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Respiratory Physiology &Acute Respiratory Failure

- ***Respiratory physiology is central to the practice of Anaesthesia***

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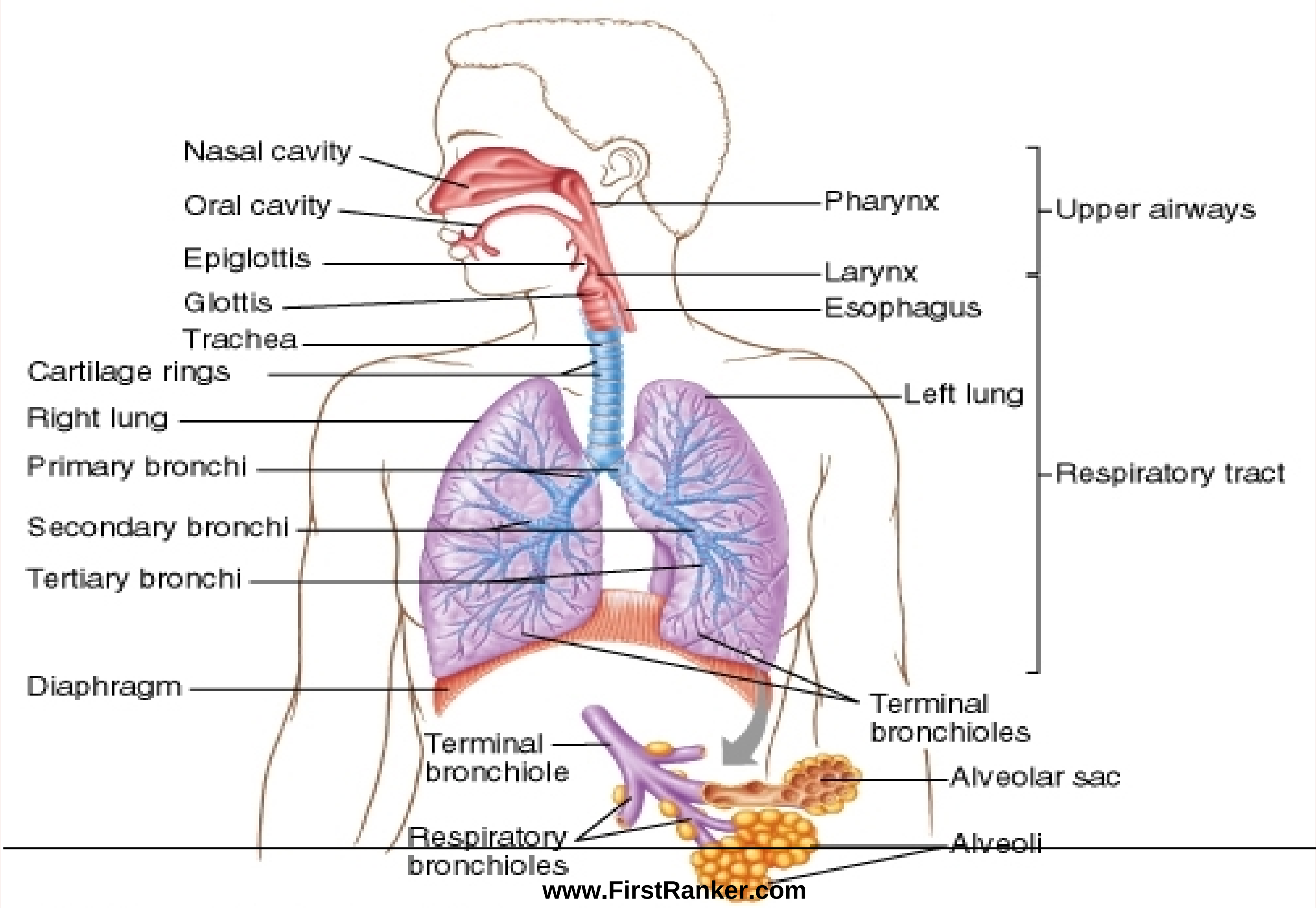
- The most commonly used anaesthetics- *the inhalational agents*- depend on the lungs for uptake and elimination.

- The most important side effects of both inhalational and intravenously administered anaesthetics are primarily respiratory.



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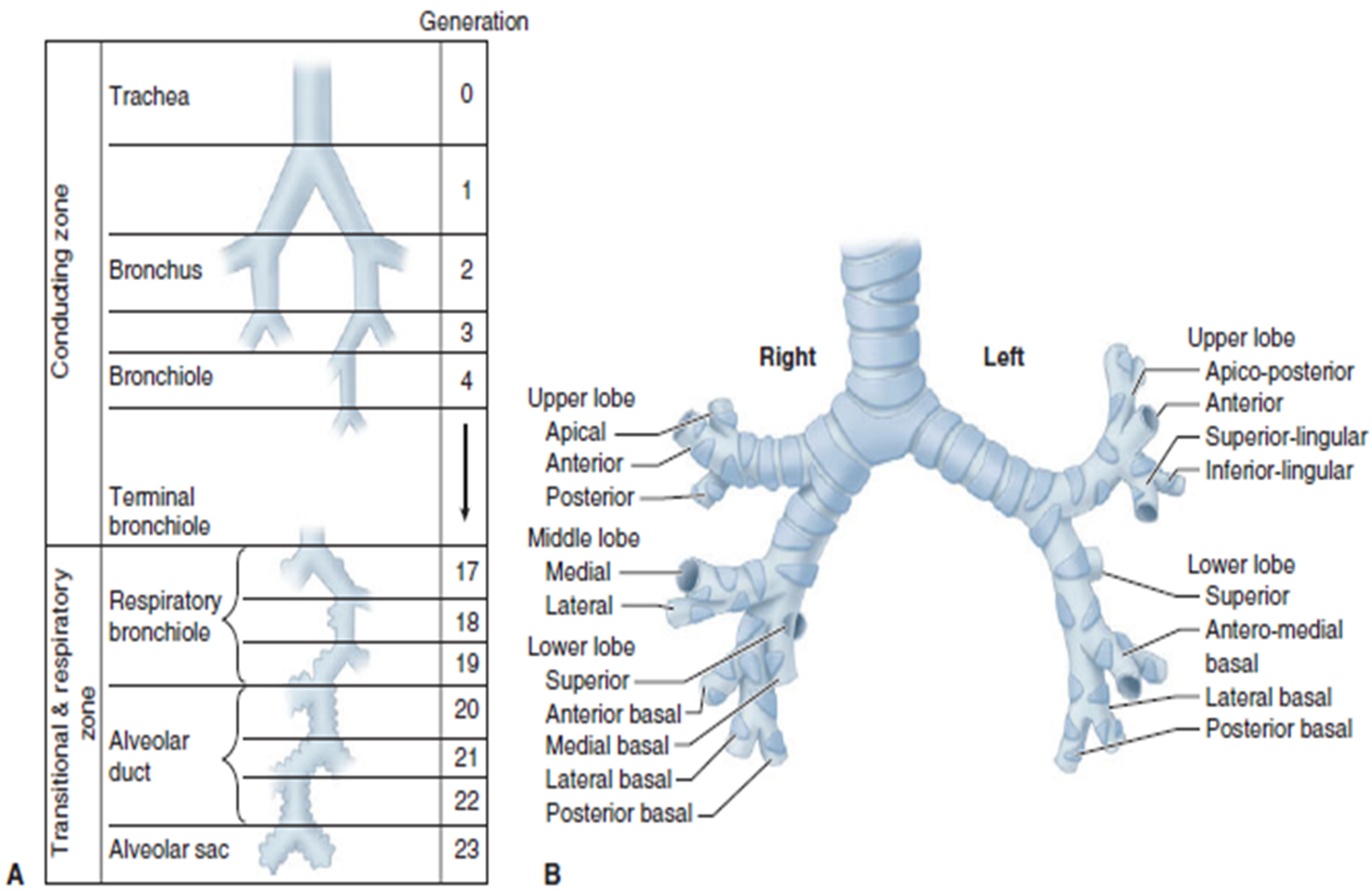

FIGURE 23–1 **A**: Dichotomous division of the airways. (Reproduced, with permission, from Guyton AC: *Textbook of Medical Physiology*, 7th ed. W.B. Saunders, 1986.) **B**: The segmental bronchi. (Reproduced, with permission, from Minnich DJ, Mathisen DJ: Anatomy of the trachea, carina, and bronchi. Thorac Surg Clin 2007 Nov;17(4):571-585.)

Functions of the Respiratory System

- Gas Exchange
  - O_2, CO_2
- Acid-base balance
  - $CO_2 + H_2O \leftrightarrow H_2CO_3 \leftrightarrow H^+ + HCO3^-$
- Phonation
- Pulmonary defense
- Pulmonary metabolism and handling of bioactive materials

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Respiration

- The term respiration includes 3 separate functions:
  - Ventilation:
    - Breathing.
  - Gas exchange:
    - Between air and capillaries in the lungs.
    - Between systemic capillaries and tissues of the body.
  - 0_2 utilization:
    - Cellular respiration.

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Ventilation

- Mechanical process that moves air in and out of the lungs.
- $[O_2]$ of air is higher in the lungs than in the blood, O_2 diffuses from air to the blood.
- $C0_2$ moves from the blood to the air by diffusing down its concentration gradient.
- Gas exchange occurs entirely by diffusion:
  - Diffusion is rapid because of the large surface area and the small diffusion distance.

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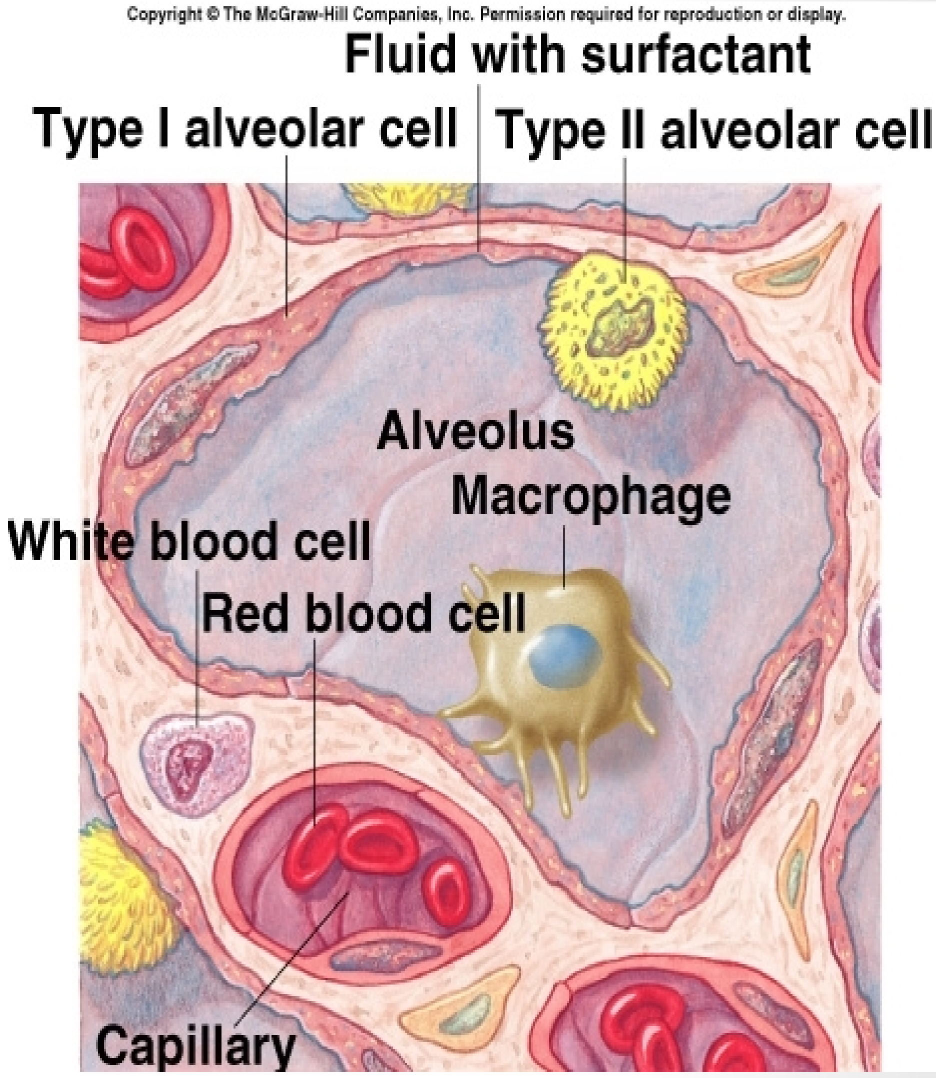

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Respiratory Zone

- Region of gas exchange between air and blood.
- Includes respiratory bronchioles and alveolar sacs.
- Must contain alveoli.

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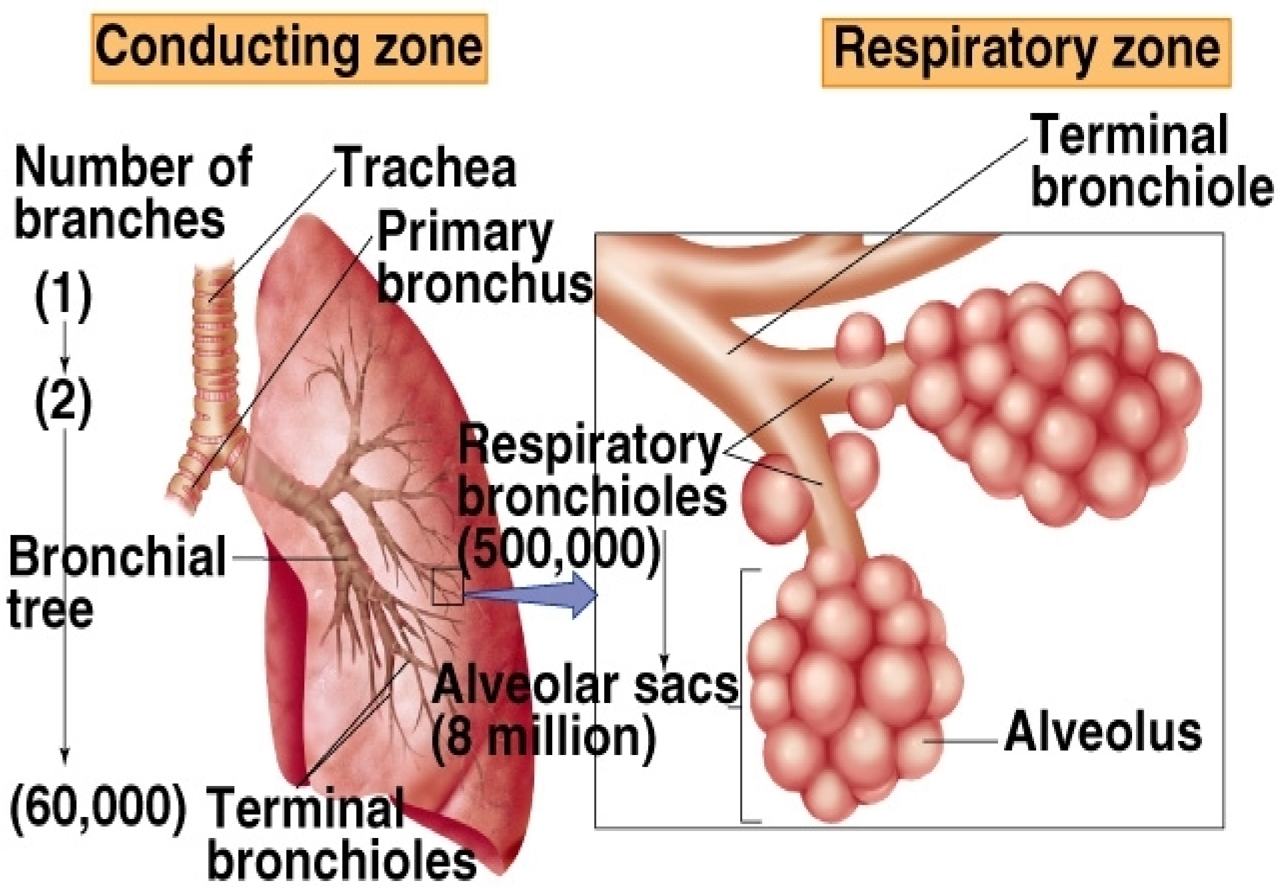

