

Pulmonary Critical Care Primer

Introduction/Purpose Statement

The lungs are responsible for oxygenating all 50 billion cells in the body and for helping in the excretion of waste products. Primary or secondary insults to the lungs can cause a rapid decline into critical illness. The purpose of this home study is to give you information on the relevant A & P, pathophysiology of the pulmonary system, ABG interpretation, and basic interventions to form a foundation for understanding the assessment and management of pulmonary embolism, pneumonia, COPD, asthma, and ARDS.

Target Audience

This home study was designed for the novice critical care or telemetry nurse; however, other health care professionals are invited to complete this packet.

Content Objectives

1. Define the process of oxygenation and ventilation.
2. Identify acid-base disturbances based on blood gas analysis.
3. Review oxygenation and ventilation modalities used for the critically ill patient.
4. Differentiate between the pathophysiologies of asthma, bronchitis, and emphysema.
5. Discuss the pathophysiology of pulmonary embolism, pneumonia, and acute respiratory distress syndrome (ARDS).

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December 31, 2017—your envelope must be postmarked on or before that day.

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Review of Pulmonary Anatomy and Physiology

Upper Respiratory Tract

The upper respiratory tract is comprised of the nose, mouth, pharynx, larynx, and trachea. Air enters the body through the nose or mouth and moves through the pharynx. The respiratory tract is lined with ciliated mucosal cells. These cells cleanse the airway by moving debris and mucus up and out. This mechanism is called the “mucociliary escalator.” The upper respiratory tract:

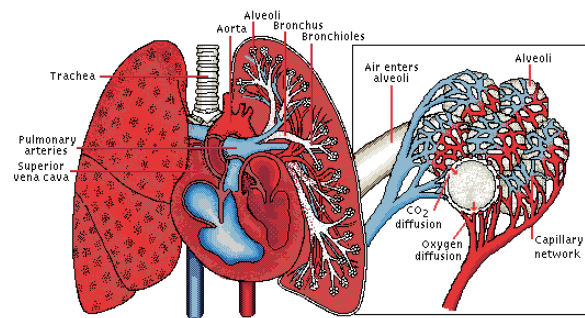
- conducts and conditions air
- protects the airways
- makes speech and smell possible

Lower Respiratory Tract

The air moves past the epiglottis, larynx, and through the trachea into the lungs. The **epiglottis** covers and protects the airway by preventing aspiration of food or foreign bodies. The **larynx** is a structure that houses the vocal cords, which are designed to produce sound through vibration and movement. The **trachea** is 10-12 cm long and consists of 16-20 C-shaped rings made of cartilage that cover its anterior side.

The lower respiratory tract begins when the trachea bifurcates into the **right and left mainstem bronchi**, at a site called the **carina**. The right mainstem bronchus is shorter, wider, and more vertical than the left. The bronchi are made of cartilage and are surrounded by muscles that run longitudinally and spirally around the bronchi. The main bronchi each branch into five lobar bronchi. The lobar bronchi branch into segmental bronchi, which divide into terminal bronchioles, which then divide into respiratory bronchioles.

At the end of the respiratory bronchioles lies a cluster of several alveoli, called an acinus. The acinus is the area where gas exchange takes place. Adults have 200-600 million alveoli with a total surface area of 40-100 square meters. The alveolar membrane is about 0.2 microns thick.



The lungs themselves are air-filled, spongy structures that are divided into lobes. The right lung has three lobes and normally accounts for 55% of total ventilation. The left lung has two lobes and accounts for 45% of ventilation.

The Mechanics of Ventilation

Inspiration is an active process -- muscles have to contract to cause air to flow into the lungs. The **diaphragm** is the major muscle of inspiration. This large muscle is located just underneath the rib cage and contracts to pull the rib cage down and out. This produces a negative pressure (vacuum) inside the thorax, which pulls air in. Nerves coming from the spinal cord at the C3-C5 level innervate the diaphragm. Seventy percent of the tidal volume is provided by the action of the diaphragm.

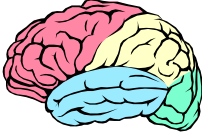
Another group of muscles that is normally used in inspiration is the **internal intercostal** group. These muscles are located between the ribs and elevate the ribs when contracted, increasing the antero-posterior diameter. If you place your hand on your ribs and deeply inspire, you will note that your ribs come up and out. These muscles are innervated at the T1-T11 level of the spinal cord.

The third group of muscles is not used in normal inspiration. The **scalene and sternocleidomastoid** muscles are called “accessory muscles,” and pull up the sternum and ribs when used. Think of a long distance runner after a race. The runner will stand with his hands on his knees and breathe deeply, so that you can see the sternocleidomastoid bulge and the clavicles rise. These muscles are used when additional volume of inspiration is needed (as in exercise), when the body’s demand for oxygen is greater than the supply, when the airway is obstructed, or there are lung compliance problems.

