Respiratory Volumes

- Tidal volume (TV) – air that moves into and out of the lungs with each breath (approximately 500 ml)
- Inspiratory reserve volume (IRV) – air that can be inspired forcibly beyond the tidal volume (2100–3200 ml)
- Expiratory reserve volume (ERV) – air that can be evacuated from the lungs after a tidal expiration (1000–1200 ml)
- Residual volume (RV) – air left in the lungs after strenuous expiration (1200 ml). Keeps alveoli open and prevents collapse
Respiratory Capacities

- Inspiratory capacity (IC) – total amount of air that can be inspired after a tidal expiration (IRV + TV)
- Functional residual capacity (FRC) – amount of air remaining in the lungs after a tidal expiration (RV + ERV)
- Vital capacity (VC) – the total amount of exchangeable air (TV + IRV + ERV)
- Total lung capacity (TLC) – sum of all lung volumes (approximately 6000 ml in males)
Dead Space

- Anatomical dead space – Volume of conducting zones where air never contributes to gas exchange, e.g. 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
Pulmonary Function Tests

- Spirometer – an instrument consisting of a hollow bell inverted over water, used to evaluate respiratory function

- Spirometry can distinguish between:
  - Obstructive pulmonary disease – increased airway resistance
  - Restrictive disorders – reduction in total lung capacity from structural or functional lung changes
Pulmonary Function Tests

- Total ventilation – total amount of gas flow into or out of the respiratory tract in one minute
- Forced vital capacity (FVC) – gas forcibly expelled after taking a deep breath
- Forced expiratory volume (FEV) – the amount of gas expelled during specific time intervals of the FVC
Pulmonary Function Tests

- Increases in TLC, FRC, and RV may occur as a result of obstructive disease.
- Reduction in VC, TLC, FRC, and RV result from restrictive disease.
Alveolar Ventilation

- Alveolar ventilation rate (AVR) – measures the flow of fresh gases into and out of the alveoli during a particular time

\[
AVR = \text{frequency} \times X \times (TV - \text{dead space})
\]

- Slow, deep breathing increases AVR and rapid, shallow breathing decreases AVR
Nonrespiratory Air Movements

- Most result from reflex action
- Examples include: coughing, sneezing, crying, laughing, hiccupping, and yawning
Basic Properties of Gases: Dalton’s Law of Partial Pressures

- Total pressure exerted by a mixture of gases is the sum of the pressures exerted independently by each gas in the mixture.

- The partial pressure of each gas is directly proportional to its percentage in the mixture.
Basic Properties of Gases: Henry’s Law

- When a mixture of gases is in contact with a liquid, each gas will dissolve in the liquid in proportion to its partial pressure.
- The amount of gas that will dissolve in a liquid also depends upon its solubility:
  - Carbon dioxide is the most soluble.
  - Oxygen is \(1/20\)th as soluble as carbon dioxide.
  - Nitrogen is practically insoluble in plasma.
  - Thus, more CO2 than O2 dissolves in a liquid and virtually no N2 goes into solution.
  - Increasing temperature decreases gas solubility.
Composition of Alveolar Gas

- The atmosphere is mostly oxygen and nitrogen, while alveoli contain more carbon dioxide and water vapor.

- These differences result from:
  - Gas exchanges in the lungs – oxygen diffuses from the alveoli and carbon dioxide diffuses into the alveoli.
  - Humidification of air by conducting passages.
  - The mixing of alveolar gas that occurs with each breath.
  - Thus, high AVR brings in more O2 increasing $P_{O_2}$ and rapidly eliminates CO2 from the lungs.
External Respiration: Pulmonary Gas Exchange

- Factors influencing the movement of oxygen and carbon dioxide across the respiratory membrane
  - Partial pressure gradients and gas solubilities
  - Matching of alveolar ventilation and pulmonary blood perfusion
  - Structural characteristics of the respiratory membrane
Partial Pressure Gradients and Gas Solubilities

- The partial pressure oxygen (PO$_2$) of venous blood is 40 mm Hg; the partial pressure in the alveoli is 104 mm Hg
  - This steep gradient allows oxygen partial pressures to rapidly reach equilibrium (in 0.25 seconds)
  - Blood can move three times as quickly (0.75 seconds) through the pulmonary capillary and still be adequately oxygenated
Partial Pressure Gradients and Gas Solubilities

