8

Respiratory Basics

Quick Contents

Most patients having a cardiovascular emergency are in need of supplemental oxygen and in some cases airway management. The knowledge and skills necessary to provide respiratory support are addressed in the next three chapters.

This chapter provides you with the basics of respiratory anatomy and physiology. It explores airway anatomy, the mechanics of ventilation, alveolar gas exchange and gas transport.

Respiration is an umbrella term for the total process of delivering oxygen to the cells and the removal of carbon dioxide, a by-product of aerobic metabolism. This includes ventilation (inspiration and expiration), gas exchange and gas transport.

Effective respiratory management begins with a good handle on respiratory anantomy and physiology. These topics are not difficult. While this chapter might seem peripheral to the core content of managing cardiac emergencies, a generous share of gems and a better grasp of the "big picture" awaits within.

Relax. Take a good cleansing breath. Now, let's get to it.

If you can walk, you can dance.
If you can talk, you can sing.

Zimbabwean Proverb

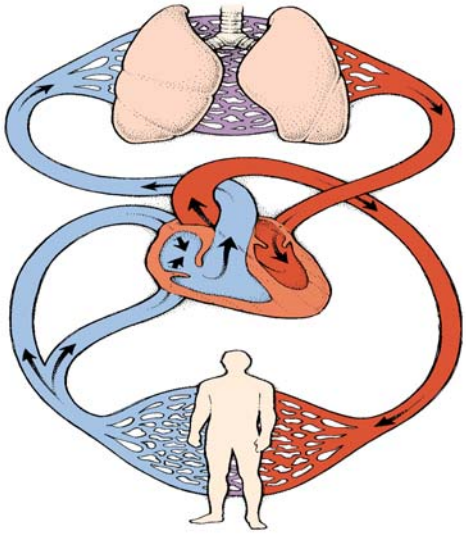
Respiratory Anatomy

The respiratory system is an intimate partner with the cardiovascular system. Together, both systems in tandem provide oxygen to tissues of the body. A sufficient oxygen supply facilitates aerobic metabolism and abundant energy production (chapter 2). A serious lack of oxygen - and thus energy - quickly impairs the organ affected.

Aerobic metabolism uses oxygen to produce energy in the form of adenosine triphosphate (ATP). When glucose is burned or oxidized, as many as 36 ATP are created. Water and carbon dioxide are the bi-products. **Anaerobic metabolism** creates only 2 ATP in the absence of oxygen with lactic acid a toxic by-product of this reaction.

At rest, a healthy adult requires about 250 ml/minute of oxygen to sustain life. Fortunately, respiration normally provides an abundance of oxygen, about 1000 ml per minute at rest. This abundance of oxygen provides a significant physiologic reserve. This physiologic reserve has an important preservation / protective function for those who are victim to a sudden cardiac death or periods of sudden hemodynamically unstable tachycardias and bradycardias.

Figure 8.1 Cardiorespiratory System



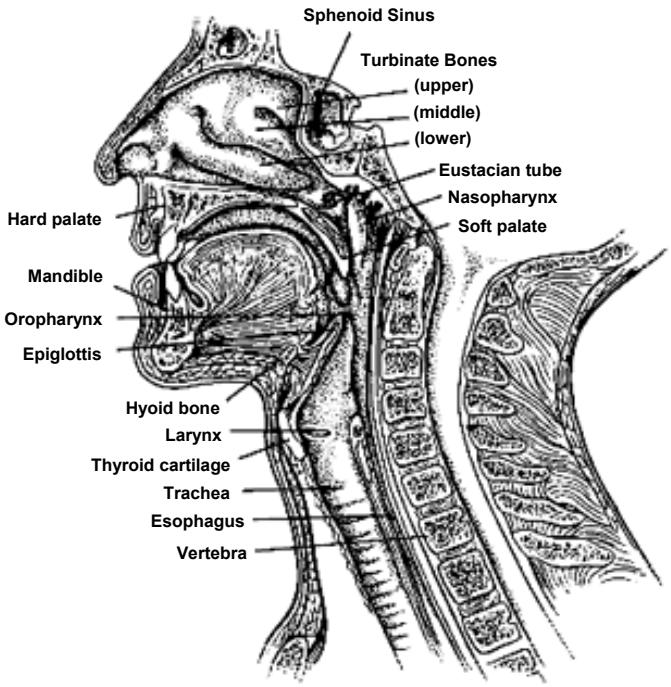
The heart, the vascular network and the lungs function interdependently to ensure an adequate oxygen supply to the tissues. The venous system and the right side of the heart delivers carbon dioxide - a biproduct of aerobic metabolism - to the lungs for speedy removal (diffuses 20 times faster than oxygen). Blood oxygenated by the lungs is pumped by the left side of the heart via the arterial system to the tissues.

In addition to ventilation and gas exchange, the respiratory system engages in several other important tasks . The lungs moderate cardiac preload, help maintain blood acid-base balance, clear toxins, serve as an endocrine organ and act as a filter for emboli. Not one of these tasks could be sustained, though, if the lungs failed in their primary function - the gas exchange of oxygen and carbon dioxide with the blood stream. This process is facilitated by the combined actions of the upper and lower airways.

The Upper Airways

The upper airways are more than just openings to the lungs. Within the first few centimeters of the mouth or nose, air is cleaned, humidified and the air temperature is regulated to within a few degrees of body temperature. Key upper airway structures are depicted in figure 8.2.

Figure 8.2 Upper Respiratory Tract



Certain structures are especially key to airway management. For example, the displacement of the tongue posteriorly against the soft palate and posterior oropharynx is the leading cause of airway blockage in the unconscious patient. Manipulation of the mandible with a modified jaw thrust (or head tilt-chin lift) is an effective method to lift the tongue and open the airway.

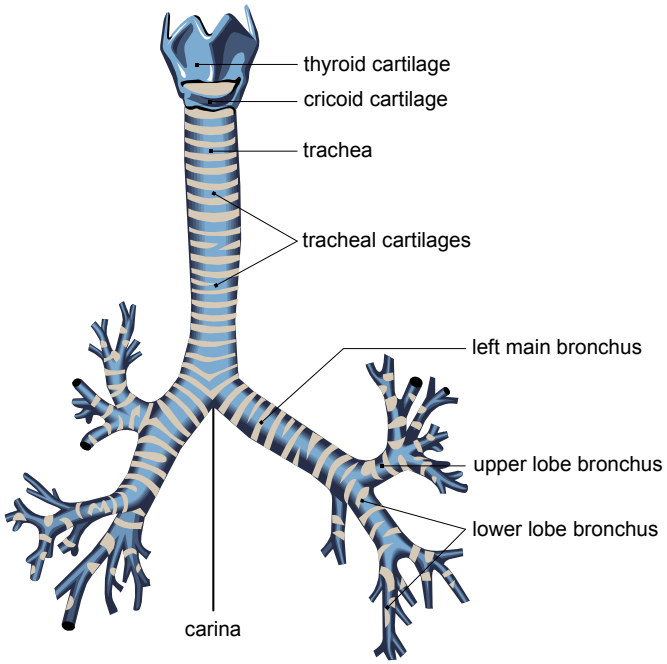
The inspiratory volume provided through the mouth can be as much as five times the volume through the nose. For those who are receiving supplemental oxygen via nasal prongs, note that the oxygen delivered to the patient may be substantially less if the person is mouth breathing. A simple face mask may be a more suitable method if hypoxia is likely.

The vallecula, the notch between the base of the tongue and the epiglottis, is an important target during intubation. The tip of the laryngoscope blade pulls up from this notch to cause the epiglottis to open (in adults). The epiglottis, a leaf- shaped cartilage that acts like a trap door covering the opening to the trachea, is another common site for airway obstruction. Note that an oversized oral airway can push the epiglottis closed and obstruct the opening to the trachea.

The Trachea and The Lungs

The trachea connects the upper airways to the lungs. It is composed of a number of cartilagous rings to provide stability and patency. Along the trachea, the cricoid cartilage is an important anatomical landmark when assisting intubation or using a bag-valve-mask. The membrane between this cartilage and the thyroid cartilage is the location for a cricothyroidotomy.

Figure 8.3 Cricoid, Trachea and Major Bronchi



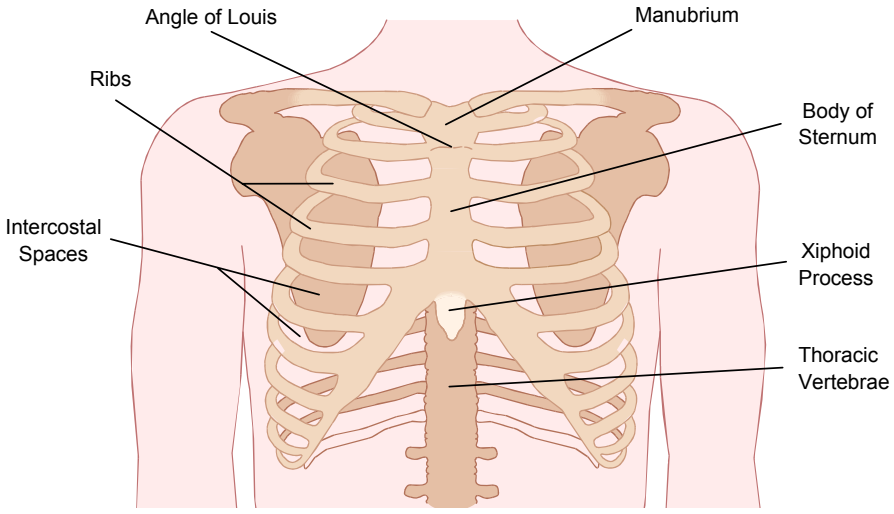
The **cricoid cartilage** rings around the trachea. Compression of the cricoid will normally not collapse the trachea (in adults) while providing an easier visual of the larynx. During ventilations using the bag-valve-mask (not intubated), cricoid pressure can compress and occlude the esophagus (forcing the trachea back against the esophagus). As a result of cricoid pressure, gastric inflation and vomiting can be minimized.

The trachea terminates at the carina branching to the right and left mainstem bronchus. The right bronchus typically dips at a steeper angle than the left bronchus (see Figure 8.3 on the previous page). As a result, an endotracheal tube that is inserted too deeply tends to go down the right bronchus. This can cause the ventilation of only the right lung. Similarly, most aspiration pneumonias tend to involve the right lung.

The 2nd intercostal space (ICS) is a significant landmark in cardiorespiratory management. For example, rapid **needle decompression** of a tension pneumothorax is performed in the 2nd ICS at the mid-clavicular line. Also, the aortic valve is often best auscultated to the right of the sternum in the 2nd ICS, the pulmonic valve to the left of the sternum in the 2nd ICS.