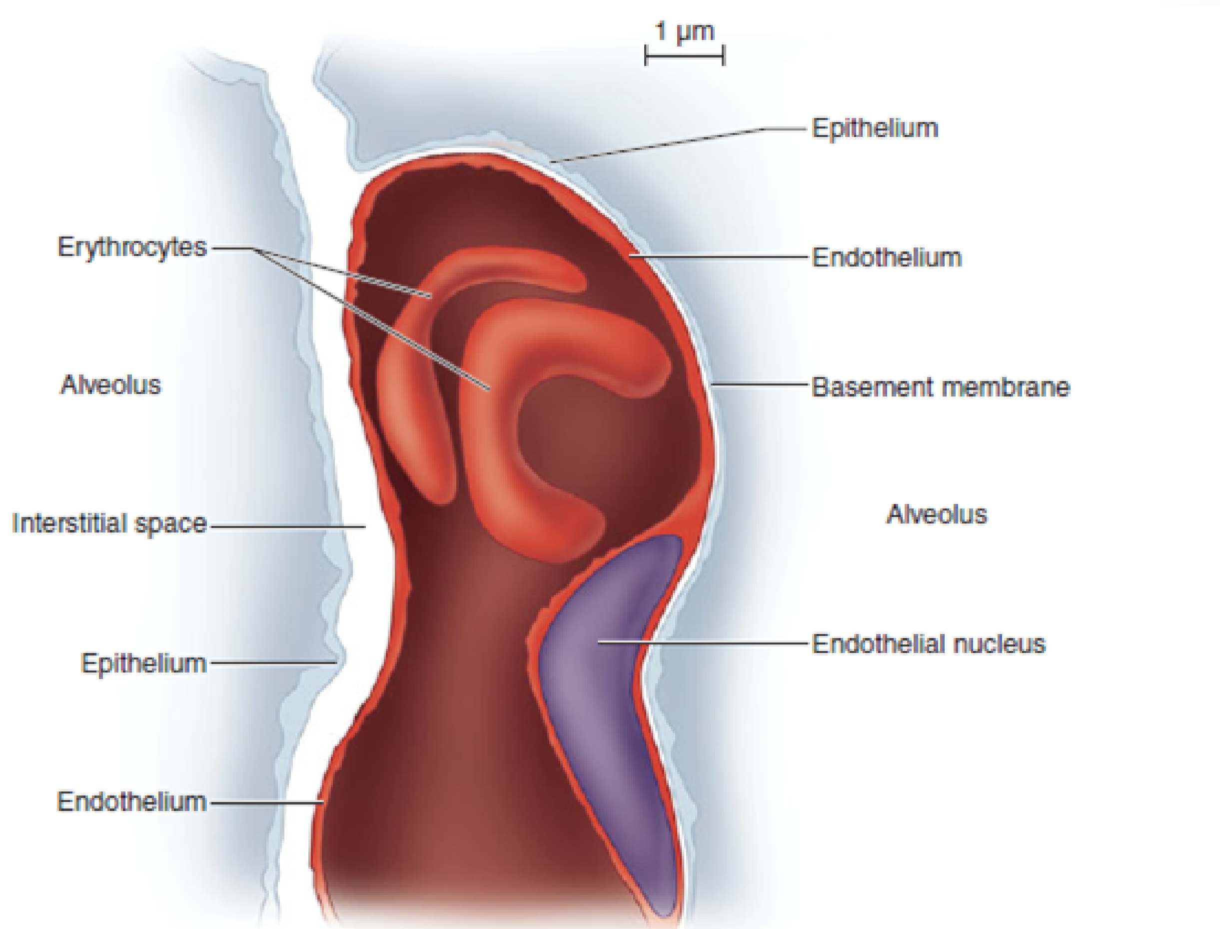

FIGURE 23–2 The pulmonary interstitial space, with a capillary passing between the two alveoli. The capillary is incorporated into the thin (gas-exchanging) side of the alveolus on the right. The interstitial space is incorporated into the thick side of the alveolus on the left. (Reproduced with permission, from Nunn JF: *Nunn's Applied Physiology*, 4th ed. Butterworth, 2000.)

Alveoli

- Polyhedral in shape and clustered like units of honeycomb.
- ~ 300 million air sacs (alveoli).
  - Large surface area (60–80 m^2).
  - Each alveolus is 1 cell layer thick.
    - Total air barrier is 2 cells across (2 μm).
- 2 types of cells:
  - Alveolar type I:
    - Structural cells.
  - Alveolar type II:
    - Secrete surfactant.

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Conducting Zone

- All the structures air passes through before reaching the respiratory zone.
- Warms and humidifies inspired air.
- Filters and cleans:
  - Mucus secreted to trap particles in the inspired air.
  - Mucus moved by cilia to be expectorated.

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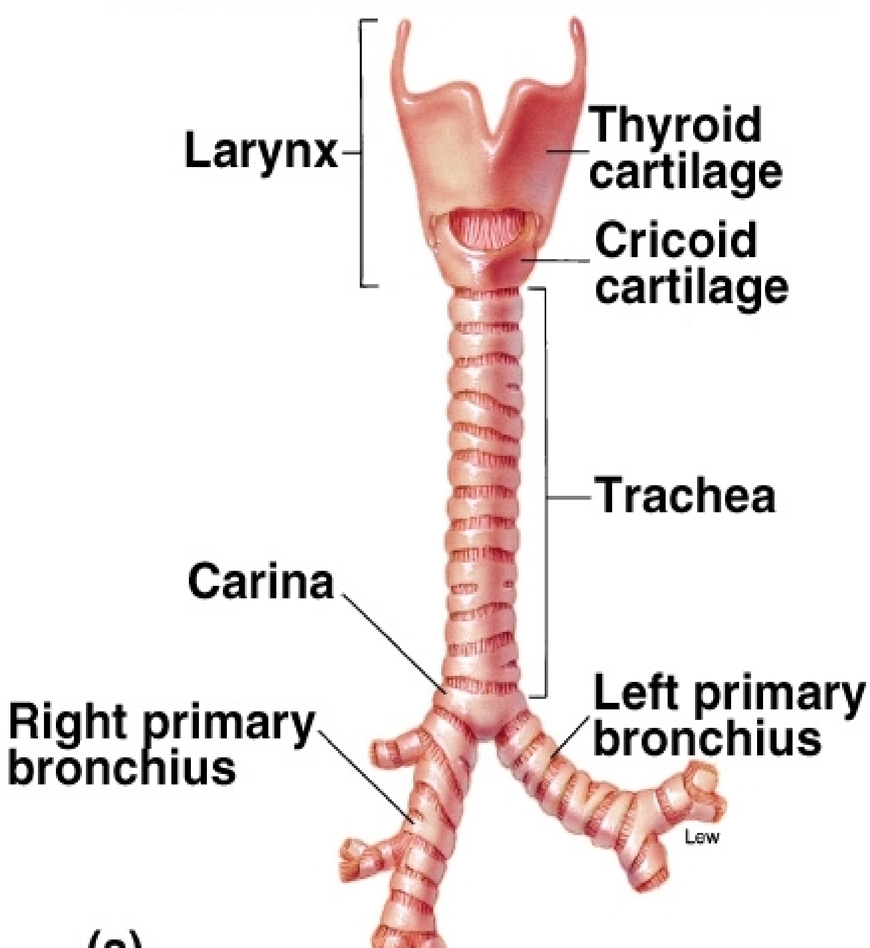

(a)

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Tracheobronchial Tree:

a) **Trachea**- conduit for ventilation

- Clearance of tracheal &bronchial secretions
- Begins at the lower border of the cricoid cartilage and extends to the level of the carina
- Average length of 10–13 cm
- The external diameters of the trachea is approximately 2.3 cm coronally and 1.8 cm sagitally in men and 2.0 cm & 1.4 cm, respectively, in women

- The trachea bifurcates at the carina into the right and left main stem bronchi
- Dichotomous division, starting with the trachea and ending in alveolar sacs, is estimated to involve 23 divisions.
- An estimated 300 million alveoli provide an enormous membrane (50–100 m^2) for gas exchange in the average adult
- Gas exchange can occur only across the flat epithelium, which begins to appear on respiratory bronchioles

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Pulmonary Circulation & Lymphatics

- The lungs are supplied by two circulations, pulmonary and bronchial
- The bronchial circulation arises from lt heart
- Along their courses, the bronchial vessels anastomose with the pulmonary arterial circulation and continue as far as the alveolar duct.
- The pulmonary circulation normally receives the total output of the right heart via the pulmonary artery, which divides into rt and lt branches to supply each lung

- Deoxygenated blood passes through the pulmonary capillaries, where O_2 is taken up and CO_2 is eliminated
- The oxygenated blood is then returned to the lt heart by 4 main pulmonary veins (two from each lung)

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Innervation

- The diaphragm is innervated by the phrenic nerves, which arise from the C3–C5 nerve roots.
- U/L phrenic nerve palsy only modestly reduces most indices of pulmonary function (~ 25%).
- B/L phrenic nerve palsies produce more severe impairment
- The vagus nerves provide sensory innervation to the tracheobronchial tree

Thoracic Cavity

- Diaphragm:
  - Sheets of striated muscle divides anterior body cavity into 2 parts.
- Above diaphragm: thoracic cavity:
  - Contains heart, large blood vessels, trachea, esophagus, thymus, and lungs.
- Below diaphragm: abdominopelvic cavity:
  - Contains liver, pancreas, GI tract, spleen, and genitourinary tract.
- Intrapleural space:
  - Space between visceral and parietal pleurae.

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Intrapulmonary and Intrapleural Pressures

- Visceral and parietal pleurae are flush against each other.
  - The intrapleural space contains only a film of fluid secreted by the membranes.
- Lungs normally remain in contact with the chest walls.
- Lungs expand and contract along with the thoracic cavity.
- Intrapulmonary pressure:
  - Intra-alveolar pressure (pressure in the alveoli).
- Intrapleural pressure:
  - Pressure in the intrapleural space.
  - Pressure is negative, due to lack of air in the intrapleural space.

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Transpulmonary Pressure

- Pressure difference across the wall of the lung.
- Intrapulmonary pressure – intrapleural pressure.
  - Keeps the lungs against the chest wall.

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Intrapulmonary and Intrapleural Pressures (continued)

- During inspiration:
  - Atmospheric pressure is > intrapulmonary pressure (-3 mm Hg).
- During expiration:
  - Intrapulmonary pressure (+3 mm Hg) is > atmospheric pressure.

Boyle's Law

- Changes in intrapulmonary pressure occur as a result of changes in lung volume.
  - Pressure of gas is inversely proportional to its volume.
- Increase in lung volume decreases intrapulmonary pressure.
  - Air goes in.
- Decrease in lung volume, raises intrapulmonary pressure above atmosphere.
  - Air goes out.

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Physical Properties of the Lungs

- Ventilation occurs as a result of pressure differences induced by changes in lung volume.
- Physical properties that affect lung function:
  - Compliance.
  - Elasticity.
  - Surface tension.

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Compliance

- Distensibility (stretchability):
  - Ease with which the lungs can expand.
- Change in lung volume per change in transpulmonary pressure.

  $\Delta V/\Delta P$
- 100 x more distensible than a balloon.
  - Compliance is reduced by factors that produce resistance to distension.

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Elasticity

- Tendency to return to initial size after distension.
- High content of elastin proteins.
  - Very elastic and resist distension.
    - Recoil ability.
- Elastic tension increases during inspiration and is reduced by recoil during expiration.

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Surface Tension

- Force exerted by fluid in alveoli to resist distension.
    - Lungs secrete and absorb fluid, leaving a very thin film of fluid.
  - This film of fluid causes surface tension.
  - Fluid absorption is driven (osmosis) by Na^+ active transport.
  - Fluid secretion is driven by the active transport of Cl^- out of the alveolar epithelial cells.
- H_20 molecules at the surface are attracted to other H_20 molecules by attractive forces.
  - Force is directed inward, raising pressure in alveoli.

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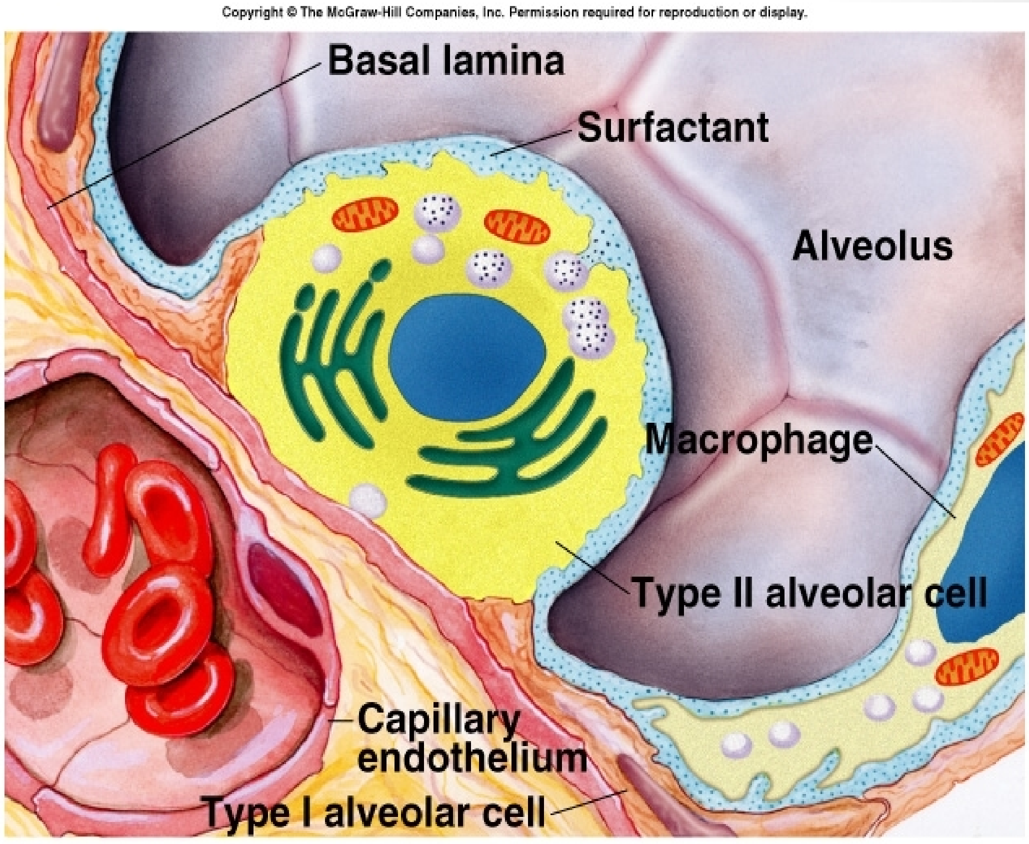
Surfactant

- Phospholipid produced by alveolar type II cells.
- Lowers surface tension.
  - Reduces attractive forces of hydrogen bonding by becoming interspersed between H_20 molecules.
    - Surface tension in alveoli is reduced.
- As alveoli radius decreases, surfactant's ability to lower surface tension increases.
- Disorders:
  - RDS.
  - ARDS.



