What It Takes To Breathe

by Julie Aberger

EMT Objectives

After reading this article, the EMT will be able to:

- list structures and functions of the conduction and respiratory portions of the respiratory system;
- distinguish respiration from ventilation;
- explain the inverse relationship of gas volume and pressure and how it affects ventilation;
- understand some basic chemical changes that occur in the bloodstream that prompt the body to change its respiratory pattern;
- identify normal as well as abnormal respiratory rate, depth and conditions;
- understand the vital relationship of pulmonary perfusion and ventilation.

Introduction

Breathing is easy: Air in, air out. It seems pretty simple.

But actually, it's not.

Breathing is part of a system – the respiratory system – whose sum of its innumerable parts makes up the whole. Knock out any individual part, and breathing falters.

As you know, the respiratory and the cardiovascular systems work together for a mutual purpose: perfusion. The body's billions of cells need a constant supply of oxygen in order to create energy needed for life. At the same time, carbon dioxide, the waste product of metabolism, must be removed from the cells to prevent damage and destruction. Perfusion, or cellular gas exchange, is essential to life.

This interdependent relationship between the two systems is obvious, but actually *all* organ systems of the body depend on the adequate functioning of the respiratory system, e.g., endocrine, nervous, urinary, digestive, immune, lymphatic, etc. And, vice versa, the respiratory system depends upon all the body's other systems to adequately function. It's an organ-wide physiological "co-dependent" relationship without any baggage!

This article is an "under the hood" piece, examining the nuts and bolts of this extraordinary engine we call the respiratory system. Although we briefly consider engine failure, it is primarily about respiratory anatomy and physiology. *(Note: Abnormal conditions are denoted with an asterisk.)*

Gas In The Air

Let's first consider what's in air itself. The atmosphere – the whole mass of air surrounding you –contains 78.09% nitrogen, 20.95% oxygen, 0.93% argon, 0.04% carbon dioxide and small amounts of other gases. *(See Figure 1.)* You inhale 20.9% oxygen; you exhale 15.3%. Therefore, your body extracts or captures 5.6% oxygen.

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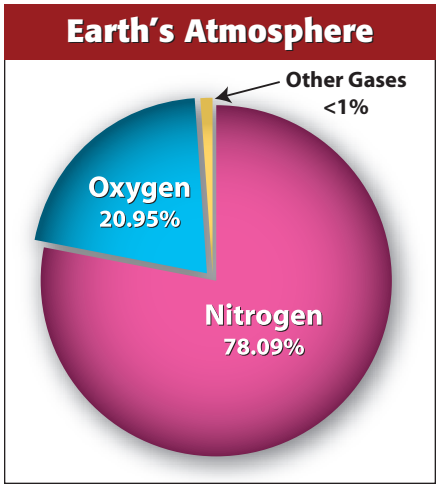
Changes in the composition of gasses in the atmosphere can lead to inadequate perfusion, and subsequent hypoxia. For instance, if there is a mass of carbon monoxide in the air, hemoglobin, the protein carrier in red blood cells, prefers to transport it instead of oxygen. Consequently, oxygen is displaced by a poisonous gas.

Figure 1:



Is adequate oxygen abundant everywhere in the earth's atmosphere?

At 7,000 feet above sea level, the percentage of oxygen is the same, but there are fewer oxygen molecules in the air and the body's O2 saturation begins to plummet. The "thinner" air means there is less oxygen to breathe. To survive at high altitude, it's necessary to breathe faster and deeper. *The climber needs to acclimatize to the changes in air pressure and oxygen levels by taking time (sometimes days) for her body to recalibrate the levels of carbon dioxide and oxygen in the bloodstream. *(See Figure 2.)* If she doesn't, she risks "altitude sickness," e.g., headache, dyspnea, weakness, nausea, vomiting, etc. Symptoms may