Exhalation is a passive process. The relaxation of the inspiratory muscles will “push” air out of the thorax. In the event of difficulty in breathing, the abdominal muscles and external intercostal muscles can contract to push up and back, which will press the air out of the lung.

Control of Ventilation

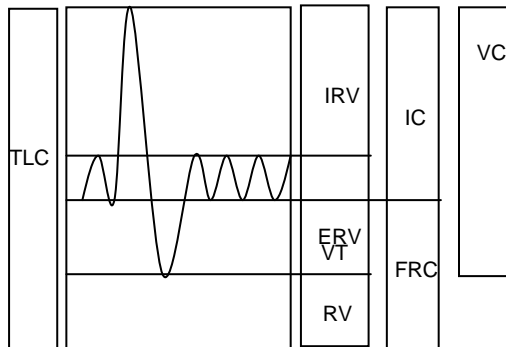
There are several mechanisms that control ventilation:

1. **The cerebral cortex** controls voluntary breathing, which makes holding breath and hyperventilation possible. 
2. **Brain stem:**
 - The lower pons (pacemaker or apneustic center) produces sustained inspiration when stimulated.
 - The upper pons (pneumotaxis center) initiates expiration when stimulated by the apneustic center.
 - The medulla (the “manager”) receives messages from the chemoreceptors to stimulate inspiration.
3. **Chemoreceptors** are receptors that are sensitive to hydrogen ion and oxygen concentration. They are located in the aorta and carotid arteries, and medulla. Changes in the PaCO₂, pH, and PaO₂ cause the respiratory rate and tidal volume to change to maintain adequate oxygenation and acid-base balance. The central chemoreceptors in the medulla are most sensitive to hydrogen ions and CO₂. Those in the aorta and the carotid bodies (peripheral chemoreceptors) are most sensitive to oxygen.

Lung Volumes and Capacities

The amount of air moving in and out of the lungs can be broken down into specific volumes. Two or more volumes combine to form a capacity. Many of these volumes and capacities, called Pulmonary Function Tests (PFT's), can be measured for diagnostic purposes.

The following chart shows the volumes and capacities and describes what each one measures.



TLC = Total Lung Capacity

- air in lungs after full inspiration ~ 6,000 ml

IRV = Inspiratory Reserve Volume

- air forcibly inhaled above VT

VT = Tidal Volume

- air inhaled or exhaled with each breath

ERV = Expiratory Reserve Volume

- air forcibly exhaled above VT

RV = Residual Volume

- air that always remains in lung

IC = Inspiratory Capacity

- max amount of air inhaled after a normal exhalation

FRC = Functional Residual Capacity

- amount of air in lungs after tidal breath

VC = Vital Capacity

- amount of air that can be forcibly inhaled and exhaled with one breath

Physiology of Ventilation

There are two parts to gas exchange: ventilation (V) and perfusion (Q). Ventilation refers to the movement of **air** in the pulmonary airways; perfusion refers to the movement of **blood** in the pulmonary vasculature. The pulmonary arteries, veins, and capillaries, a low-pressure system, together contain about 500-750 ml of blood, 10-15% of the cardiac output.

The capillary bed in the lung is a network of very thin, fine vessels that enclose each alveolus. The alveolar-capillary membrane is approximately 2 microns thick. This extremely fine membrane allows the easy diffusion of gases. Gases move from areas of high pressure to areas of lower pressure.

The amount of ventilation (V) and perfusion (Q) is expressed by the ratio V/Q. The normal amount of ventilation is 4 LPM, and normal amount of perfusion is about 5 LPM, so V/Q overall = 4/5 or 0.8. There are, however, regional differences in the different parts of the lung depending on position. With normal lung function, ventilation and perfusion is greater in the bases of the lungs when the person is in an upright position.

Changes in the V/Q ratio occur when perfusion does not match ventilation. There are two reflexes that work to keep V/Q normal:

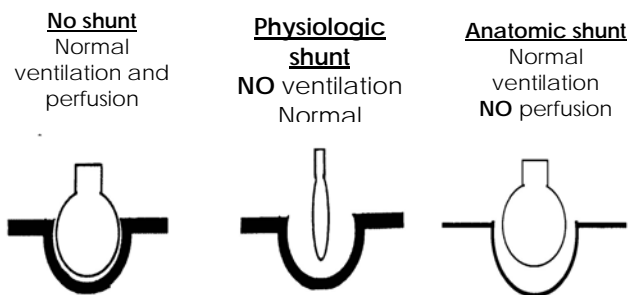
1. **Pulmonary vasoconstriction:** when there are alveoli not helping with gas exchange, the blood vessels supplying those alveoli constrict (e.g., pneumonia, COPD)
2. **Terminal bronchiole constriction:** When blood is not flowing past the alveoli, the smooth muscle in that area constricts (e.g., pulmonary embolism)

Alterations in the VQ ratio can occur from one of two physiologic mechanisms: shunting or increased dead space.

