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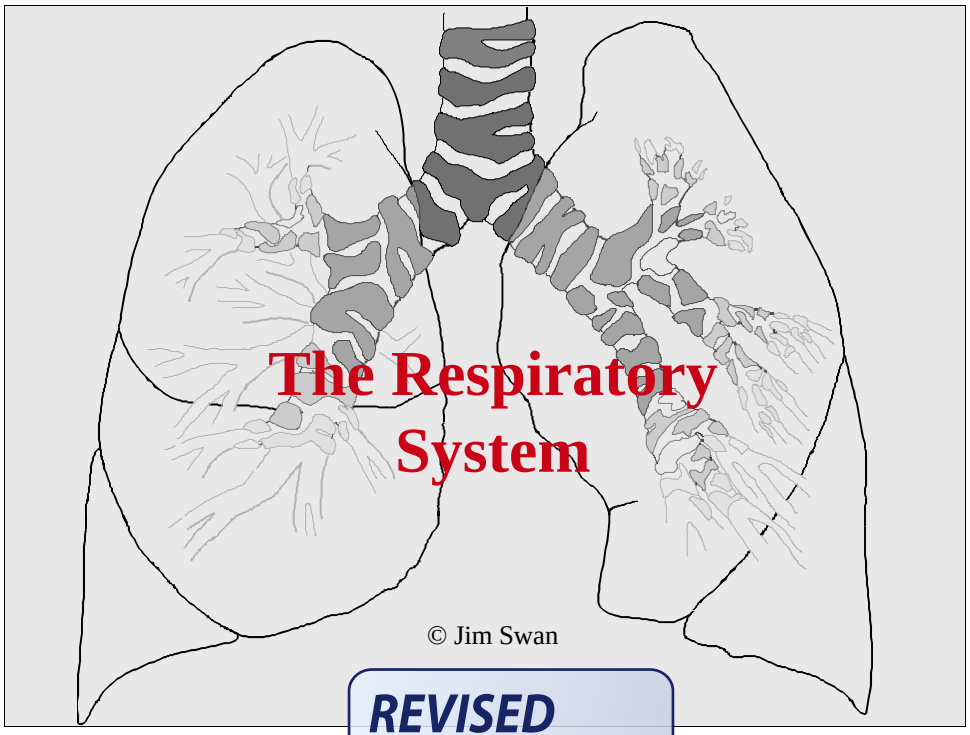

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Respiratory System Topics

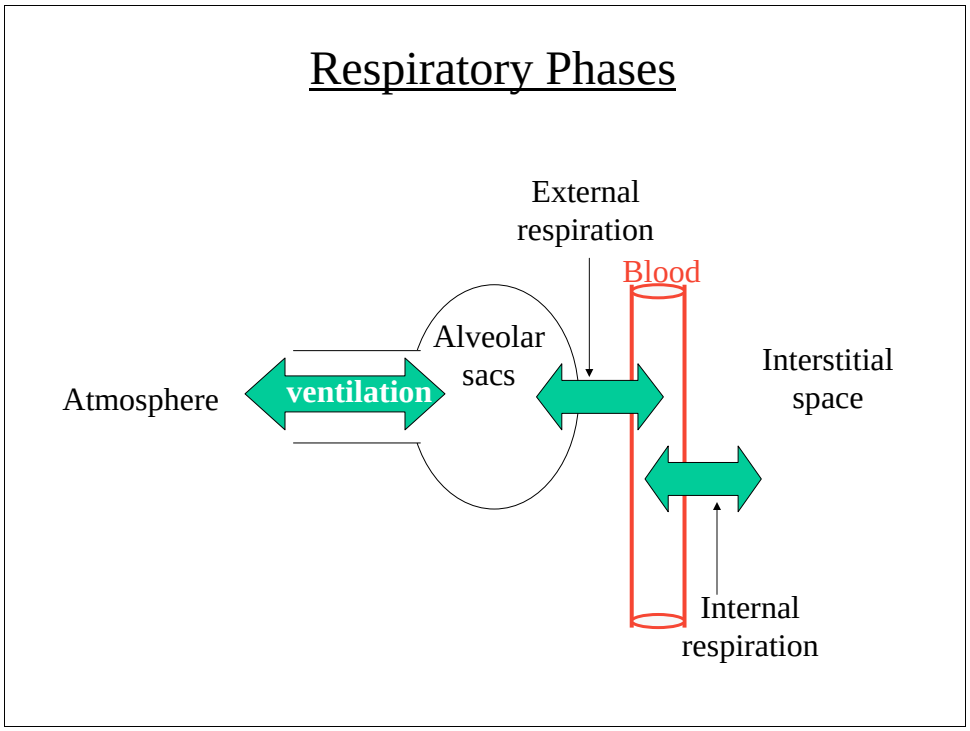
1) Ventilation (breathing) – the exchange of gases between the atmosphere and the lungs.

2) External respiration – the exchange of gases between the lungs and the blood.

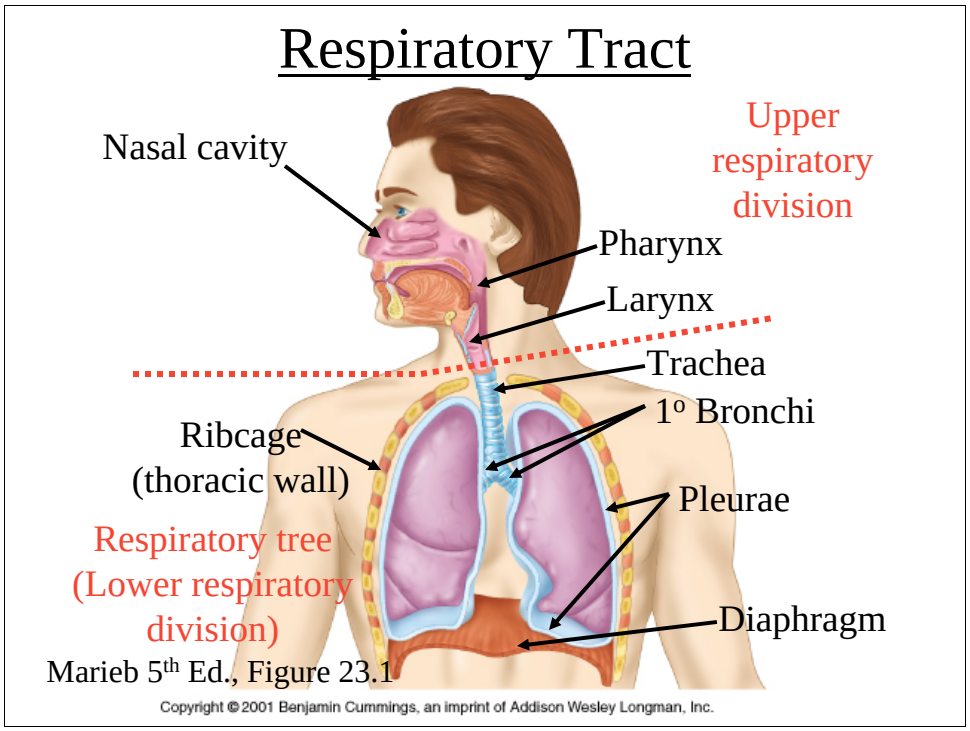
3) Internal respiration – the exchange of gases between the blood and the systemic tissues.

4) Cellular respiration – aerobic metabolism

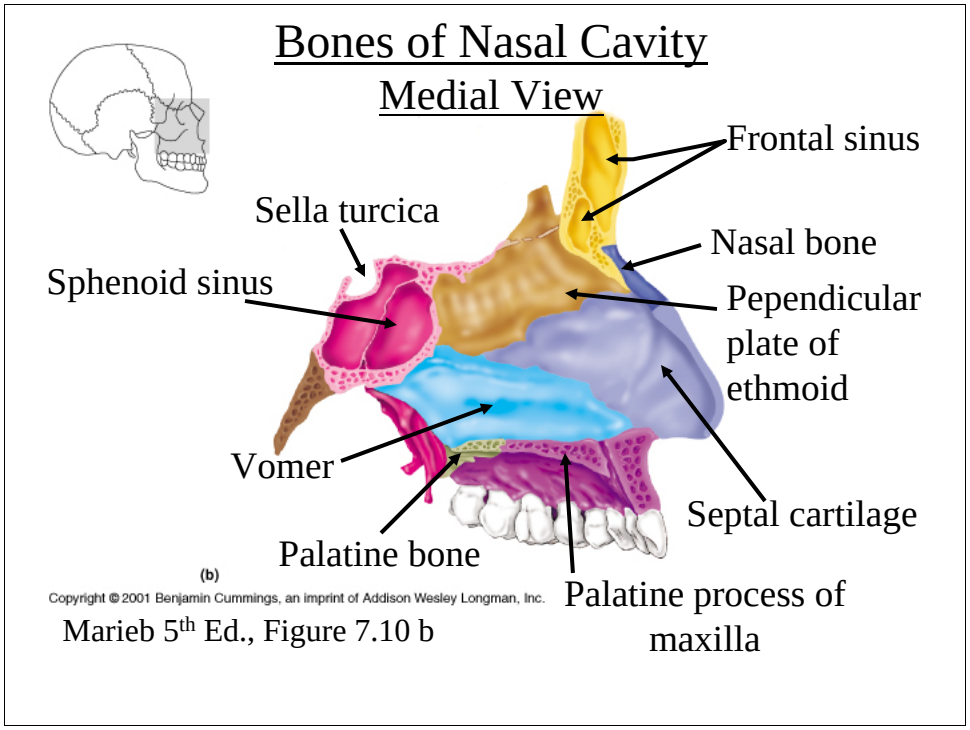
We will cover the first three topics in this unit. Cellular respiration will be briefly touched upon in the section on **Nutrition and Metabolism.**



The above is a diagramatic representation of the three respiratory topics to be covered.

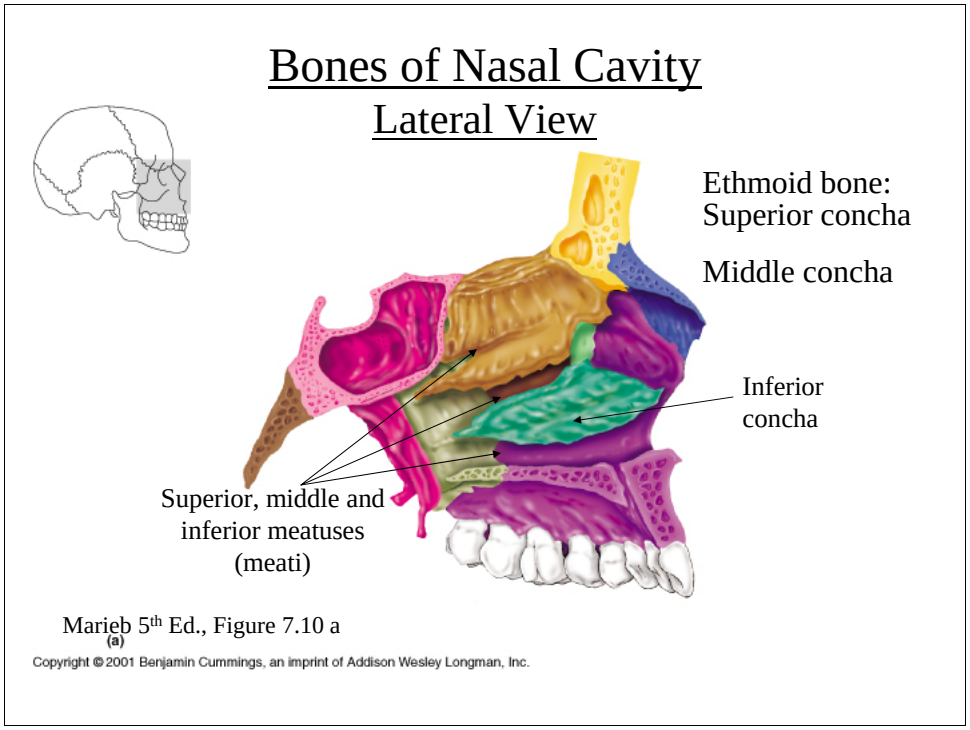


The two basic divisions of the respiratory tract are shown above.



Marieb 5th Ed., Figure 7.10 b

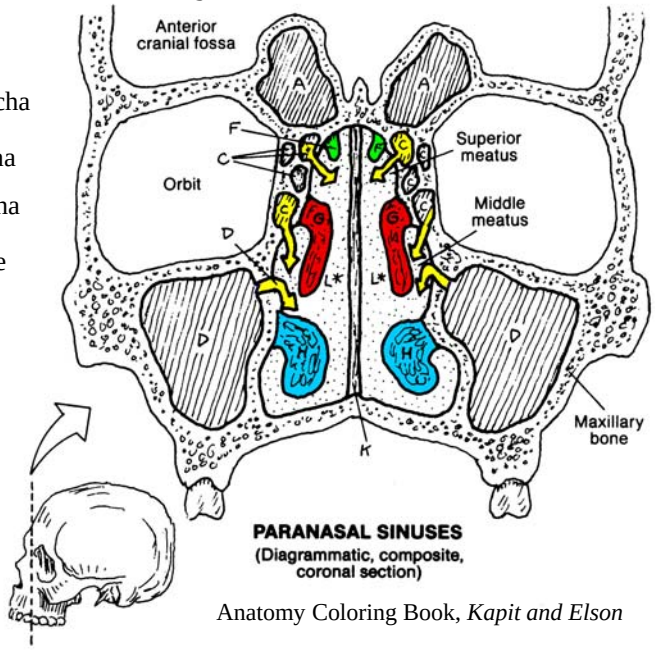
Note the relationship of paranasal sinuses to the nasal cavity.



The **conchae** force the air to pass through narrow channels, the **meati**. This exposes the air to the mucosal surface.

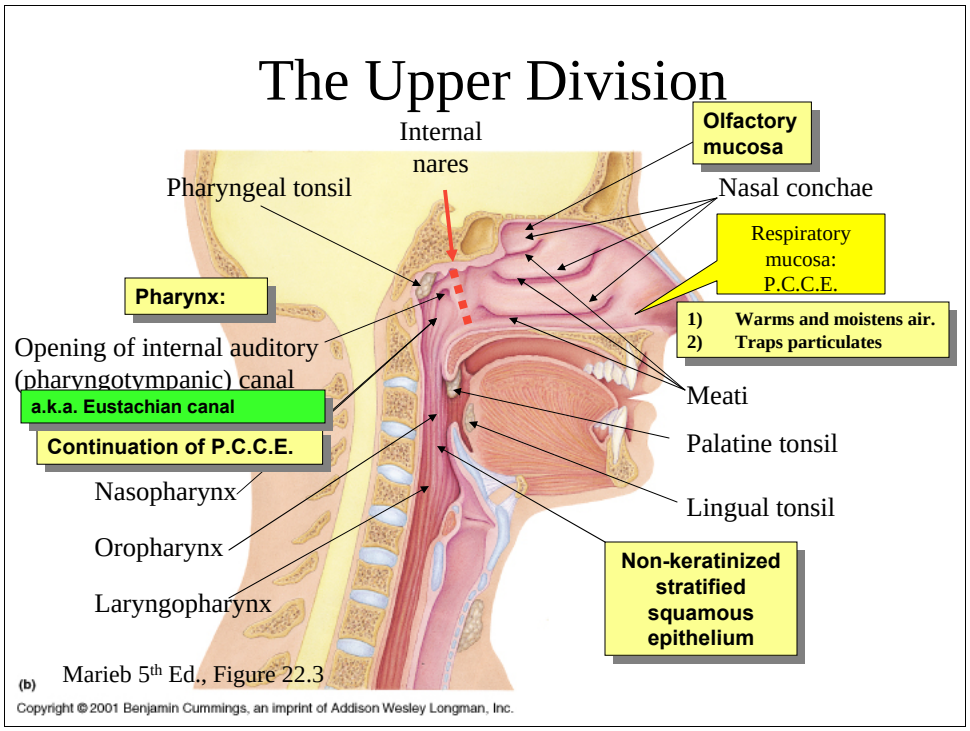
Nasal Cavity, Coronal Section

- Superior concha
- Middle concha
- Inferior concha
- Sinus drainage



Anatomy Coloring Book, *Kapit and Elson*

The **paranasal sinuses** drain into the nasal cavity. Mucosa is a continuous lining throughout the sinuses and nasal passages, and into the nasopharynx.

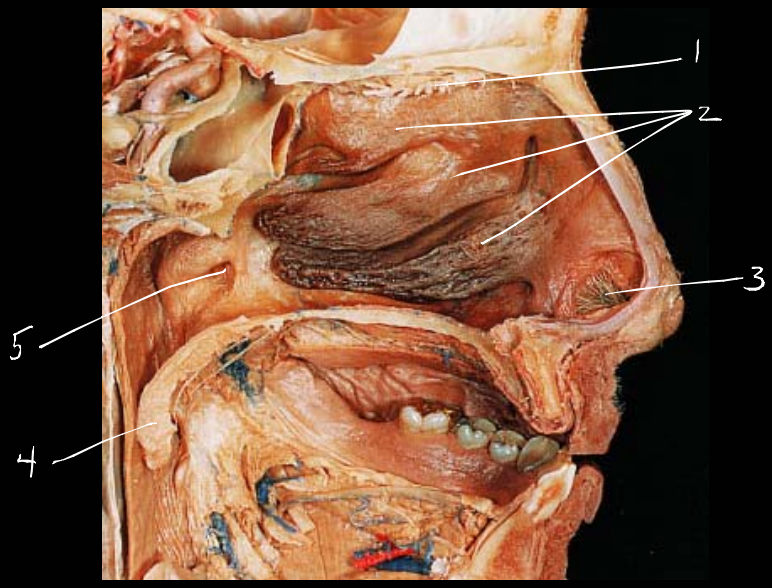


The **upper respiratory division** (See Figure 22.3) includes the nasal cavity and **pharynx**. Air enters the nasal cavity through the **external nares** and passes through the narrow channels (meati) created between the **nasal conchae**. This exposes the air to the mucosa which warms and moistens the air as it passes through the entire system. The mucosa also removes particulates, dust, pollen, etc. and moves it toward the esophagus. In the nasal cavity the respiratory mucosa is partly ciliated and partly non ciliated and the cilia beat downward. The upper portion of the nasal cavity has olfactory receptors whose nerves pass through the foramina in the **cribriform** plate to the olfactory bulb of the brain. The nasal cavity narrows at the internal nares before joining with the pharynx. The pharynx is divided into three portions: the **nasopharynx**, the **oropharynx** and the **layngopharynx** which connect to the nasal cavity, oral cavity and larynx respectively.