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Quiet Inspiration

- Active process:
  - Contraction of diaphragm, increases thoracic volume vertically.
- Parasternal and external intercostals contract, raising the ribs; increasing thoracic volume laterally.
- Pressure changes:
  - Alveolar changes from 0 to –3 mm Hg.
  - Intrapleural changes from –4 to –6 mm Hg.
  - Transpulmonary pressure = +3 mm Hg.

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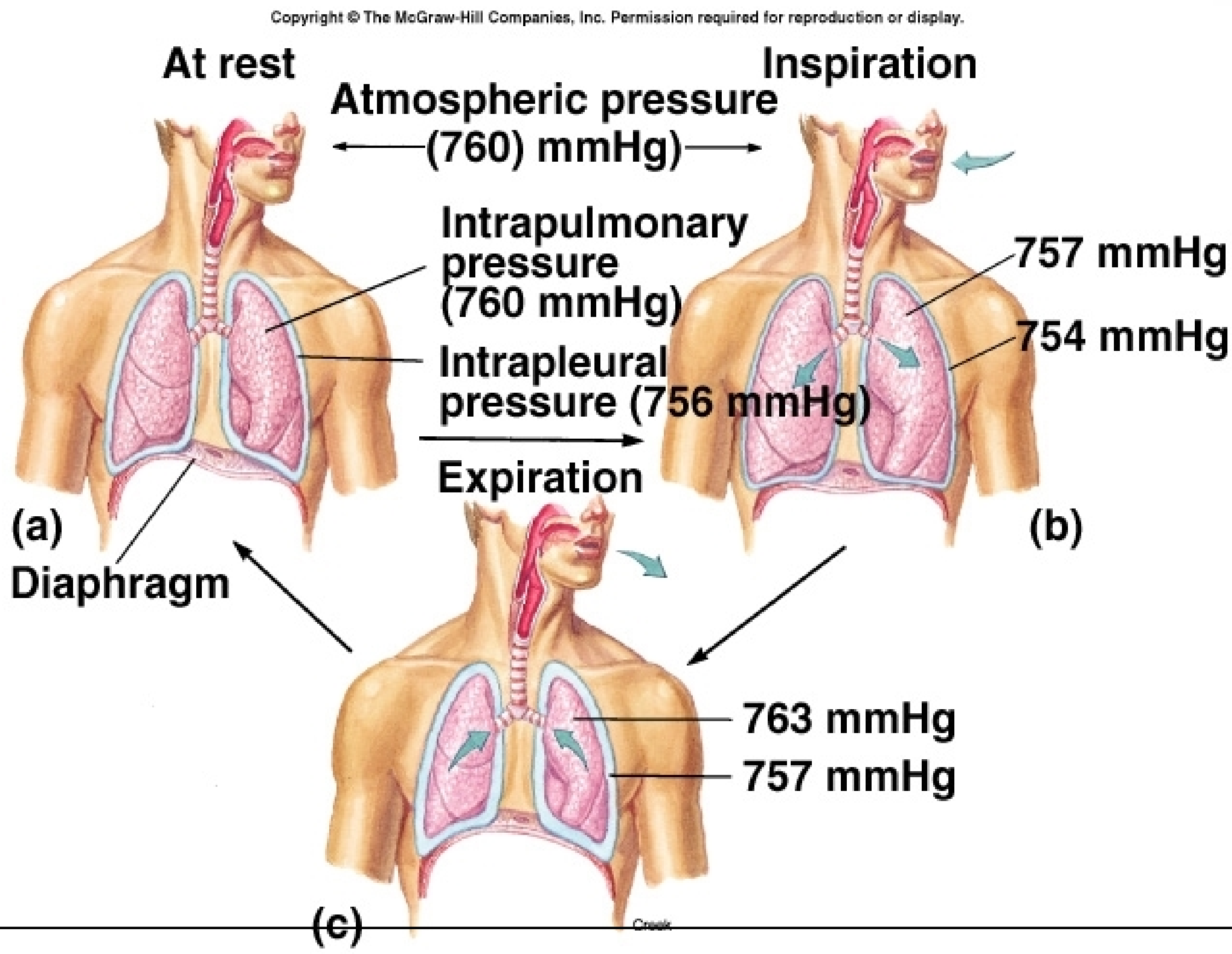
Expiration

- Quiet expiration is a passive process.
  - After being stretched by contractions of the diaphragm and thoracic muscles; the diaphragm, thoracic muscles, thorax, and lungs recoil.
  - Decrease in lung volume raises the pressure within alveoli above atmosphere, and pushes air out.
- Pressure changes:
  - Intrapulmonary pressure changes from –3 to +3 mm Hg.
  - Intrapleural pressure changes from –6 to –3 mm Hg.
  - Transpulmonary pressure = +6 mm Hg.

Pulmonary Ventilation



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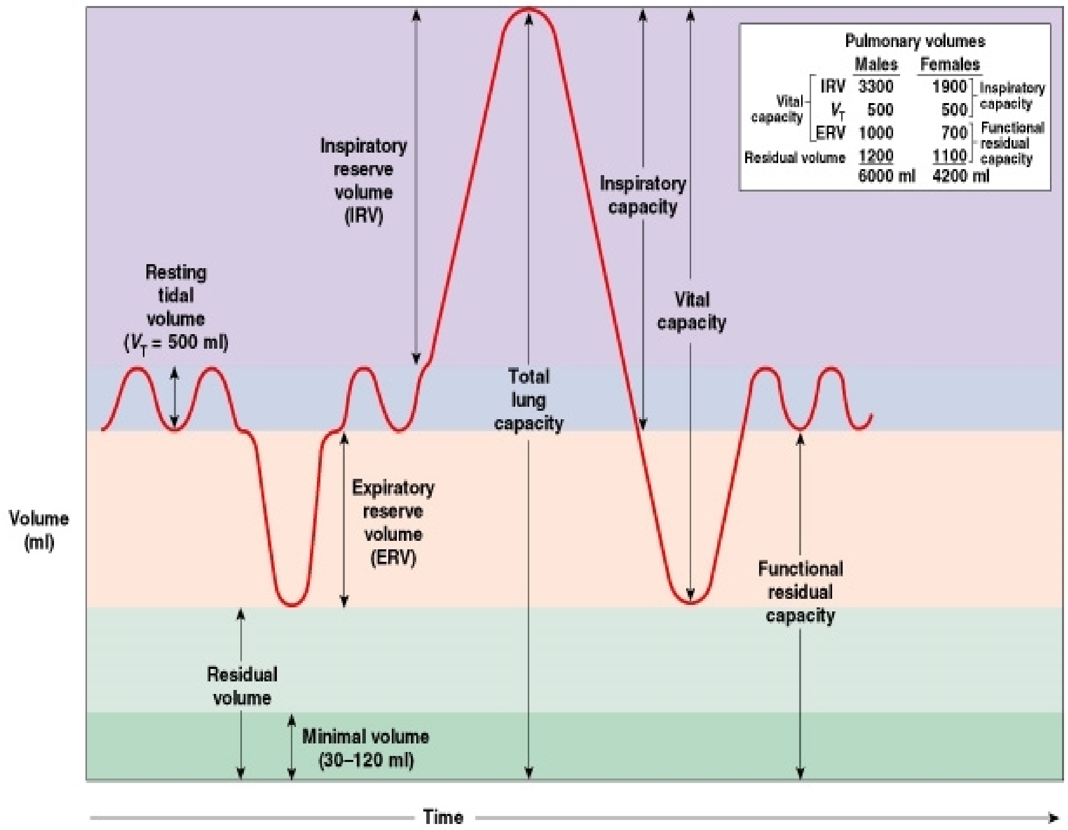
Lung volumes and capacities

4 lung volumes:

- tidal (~500 ml)
- inspiratory reserve (~3100 ml)
- expiratory reserve (~1200 ml)
- residual (~1200 ml)

4 lung capacities

- inspiratory (~3600 ml)
- functional residual (~2400 ml)
- vital (~4800 ml)
- total lung (~6000 ml)



Terms Used to Describe Lung Volumes and Capacities

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Table 16.3 Terms Used to Describe Lung Volumes and Capacities

Term	Definition
Lung Volumes	The four nonoverlapping components of the total lung capacity
Tidal volume	The volume of gas inspired or expired in an unforced respiratory cycle
Inspiratory reserve volume	The maximum volume of gas that can be inspired during forced breathing in addition to tidal volume
Expiratory reserve volume	The maximum volume of gas that can be expired during forced breathing in addition to tidal volume
Residual volume	The volume of gas remaining in the lungs after a maximum expiration
Lung Capacities	Measurements that are the sum of two or more lung volumes
Total lung capacity	The total amount of gas in the lungs after a maximum inspiration
Vital capacity	The maximum amount of gas that can be expired after a maximum inspiration
Inspiratory capacity	The maximum amount of gas that can be inspired after a normal tidal expiration
Functional residual capacity	The amount of gas remaining in the lungs after a normal tidal expiration

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Anatomical Dead Space

- Not all of the inspired air reached the alveoli.
- As fresh air is inhaled it is mixed with air in anatomical dead space.
  - Conducting zone and alveoli where $[O_2]$ is lower than normal and $[CO_2]$ is higher than normal.
- Alveolar ventilation = F x (TV- DS).
  - F = frequency (breaths/min.).
  - TV = tidal volume.
  - DS = dead space.

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Restrictive and Obstructive Disorders

- Restrictive disorder:
  - Vital capacity is reduced.
  - FVC is normal.
- Obstructive disorder:
  - Diagnosed by tests that measure the rate of expiration.
    - VC is normal.
    - FEV_1 is < 80%.

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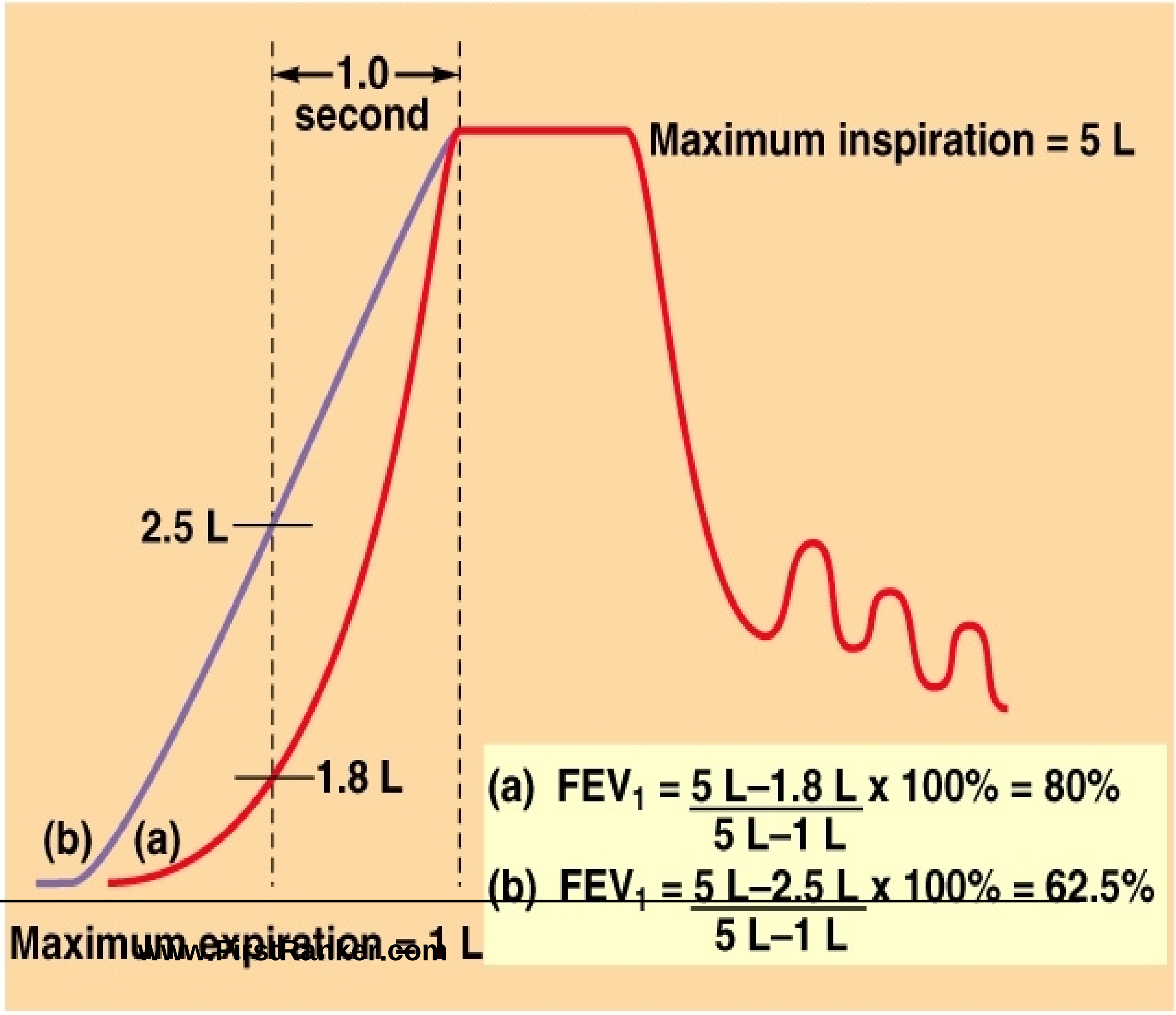

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Gas Exchange in the Lungs

- Dalton's Law:
  - Total pressure of a gas mixture is = to the sum of the pressures that each gas in the mixture would exert independently.
- Partial pressure:
  - The pressure that an particular gas exerts independently.
- $P_{ATM} = P_{N_2} + P_{O_2} + P_{CO_2} + P_{H_2O}$= 760 mm Hg.
  - O_2 is humidified = 105 mm Hg.
    - H_2O contributes to partial pressure (47 mm Hg).
      - P_{O_2} (sea level) = 150 mm Hg.
  - P_{CO_2} = 40 mm Hg.

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Partial Pressures of Gases in Inspired Air and Alveolar Air

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

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Partial Pressures of Gases in Blood

- When a liquid or gas (blood and alveolar air) are at equilibrium:
  - The amount of gas dissolved in fluid reaches a maximum value (Henry's Law).
- Depends upon:
  - Solubility of gas in the fluid.
  - Temperature of the fluid.
  - Partial pressure of the gas.
- [Gas] dissolved in a fluid depends directly on its partial pressure in the gas mixture.