- The partial pressure carbon dioxide (PCO$_2$) of venous blood is 45 mm Hg; the partial pressure in the alveoli is 40 mm Hg
- CO$_2$ travels down the gradient into the alveoli
- Although carbon dioxide has a lower partial pressure gradient:
  - It is 20 times more soluble in plasma than oxygen
  - It diffuses in equal amounts with oxygen
Figure 22.17

Inspired air:
\[ P_{O_2} = 160 \text{ mm Hg} \]
\[ P_{CO_2} = 0.3 \text{ mm Hg} \]

Expired air:
\[ P_{O_2} = 120 \text{ mm Hg} \]
\[ P_{CO_2} = 27 \text{ mm Hg} \]

Alveoli of lungs:
\[ P_{O_2} = 104 \text{ mm Hg} \]
\[ P_{CO_2} = 40 \text{ mm Hg} \]

Blood entering alveolar capillaries:
\[ P_{O_2} = 40 \text{ mm Hg} \]
\[ P_{CO_2} = 45 \text{ mm Hg} \]

Blood leaving alveolar capillaries:
\[ P_{O_2} = 104 \text{ mm Hg} \]
\[ P_{CO_2} = 40 \text{ mm Hg} \]

Pulmonary veins (\( P_{O_2} = 100 \text{ mm Hg} \))

Blood leaving tissue capillaries:
\[ P_{O_2} = 40 \text{ mm Hg} \]
\[ P_{CO_2} = 45 \text{ mm Hg} \]

Blood entering tissue capillaries:
\[ P_{O_2} = 100 \text{ mm Hg} \]
\[ P_{CO_2} = 40 \text{ mm Hg} \]

Tissues:
\[ P_{O_2} \text{ less than } 40 \text{ mm Hg} \]
\[ P_{CO_2} \text{ greater than } 45 \text{ mm Hg} \]
Oxygenation of Blood

Figure 22.18

The graph illustrates the change in oxygen tension ($P_O_2$) over time as a blood capillary moves through the pulmonary capillary bed. The $P_O_2$ starts at 0 and increases to 104 mm Hg, the saturation level in the pulmonary capillary. The time scale indicates the progression from the start to the end of the capillary.
Ventilation-Perfusion Coupling

- Ventilation – the amount of gas reaching the alveoli
- Perfusion – the blood flow reaching the alveoli
- Matching amounts of gas reaching alveoli and blood flow in pulmonary capillaries
- Local autoregulatory mechanisms respond to alveoli conditions
- Low PO2 results in arteriole constriction to redirect blood flow to alveoli that are more efficient in gas exchange
- High PO2 results in arteriole dilation increasing blood flow to capillary bed
Ventilation-Perfusion Coupling

- Changes in $P_{CO_2}$ in the alveoli cause changes in the diameters of the bronchioles
  - Passageways servicing areas where alveolar carbon dioxide is high dilate allowing CO2 to be eliminated rapidly
  - Those serving areas where alveolar carbon dioxide is low constrict
Ventilation-Perfusion Coupling

- However, this does not balance ventilation-perfusion in every alveoli because:
  
  - Gravity causes regional variations in blood and air flow
  
  - Unventilated areas arise due to mucus plugged alveolar ducts
Ventilation-Perfusion Coupling

Reduced alveolar ventilation; excessive perfusion
Reduced alveolar ventilation; reduced perfusion
Enhanced alveolar ventilation; inadequate perfusion
Enhanced alveolar ventilation; enhanced perfusion

Pulmonary arterioles serving these alveoli constrict
Pulmonary arterioles serving these alveoli dilate

$P_{O2}$ in alveoli
$P_{CO2}$ in alveoli

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Figure 22.19
Internal Respiration: Capillary Gas Exchange in Body Tissues

- The factors promoting gas exchange between systemic capillaries and tissue cells are the same as those acting in the lungs
  - The partial pressures and diffusion gradients are reversed
  - \( P_{O_2} \) in tissue is always lower than in systemic arterial blood (40 vs. 100 mm Hg)
  - Thus, \( O_2 \) moves rapidly from blood to tissues until equilibrium is met
  - \( CO_2 \) is the reverse
Oxygen Transport

- Molecular oxygen is carried in the blood:
  - Bound to hemoglobin (Hb) w/i RBCs (98.5%)
  - Dissolved in plasma (1.5%)
Oxygen Transport: Role of Hemoglobin

- Each Hb molecule binds four oxygen atoms in a rapid and reversible process.