The nasopharynx has the pharyngeal tonsils (lymph nodes) and the opening into the **eustachian canal** (a.k.a. pharyngotympanic tube or internal auditory canal). This canal allows equalization of pressure between the atmosphere and the middle ear. This is important when increasing or decreasing in altitude and when atmospheric pressure changes for any reason. Inability to equalize pressure can impair hearing as occurs when the mucosa is inflamed due to a respiratory infection.

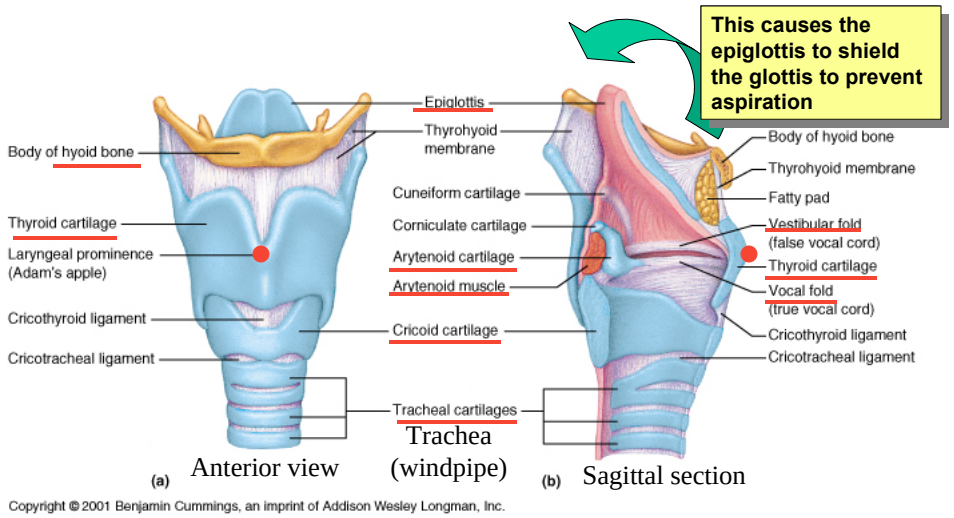
The lining continues to be ciliated into the **nasopharynx** and then changes to non-keratinized stratified squamous in the **oropharynx** and **laryngopharynx** where protection from ingested food is important.

Upper Respiratory Division



Identify the structures labeled above

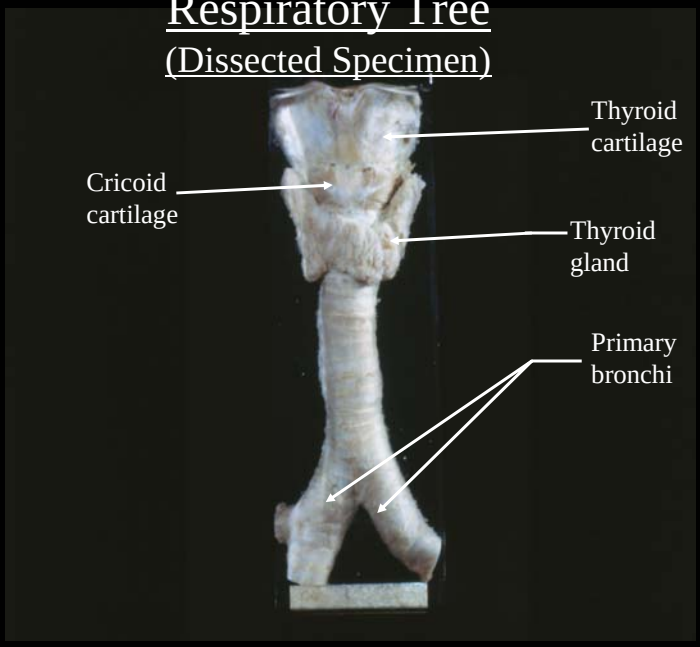
The Larynx (*lair-inks*)



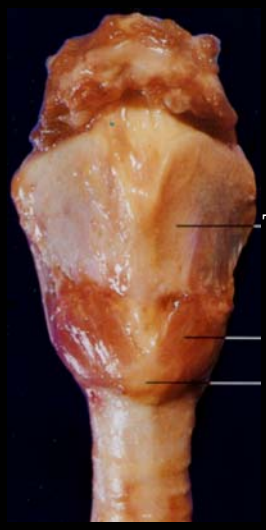
Marieb 5th Ed., Figure 22.4

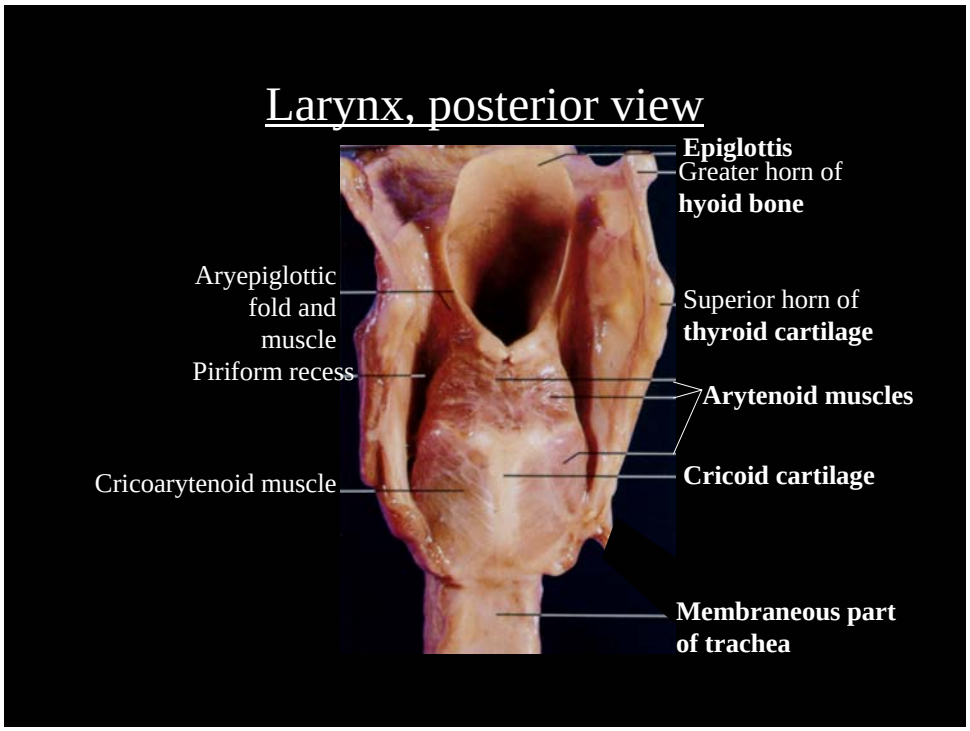
The **larynx** lies atop the respiratory tree and contains the **voice box**. Supporting the larynx is the **hyoid** bone which connects to the tongue above and to the thyroid cartilage below. When swallowing occurs, the hyoid bone, which does not articulate with any other bones, hinges upward and the larynx tilts backward. This causes the **epiglottis** to more effectively shield the **glottis** and prevent aspiration of food or liquid. The thyroid cartilage is the largest laryngeal cartilage and projects anteriorly as the "Adam's Apple". The thyroid cartilage is open posteriorly and the vocal folds (true vocal cords) run along its inside antero-posteriorly. Men tend to have deeper voices and more pronounced "Adam's Apples" due to the increased length of the thyroid cartilage and vocal fold. Tension on the vocal cords determines the pitch of the voice as air is pushed up between them. This tension in turn is controlled by cartilages attached to the back of the cords called the **arytenoid cartilages**. These cartilages swivel to change vocal cord tension. Vocal cord tension is controlled by **arytenoid and other muscles** [See laryngeal muscles] which contract or relax to increase (for high pitches) or decrease (for low pitches) tightness. Lying above the vocal folds are the vestibular folds (false vocal cords) which are narrow ridges protecting the vocal cords from aspirated materials. The glottis is the opening between the vocal cords and the passageway for air into the trachea.

Respiratory Tree
(Dissected Specimen)

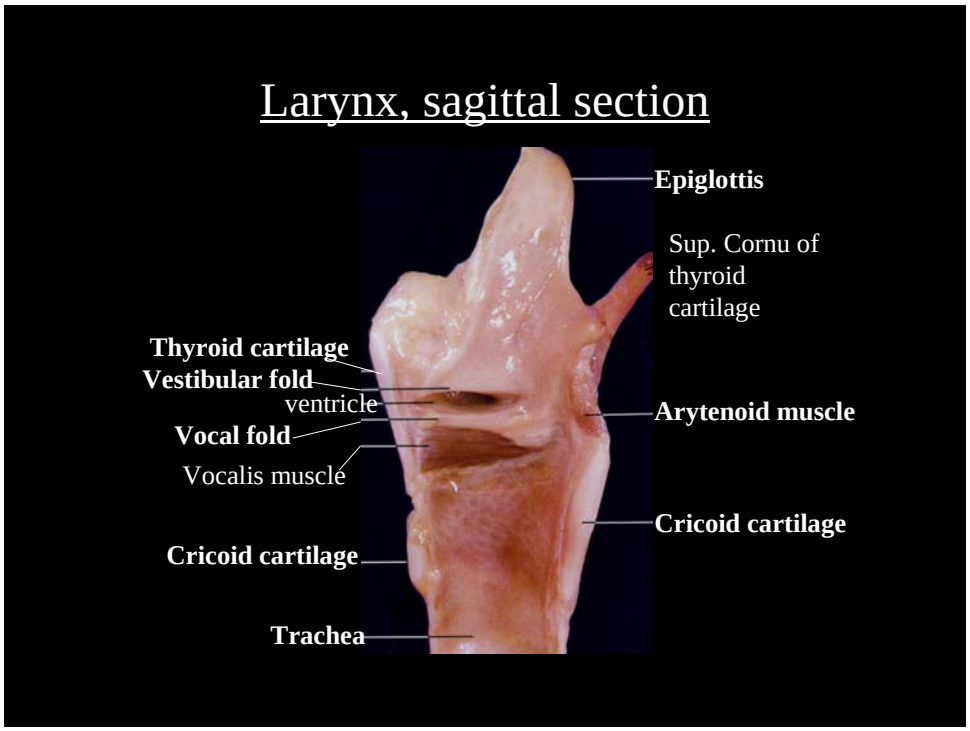


Larynx, anterior view

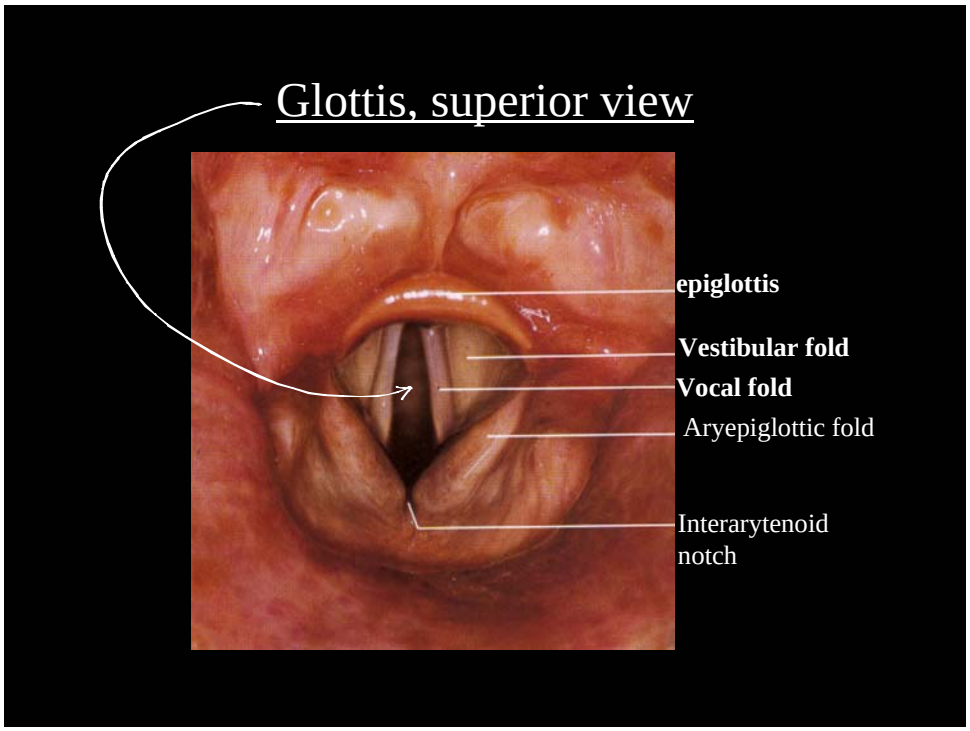




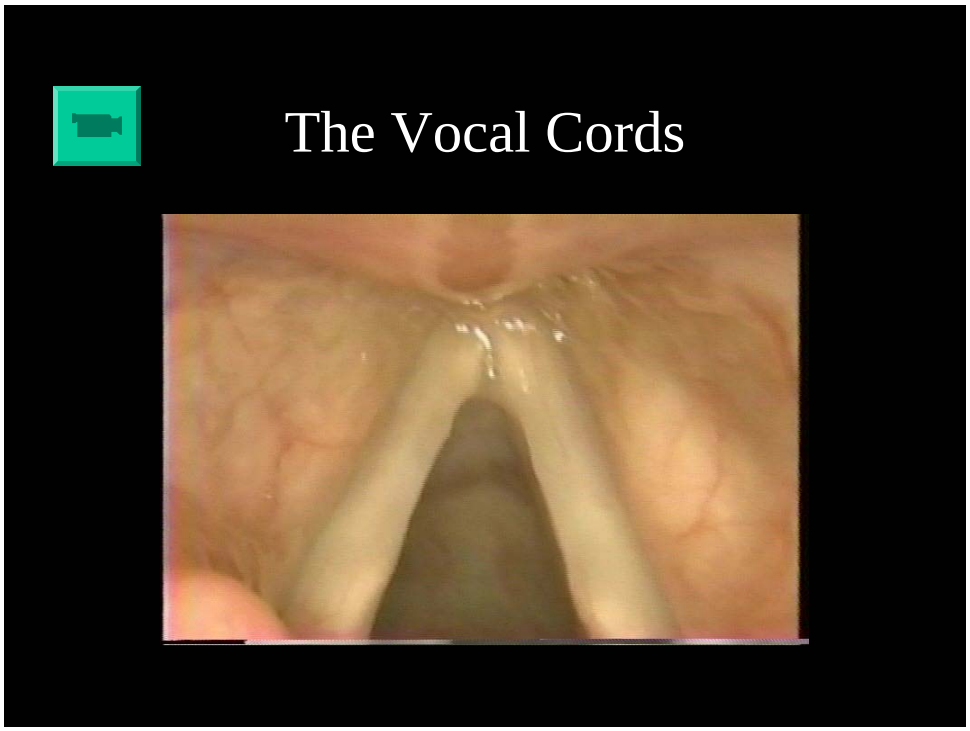
Notice the “tulip shape” of the epiglottis and aryepiglottic folds. These structures curl backwards when swallowing to prevent aspiration.



The vocal folds (true vocal chords) are protected by the overhanging vestibular folds.

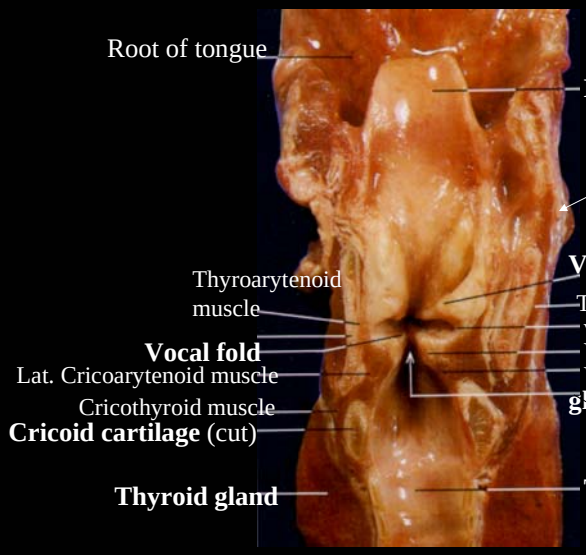


The glottis is the opening between the vocal folds, into the respiratory tree.

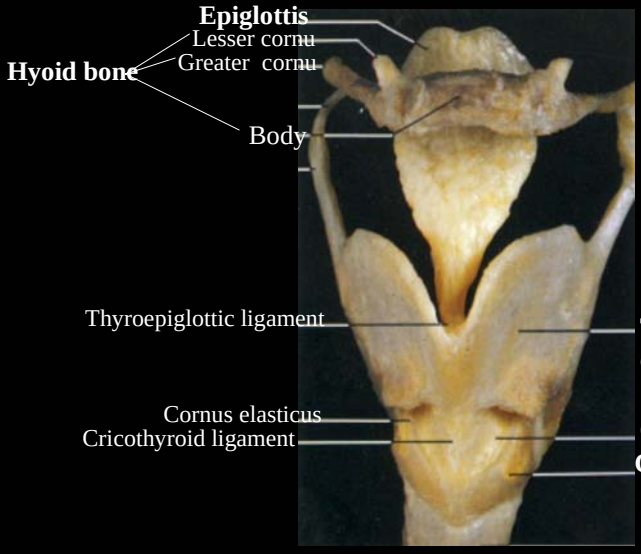


Listen to the function of the vocal folds in action.

Larynx, coronal section



Laryngeal Cartilages, anterior view



Laryngeal Cartilages, posterior view

Epiglottis

Thyroepiglottic lig.

Thyroid cartilage

Cricoarytenoid joint

Cricoid cartilage

Trachea

Lesser cornu of hyoid bone

Greater cornu of hyoid bone

Thyrohyoid ligament

Sup. Cornu of thyroid cartilage

Corniculate cartilage

Arytenoid cartilage

Post. Cricoarytenoid lig.