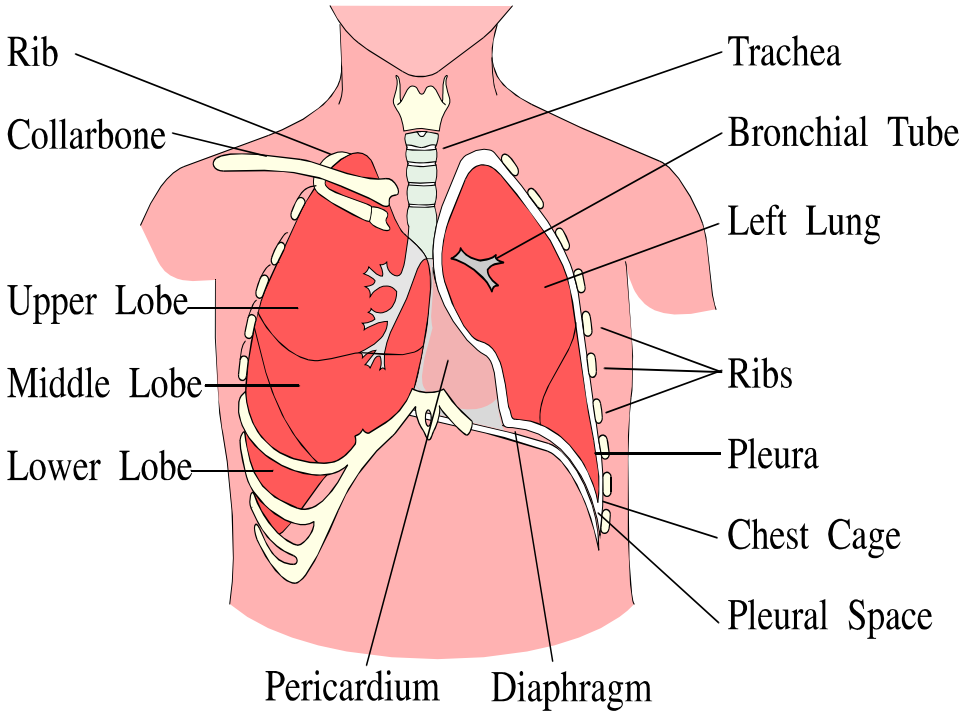
The lungs are housed in a skeletal cage shaped by the sternum, the ribs and the spine (see figure 8.4). The sternum is composed of 3 structures: the manubrium, the sternal body and the xiphoid process. The angle of Louis is the juncture of the manubrium and the sternal body. This landmark is significant for monitoring central venous pressure and for locating the second costal cartilage (and the 2nd intercostal space).

Figure 8.4 Skeletal Landmarks of the Chest Cage



The lungs are protected by 2 layers, the outside parietal pleura and the inner visceral pleura. Surfactant produced between the pleura minimizes friction and surface tension between these 2 layers allowing for unimpeded separation during expiration. The accumulation of blood or exudate in the pleural space can negate the effects of the surfactant, increase the work of breathing and restrict lung expansion. A tear in the pleura can lead to a tension pneumothorax.

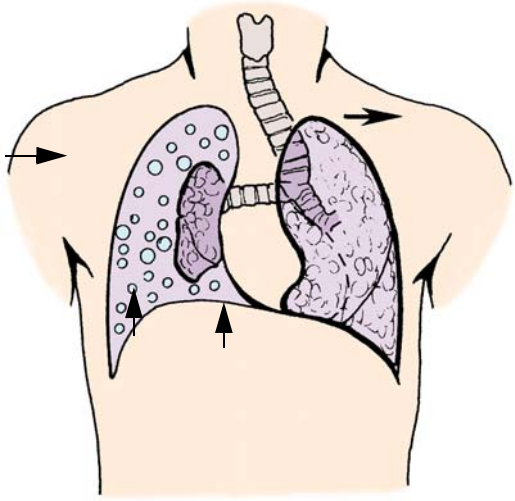
Figure 8.5 Lung Anatomy



The lungs and the skeletal structures that encase the lungs have an important relationship. The lungs tend to recoil on expiration inwards while the rib cage (i.e. ribs, sternum and clavicles) restricts the extent of this recoil. This creates a negative intrapleural pressure between the visceral and parietal pleura during expiration. The diaghragm's role is to draw the lungs down and thus expand the lungs causing a fall in lung pressures. As a result, air rushes in from the atmosphere.

For a tension pneumothorax to occur, air is inspired into both the lungs and the pleural space. On expiration, this air is often trapped by a flap established by the original tear thus blocking the exit hole. With each subsequent breath, the volume between the pleural linings increases. An expanding pleural space can collapse the affected lung and exert pressure on the heart, restricting diastolic filling. A severe reduction in stroke volume can result, leading to low cardiac output and even pulseless electrical activity.

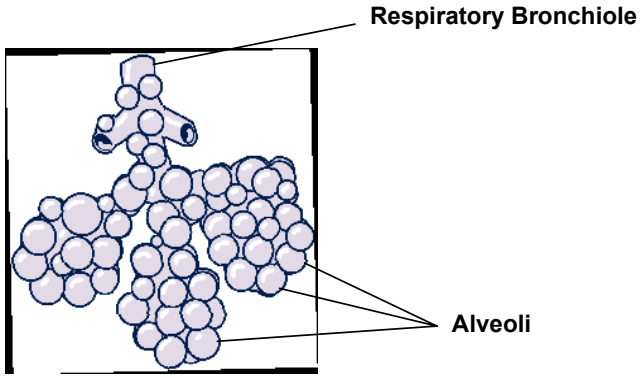
Figure 8.6 Tension Pneumothorax to the Right Lung



Note the effects of the an expanded pleural space (tension pneumothorax) on the heart. The filled pleural space can collapse the effected lung and exert high pressures to the surfaces of the heart. As a result, the heart becomes wrapped in a confining straight jacket, preventing it from opening and filling. As a result, stroke volumes decrease markedly.

Within the lungs, the respiratory tree branches from the main bronchi to the smaller bronchioles becoming narrower but exponentially more plentiful with each successive bifurcation. After about 23 divisions, the respiratory brochioles terminate at about 300 million alveoli. The alveoli facilitate gas exchange between the atmosphere and the pulmonary capillaries. The alveoli are the functional respiratory units of the lungs.

Figure 8.7 The Acinus



After about the 17th generation of branching from the trachea, alveoli begin to appear along the respiratory bronchioles and later as terminal structures of the alveolar ducts. The alveolar ducts and the alveolar sacs (which form the acinus) are the only airways that can engage in gas exchange. All airways prior to the acini are called the conducting airways. This conducting zone is also referred to as the anatomical dead space.

The alveoli are typically situated in clusters sharing an alveolar septum (wall). The alveolar septa contribute significantly to maintaining the shape of the alveoli. This is an important function in reducing breathing workload as collapsed alveoli require considerable energy expenditures to re-open.

An alveolus consists of Type I and Type II cells. The Type I cells are larger cells that provide the majority of the alveolar surface, which is only one cell thick. The Type II cells are responsible for secreting surfactant, a necessary liquid medium to reduce water tension (a tendency of water coated surfaces to attract each other) within the alveolus.

Pulmonary and Bronchial Circulation

In order for gas exchange between the atmosphere and the blood stream to occur, the alveoli must come in contact with an ample blood supply. This is accomplished by the pulmonary circulation. The entire blood volume ejected from the right ventricle enters the pulmonary circulation. Each minute the pulmonary blood flow is virtually equal to the cardiac output ejected by the left ventricle (about 5 litres/minute).

The right lung pumps mixed venous blood collected from the body to the left and right pulmonary arteries. The pulmonary arteries branch successively to form abundant smaller arteries that culminate in about 280 billion pulmonary capillaries that envelop the alveoli. Therefore each of the 300 million alveoli are perfused by about1000 capillaries. Wow!

The pulmonary capillaries are highly distensible and collapsible because of the scarcity of vascular smooth muscle and elastic fibers that are common along the systemic arteries. Thus, pulmonary vascular resistance and pulmonary arterial pressures are quite low when compared to systemic vascular resistance and systemic arterial pressures. For example, a pulmonary artery pressure could typically be only 24/8 mm Hg.

A low pulmonary vascular resistance (PVR) is the key to why the right ventricular myocardium is about 1/3 the thickness of the left ventricle. Because of a low PVR (low afterload), the right ventricle requires less force and less muscle mass to eject blood into the pulmonary circulation. Because of the relatively high systemic vascular resistance of the systemic arterial system, the left ventricle must exert a much more substantial contractile force to eject an adequate blood supply to the tissues. This requires a larger myocardial muscle mass.

While PVR is generally low compared to systemic vascular resistance, various factors increase PVR and the perfusion of the pulmonary capillaries. For example, with increased lung volumes, the pulmonary capillaries are stretched and elongated, thus reducing the diameter of the vessel. A narrowed vessel is much more resistive to blood flow (resistance is inversely proportional to decreases in vessel radius to the fourth power - R^4). Table 8.1 provides a annotated list of factors that affect PVR.

Table 8.1 Factors That Affect Pulmonary Vascular Resistance

Factor	Effect	Rationale
gravity	decreased PVR in dependent regions of the lungs	the increased vessel pressure caused by gravity in dependent lung regions open capillaries further
lung volumes	increased PVR with increased lung volumes	increased lung volume stretches, elongates and thus narrows the pulmonary capillaries
interstitial congestion	increased PVR	interstitial congestion compresses and narrows the pulmonary capillaries
positive-pressure ventilation	increased PVR	positive-pressure ventilation compresses and narrows the pulmonary capillaries
histamine	increased PVR	pulmonary vasoconstrictor
alveolar hypoxia	increased PVR	pulmonary vasoconstriction occurs in an effort to redirect blood to more functional alveoli
alveolar hypercapnia	increased PVR	pulmonary vasoconstriction occurs in an effort to redirect blood to more functional alveoli

The lungs also requires a supply of oxygenated arterial blood to sustain the lung tissues. This is provided by bronchial arteries that branch off of either the aorta or the intercostal arteries. This is called the bronchial circulation. Normally, the bronchial circulation accounts for about 2% of cardiac output from the left ventricle. Bronchial blood flow increases during periods of high oxygen demand i.e. conditions such as asthma that cause high breathing workloads.

The desaturated venous return from the bronchial circulation mixes largely with the oxygenated blood returning from the lungs to the left atrium. This forms a natural right-to-left shunt of blood that bypasses the lungs. As a result, the oxygen saturation of systemic arterial blood (obtained by pulse oximetry) is slightly less than the expected 100% saturation that normally occurs at the pulmonary capillaries.

Flash Quiz 8.1

1. Obstruction of the upper airways is most often caused by:

a) cut up hotdogs
b) the tongue
c) the epiglottis
d) hard candy

2. Cool air that enters the nose and mouth is warmed to within a few degrees of core body temperature by the time it enters the lungs.

True or False

3. Each alveolus is enveloped in about (1, 10, 100, 1000) pulmonary capillaries.

4. Compression of the cricoid cartilage can be useful during:

a) intubation
b) forceful vomiting
c) positive pressure ventilations using a bag-valve-mask
d) a and c

5. Surfactant :

a) is produced by Type I cells in an alveolus
b) is a substance that reduces surface tension within an alveolus
c) production increases the work of breathing
d) all of the above

6. Gas exchange occurs at the (circle all that are correct):

a) alveolar ducts
b) conducting airways
c) alveoli
d) acini

7. Anatomical dead space (circle all that are correct):

a) is the space between the visceral and parietal pleura
b) does not engage in gas exchange
c) is reduced with an asthma attack
d) is a term that refers to the conducting airways

Answers: ***1.*** *b)* ***2.*** *true* ***3.*** *1000* ***4.*** *d) Note that cricoid pressure during forceful vomiting can result in an esophageal rupture* ***5.*** *b)* ***6.*** *a), c), d)* ***7.*** *b), c), d)*

8. Blood flow to the lungs (circle all that are correct):

a) includes both the pulmonary circulation and bronchial circulation
b) is greater than the cardiac output from the left ventricle
c) is greater than the blood flow to any other organ in the body
d) exits through the pulmonary veins

9. The left ventricle has a larger muscle mass than the right ventricle because the left ventricle must pump blood further into the body.