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Significance of Blood P_{O_2} and P_{CO_2} Measurements

- At normal P_{O_2} arterial blood is about 100 mm Hg.
- P_{O_2} level in the systemic veins is about 40 mm Hg.

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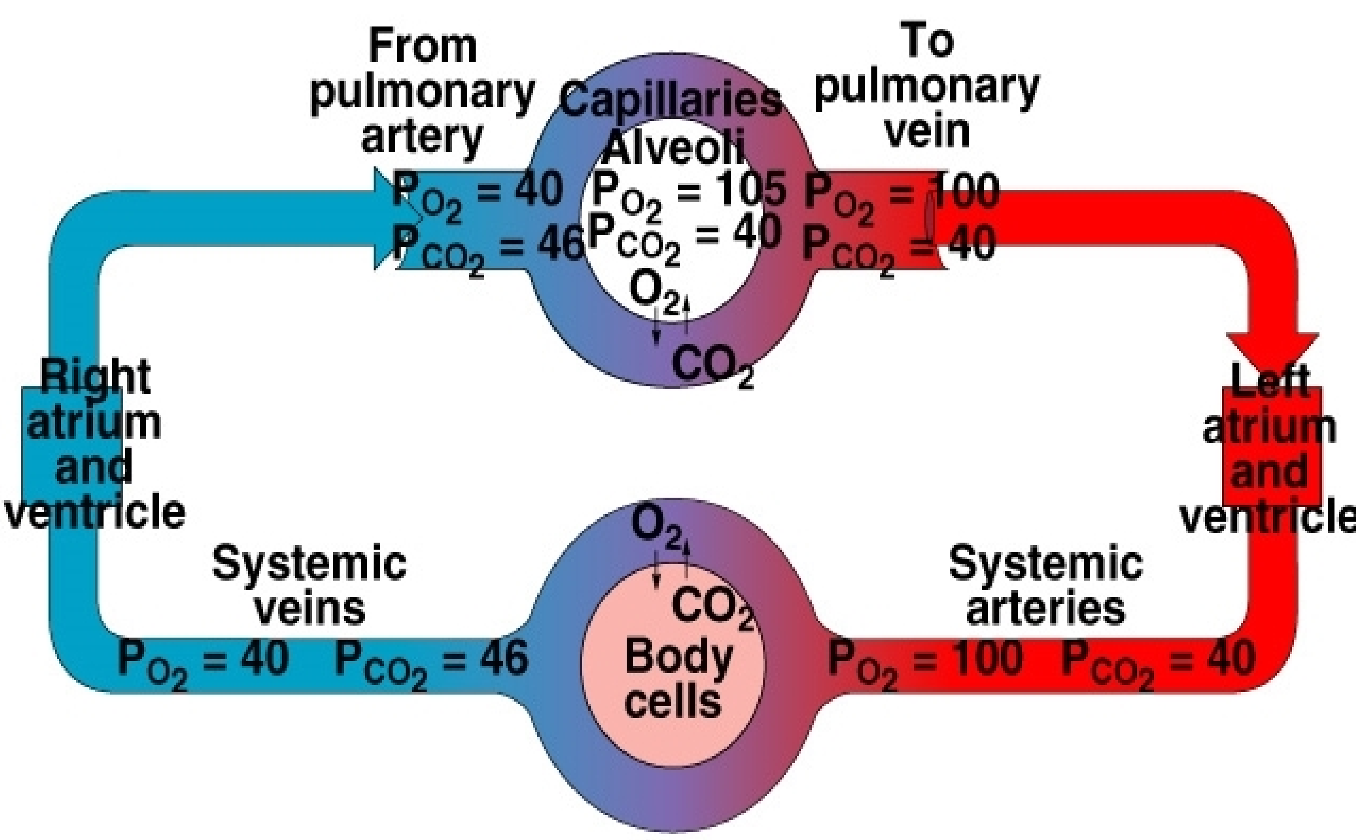

- P_{CO_2} is 46 mm Hg in the systemic veins.
- Provides a good index of lung function.

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Pulmonary Circulation

- Rate of blood flow through the pulmonary circulation is = flow rate through the systemic circulation.
  - Driving pressure is about 10 mm Hg.
- Pulmonary vascular resistance is low.
  - Low pressure pathway produces less net filtration than produced in the systemic capillaries.
    - Avoids pulmonary edema.
- Autoregulation:
  - Pulmonary arterioles constrict when alveolar P_{O_2} decreases.
  - Matches ventilation/perfusion ratio.

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Pulmonary Circulation (continued)

- In a fetus:
  - Pulmonary circulation has a higher vascular resistance, because the lungs are partially collapsed.
- After birth, vascular resistance decreases:
  - Opening the vessels as a result of subatmospheric intrapulmonary pressure.
  - Physical stretching of the lungs.
  - Dilation of pulmonary arterioles in response to increased alveolar P_{O_2}.

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Lung Ventilation/Perfusion Ratios

- Functionally:
  - Alveoli at apex are underperfused (overventilated).
  - Alveoli at the base are underventilated (overperfused).

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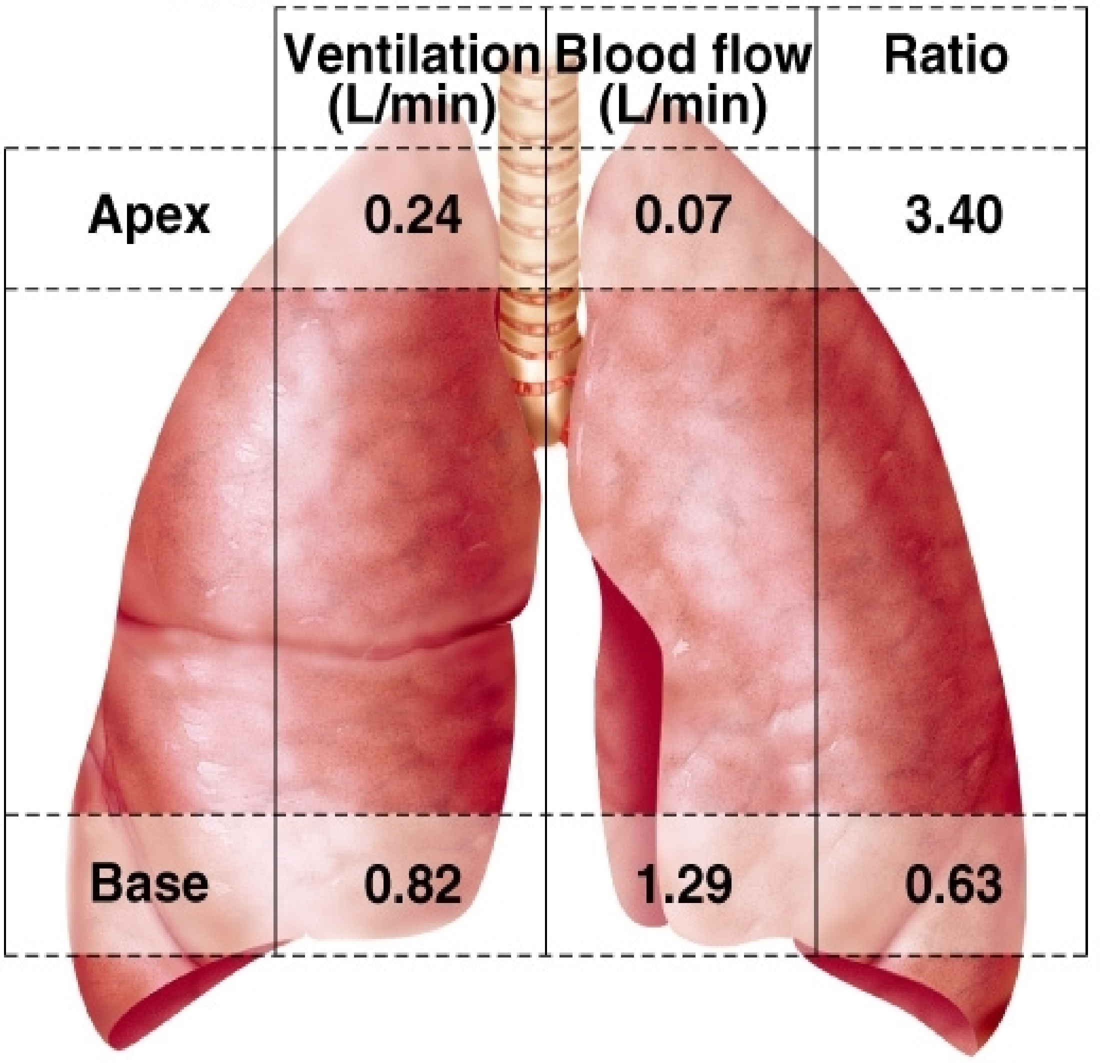

	Ventilation (L/min)	Blood flow (L/min)	Ratio
Apex	0.24	0.07	3.40
Base	0.82	1.29	0.63

Brain Stem Respiratory Centers

- Neurons in the reticular formation of the medulla oblongata form the rhythmicity center:
  - Controls automatic breathing.
  - Consists of interacting neurons that fire either during inspiration (I neurons) or expiration (E neurons).

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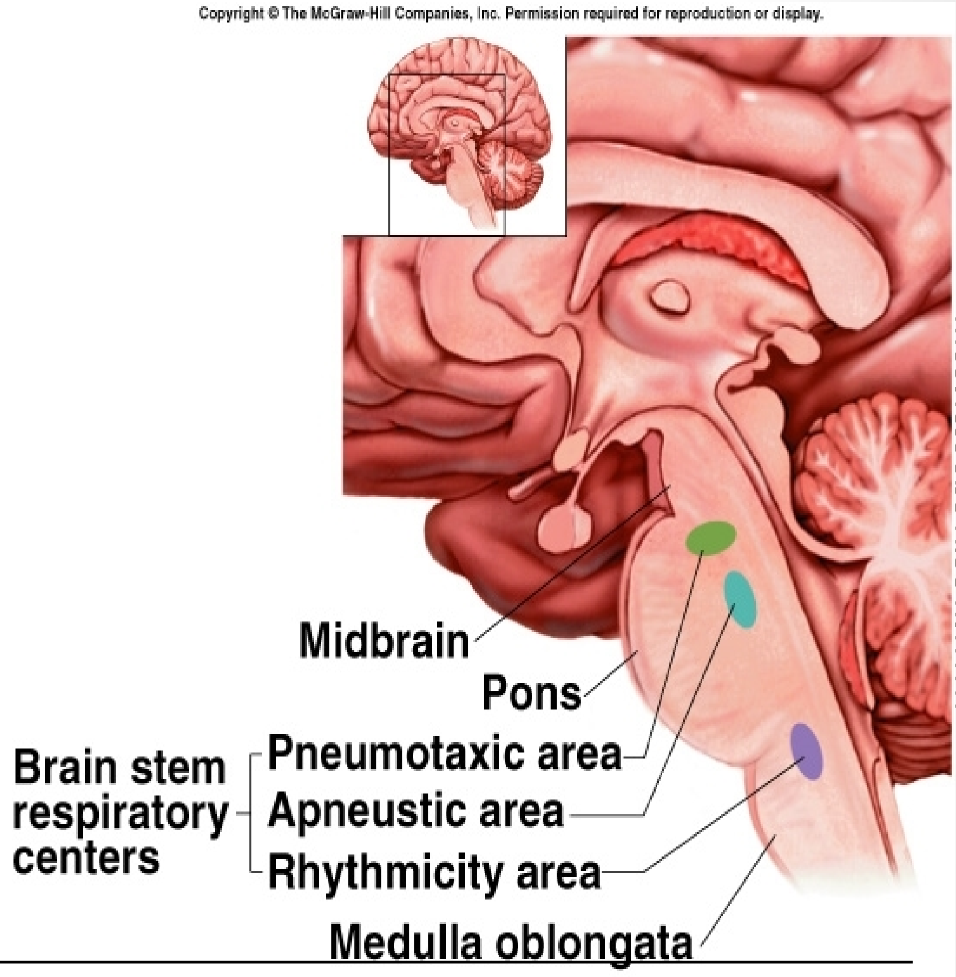

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Brain Stem Respiratory Centers (continued)

- I neurons project to, and stimulate spinal motor neurons that innervate respiratory muscles.
- Expiration is a passive process that occurs when the I neurons are inhibited.
- Activity varies in a reciprocal way.

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Rhythmicity Center

- I neurons located primarily in dorsal respiratory group (DRG):
  - Regulate activity of phrenic nerve.
    - Project to and stimulate spinal interneurons that innervate respiratory muscles.
- E neurons located in ventral respiratory group (VRG):
  - Passive process.
    - Controls motor neurons to the internal intercostal muscles.
- Activity of E neurons inhibit I neurons.
  - Rhythmicity of I and E neurons may be due to pacemaker neurons.

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Pons Respiratory Centers

- Activities of medullary rhythmicity center is influenced by pons.
- Apneustic center:
  - Promotes inspiration by stimulating the I neurons in the medulla.
- Pneumotaxic center:
  - Antagonizes the apneustic center.
  - Inhibits inspiration.

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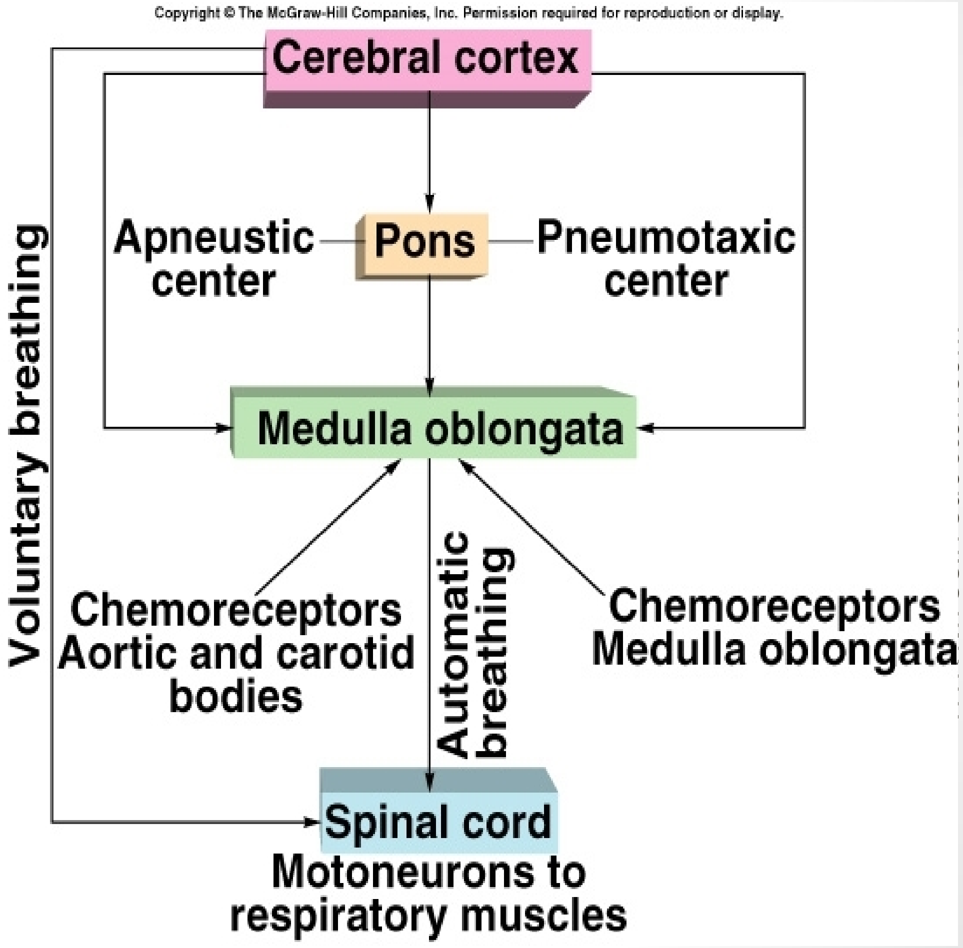
Chemoreceptors

- 2 groups of chemo-receptors that monitor changes in blood P_{CO_2}, P_{O_2}, and pH.
- Central:
  - Medulla.
- Peripheral:
  - Carotid and aortic bodies.
    - Control breathing indirectly via sensory nerve fibers to the medulla (X, IX).