Shunting

A “shunt” occurs when a portion of the cardiac output (**blood**) does not participate in gas exchange. An “anatomical shunt” occurs when a portion of the cardiac output bypasses the alveolar-capillary unit. This is normal with bronchial, pleural, and thesbian vasculature.

Abnormal shunting occurs with structural abnormalities, such as pulmonary AV fistula (intrapulmonary), Tetralogy of Fallot (intracardiac), or shunts related to neoplasms. In a “physiological shunt,” blood is circulating through non-ventilated alveoli, as is seen with atelectasis, pleural effusions, pulmonary edema, or pneumonia.



Dead Space

Dead space is the volume of **air** not participating in gas exchange. Dead space is normally 2 ml/kg. In **anatomical dead space**, a portion of each breath fails to reach the alveoli for perfusion. This is normal in the trachea and large airways, such as the bronchi. Anatomical dead space is increased in persons with large airways or long ventilator tubing. **Physiological dead space** exists when the alveoli receive air but do not connect with the capillary membrane. An example of increased physiologic dead space is emphysema -- the alveolar walls and capillary beds have been destroyed, leaving a large amount of air space that doesn't connect with a capillary.

Compliance

Compliance is to the ability of the lungs to stretch (elasticity or distensibility) and recoil. It is measured as the volume of air per unit of pressure change (i.e., ml/cm H₂O). Normal lung compliance is 200 ml/cmH₂O. **Increased compliance** indicates that the **pressure** needed to stretch the lungs is **less** than normal, from:

- “stretched out” lungs (as is seen in emphysema) - leading to increased expiratory work
- the patient assisting the pressure-controlled ventilator

Decreased compliance means greater inspiratory work because of “stiff” lungs (e.g. ARDS). As compliance decreases, the **pressure** required to deliver the same volume **increases**.

Resistance

Resistance is the pressure inside the airways as air flows into the lungs. To a certain extent, the normal airway “resists” the entrance of air, simply because the airways become smaller. Resistance is measured in terms of cm H₂O/liters of flow. An increase in the resistance to air flow can be measured with a **peak pressure**. Factors that influence resistance include:

Airway:

- flow rate of the gas: noninvasive oxygen delivery, CPAP, or mechanical ventilation
- size/diameter: bronchospasm
- obstruction: kinks, H₂O in tubing, secretions

Lung:

- chest size
- volume of gas
- elasticity

Chest wall:

- deformities
- position of patient
- external compression of chest wall or diaphragm (ascites, obesity, pregnancy)

Neuromuscular disorders:

- Guillain-Barré
- ALS (amyotrophic lateral sclerosis/Lou Gehrig’s disease)

Work of Breathing

The work of breathing (WOB) refers to the how much energy the ventilatory muscles require. At rest, the work of breathing consumes 1-3% of the cardiac output. The work of breathing can be either increased or decreased;

however, we are more concerned about increases in the work of breathing. Work of breathing can be increased by a variety of factors:

1. Hypoxemia, acidosis, hypercarbia
2. Airway resistance problems: secretions, bronchospasm, artificial airway
3. Lung compliance problems: ARDS
4. Increased metabolic work: hyperthermia, hyperthyroidism

Increased WOB may lead to respiratory muscle fatigue and decompensation. If the oxygen demands of the body continue to be higher than the supply, the patient may exhibit hypoxemia, tissue hypoxia, acidosis, and hypercarbia, resulting in arrhythmias and cardiac arrest.

Oxygenation

The amount of oxygen can be measured three different ways in the blood: the partial pressure of oxygen (PaO₂), O₂ content, and O₂ saturation.

1. The **PaO₂** is the pressure (P) exerted by oxygen (O₂) dissolved in the arterial blood (a).
2. **Oxygen content** (CaO₂) is the number of milliliters of oxygen carried by 100 ml of whole blood.
3. **Oxygen saturation** (SaO₂) is the percent (%) of oxygen that the hemoglobin is carrying.

PaO₂

The PaO₂ (pO₂) represents the amount of oxygen that is physically dissolved in the blood -- about 3% of the total oxygen. The greater portion of oxygen (about 97%) is chemically bound to hemoglobin as **oxyhemoglobin**. The PaO₂ reflects gas exchange in the lung and is the driving force behind hemoglobin saturation.

A normal range for PaO₂ on room air is 70-100 mm Hg. This measurement can be affected by:

- **age**: as people age, their "normal" PaO₂ decreases
- **altitude**: the higher the altitude, the lower the pressure to push oxygen into the blood
- **FiO₂**: the Fraction of inspired oxygen (FiO₂) is the amount of oxygen that is being inhaled. Decreases in the amount of oxygen will lead to a decrease in the PaO₂.

