of hemoglobin is crucial
  - It is the amount of oxygen bound to available hemoglobin
  - Saturation increases as P_{O_2} increases
    - **Cooperative binding effect:** each O_2 that binds causes a change in hemoglobin making it easier for next O_2 to bind
  - Graphed in the **oxygen-hemoglobin saturation curve**
    - S-shaped, nonlinear relationship

Oxygen-Hemoglobin Saturation Curve



Po_2 (mm Hg)	% O_2 Saturation of Hb
10	14
20	35
30	57
40	75
50	85
60	90
70	93
80	95
90	97
100	98
105	98

Figure 23.28

23.7c Hemoglobin as a Transport Molecule

Oxygen-hemoglobin saturation curve (*continued*)

- Large changes in saturation occur with small increases of P_{O_2} at lower partial pressures (i.e., curve is initially steep)
- At P_{O_2} higher than 60 mm Hg only small changes in saturation occur
  - About 90% saturation at 60 mm Hg
- Hemoglobin saturation is about 98% at pulmonary capillaries as P_{O_2} is 104 mm Hg
- Saturation can only reach 100% at pressures above 1 atm (e.g., in hyperbaric oxygen chambers)

23.7c Hemoglobin as a Transport Molecule

Oxygen-hemoglobin saturation curve (*continued*)

- Can use graph to determine saturation at a given P_{O_2}
  - At 5000 ft, alveolar P_{O_2} is 81 mm Hg
    - Corresponds to a hemoglobin saturation of 95%
  - At 17,000 ft, alveolar P_{O_2} is 40 mm Hg
    - Corresponds to a hemoglobin saturation of 75%
- **Altitude sickness**
  - Adverse physiologic effects from a decrease in alveolar P_{O_2} and low oxygen saturation
  - Includes symptoms of headache, nausea, pulmonary edema, and cerebral edema

23.7c Hemoglobin as a Transport Molecule

Oxygen-hemoglobin saturation curve (*continued*)

- Some (not all) oxygen released from hemoglobin at systemic capillaries
  - 98% saturation as it leaves the lungs (at sea level)
  - About 75% saturation after passing systemic cells at rest
    - Only 20–25% of transported oxygen is released
  - **Oxygen reserve:** O_2 remaining bound to hemoglobin after passing through systemic circulation
    - Provides a means for additional oxygen to be delivered under increased metabolic demands (e.g., exercise)
    - Vigorous exercise produces a significant drop in saturation
      - Blood leaving capillaries in active muscles only about 35% saturated

23.7c Hemoglobin as a Transport Molecule

Other variables that influence oxygen release from hemoglobin during systemic exchange

- Temperature
  - Elevated temperature diminishes hemoglobin's hold on oxygen
- H^+ binding to hemoglobin
  - Hydrogen ion binds to hemoglobin and causes a conformational change
  - This causes decreased affinity for O_2 and oxygen release
    - Called the **Bohr effect**

23.7c Hemoglobin as a Transport Molecule

Other variables that influence oxygen release from hemoglobin during systemic exchange (*continued*)

- **Presence of 2,3-BPG:** a molecule in erythrocytes
  - Molecule binds hemoglobin, causing release of additional oxygen
  - Certain hormones stimulate erythrocytes to produce 2,3-BPG
    - Thyroid, epinephrine, growth hormone, and testosterone
- **CO_2 binding to hemoglobin**
  - Binding causes release of more oxygen from hemoglobin
- **Haldane effect**
  - Release of oxygen causes a conformational change in hemoglobin
  - Conformational change increases the amount of carbon dioxide that can bind

23.7c Hemoglobin as a Transport Molecule

Other variables that influence oxygen release from hemoglobin during systemic exchange (*continued*)

