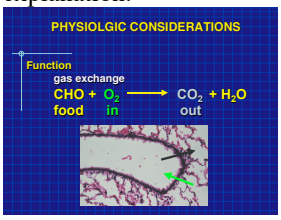
Draft

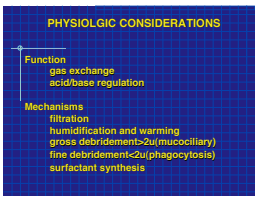
Respiratory Physiology Draft

Fred Quarnstrom, DDS

Respiratory Physiology for most dentists is a bad dream that occurred early in dental school. Our knowledge of this topic most simply stated is: people breath in and out. When they breathe in, they take in oxygen. When they breathe out the get rid of carbon dioxide. If we are going to use nitrous oxide we must go a little beyond this simplistic explanation.

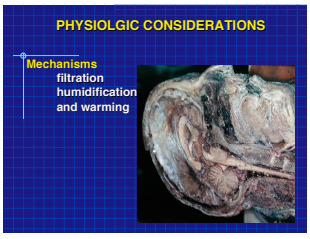


The challenge of this chapter was how far it should go. The physics, chemistry and physiology of respiration are exquisitely complex and very well understood. However, it is not necessary to become a physiologist to administer nitrous oxide. This chapter will attempt to achieve a balance and present enough to understand the system as it pertains to the use of nitrous without getting too complex.



Nasal Passages

When we breathe in, there are two passages we can use to inhale air into our lungs-- the nose, nasal passages and nasal pharynx or the mouth and oral pharynx. Normally, in general anesthesia, we would use both with the gas being administered from a full-face mask that covers the nose and mouth. Since dentists need access to the mouth, only the nasal passages, through a nose mask, or nasal hood, can be used.



(A line drawing would be better)

In the normal, healthy adult the nose is the preferred passageway for breathing. The nares are smaller than the mouth and there fore will prevent the passage of small particles in to the respiratory system. The hair at the entrance to the nose acts as a gross filter of air. The mucosal lining of the nares has tiny cilia supported by pseudo columnar epithelium interspersed with mucous cells. The mucous entraps any fine particles and the rhythmic beating of the cilia passes the entrapped particles to the back of the nasal cavity and into the pharynx where it is swallowed. [1] <#_edn1>

In addition, the nasal passages increase their surface area through the presence of nasal turbinates or conchae. These thin sheets of folded bone covered by nasal mucosa allow greater volumes of air to be exposed to cilia and mucous and, therefore, increase the efficiency of the filtering system.

This structure also increases the efficiency of the second function of the nasal passages that of heat exchange. The inhaled air is converted to body temperature by the time it approaches the oral pharynx. This can be important in very hot or cold climates, where temperature extremes would be at the least uncomfortable and perhaps damaging to the upper respiratory tract.

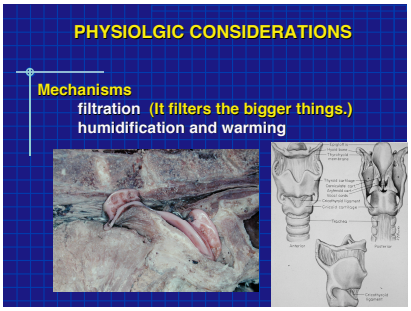
The nasal passages with their extensive surface area, also completely saturate the air with moisture so that the upper respiratory tract is not dried out even in the driest climate. In a very dry, hot location such as a desert, a certain amount of this moisture will be recaptured and heat will be exchanged back into the exhaled air. The camel has adapted this to survive its hostile atmosphere. It exhales air that is very close to the ambient air temperature with almost all of the moisture having been recaptured. This adaptation makes the camel a very efficient desert dweller. It does not absorbed the deserts heat and it preserves its bodily fluids that other animals would loose with their exhaled gases. This does however; require a rather spectacular large nose to achieve this exchange. The efficiency of the camel's nose achieves levels not often achieved by heating engineers.

While the nasal passages provide the important beneficial functions of air filtration, heat exchange and humidity control, they also can be an impediment to the passage of air. As many boxers know trauma to the nose can lead to passages that are closed and necessitate mouth breathing. In addition allergic reactions and infection can cause the nasal tissues to engorge with fluids and close off these passages. There may also be anatomical blockage such as a deviated nasal septum. Without patent nasal passages it is impossible to administer gas via a nosepiece and the above examples are all contra-indications to its use.

In addition, nasal infections may cause the discomfort of postnasal drip, while the infecting agents can be released from the tissues to contaminate the nosepiece or infect the dental staff. For this reason a person with an upper respiratory tract infection would best be not treated with nitrous oxide sedation.

Pharynx

Once the air leaves the nasal passages it traverses the nasal pharynx and pharynx. The pharynx connects with openings from the oral cavity, nasal cavity, larynx and esophagus. Because it serves as a passageway for food water and air some complex reflexes exist to close the epiglottis when we are swallowing and thus close off the larynx.