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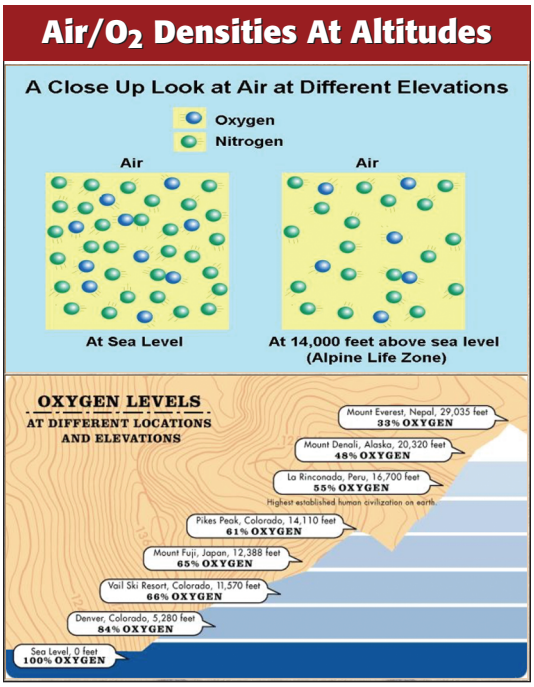
be mild at 8,000 ft above sea level, but above 12,000 ft, the affliction can become critical and even life threatening, e.g., cyanosis, shortness-of-breath at rest, inability to walk, confusion, and pulmonary edema.

Figure 2:



Conduction

In order to move air to the lungs, we look to the structures of the respiratory system; all its parts must be sound for the whole to function. *(See Figure 3.)*

The respiratory system can be divided into the *conducting* portion that conditions air – warms, humidifies, and filters it – before it reaches the bronchioles, and the *respiratory* portion where passive gas exchange occurs between the alveoli and the blood.

● Nasopharynx, oropharynx and pharynx, the vestibules of respiration: Air first enters the nose or mouth (or both) and moves into the back of the throat, or pharynx. *The passageway is wide; there's plenty of opportunity here for airway obstruction, or aspiration: food, teeth, vomit, blood, bone, and secretions. But the most common obstruction is the tongue. When you lose consciousness, the muscles in your mandible relax, causing your tongue to fall into the back of your throat, blocking your airway.

● Epiglottis: the structure that covers the windpipe when you are swallowing and opens when you are speaking or breathing, a protective device to keep food (and any other foreign body) out of your lungs.

*The epiglottis may fall forward and occlude the glottic opening when a patient is unconscious. *It can also become infected and swell, and in case of epiglottitis, be fatal. This condition can affect adults as well as children.

● Larynx, or voice box, is where air passes from the pharynx to the trachea. *Laryngospasm, a condition caused by smoke, asthma, allergies, exercise or even stress, can cause the larynx to suddenly shut, closing off air. The patient may feel as if he is choking and can't breathe. *Gastroesophageal reflux disease, or GERD, can also provoke laryngospasm that produces stridor, the high-pitched breathing sound.

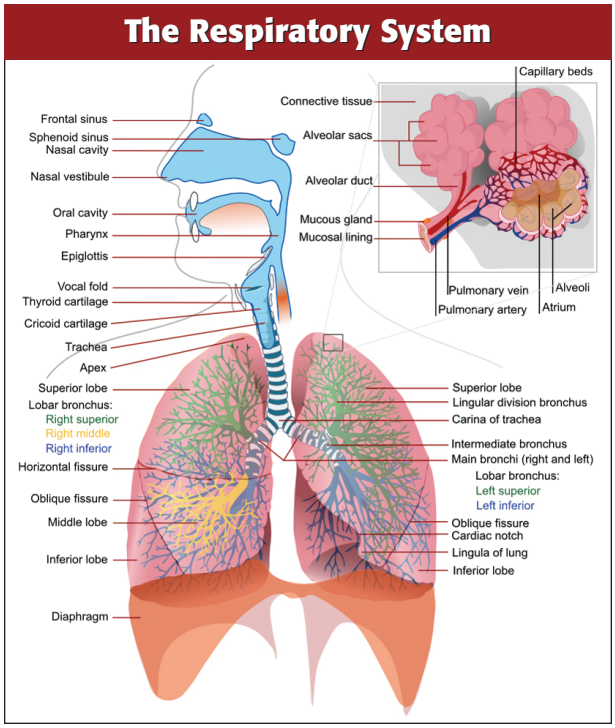
● Trachea, bronchi, bronchioles: The multi-passageways, or tubes, from largest to smallest, that carry air to the alveoli.