- The hemoglobin-oxygen combination is called oxyhemoglobin (HbO$_2$).

- Hemoglobin that has released oxygen is called reduced hemoglobin or deoxyhemoglobin (HHb).

\[
\text{Lungs} \\
\text{HHb} + \text{O}_2 \rightleftharpoons \text{HbO}_2 + \text{H}^+ \\
\text{Tissues}
\]
Hemoglobin (Hb)

- Saturated hemoglobin – when all four hemes of the molecule are bound to oxygen

- Partially saturated hemoglobin – when one to three hemes are bound to oxygen
  
  - 1st O2 binds resulting in a conformational change in Hb allowing 2 more O2 to bind which results in yet another conformational change in Hb allowing 4th O2 to bind (fully saturated)
  
  - Unloading of O2 works the same way via affinity binding with the last bound O2 being the first released and so on
Hemoglobin (Hb)

- The rate that hemoglobin binds and releases oxygen is regulated by:
  - $P_{O_2}$, temperature, blood pH, $P_{CO_2}$, and the concentration of BPG (an organic chemical)
  - These factors ensure adequate delivery of oxygen to tissue cells
Influence of $P_{O_2}$ on Hemoglobin Saturation

- Hemoglobin saturation plotted against $P_{O_2}$ produces a oxygen-hemoglobin dissociation curve.

- 98% saturated arterial blood contains 20 ml oxygen per 100 ml blood (20 vol %).

- As arterial blood flows through capillaries, 5 ml oxygen are released.
  - 75-80% remains bound as the venous reserve that can be unloaded during vigorous exercise.
Hemoglobin Saturation Curve

- Hemoglobin is almost completely saturated at a \( P_{O_2} \) of 70 mm Hg
- Further increases in \( P_{O_2} \) produce only small increases in oxygen binding
- Oxygen loading and delivery to tissue is adequate when \( P_{O_2} \) is below normal levels
Hemoglobin Saturation Curve

- Only 20–25% of bound oxygen is unloaded during one systemic circulation

- If oxygen levels in tissues drop:
  - More oxygen dissociates from hemoglobin and is used by cells
  - Respiratory rate or cardiac output need not increase
Hemoglobin Saturation Curve

Figure 22.20
Other Factors Influencing Hemoglobin Saturation

- Temperature, H⁺, PCO₂, and BPG (2,3-bisphosphoglycerate)
  - Note: BPG binds reversibly with Hb and is produced by RBCs as they breakdown glucose by glycolysis
  - Modify the structure of hemoglobin and alter its affinity for oxygen
  - Increases of these factors:
    - Decrease hemoglobin’s affinity for oxygen
    - Enhance oxygen unloading from the blood
  - Decreases act in the opposite manner
- These parameters are all high in systemic capillaries where oxygen unloading is the goal
Other Factors Influencing Hemoglobin Saturation

Figure 22.21

(a) Normal body temperature

(b) Decreased carbon dioxide ($P_{CO_2}$ 20 mm Hg) or $H^+$ (pH 7.6)

Increased carbon dioxide ($P_{CO_2}$ 80 mm Hg) or $H^+$ (pH 7.2)

Normal arteriole carboxylic acid ($P_{CO_2}$ 40 mm Hg) or $H^+$ (pH 7.4)
Factors That Increase Release of Oxygen by Hemoglobin

- 1) Increase in temperature, PCO2 and BPG
- 2) And decrease in pH
  - All decrease Hb’s affinity for O2
  - Causes curve to shift to the right thus enhancing O2 unloading from the blood
- When the above items in 1 and 2 reverse, curve shifts to the left
Hemoglobin-Nitric Oxide Partnership

- Nitric oxide (NO) is a vasodilator secreted by vascular endothelial cells that plays a role in blood pressure regulation.
- Hemoglobin is a NO scavenger (heme group destroys NO) resulting in vasoconstriction.
- However, blood vessels dilate at site of gas exchange. Why?
  - Hb transports O2 and NO (attached to globin)
  - NO unloads and causes vasodilation.
- As deoxygenated hemoglobin picks up carbon dioxide, it also binds nitric oxide and carries these gases to the lungs for unloading.
Carbon monoxide

- CO competes with O2 for heme sites
- CO has 200x greater affinity than O2 for heme sites
- The result is hypoxia: inadequate O2 delivery to tissues
Carbon Dioxide Transport

- Carbon dioxide is transported in the blood in three forms
  - Dissolved in plasma – 7 to 10%
  - Chemically bound to hemoglobin – 20% is carried in RBCs as carbaminohemoglobin
  - Bicarbonate ion in plasma – 70% is transported as bicarbonate (HCO$_3^-$)
Transport and Exchange of Carbon Dioxide

- Carbon dioxide diffuses into RBCs and combines with water to form carbonic acid ($H_2CO_3$), which quickly dissociates into hydrogen ions and bicarbonate ions.