Cricothyroid joint

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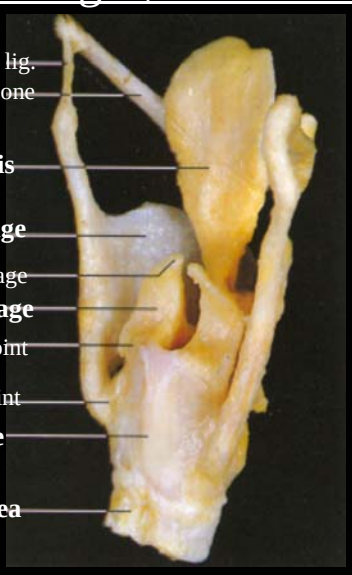
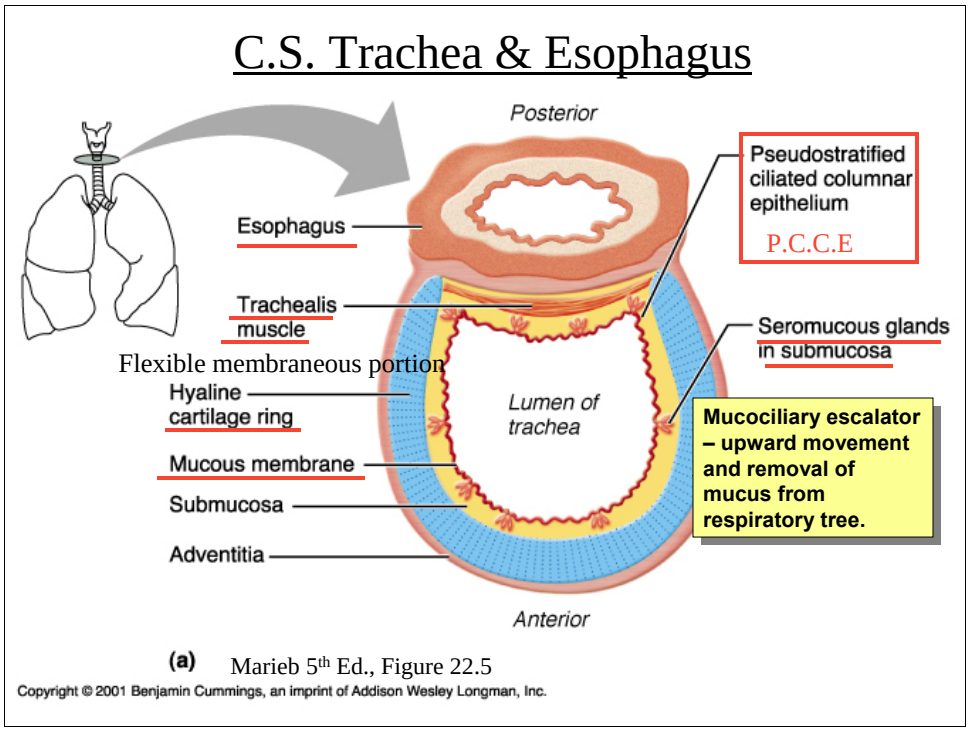
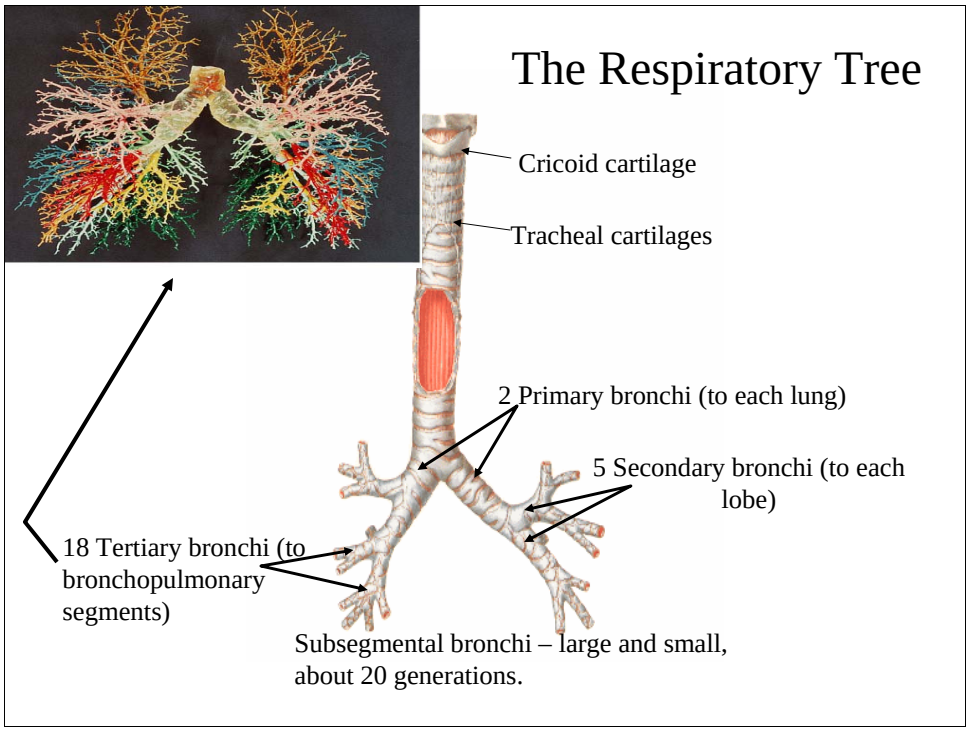
Laryngeal Cartilages, lateral view



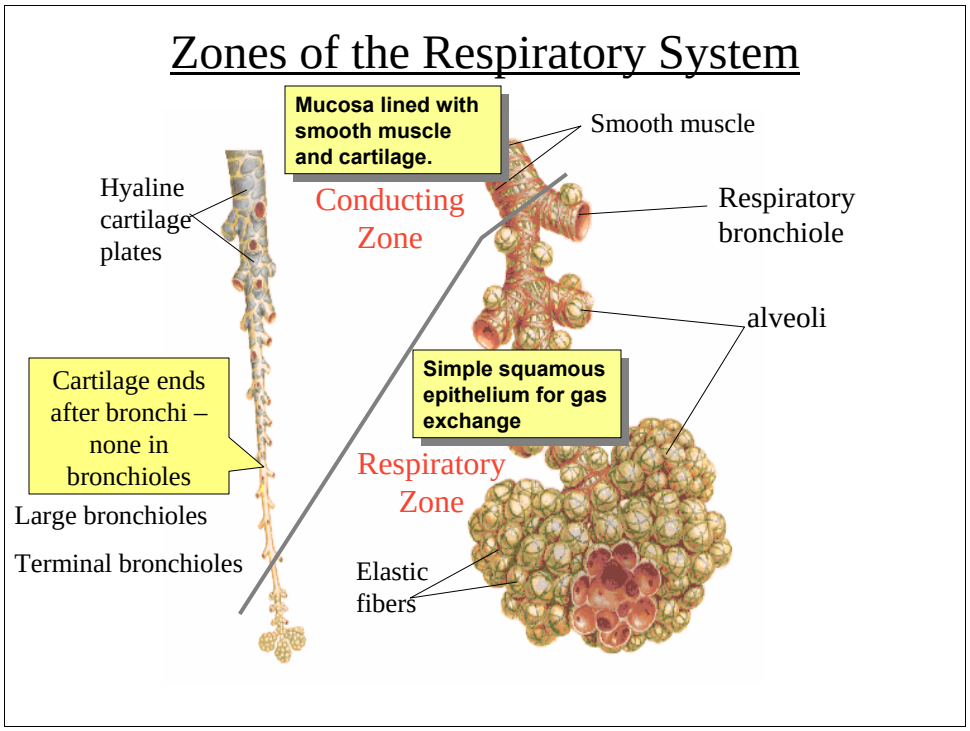


The **trachea**, See or windpipe, is the beginning of the respiratory tree. It has cartilages shaped like Cs (or Us) with the open portion posterior. The trachea is lined with pseudostratified ciliated columnar epithelium (p.c.c.e.) which continues into the bronchi. The cilia of this mucosa beat upward to carry particulates up and out into the esophagus. The submucosa of the trachea contains seromucous glands to produce an abundance of mucus for the **mucociliary escalator**, the mechanism by which the mucus is moved up and out of the respiratory tract. The membraneous portion of the trachea on the posterior side contains the majority of the trachealis muscle and is flexible to accommodate the esophagus nestled immediately behind the trachea.



The Conducting Zone - area which conducts air into the lungs. No gas transport occurs here. Also called the **anatomical dead space**.

The trachea, branches into the **primary bronchi** (which lead to each lung, then branching to the **secondary bronchi** leading to the lobes (2 on the left, three on the right), then the **tertiary bronchi** to the 18 broncho-pulmonary segments (8 on the left, 10 on the right). Each broncho-pulmonary segment includes a branch of the pulmonary artery, pulmonary vein, and a tertiary bronchus. These segments act as structural and functional units in the lung to maintain blood flow and gas transport. The cartilages change from C-rings to plate cartilages in the bronchi while the walls get thinner. PCCE continues. Tertiary bronchi lead to the **large bronchioles**. Large bronchioles have little or no cartilage and the mucosa becomes simple ciliated columnar epithelium. Large bronchioles lead to the **terminal bronchioles** which have simple cuboidal epithelium. **Goblet cells** continue through the mucosa providing continued mucus secretion.



The difference between the respiratory and conducting zones is their function: the **conducting zone** transports the air into and out of the lungs; the **respiratory zone** allows gas exchange between the lungs and the blood.

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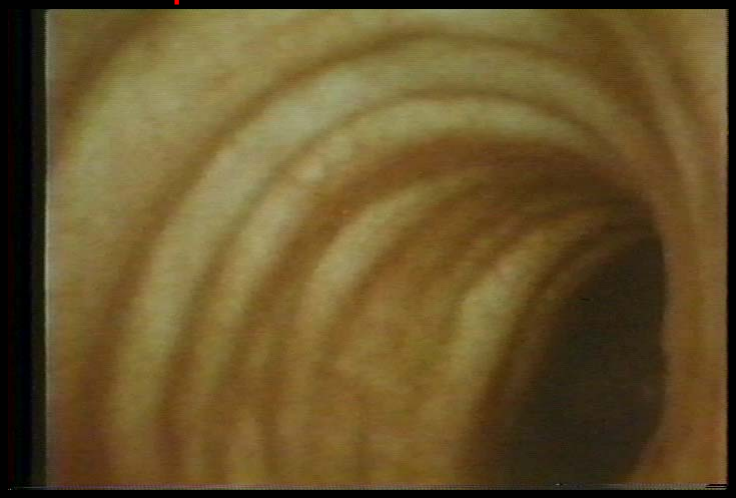
Respiratory Tree

Respiratory Tract Video Clip



Conducting Zone Hierarchy

Nasal mucosa — P.C.C.E. (partly ciliated) cilia beat down

Pharynx:

- nasopharynx — P.C.C.E. (partly ciliated) cilia beat down
- oropharynx — Non-keratinized stratified squamous
- laryngopharynx — Non-keratinized stratified squamous

Larynx — P.C.C.E. (partly ciliated) cilia beat up

Trachea — P.C.C.E. (partly ciliated) cilia beat up

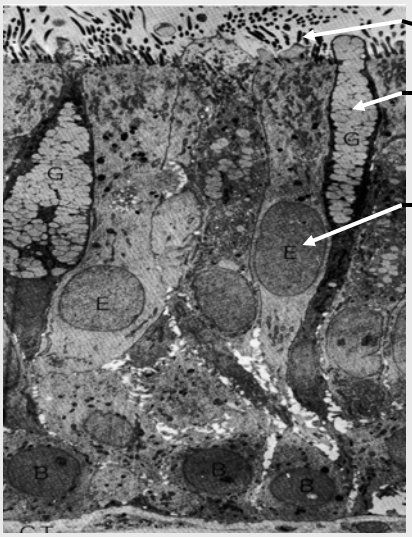
Bronchi (1^{o},2^{o},3^{o}, sub) — P.C.C.E. (partly ciliated) cilia beat up

Large bronchioles — Simple ciliated columnar

Terminal bronchioles — Simple cuboidal

Tracheal Lining

Transmission Electron Micrograph (TEM)



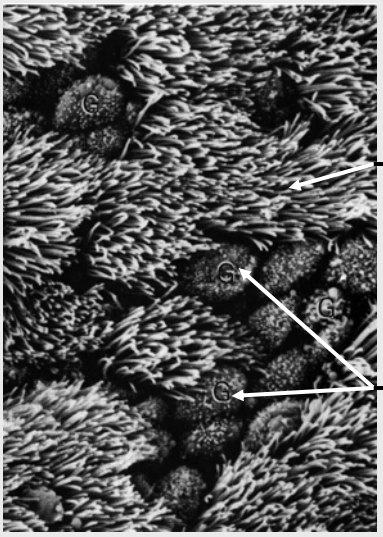
cilia

Goblet cell with mucinogen granules (G)

Ciliated Epithelial Cell (E)

Bronchial Lining Surface View

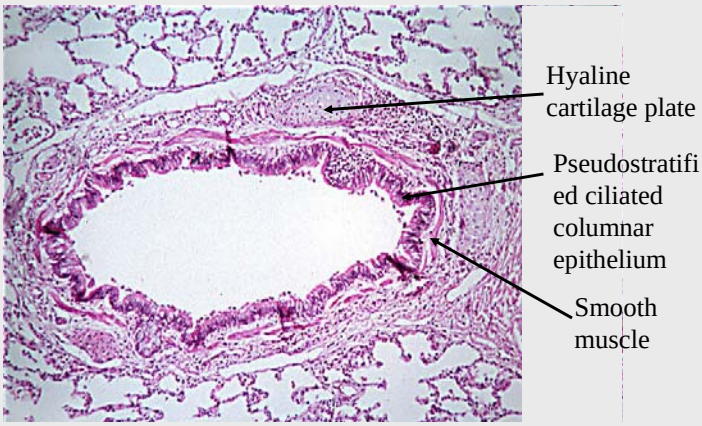
Scanning Electron Micrograph (SEM)



Cilia of the pseudostratified ciliated columnar epithelium. Note how the cilia lean uniformly in the same direction, appearing just as they were when fixed at a specific moment in their wave-like movement.

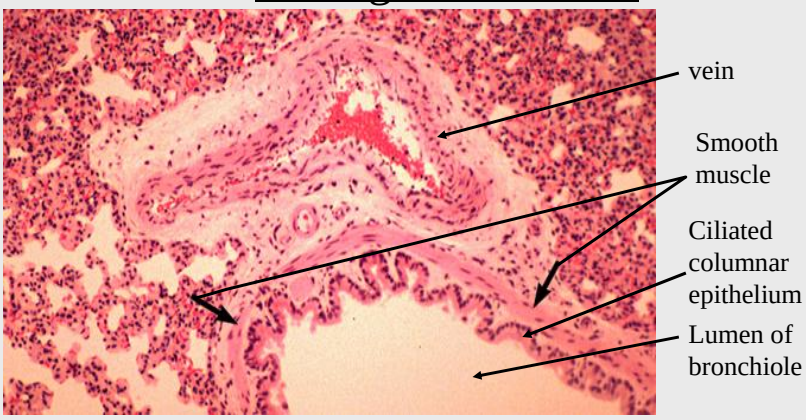
Goblet cells.

A Bronchus



See the [Virtual Microscope] for histology slides.

A Large Bronchiole



Notice the difference between the wall of bronchiole (cut, at bottom) and the vein above.

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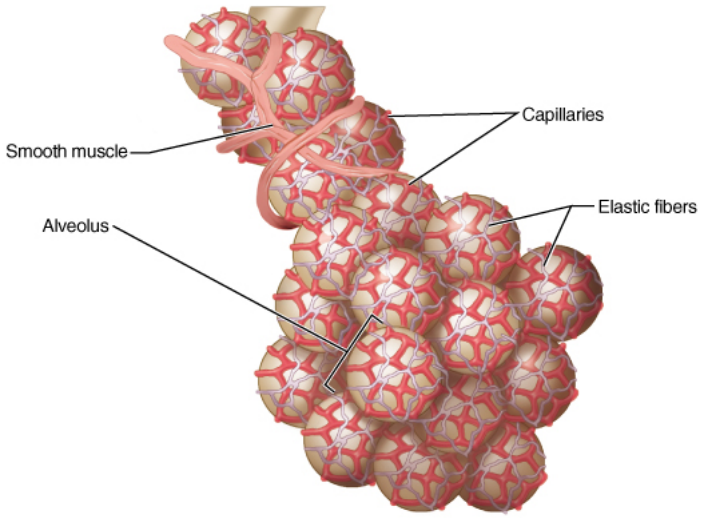
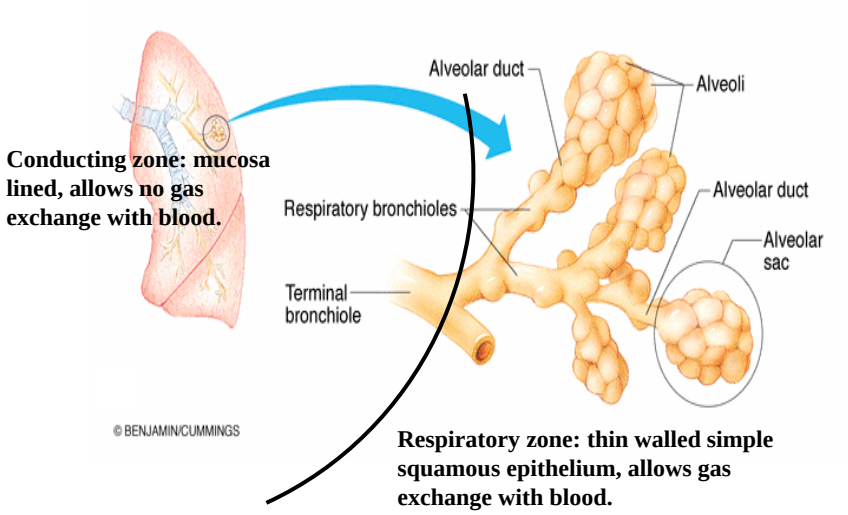
Alveoli



(b)

Alveoli are thin-walled chambers surrounded by capillaries for gas transport. Elastic fibers, part of the stroma of the lungs, provide support and elasticity (recoil) for the lungs.