Effects of Blood P_{CO_2} and pH on Ventilation

- Chemoreceptor input modifies the rate and depth of breathing.
  - Oxygen content of blood decreases more slowly because of the large "reservoir" of oxygen attached to hemoglobin.
  - Chemoreceptors are more sensitive to changes in P_{CO_2}.

$$H_20 + CO_2 \longleftrightarrow H_2CO_3 \longleftrightarrow H^+ + HCO_3^-$$

- Rate and depth of ventilation adjusted to maintain arterial P_{CO_2} of 40 mm Hg.

Chemoreceptor Control

- Central chemoreceptors:
  - More sensitive to changes in arterial $PC0_2$.

$$H_20 + CO_2 \longrightarrow H_2C0_3 \longrightarrow H^+$$

- H^+ cannot cross the blood brain barrier.
- $C0_2$ can cross the blood brain barrier and will form H_2C0_3.
  - Lowers pH of CSF.
    - Directly stimulates central chemoreceptors.

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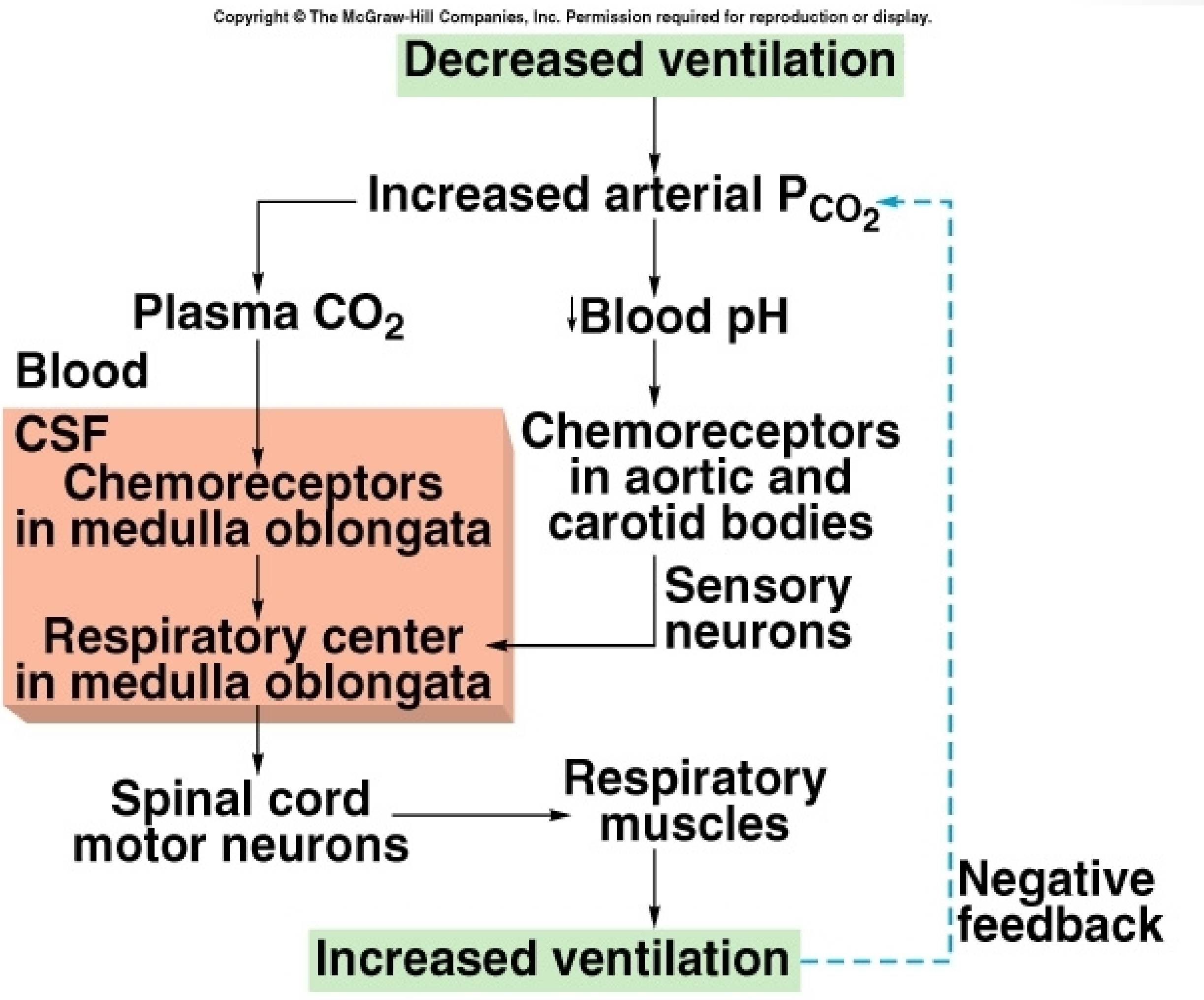
Chemoreceptor Control of Breathing



Effects of Blood P_{O_2} on Ventilation

- Blood PO_2 affected by breathing indirectly.
  - Influences chemoreceptor sensitivity to changes in PCO_2.
- Hypoxic drive:
  - Emphysema blunts the chemoreceptor response to PCO_2.
  - Choroid plexus secrete more HCO_3^- into CSF, buffering the fall in CSF pH.
  - Abnormally high PCO_2 enhances sensitivity of carotid bodies to fall in PO_2.

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Effects of Pulmonary Receptors on Ventilation

- Lungs contain receptors that influence the brain stem respiratory control centers via sensory fibers in vagus.
  - Unmyelinated C fibers can be stimulated by:
    - Capsaicin:
      - Produces apnea followed by rapid, shallow breathing.
    - Histamine and bradykinin:
      - Released in response to noxious agents.
  - Irritant receptors are rapidly adaptive receptors.
- Hering-Breuer reflex:
  - Pulmonary stretch receptors activated during inspiration.
    - Inhibits respiratory centers to prevent undue tension on lungs.

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Hemoglobin and 0_2 Transport

- 280 million hemoglobin/RBC.
- Each hemoglobin has 4 polypeptide chains and 4 hemes.
- In the center of each heme group is 1 atom of iron that can combine with 1 molecule 0_2.

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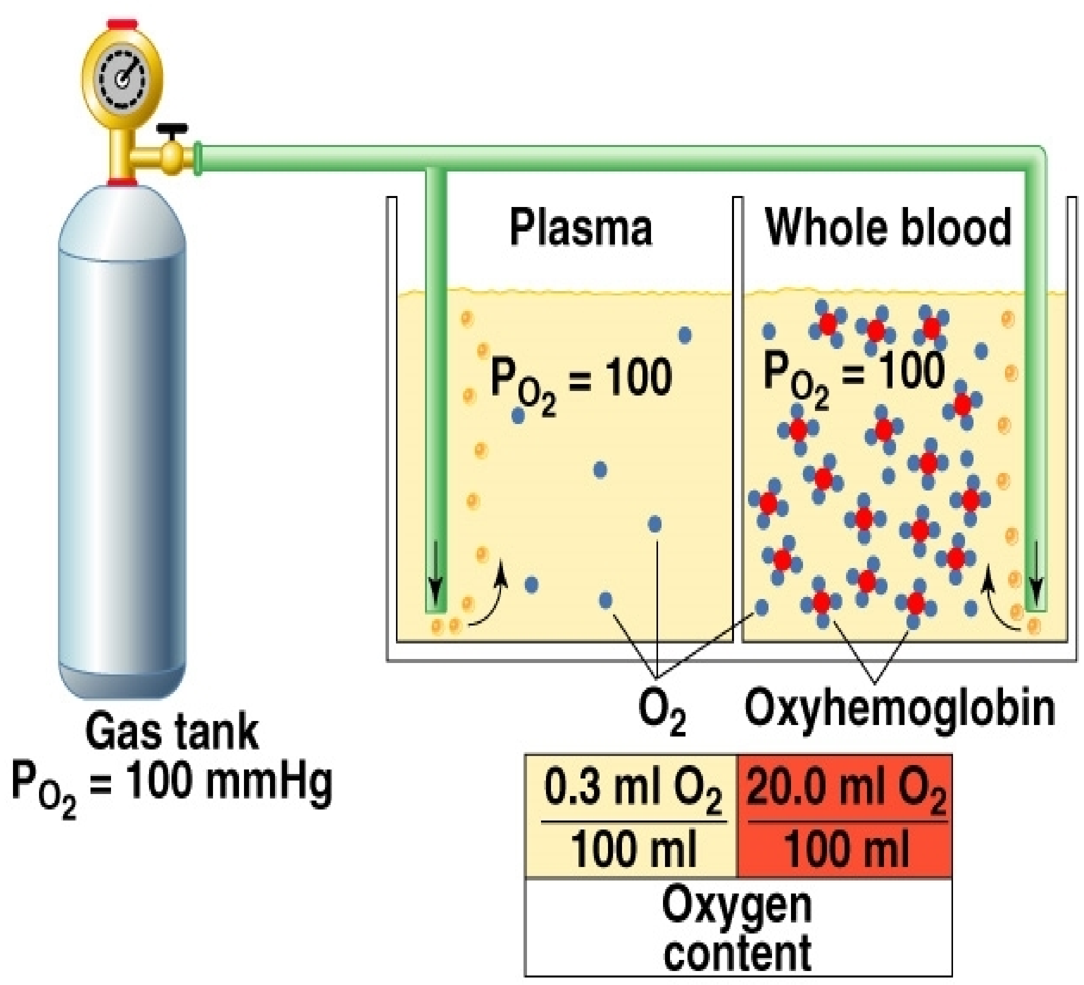

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Hemoglobin

- Oxyhemoglobin:
  - Normal heme contains iron in the reduced form (Fe^{2+}).
  - Fe^{2+} shares electrons and bonds with oxygen.
- Deoxyhemoglobin:
  - When oxyhemoglobin dissociates to release oxygen, the heme iron is still in the reduced form.
  - Hemoglobin does not lose an electron when it combines with 0_2.

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Hemoglobin (continued)

- Methemoglobin:
  - Has iron in the oxidized form (Fe^{3+}).
    - Lacks electrons and cannot bind with 0_2.
      - Blood normally contains a small amount.
- Carboxyhemoglobin:
  - The reduced heme is combined with carbon monoxide.
  - The bond with carbon monoxide is **210** times stronger than the bond with oxygen.
    - Transport of 0_2 to tissues is impaired.

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Hemoglobin (continued)

- Oxygen-carrying capacity of blood determined by its [hemoglobin].
  - Anemia:
    - [Hemoglobin] below normal.
  - Polycythemia:
    - [Hemoglobin] above normal.
  - Hemoglobin production controlled by erythropoietin.
    - Production stimulated by P_{CO_2} delivery to kidneys.
- Loading/unloading depends:
  - PO_2 of environment.
  - Affinity between hemoglobin and O_2.

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Oxyhemoglobin Dissociation Curve

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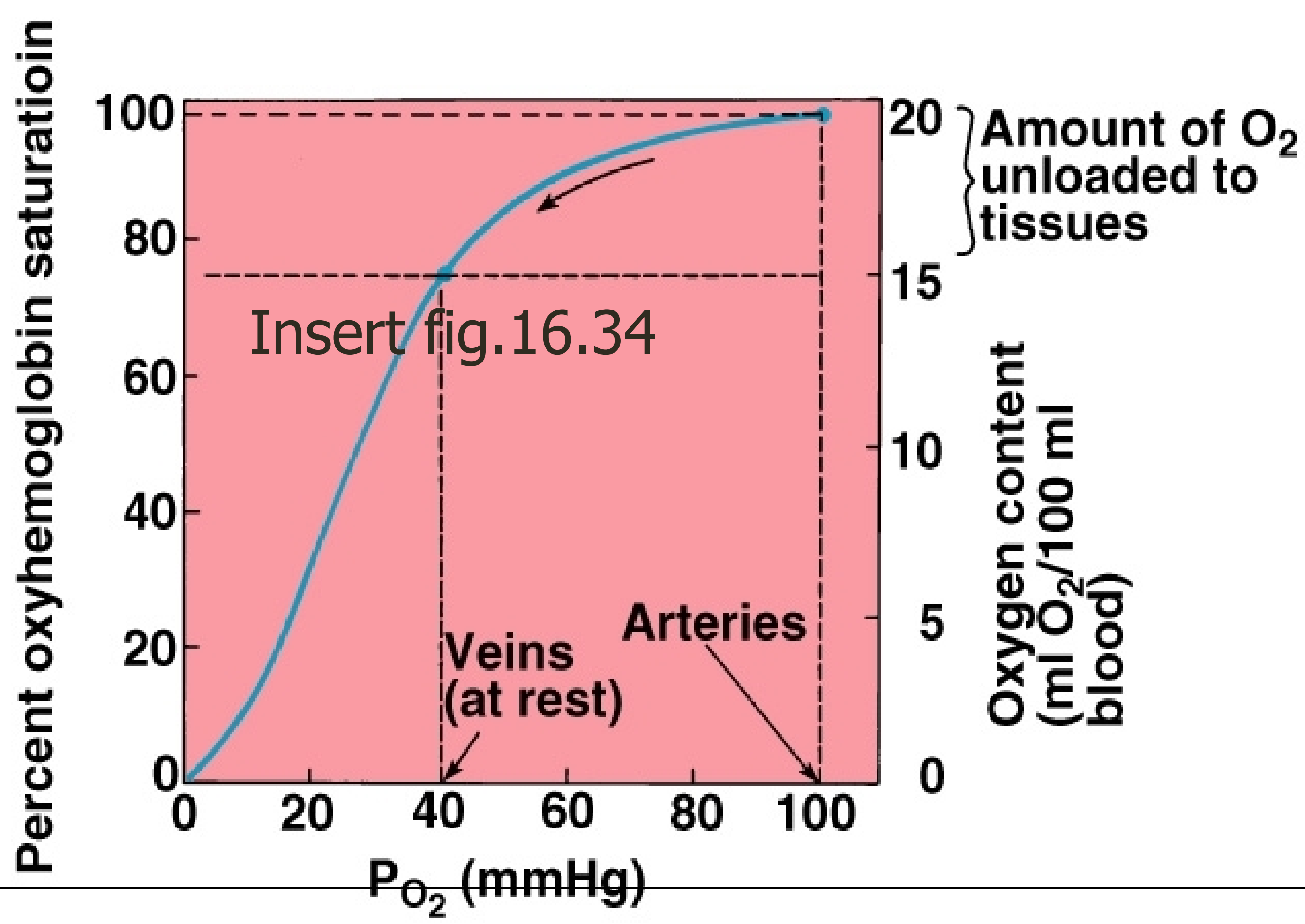