The Larynx is comprised of 9 cartilaginous plates, the vocal cords and a series of small muscles that control the cartilage and vocal cords. While its functions of speech and air transportation are easily understood, the actual anatomy and nervous innervations are a challenge to all dental students. Basically, however, air, food and water pass through the pharynx, and the epiglottis, a cartilage and mucosal flap, occludes the airway when we swallow. In turn it opens and allows the passage of air when we breathe and talk.[2] <#_edn2>

Basically, speech is possible because of two fibrous strands of tissue that vibrate when we exhale air. In a very

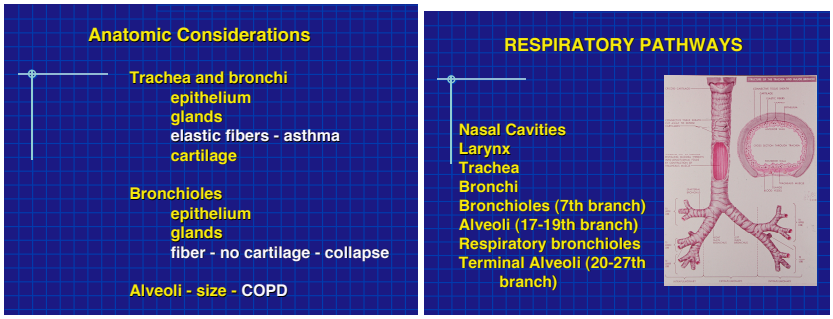
complex process we can make sounds using these vibrating structures and form these sounds into words for speech. If a foreign body manages to evade the epiglottis and lodges on the vocal cords a laryngeal spasm can be precipitated causing the vocal cords to come together and thus close off the airway, making it impossible to breathe. This is one of the dangers of deep sedation or anesthesia. In the worst-case scenario, food can actually block the air passage or be aspirated into the lung occluding smaller airways.[3] <#_edn3>

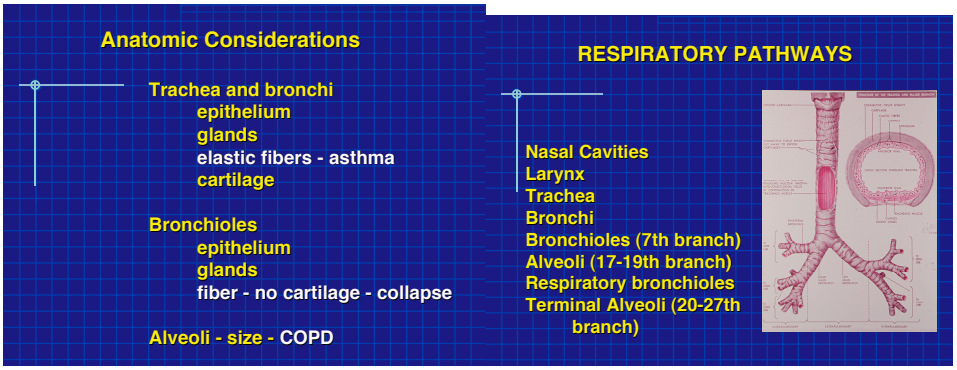
Under normal circumstances a tight muscle band closes the upper end of the stomach attached to the esophagus. Any food in the esophagus triggers peristalsis, a rhythmic contraction of the muscles that moves food down the esophagus into the stomach. If food is in the upper end of the esophagus as is the case when we swallow, the epiglottis closes to keep this food out of the breathing passages. This area is of vital interest to those who administer sedation and general anesthesia. As sedation increasingly approaches general anesthesia these reflexes are lost.

First to go is the stomach-closing reflex allowing the stomach contents to move out of the stomach and up through the esophagus. The swallowing reflex is next to be lost. This allows the stomach contents to migrate higher. Finally the epiglottal reflex is lost. If these stomach contents ascend to this level they can then be aspirated down the breathing passages into the lungs.

Because this material has been in the stomach and the stomach secretes acid to digest food, the, acidity of this material is very high and will precipitate aspiration pneumonia. Which may lead to death.

Nitrous oxide sedation, administered correctly, with a minimum of 30 to 50 percent oxygen, should never achieve this level of deep sedation. Problems can arise however where a patent takes a prescription or illegal self prescribed sedative medication that might potentate the anesthetic potency of nitrous oxide.

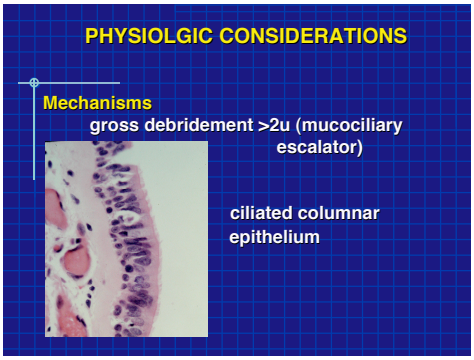




Trachea

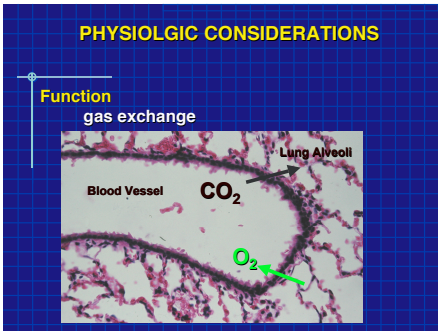
Once air enters the trachea it travels down some 10 inches where it branches into the right and left main stem bronchi. The trachea is kept open by cartilaginous horseshoe shaped structures and the bronchi by cartilaginous rings. All are still lined by a ciliated columnar epithelium and mucus cells. The cilia beat rhythmically, this time bringing particles that are greater the 2 microns in size embedded in mucus, up through trachea, away from the lungs and towards the pharynx. This mucocillary escalator provides a mechanism of removing particulate matter too large

for the macrophages present in the lumen of the trachea to remove. In this way dust particles are removed.