*The trachea is about 4.3 inches long, and an inch in diameter. It extends from the sixth cervical vertebra to the fifth thoracic vertebra. It divides into the right and left main-stem bronchi that further subdivide into approximately 23 "generations," or groups of branching airways. The trachea is kept open by 20 C-shaped cartilage rings. Between these rings, a layer of smooth muscle allows for flexibility during inhalation and exhalation. The two main stem bronchi and bronchioles are also lined with smooth muscle that allows them to constrict. *After prolonged contraction, the muscle may suddenly spasm, narrowing, or completely constricting the airway. *The trachea, bronchi and bronchioles can become obstructed, infected, inflamed, and swell, occluding the passageway and increasing the work of breathing. Infection also causes an increase in mucous production, decreasing the diameter of the airway and causing more airway resistance.

Laryngospasm, a condition caused by smoke, asthma, allergies, exercise or even stress, can cause the larynx to suddenly shut, closing off air.

A word about the gross anatomy of the lungs: They comprise five lobes, three on the right, two on the left. *(See Figure 4, next page.)* Anteriorly, both lungs extend from the base of the neck above the clavicle of the first rib, down to the sixth rib at the mid-clavicular line.

Figure 3:



Respiratory

Respiration is the process by which oxygen and

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carbon dioxide diffuse in and out of the blood. It occurs in two areas of the body. *(See Figure 5.)*

- External respiration refers to gas exchange in the alveoli, the 300 million microscopic air sacs that resemble clusters of grapes and make up the lungs. *(See Figure 6.)* Their surfaces secrete "surfactant" which decreases the surface tension between the thin alveolar walls, and prevents them from collapsing on exhalation.
- Internal respiration refers to gas exchange, or perfusion, that takes place between the cells and the blood.

Pulmonary respiration takes place through diffusion; we inhale oxygen; we exhale carbon dioxide. The two gases diffuse simultaneously, going from an area of greater concentration to an area of lower concentration. The oxygen then attaches itself to hemoglobin in the red blood cells that then carry it to the body's cells. After the oxygen and other nutrients have metabolized, the cell disposes of the waste product – CO_2 – by sending it back into the bloodstream to be dumped at the lungs. Carbon dioxide can be transported back to the lungs in three ways: dissolved in plasma; chemically bound to hemoglobin; or converted to bicarbonate ions in plasma.

How We Breathe: Ventilation

Ventilation is the movement of air into and out of the lungs. *Inhalation* is an active process that depends upon the contraction of the respiratory muscles, primarily the diaphragm. *Exhalation* is a passive movement of air out of chest that depends primarily on the elasticity of the lungs. Both

Figure 6:

Gas Exchange In Alveoli

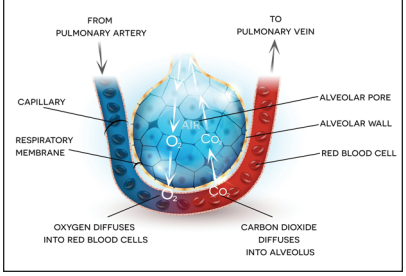
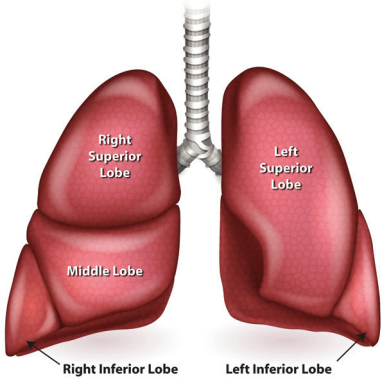


Figure 4:

Five Lobes Of The Lungs



adhere to Boyle's Law of physics that defines how gases behave with respect to pressure and volume.

Boyle's Law says: "a volume of gas is inversely proportionate to the pressure." The increase or decrease of the pressure within the chest cavity is dependent upon its internal size, or volume. (Recall that the chest cavity is essentially a closed chamber.) Volume leads to pressure changes that lead to the flow of gasses to equalize the pressure.

- When you inhale, your diaphragm contracts, and moves downward, flattening. At the same time, the external intercostal muscles pull the rib cage up and out. Both actions increase the size or *volume* of the space within the chest cavity.

Respiration is the process by which oxygen and carbon dioxide diffuse in and out of the blood. Ventilation is the movement of air into and out of the lungs.