- Shifts to the saturation curve
  - Some variables decrease oxygen affinity for hemoglobin
    - Known as a **shift right**
    - E.g., increased temperature, increase in hydrogen ion
  - Other variables increase oxygen affinity to hemoglobin
    - Known as a **shift left**
    - E.g., decreased temperature, decrease in hydrogen ion

Hemoglobin and Oxygen Release

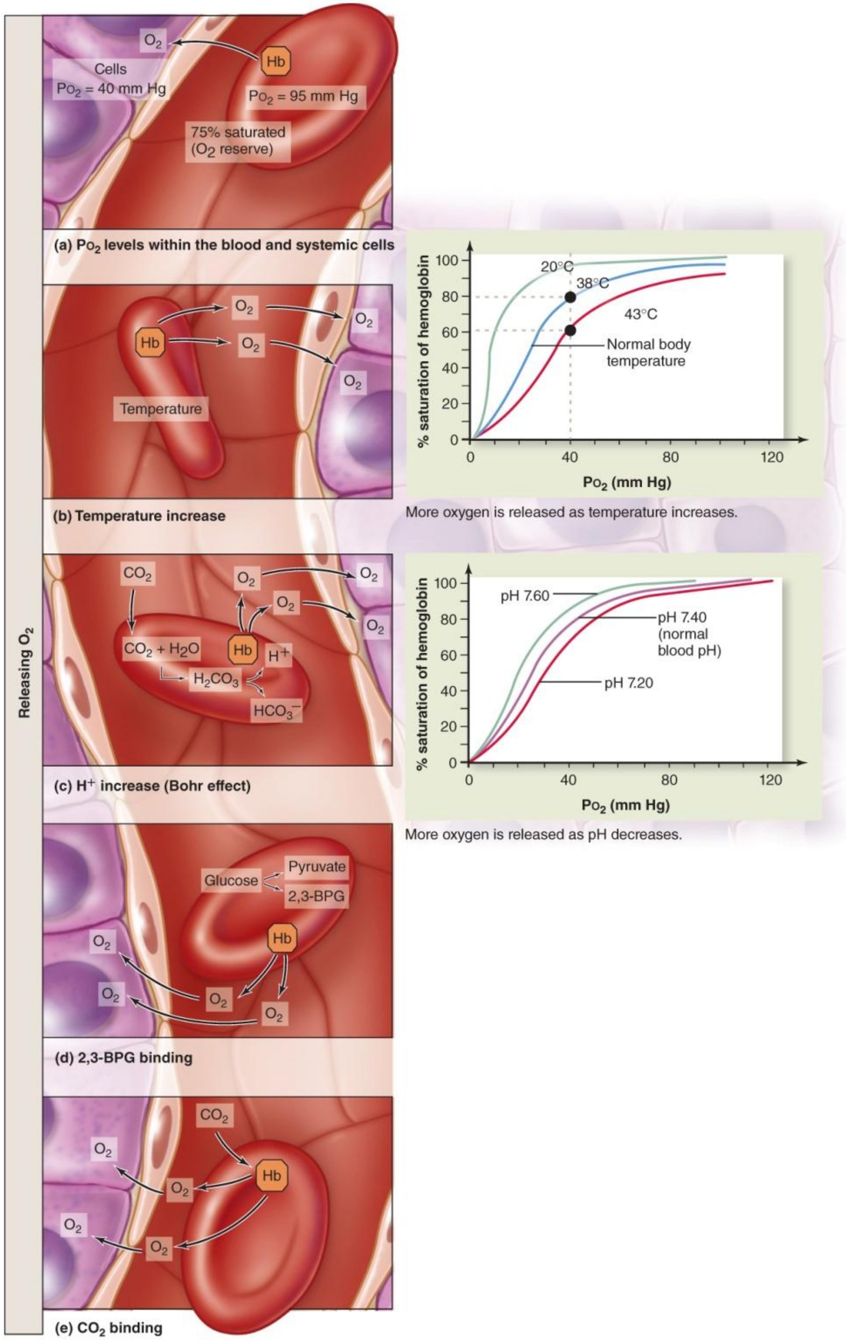


Figure 23.29

Summary of Respiration

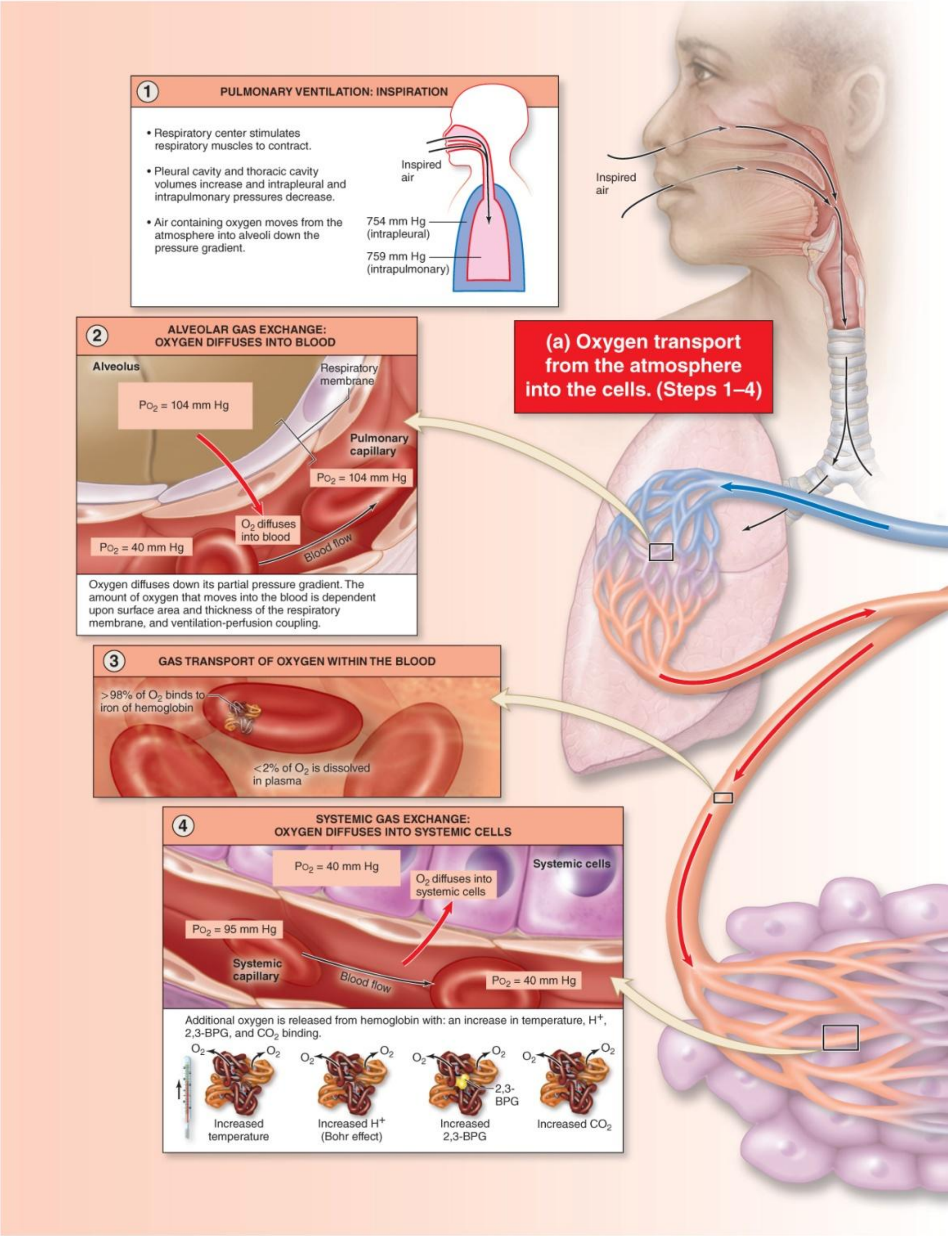


Figure 23.30a

Summary of Respiration

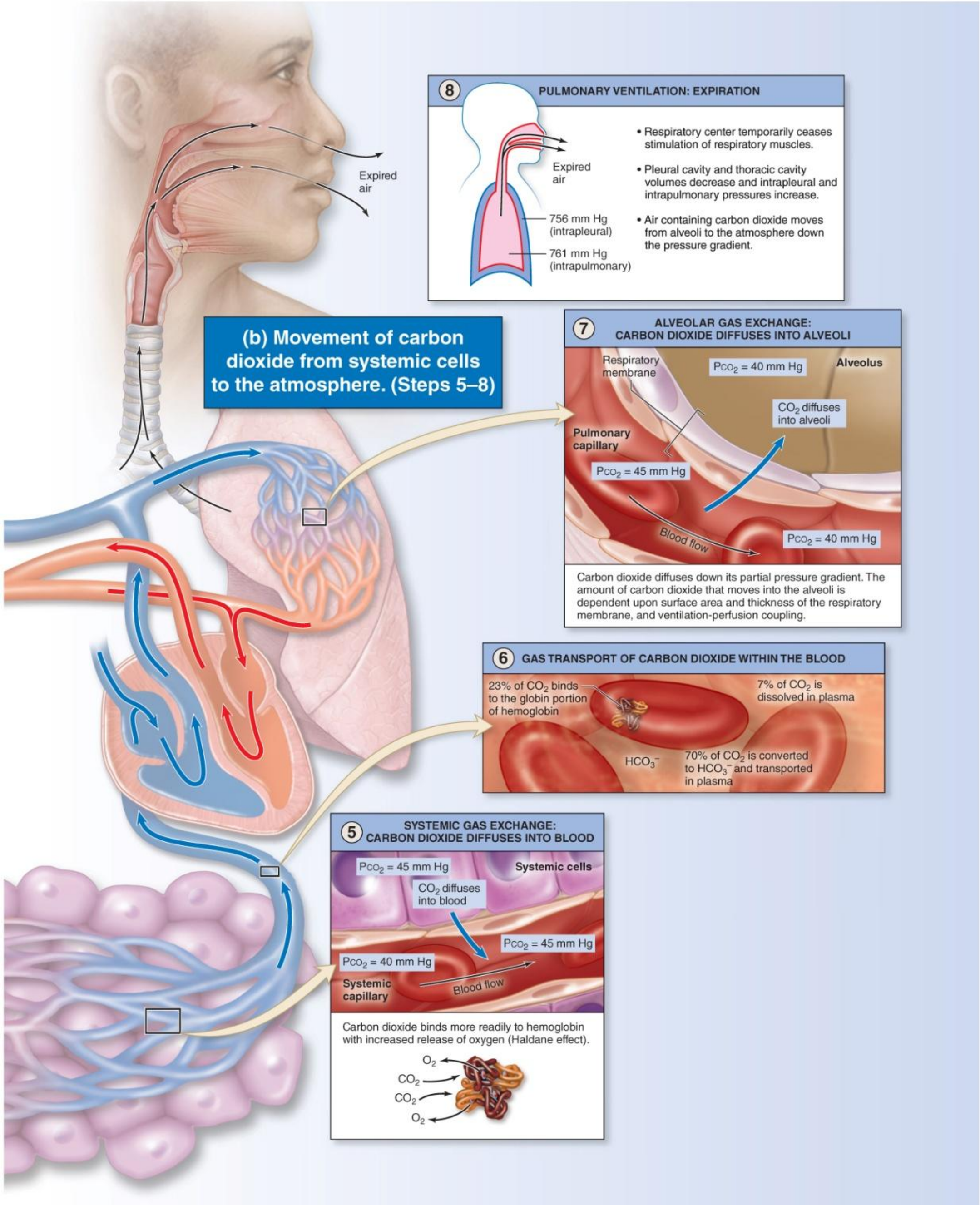


Figure 23.30b

Clinical View: Fetal Hemoglobin and Physiologic Jaundice

- Unborn babies have a different type of hemoglobin molecule
  - **Fetal hemoglobin** has a greater affinity for binding oxygen than adult hemoglobin
  - This ensures a net movement of oxygen from the blood of the mother to the blood of the fetus
  - Infants may have **physiologic jaundice** as fetal hemoglobin breaks down
    - Yellowish tinge to skin due to elevated levels of bilirubin

What did you learn?