Bronchi

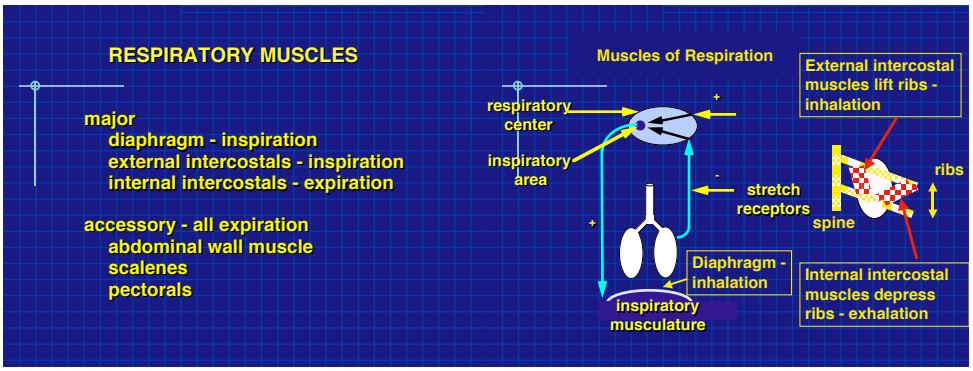
The bronchi branch again and again becoming smaller and smaller until at about the 17th branching the cartilage rings are replaced by muscles and small pouches of alveoli begin to form. The contraction of the muscles can constrict these airways causing difficulty in breathing as seen in asthma. These are referred to as respiratory bronchioles. It is at this level where gaseous exchange begins to take place.



Alveoli

Branching continues ending with alveoli at the 20th to 27th branching by which time, there is in excess of 100,000,000 alveoli. These alveoli are very small but because there are so many they have a large surface area to act as a diffusing surface for gas. The surface area where gaseous exchange takes place is about the same as a tennis court in the healthy adult lung.

We would expect that the small alveoli would collapse due to the surface tension but the pressure caused by a given surface tension is greater in the small alveoli. As described by the law of Laplace, pressure in a sphere is inversely proportional to the radius of a sphere. The pressures are greater in the small alveoli than larger alveoli. One would expect this great pressure to cause the gas to migrate to the larger alveoli with the lower pressure. This does not happen because of a surface-active agent called pulmonary surfactant that lowers the surface tension.[4] <#_edn4> [5] <#_edn5>



The Muscles of Inspiration

The mechanism of moving air is an exquisite system of muscle stimulation, contraction, and inhibition, relaxation. Air is brought into the lungs by increasing the size of the chest cavity. This increase occurs due the contraction of muscles. This action falls into two classifications passive respiration that exchange of gas that occurs at rest and active respiration that more vigorous exchange of higher volumes required when exercising.

Muscles of inspiration

The primary muscle of respiration is the diaphragm, a large dome shaped muscle found at the base of the lung separation the thoracic cavity from the abdominal cavity. The phrenic nerve coming from 3rd and 5th cervical segments of the spinal cord stimulates contraction of this muscle. [6] <#_edn6> This muscle is anchored around the circumference of the lower thoracic cage. As this muscle contracts the dome lowers increasing the volume of the chest cavity. The dome is lowered by as much as 10 cm. When a person breathes quietly.

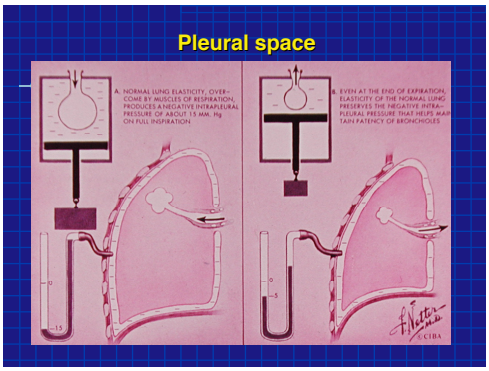
The intercostals muscles are attached to the ribs they are innervated by the intercostals nerves leaving the spinal cord between T 1 and T11. There two sets of intercostals muscles. The external intercostals elevate the anterior end of the ribs increasing the thoracic volume and cause inspiration. If these are paralyzed it does not effect inspiration to a great extent. Even if the chest is immobilized maximum voluntary ventilation is only decreased by 20 to 30%.

The other muscles involved in inspiration are the scalene and sternomastoids. These and other accessory muscles only function with strenuous muscular activity. Other accessory muscles include the posterior neck, trapezius and back muscles. The muscles aid inspiration by reducing resistance to air flow but do not increase the thorax volume, these include the mylohyoid, digastric, alae nasi, platysma, cheek muscles, levator palati, laryngeal muscles tongue muscles and the posterior neck muscles. [7] <#_edn7>

Muscles of expiration

Expiration is usually passive, the elastic fibers of the lung contract and squeeze out the air decreasing the volume of the thorax and retracting the diaphragm. There are some muscles that can be active is expiration, the abdominal muscles including the external oblique, rectus abdominis, internal oblique and tranversus abdominis. These are innervated from T6 to L1.

The internal intercostals muscles are innervated from T1 to T11. Contraction of these muscles depress the ribs and as the ribs move the downward the volume of the thoracic cavity decreases.[8] <#_edn8>

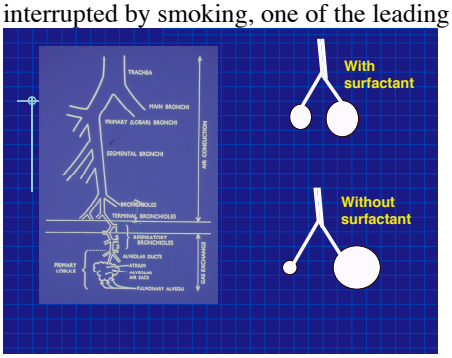


The plural Space.

Surrounding the lungs between the ribs and the lungs is a plural sack. This can be though of much the same as a large collapsed balloon. The elastic fibers of the lung try to pull the lung tissue away from the plural membrane adjacent the rib cage. This creates a negative pressure in the plural space. While the two plural membranes touch each other they can slide against each other with very little effort, if you try to separate the two membranes the negative pressure prevents any separation. However if the plural sack is separated or if there is a bleed into the space it will fill with air or blood. As this happens the effected lung will collapse due to the pull of the elastic tissue of the lung. This negative pressure is 4 to 5 cm of water.