Respiratory Zone

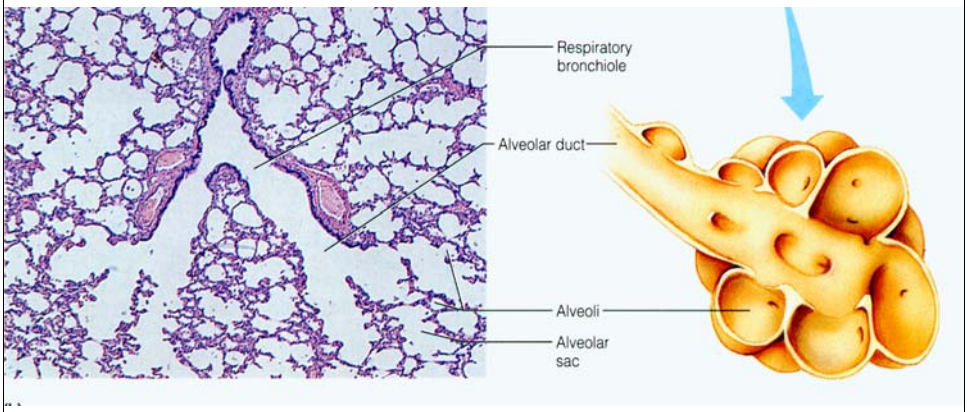


Conducting zone: mucosa lined, allows no gas exchange with blood.

Respiratory zone: thin walled simple squamous epithelium, allows gas exchange with blood.

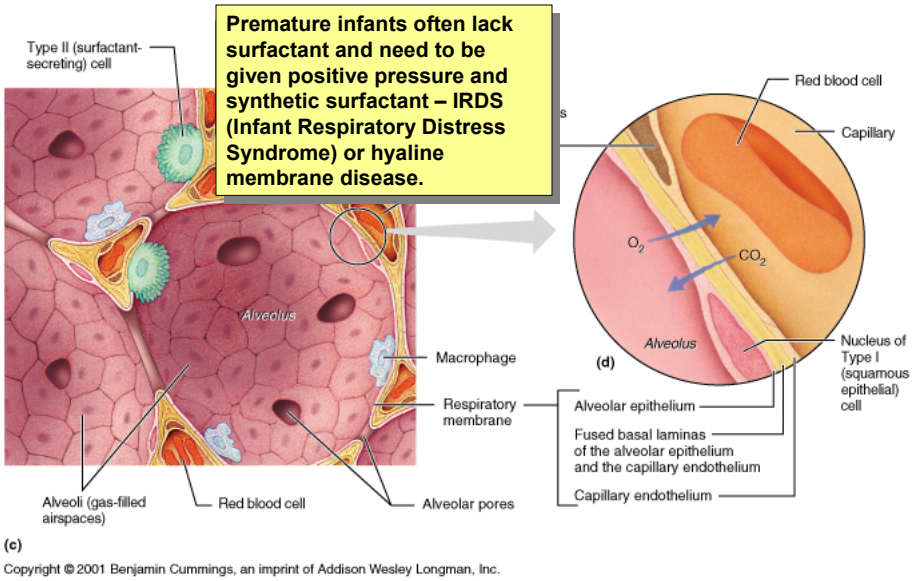
The Respiratory Zone - this area has thin simple squamous epithelial walls (the respiratory bronchioles start out as simple cuboidal and soon become simple squamous), no mucosa, and permits gas transport into and out of the blood. Respiratory bronchioles begin this zone, leading to alveolar ducts and then alveolar sacs. These sacs are a connected system of thin-walled chambers called alveoli. The alveoli increase the surface area for contact with blood vessels. The structure of the lungs is composed of these spongy-appearing alveolar sacs, together with bronchial passageways and blood vessels. See the following for highly magnified views of alveoli and associated cells and tissues: Look for the numerous capillaries in these closeups. They are indicated by the red blood cells inside them.

Branch into Respiratory Bronchiole



In this unlikely lung section you can see the passageways from a terminal bronchiole to the alveoli.

The Respiratory Membrane

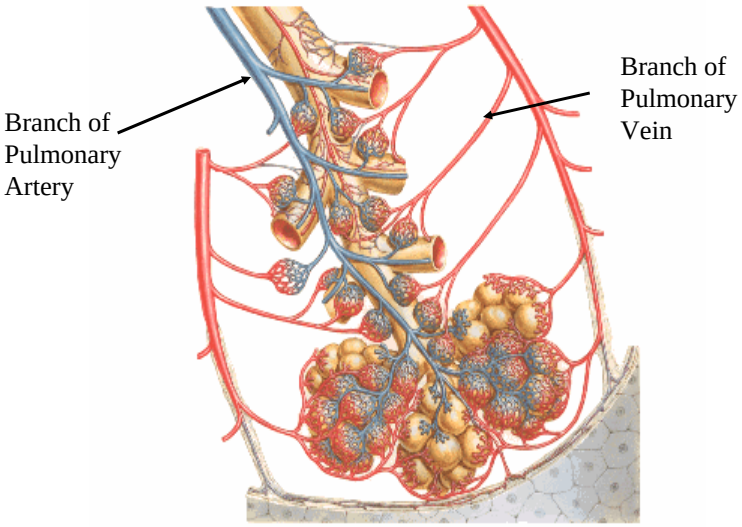


Marieb 5th Ed., Figure 22.9c

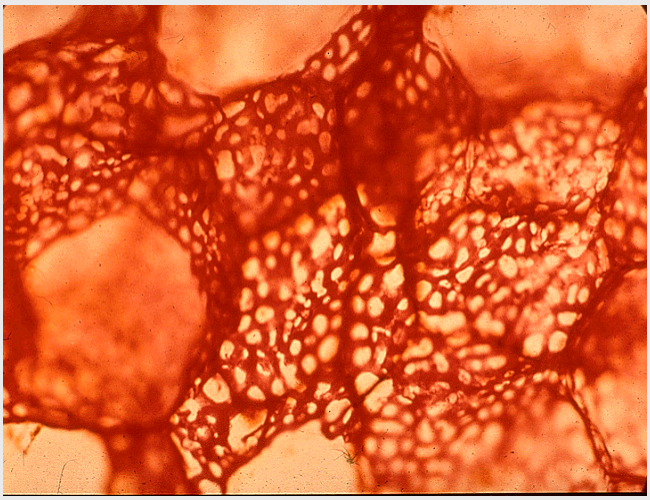
Alveolar capillaries are more numerous and densely arranged than in most any other organ. They form an area of contact with the alveolar walls of (depending on which source you read) from 50 to 400 sq. meters. Upon this area, at rest, approximately a liter and a half of blood is distributed. (This amount increases with exercise).

The structure of the respiratory membrane permits gas transport while preventing much water from entering the alveoli from the capillaries. Since interstitial fluid in the lungs would be disastrous to respiratory function, any fluid which does enter is removed by the lymph system. All the moisture in the alveolar sacs is the result of evaporation from the mucosal lining of the respiratory passages above. In addition to the **Type I** alveolar cells (the simple squamous cells of the alveolar wall) there are **Type II** alveolar cells present which secrete **surfactant**. Surfactant acts to break the surface tension (cohesiveness) of water which would cause collapse of the alveoli during expiration. This happens in premature infants whose respiratory systems have not matured, a condition called **Infant Respiratory Distress Syndrome** (**IRDS**) or **Hyaline Membrane Disease** . Macrophages are also present which phagocytize bacteria that make it into the alveoli.

Respiratory Zone Circulation

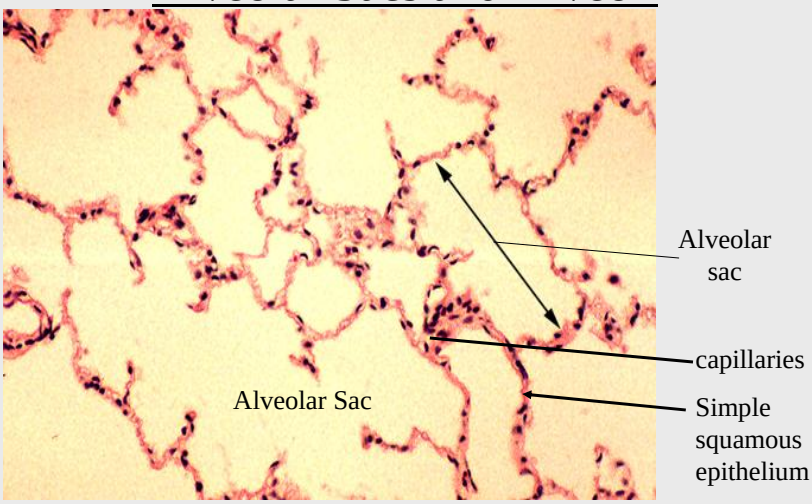


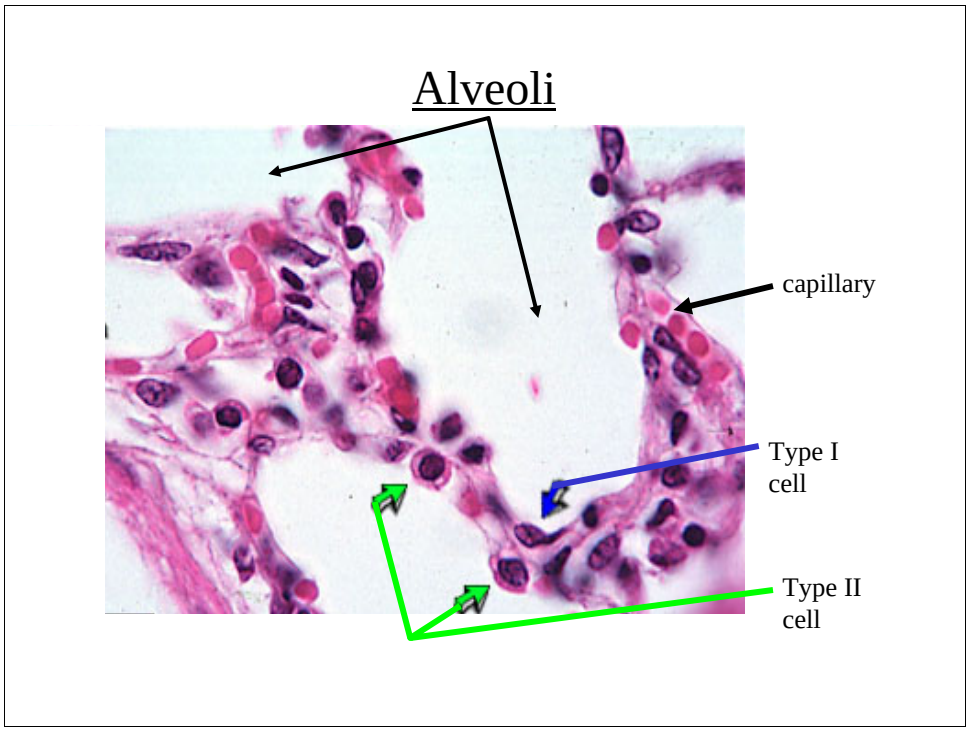
Alveolar Capillaries



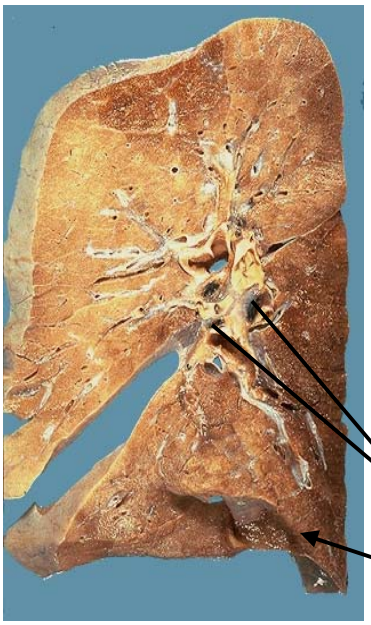
A dense network of capillaries surrounds each alveolus to produce the **respiratory membrane** consisting of the capillary wall (endothelium), the alveolar wall (simple squamous epithelium), and the basement membranes of each. These basement membranes are composed of a basal lamina with collagen fibers and a small amount of elastic tissue. The elastic tissue gives the lungs their elasticity or recoil.

Alveolar Sacs and Alveoli





A high magnification view of the alveoli.

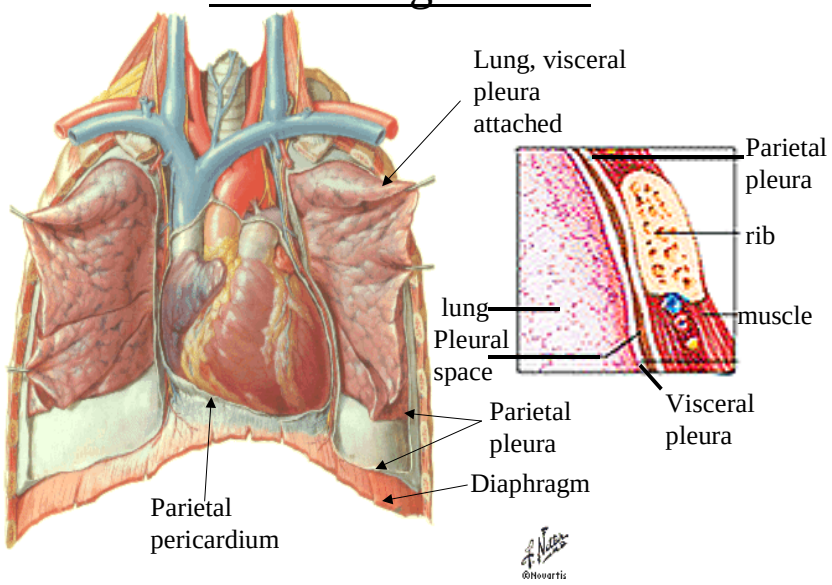


Normal Lung, Sec.

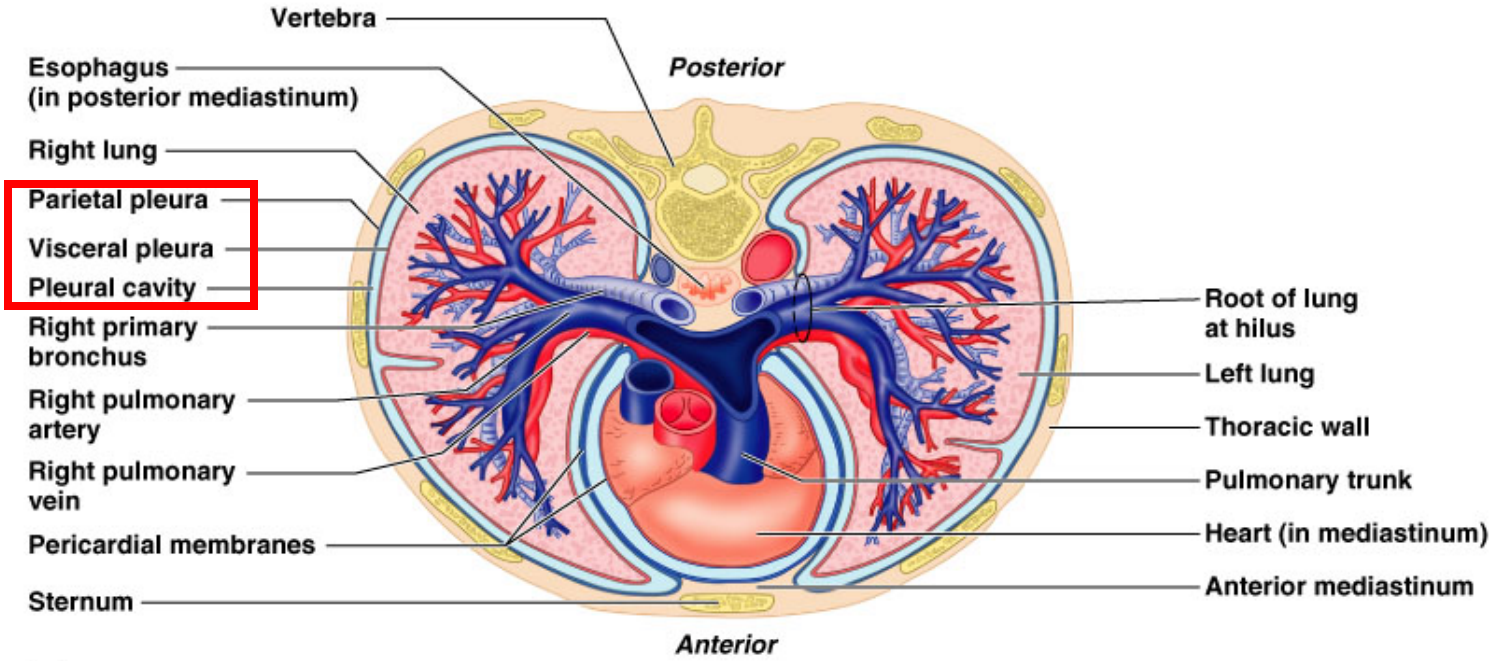
Notice the hilar lymph nodes which are small and have enough pigment (from dusts in the air breathed in, scavenged by pulmonary macrophages, transferred to lymphatics, and collected in lymph nodes) to make them appear greyish-black.

The majority of lung tissue, the “spongy” part, is composted of the alveoli and associated capillaries. The remainder consists of the larger blood vessels and respiratory passageways. In the lung specimen seen above some fluid has accumulated in the lower lobe.