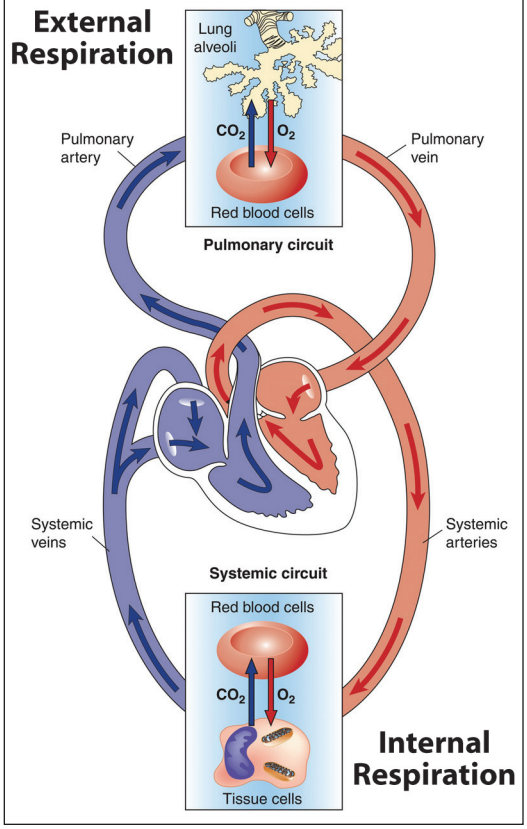
As the size or volume increases, the air pressure within decreases, and you have a negative pressure, called a pressure differential, i.e., a difference in air pressures. This leads to the flow of about 500 mL of air into the lungs until the intrapulmonary and atmospheric pressures equalize. Then, inhalation stops and exhalation begins. As the respiratory muscles relax passively, the elastic alveoli recoil, expelling the air up and out of the lungs.

Thoracic volume depends largely on the diaphragm, the main muscle of respiration that longitudinally separates the thoracic cavity from the abdominal cavity. (Several structures pass through openings in it: the aorta, the vena cava and the esophagus.) When you inhale, the diaphragm contracts and flattens, increasing volume for the spongy lungs to inflate. When you exhale, the diaphragm relaxes and resumes its dome shape, decreasing volume and squeezing air out of the lungs. The lungs, much like sponges, diminish in size.

The diaphragm depends on the brain stem to control respirations through the phrenic nerve, which originates in the neck and descends through the thorax to the diaphragm. *Traumatic injury to the head or brain stem is often catastrophic to the phrenic nerve destroying respiratory control. *Other disorders that may critically affect this essential muscle are disease, and congenital and

Figure 5:

External/Internal Respiration



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acquired anatomical development. The diaphragm can also be ruptured during blunt force injury.

Accessory Muscles: What are they? And where are they found? These muscles are the superchargers of the respiratory system that are used when it is necessary to either inhale or exhale forcefully to get additional air in or out.

*For instance, consider a COPD patient whose alveoli have lost their elasticity and no longer recoil to expel air. To compensate, the patient engages his accessory muscles to exhale forcibly. *(See Figure 7.)* The continuous additional muscular exertion causes labored breathing and the distinct facial flushing of "pink puffers."

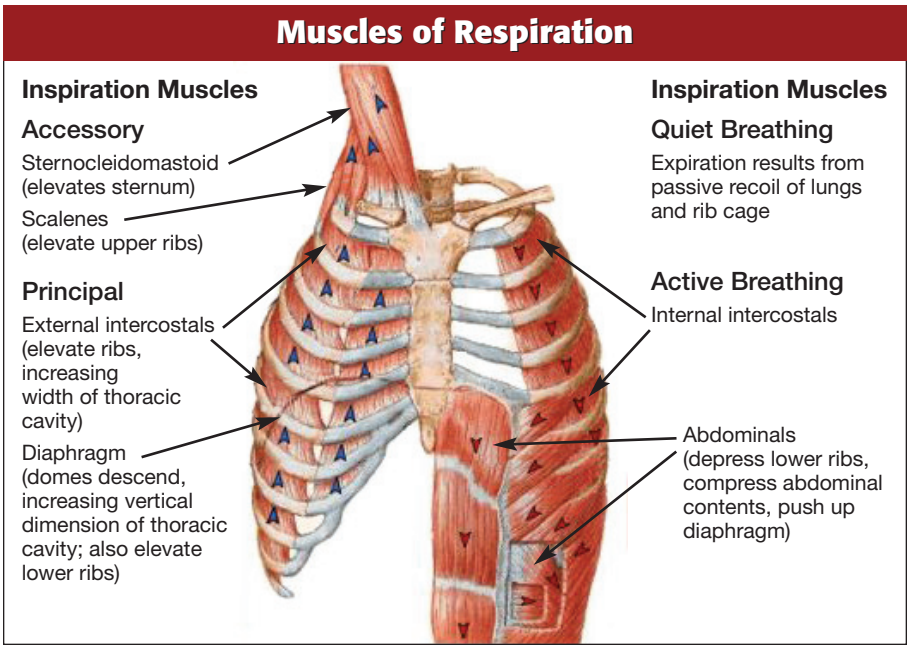
Thoracic volume depends largely on the diaphragm, the main muscle of respiration that longitudinally separates the thoracic cavity from the abdominal cavity.