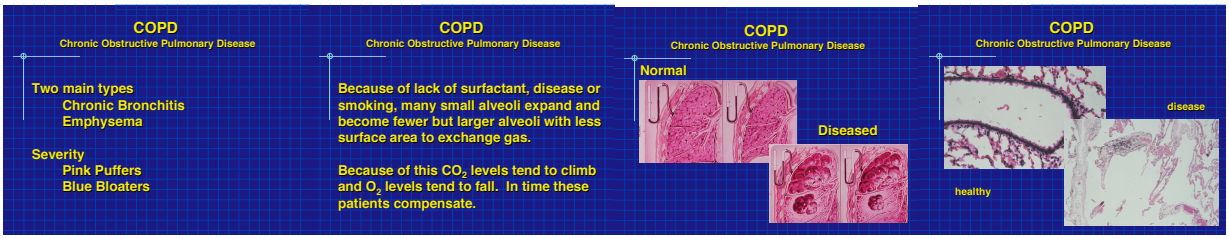
In addition to the elastic tissue of the lung there is another force trying to collapse the alveoli of the lungs. This is surface tension. When you make a soap bubble the smaller the bubble the closer the molecules are together in film of the bubble's surface. The molecules attract and the force of attraction is greater the closer the molecules are. Thus the pressure in a small soap bubble is greater than that of a large soap bubble. Similarly the alveoli of the lungs are very small spheres. The surface tension tries to collapse them. If this were allowed to happen the small alveoli would collapse and the larger ones would enlarge. This would decrease the surface area of the lung. With this decrease in area gas diffusion would decrease. This is exactly what happens with the Chronic Obstructive Pulmonary Disease, COPD, patient.

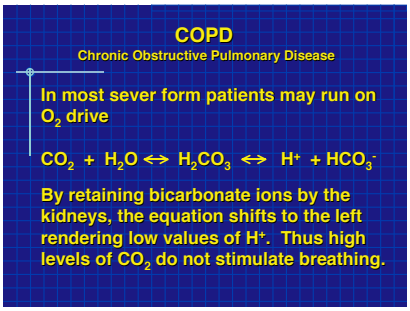
Fortunately, our lungs have a chemical, surfactant. Surfactant decreases surface tension and helps counter act the effect of surface tension keeping the small alveoli open. Surfactant is unique in that it has a greater effect the smaller the alveoli and a less effect on the larger alveoli. Obviously all alveoli are not the same size and without the surfactant the small alveoli would collapse. The alveolar cells synthesize surfactant and this synthesis is probably interrupted by smoking, one of the leading causes of COPD.



The COPD patient has other problems. To exhale they often depend on accessory muscles to apply further pressure on the lung tissue to move more air. This causes the plural pressure to become positive and can compress the small bronchioles preventing air spaces from empting. For this reason they will often purse their lips to increase the air pressure in these air passages to keep them open. They will have areas of the lung with very little exchange because the alveoli have collapsed. They will have blood shunt through these areas without taking on any oxygen or releasing any carbon dioxide. Some of the alveoli can become blebs that are quite large and can rupture into the plural space causing a lung to collapse. These blebs can be stressed if nitrous oxide is administered as nitrous like

any other gas will diffuse into close spaces in an effort to equalize the gas concentration inside the bleb. This results in an increase in total pressure and can cause the bleb to rupture.

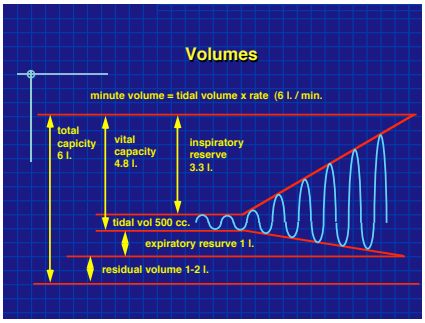




The function of the alveoli is to provide the gas that has been transported to them to be transferred to the blood stream. At the alveolar level there is only two cells separating the air from the blood. For the gas to enter the blood stream it must diffuse across the two cell membranes of the cells lining the alveoli and then across the two cells that make up the wall of the capillaries that carry the blood. The4 cell membranes of these two cells transfer most gases very rapidly from the air of the alveoli to the liquid of the blood.

Lung volumes

Adults breathe, on the average, 12 to 15 times a minute. Each breath is about 500 cc. Minute volume that amount of air we use each minute is found by multiplying the size of each breath by the number of breathes per minute. Our resting minute volume is between 6 and 7.5 L. / minute. As we exert we will inhale a larger volume of gas, the inspiratory reserve and when we exhale we will more of the gas from our lungs, the expiratory reserve. By increasing the inflation of the lung and exhaling gas that normally does not leave the lung the volume of each breath can increase to 4.8 L. If you breathed 30 times a minute at this volume you would be moving over 144 L./minute. There is a residual volume that can never be exhaled. In the young healthy adult it is about 1.2 L.



From this we see the total lung volume is about 6L. 1.2 L. is residual volume that can never be exhaled. At rest, the average adult breathes only 0.5L. per breath. With heavy exertion this can be increased to 4.8 L. per breath. The minute volume can very between 6 L./min and over 100 L./min with heavy exertion. Patients using nitrous oxide

will rarely move more than 6 L./ minute. It is important however, to provide that volume of fresh gas equal to or greater than their minute volume.

Not all air that gets to the alveoli came directly from the outside. This would only happen if on exhalation all gas spaces completely collapsed. This does not happen. Consequently some of the air in the alveoli is that left in the breathing spaces after the last exhalation. This is known as dead space. This volume is about .0.15L or 150 ml. Thus every 500 ml. Breath is about 350 ml. of fresh air and 150 ml. of air that was left after the last breath. In the worst case, if a person was breathing very shallow, 150 ml. per breath, no fresh air could get to the alveoli. In a healthy individual this is not an issue. If you depress respiration or increase the dead space because of pathology it can be lethal.[9] <#_edn9>

We have described tidal volume and minute volume the most important volume is alveolar ventilation, tidal volume minus dead-space volume times respiratory rate. This is the fresh air that gets to the alveoli each minute.