Heart-Lungs in Situ

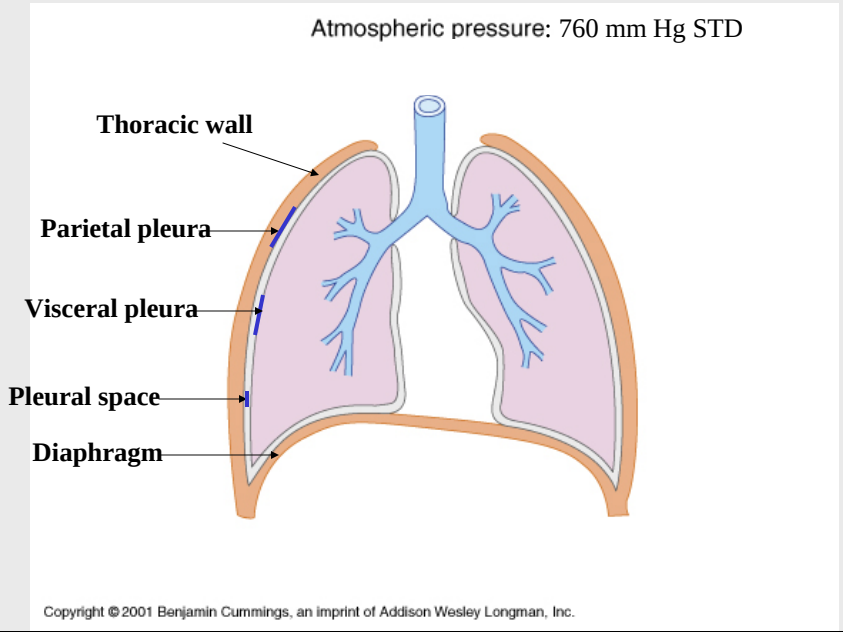


Pleural membranes are serous membranes: **Parietal pleura** – attached to the thoracic wall. **Visceral pleura** – attached to the lungs. **Pleural cavity** (space) – a fluid filled space which adheres the two pleural membranes. Because of the serous fluid these membranes adhere to one another, and when the parietal membrane is displaced due to movement of diaphragm or ribcage, so is the visceral layer, and so are the lungs.



(c)

Thorax-Lung Relationships



The lungs are surrounded by a double layered **pleural membrane or sack**. The inner layer is called the **visceral pleura** and is attached to the surface of each lung. The outer layer is the **parietal pleura** and is attached to the lining of the thorax and the mediastinum. It adheres to the inner wall of the ribcage and to the upper surface of the diaphragm. The space between these layers, sometimes called the pleural space or cavity, is really only a potential space, normally just the layer of serous fluid secreted by the membranes. The serous fluid binds the two layers together so that if the parietal layer is expanded by movement of the ribcage or diaphragm, so is the visceral pleura and so are the lungs.

When radial fibers contract the diaphragm flattens, pulling down and enlarging the thorax. Lowered pressure in the lungs following Boyle's Law pulls air down the pressure gradient into the lungs.

Contraction of the Diaphrgam in Inspiration

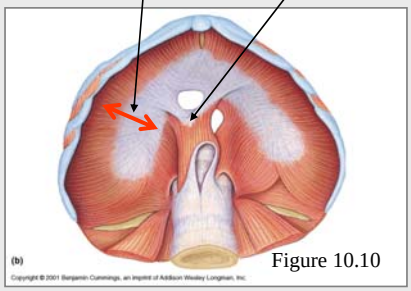
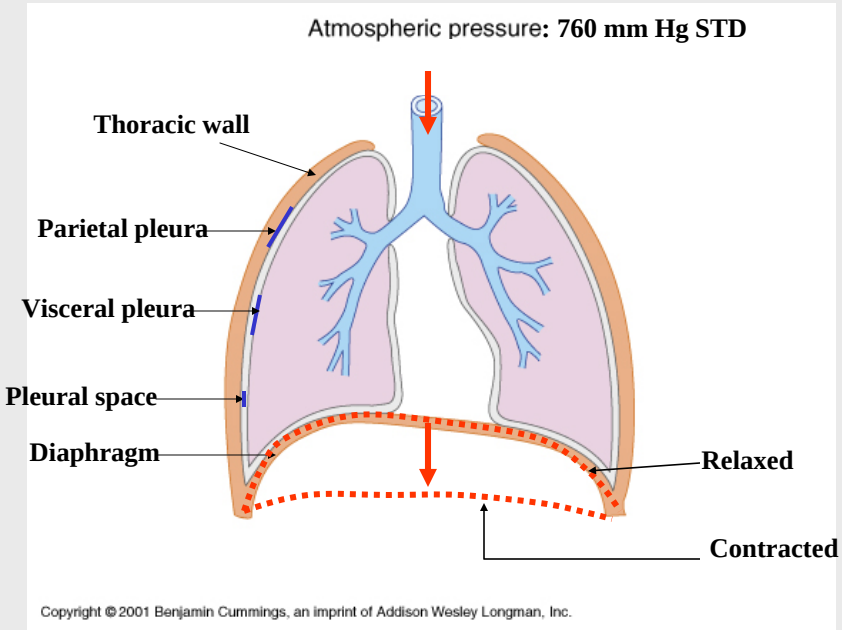


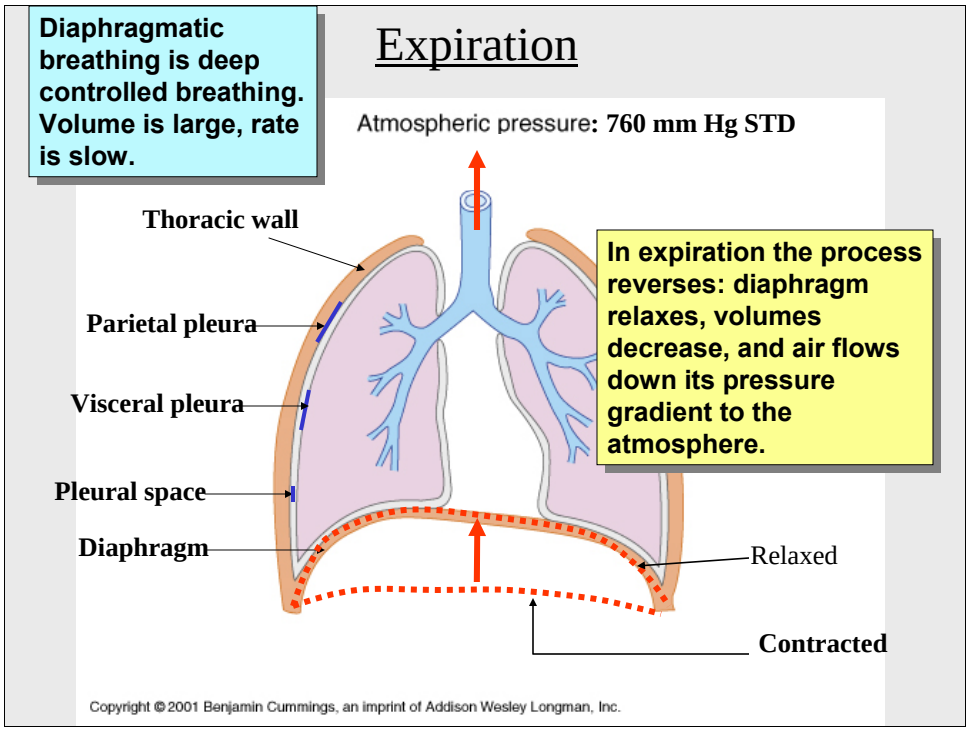
Figure 10.10

Ventilation is composed of two parts: inspiration and expiration. Each of these can be described as being either **quiet**, the process at rest, or **forced**, the process when active such as when exercising. **Boyle's Law** states that the volume and **pressure** of a gas are inversely proportional. If the volume of the gas increases, its pressure will decrease. If the volume decreases, its pressure will increase. The movement of air in ventilation occurs as a result of the pressure gradient produced when the volume of the lungs increases or decreases. The following table describes the events which produce this pressure gradient:

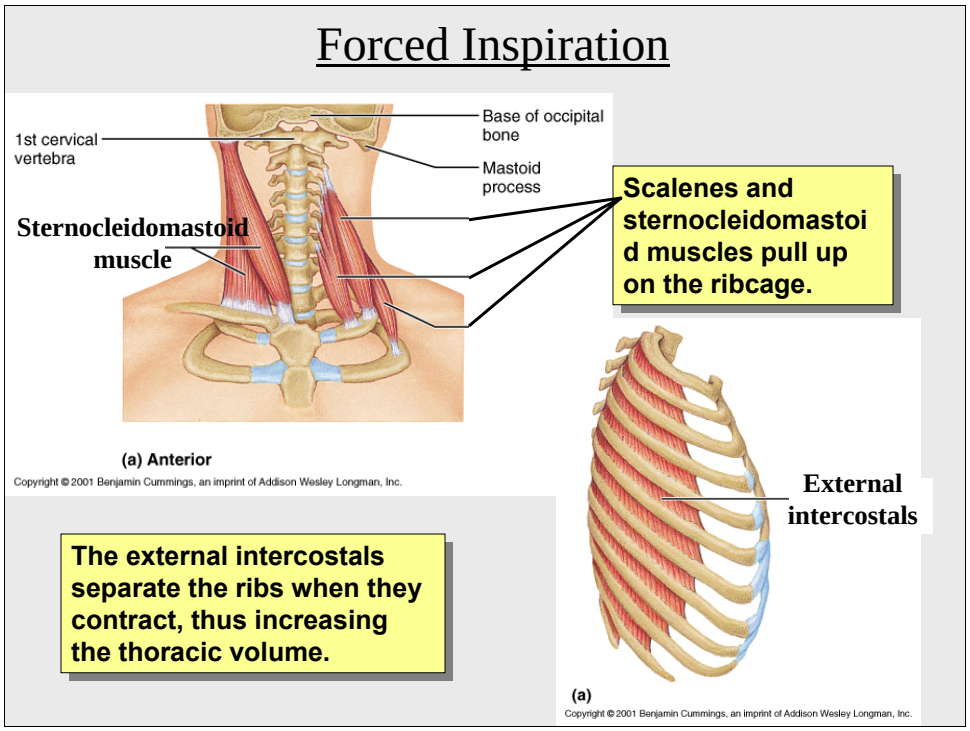
Inspiration



Quiet inspiration: The **diaphragm contracts**, this causes an **increase in volume** of the thorax and the lungs, which causes a **decrease in** pressure of the thorax and lungs, which causes air to enter the lungs, moving down its pressure gradient. Air moves into the lungs to fill the partial vacuum created by the increase in volume.



Quiet expiration: The diaphragm relaxes. The elasticity of the muscle tissue and of the lung stroma causes recoil which returns the lungs to their volume before inspiration. The reduced volume causes the pressure in the lungs to increase thus causing air to leave the lungs due to the pressure gradient.



Forced inspiration: Other muscles aid in the increase in thoracic and lung volumes.

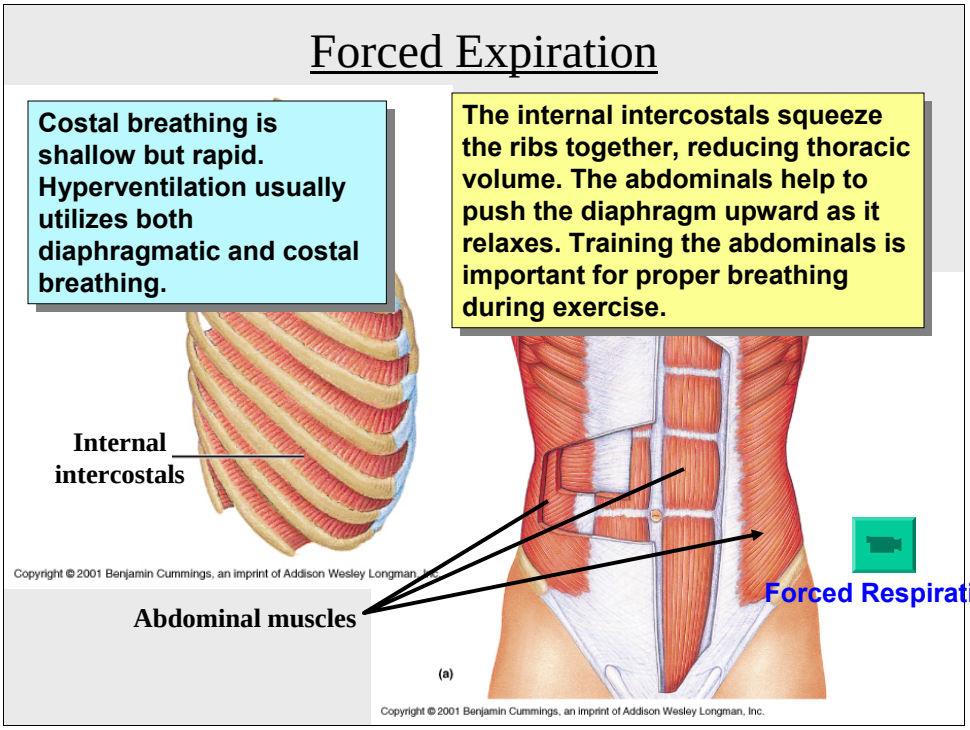
The **scalenes** - pull up on the first and second ribs.

The **sternocleidomastoid muscles** pull up on the clavicle and sternum.

The **pectoralis minor** pulls forward on the ribs.

The **external intercostals** are especially important because they spread the ribs apart, thus increasing thoracic volume. (See Figure 22.12) It's these muscles whose contraction produces the "costal breathing" during rapid respirations.

Forced Expiration



Forced Expiration: The following muscles aid in reducing the volume of the thorax and lungs:

The **internal intercostals** - these compress the ribs together.

The **abdominus rectus** and **abdominal obliques: internal obliques, external obliques-** these muscles push the diaphragm up by compressing the abdomen.

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Ventilation

	inspiration	expiration
Restful	Diaphragm contracts, volumes of thorax and lungs increase, pressures decrease: air flows inward along pressure gradient	Diaphragm relaxes, volumes of thorax and lungs decrease, pressures increase: air flows outward along pressure gradient
Forced (active)	Additional muscles help to increase the volume of the thorax and lungs: scalenes, pectoralis minor, sternocleidomastoid, external intercostals.	Additional muscles aid in decreasing the volume of the thorax or in pushing the diaphragm upward.

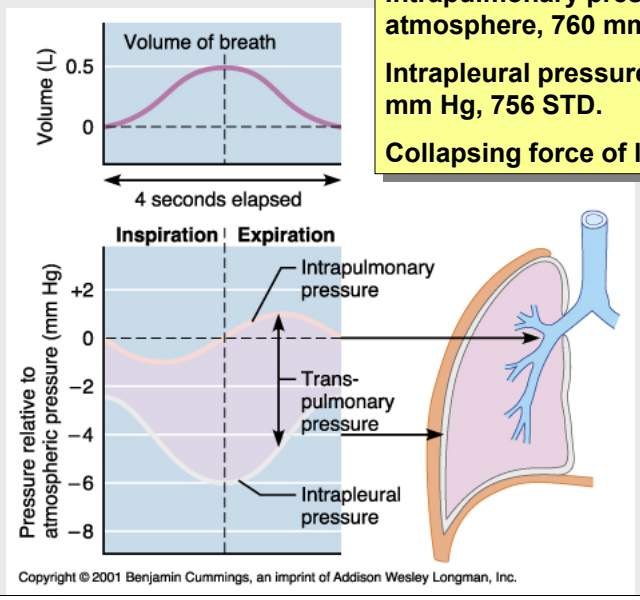
Quiet Respiration

Pressure Changes During Ventilation

Intrapulmonary pressure averages atmosphere, 760 mm Hg STD.

Intrapleural pressure averages –4 mm Hg, 756 STD.

Collapsing force of lungs = 4 mm Hg.



Intrapulmonary Pressures

Intrapleural Pressures

During inspiration the pressure inside the lungs (the **intrapulmonary pressure**) decreases to -1 to -3 mmHg compared to the atmosphere. The variation is related to the forcefulness and depth of inspiration. During expiration the intrapulmonary pressure increases to +1 to +3 mmHg compared to the atmosphere. The pressure oscillates around zero or atmospheric pressure. The **intrapleural pressure** is always negative compared to the atmosphere. This is necessary in order to exert a pulling action on the lungs. The pressure varies from about -4 mmHg at the end of expiration, to -8 mmHg and the end of inspiration.

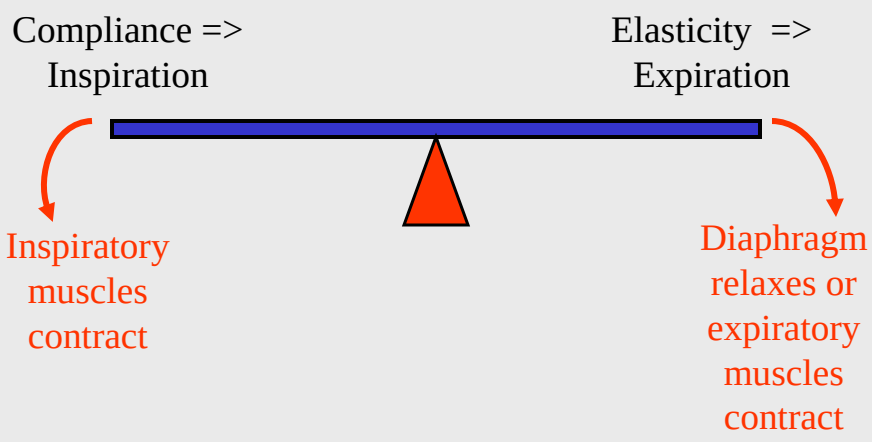
Balance Between Two Processes

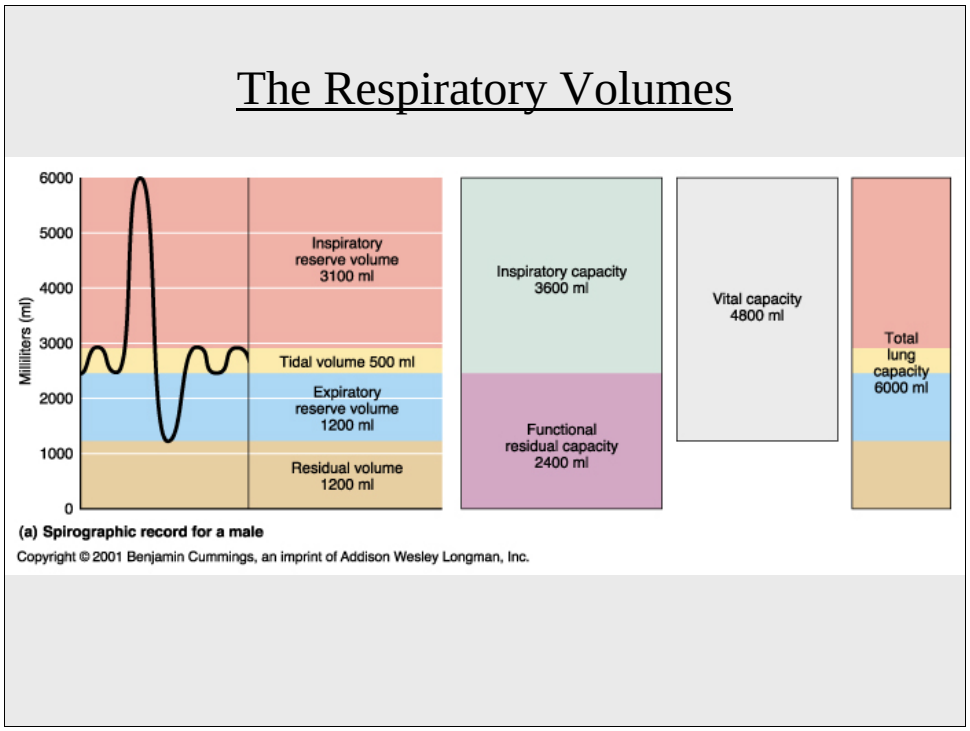
Compliance (distensibility) – the tendency of the lungs to expand. This is made possible by the negative pleural pressure and surfactant which prevents the alveoli from collapsing upon expiration. Compliance is the force responsible for inspiration.