● Located in the neck, the sterno/cleido/mastoid (separated for easier reading) muscles and the scalene muscles as well as other muscles in the rib cage, provide strength when you forcefully inhale, increasing the volume of air in your lungs.

● Located between the ribs: When you exhale, the intercostal muscles contract and strengthen your ability to quickly decrease the volume of air in your lungs.

You have eleven internal and eleven external intercostal muscles. Each rib is connected to the rib below it by both an external and an internal intercostal muscle. (The twelfth rib is the exception as it is the inferior-most rib.) When you inhale, your external intercostal muscles contract and elevate the ribs, spreading them apart. When you exhale, your internal intercostal muscles pull the ribs down and bring them closer together. The compression of the ribs decreases the volume of the thoracic cavity, resulting in the forced exhalation of air from the lungs. *(See Figure 8.)*

Figure 7:



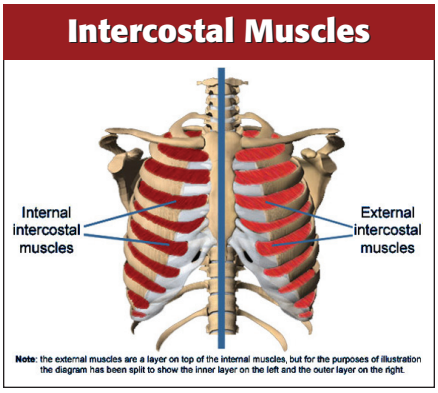
● The abdominal muscles are normally relaxed. But when you exhale forcefully, they contract, pushing the diaphragm upward, and forcing the air out of the chest cavity.

Compliance, Resistance, Pleural Space, Minute Volume & Dead Space

What else must function for ventilation to occur?

● Lung tissue must be compliant, that is, it must be able to stretch and continuously inflate and deflate. The alveoli must be elastic enough to recoil and expel air. *Lung tissue can be damaged by smoking, GERD, tuberculosis and pneumonia and exposure to toxic substances. Pulmonary fibrosis patients lose their lung compliance after years of exposure to toxic gases, and their air sacs stiffen. *Some emergency responders working at the World Trade Center in 2011 suffered subsequent pulmonary damage from toxic inhalation that necessitated lung transplants.

Figure 8:



The structures in the thoracic cage must also be flexible. *Aging can cause costal cartilages to ossify, or turn into bony tissue, which hinders chest expansion. *In COPD patients, the lungs become progressively more fibrous making ventilation difficult.

● The bronchi and bronchioles must be open and not occluded by obstructions such as mucus or swelling, commonly seen (and heard) in asthma and bronchitis patients. *Resistance* is determined by the internal diameter of the lower airways leading to the alveoli. High resistance can lead to dyspnea and apnea: The patient works harder to move air in and out, which causes him to tire and eventually, stop breathing.

● The (potential) space between the inner wall of the chest cavity – the parietal pleura – and the outer wall of the lung – the visceral pleura – must be intact.