True or False

10. Pulmonary vascular resistance is usually equal to systemic vascular resistance.

True or False

Respiratory Physiology

Knowing about the structures of the upper and lower airways is important to be able to differentiate between normal and pathological findings. Understanding why and how these structures function together to facilitate respiration is at least as important.

In general, answering the how and why things work demands attention to the finer details of a process. The next few sections begin with a look at the mechanics of spontaneous and assisted breathing. We will then delve into the microscopic processes that connect breathing with energy production: gas exchange and gas transport.

Mechanics of Ventilation

Ventilation involves inspiration and expiration, moving air into and out of the lungs. Most of us simply call it breathing. An average adult breathes in about 500-700 ml of air with each breath at a rate of about 15 breaths a minute for a daily volume of about 13,000 litres. How does air move into and out of our lungs? What factors influence the efficiency and effectiveness of breathing? This section addresses these questions.

Air moves from an area of high pressure to an area of lower pressure. As the diaphragm contracts and pulls down, the lungs are also drawn down (inspiration). The intrapleural pressure generated between the visceral and parietal pleural layers

***Answers:* 8.** *a), b), d) The lungs receive about 2% more blood than the cardiac output from the left ventricle due to the contributions from both the right ventricle and the bronchial arteries. The heart receives about 10 litres of blood each minute - 5 litres to the right and 5 litres to the left.* **9.** *d) Note that cricoid pressure during forceful vomiting can result in an esophageal rupture* **10.** *True*

becomes increasingly negative. The visceral pleural layer connects with the lung wall while the parietal pleural layer connects with the chest wall (i.e. ribs). A more negative intrapleural pressure pulls upon and opens the alveoli further.

In a closed system pressure decreases as volume increases. The expanded alveolar volume from inspiration causes a reciprocal fall in alveolar pressure below atmospheric pressure, and air rushes into the lungs to equalize this pressure gradient.

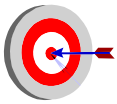
In a closed system, pressure is inversely related to volume.

This negative-pressure ventilation, better known as breathing spontaneously, is the mechanism behind normal breathing. The external intercostals and scalene muscles are also used in normal inspiration. The sternocleidomastoid muscle is an accessory breathing muscles used during periods of rapid breathing and dyspnea.

Expiration is usually a passive process requiring minimal work during normal breathing. As inspiratory muscles relax, the inward elastic recoil (see page 167) of the alveolar wall reduces alveolar volume. Alveolar pressure begins to surpass atmospheric pressures and air exits the lungs. Forced expiration, as might occur with increased breathing rates, actively engages the abdominal and intercostal muscles to reduce the size of the chest cavity more rapidly than the time taken at rest.

The diaphragm is the main muscle responsible for inspiration. It is innervated by the **phrenic nerve**, arising from the 3rd-5th cervical segments of the spine. The accessory breathing muscles (i.e. intercostals) are innervated by nerves that arise low in the thoracic and lumbar segments. These simple facts help explain why spinal cord injuries in the lower back generally do not adversely affect one's breathing (the diaphragm remains functional); whereas, high cervical spinal injuries typically result in the inability to breathe.

To review, spontaneous inspiration causes the pleural cavity to increase, intrapleural pressures to become negative, and air to move from an area of higher pressure (the atmosphere) to an area of lower pressure (the lungs). During expiration the intrapleural pressures are increased in relation to atmospheric pressure as the closed thoracic cavity decreases in size. This causes air to flow back out to the atmosphere.



Positive pressure ventilation does have its disadvantages. Positive pressure ventilation can rupture the lung causing a tension pneumothorax. Positive pressure ventilation with positive end expiratory pressure (PEEP) can increase the internal pressures on the pulmonary vasculature and reduce the venous return (preload) to the left side of the heart. A reduction of preload may benefit those in left ventricular failure or compromise those who are critically dependent on preload (i.e. acute right ventricular infarction).

For patients who require assistance breathing, a ventilator or bag valve mask (BVM) delivers air forcefully into the lungs. The air pressure created by the action of these devices is higher than the air pressure in the lungs. This type of ventilation is called positive-pressure ventilation (PPV) and it has many benefits in cardiovascular emergencies. For example, PPV can help expand collapsed alveoli or cause fluids to be forced out of the lungs and into the blood stream i.e. to treat pulmonary edema.

What Drives Breathing?

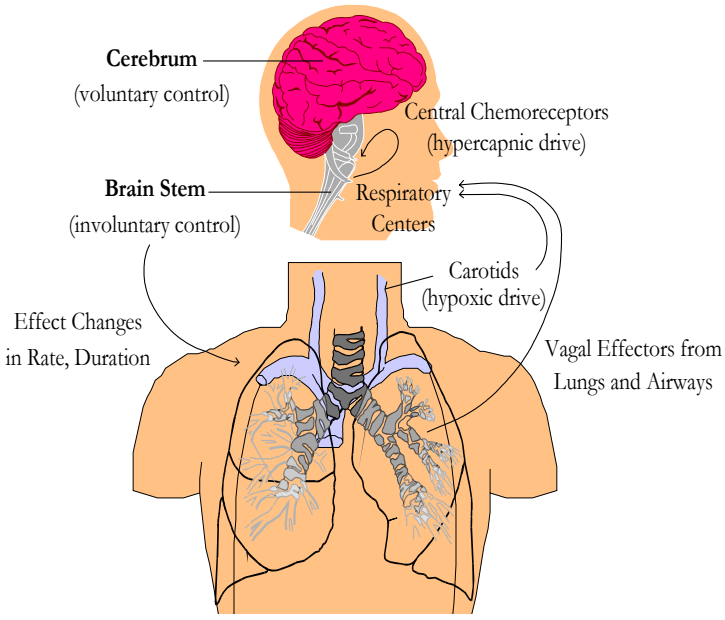
Spontaneous breathing is unique in that it can be both a conscious and an unconscious process. We can consciously speed, slow or stop breathing at will. Most often, though, breathing is an unconscious activity with rate and depth closely monitored by a host of sensors throughout the body. With varying energy demands and stimuli, the volume of air breathed over a minute (minute ventilation) fluctuates in kind.

Voluntary control of breathing is accomplished by the cerebrum while the involuntary or automatic control of breathing is provided by the brain stem. A host of chemoreceptors stimulate the brain stem to innervate the muscles responsible for ventilation i.e. diaphragm. These receptors influence the rate and duration of breaths (see Figure 8.8).

The drive to breathe is normally stimulated by falls in the blood pH (more acidotic) most often attributed to increased carbon dioxide levels (see the section on acid-base balance in chapter 9). Chemoreceptors in the brainstem react to hypercapnic (high CO_2) blood levels with increases in breathing rates to blow off excess CO_2. As a result, blood acid levels return to a target zone.

The hypercapnic drive to breathe is quite powerful. For example, minute ventilation volumes usually increases by 2-3 litres/minute for every 1 mm Hg increment in CO_2 levels in an attempt to normalize blood pH. Therefore, CO2 levels of 45 mm Hg (40 mm Hg is an accepted mean) can cause the minute ventilation to increase by up to 15 litres/minute, almost quadrupling minute ventilation volumes.

Figure 8.8 Regulation of Ventilation



Low blood oxygen levels can also drive breathing. Chemoreceptors in the carotid bodies respond to hypoxemia, sensing falls in blood oxygen levels (PaO_2) with the stimulation of the breathing centers in the mid-brain. They react most strongly to falls in PaO_2 less than 60 mm Hg, corresponding to oxygen saturations below 90% (normal PaO_2 is 80-100 mm Hg at sea level).

Because the hypercapnic chemoreceptors in the brain stem usually respond first, ventilations are generally driven by hypercapic chemoreceptors. Oxygen levels generally never fall below 60 mm Hg. As a result, the hypoxic drive is seldom called upon to stimulate breathing. For those with advanced lung disease, though, the ability to lower CO_2 levels decrease. Chronically high CO_2 levels may desensitize the hypercapnic chemoreceptors, with the hypoxia chemoreceptors called upon to stimulate breathing. For these people, the administration of high flow rates of oxygen can increase their PaO_2 to a level where the hypoxic drive falters. With both hypoxic and hypercapnic drives now inactive, respiratory failure is quite possible. **For those with advanced lung disease and chronically high CO_2 levels, the goal is often to improve their hypoxia without relieving it completely.** This might be achieved by delivering a sufficient supply of oxygen to maintain oxygen saturations near 90-92%.

The drive to breathe is also affected by stretch receptors (associated with the vagus nerve) in the smooth muscle and mucosa of the airways that respond to increases in lung volumes. As a result of the stimulation of these receptors, respiratory rate decreases. This is known as the Hering-Breuer reflex.

Some aspects of the regulation of ventilation remain a mystery. For example, minute ventilation volumes can expand to over twenty times normal during exercise even though arterial pH, PaO_2, and CO_2 would largely remain unchanged with only marginal increases in breathing rates. Physiologists point to possible respiratory receptors in muscles, joints, tendons and the diaphragm as possible triggers.

Factors That Influence The Work of Breathing

Ventilations are most efficient when optimal gas exchange is achieved with a minimal breathing workload. Disorders of the respiratory system can impair gas exchange and increase the work of breathing. Several factors affect the efficiency and the effectiveness of breathing whether breaths are self-delivered (negative pressure) or delivered by a breathing device (positive pressure). These factors include:

- **inspired volume** - the amount of air breathed during inspiration
- **airway resistance** to air flow
- **tissue resistance** - frictional resistance of lung tissues and chest wall
- **elastic recoil** - the ability of the airways to maintain their baseline shape rather than collapse on exhalation
- **compliance** - the distensibility of the lungs to accommodate adequate volumes of air
- **breathing rate** - increased minute ventilation volumes are achieved over time with an increased breathing rate

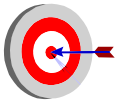
The work of breathing is a product of the work necessary to cause changes in lung volume and pressure. For each breath, it is the pressure changes within the lungs that cause increased lung volumes (inspiration). The primary work for each breath, then, is the work done to produce a pressure change between the lungs and the atmosphere.

In order to cause a pressure change in the lungs, effort (work) is necessary to overcome airway resistance, tissue resistance and elastic recoil. The work of breathing, then, is primarily the work necesary to overcome airway resistance, tissue resistance and elastic recoil. The work of several breaths is cumulative. As a general rule, then, the greater the lung volumes and the faster the breathing rate, the work of breathing increases.