The Gas Composition

Up to the point we have discussed the Anatomy of the Respiratory system, the pipes and tubes that carry gas and the muscles the cause this to happen. Just what is the composition of that gas and how does that composition change as the gas go from the air to the alveoli, enters the blood and is transported to the various tissues of the body?

Air is approximately 21% oxygen and 79% nitrogen. . Standard sea level pressure is assumed to be 760 TORR (mm Hg), which equals 1013 millibars, a common unit of measurement in meteorology. It is also equal to 14.7 pounds per square inch of pressure.[10] <#_edn10> The pressure exerted by either gas can be determined from Dalton's law by multiplying the percentage of that gas by the pressure, 760 TORR times 0.21 equals a partial pressure of 160 TORR, which is read as a PO2 of 160. For nitrogen the partial pressure is 760 TORR times 0.79 or 600 TORR, or Pn2 = 600. This is the pressure either gas would exert if place in a sealed container and the other gas was removed without changing the volume of the container.

Once the air gets to the trachea it is completely saturated with water vapor. The partial pressure of water at body temperature, 37 degrees C, is 47 TORR. If we apply this new pressure we find the total pressure is 760 TORR. The pressure of water is 47 TORR. This leaves a partial pressure of 713 TORR for oxygen, nitrogen and carbon dioxide. We are now getting some mixing of the carbon dioxide that is coming out of the blood at the alveoli level. The PO2 is 713 times 0.21 or 149 TORR the Pn2 is 713 times 0.79 or 563 TORR and the new Pco2 is 0.3 TORR. Total pressure is still 760 TORR.[11] <#_edn11>

At the alveoli, the pressures changes. CO2 is arriving at the alveoli from the venous blood and defusing into the alveoli mixing with the air coming from out side the body. The partial pressure of CO2 is reflected in the pressure of CO2 in the venous and arterial blood. Gas will defuse from a greater partial pressure to a lesser partial pressure. Venous blood has a Pco2 of 46. Arterial blood carries a PaCO2 of 40. The PO2 has dropped from 149 to 104 because oxygen is defusing into the blood from the alveoli. The partial pressures in the alveolar gas for oxygen is 104 TORR, 40 TORR for carbon dioxide is still 47 TORR for water vapor and is now 569 TORR for nitrogen. The total is 760 TORR.

The arterial blood has a partial pressure PaO2 of 100. The gradient of 104 TORR in the alveoli to 100TORR attests to the efficiency of transfer from the alveoli to the blood. The venous blood returning to the lungs will have a PO2 of 40 TORR. The passage through the lungs will raise this to the 100 TORR of the arterial blood. Gas will diffuse from a higher partial pressure to a lower pressure. It does not reflect the true picture because much of the oxygen is carried on the hemoglobin molecule. Much more oxygen is carried that you would expect from a partial pressure of 100 TORR in a pure liquid system.

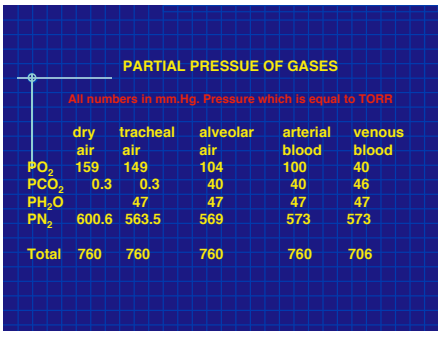
The body's tissues have a pCO2 of 46 TORR this compares to the PaCO2 of 40 TORR of the arterial blood. Carbon dioxide defuses from the tissue to the blood bringing the PCO2 in venous blood to the 46 TORR seen at the alveoli. Oxygen saturation of tissue, PO2 is 40 TORR. The PaO2 of 105 TORR causes a shift of oxygen from the blood into the tissues.

Some tissue is more sensitive to lower levels of oxygen than others. It has been shown that pilot's night vision is

affects at altitudes as low as 5,000 feet. The atmospheric pressure at 5,000 feet is about 630 TORR Oxygen saturation would be about 132 TORR in the air, and Alveolar PO2 about 88 TORR. The retina of the eye is affected at mild hypoxia. At 8,000 feet forced concentration, fatigue and headache may occur. The atmospheric pressure is about 580 TORR PO2 would be about 122 TORR, Alveolar PO2 about 81TORR. At 14,000 feet forgetfulness, incompetence and indifference has been reported in pilots.

The pulse oximeter reading was taken on a person who lives at sea level and was at 14,000 ft in the Andes of Bolivia in South America. It shows a saturation of hemoglobin of 75% and a pulse rate of 66. The person was on a beta-blocker for hypertension that kept the pulse low. A more normal reading would have been 85 to 90 beats per minute at this saturation. She reported being out of breath and having a difficult time "catching her breath.

The Atmospheric pressure is about 470 TORR; PO2 is about 99 TORR, Alveolar PO2 about 66 TORR. Women seem to see these effects a bit lower than men.[12] <#_edn12> For these reasons, pilots and aircrews are required to be on supplemental oxygen above 11,500 feet.



The Venus blood coming to the lungs has a partial pressure of carbon dioxide of 46 TORR the partial pressure of carbon dioxide in the alveoli is 40 TORR so the carbon dioxide defuses into the alveoli. The pressure equalizes in the alveoli at 40 TORR and this same level is found in the arterial blood. This becomes important because the carbon dioxide determines the pH of the blood. The pH or the acidity of the blood is a major stimulus to breathing.

The body has decided that 100 TORR of oxygen and 40 TORR of carbon dioxide is that appropriate levels for arterial blood and has a rather exquisite set of monitors and neural mechanisms to maintain these levels.