There must also be adequate surfactant, the serous fluid produced by the alveoli that acts as a lubricant when the chest cage and lungs slide past each other during ventilation. These two linings are in close contact; they have a molecular attraction and

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oppose separation, like two flat wet panes of glass stacked on top of one another. *If air enters the potential space between the two linings, the lung(s) loses its ability to inflate and the patient becomes dyspneic. This is called a pneumothorax. *(See Figure 9.)*

How does a pneumothorax occur? A lung loses its integrity when it is ruptured, resulting in either a closed or open injury. (The chest wall is intact in a closed pneumo, broken in an open. *See Figure 10.*) In a closed pneumo, the victim draws air into the lungs, which escapes through the hole into the pleural space between the two linings. The air is trapped and does not escape on exhalation. With every breath, the presence of air in the pleural space slowly exceeds the alveolar pressure, and the lung begins to compress. As the patient continues to inhale, the trapped air continues to amass and its pressure increases causing the affected lung to collapse (atelectasis) and shift towards the unaffected side, eventually pushing the heart, trachea, esophagus, and great vessels with it as well. This is a tension pneumothorax. Blood flowing into the chest cavity from an internal injury can also accumulate in this space causing a hemothorax pneumothorax, aka hemo-pneumothorax.

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Figure 9:

Three Types Of Pneumothorax

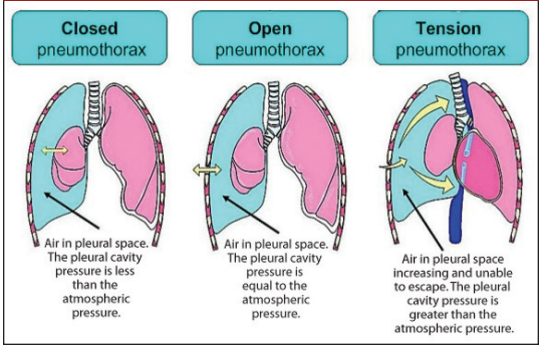
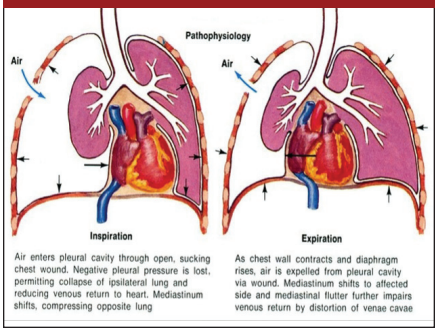


Figure 10:

Open (Sucking) Pneumo



- There must be an adequate minute volume. At rest, an average adult inhales approximately 500 mL per breath at a rate of 12 breaths-per-minute. **Minute volume** is calculated by multiplying the patient's breaths-per-minute (12) times the amount of inhaled air, or the tidal volume (500 mL) 12 x 500 mL= 6,000 mL or 6 liters/minute. Change the rate or amount of inspired air, and the patient risks dyspnea.
- An adequate amount of air must reach the *alveoli*. This is called alveolar ventilation and it depends upon volume and rate of respiration. Of the 500 mL of air you inhale, only about 350 mL reach the alveoli. About 150 mL of air fill and remain in the **dead spaces**, such as the pharynx, larynx, trachea, bronchi and bronchioles. If there are changes in the respiratory volume and/or rate, the alveolar ventilation is affected. If you ventilate a patient with too little volume, too little air reaches the air sacs, gas exchange is inadequate and the patient becomes hypoxic.

Primary Drive to Breathe: Chemical Equilibrium

Before we look at perfusion, we need to know one more major fact about ventilation. What activates the respiratory system to breathe?

The primary drive to breathe is based on the level of CO_2 in your arterial blood. If that fails, your respiratory drive depends secondarily on the level of *oxygen* in your arterial blood.

Your blood is kept in a chemical equilibrium by "receptors," or nerve endings that respond to particular types of stimuli. These specialized receptors are found in the medulla in the brainstem, and within the walls of the aortic arch and the carotid artery.

- The medulla contains *central* chemoreceptors that are extremely sensitive to changes in the chemistry of the blood and cerebral spinal fluid.
- Chemoreceptors in the aortic arch and the carotid artery are called *peripheral* chemoreceptors and are extremely sensitive to changes in oxygen levels. This is called hypoxic drive. *A person who has smoked or been exposed to other toxic gases, may eventually lose the elasticity of his alveoli; the airsacs can no longer recoil effectively to expel carbon dioxide. Consequently, his body's "normal" arterial blood level of CO_2 becomes chronically elevated. The central chemoreceptors, now desensitized to fluctuations in these gas levels, finally fail.

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This is when the secondary drive to breathe kicks-in; the peripheral chemoreceptors now monitor the level of *oxygen* in the arterial blood. When the oxygen level is too low, the respiratory drive is stimulated and you take a breath. This is called "hypoxic drive," no doubt a familiar term to most EMTs. When the oxygen level in the arterial blood is adequate, no breath is needed.