While the work of quiet breathing consumes only about 5% of the body's oxygen content, difficult and rapid breathing can cost as much as 25-30% of the body's oxygen content. For a healthy adult during exercise, for example, an abundant physiological reserve satisfies this extra need (see page 152). For the cardiac patient with a low cardiac output and pneumonia, this is likely critical.

The Resistive Work of Breathing

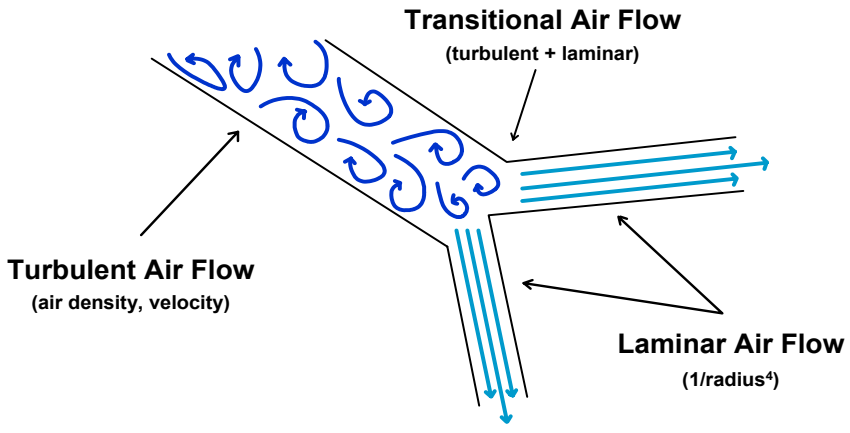
The resistive work of breathing - overcoming tissue resistance and airway resistance - adds significantly to breathing workload. Normally, airway resistance accounts for the majority of the resistive work of breathing. Tissue resistance only accounts for about 20% of the total resistance of breathing. Tissue resistance occurs due to the friction produced between lung tissues during lung inflation. Airway resistance is the friction between inspired air and the airway walls.



Airway resistance is significant in the upper airways. When breathing through the nose, the upper airways (nasopharynx, oropharynx and larynx) account for about 40% of the total airway resistance. When mouth breathing, the upper airway resistance is about 25% of the total airway resistance. For those who are experiencing an extended and difficult wean from mechanical ventilation (i.e. a ventilator), a common practice is the creation of a **tracheostomy**. Note that a tracheostomy bypasses the upper airways making breathing easier.

Three types of air flow occur, determined generally by the size of the airways. Air flows through the larger airways in a turbulent fashion while air flow through the narrow bronchioles is more laminar (where air in the center of the airway moves faster than the air at the walls). As airways branch off, transitional air flow results from a mix of laminar and turbulent air flow. The factors that determine the degree of airway resistance change with the type of air flow.

Figure 8.9 Turbulent and Laminar Air Flow



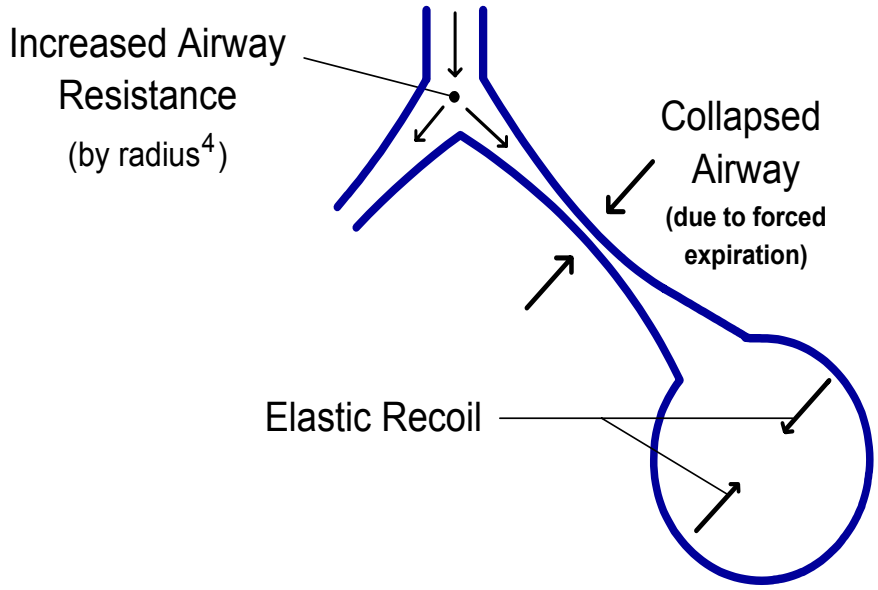
With turbulent air flow, airway resistance is proportional to inspired air density and air velocity. For example, denser air at sea level inspired at greater velocities (i.e. fast breathing rate) increases airway resistance in the larger airways where turbulent air flow occurs. Laminar airflow occurs in the smaller airways. Here airway resistance is a function of the airway length and the inverse of the airway radius to the fourth power. Airway resistance in smaller airways would then increase 16 times for a radius that is reduced by half. Conversely, an airway with a radius that doubles in size would have 16 times less (2^4) airway resistance.

Turbulent air flow resistance is more pronounced when the inspired air is more dense and moves with greater velocity. Turbulent air flow is noisy, providing the breath sounds audible with a stethoscope. Laminar airway resistance increases dramatically as the airway lumen narrows. Laminar airway resistance is calculated by Poiseuille's law:

$$\text{Airway Resistance} = (8 \times \text{viscosity} \times \text{length}) / \pi \times \text{radius}^4$$

The important point to take from this formula is that a reduced airway lumen has a dramatic affect on airway resistance (increases it to the 4th power). For example, if a smaller airway is narrowed by half its original radius by such events as infection, inflammation, or bronchospasm, the airway would be 16 times ($(1/0.5)^4$) more resistant to air flow.

Figure 8.10 Emphysema



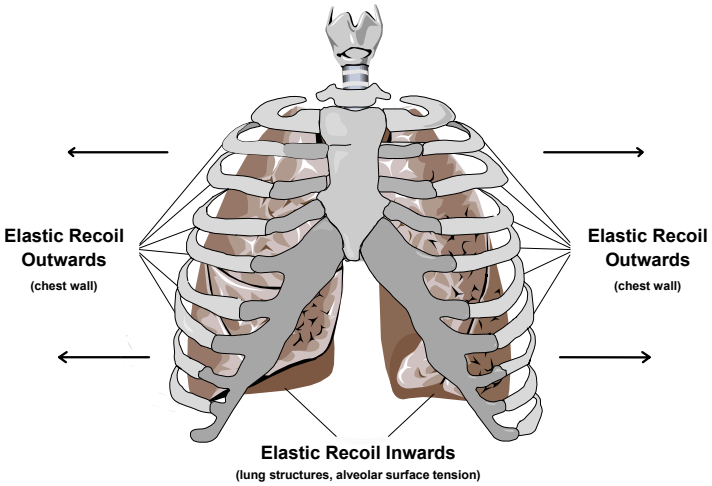
With emphysema, the smaller airways and the alveoli tend to collapse during forced expiration due to diminished elastic recoil. The work to re-expand these airways can be tremendous.

Medium-sized bronchi are the prime contributors to airway resistance. Parasympathetic stimulation, increased mucous production (infection, asthma), foreign body obstruction and inflammation can narrow the bronchi, increase airway resistance and increase the work of breathing. Conversely, sympathetic stimulation tends to dilate the bronchi. Localized hypoxia and hypercapnia dilates the smaller airways in an effort to increase ventilation, increase PO_2 and reduce CO_2.

The Elastic Work of Breathing

Since lung volume is directly related to pressure change (i.e. the difference between atmospheric and alveolar pressures drive volume changes), the work required for each breath is primarily the resistive and elastic work necessary to produce a pressure change. Besides air and tissue resistance, factors such as the elastic recoil of the lungs and the surface tension of the alveoli must also be overcome.

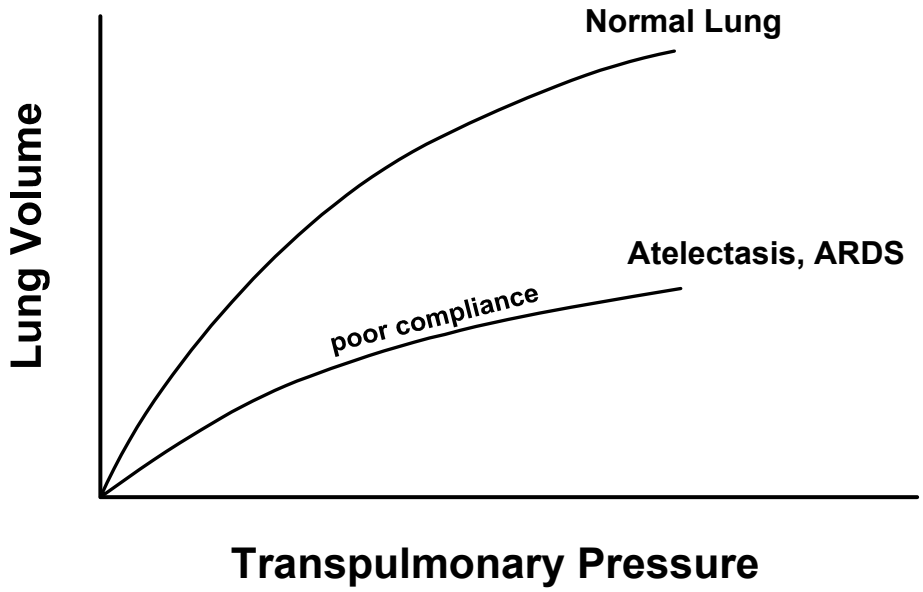
Figure 8.11 Elastic Recoil of the Lung and the Chest Wall



The elastic recoil of the lungs refers to the tendency of the alveoli and other lung structures to return to their original size. As elastic recoil increases, so does the work of breathing. Factors that contribute to elastic recoil include alveolar surface tension (see page 170) as well as elastin and collagen in lung tissue. Alveolar surface tension accounts for about 2/3 of the total elastic recoil of the lungs.

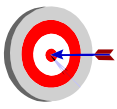
The inverse of elastic recoil is lung compliance. Lung compliance refers to the ease by which the lungs can increase in volume. A more compliant lung distends with less work of breathing. Technically, lung compliance is the slope between two points on a pressure-volume curve. The steeper the slope, the greater the lung volume changes per unit pressure change. This is a useful concept when trying to understand how either compliance or elastic recoil affects the work of breathing (see Figure 8.10).

Figure 8.12 Lung Compliance and Lung Disease



The steeper the slopes of the pressure-volume curves, the more compliant the lungs. Transpulmonary pressure is the difference between the air pressures in the trachea and the alveoli. When alveolar pressure falls below the tracheal pressure, air enters the lungs. With lung diseases that cause interstitial congestion in the lungs and fibrosis (i.e. ARDS), elastic recoil of the lung tissues increases, lung compliance decreases and the elastic work of breathing becomes more arduous.

As lung compliance increases, the elastic work of breathing is reduced. For many lung diseases, particularly restrictive lung disease such as pulmonary fibrosis, compliance decreases and breathing becomes more arduous. Other conditions that decrease lung compliance include acute respiratory distress syndrome (ARDS), pulmonary edema, and pneumonia. Note that each of these diseases produce an increase in elastic recoil. and the elasic work of breathing.