Elasticity or **recoil** – this is the tendency of the lungs to return to their size before inspiration. Elasticity is due to the elastic stroma of the lungs and elasticity of respiratory muscles. Elasticity is necessary for expiration.

The tendency of the lungs to expand, called **compliance** or **distensibility**, is due to the pulling action exerted by the pleural membranes. Expansion is also facilitated by the action of surfactant in preventing the collapse of the alveoli. The opposite tendency is called **elasticity** or **recoil**, and is the process by which the lungs return to their original or resting volume. Recoil is due to the elastic stroma of the lungs and the series elastic elements of the respiratory muscles, particularly the diaphragm.

Balance of Compliance and Elasticity





Respiratory volumes **Tidal Volume TV**: Volume of a single breath, usually at rest.

Inspiratory Reserve Volume IRV: Volume which can be inspired beyond a restful inspiration.

Expiratory Reserve Volume ERV: Volume which can be expired beyond a restful expiration.

Residual Volume RV: Volume remaining in the lungs after a maximum expiration. This volume keeps the alveoli inflated.

Vital Capacity: The vital capacity (**VC**) is the maximum volume which can be ventilated in a single breath. VC= IRV+TV+ERV. VC varies with gender, age, and body build. Measuring VC gives a device for diagnosis of respiratory disorder, and a benchmark for judging the effectiveness of treatment.

The Minute Volume

Minute Volume = Respiratory Rate X Tidal Volume

Rates vary from 12 to 15 bpm (breaths per minute)

Tidal volume averages 500 cc.

For example:

Minute Volume = 12 bpm X 500 cc = 6000 cc/minute

Minute Volume = Rate (breaths per minute) X Tidal Volume (ml/breath)

Rate of respiration at rest varies from about 12 to 15 bpm. Tidal volume averages 500 ml (See Figure 22.16 or Lung Volumes Table below)

Assuming a rate of 12 breaths per minute and a tidal volume of 500, the restful minute volume is 6000 ml. Rates can, with strenuous exercise, increase to 30 to 40 bpm and volumes can increase to around half the vital capacity.

The Alveolar Ventilation Rate

Out of each breath approximately 150 cc is in the anatomical dead space (the conducting zone) where gas transport does not occur. In order to calculate the volume of air actually entering the alveoli the dead space volume must be subtracted.

AVR = rate X [tidal volume – 150 cc]

For example:

AVR = 12 X [500 - 150 cc] =

AVR = 12 X 350 cc = 4200 cc

Which would raise the AVR more, increasing the rate or volume of respirations?

Not all of this air ventilates the alveoli, even under maximal conditions. The conducting zone volume is about 150 ml and of each breath this amount does not extend into the respiratory zone. The **Alveolar Ventilation Rate, AVR,** is the volume per minute ventilating the alveoli and is calculated by multiplying the rate times the (tidal volume-less the conducting zone volume).

AVR = Rate X (Tidal Volume - 150 ml)

For a calculation using the same restful rate and volume as above this yields 4200 ml.

Since each breath sacrifices 150 ml to the conducting zone, more alveolar ventilation occurs when the volume is increased rather than the rate.

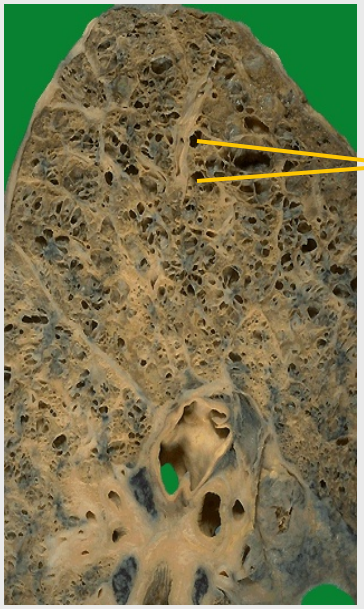
Restrictive Disorders

Any disorder which interferes with compliance or elasticity is a **restrictive disorder**. Examples are emphysema and fibrosis.

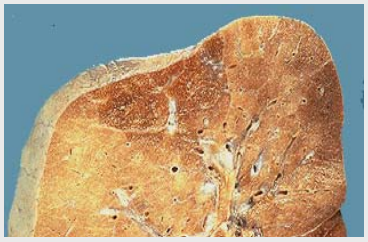
Restrictive disorders reduce the Vital Capacity by changing the volume of air which can be inspired or expired.

Conditions which interfere with compliance or elasticity are called **restrictive disorders**. Examples are **emphysema,** which increases compliance and decreases elasticity, and **fibrosis,** which reduces both.

Emphysema Lung Section



In this section of lung, dilated airspaces with emphysema are seen. Compare to normal lung below.

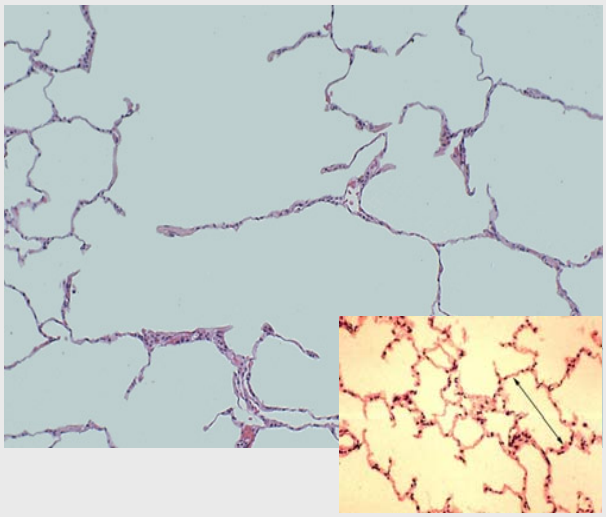


In emphysema the buildup of toxins from cigarette smoke and resulting mucus production leads to destruction of the alveolar and capillary walls and fibrosis of the tissue. This produces large thick-walled chambers replacing the normal small thin-walled alveoli. It results in a larger volume in the lungs but impaired gas transport and reduced ability to expire the trapped air. Many emphysema sufferers have the characteristic "barrel chest" as a result. Carbon dioxide tends to increase in alveolar air and in the blood, in some individuals interfering with normal respiratory stimuli and responses.

Pulmonary or **cystic fibrosis** (def) produces thickened mucus secretions which block the airways and scar tissue often develops in place of the normal elastic stroma. This is both restrictive and obstructive in its effects.

Restrictive disorders reduce the volume which can be ventilated as seen in a reduced **vital capacity**.

Emphysema, micrograph



Microscopically at high magnification, the loss of alveolar walls with emphysema is demonstrated. Remaining airspaces are dilated. Compare to normal lung.

Note the enlarged alveolar sacs compared with the normal tissue (lower right).

Alpha 1 anti-trypsin

Alpha-1 Antitrypsin is a glycoprotein produced in the liver which is the major antiprotease in the blood, serving mainly to inhibit leukocyte elastase. Hereditary antitrypsin deficiency is one of the most common genetic disorders among males of European descent. Alpha-1-antitrypsin deficiency results in leukocyte elastase building up and breaking down the lining of the lung, which results in a severe form of emphysema. Oxidizing agents in cigarette smoke are also known to inactivate antitrypsin, thus causing the high rates of emphysema among long-term smokers.

Leucocytes protect the body against invasion by releasing digestive enzymes. Normally the antitrypsin prevents the enzyme released by leucocytes from damaging the body's own tissue. But when the antitrypsin is deficient or inhibited, lung tissue is broken down.

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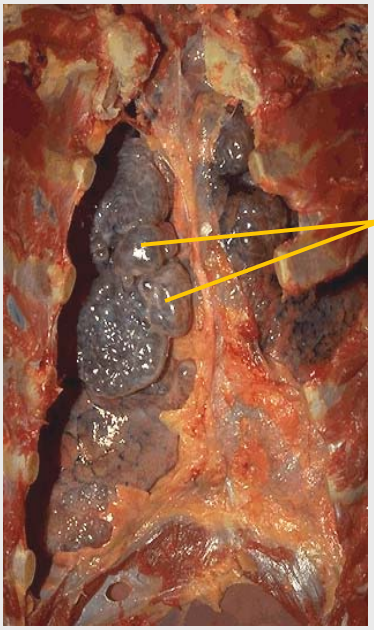


Emphysema, Gross

The chest cavity is opened at autopsy to reveal numerous large bullae (air bubbles) apparent on the surface of the lungs in a patient dying with emphysema. Bullae are large dilated airspaces that bulge out from beneath the pleura. Emphysema is characterized by a loss of lung parenchyma by destruction of alveoli so that there is permanent dilation of airspaces.

Obstructive Disorders

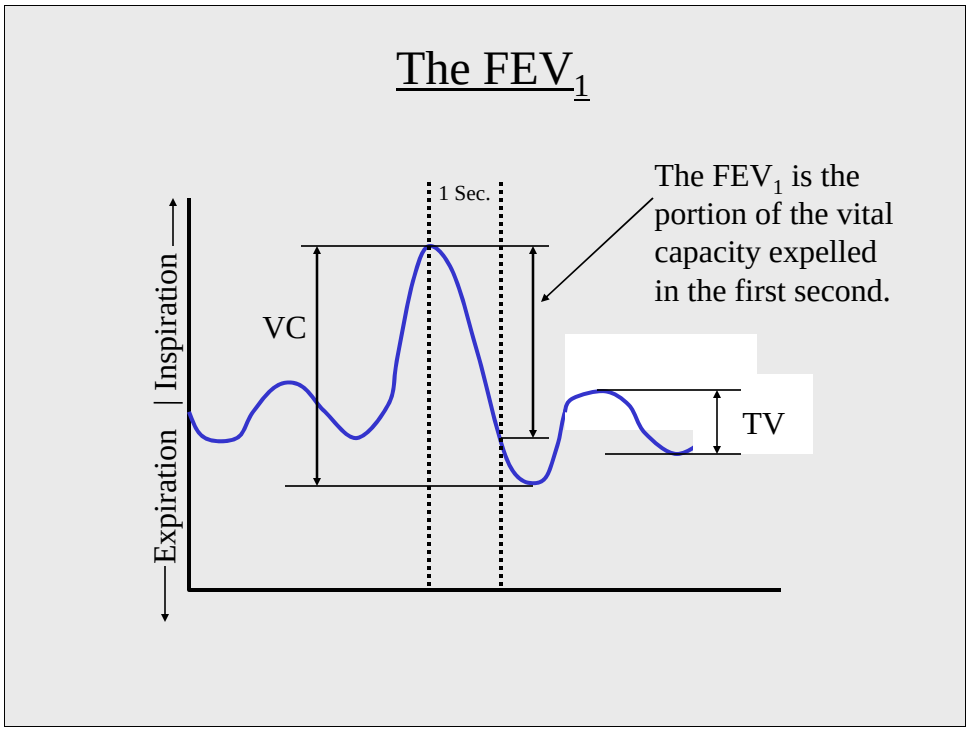
Disorders which obstruct the airflow are called **obstructive disorders**: **inflammation**, **mucus secretion**, **constriction**.

Obstructive disorders do not in themselves reduce the Vital Capacity. Instead they reduce the rate at which the VC is expelled. This is measured by the **FEV_1**. FEV stands for Forced Expiratory Volume, the 1 is for the first second.

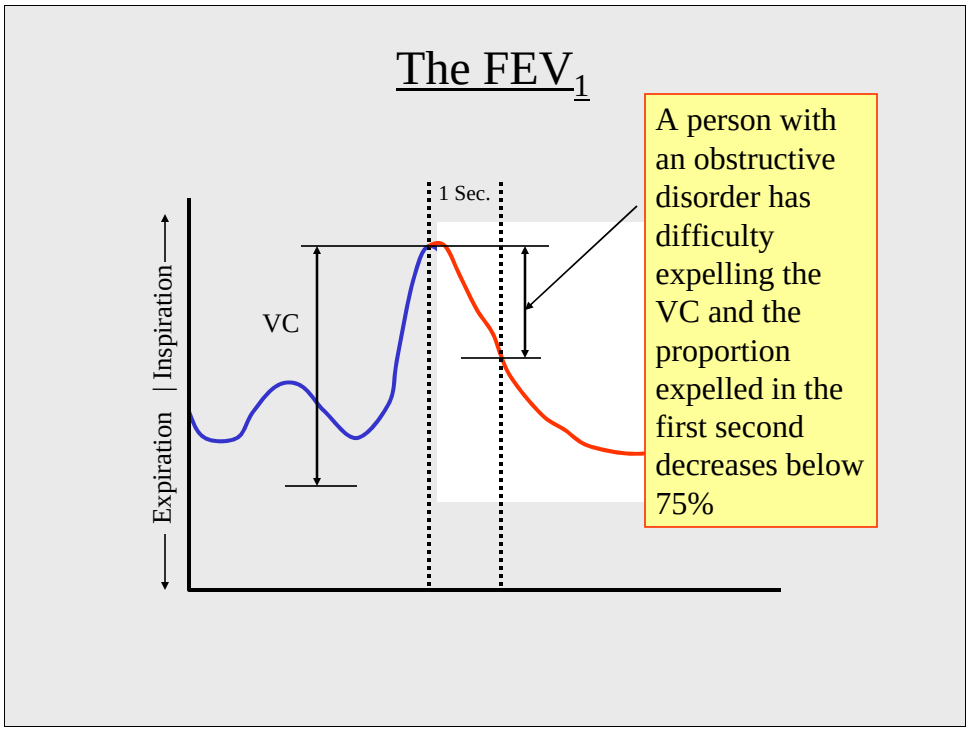
FEV_1 = % of the Vital Capacity expired in the first second.

Normal is 75 to 80%. Below 65% is considered severe.

In contrast, **obstructive disorders** such as **bronchitis** and **asthma** reduce the size of the bronchial passages thus interfering with the airflow. In bronchitis an inflammation results from respiratory infection or irritation from smoke or pollution.

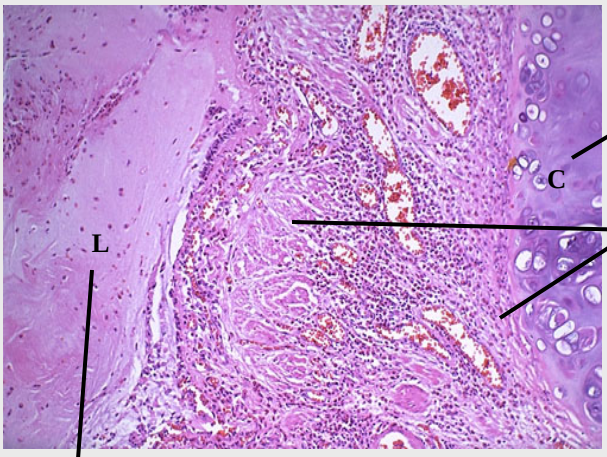


Purely obstructive disorders do not technically reduce the volume which can be ventilated but they *reduce the rate of ventilation* by increasing the resistance to airflow. They will show a normal vital capacity but a reduced **FEV_1, (Forced Expiratory Volume)** the percentage of the vital capacity which can be expelled in the first second. Normal FEV_1 is 75% or greater.



Note the shallow slope of the expiration. This indicates the reduced rate of an obstructive disorder such as **bronchitis** or **asthma**.

Bronchial Asthma, L.P.



Bronchial cartilage

Submucosa widened by smooth muscle hypertrophy, edema, and inflammation (mainly eosinophils).

Bronchial lumen (filled with mucus)

Asthma is like an allergic reaction in which inflammation occurs together with a constriction of the bronchial passages.

Disorders Which Are Both Restrictive and Obstructive

Disorders such as COPD (Chronic Obstructive Pulmonary Disease) and obstructive emphysema are both restrictive and obstructive and reduce both the Vital Capacity and the FEV_1.

COPD is a combination of emphysema, bronchitis, and asthma. Obstructive emphysema is dilation of the alveolar sacs together with the collapse of alveolar ducts and air trapping.

Many times disorders are both obstructive and restrictive. For example:

obstructive emphysema - in addition to the enlarging and thickening of alveolar walls many of the passageways collapse producing obstructive effects as well.

COPD Chronic Obstructive Pulmonary Disease - a combination of emphysema, bronchitis and asthma, has components of each.

Dalton's Law of Partial Pressures

The partial pressure of a gas in a mixture is equal to the fraction of the gas multiplied by the total pressure of the mixture.

For STP of 760 mm Hg: (Table 23.4)

O_2 = 21% X 760 = **pO_2 159** (160)

CO_2 = .04% X 760 = **pCO_2 .3**

N_2 = 79%

H_2O = .46% ?