*You're administering 15 lpm oxygen via nonrebreather to a patient with an unknown history of emphysema when he suddenly quits breathing. What happened? By flooding the

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system with almost 100% oxygen, the patient's stimulus to breathe – low oxygen levels – is deactivated. *But never withhold oxygen from a patient in respiratory distress. In this case, be ready to ventilate with a bag valve mask.*

What happens when chemoreceptors detect significant changes in blood gas levels? Truth is, the chemistry is complex and too onerous to explain in a short CEU article. But be aware, that when chemical imbalances occur, they prompt the body (the lungs and the kidneys) to reflexively take protective action, such as changing the heart rate, respiratory rate, and blood pressure, to restore homeostasis. Here are some of the conditions our EMS patients experience and we witness:

- An increase in CO_2 triggers the central chemoreceptors to dump (expel) the excess gas. The brain signals the body to increase its respiratory rate and depth to blow-off more CO_2. *Consider an elderly bedridden patient in a nursing home whose breaths are slow and shallow.

Chemical imbalances prompt the body to take protective action, such as changing the heart rate and blood pressure, to restore homeostasis.

After a time, she begins to retain CO_2 from hypoventilation. Eventually, her breathing becomes abnormal, deep, rapid and labored in an attempt to rid the body of excessive carbon dioxide. This is called Kussmaul breathing and occurs in central nervous system disorders, diabetic ketoacidosis as well as kidney failure.

- A decrease in CO_2 produces hyperventilation. *When a patient experiences anxiety or panic, and breathes too deeply and rapidly ("excessive breathing"), too much carbon dioxide is removed from the bloodstream, leaving the cells depleted. Hypocapnia, or low arterial CO_2, causes a chemical reaction that causes vasoconstriction of the cerebral blood vessels, and results in hypoxia. The patient feels confused, lightheaded, faint, has tingling in his hands and feet, and around his mouth as his muscles contract and spasm.

Hypocapnia, or low arterial CO_2, causes a chemical reaction that causes vasoconstriction of the cerebral blood vessels, and results in hypoxia.

This patient does ***not*** need oxygen! He needs to restore his arterial CO_2. In addition, check the patient's pulse oximetry. If those numbers are good, the EMT should first urge him to slow his breathing and coach him for a minute or so in doing it. No paper bags! There are other acute critical illnesses, like pulmonary emboli, that act like hyperventilation. You need to know patient history!

Ventilation/Perfusion Ratio

In order for cellular perfusion to occur, there must be adequate ventilation and blood flow, i.e., there must be enough blood flowing through the pulmonary capillaries while at the same time, there must be adequate oxygen in the alveoli during ventilation.

Let's consider this vital relationship briefly:

The ratio that describes the amount of ventilation, or "V," with the corresponding amount of perfusion, or "Q," is V/Q. This ratio is never perfect (1-1). Conditions that cause alterations of V/Q include gravity, pressure imbalances and ventilation disturbances.

- The apexes, or tops of the lungs, contain fewer alveoli, but they have a greater capacity than the alveoli at the bases. That means they can hold more air, but because of gravity and surface tension, make them less compliant and difficult to fill. The greatest amount of ventilation and corresponding blood flow occur in the more numerous alveoli in the bases. Consequently the bases are much better perfused. **A good reason to keep patients with inadequate breathing sitting up!*
- Inadequate ventilation, resulting from bronchospasm or mucous plugging for example, leads to inadequate gas exchange. There's not enough air/oxygen reaching the alveoli to diffuse into the capillaries.

Inadequate ventilation, resulting from bronchospasm or mucous plugging, for example, leads to inadequate gas exchange.

- Inadequate perfusion, resulting from hypotension or pulmonary emboli, for example, leads to inadequate gas exchange. There's not enough blood in the capillaries to diffuse with the oxygen in the alveoli.

*A patient who is hemorrhaging loses blood pressure; less blood is shunted through the capillaries creating hypoperfusion.

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Why Do I Have To Know A&P?

What importance is all this nuts-and-bolts information to an EMT?

As you may remember, our 2009 National EMS Education Standards omitted the "B" in EMT-B and we were once again plain old EMTs. The implication now is that we are required to "think critically" beyond *basic* protocols, and apply that thinking to clinical decisions. In order to do that, we must know more science, i.e., medicine, including pathophysiology. To do that, you must know the anatomy and physiology of the human body.