Normally, the highly compliant lungs expand easily. As a result, the work of breathing at rest uses only about 2% of the body's oxygen consumption. For a healthy person, rapid breathing does not increase the work of breathing significantly. For those with obstructive or restrictive lung disease, though, the work of breathing at rest and with exertion can tax the muscles involved. Respiratory muscle fatigue and failure are more likely to occur for those with lung disease.

Elastic recoil does more than just add to the breathing workload. Elastic recoil takes the work out of expiration and maintains airway patency during sudden periods of changing pressures. While the larger airways contain cartilagous rings to maintain patency, the smaller airways depend on a matrix of elastin and connective tissue to remain open.

Surfactant

The main contributor to the inward elastic recoil of the lungs is the surface tension within the alveoli. Water is generally more attracted to other water molecules than it is to air (this attraction is what keeps a water droplet together). Along the inside surface of an alveolus, this attraction of water for itself creates a **surface tension,** drawing the walls of the alveolus towards each other.

Surfactant is a chemical produced by the type II cells within the alveoli that effectively reduces surface tension within the alveoli. The presence of adequate amounts of surfactant, then, is necessary to minimize both the work of breathing and the fluid within the alveoli.

During periods of hypoxia and hypoxemia, the production of surfactant is curtailed. Possible complications of this lower surfactant production are increased surface tension, collapsed alveoli (atelectasis) and fluid accumulation in the alveoli. Pulmonary edema is but one disease that can cause low surfactant production.

If the work of breathing is significant, respiratory fatigue can lead to precipitous drops in both breathing rates and inspired volumes - both signs of respiratory failure. Note that signs of respiratory failure such as an altered level of consciousness, paradoxical breathing and elevated CO_2 levels may coincide with an apparently normal breathing rate and oxygen saturation. Being aware of the factors that influence breathing workload is useful for predicting and treating respiratory fatigue

Being aware of the factors that affect the workload of breathing influence respiratory management. For example, a hypoxemic episode during an asthma attack often responds well to agents that open the airway lumens, reduce airway resistance, decrease the work of breathing and increase air flow to the alveoli. Supplemental oxygen may help increase oxygen supply to the tissues. A reduction in hypoxia can also help ensure adequate surfactant production.

It's time to review the mechanics of breathing and the factors that affect the work of breathing with a brief quiz before we move on to the intricacies of gas exchange and gas transport.

Flash Quiz 8.2

1. The muscles involved in normal inspiration usually include (circle all that apply):

a) sternocleidomastoid
b) internal intercostals
c) external intercostals
d) abdominal
e) diaphragm

2. Compliance is the inverse of elastic recoil.

True or False

3. Which of the following conditions result in an increased breathing workload (circle all that apply):

a) decreased lung compliance
b) decreased elastic recoil
c) narrowed airways
d) pleural effusion
e) kyphoscoliosis (curvature and rotation of spine)
f) restrictive lung disease
g) obstructive lung disease
h) obesity
i) rapid breathing rates

4. The alveoli open during inspiration due to the muscle tissue that surrounds each alveoli.

True or False

5. The main muscle used for breathing is the (diaphragm, sternocleidomastoid) which is innervated by the (vagal verve, phrenic nerve) which leaves the spine at the level of the level of the (lumbar vertebrae, #3-5 cervical vertebrae, 6-9 thoracic vertebrae).

6. Which of the following statements about the mechanics of breathing is false?

a) inspiration is an active process
b) exhalation is a passive process at rest and during physical exertion
c) accessory breathing muscles are used to increase inspiratory volumes
d) negative pressure ventilations are spontaneously performed at rest

Answers: ***1.*** *c), e)* ***2.*** *True* ***3.*** *all but b)* ***4.*** *False* ***5.*** *diaphragm, phrenic nerve, #3-5 cervical vertebrae* ***6.*** *b)*

7. In a closed system, as volume increases, pressure increases.

True or False

8. The chemoreceptors that stimulate breathing as a response to hypoxia are located:

a) along the inner surface of the airways in the lungs
b) centrally near the respiratory center in the midbrain
c) in the carotid bodies
d) all of the above

9. For those without advanced lung disease, breathing is driven primarily by chemoreceptors that monitor blood acidity (and carbon dioxide levels).

True or False

10. Respiration is accomplished with the physiological goal to provide a sufficient lung volume with minimal breathing workload. As (compliance, air resistance) increases, so does breathing workload. In conditions such as emphysema, an (increased, decreased) elastic recoil can result in collapsed airways, particularly during (inspiration, expiration).

Gas Exchange and Transport

Gas exchange is vital for aerobic metabolism and energy production. Without oxygen, energy production is terribly less effective. No energy - no life. Effective oxygenation occurs if three mechanisms occur: 1) oxygenation of the blood at the alveoli; 2) the transport of oxygen to the tissues; and 3) the release of oxygen at the cellular level. A breakdown in any these tasks can result in hypoxia, a lack of oxygen at the cellular level (where it really counts).

A term used perhaps as often as hypoxia is ischemia. Ischemia is a lack of oxygen to the tissues due to inadequate flow of blood (oxygen transport system). The end result, though, is similar to hypoxia - a lack of oxygen (and therefore energy) at the cellular level. Both hypoxia and ischemia can quickly cause cellular damage and, soon after, cellular death. Hypoxemia is a low arterial partial pressure of oxygen (i.e. low PO_2 at high altitudes is one example). Hypoxemia can cause hypoxia.

While discussions of respiratory physiology may tend to be biased towards the role of oxygen, the transport and exchange of carbon dioxide are also important factors. Carbon dioxide levels affect oxygen transport, oxygen affinity and acid-base balance. Through O_2 and CO_2 transport and exchange, respiration strives to maintain homeostasis and drive energy production.

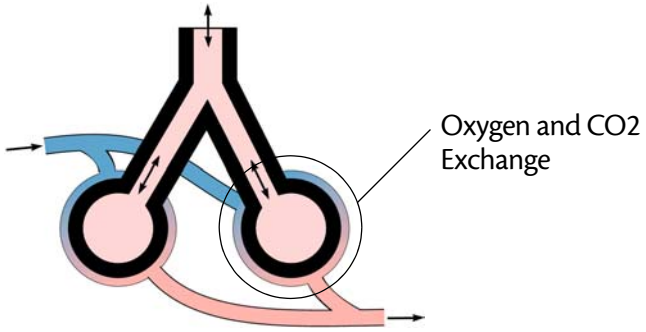
Answers: ***7.*** *True* ***8.*** *c)* ***9.*** *True* ***10.*** *air resistance, decreased, expiration*

The structures and the mechanics of the respiratory system, as just outlined, are fine tuned to minimize the workload of breathing while maximizing gas exchange. Delving deeper into respiratory physiology reveals many more evolutionary wonders. The rationale behind many clinical respiratory and cardiac interventions rests in the intricacies of gas exchange and transport.

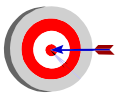
Alveolar Gas Exchange

Let's begin at the top. Air enters the trachea rapidly during inspiration. As the air passes the bronchi on to the bronchioles about 23 successive divisions exponentially increase the overall cross-section that the air occupies. As a result, air speed begins to slow as the air reaches the deeper bronchioles. At the alveoli, air velocity approaches zero. Here, gas moves primarily due to diffusion alone.

Figure 8.13 The Alveoli and Oxygen Exchange



A respiratory bronchiole, the alveolar duct and the alveoli form the terminal respiratory unit of the lungs. It is here that gas exchange occurs. About 80% of the surface area of the alveoli is in contact with pulmonary capillaries. The remaining 20% is commonly called physiologic dead space (PDS). Along areas not perfused by capillaries, gas exchange cannot occur.

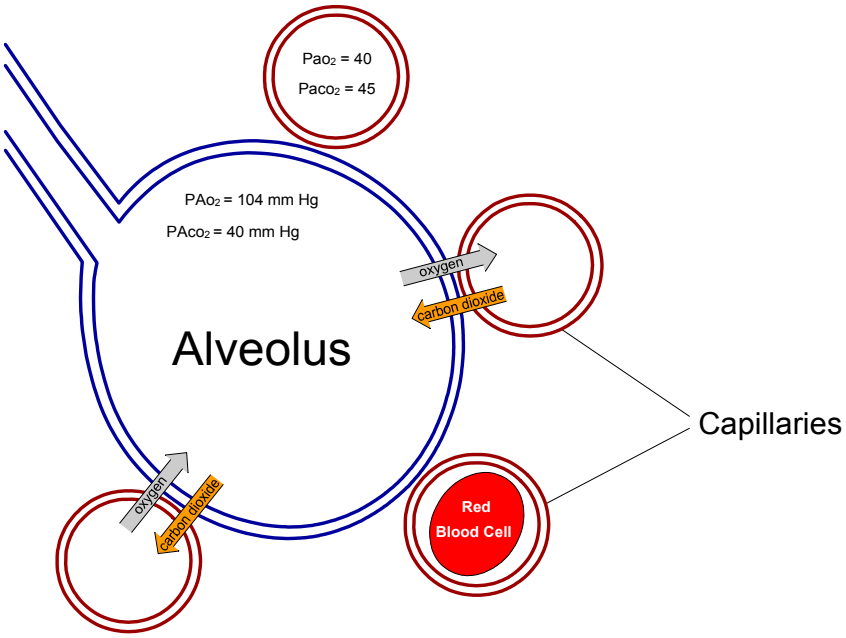


The total surface of the 300 million **alveoli** is an impressive 80 m^2 - about 40 times the surface area of the skin. The lungs have significantly more contact with the atmosphere than does the skin (or any other organ for that matter). As a result, the alveoli provide ample surface area for gas exchange.

The ventilation/perfusion ratio (V/Q) reflects the adequacy of inhaled volume (V) as measured against the amount of blood perfused (Q). Since we breathe about 4 litres of air at rest (after anatomical dead space is subtracted) and circulate about 5 litres of

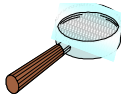
blood/minute, a normal V/Q ratio then would be about 4/5 or 0.8 or 80%. As ventilation increases and lung perfusion decreases (i.e. shock states or pulmonary embolus), the V/Q ratio increases.

Figure 8.14 Gas Exchange at the Alveolus



The capillaries carry blood that is low in oxygen (PaO_2=40 mm Hg) and high in carbon dioxide ($PaCO_2$=45 mm Hg). With a higher partial pressure for oxygen (104 mm Hg) in the alveoli, oxygen tends to diffuse into the capillaries and the red blood cells. Carbon dioxide diffuses into the alveoli. Red blood cells must squeeze through the capillaries. This provides ample surface area for the oxygen to diffuse through and bond to Hgb for transport.

Gas exchange at the alveoli occurs due to diffusion alone. Only a 2 cell thickness separates the air within the alveoli from the blood in the capillaries. The red blood cells, the primary transport units of both oxygen and carbon dioxide, must squeeze through the tiny capillaries. The resulting close proximity of the red blood cell wall to the alveolus helps to facilitate rapid gas exchange.