Oxygen Saturation and Oxyhemoglobin

Oxygen saturation (SaO₂) calculates the percentage of oxygen that hemoglobin is transporting. Each gram of hemoglobin can carry 1.34 ml of oxygen. **Oxyhemoglobin** can be determined when the hemoglobin

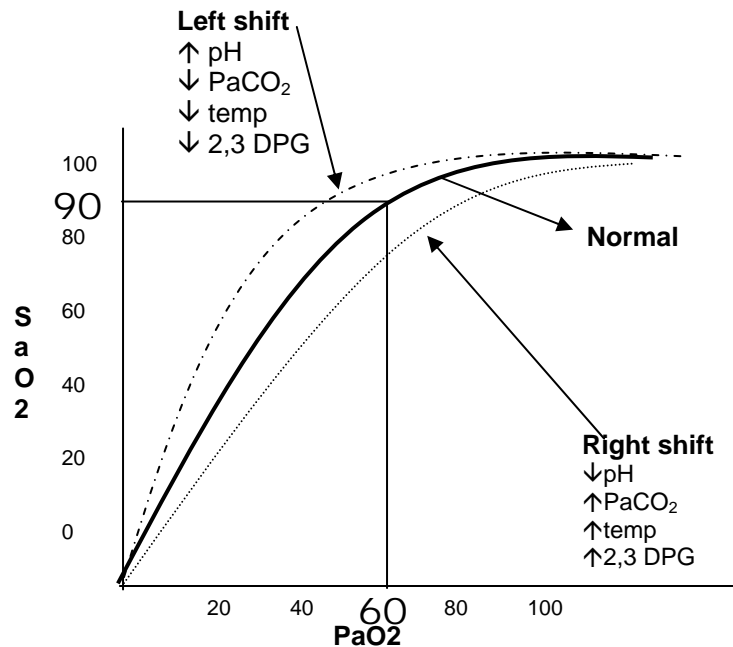
(Hgb), SaO₂, and the cardiac output (CO) are known. The formula is:

$$\text{Oxyhemoglobin saturation} = (1.34 \times \text{Hgb} \times \text{SaO}_2) \times \text{CO}/100$$

Pulse oximetry uses light-emitting devices to detect the saturated hemoglobin and non-saturated hemoglobin. The percentage of saturated hemoglobin is usually 90-100%. Because it is a percentage, the SaO₂ can never be more than 100%. The SaO₂ reflects oxygenation status, not ventilation status (pH, PaCO₂).

Benefits of SaO₂ Monitoring

- ♦ Noninvasive
- ♦ Accurate with an SaO₂ > 70%



- ♦ Can have continuous monitoring

Limitations of SaO₂ Monitoring

- ♦ Affected by a poor pulsatile signal (hypotension, shock, vasopressors)
- ♦ Affected by high bilirubin levels (such as in liver failure)
- ♦ Excessive movement will limit accuracy of monitor

Oxygen Content (CaO₂)

The CaO₂ represents the total amount of oxygen in the body - both dissolved and bound to hemoglobin. Some intensive care units and physicians calculate this figure to determine how well oxygenated the patient is. The calculation for the CaO₂ is:

$1.34 \text{ ml O}_2/\text{gm Hgb} \times \text{gm/Hgb} \times \text{SaO}_2 + 0.003 \times \text{PaO}_2$

The normal CaO_2 is 20 ml/100 ml blood. Here are some examples:

1. A patient with hemoglobin of 14.6, an SaO_2 of 98%, and PaO_2 of 99 would have a CaO_2 of:
 $1.34 \times 14.6 \times .98 + 0.003 \times 99 = 19.99$
2. A patient with a hemoglobin of 11, an SaO_2 of 90%, and PaO_2 of 65 would have a CaO_2 of:
 $1.34 \times 11 \times .90 + 0.003 \times 65 = 13.47$

The CaO_2 is affected by changes in the amount of hemoglobin, changes in the saturation of hemoglobin with oxygen, and the PaO_2 .

Oxyhemoglobin Dissociation Curve

The oxyhemoglobin dissociation curve defines the relationship between dissolved oxygen (PaO_2) and the oxygen actually carried by the hemoglobin (oxyhemoglobin). This curve reflects how easily Hgb gives up oxygen to the tissues.

The flat upper portion of the curve illustrates that if the PaO_2 drops from 100 to 70, the saturation decreases only slightly. Adequate amounts of oxygen will be carried to the tissues even with a lower PaO_2 . The steep midportion of the curve demonstrates that slight reductions in PaO_2 result in large reductions in the saturation of Hgb.

Many physicians write orders to keep the $\text{SaO}_2 > 90\%$. This is because an SaO_2 of 90% is roughly equal to a PaO_2 of 60. A PaO_2 of 60 will keep tissues alive.

The relationship between PaO_2 and SaO_2 is affected by alterations in the pH, temperature, CO_2 , and 2,3 DPG (a substance that facilitates dissociation of O_2 from hemoglobin at the tissues). If the hemoglobin-oxygen affinity is high, oxygen is easily bound to hemoglobin and does not want to release to the tissues. This is called a “**shift to the left**”. When the hemoglobin-oxygen affinity is low, oxygen is not easily bound to hemoglobin; however, the hemoglobin readily unloads its O_2 at the tissue level. This is called a “**shift to the right**”.