- Why is so little O_2 dissolved in plasma?
- How is most CO_2 transported in blood?
- What does increased temperature do to hemoglobin's hold on oxygen?
- What sort of shift to the saturation curve is caused by factors that decrease the affinity between O_2 and Hb?

23.8 Breathing Rate and Homeostasis

Learning Objectives:

1. Explain how hyperventilation and hypoventilation influence the chemical composition of blood.
2. Describe how breathing rate and depth effects venous return of blood and lymph.
3. Explain the changes in breathing that accompany exercise.

23.8a Effects of Hyperventilation and Hypoventilation on Cardiovascular Function

- **Hyperventilation:** breathing rate or depth above body's demand
  - Caused by anxiety, ascending to high altitude, or voluntarily
  - PO_2 rises and PCO_2 fall in the air of alveoli
  - Additional oxygen does not enter blood because hemoglobin is already 98% saturated
  - There is greater loss of CO_2 from blood, called **hypocapnia**

23.8a Effects of Hyperventilation and Hypoventilation on Cardiovascular Function

- **Hyperventilation** (*continued*)
  - Low blood CO_2 causes vasoconstriction
    - Brain vessel constriction can decrease oxygen delivery to the brain
  - May decrease blood hydrogen ion concentration
    - If buffers cannot compensate, result is **respiratory alkalosis**
  - Hyperventilation may cause
    - Feeling faint or dizzy, numbness, tingling, cramps, and tetany
    - If prolonged, disorientation, loss of consciousness, coma, possible death

23.8a Effects of Hyperventilation and Hypoventilation on Cardiovascular Function

- **Hypoventilation:** breathing too slow **(bradypnea)** or too shallow **(hypopnea)**
  - Causes include: airway obstruction, pneumonia, brainstem injury, other respiratory conditions
  - O_2 levels down, CO_2 levels up in alveoli
  - Blood P_{O_2} decreases **(hypoxemia);** and can lead to low oxygen in tissues **(hypoxia)**
  - Blood P_{CO_2} increases **(hypercapnia)**

23.8a Effects of Hyperventilation and Hypoventilation on Cardiovascular Function

- **Hypoventilation** (*continued*)
  - May result in inadequate oxygen delivery
  - May result in increased hydrogen ion concentration due to high blood P_{CO_2}
    - Might result in respiratory acidosis
  - May cause
    - Lethargy, sleepiness, headache, polycythemia, cyanotic tissues
    - If prolonged, convulsions, loss of consciousness, death

23.8b Breathing and Exercise

- While exercising, breathing shows **hyperpnea** to meet increased tissue needs
  - Breathing depth increases while rate remains the same
- Blood PO_2 and Blood PCO_2 remain relatively constant
  - Increased cellular respiration compensated for by deeper breathing, increased cardiac output, greater blood flow
  - The respiratory center is stimulated from one or more causes
    - Proprioceptive sensory signals in response to movement
    - Corrollary motor output from cerebral cortex relayed to respiratory center
    - Conscious anticipation of exercise

What did you learn?

- If hyperventilation is excessive, what change occurs in blood pH?
- What distinguishes hyperventilation from hyperpnea?
- What happens to blood levels of oxygen and carbon dioxide during exercise?