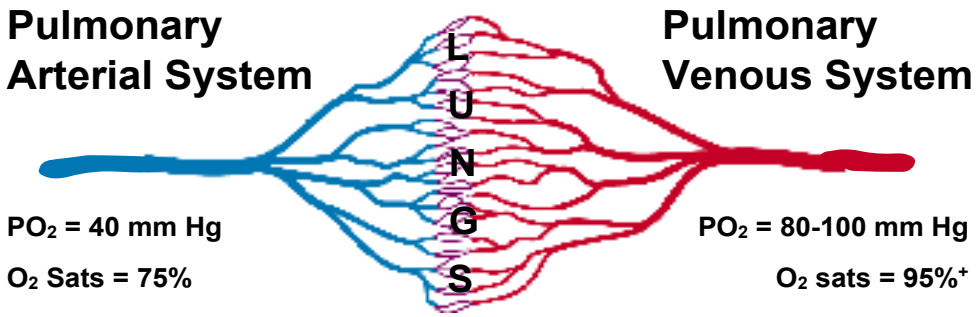


Low ventilation with normal or high perfusion yields a low V/Q ratio. For disease processes such as pneumonia and tension pneumothorax, collapsed airways are not ventilated, perfusion is maintained and gas exchange is hampered. As a result, some blood shunts to the left atrium without the benefit of gas exchange. If the shunt becomes significant (i.e. large part of the lung is affected), arterial oxygen saturation falls.

With only a 2 cell separation between the inside of the alveoli and the blood, it is not surprising that increased pressure within the capillaries (hydrostatic pressure due to back up of blood volume into the lungs i.e. left ventricular failure) can lead to fluid accumulation in the lungs.

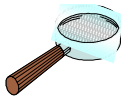
Gases such as oxygen and carbon dioxide freely cross between the blood and the alveoli. Gas diffusion is dependant on a difference in partial pressures of gases between the alveoli and the pulmonary capillaries. Gases quickly diffuse from high pressure zones to low pressure zones. As a result, oxygen enters into the capillaries and carbon dioxide exits through the alveoli.

Figure 8.15 Normal Arterial and Venous Oxygen Values



The pulmonary arterial system brings blood to the lungs that is oxygen-depleted and carbon dioxide rich. After gas exchange occurs at the lungs, the pulmonary venous system delivers oxygenated blood to the left atrium.

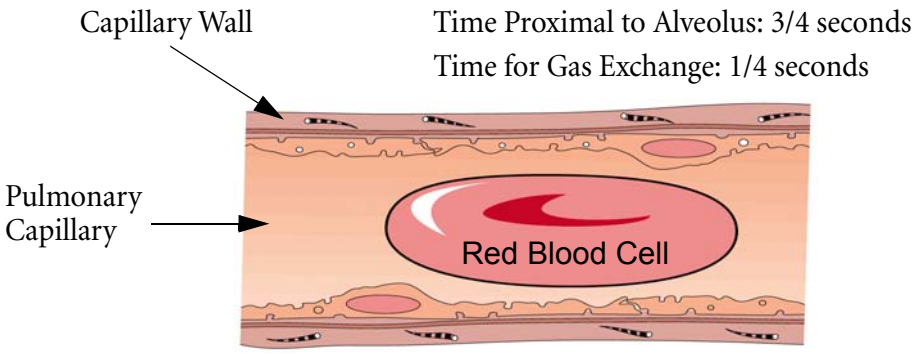
Diseases such as asthma and emphysema restrict the flow of air into and out of the lungs, impairing gas exchange at the alveoli. Pneumonia causes gas exchange impairment along regions of the pulmonary capillary bed leading to shunting, elevated blood CO_2 levels and falling blood oxygen levels.



Dalton's law of partial pressure states that the total pressure of a volume of gas equals the sum of the partial pressures of each gas that constitute the gas mixture. At sea level, the pressure of air (atmospheric pressure) is 760 mm Hg. Since roughly 21% of air is oxygen, the partial pressure of oxygen is equal to 21% of 760 mm Hg (answer is 158 mm Hg). If a person is breathing 50% oxygen via a face mask, then the partial pressure of oxygen inspired is equal to 50% of 760 mm Hg (answer is 360 mm Hg).

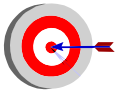
Normally this gas exchange (diffusion) occurs in 1/4 second. Because the red blood cell generally takes about 3/4 of a second to flow (or squeeze) by the alveoli, the red blood cell has more than sufficient time to complete the gas exchange. In high output states (i.e. extreme exercise), the increased speed of blood flow can reduce the opportunity for gas exchange if red blood cells are across the alveolar membrane for a period less than 1/4 second.

Figure 8.16 The Red Blood Cell and the Pulmonary Capillaries



The red blood cell (about 8 microns in diameter) must re-shape and squeeze through the narrow capillaries (about 6 microns in diameter). Gas exchange occurs in just 1/4 second. The red blood cell (RBC) is proximate to alveoli for approximately 3/4 second during normal conditions. During periods of high cardiac output (i.e. extreme exercise), gas exchange may be incomplete as the faster moving RBC may be proximate to alveoli for less than 1/4 second.

Other causes of failure to oxygenate the blood include airway obstruction, respiratory failure, carbon monoxide poisoning and low oxygen gradients (i.e. high altitude). Respiratory failure is usually diagnosed with the partial pressure of oxygen in the arterial system (Pao_2) falling below 50 mm Hg or the partial pressure of carbon dioxide ($Paco_2$) being over 50 mm of Hg.



While most instances of poor oxygenation produces cyanosis, **carbon monoxide (CO) poisoning** is the exception. Hemoglobin's affinity for carbon monoxide is about 240 times that of oxygen. Carbon monoxide quickly binds to hemoglobin at the sites usually reserved for oxygen. This causes the hemoglobin to reflect a bright red hue typical of full oxygen saturation. While CO poisoning is indeed responsible for severe oxygen depletion, the patient presents without cyanosis (white skinned people become a cherry red). Treatment includes the provision of high flow oxygen.

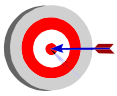
As mentioned, respiratory failure is usually associated with both low oxygen levels and high CO_2 levels. The high CO_2 levels are associated with increased carbonic acid in the blood. The lower blood pH levels associated with high CO2 concentrations is called

respiratory acidosis. Virtually every extended cardiac arrest causes respiratory acidosis (CO_2 is not being breathed off effectively with cardiac output from CPR only about 25% that of normal output).

Low oxygen values often lead to a shift towards anaerobic energy metabolism with by-products such as lactic and pyruvic acids. This increased acidity (lower pH) in the blood is called metabolic acidosis. Similarly, most extended cardiac arrests result in metabolic acidosis. A return of spontaneous circulation and breathing most often self-corrects both the respiratory and metabolic acidosis (see the section on blood gas analysis later in this chapter).

Oxygen Transport

Hemoglobin carries about 97% of the body's oxygen to the tissues. Only about 3% of oxygen makes it's way to the tissues as dissolved oxygen. In order for the oxygen to be picked up and delivered to the tissues, sufficient quantities of hemoglobin must be available.



Oxygen saturation measures the amount of oxygen bound to the hemoglobin. A hemoglobin that is 100% saturated is carrying a full load of oxygen. Hemoglobin that are carrying only half the maximum number of oxygen would have an oxygen saturation of 50%. Note that an oxygen saturation of 75% is typical of venous blood returning to the heart. Each hgb binds with as many as 4 oxygen molecules. So with an O_2 saturation of 75%, on average only 1 oxygen molecule has been released per hgb leaving most of the oxygen in reserve.

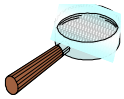
The normal range of hemoglobin is 120-160 grams of hemoglobin per litre of blood. Each gram can bind with as much as 1.34 ml of oxygen (at 100% saturation). The amount of oxygen carried by a litre of blood can be calculated as follows:

hemoglobin x 1.34 x O_2 saturation = oxygen carried / litre of blood

The central parameters that influence blood oxygen content are hemoglobin and oxygen saturation levels. The next three scenarios explore how these parameters as well as cardiac output can affect the supply of oxygen to the tissues.

Scenario 1: The best case scenario might be a hemoglobin of 150 g and 100% oxygen saturation. A calculator quickly informs us that 1 litre of blood with a hemoglobin of 150 g/litre and a saturation of 100% carries about (150 x 1.34 x 1.0) 200 ml of oxygen. With an average cardiac output of 5 litres/minute, about 1 litre (5 x 200 ml) of oxygen is delivered to the body every minute.

Scenario 2: But what if the hemoglobin (hgb) dropped to 100 g/litre, a level that is often only monitored, seldom treated. With an oxygen saturation of 100% the oxygen transported per litre would be (100 x 1.34 x 1.0) about 134 ml. A drop in the hemoglobin by a third correlated to an identical third less oxygen transported to the tissues! And don't figure on dissolved oxygen coming to the rescue. Dissolved oxygen would account for only 20 ml delivered per minute.



Oxygen saturation measurement can be invasive (i.e. blood gas measurement) or non-invasive (i.e. pulse oximetry). Also called pulse oximetry, a probe often placed on a finger shines infrared (and sometimes ultraviolet) light into the finger. The absorption of the light in the capillaries depends on the saturation of the hgb. **Pulse oximetry correlates light absorption with blood pulsations.** Therefore, a reliable reading is provided only if the oximeter can determine a pulse. Poorly perfused fingers (cold, pale or blue) tend to provide inaccurate readings. **During profound shock states and cardiac arrest, a pulse oximeter is much less reliable.**

Scenario 3: An elderly patient with a cardiac history, a borderline cardiac output status of 3800 ml/minute at rest, hgb of 100 g/litre converts from sinus rhythm to atrial fibrillation. Cardiac output could be cut by as much as 35%. From the earlier example, a hgb of 100 g/litre provides 134 ml of oxygen per litre. But with poor cardiac output, less blood and therefore less oxygen is transported. In this case, only (3.8 litres/min x 134) 509 ml of oxygen is available to the tissues each minute.

While the last example is a gross generalization of what could occur (i.e. perhaps loss of atrial kick only results in 20% less cardiac output), it does illustrate how oxygen delivery is impacted by three main factors:

1. oxygen saturation (number of oxygen bound to each hemoglobin)
2. hemoglobin levels
3. cardiac output

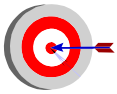
Compare a healthy blood oxygen volume of (150 g hgb/litre x 1.34 x 100% oxygen saturation x resting average cardiac output of 5 litres) of 1005 ml in the first scenario with roughly half of the blood oxygen volume (509 ml) in the third scenario. Note that in both scenarios the oxygen saturation was 100%! The oxygen volume delivered to the body is dependent on all three of the factors listed above. A fall in any one or more of these values will negatively impact oxygen delivery.

Tissue Oxygenation

The microcirculation system of arterioles and capillaries finish the job that the lungs and heart began, bringing oxygen to the cells. Oxygen is converted from a deliverable to a vital consumable at the cellular level. The mitochondria within the cells make use

of oxygen and glucose to produce adenosine triphosphate (ATP), a molecule that is used in any activity that requires energy. As mentioned several times already, ATP provides the energy necessary for life.