<table>
<thead>
<tr>
<th>CO$_2$</th>
<th>+</th>
<th>H$_2$O</th>
<th>$\leftrightarrow$</th>
<th>H$_2$CO$_3$</th>
<th>$\leftrightarrow$</th>
<th>H$^+$</th>
<th>+</th>
<th>HCO$_3^-$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Carbon dioxide</td>
<td></td>
<td>Water</td>
<td></td>
<td>Carbonic acid</td>
<td></td>
<td>Hydrogen ion</td>
<td></td>
<td>Bicarbonate ion</td>
</tr>
</tbody>
</table>

- In RBCs, carbonic anhydrase reversibly catalyzes the conversion of carbon dioxide and water to carbonic acid.
Transport and Exchange of Carbon Dioxide

(a) Oxygen release and carbon dioxide pickup at the tissues
Transport and Exchange of Carbon Dioxide

- At the tissues:
  - Bicarbonate quickly diffuses from RBCs into the plasma
  - The chloride shift – to counterbalance the outrush of negative bicarbonate ions from the RBCs, chloride ions ($Cl^-$) move from the plasma into the RBCs
Transport and Exchange of Carbon Dioxide

- At the lungs, these processes are reversed
  - Bicarbonate ions move into the RBCs and bind with hydrogen ions to form carbonic acid
  - Carbonic acid is then split by carbonic anhydrase to release carbon dioxide and water
  - Carbon dioxide then diffuses from the blood into the alveoli
Transport and Exchange of Carbon Dioxide

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

- The amount of carbon dioxide transported is markedly affected by the $P_{O_2}$

- Haldane effect – the lower the $P_{O_2}$ and hemoglobin saturation with oxygen, the more carbon dioxide can be carried in the blood
Haldane Effect

- At the tissues, as more carbon dioxide enters the blood:
  - More oxygen dissociates from hemoglobin (Bohr effect)
  - More carbon dioxide combines with hemoglobin, and more bicarbonate ions are formed
- This situation is reversed in pulmonary circulation
Haldane Effect

Figure 22.23

- O₂ saturation of Hb = 0%
- O₂ saturation of Hb = 100%

Blood CO₂ content (ml/100 ml)

P CO₂ (mm Hg)
Influence of Carbon Dioxide on Blood pH

- The carbonic acid–bicarbonate buffer system resists blood pH changes
- If hydrogen ion concentrations in blood begin to rise, excess $H^+$ is removed by combining with $\text{HCO}_3^-$
- If hydrogen ion concentrations begin to drop, carbonic acid dissociates, releasing $H^+$
Influence of Carbon Dioxide on Blood pH

Changes in respiratory rate can also:

- Alter blood pH
- Provide a fast-acting system to adjust pH when it is disturbed by metabolic factors
  - E.g. slow shallow breathing leads to increased CO2 levels which leads to increased H2CO3 which leads to a decrease in pH
  - E.g. rapid deep breathing leads to decreased CO2 levels which leads to decreased H2CO3 which leads to an increase in pH
Control of Respiration: Medullary Respiratory Centers

- Two areas of medulla oblongata are important in respiration:
  - dorsal respiratory group (DRG)
  - ventral respiratory group (VRG)

VRG is the rhythm-generating and integrative center

- Contains neurons that fire during inspiration and a different neural group that fire during expiration

- Inspiration: VRG neural impulse travels down the phrenic & intercostal nerves exciting the diaphragm & intercostal muscles, respectively
  - The thorax expands and air rushes in

- Expiration: when the VRG expiratory neurons fire, the inspiratory neurons stop firing and passive expiration occurs

- Mutual inhibition of the two neural areas

- Function of DRG: integrates input from peripheral stretch and chemoreceptors and communicates this information to the VRG
Depth and Rate of Breathing

- Chemical factors: CO2, O2, H+ sensed by chemoreceptors found in the ventrolateral medulla (central receptors) and aortic arch and carotid arteries (peripheral receptors)
Medullary Respiratory Centers