Hypoxemia/Hypoxia

The terms “hypoxia” and “hypoxemia” are sometimes used interchangeably, but they represent different concepts.

- **Hypoxia** is an inadequate amount of oxygen available at the tissue level (*We can't measure this*)
- **Hypoxemia** is an inadequate amount of oxygen in the blood (*We can measure this*)

Hypoxia occurs for a variety of reasons:

- Pulmonary causes:
 1. Alveolar hypoventilation
 2. Diffusion defects at the alveolar-capillary level
 3. Right to left shunt
 4. V/Q mismatch (the most common cause)
- Nonpulmonary causes:
 1. Reduced blood flow: myocardial infarction, shock, or dysrhythmias
 2. Anemia
 3. Nonfunctioning hemoglobin: Hgb is bound to other substances, such as CO poisoning
 4. Mitochondrial failure: cyanide poisoning

Compensatory Mechanisms to Prevent Hypoxia

The body has a number of compensatory mechanisms that it uses to correct hypoxia.

1. The respiratory system will increase the minute ventilation by increasing the respiratory rate and/or the tidal volume, and will change the blood flow to optimize the VQ ratio.
2. The heart rate and contractility will increase, and selective vasoconstriction and vasodilation will take place to pump oxygenated blood to the priority organs.
3. The kidneys will excrete erythropoietin, which increases red blood cell production in the bone marrow (erythrocytosis).

With the exception of the kidneys' response, all of the compensatory mechanisms to correct hypoxemia increase the tissue demand for oxygen, thus increasing workload.

Measures to Increase Oxygenation

Administration of Oxygen

The fastest way to increase oxygenation is to administer oxygen! Oxygen therapy is used to treat hypoxemia, to decrease the work of breathing, and to decrease the work of the heart.

Nasal Prongs (Cannula)

Nasal prongs (cannula) are used when an exact concentration of oxygen does not need to be guaranteed.

Adults and pediatric patients are put on a flow of 1-6 liters per minute (LPM), while infants can be put on up to 2 LPM. Nasal prongs are indicated for supplemental oxygen, not patients in acute distress. The approximate concentrations of oxygen per liter of flow per minute are:

1 L = 24%	2 L = 28%	3 L = 32%
4 L = 36%	5 L = 40%	6 L = 44%

A "bubbler" humidifier can be used for flow rates of 4 liters or higher. **CAUTION:** a bubbler humidifier should not be used with any device other than a nasal cannula, as it may cause harm to the patient.

Simple mask

The simple mask can be used to deliver 6 - 10 LPM of oxygen, which approximates 35-55% fraction of inspired oxygen (FiO₂). The actual amount of oxygen delivered can vary greatly with changes in the patient's ventilatory pattern.

Non-rebreather (partial) mask

The mask of choice for emergency situations is the non-rebreather mask. This mask delivers nearly 100% oxygen as long as the following criteria are met:

1. The mask fits the patient's face snugly
2. The flow to the reservoir bag is adjusted so that the bag does not totally collapse when the patient inhales (the bag is always partially inflated)

The partial non-rebreather mask consists of a pliable mask with a reservoir bag and two one-way valves. For safety's sake, there is only one valve on the side of the mask so if the source of the oxygen fails, the patient can entrain room air. The second one-way valve is located on the reservoir bag so the patient cannot "rebreath" exhaled gas.

Venturi mask

The Venturi mask is considered a "high flow system." This mask is used when a consistent FiO₂ is needed and the patient does not require added humidity. The mask has either an adjustable Venturi, or individual Venturis that can be changed to allow different oxygen concentrations. Be sure to set the flowmeter to the appropriate liter flow. Each Venturi requires different flows; this is usually stamped with the concentration of oxygen it will deliver; e.g. 50% FiO₂/15 LPM.

High flow humidifiers

A high flow humidifier is indicated for patients whose natural mechanism for heating and humidifying inspired

gas has been bypassed (i.e. intubated or tracheostomy patients). It can also be used for patients whose natural mechanism is not sufficient to prevent retention of secretions due to mucosal drying. The tubing, reservoir, sterile water, and "mask" must be changed at the frequency required by Infection Control policies and prn. Heated humidity devices will cause condensation in the tubing; this condensate must be drained from the circuit and not drained back into the reservoir to prevent contamination.

Aerosol (nebulizers)

Aerosol therapy is indicated for the following conditions:

- ◆ The presence of upper airway edema; i.e. laryngotracheobronchitis, subglottal edema, post-extubation edema, post-operative management of the upper airway.
- ◆ The presence of one or more of the following: stridor, brassy croup-like cough, hoarseness following extubation, upper airway irritation (smoke inhalation) or airway insult.

Continuous aerosol therapy may be delivered via face mask, face tent, hood, or blow-by. An oxygen analyzer **must** be used on all infant hoods. Condensate must be drained frequently from tubing to avoid contamination. Contamination with an aerosol can cause the contaminates to become airborne.