The transportation of Gases

Once the gas has been dissolved in the blood it is transported to all the tissues of the body. The gases are not carried

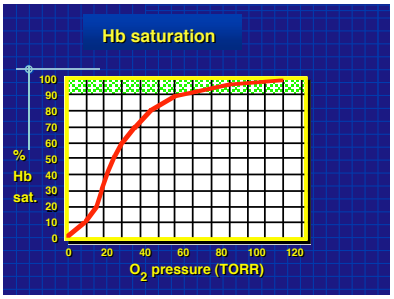
in simple solution. CO2 is about 20 times more soluble and diffuses about 20 times as fast as Oxygen. [13] <#_edn13> Oxygen does not easily dissolve in water. Only about 1.5% of oxygen is carried in solution. The rest, 98% is carried on hemoglobin molecules in the red blood cells.[14] <#_edn14>

The iron can be reduced or oxidized. If it is reduced and has no oxygen it is called deoxyhemaglobing, which is common to venous blood. With oxygen it is referred to as oxyhemaglobin common to arterial blood. If the iron has a molecule of carbon monoxide it will not transport oxygen and is known as carboxyhemoglobin. If the iron is oxidized it is known as methemoglobin. This can be reversed with methylene blue and can result from some local anesthetics.[15] <#_edn15>

Hemoglobin is a protein called globin and an iron containing iron called heme. Each molecule of hemoglobin has four heme groups and each heme can carry one oxygen molecules. Each red blood cell contains 200 to 300 million molecules of hemoglobin.

The percentages of the heme groups that hold an oxygen molecule is know as the oxygen saturation. This can be measured by a pulse oximeter and can be represented by the Oxygen-hemoglobin dissociation curve. This curve shows 35% saturation PsO2 in contracting muscle tissue with a PO2 of 20 TORR. Venous blood has PsO2 75% at a PO2 40 TORR. Arterial blood will be 100% saturated PsaO2 of 100 at a PO2 of 105 TORR.

The saturation of oxygen is not a linear relationship with oxygen concentration. Doubling the oxygen concentration does not double the % of hemoglobin saturation. The relationship is depicted by and S-shape cure know as the oxygen-hemoglobin curve.

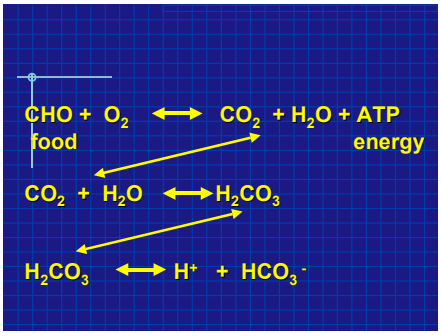


This SaO2, oxygen hemoglobin saturation can be read noninvasively with a pulse oximeter. The oximeter shines a light through a finger, ear or toe. The blood was change color depending on the percentage saturation. Sensors read the change in color of the light that has passed through the body part and reads saturation on a digital scale. You should realize it may take up to a minute or more to see a drop in saturation if a patient stops breathing. The information is most important to identify trends in saturation early before low levels of saturation occur. Most machines also read pulse rate. Some have alarms to warn of low saturations or high or low pulse rates. The more expensive modes also show a graphic display of the pulse.

The oxygen is bound more tightly to the hemoglobin molecule with a high pH (basic) blood and less tightly with a low pH (more acidic) blood. The more carbon dioxide dissolved in the blood the lower the pH (more acidic) the blood is. So more carbon dioxide as we would expect in muscles that were working would lead to the release of

more oxygen necessary for the muscles metabolism. A similar shift occurs in the presence of heat. When muscles work they create heat and this heat aids the metabolism of the muscle by releasing more oxygen from the hemoglobin.

Carbon dioxide is responsible for the pH of the blood. 10% of CO2 is carried dissolved in blood. 30% is bound to hemoglobin and 60% is carried as a bicarbonate ion.[16] <#_edn16>



CHO (food) + O2 ----- CO2 + H20 +ATP[17] <#_edn17>

CO2 + H20 ------ H2CO3 -------- H+ + HCO3-

This reaction is catalyzed by carbonic anhydrase

H2CO3 -------- H+ + HCO3-

The H+ is buffered by the hemoglobin of the red blood cells. Much of the bicarbonate diffuses outward. This causes Cl- ions to be drawn into the red blood cells and is known as Chloride shift. Chlorine is brought into the red blood cells in the tissue and released from the red blood cells in the lungs[18] <#_edn18>

pH is the negative log of the H+ (hydrogen ion concentration). The greater the level of the carbon dioxide the greater the amount of carbonic acid, H2CO3. The more carbonic acid leads to a higher concentration of hydrogen ions, H+ and the more bicarbonate ions. This last equation can be shifted to the left by increasing the concentration of the bicarbonate ions.

The pH of the blood can be calculated from the Henderson-Hasselbach equation,

pH+pKa + log([A-]/[HA])

pH =-log[h+] pH equals the negative log of the hydrogen ion concentration, in blood about 7.4

pKa=-log(Ka) Ka is the equilibrium constant pKa for this week acid is 6.1

There is about 20 times as much HCO3- (A-) as there is H2CO3 (HA)

Thus we see pH(7.1)= pK(6.1)+log(20)

This buffer system helps with the second to second control of the pH of the blood. It is effective only because the lungs are so effective in control of the pH on a minute-to-minute basis and the kidneys are able to make adjustment on a longer-term basis.[19] <#_edn19>