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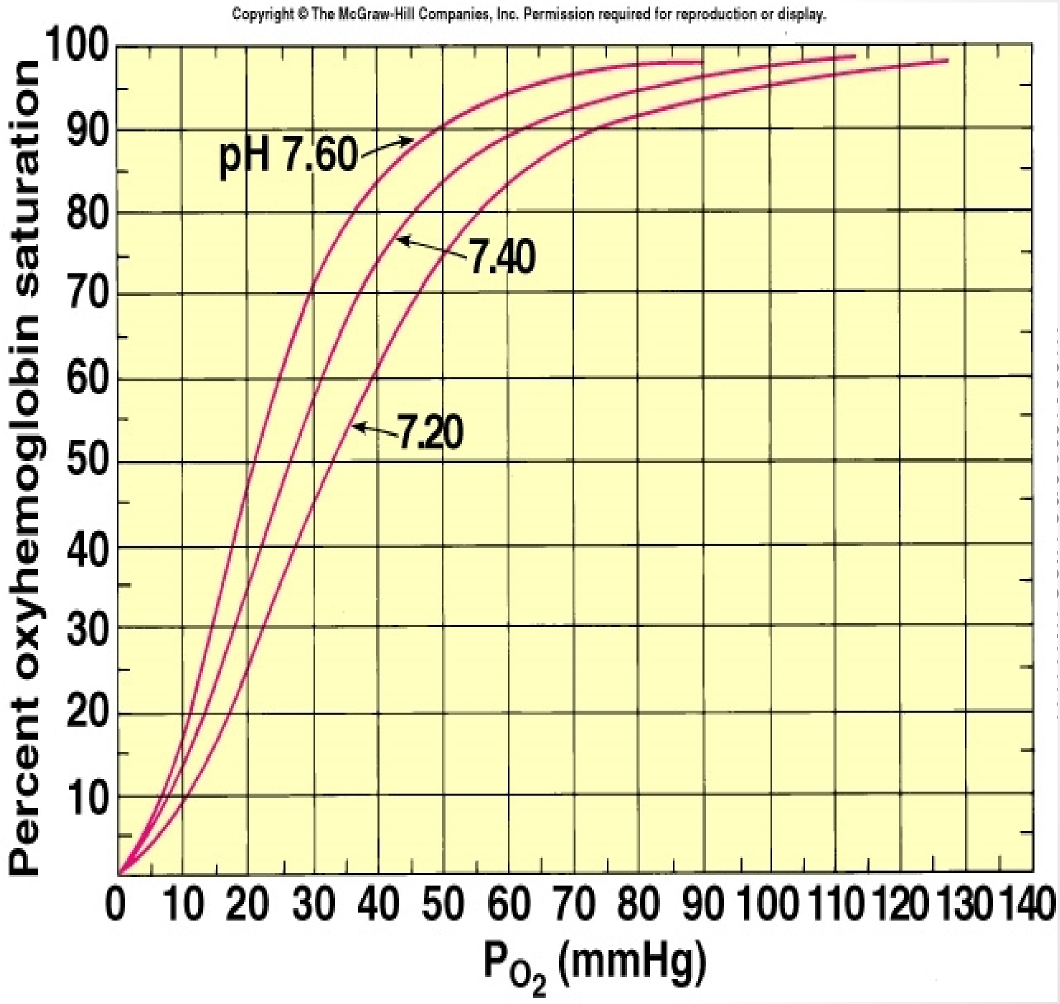
Effects of pH and Temperature

- The loading and unloading of O_2 influenced by the affinity of hemoglobin for 0_2.
- Affinity is decreased when pH is decreased.
- Increased temperature and 2,3-DPG:
  - Shift the curve to the right.



Effect of 2,3 DPG on 0_2 Transport

- Anemia:
  - RBCs total blood [hemoglobin] falls, each RBC produces greater amount of 2,3 DPG.
    - Since RBCs lack both nuclei and mitochondria, produce ATP through anaerobic metabolism.
- Fetal hemoglobin (hemoglobin f):
  - Has 2 γ-chains in place of the β-chains.
    - Hemoglobin f cannot bind to 2,3 DPG.
      - Has a higher affinity for 0_2.

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CO_2 Transport

- CO_2 transported in the blood:
  - HCO_3^- (70%).
  - Dissolved CO_2 (10%).
  - Carbaminohemoglobin (20%).

$$H_2O + CO_2 \overset{ca}{\longleftrightarrow} H_2CO_3$$

High P_{CO_2}

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Chloride Shift at Systemic Capillaries

- **$H_2O + CO_2 \longleftrightarrow H_2CO_3 \longleftrightarrow H^+ + HCO_3^-$**
- At the tissues, CO_2 diffuses into the RBC; shifts the reaction to the right.
  - Increased [HCO_3^-] produced in RBC:
    - HCO_3^- diffuses into the blood.
  - RBC becomes more +.
    - Cl^- attracted in (Cl^- shift).
  - H^+ released buffered by combining with deoxyhemoglobin.
- $HbCO_2$ formed.
  - Unloading of O_2.

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Carbon Dioxide Transport and Chloride Shift

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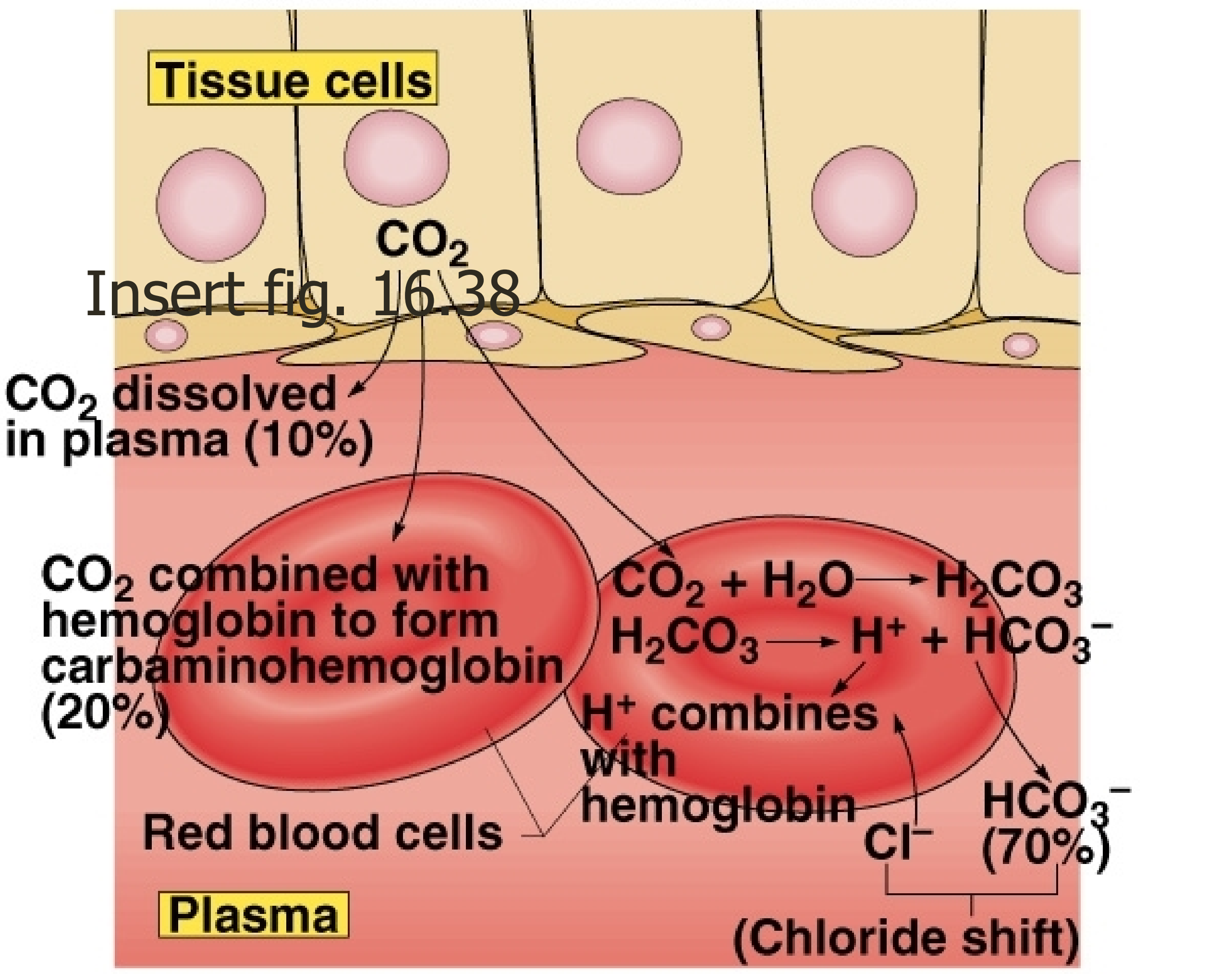

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At Pulmonary Capillaries

- **$H_20 + C0_2 \leftrightarrow H_2C0_3 \leftrightarrow H^+ + HC0_3^-$**
- At the alveoli, $C0_2$ diffuses into the alveoli; reaction shifts to the left.
- Decreased $[HC0_3^-]$ in RBC, $HC0_3^-$ diffuses into the RBC.
  - RBC becomes more -.
    - Cl^- diffuses out (reverse Cl^- shift).
- Deoxyhemoglobin converted to oxyhemoglobin.
  - Has weak affinity for H^+.
- Gives off $HbC0_2$.

Reverse Chloride Shift in Lungs

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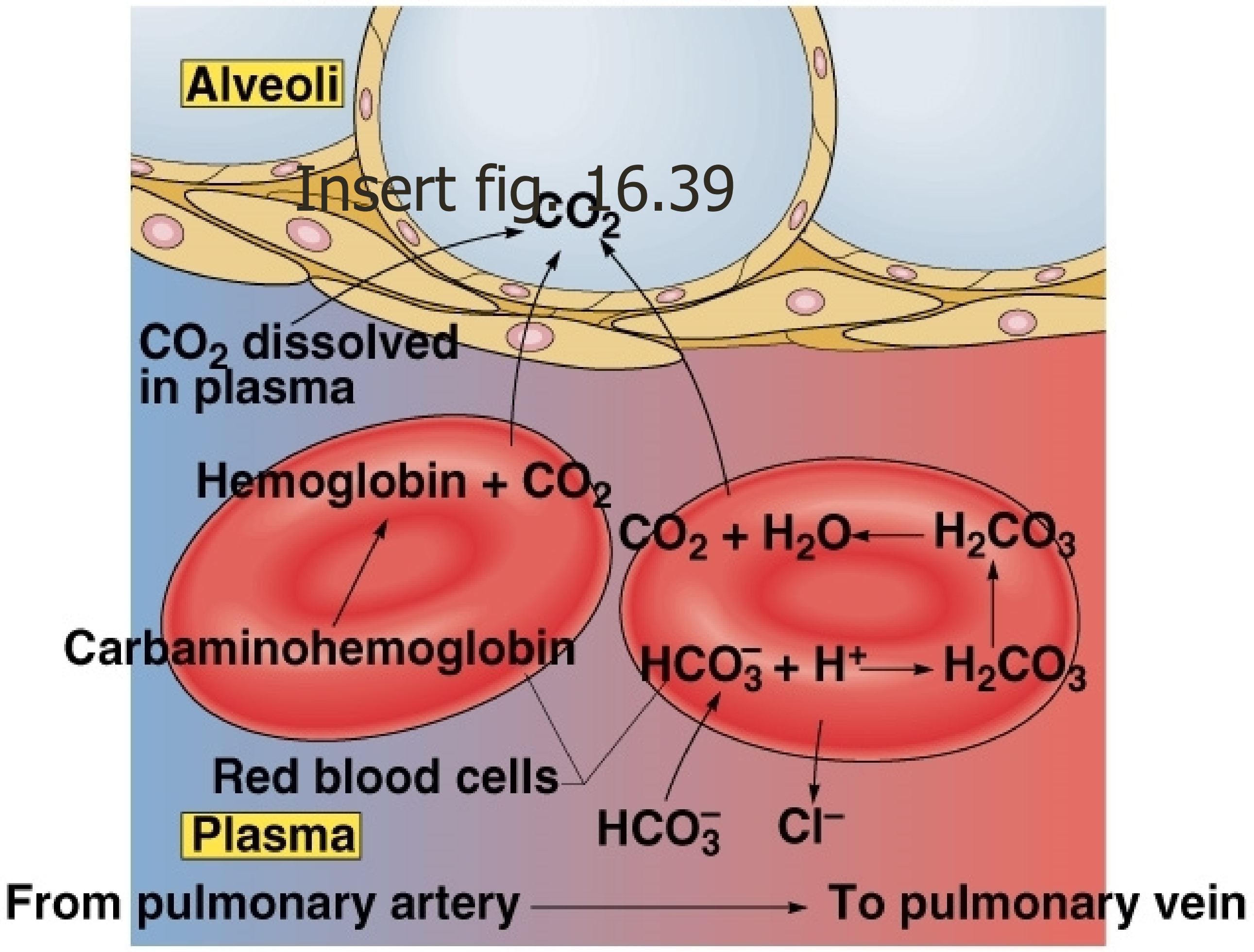

Respiratory Acidosis

- Hypoventilation.
- Accumulation of CO_2 in the tissues.
  - PCO_2 increases.
  - pH decreases.
  - Plasma HCO_3^- increases.

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Respiratory Alkalosis

- Hyperventilation.
- Excessive loss of CO_2.
  - PCO_2 decreases.
  - pH increases.
  - Plasma HCO_3^- decreases.