Ventilation

Ventilation is the movement of air, both into and out of the lungs. Ventilation is dependent on:

- ◆ Respiratory effort
- ◆ Respiratory rate
- ◆ The distance between the blood and the gas exchanging part of the alveolus

Ventilation is measured by the PaCO₂ on the ABG and by end-tidal CO₂ monitoring (capnography).

PaCO₂

Measurement of the PaCO₂ is done on the arterial blood gas. Like the PaO₂, the PaCO₂ is measured as a pressure, in mm of Hg. The normal PaCO₂ is between 35 and 45 mm Hg.

End-Tidal CO₂ Monitoring

End-tidal CO₂ monitoring or "capnography" is useful for determining that tracheal, rather than esophageal,

intubation has taken place. Capnography can also be used to evaluate the efficiency of mechanical ventilatory support and for monitoring the severity of pulmonary disease and response to therapy.

Ways to Improve Ventilation

Effective Coughing Techniques

In the old days, we were taught to have the patient cough with every deep breath, and to have patients blow up balloons in an effort to prevent pneumonia. We know now that those techniques are not helpful. Coughing is effective only when there is something to cough out. Deep exhalation and needless coughing cause the alveoli to collapse, causing more atelectasis. Coughing also causes an increase in the intracranial pressure and pain in surgery patients.

There are several methods of coughing that are effective in clearing the smaller airways of mucous. They are:

1. *Cascade cough*: have the patient inhale deeply through the nose, hold for 1-3 seconds, then cough forcefully several times.
2. *Huff cough*: have the patient inhale deeply through the nose, hold for 1-3 seconds, then say "huff" forcefully several times.
3. *End-expiratory cough*: have the patient take a normal breath, then at the end of a normal exhalation, have the patient cough once. Follow by a cascade or huff cough.

Deep Breathing and Incentive Therapy

Effective deep breathing and incentive therapy will aid in alveolar expansion, will help to clear the smaller airways, and will improve stress. The patient should be encouraged to breathe deeply at least ten times every hour. For patients who cannot breathe and are on the ventilator, the "sigh" button can be used to give them a breath that is 1 1/2 times the normal tidal volume. Check with the physician before doing this.

Chest Physiotherapy

Often done by respiratory therapists, chest physiotherapy is designed to mobilize secretions by a sequential application of "cupping" or "thumping" the posterior, and sometimes anterior, chest.

Nasal Pharyngeal Airway (Trumpet)

The trumpet is an excellent ventilation and suctioning aid. It can be used in conscious or unconscious patients. The

trumpet is placed, using a water-soluble lubricant, into the patient's nare, using a gentle back and forth motion. Optimally, the trumpet should be moved from nare to nare every 24 hours to prevent skin breakdown.

Oral Airway

The oral airway is a good tool to keep the tongue out of the airway. It can be used only for patients who are unconscious and who are not gagging. The oral airway can be dangerous to use in patients who may vomit, as it provides a more open channel for aspiration. Generally, an oral airway is used as a temporary measure until the patient wakes up or is intubated.

Tracheostomy

The ultimate in invasive ventilation techniques, the tracheostomy is used for a wide variety of purposes. Patients may require a tracheostomy for long-term mechanical ventilation or as a result of neck, throat, or mouth surgery.

CPAP or BiPAP

Another way of improving oxygenation and ventilation is by using non-invasive positive pressure, such as CPAP or BiPAP. CPAP stands for "Continuous Positive Airway Pressure," and BiPAP stands for "Bilevel Positive Airway Pressure." Both will provide positive pressure or "PEEP" for spontaneously breathing people. It is important to differentiate between CPAP and BiPAP. BiPAP is essentially "Bi-level" ventilation - or CPAP with pressure support. BiPAP is for spontaneously breathing patients who require additional positive pressure for their inspiratory cycle.

Indications for positive airway pressure are to:

1. Reduce air trapping in patients with asthma and/or COPD.
2. Help mobilize retained secretions in patients with cystic fibrosis or chronic bronchitis.
3. To prevent or reverse atelectasis.
4. Optimize bronchodilator delivery in patients receiving bronchial medication therapies.
5. Help redistribute extra-vascular water, such as in pulmonary edema.
6. Assist with breathing for those with ventilatory muscle weakness, but who do not wish to be intubated.

CPAP can be delivered a number of ways, either with a nasal or full-face mask or with a mechanical ventilator via

an endotracheal tube or tracheostomy. Patients, such as those with obstructive sleep apnea (OSA), may also have a small bedside CPAP machine for the home care setting. These patients use the CPAP at night and when napping.

One of the major complications of CPAP and BiPAP use relates to skin breakdown because of the tight-fitting nasal or facial mask. For this reason, these machines have been designed to have a small leak when placed on the patient.

In the critical care setting, patients who are alert and cooperative may benefit greatly from this type of ventilation. Patients who are at risk for vomiting, who have facial trauma, or who are not able to comply with the treatment should not be on CPAP/BiPAP treatment.