Higher brain centers (cerebral cortex—voluntary control over breathing)

Other receptors (e.g., pain) and emotional stimuli acting through the hypothalamus

Respiratory centers (medulla and pons)

Peripheral chemoreceptors
$O_2$, $CO_2$, $H^+$

Central chemoreceptors
$CO_2$, $H^+$

Receptors in muscles and joints

Stretch receptors in lungs

Irritant receptors

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Figure 22.25
Depth and Rate of Breathing: $P_{\text{CO}_2}$

- CO\textsubscript{2} is the most potent chemical influence and most controlled

- Changing PCO\textsubscript{2} levels are monitored by chemoreceptors of the brain stem

- As PCO\textsubscript{2} levels rise (hypercapnia), cerebrospinal fluid pH drops (CSF lacks pH buffering system) exciting the central chemoreceptors: depth & rate of breathing increase

- As PCO\textsubscript{2} levels fall, respiration is inhibited and apnea (breathing cessation) may occur until PCO\textsubscript{2} rises and stimulates respiration
Increased arterial $\text{PCO}_2$

- Increased $\text{PCO}_2$ decreases pH in cerebrospinal fluid (CSF)
- Central chemoreceptors in medulla respond to $\text{H}^+$ in CSF (mediate 70% of the $\text{CO}_2$ response)
- Peripheral chemoreceptors (carotid and aortic bodies) (mediate 30% of the $\text{CO}_2$ response)

- Afferent impulses
  - Medullary respiratory centers
  - Efferent impulses
    - Respiratory muscles
    - Increased ventilation (more $\text{CO}_2$ exhaled)
      - Arterial $\text{PCO}_2$ and pH return to normal

**Key:**
- Initial stimulus
- Physiological response
- Result

*Figure 22.26*
Depth and Rate of Breathing: $P_{CO_2}$

- Hyperventilation – increased depth and rate of breathing that:
  - Quickly flushes carbon dioxide from the blood
  - Occurs in response to hypercapnia
- Though a rise $CO_2$ acts as the original stimulus, control of breathing at rest is regulated by the hydrogen ion concentration in the brain
Depth and Rate of Breathing: $P_{CO_2}$

- Hypoventilation – slow and shallow breathing due to abnormally low $P_{CO_2}$ levels
  - Apnea (breathing cessation) may occur until $P_{CO_2}$ levels rise
Depth and Rate of Breathing: $P_{CO_2}$

- Arterial oxygen levels are monitored by the aortic and carotid bodies.

- Substantial drops in arterial $P_{O_2}$ (to 60 mm Hg) are needed before oxygen levels become a major stimulus for increased ventilation.

- If carbon dioxide is not removed (e.g., as in emphysema and chronic bronchitis), chemoreceptors become unresponsive to $P_{CO_2}$ chemical stimuli.

- In such cases, $P_{O_2}$ levels become the principal respiratory stimulus (hypoxic drive).
Depth and Rate of Breathing: Arterial pH

- Changes in arterial pH can modify respiratory rate even if carbon dioxide and oxygen levels are normal

- As pH falls, respiratory system controls attempt to compensate and increase pH by eliminating CO2 and carbonic acid from the blood by increasing respiration rate and depth
Peripheral Chemoreceptors

- Brain
- Sensory nerve fiber in cranial nerve IX (pharyngeal branch of glossopharyngeal)
- External carotid artery
- Internal carotid artery
- Carotid body
- Common carotid artery
- Cranial nerve X (vagus nerve)
- Sensory nerve fiber in cranial nerve X
- Aortic bodies in aortic arch
- Aorta
- Heart

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Figure 22.27
Summary of Interactions of PCO2 and Arterial pH

- 1) Rising CO2 levels are the most powerful respiratory stimulant

- 2) Blood PO2 affects breathing indirectly

- 3) When arterial PO2 falls below 60 mm Hg, PO2 becomes the major stimulus for respiration

- 4) Changes in arterial pH resulting from CO2 retention or metabolic factors act indirectly thru the peripheral chemoreceptors to promote changes in ventilation which in turn modify PCO2 and pH
Depth and Rate of Breathing: Higher Brain Centers

- Hypothalamic controls act through the limbic system to modify rate and depth of respiration
  - Example: “gasp” with a fright
- Cortical controls are direct signals from the cerebral motor cortex that bypass medullary controls
  - Examples: voluntary breath holding, taking a deep breath
  - However, when CO2 is at critical levels, involuntary breathing begins again
Depth and Rate of Breathing: Reflexes