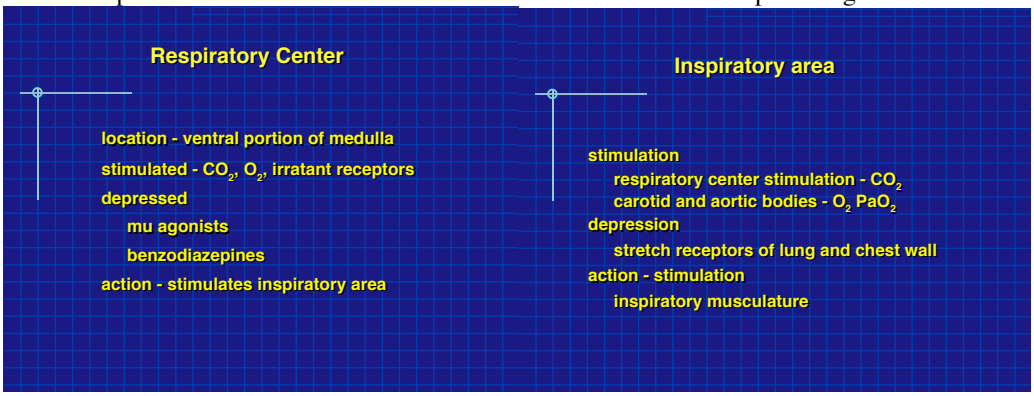
The normal pH of the blood is 7.40, if it is less than 7.35 it is termed acidosis and greater than 7.45 alkalosis. Hyperventilation will blow off CO2 and cause a respiratory alkalosis. Holding you breath or breathing less than ideal will cause respiratory acidosis. Acidosis can also occur as a result of metabolic problems. Hyperventilation can partially compensate for this alkalosis.

The response to high carbon dioxide levels of a high hydrogen ion concentration (H+), a low pH can be reversed if additional bicarbonate ions are in the blood. In the case of emphysema or chronic bronchitis patients (COPD) where there is a decrease in the surface area of the lung, CO2 is retained at higher concentrations in the blood. This leads to higher concentrations of hydrogen ions and a lower (more acidic) pH. In response to this increase in H+ the kidneys excrete hydrogen ions and retain bicarbonate ions. The buffering that occurs because of the retention of bicarbonate ions blunts the effect of lower pH that would normally stimulate breathing. These patients frequently will have abnormal high lung volume, they have difficulty exhausting air from their lungs

A further problem can occur. Inspired air is not necessarily evenly distributed through out the lung. Some areas may not inflate or may not be able to deflate causing a lowered concentration of oxygen. The lung has a mechanism of vasoconstriction to cut down the blood flow to these areas of low oxygen. There can also be areas of scaring where little if any blood flows. It is also possible to have areas where the vessels are too large to effective defuse gases so blood my shunt buy alveoli with out being properly oxygenated.[20] <#_edn20>

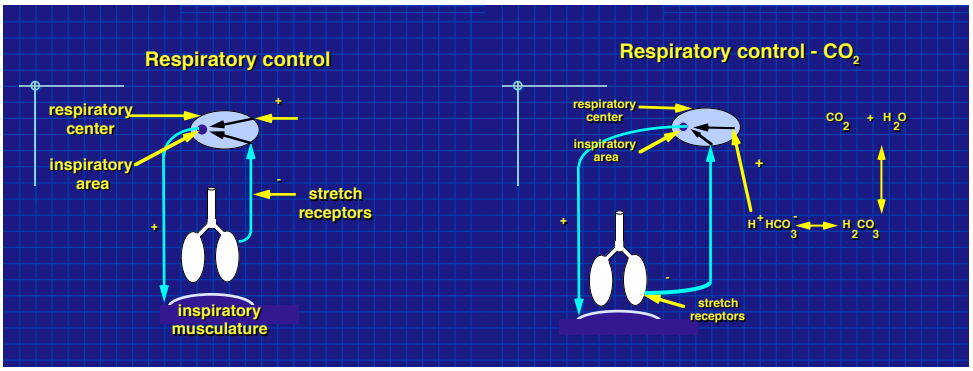
The Control of the System

Breathing control can be automatic controlled by the concentration of gases in the blood or it can be controlled consciously under direct control of the cerebral cortex. There are also some reflex mechanisms that come into play for sneezing, coughing, sighing, yawning, sobbing, crying laughing and hiccupping.[21] <#_edn21> CO2 and the resultant pH changes of the blood is the primary stimulus to breathing. Chemical receptors in the medulla oblongata are sensitive as the result of the changes of the pH of the cerebrospinal fluid. There are also peripheral chemoreceptors in the aortic and carotid bodies that are sensitive to the pH changes.



A decrease in PO2 directly stimulates breathing but only if the PO2 is lower than 50 TORR. A drop in oxygen concentration also causes the chemoreceptors to be more sensitive to changes in PCO2 and pH. [22] <#_edn22>

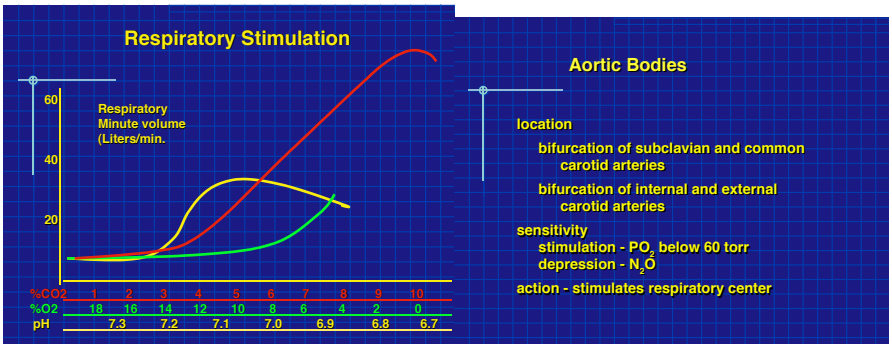
Once the lung is expanded to a tidal volume greater than 1L inspiration is inhibited by stretch receptors in the lungs, the Hering, Breauer reflex.[23] <#_edn23>



The body uses 2 to 300 ml of oxygen each minute. This produces an equal amount of CO2. To remove this CO2 and to replenish the O2 we breathe. This rhythmic contraction and relaxation of muscles are controlled by the nervous system. The area of the brain responsible for the stimulatory nerve impulses is the reticular formation of the brain stem; it is referred to as the respiratory center. It has three functional divisions, a rhythmicity area, a pneumotaxic are and an apneustic area.