Arterial Blood Gas Analysis

Arterial blood gases (ABG's) provide information about oxygenation and acid-base balance. Acid-base status reflects physiologic processes and chemical reactions. Acid-base balance refers specifically to the regulation of hydrogen ion concentration in the body.

Obtaining an ABG

1. Identify the pulsating arterial site.
2. Perform the Allen's test.
3. Thoroughly cleanse area with Betadine solution and let dry for three minutes (can also scrub area for one minute with alcohol prep).
4. Stabilize the artery by pulling the skin taut and bracketing the pulsating area with the first two fingers of your non-dominant hand.
5. Holding the syringe like a pencil, puncture the skin slowly (at about a 45 degree angle). Advance the needle with the bevel up.
6. Wait for flash of arterial blood to occur.
7. If no flash occurs, withdraw slowly until the needle is almost out, and redirect.
8. When flash occurs, allow syringe to fill with at least one ml of blood.
9. Withdraw needle and apply pressure to the site for five minutes. While holding pressure, carefully rotate the syringe to mix the blood and heparin.
10. Using universal precautions, remove the needle from the syringe and place cap (see your unit policy). Immediately place on ice and send to the lab. The ABG is no longer valid after 30 minutes.

Do's and Don'ts

- ◆ **DO** document the SaO₂ at the exact time the ABG is drawn. The SaO₂ is calculated on the ABG from the pH and HCO₃⁻ and should correspond closely with the oximeter measurement.

- ◆ **DO** document the respiratory rate, effort, and use of accessory muscles.
- ◆ **DO** document patient temperature
- ◆ **DO** document amount of oxygen the patient is on.
- ⊘ **DON'T** draw ABG's if patient just suctioned.
- ⊘ **DON'T** draw ABG's if patient receiving nebulizer treatment.
- ⊘ **DON'T** draw ABG's if patient became short of breath doing an activity (or if SaO₂ dropped while YOU were doing something to the patient).
- ⊘ **DON'T** draw ABG's if patient is not on the amount of oxygen you want to assess (wait 20 minutes after any O₂ change).

Acid-Base Balance and the ABG

pH

The pH on the ABG is inverse logarithmic number of hydrogen ions in the blood. **Normally, the pH should be 7.35-7.45.** If the number of hydrogen ions rises, the blood is more acidotic. If the number of hydrogen ions falls, the blood is more alkalotic.

Maintaining a Normal pH

The body really likes to keep a normal pH. In order to maintain the blood pH between 7.35-7.45, the body has a buffering system. There are two major chemical buffers, regulated by the respiratory and renal systems, in the body:

- carbon dioxide (CO₂): **the normal PaCO₂ on the ABG is 35 - 45 mm/Hg**
- bicarbonate (HCO₃⁻): **the normal HCO₃⁻ level on the ABG is 22 - 26 mEq/L**

The respiratory system responds within 1-3 minutes to changes in acid-base balance. If the chemo-receptors sense too many hydrogen ions (acidosis), it will stimulate the respiratory center to breathe faster and deeper – to “**blow off**” CO₂. If the chemoreceptors sense too few hydrogen ions (alkalosis), it will depress the respiratory center to **keep** CO₂.

The kidneys compensate over 24-48 hours to correct imbalances. If the kidneys see acidosis, they will retain, regenerate or synthesize HCO₃⁻ and excrete H⁺. If the

kidneys see alkalosis, they will excrete HCO_3^- and retain H^+ .

If the body sees acidosis, it will:

Increase the respiratory rate to blow off CO_2
Retain, regenerate or make bicarbonate
Excrete hydrogen ions

If the body sees alkalosis, it will:

Decrease the respiratory rate to keep CO_2
Excrete bicarbonate
Retain hydrogen ions

When there is an acid-base disturbance and either the lungs or kidneys react, it is called **compensation**. **Compensation** can be complete or partial. The body will compensate so that the pH reaches the edges of normal. For example, if the pH is 7.10 (acidosis), the body will try to compensate so that the pH will reach 7.35, not greater than 7.35. Partial compensation means that the pH has not reached a normal level.

Respiratory Acidosis

In **acute** respiratory acidosis, the lungs don't get rid of enough CO_2 .

Causes: oversedation, head trauma, respiratory and cardiac arrest

What to look for: $\uparrow \text{PaCO}_2$, $\downarrow \text{pH}$, normal HCO_3^-

Examples:

- pH 7.29, PaCO_2 57, HCO_3^- 28
- pH 7.06, PaCO_2 98, HCO_3^- 28

In **compensated** respiratory acidosis, the lungs still don't get rid of enough CO_2 , but the kidneys have had enough time to save bicarbonate.

Causes: COPD, spinal cord injury, respiratory muscle paralysis

What to look for: $\uparrow \text{PaCO}_2$, $\downarrow \text{pH}$, $\uparrow \text{HCO}_3^-$

Examples:

- pH 7.31, PaCO_2 76, HCO_3^- 39
- pH 7.34, PaCO_2 60, HCO_3^- 33

Respiratory Alkalosis

In **acute** respiratory alkalosis, the lungs are "blowing off" too much CO_2 , leading to an increased pH.