- Pulmonary irritant reflexes – irritants promote reflexive constriction of air passages

- Inflation reflex (Hering-Breuer) – stretch receptors in the lungs are stimulated by lung inflation
  - Signal the medullary respiratory centers via afferent fibers of the vagus nerves sending inhibitory impulses that end inspiration
  - Upon inflation, inhibitory signals are sent to the medullary inspiration center to end inhalation and allow expiration (protective response)
Respiratory Adjustments: Exercise

- Respiratory adjustments are geared to both the intensity and duration of exercise

- During vigorous exercise:
  - Ventilation can increase 20 fold
  - Hyperpnea: increased ventilation in response to metabolic needs.
    - Different from hyperventilation in that hyperpnea does not lead to changes in blood O2 & CO2 levels whereas hyperventilation leads to low PCO2
  - Exercise-enhanced breathing is not prompted by an increase in $P_{CO2}$ or a decrease in $P_{O2}$ or pH
    - These levels remain surprisingly constant during exercise
Respiratory Adjustments: Exercise

- As exercise begins:
  - Ventilation increases abruptly. Due to:
    - 1) Psychological stimuli (anticipation)
    - 2) cortical motor activation of skeletal muscles and respiratory centers
    - 3) proprioceptors in muscles, tendons, joints

- When exercise stops:
  - 1-3 above turn off
Respiratory Adjustments: High Altitude

- The body responds to quick movement to high altitude (above 8000 ft) with symptoms of acute mountain sickness – headache, shortness of breath, nausea, and dizziness
Respiratory Adjustments: High Altitude

- Acclimatization – respiratory and hematopoietic adjustments to altitude include:
  - Increased ventilation – 2-3 L/min higher than at sea level
  - Chemoreceptors become more responsive to $P_{CO_2}$
  - Substantial decline in $P_{O_2}$ stimulates peripheral chemoreceptors
  - Produce more RBCs
Homeostatic Imbalances of the Respiratory System

- Chronic Obstructive Pulmonary Disease (COPD)
  - Exemplified by chronic bronchitis and obstructive emphysema
  - Patients have a history of:
    - Smoking
    - Dyspnea, where labored breathing occurs and gets progressively worse
    - Coughing and frequent pulmonary infections
  - COPD victims develop respiratory failure accompanied by hypoxemia, carbon dioxide retention, and respiratory acidosis
Pathogenesis of COPD

- Tobacco smoke
- Air pollution
- \( \alpha-1 \) antitrypsin deficiency

- Continual bronchial irritation and inflammation
- Breakdown of elastin in connective tissue of lungs

**Chronic bronchitis**
Bronchial edema, chronic productive cough, bronchospasm

**Emphysema**
Destruction of alveolar walls, loss of lung elasticity, air trapping

- Airway obstruction or air trapping
- Dyspnea
- Frequent infections

- Abnormal ventilation-perfusion ratio
- Hypoxemia
- Hypoventilation
Emphysema

- Permanent enlargement of the alveoli and destruction of alveolar walls leading to loss of elasticity of the lungs
- Three traits:
  - 1) accessory muscles must be enlisted to breathe using a lot of energy
  - 2) bronchioles collapse during expiration trapping air in the alveoli
  - 3) damage to pulmonary capillaries thus increasing air resistance which makes the right ventricle work harder
Asthma

- Characterized by dyspnea, wheezing, and chest tightness
- Active inflammation of the airways precedes bronchospasms
- Airway inflammation is an immune response caused by release of IL-4 and IL-5, which stimulate IgE and recruit inflammatory cells
- Airways thickened with inflammatory exudates magnify the effect of bronchospasms
Tuberculosis

- Infectious disease caused by the bacterium *Mycobacterium tuberculosis*
- Symptoms include fever, night sweats, weight loss, a racking cough, and splitting headache
- Treatment entails a 12-month course of antibiotics
Lung Cancer

- Accounts for 1/3 of all cancer deaths in the U.S.
- 90% of all patients with lung cancer were smokers
- The three most common types are:
  - Squamous cell carcinoma (20-40% of cases) arises in bronchial epithelium
  - Adenocarcinoma (25-35% of cases) originates in peripheral lung area
  - Small cell carcinoma (20-25% of cases) contains lymphocyte-like cells that originate in the primary bronchi and subsequently metastasize