The basis of rhythm is found in the medulla. Inspiration lasts about 2 seconds, expiration 3 seconds. After two seconds the signals stop and the muscles become inactive. Expiration is passive elastic recoil of the tissues. At high levels of activity expiration becomes active and signals are sent to the muscles that can assist expiration.

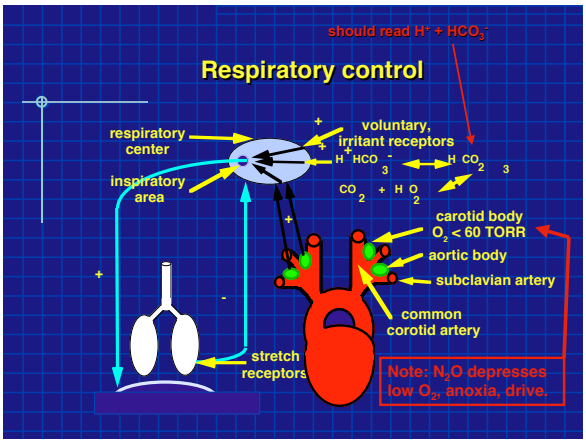
The Pneumotaxic area sends inhibitory signals to the inspiratory area. These signals turn off the inspiratory area so as to prevent over inflation of the lungs. This are receives signals from stretch receptors in the bronchi and bronchioles. This causes inspiration to stop and expiration begins. This reflex is known as the Hering-Breuer reflex.



The apneustic control occurs from several sensory areas. The central chemosensitive area is in the medulla and is sensitive to pH changes. Peripheral chemoreceptors are sensitive to H+(pH), CO2 and O2 levels in the blood ad are found in the carotid bodies near the bifurcation of the common carotid arteries and in the aortic bodies that are clustered in a region near the arch of the aorta.

If there is a slight change in concentration of CO2, the central chemosensitive area is stimulated. pH changes occur more rapidly in the cerebral spinal fluid because there are fewer buffers. The peripheral receptors are sensitive to both CO2 and H+. Signals from both of these areas cause the depth and rate of respiration to increase. This is true up to about 10% CO2. Beyond 10% carbon dioxide depresses respiration and can be a general anesthetic.

Peripheral receptors are also responsive to low concentrations of O2. However, the pressure of oxygen must drop below 50 TORR to see stimulation. If it drops much below 50 TORR the cells of the inspiratory area suffer oxygen starvation and do not respond well. As the rate falls breathing stops and pO2 falls lower yet. This areas sensitivity to low oxygen levels can be depressed by nitrous oxide.



When administering nitrous oxide oxygen sedation there are several parts of the foregoing discussion that are very important:

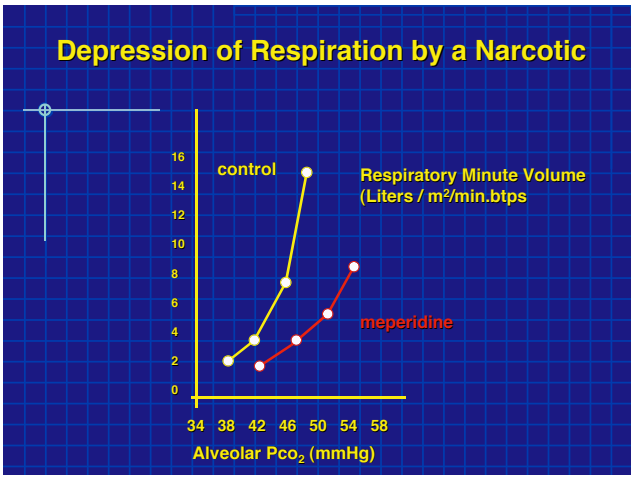
We must be sure we do not allow any rebreathing of exhaled gas. If this were to occur, carbon dioxide levels would climb and stimulate breathing because of the change in the bloods and cerebral spinal fluids pH. It is not necessary to give the patient carbon dioxide to stimulate breathing.

We must be sure we are always providing an adequate supply of Oxygen. Practically there is very rarely a reason go give more than 50% nitrous oxide or less than 50% oxygen.

We must be sure we are providing an adequate volume of fresh, no carbon dioxide, gas to at least match the patient's minute volume. 5-6 L per minute are adequate for most patients.

We must be aware of those conditions that do not tolerate nitrous oxide. From a respiratory standpoint that is COPD, the chronic bronchitic or emphysematic patient. Both can have defects in their lungs that could be expanded and rupture if nitrous oxide defuses into these closed blebs. In addition those patients in the most sever forms of these diseases may be breathing on oxygen drive because of their heavily buffered carbon dioxide-pH drive has been depressed. The high levels of oxygen typically given with nitrous oxide mixtures could depress their oxygen drive.

We never want to approach general anesthesia because of the effect this level of sedation has on the swallowing and laryngeal reflexes. Fortunately nitrous oxide is a very week anesthetic and with the exception of a very few very sensitive individuals the author has seen who were under general anesthesia at 30% nitrous oxide. Giving nitrous oxide in combination with other central nervous system depressants can also lead to general anesthesia and all its complications.



It would be difficult to design a better sedative than nitrous oxide. This is largely true because it is such a week general anesthetic at atmospheric pressures. It is a very potent general anesthetic when given in a hyperbaric chamber with several atmospheres of pressure. The fact that nitrous is administered via the respiratory system is another very large plus. It crosses readily into the blood stream and once the source of gas is removed it leaves the blood equally quickly.

All of this contributes to the ease and effectiveness of nitrous oxide as a dental sedative.

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