Because the supply of oxygen and the production of ATP is of primary importance, it should come as no surprise that several mechanisms ensure that oxygen supply more than satisfies oxygen (and energy) demand. Respiratory rate, tidal volumes, heart rate and stroke volumes all increase to bring more oxygen to the tissues. At the cellular level several other triggers can cause the hemoglobin to release more oxygen.

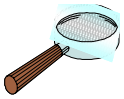


For healthy individuals at rest, the oxygen supplied is far greater than the oxygen required. On average, adults require about 250 ml of oxygen each minute to satisfy metabolic demands (energy demands). In the previous section, we determined that oxygen content for a healthy adult is about 200 ml per litre. With an average cardiac output of 5 litres/minute, the body receives about 1000 ml of blood each minute - about 3 times more oxygen than necessary.

The amount of oxygen released to the cells fluctuates with increased energy demand. As aerobic metabolism increases as a response to high demand states (i.e. exercise or fever), CO_2 - the bi-product of aerobic metabolism - also increases. In response to high energy demand states, the chemical 2,3 - diphosphoglycerate (2,3-DPG) also increases. Not surprisingly, hemoglobin releases more oxygen to the cells in the presence of elevated amounts of 2,3-DPG, temperature, and CO_2 (which causes increased acidity and lower pH). Conversely, low levels of any of these factors results in hemoglobin having a higher affinity for oxygen (less oxygen is released).

This all seems rather academic, you might say. How can you apply this information? Oxygen saturation, a topic we will explore further in the next chapter, measures the percentage of hemoglobin binding sites (four sites/hemoglobin) that are bound to oxygen. When hemoglobin's affinity for oxygen decreases, the Pao_2 is higher for every oxygen saturation reading i.e. more oxygen is dissociated from the hemoglobin at any oxygen saturation level.

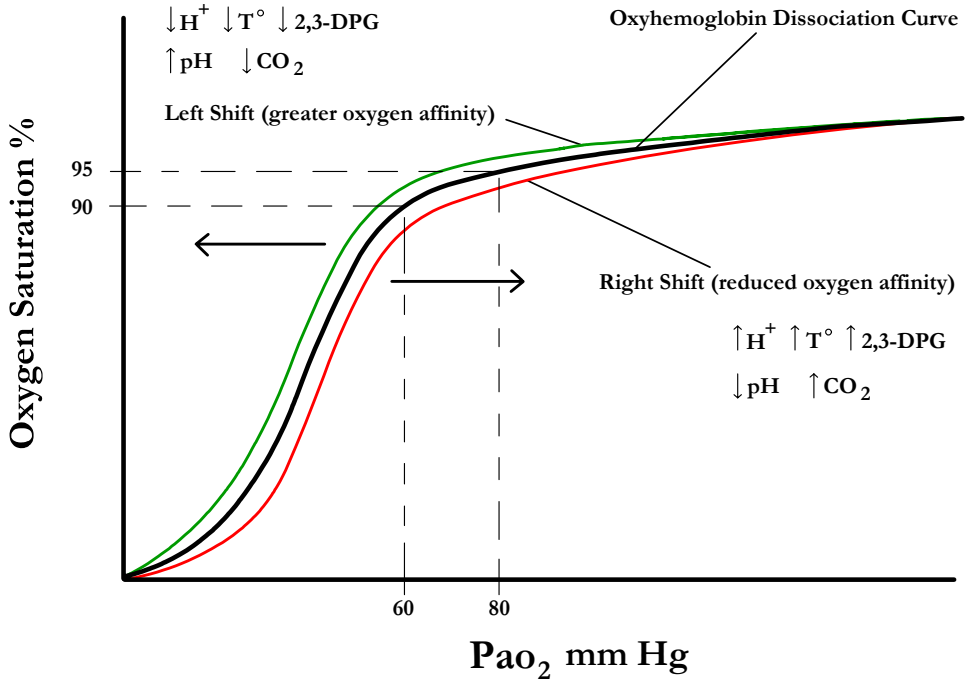
The good news is that hemoglobin most often has a whole lot more oxygen to share than is provided during normal resting states. Venous blood has an oxygen saturation of about 75% (3 out of every 4 oxygen binding sites are occupied by oxygen). During episodes of low oxygen content (i.e. anemia, respiratory failure, pneumonia, pulmonary edema) or low oxygen delivery (low cardiac output states such as extreme tachycardias or bradycardias and acute heart failure) the body continues to respond by making more oxygen available to the tissues.



During cardiac arrests, ensuing respiratory and metabolic acidosis (high CO_2 and low pH) may initially cause the hemoglobin to release more oxygen reserve at the cellular level. Many scientists believe that many victims of sudden cardiac arrests (caused by dysrhythmias) have sufficient stores of oxygen and ATP to help preserve cell function for at least 4-6 minutes. One caveat: effective cardiac compressions are necessary to maintain some degree of circulation.

A standard oxyhemoglobin dissociation curve (OHDC) reflects the relationship between hemoglobin oxygen saturation and the partial pressure of arterial oxygen (Pao_2). Since oxygen equalizes across the alveolar-capillary membrane quite quickly, the partial pressure of arterial oxygen (Pao_2) comes close to equalizing with the partial pressure of alveolar oxygen (PAo_2). The ODHC is a graphical relationship between Pao_2 and the amount of oxygen carried to the cells (hemoglobin's oxygen saturation - see Figure 8.14).

Figure 8.17 Oxyhemoglobin Dissociation Curve



The oxyhemoglobin dissociation curve (OHDC) reflects the amount of oxygen released by the hemoglobin as a function of the arterial oxygen pressures. Certain conditions shift this curve to the left (hgb holds on to more oxygen) or to the right (hgb releases more oxygen). Shifts in the OHDC left, for example, result in less oxygen released to the tissues for any given PaO_2. If you follow the broken line up from a PaO_2 of 60, oxygen saturations with a shift left would increase from 90% to perhaps 93%. With higher hgb saturation, more oxygen is bound - less oxygen is released.

The distinct S shape of the OHDC and the tendency of the OHDC to shift left or right during certain conditions are important points worth exploring.

The S shaped curve reveals important phenomena. Only a 5% increase in oxygen saturations (from 90% to 95%) occurs with a 33% increase in Pao_2 (from 60 to 80 mm Hg). After a Pao_2 of 60 mm Hg, increasing inspired oxygen comes with diminishing returns as the curve plateaus. Breathing high flow oxygen to increase oxygen saturations to more than 95% will not result in substantial increases in blood oxygen volume (see section on oxygen transport).

The steep slope of the oxyhemoglobin curve reveals precipitous drops in oxygen saturations with Pao_2 below 60 mm Hg (or oxygen saturations below 90%). From a clinical perspective, most practitioners consider an oxygen saturation below 90% a significant finding requiring prompt attention. The steep slope of the oxyhemoglobin curve supports this concern. Without a rapid response, oxygen saturations could quickly plummet to levels incompatible with life.

Oxygen saturations for each Pao_2 level are not always linear, following the standard curve. The OHDC can shift to the right or left depending on certain conditions. Increased blood acidity, elevated Pco_2, and fever decrease hemoglobin's affinity for oxygen with more oxygen released. The OHC shifts right.

Hemoglobin also tends to release more oxygen with increases in the presence of 2,3 - diphosphoglycerate (2,3-DPG). High levels of 2,3-DPG is associated with chronic obstructive pulmonary disease (COPD), anemia, hypoxemia and congestive heart failure. Each of these can negatively affect blood oxygen volumes. An enhanced production of 2,3-DPG apparently serves as an adaptive mechanism, causing the hgb to release more oxygen to the cells.

With a shift of the oxyhemoglobin curve to the right, the hemoglobin is less saturated at any corresponding Pao_2 level. Conversely, increased affinity for oxygen is reflected in a shift to the left for the oxyhemoglobin curve. Here, hemoglobin is more saturated with oxygen for every Pao_2 level resulting in less oxygen availability at the tissues. This left shift occurs with alkalosis, low Co_2 levels, depleted 2,3-DPG (i.e. blood transfusions) and hypothermia.

Tissue oxygenation rarely suffers from just shifts in the OHDC to the left. More commonly, tissue oxygenation falls due to abnormally narrow blood vessels and ruptured plaques that acutely occlude vessel lumens. An increase in oxygen demand beyond the limits of oxygen supply for these obstructed vessels can result in ischemia and infarction. Other conditions that impair tissue oxygenation include shock states, carbon monoxide poisoning, myoglobinemia and sickle cell anemia.

Flash Quiz 8.3

1. Effective oxygenation requires:

a) gas exchange at the alveoli
b) transport of oxygen to the cells
c) gas exchange at the cellular level
d) all of the above

2. The term that denotes a low partial pressure of oxygen in the blood is called hypoxemia.

True or False

3. Gas exchange at the alveoli occurs by:

a) osmosis
b) diffusion
c) filtration
d) all of the above

4. The main parameters that affect the supply of oxygen to the tissues are:

a) oxygen saturations, cardiac output and fever
b) cardiac output, age and respiratory rate
c) hemoglobin, respiratory rate and cardiac output
d) hemoglobin, oxygen saturations and cardiac output

5. An oxygen saturation of 95% or more guarantees sufficient blood oxygen content.

True or False

6. A person who suffers an extended cardiac arrest usually develops (respiratory acidosis, metabolic acidosis, both respiratory and metabolic acidosis). Upon a return to spontaneous circulation, acidosis is usually corrected within (minutes, days) with adequate (ventilation, sedation, buffer administration).

7. A person with carbon monoxide poisoning:

a) may be suffocating with oxygen saturation values of 98-100%
b) has cyanosis and shortness of breath
c) can be cherry red in colour
d) a and c

Answers: ***1.*** *d)* ***2.*** *True* ***3.*** *b)* ***4.*** *d)* ***5.*** *False* ***6.*** *both respiratory and metabolic acidosis, minutes, ventilation* ***7.*** *d)*

8. Hemoglobin carries about (3%, 60%, 97%) of the oxygen supplied to the body. A shift (right, left) in the oxyhemoglobin dissociation curve occurs when the hemoglobin's affinity for oxygen decreases (more oxygen is released).

9. The oxyhemoglobin dissociation curve follows an S curve. The steep slope of the curve signifies the precipitous drop in oxygen content for oxygen saturations below 90%.

True or False

10. Hemoglobin releases less oxygen with the following conditions (circle all that are correct):

a) alkalosis
b) low CO_2 levels
c) high 2,3 DPG levels
d) hypothermia

Summary

The structure and function of the respiratory system was explored in this chapter. The focus may have sided with how oxygen makes its way to the cells but the effects of carbon dioxide are also worth considering. Further discussion on the impact of carbon dioxide is provided in the next chapter.

The anatomy of the upper airways efficiently warm, filter and moisturize incoming air. The upper airways account for about 40% of the total airway resistance. Structures such as the larynx and the cricothyroid cartilage are particularly important during respiratory procedures such as endotracheal intubation.

From the trachea, 23 successive branches through each lung takes air flow to the terminal respiratory unit, a respiratory bronchiole and an alveolus. The branches above this terminus facilitate air movement but do not engage in gas exchange with the blood stream. Approximately 300 million alveoli provide ample surface area (about 80 m^2) to enable gas exchange with 280 billion pulmonary capillaries. Most of the alveoli (80%) are completely enveloped by pulmonary capillaries.