Movement of the respiratory gases is due to diffusion. Diffusion results from a concentration gradient which is expressed for gases as the difference in **partial pressures**. **Dalton's Law of Partial Pressures** states that ***The partial pressure of a gas in a mixture is calculated by multiplying the fraction occupied by the gas times the total pressure of the mixture*.**

The mixture of gases under consideration is the air and Table 22.2 shows the partial pressures of its gases at STP (standard temperature and pressure at sea level).

Oxygen is present at nearly 21% of ambient air. Multiplying .21 times 760 mmHg (standard pressure at sea level) yields a **pO_2** of about 160. Carbon dioxide is .04% of air and its partial pressure, pCO_2, is .3.

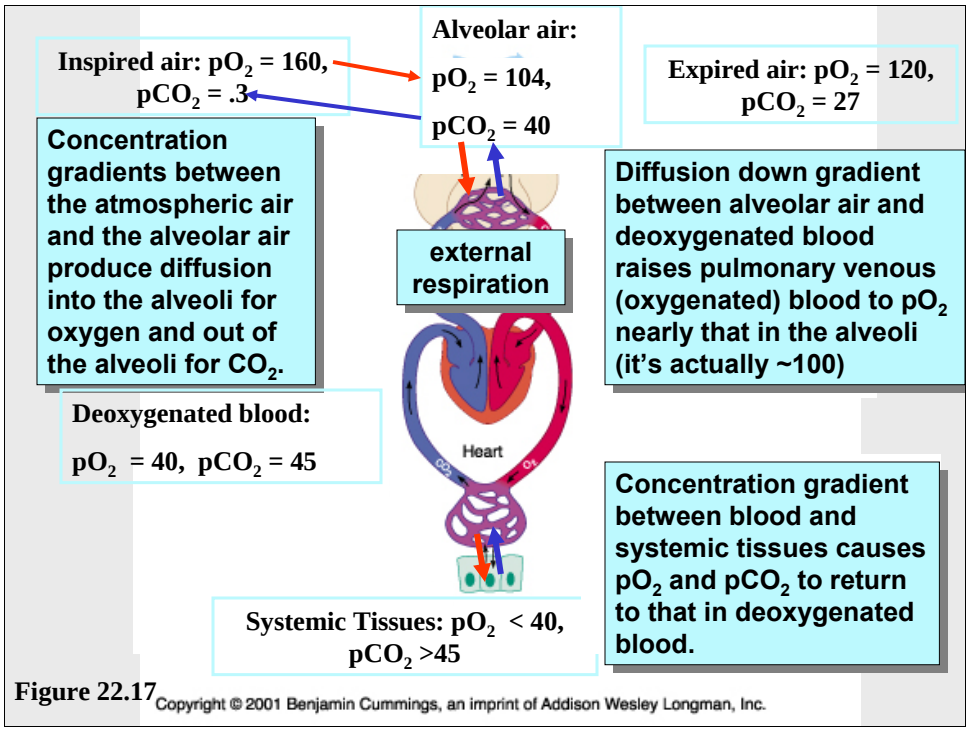
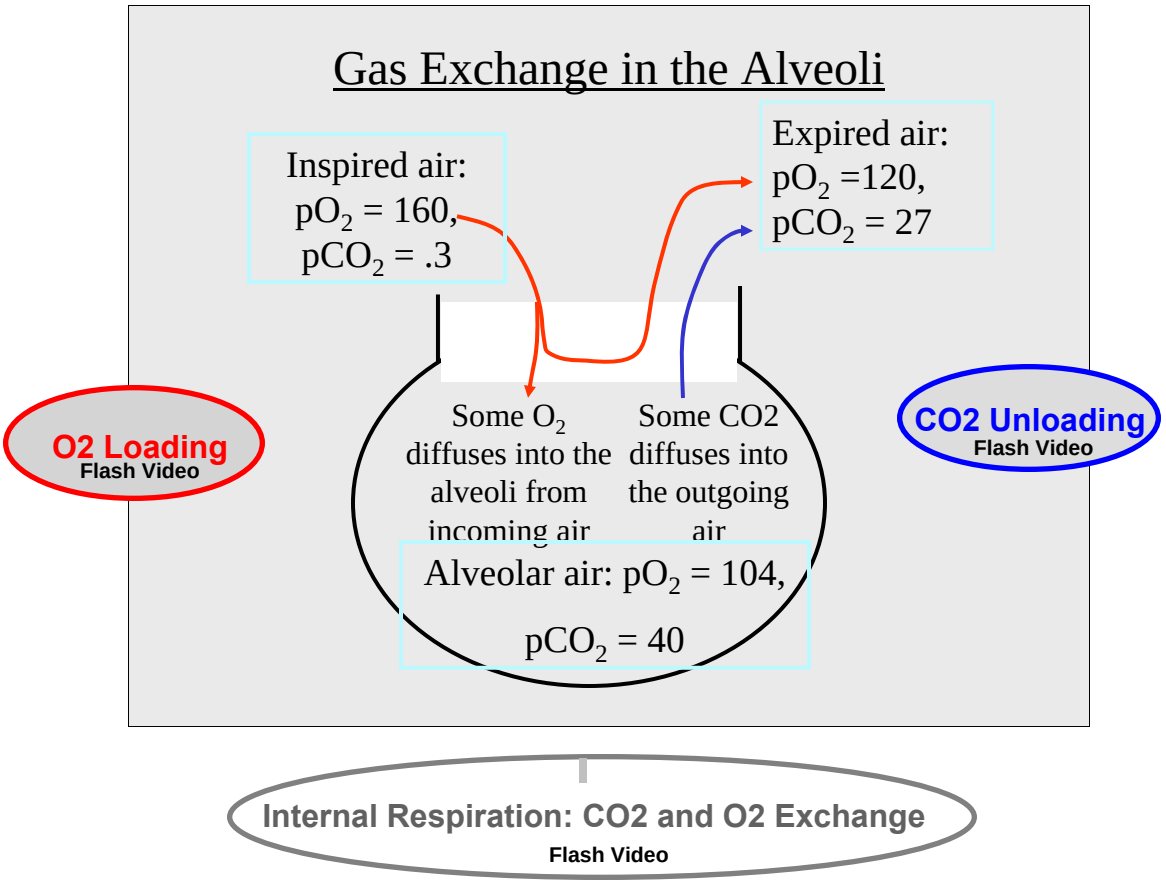
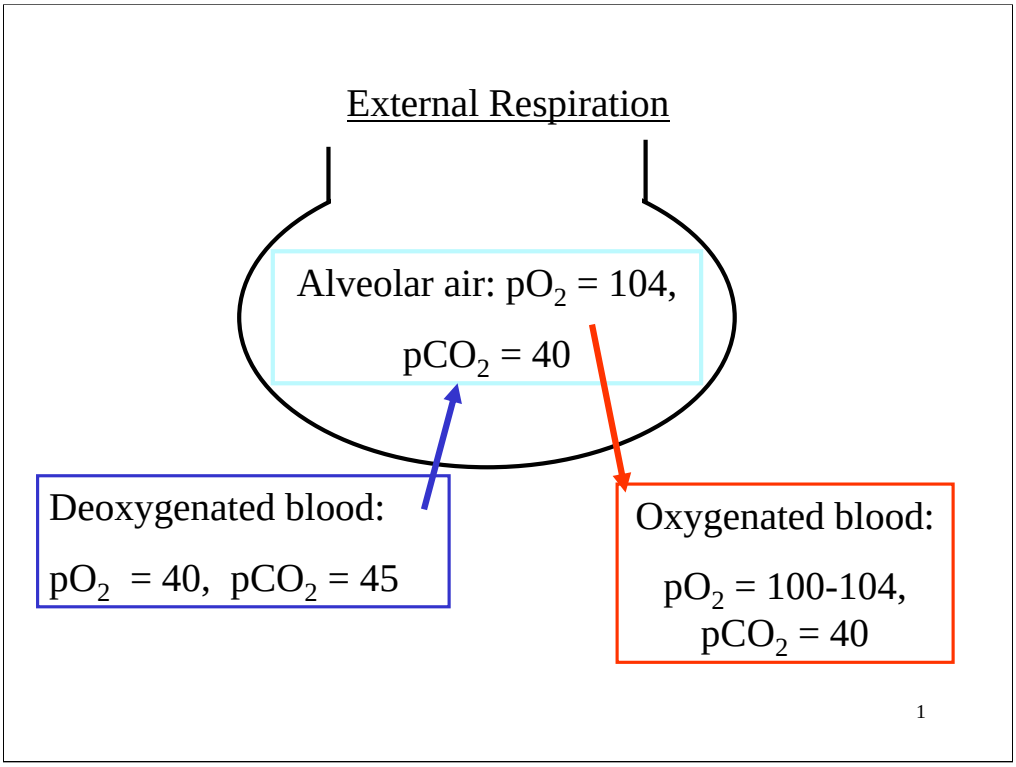


Figure 22.17

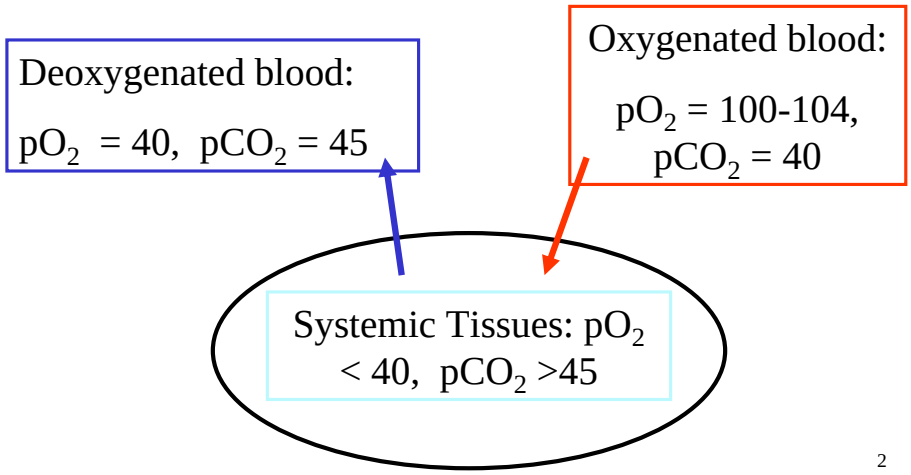
As Figure 22.17 shows there is a partial pressure gradient between inspired air and the alveolus with alveolar air having a pO_2 of 104 and a pCO_2 of 40. So oxygen diffuses into the alveoli from inspired air and carbon dioxide diffuses from the alveoli into air which will be expired. This causes the levels of oxygen and carbon dioxide to be intermediate in expired air when compared to inspired air and alveolar air. Some oxygen has been lost to the alveolus, lowering its level to 120, carbon dioxide has been gained from the alveolus raising its level to 27.

Likewise a concentration gradient causes oxygen to diffuse into the blood from the alveoli and carbon dioxide to leave the blood. This produces the levels seen in oxygenated blood in the body. When this blood reaches the systemic tissues the reverse process occurs restoring levels seen in deoxygenated blood.


Gas Exchange in the Alveoli
Inspired air:
pO_2 = 160,
pCO_2 = .3
Expired air:
pO_2 =120,
pCO_2 = 27
Some O_2 diffuses into the alveoli from incoming air
Some CO2 diffuses into the outgoing air
Alveolar air: pO_2 = 104,
pCO_2 = 40
O2 Loading
Flash Video
CO2 Unloading
Flash Video
Internal Respiration: CO2 and O2 Exchange
Flash Video


External Respiration
Alveolar air: pO_2 = 104,
pCO_2 = 40
Deoxygenated blood:
pO_2 = 40, pCO_2 = 45
Oxygenated blood:
pO_2 = 100-104,
pCO_2 = 40
1

Internal Respiration



2

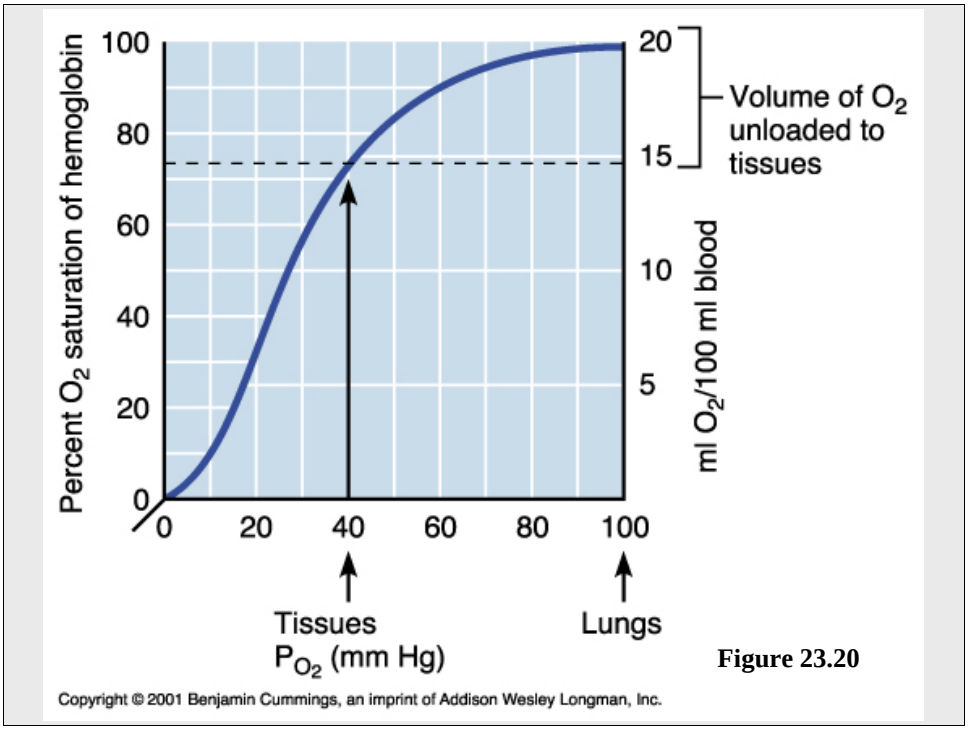
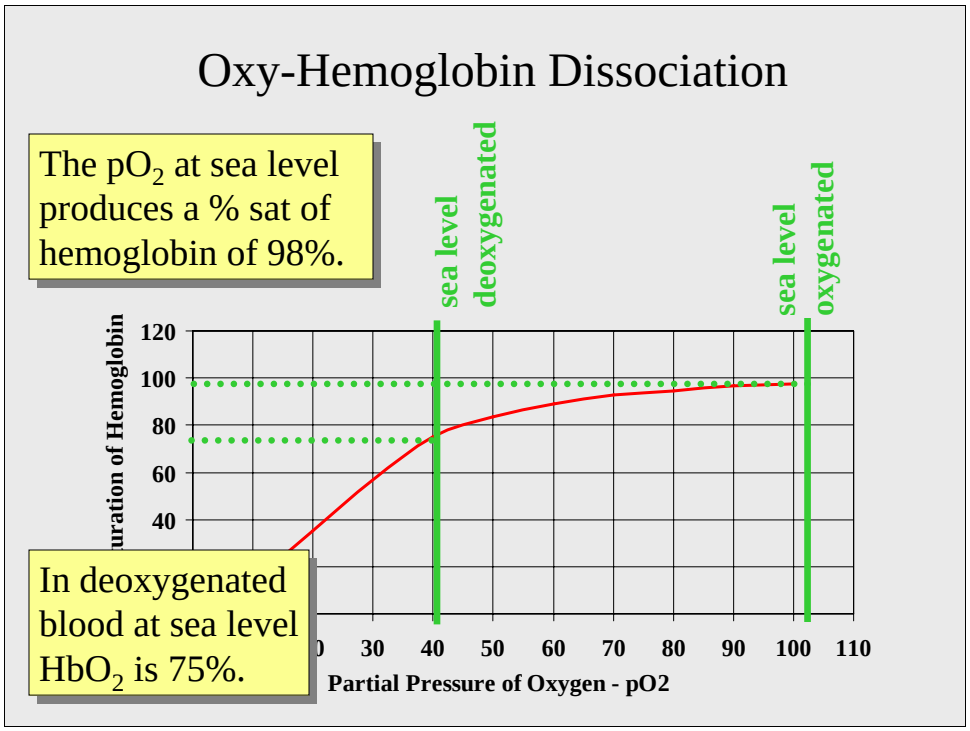


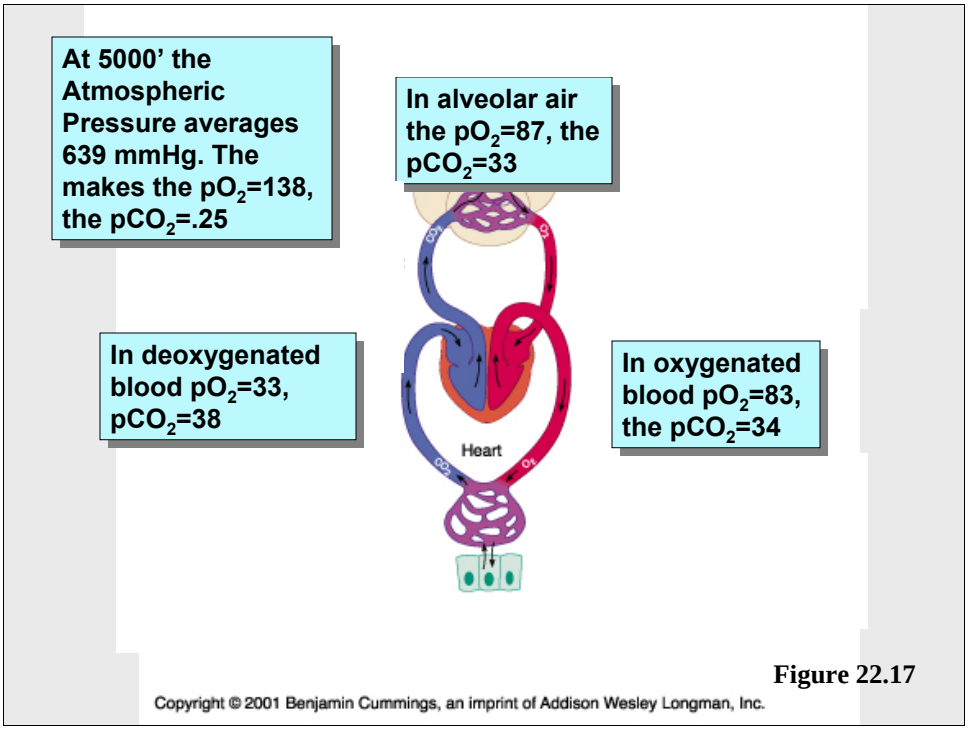
Figure 23.20

This curve illustrates the % saturation of hemoglobin at different levels of pO_2 . Because the curve is flat on top, changes in pO_2 in this region do not result in significant differences in % saturation of hemoglobin.