It's vital to understand not only what to do about an airway or breathing problem, but what is causing it. EMTs have been tasked with a higher duty, one that necessitates more study and medical insight. Continual training and study are key.

***Julie Aberger** is an EMT instructor and an active member of the **Pennington First Aid Squad.** Julie is also the editor emerita of* The Gold Cross.

Many thanks to my many friends who helped with this article including Dr. Alice Freeman, Doug Kabay, Jon Politis, Andy Jefferson and reader Joan Schwarzwalder.

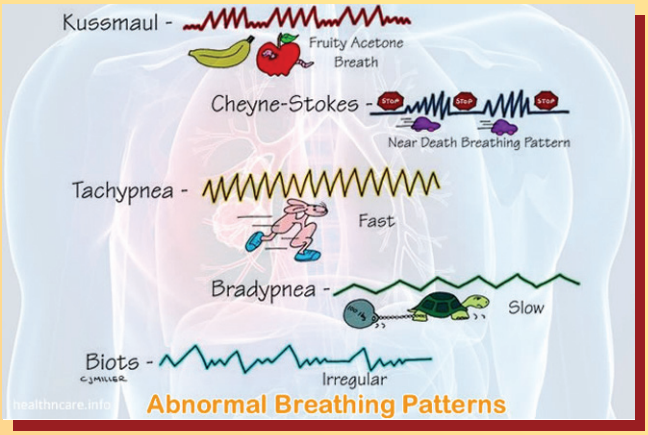
A BIT OF BASIC RESPIRATORY REVIEW

- Normal respiratory rates in breaths-per-minute:

Adult 12-20	Child 15-30
Infant 25-50	Newborn 40-60

A patient with respirations less than 8 or more than 24 BPM needs your immediate attention: she may need ventilation. But first, consider history, i.e., what happened, or her chief complaint. Is the patient breathing rapidly because she's in respiratory distress or did she just jog several miles?

- Normal respiratory depth: Check for adequate chest expansion, its rise and fall. If breathing is shallow and the chest is barely rising, the patient needs ventilation.



- Recognize inadequate breathing patterns: rapid, irregular breathing is a sign of respiratory distress. Head injuries and strokes may produce Cheyne-Stokes respirations; a repetition of increasing, decreasing then total cessation of breath which alone can last up to a minute. Kussmaul's respirations occur as deep, gasping breaths, commonly seen in patients trying to blow off excessive CO_2. Biot's breathing occurs with increased intracranial pressure and produces two-to-three short breaths with long, irregular periods of apnea. Agonal describes those last gasping breaths before death.

- Recognize signs & symptoms of respiratory distress or impending failure: tripoding, labored breathing, use of accessory muscles, inability to speak in full sentences, agitation, confusion, somnolence, and unconsciousness. Diminished or absent breath sounds are abnormal, so is the presence of rales, rhonchi and wheezing.

- Skin color and condition may be one of the most obvious signs of poor perfusion. A patient breathing inadequately may be cool, clammy and cyanotic.

Pink is good, blue is bad! If your patient is pale, ashen, or dusky blue or gray, give her oxygen.

- Oxygen Therapy:

*Pulse oximetry measures the oxygen saturation of arterial capillary blood. It can also be used to monitor the effectiveness of the EMT's oxygenation therapy (NC, NRB or BVM). *This device is used as an adjunct to patient assessment! Its results alone are not significant.* Conditions that may affect the pulse ox reading: hypothermia, hypovolemia, carbon monoxide or cyanide poisoning, some fingernail polish, anemia, Sickle Cell disease, even bright lights. A normal pulse ox reading falls between 96-100%.

*Adequate oxygen delivery to an adult with a bag-valve-mask device requires you to deliver 600 ml of air; squeezing two-thirds of the bag over one second into the patient's lungs every five seconds. Do not squeeze the bag too hard or too fast! Excess air ends up in the belly, forcing the diaphragm upward and creating more difficulty breathing.

- Nasal cannulas are still used primarily when the patient cannot tolerate a nonrebreather mask. Used together with the pulse ox, they are currently also used for patients complaining of chest pain, but no difficulty breathing. Nasal cannulas play a minor role in EMS, and are actually designed for long-term hospital use.