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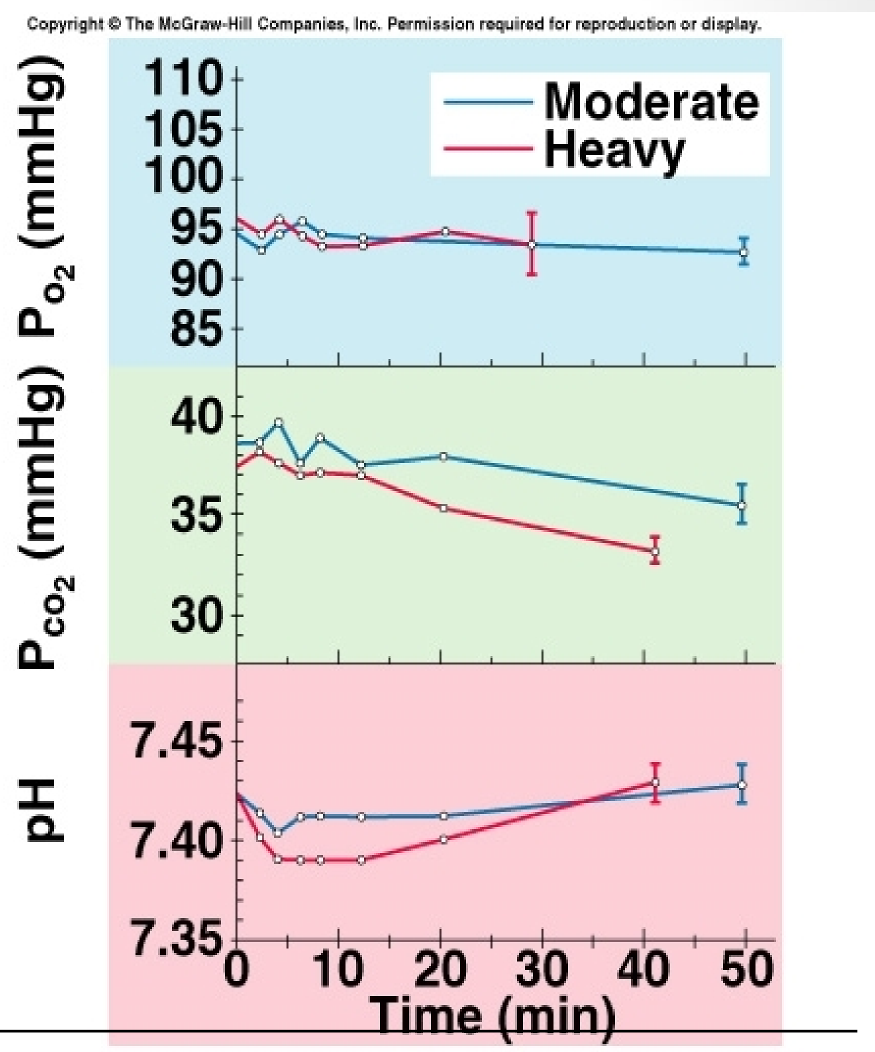
Ventilation During Exercise

- During exercise, breathing becomes deeper and more rapid.
- Produce > total minute volume.
- Neurogenic mechanism:
  - Sensory nerve activity from exercising muscles stimulates the respiratory muscles.
  - Cerebral cortex input may stimulate brain stem centers.
- Humoral mechanism:
  - P_{CO_2} and pH may be different at chemoreceptors.
  - Cyclic variations in the values that cannot be detected by blood samples.



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Acute Respiratory Failure

- Results from inadequate gas exchange
  - Insufficient O_2 transferred to the blood
    - Hypoxemia
  - Inadequate CO_2 removal
    - Hypercapnia

Classification of RF

- Type 1
- Hypoxemic RF
- PaO2 < 60 mmHg with normal or ↓ PaCO2
- ❑ Associated with acute diseases of the lung
- ❑ Pulmonary edema (Cardiogenic, noncardiogenic (ARDS), pneumonia, pulmonary hemorrhage, and collapse

- Type 2
- Hypercapnic RF
- PaCO2 > 50 mmHg
- Hypoxemia is common
- Drug overdose, neuromuscular disease, chest wall deformity, COPD, and Bronchial asthma

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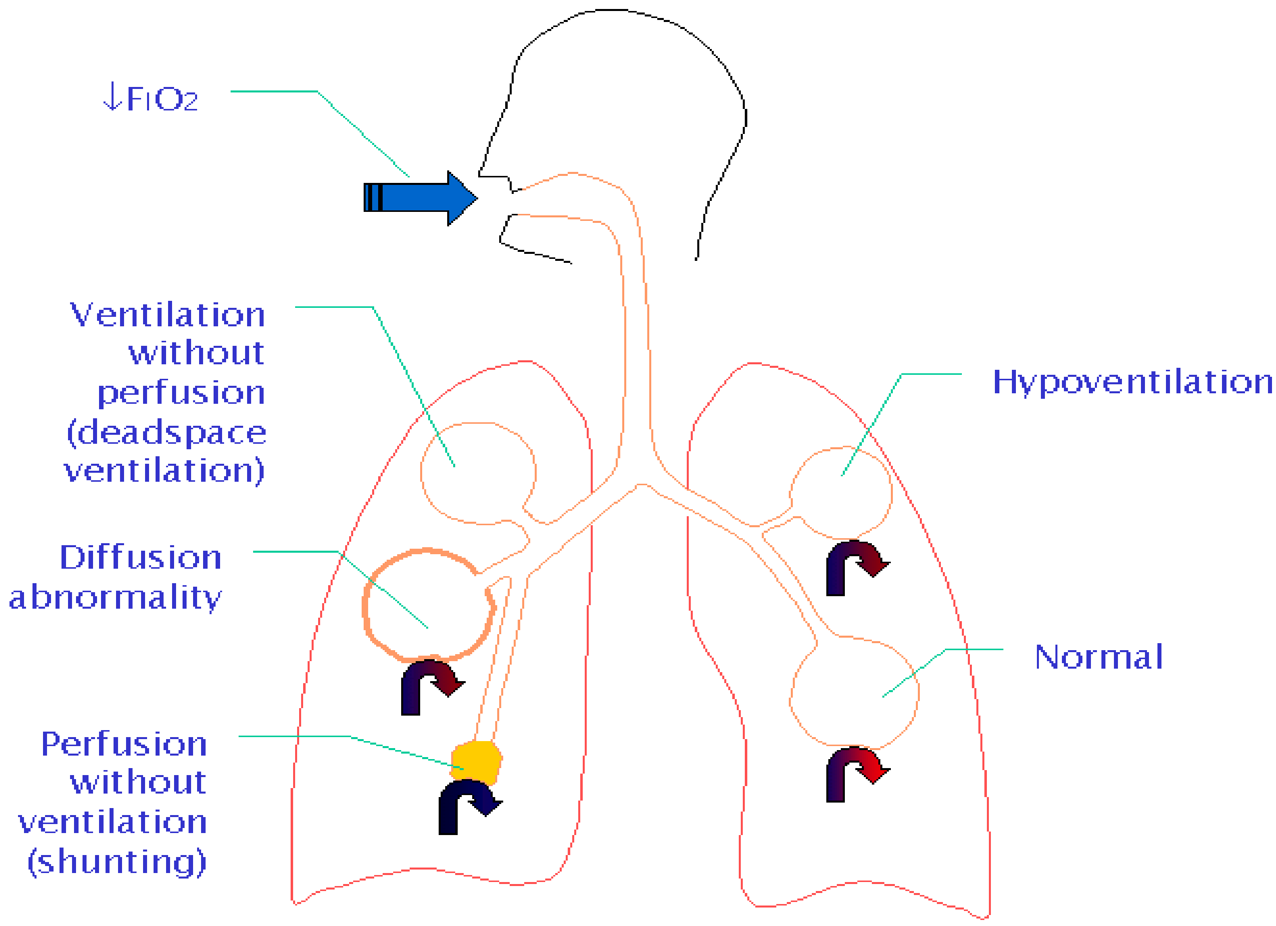
Pathophysiologic causes of Acute RF

- Hypoventilation
- V/P mismatch
- Shunt
- Diffusion abnormality



Classification of Respiratory Failure

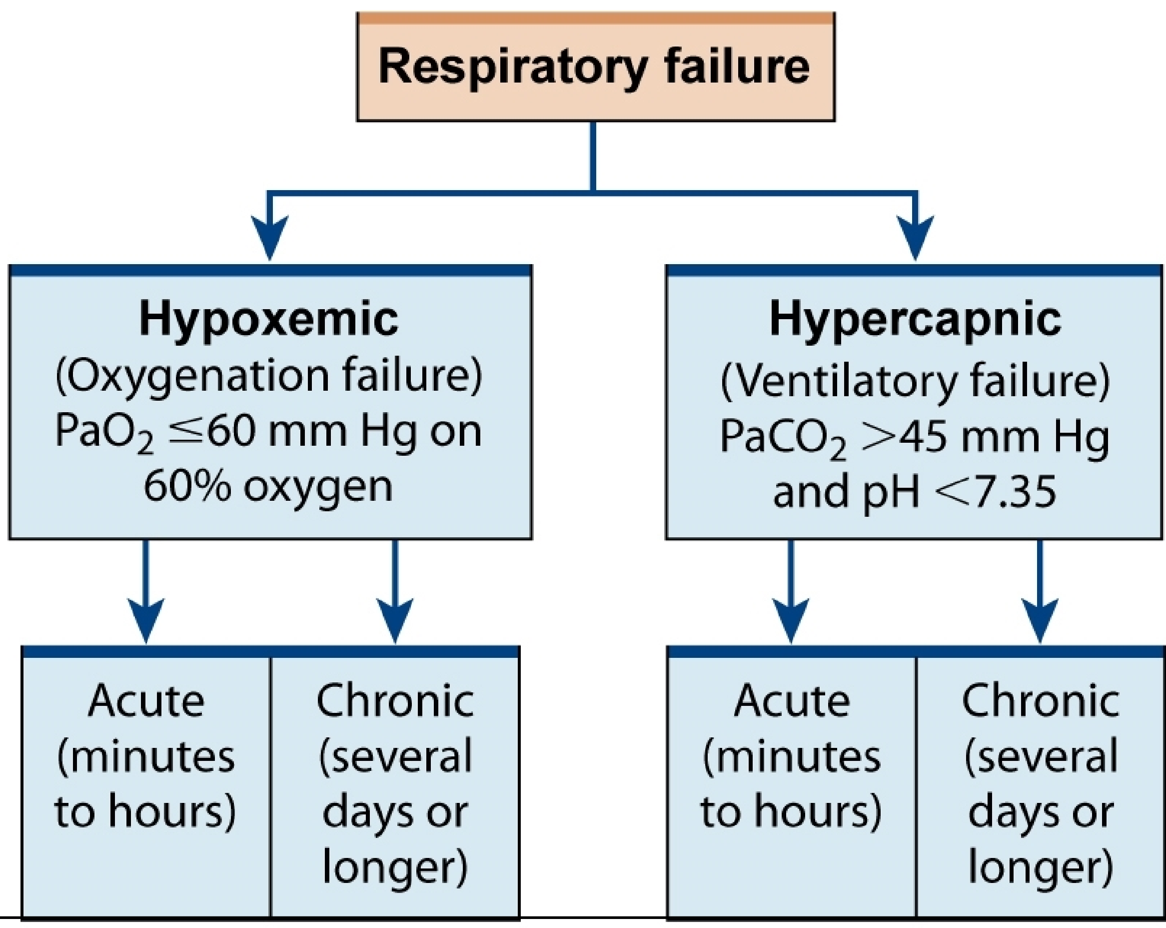


Fig. 68-2

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Mechanisms of hypoxemia

- Alveolar hypoventilation
- V/Q mismatch
- Shunt
- Diffusion limitation

Alveolar Hypoventilation

- Restrictive lung disease
- CNS disease
- Chest wall dysfunction
- Neuromuscular disease

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Perfusion without ventilation (shunting)

Intra-pulmonary

- Small airways occluded (e.g asthma, chronic bronchitis)
- Alveoli are filled with fluid (e.g pulm edema, pneumonia)
- Alveolar collapse (e.g atelectasis)

Dead space ventilation

- DSV increase:
- Alveolar-capillary interface destroyed e.g emphysema
- Blood flow is reduced e.g CHF, PE
- Overdistended alveoli e.g positive-pressure ventilation

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Diffusion limitation

- Severe emphysema
- Recurrent pulmonary emboli
- Pulmonary fibrosis
- Hypoxemia present during exercise

Diffusion Limitation

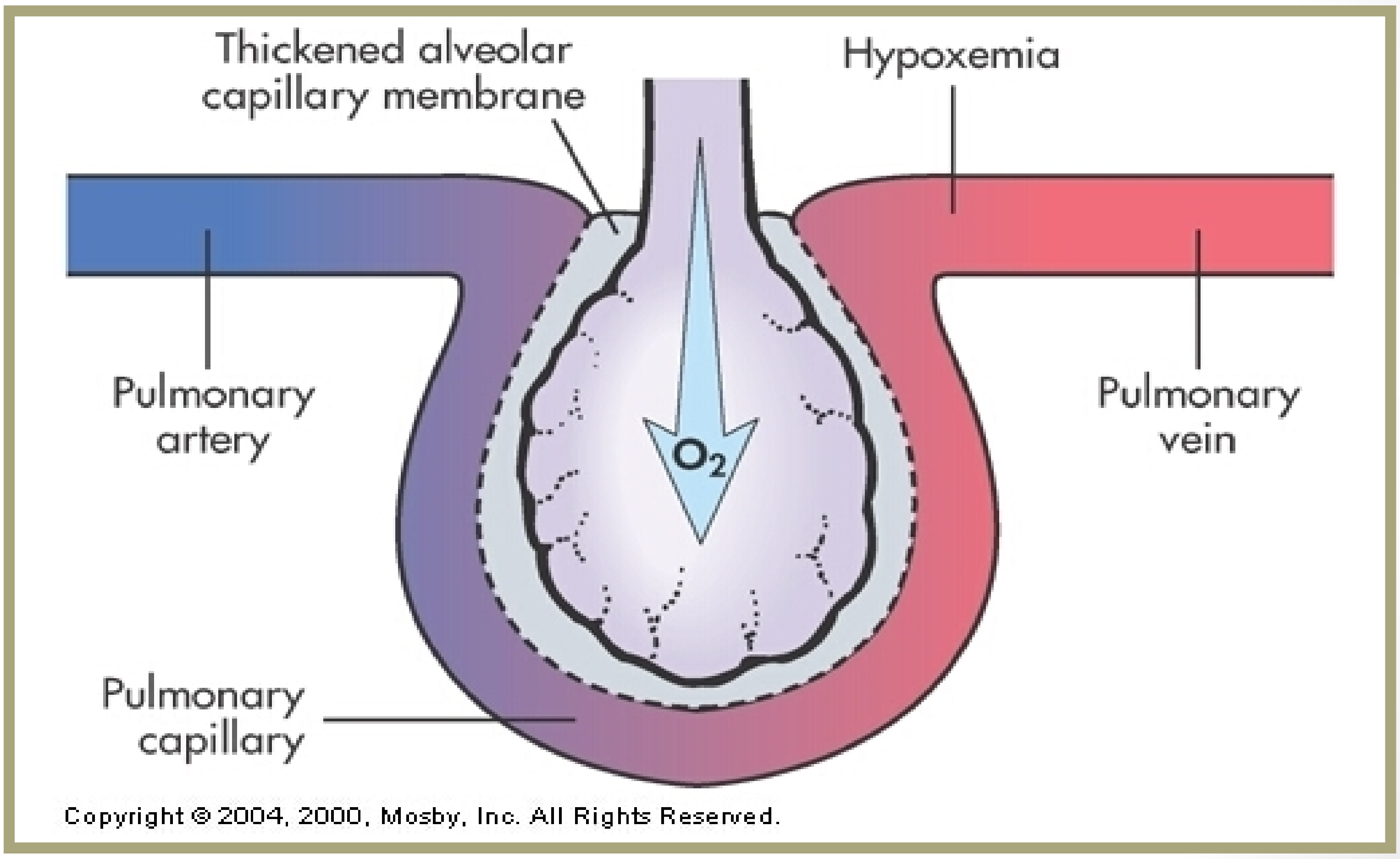

Fig. 68-5

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Hypercarbia

- Hypercarbia is always a reflection of inadequate ventilation
- $PaCO_2$ is
  - directly related to CO_2 production
  - Inversely related to alveolar ventilation

$$PaCO_2 = \frac{k \times VCO_2}{VA}$$

Hypercarbia

- When CO_2 production increases, ventilation increases rapidly to maintain normal $PaCO_2$
- Alveolar ventilation is only a fraction of total ventilation

$$V_A = V_E - V_D$$

- Increased deadspace or low V/Q areas may adversely effect CO_2 removal
- Normal response is to increase total ventilation to maintain appropriate alveolar ventilation