Oxygen diffuses from the alveoli through to the pulmonary capillaries to increase the PO_2 from 40 mm Hg to 80-106 mm Hg. Carbon dioxide exits the pulmonary capillaries to be breathed out into the atmosphere. Oxygen quickly binds to

***Answers:* 8.** *97%, right* **9.** *True* **10.** *a,b,d*

hemoglobin within red blood cells to be carried to the cells of the body. Only about 3% of the blood oxygen content is freely dissociated. Oxygen content is primarily a function of hemoglobin levels and oxygen saturation of hemoglobin.

At the cellular level, oxygen is released to migrate freely to the mitochondria where aerobic metabolism occurs. The amount of oxygen released by the hemoglobin is directly related to blood acidity, increased temperature and levels of CO_2 or 2,3-DPG. Hemoglobin's reduced oxygen affinity is represented by a shift of the oxyhemoglobin dissociation curve to the right.

Normally, the work of breathing requires only about 2% of cardiac output. Breathing becomes more arduous with increases in airway resistance and reduced lung compliance. Reduced elastic recoil, such as occurs to the alveolar walls from emphysema, leads to airway collapse with rapid or forced expiration. The effort to re-expand collapsed airways increases the work of inspiration.

Chapter Quiz

1. We breathe to create energy.

   True or False

2. The lungs accomplish more than gas exchange. The lungs also:

   a) filter the blood of emboli
   b) have an endocrine function,
   c) clear toxins from the body
   d) all of the above

3. Anaerobic metabolism (with, without) oxygen yields (2, 10, 20, 36) ATP.

4. The many functions of the upper airways include (circle all that are correct):

   a) filter air of particulates
   b) humidify air
   c) temperature regulation of air (i.e. warm air)
   d) allow the ingestion of food and fluids
   e) aid digestion
   f) verbal communication
   g) safety and security via the sense of taste and smell
   h) prevent aspiration during eating and during submersion

Answers: ***1.*** *True* ***2.*** *d)* ***3.*** *without, 2* ***4.*** *a-h*

5. The role of surfactant is to (increase, decrease) surface tension. This in turn (increases, decreases) elastic recoil of the alveoli.

6. A 68 year old man with end-stage chronic obstructive pulmonary disease (COPD) is experiencing shortness of breath and cardiac ischemia. He is admitted to the ER with an oxygen saturation of 84%. The goal of oxygen therapy for this patient is to deliver sufficient oxygen to maintain their oxygen saturation levels at:

a) greater than 95% to aid both the shortness of breath and the cardiac ischemia
b) 90-92% to help both shortness of breath and cardiac ischemia while preventing respiratory depression
c) 86-89% to help both shortness of breath and cardiac ischemia while preventing respiratory depression
d) do not provide supplemental oxygen because of the risk of reducing respiratory drive that can occur with higher oxygen levels

7. Positive pressure ventilation can (increase, decrease) preload to the left side of the heart.

8. Upper airway resistance accounts for about 25-40% of total airway resistance.

True or False

9. If the radius of the bronchioles is reduced by 1/2, bronchiolar airway resistance increases by:

a) 50%
b)200%
c) 400%
d)1600%

10. For those with emphysema, reduced elastic recoil together with forced expiration can result in collapsed airways and significantly increased breathing workload.

True or False

11. The drive to breathe is normally linked primarily to blood (O_2, CO_2) levels. Increases in (O_2, CO_2) of only 1 mm Hg above a normal mean of (40, 80) mm Hg tends to increase minute ventilatory volumes by 2-3 litres.

12. The terms respiration and ventilation are interchangeable.

True or False

Answers: ***5.*** *decrease, increases* ***6.*** *b)* ***7.*** *decrease* ***8.*** *True* ***9.*** *d)* ***10.*** *True* ***11.*** *CO_2, CO_2, 40* ***12.*** *False*

13. The Hering-Breuer reflex is:.

a) a response to increased lung volumes with a reduced respiratory rate
b) also known as the 'diving reflex' associated with the closure of the epiglottis during submersion
c) the slowing of heart rate that can occur with the immersion of one's face in ice cold water
d) triggered by receptors in the tendons and joints producing an increased breathing rate beyond what is physiologically necessary

14. The work of breathing becomes more arduous with increased:

a) inspired volume
b) airway and tissue resistance
c) elastic recoil and lung compliance
d) a and b
e) all of the above

15. Effective tissue oxygenation depends on sufficient:

a) cardiac output
b) oxygen saturation
c) hemoglobin levels
d) all of the above

16. Does an arterial blood gas value for partial pressure of oxygen (Pao_2) provide a good indication of arterial oxygen content?

Yes or No

17. Which of the following conditions will dramatically impair oxygen transport?

a) slow breathing rates
b) low cardiac output states
c) anemia
d) b and c only
e) all of the above

18. At the alveolar level, air velocity is virtually static with the process of diffusion the primary mobility agent.

True or False

Answers: 13. *a)* ***14.*** *d)* ***15.*** *d)* ***16.*** *No* ***17.*** *d)* ***18.*** *True*

19. About 20% of the alveolar surface (as a whole) is not in contact with pulmonary capillaries. This non-perfused area is known as:

a) shunting
b) anatomical dead space
c) physiological dead space
d) there is no term for this phenomenon

20. Each day we breathe volumes equivalent to that of a swimming pool and transfer gases across a barrier the size of a tennis court.

True or False

21. A V/Q ratio evaluates:

a) blood oxygen content
b) minute ventilation volume
c) efficiency of ventilation as a function of $PaCO_2$ levels
d) adequacy of ventilation as a function of pulmonary perfusion

22. A V/Q ratio increases with which of the following conditions? (Circle all that apply)

a) emphysema
b) pneumonia
c) atelectasis
d) low cardiac output states
e) low minute ventilation volume
f) pulmonary embolus
g) tension pneumothorax

23. Red blood cells change their shape to fit through the pulmonary capillaries.

True or False

24. What blood values are typical of the pulmonary arteries for a healthy person at rest? (Circle all that apply)

a) oxygen saturations of 75%
b) oxygen saturations of 98%
c) PO_2 of 40 mm Hg
d) PO_2 of 80 mm Hg
d) PCO_2 of 80 mm Hg
e) PCO_2 of 45 mm Hg

Answers: ***19.*** *c)* ***20.*** *True* ***21.*** *d)* ***22.*** *f* ***23.*** *True* ***24.*** *a), c), e)*

25. With an atmospheric pressure of 760 mm Hg at sea level, the inspired partial pressure of oxygen (PO_2) of room air is approximately (304, 180, 160, 137, 80) mm Hg. Various face masks that provide approximately 40% inspired oxygen result in an inspired PO_2 of (304, 180, 160, 137, 80) mm Hg.

26. Inspired air is soon moisturized in the large upper airways. This humidified air:

a) reduces the PO_2 further
b) helps keep mucous produced in the airways loose
c) helps to reduce elastic recoil
d) none of the above

27. Gas exchange is normally completed at the alveoli in about (1/4, 1/2, 3/4, 1) second. This compares favorably to the (1/4, 1/2, 3/4, 1) second that a red blood cell circulates about the alveoli.

28. Respiratory failure is commonly diagnosed as:

a) PaCO2 > 60 mm Hg
b) PaCO2 < 30 mm Hg
c) PO2 < 50 mm Hg
d) none of the above

29. Carbon monoxide poisoning may not be immediately recognizable. Oxygen saturations are normally (high, low) while nail bed colour is (pink, blue). This can be a respiratory emergency, nevertheless. The hemoglobin's high affinity for carbon monoxide, (10, 100, 200, 400) times that of oxygen, causes significant deterioration in (alveolar gas exchange, oxygen transport).

30. Blood oxygen content is largely determined by:

a) dissociated oxygen values
b) hemoglobin levels
c) oxygen saturation
d) cardiac output
e) d only
g) b and c only
f) all of the above

31. A pulseless patient quickly develops both respiratory and metabolic acidosis.

True or False

***Answers:* 25.** *160, 304* **26.** *a)* **27.** *1/4, 3/4* **28.** *a)* **29.** *high, pink, 200, oxygen transport* **30.** *g)* **31.** *True*

32. Each gram of hemoglobin when fully bound with oxygen can carry about 1.34 ml of oxygen. The oxygen content (oxygen volume per litre of blood) for a person with a hemoglobin of 160 g/litre and an oxygen saturation of 90% is:

a) 214 ml of oxygen per lire of blood
b) 193 ml of oxygen per lire of blood
c) 162 ml of oxygen per lire of blood
d) 141 ml of oxygen per lire of blood

33. An average adult requires about 250 ml of oxygen per minute to sustain life.

True or False

34. Effective oxygen delivery is dependent on the following factors:

a) cardiac output
b) hemoglobin concentration
c) oxygen saturations
d) all of the above

35. A standard oxyhemoglobin dissociation curve depicts the relationship between oxygen saturations and oxygen actually delivered to the tissue.

True or False

36. The oxyhemoglobin dissociation curve provides the following important observations:

a) incremental increases in oxygen saturations above 90% does not result in significant increases in available oxygen at the tissues
b) there is not much value in providing supplemental oxygen for those with oxygen saturations over 95%
c) oxygen saturations decrements below 90% result in plummeting oxygen availability at the tissues
d) all of the above

37. Many experts believe that for those who experience a sudden cardiac death (i.e. due to ventricular fibrillation), oxygen reserves are sufficient to sustain life for the first 4-6 minutes in the presence of effective chest compressions.

True or False

***Answers:* 32.** *b)* **33.** *True* **34.** *d)* **35.** *False* **36.** *d)* **37.** *True*

38. Circle all parameters that result in more oxygen released to the tissues.

a) low pH
b) high pH
c) high PCO2
d) hypothermia
e) low body temperature
f) increased 2,3-DPG levels

39. More oxygen is released to the tissues for every oxygen saturation when the oxyhemoglobin dissociation curve shifts to the (right, left).

40. On average, there is about (1, 10, 100, 1000) pulmonary capillaries for every alveolus.

Suggested Readings and Resources

Ball, Dr. Wilmot. (1996). Interactive Respiratory Physiology. John Hopkins School of Medicine. Found at http://oac.med.jhmi.edu/res_phys/

Levitzky, Michael G. (2003). Pulmonary Physiology. 6th ed. New York: McGraw-Hill

Lingappa, Vishwanath R. (2000). Physiological Medicine. New York: McGraw-Hill

Pulmonary Critical Care Medicine Tutorials. (2003) CCM Tutorials.com. Found at http://www.ccmtutorials.com/rs/oxygen/index.htm

What's Next?

This chapter covered pulmonary anatomy, the mechanics of breathing, gas exchange, oxygen transport, and the oxyhemoglobin dissociation curve: a lot to digest. Chapter 9 builds on this ground work, providing a closer look at three respiratory technologies that are commonly used during the cardiac periarrest period: pulse oximetry, end-tidal carbon dioxide monitoring and blood gas analysis. The basics would not be complete without at least a brief look at these useful respiratory assessment tools.

***Answers:* 38.** *a), c), f)* **39.** *right* **40.** *1000*