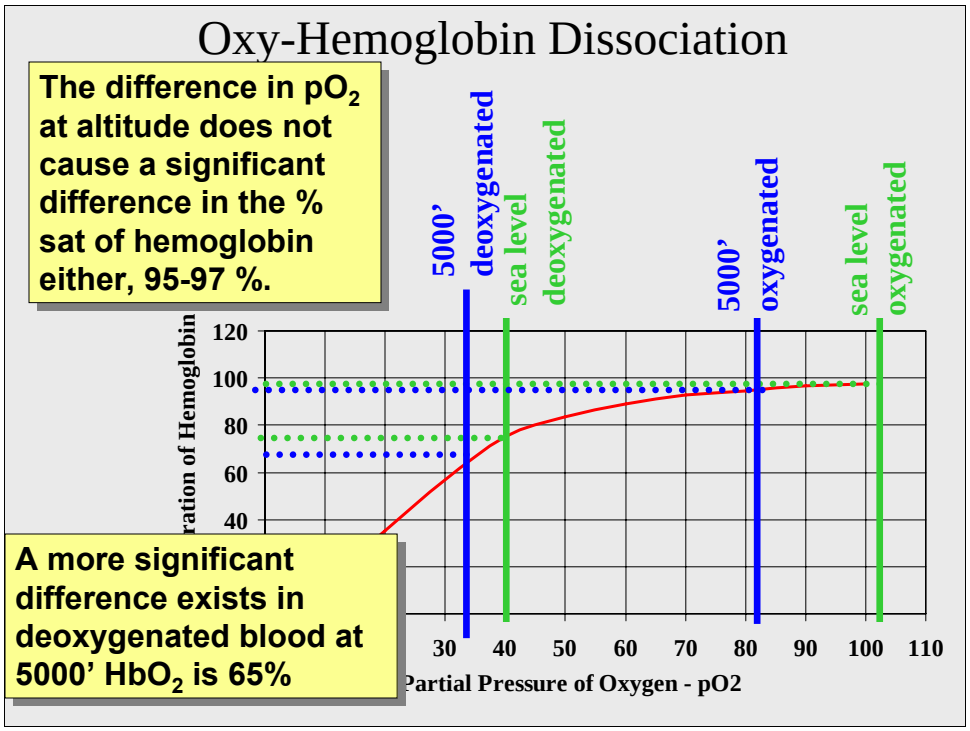


At sea level with a pO_2 in oxygenated blood of 100+ (as seen earlier) the % saturation of hemoglobin is 98%. At 5000' the pO_2 in oxygenated blood has decreased to about 83, but the saturation of hemoglobin has gone down only a few % to 95-97.

Deoxygenated blood is in the steeper portion of the curve. At sea level the pO_2 in deoxygenated blood is 40. This results in saturation of hemoglobin of about 75%. This means that about 1/4 of the oxygen carried by hemoglobin has been released to the tissues. The remaining oxygen on hemoglobin represents a significant reservoir which can be delivered to the tissues during hypoxic stress. At 5000' the pO_2 is about 33. This results in a saturation of hemoglobin of 65%. More oxygen has been unloaded (dissociated) from hemoglobin at altitude. The result of lowered pO_2, whether as a result of altitude or hypoxic stress is that more oxygen is unloaded to the tissues from the oxygen reservoir.



As altitude increases, the total pressure and partial pressures decrease. Here are the values for partial pressure at 5000' altitude.

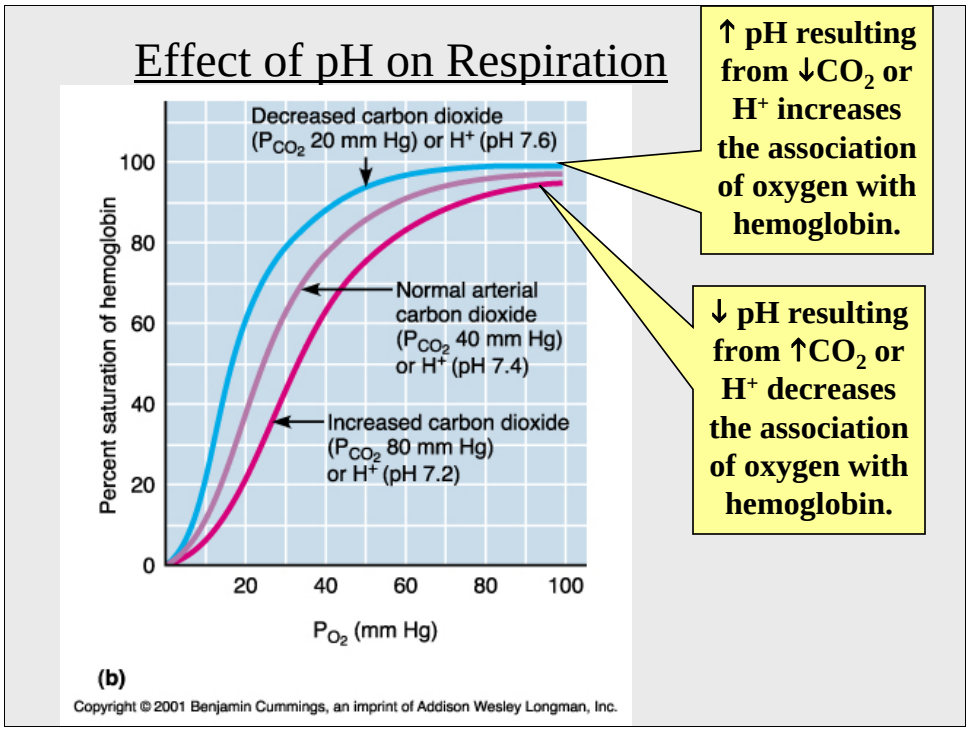


Here you see the effects on hemoglobin saturation of the changes in oxygen partial pressures at altitude.

2,3 Diphosphoglycerate

2,3 DPG (or BPG) is an intermediate in glycolysis which builds up in erythrocytes during periods of reduced oxygen supply. It attaches to hemoglobin, lowering its affinity for oxygen, thus driving oxygen off the hemoglobin molecule (it increases oxygen dissociation). It is especially important in deoxygenated blood in the systemic capillaries, and is the first response to altitude, increasing the oxygen available to the tissue.

The main factor responsible for increased dissociation of oxygen from hemoglobin at altitude is 2,3, DPG, also known as BPG.



The effect of pH on the Oxyhemoglobin Dissociation Curve. The effect of lowered pH steepen the curve resulting in greater dissociation or unloading of oxygen from hemoglobin. Higher pH flattens the curve resulting is reduced dissociation or unloading of oxygen from hemoglobin. This is due to a coupling of chemical reactions in which the reduction in pH which occurs as the blood picks up CO2 in the systemic tissues enhances the unloading of oxygen from hemoglobin. Likewise the increase in pH accompanying release of CO2 from the blood to alveolar air causes hemoglobin to load up with oxygen.

The Bohr Effect:

An increase in CO_2 in the blood results in a decrease in hemoglobin association with oxygen. (Oxygen **dissociation** increases).

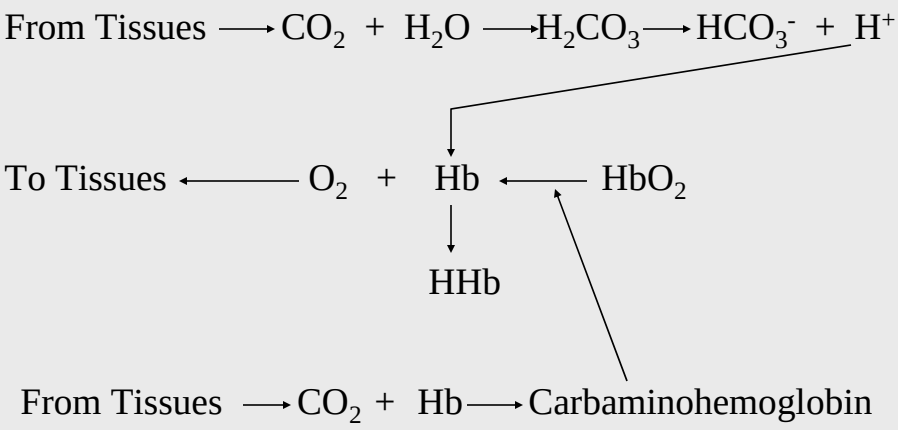
1) As a result of the H+ produced by the reaction of CO_2 and water.

2) Due to carbaminohemoglobin production.

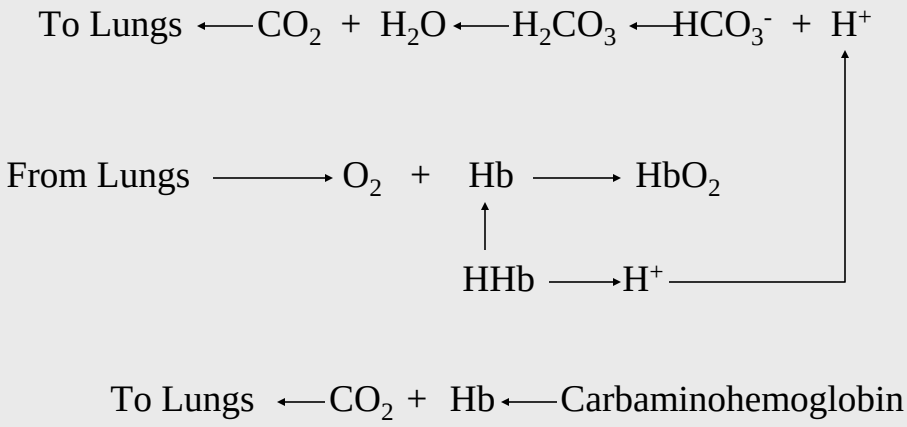
Summary of the Bohr Effect: As the blood picks up more carbon dioxide, hemoglobin can carry less oxygen.

The **Bohr Effect** describes the result of increasing CO_2 in causing more oxygen unloading from hemoglobin. [See Bohr Effect, Figure 22.22] It results from two circumstances: 1) the effect of lowering pH as described above, and 2) the effect of carbaminohemoglobin in stimulating oxygen unloading.

The Bohr Effect Occurs in the Systemic Capillaries



In the Pulmonary Capillaries the Process Reverses

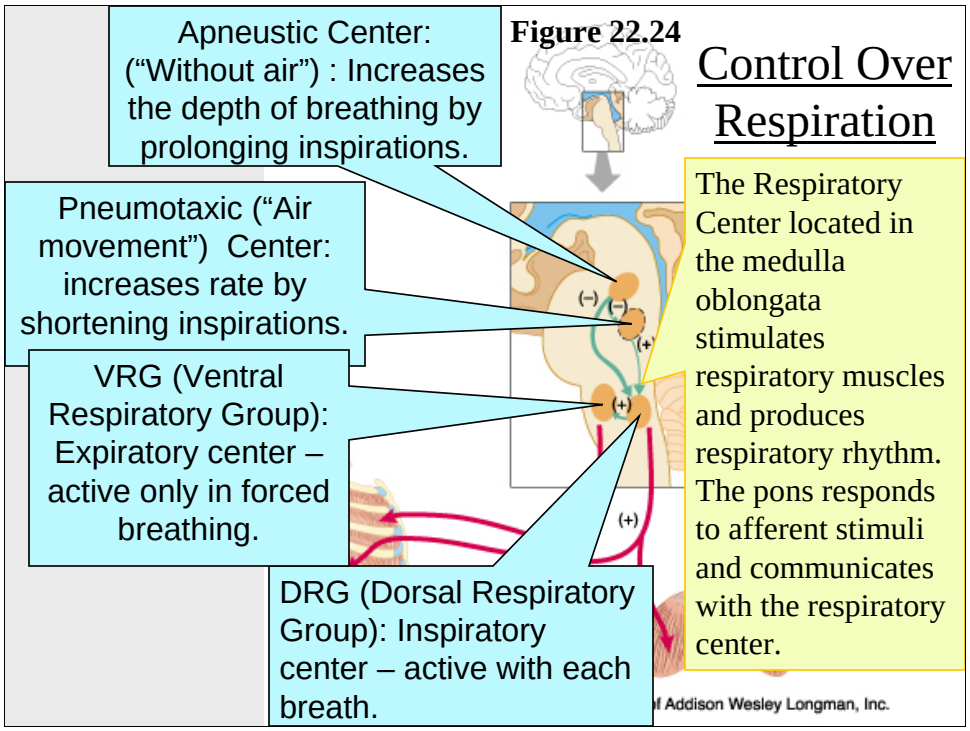


Ventilation-Perfusion Coupling

Autoregulation – ↑ O_2 → vasodilation of incoming arteries

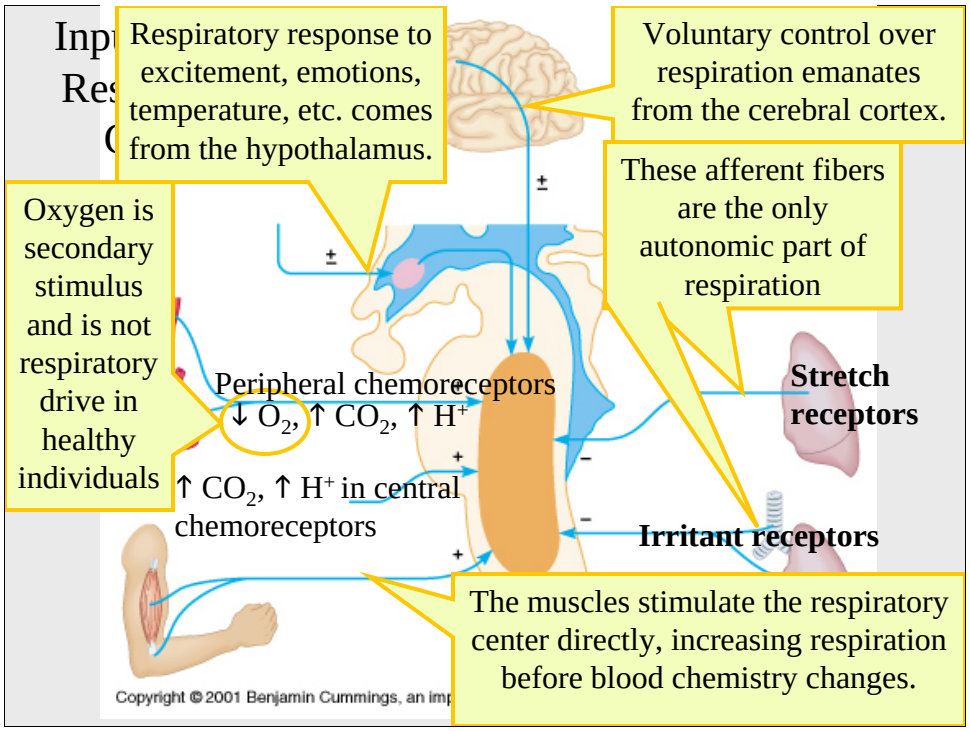
↓ O2 → vasoconstriction of incoming arteries

↑ CO_2 → dilation of bronchial passages



Control of respiration: The respiratory center is located in the **medulla** of the brainstem, with contributions from the **pons**. Within the medulla are the DRG (dorsal respiratory group) and the VRG (ventral respiratory group). The DRG sends stimuli to the muscles of inspiration: the diaphragm, external intercostals, and other. The VRG sends stimuli to the muscles of expiration: the internal intercostals and abdominal muscles. Since these muscles act only in forced expiration the VRG is only active then, while the DRG acts in both quiet and forced respiration. These muscles are all skeletal muscles and the motor control of respiration, whether quiet or forced, is a **voluntary** function. **The autonomic nervous system does not control the muscles of respiration.** The medulla controls the **rhythmicity** of respiration.

The **pons** sends stimuli to the medulla to regulate the **rate** and **depth**. The **pneumotaxic** center increases the rate by shortening inspirations. The **apneustic** center (its function is established but its location is not) increases the depth and reduces the rate by prolonging inspirations.



Inputs to these centers come from several locations:

1) peripheral chemoreceptors - located in the aortic sinus and carotid sinus, respond to increased carbon dioxide and decreased pH (which are related as discussed earlier).

Although the peripheral chemoreceptors include receptors to oxygen, oxygen levels are **secondary stimuli** at best, normally not important in triggering increased respiration. For healthy people the levels of oxygen would never decrease to the point of stimulating respiration. However, this can apparently happen in certain disorders such as emphysema, in a condition known as **hypoxic drive**. This happens because CO_2 levels become permanently elevated and the CO_2 receptors cease responding. This is somewhat controversial, but some believe that administering oxygen can actually suppress respiration in someone with hypoxic drive.

2) central chemoreceptors - located in the medulla. Their primary stimuli are decreased pH and increased carbon dioxide. pH is especially important since carbon dioxide does not readily diffuse into brain tissue but affects it through its impact on hydrogen ions.

3) muscle contraction - when you exercise there is a direct stimulus to the respiratory center from active muscles and joint receptors. This causes increased respiration before blood chemistry actually changes enough to demand it.

4) higher brain centers - from the voluntary motor center for voluntary control over respiration, and from the hypothalamus for control in response to emotional stimuli and body temperature.

Summary of Respiratory Stimuli

Stimulus	Response
↑pCO_2, ↓ pH, ↑ H^+, 2ndary ↓pO2 Hypoventilation, acidosis	Hyperventilation
↓ pCO_2, ↑ pH, ↓ H^+ Hyperventilation, alkalosis	Hypoventilation
Voluntary hypoventilation	↑pCO_2, increased need to breathe
Voluntary hyperventilation	↓pCO_2, decreased need to breathe