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Hypercapnic Respiratory Failure

- ***Imbalance between ventilatory supply and demand***

Etiology and Pathophysiology

- Airways and alveoli
  - Asthma
  - Emphysema
  - Chronic bronchitis
  - Cystic fibrosisS

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Etiology and Pathophysiology

- Central nervous system
  - Drug overdose
  - Brainstem infarction
  - Spinal chord injuries

Etiology and Pathophysiology

- Chest wall
  - Flail chest
  - Fractures
  - Mechanical restriction
  - Muscle spasm

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Etiology and Pathophysiology

- Neuromuscular conditions
  - Muscular dystrophy
  - Multiple sclerosis

Diagnosis of RF

1 – Clinical (symptoms, signs)

- **Hypoxemia**
- Dyspnea, Cyanosis
- Confusion, somnolence, fits
- Tachycardia, arrhythmia
- Tachypnea (good sign)
- Use of accessory ms
- Nasal flaring
- Recession of intercostal ms
- Polycythemia
- Pulmonary HTN, Corpulmonale, Rt. HF

- **Hypercapnia**
- ↑Cerebral blood flow, and CSF Pressure
- Headache
- Asterixis
- Papilloedema
- Warm extremities, collapsing pulse
- Acidosis (respiratory, and metabolic)
- ↓pH, ↑ lactic acid

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Respiratory Failure Symptoms

- CNS:
- Headache
- Visual Disturbances
- Anxiety
- Confusion
- Memory Loss
- Weakness
- Decreased Functional Performance

Respiratory Failure Symptoms

Cardiac:

Orthopnea

Peripheral edema

Chest pain

Other:

Fever, Abdominal pain, Anemia, Bleeding

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Clinical

- Respiratory compensation
- Sympathetic stimulation
- Tissue hypoxia
- Haemoglobin desaturation

Clinical

- Respiratory compensation
  - Tachypnoea RR > 35 Breath /min
  - Accessory muscles
  - Recesssion
  - Nasal flaring
- Sympathetic stimulation
- Tissue hypoxia
- Haemoglobin desaturation

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Clinical

- **Respiratory compensation**
- **Sympathetic stimulation**
  - ↑HR
  - ↑BP
  - Sweating

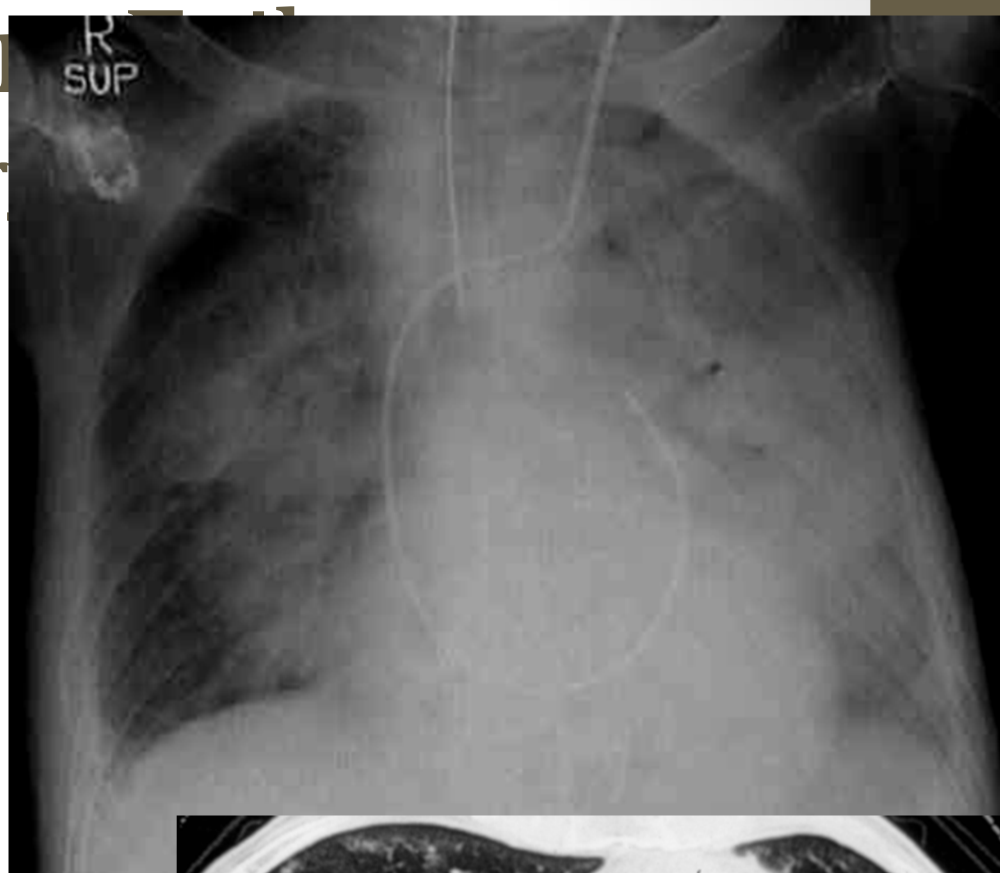
Tissue hypoxia

  - Altered mental state
  - ↓HR and ↓BP (late)

- **Haemoglobin desaturation** cyanosis

Respirator

Laborator



Arterial blood gas

PaO2

PaCO2

PH

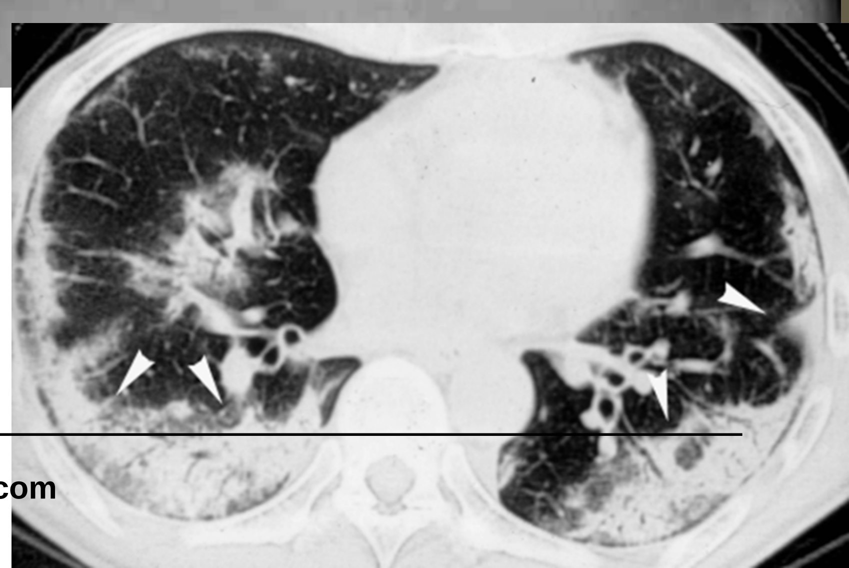
Chest imaging

Chest x-ray

CT sacn

Ultrasound

Ventilation–perfusion scan



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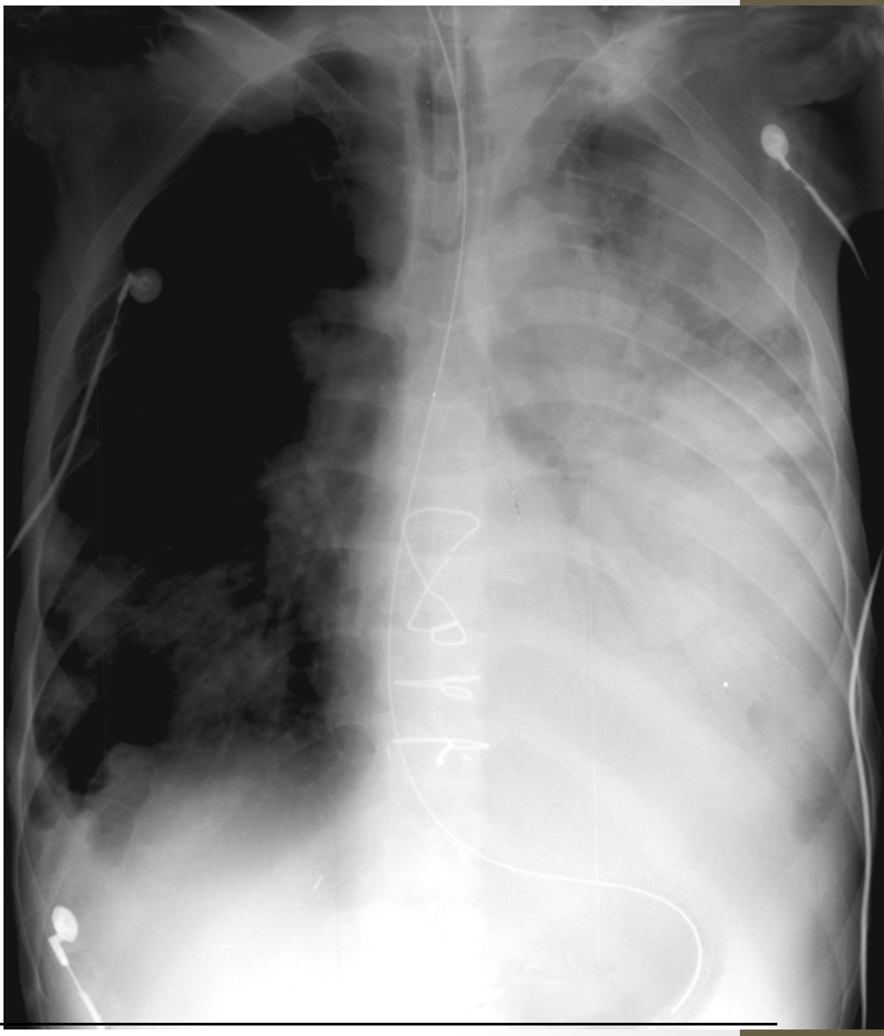
Management of ARF

- ICU admission
- 1 -Airway management
  - Endotracheal intubation:
    - Indications
      - Severe Hypoxemia
      - Altered mental status
  - Importance
    - precise O2 delivery to the lungs
    - remove secretion
    - ensures adequate ventilation

Management of ARF



- Correction of hypoxemia
  - O2 administration via nasal prongs, face mask, intubation and Mechanical ventilation
  - Goal: Adequate O2 delivery to tissues
  - PaO2 = > 60 mmHg
  - Arterial O2 saturation >90%

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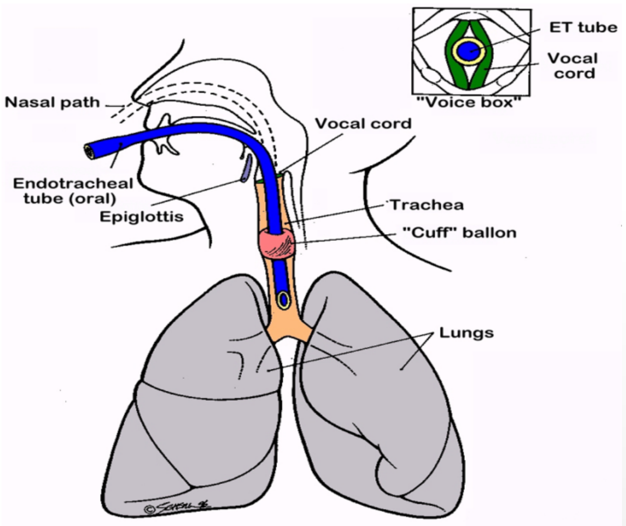
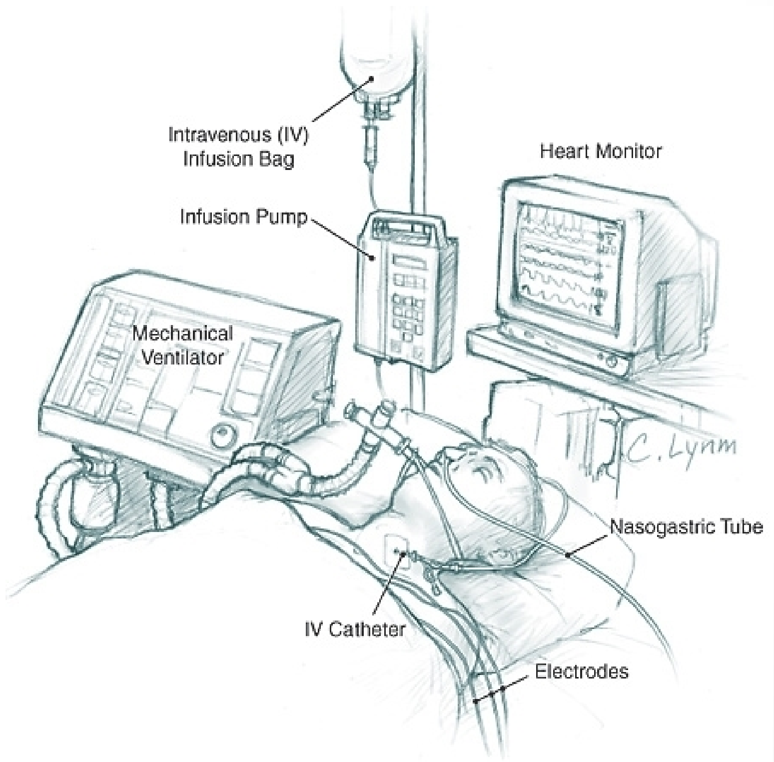
Indications for intubation and mechanical ventilation

- Innability to protect the airway
- Respiratory acidosis (pH<7.2)
- Refractory hypoxemia
- Fatigue/increased metabolic demands
  - impending respiratory arre
- Pulmonary toilet



Management of ARF

- Mechanical ventilation
  - Increase PaO2
  - Lower PaCO2
  - Rest respiratory ms (respiratory ms fatigue)
  - Ventilator
    - Assists or controls the patient breathing
  - The lowest FIO2 that produces SaO2 >90% and PO2 >60 mmHg should be given to avoid O2 toxicity



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Management of ARF

- PEEP (positive End-Expiratory pressure
- Used with mechanical ventilation
  - Increase intrathoracic pressure
  - Keeps the alveoli open
  - Decrease shunting
  - Improve gas exchange
- Hypoxemic RF (type 1)
  - ARDS
  - Pneumonias

Management of ARF

- Noninvasive Ventilatory support (IPPV)
- Mild to moderate RF
- Patient should have
  - Intact airway,
  - Alert, normal airway protective reflexes
- Nasal or full face mask
  - Improve oxygenation,
  - Reduce work of breathing
  - Increase cardiac output
- AECOPD, asthma, CHF

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Management of ARF

- Treatment of the underlying causes
- After correction of hypoxemia, hemodynamic stability
- Antibiotics
  - Pneumonia
  - Infection
- Bronchodilators (COPD, BA)
  - Salbutamol
    - reduce bronchospasm
    - airway resistance

Management of ARF

- Treatment of the underlying causes
- Physiotherapy
  - Chest percussion to loosen secretion
  - Suction of airways
  - Help to drain secretion
  - Maintain alveolar inflation
  - Prevent atelectasis, help lung expansion

Management of ARF

- Weaning from mechanical ventilation
  - Stable underlying respiratory status
  - Adequate oxygenation
  - Intact respiratory drive
  - Stable cardiovascular status
  - Patient is a wake, has good nutrition, able to cough and breath deeply

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