Causes: stress, pain, fever, and hypoxemia

What to look for: $\downarrow \text{PaCO}_2$, $\uparrow \text{pH}$, normal HCO_3^-

Examples:

- pH 7.52, PaCO_2 27, HCO_3^- 22
- pH 7.65, PaCO_2 23, HCO_3^- 24

Compensated respiratory alkalosis occurs when the lungs "blow off" too much CO_2 , but the kidneys have time to excrete bicarbonate and save hydrogen ions.

Causes: uncommon, but can occur in the patient with neurological damage

What to look for: $\downarrow \text{PaCO}_2$, $\uparrow \text{pH}$, $\downarrow \text{HCO}_3^-$

Examples:

- pH 7.49, PaCO_2 16, HCO_3^- 11
- pH 7.45, PaCO_2 23, HCO_3^- 16

Metabolic Acidosis

Metabolic acidosis occurs where there is either too much acid (such as in shock, hypoxemia, diabetes, overdose, renal failure) in the system, or when there is a loss of bicarbonate (diarrhea, NG suction, renal tubular acidosis).

Acute metabolic acidosis without compensation may be seen in the mechanically ventilated, sedated, or comatose patient. Because of the altered mental status, there is no compensatory response by the respiratory system.

What to look for: normal PaCO_2 , $\downarrow \text{pH}$, $\downarrow \text{HCO}_3^-$

Examples:

- pH 7.05, PaCO_2 37, HCO_3^- 7
- pH 7.23, PaCO_2 40, HCO_3^- 12

Compensated metabolic acidosis is much more common. The respiratory rate and depth increases to blow off CO_2 . There is a limit to how much the respiratory system can compensate. The PaCO_2 may be quite low, but it is still not able to bring the pH back to normal.

What to look for: $\downarrow \text{PaCO}_2$, $\downarrow \text{pH}$, $\downarrow \text{HCO}_3^-$

Examples:

- pH 7.19, PaCO_2 22, HCO_3^- 8
- pH 6.96, PaCO_2 9, HCO_3^- 2

Metabolic Alkalosis

In metabolic alkalosis, there is a gain of base or increased loss of acid, resulting in an increased pH. If there is a gain of base, such as in sodium bicarbonate (baking soda) ingestion or administration of NaHCO_3 during CPR, the HCO_3^- will be elevated. If there is loss of an acid, such as in vomiting or NG suction, the HCO_3^- will be normal in the acute phase.

Acute metabolic alkalosis is uncommon, but can be seen if the patient is not neurologically intact and is unable to increase the respiratory rate.

What to look for: normal PaCO_2 , \uparrow pH, \uparrow HCO_3^-

Examples:

- Gain of a base: pH 7.55, PaCO_2 40, HCO_3^- 42
- Loss of acid: pH 7.52, PaCO_2 37, HCO_3^- 28

Compensated metabolic alkalosis can look like:

What to look for: \uparrow PaCO_2 , \uparrow pH, \uparrow HCO_3^-

Examples:

- Gain of a base: pH 7.47, PaCO_2 46, HCO_3^- 42
- Loss of acid: pH 7.46, PaCO_2 44, HCO_3^- 26

There is also a limit to the compensation of the respiratory system in metabolic alkalosis. The body will not tolerate CO_2 levels over 50-55 mm Hg, and will increase the rate and depth of breathing after that point.

Now, you might notice that metabolic alkalosis from loss of an acid and respiratory acidosis look a lot alike. Here's how to tell the difference. There is an increase in CO_2 in both metabolic alkalosis and respiratory acidosis, but the pH will be relatively normal. In compensated respiratory acidosis, though, the pH will be on the **low** side of normal, not the high, and the HCO_3^- level will be high, not normal.

Analyzing the ABG

1. Look at the PaO_2 .

2. Look at the pH.

- a) Is it normal?
- b) Is it low normal or high normal? Look for changes in the PaCO_2 and HCO_3^- to see if there is compensation for a problem.

- c) If it is low (less than 7.35), the patient is in acidosis.
- d) If it is high (more than 7.45), the patient is in alkalosis.

3. Look at the PaCO_2 .

- a) The pH and PaCO_2 have a "teeter-totter" relationship. If the problem is **respiratory**, one will be up, and the other will be down.
- b) If the pH and PaCO_2 are both up or both down, the problem is **metabolic**. The teeter-totter isn't there, so it can't be a primary respiratory problem, instead, it is a metabolic problem with respiratory compensation.

4. Look at the HCO_3^- .

- a) The pH and HCO_3^- go up and down together in a **metabolic** problem.
- b) If the pH and the HCO_3^- are opposite (one is up and the other is down), the problem is primarily respiratory, and the HCO_3^- is trying to compensate.

Examples:

1) pH 7.01; PaCO_2 69; HCO_3^- 24

- a) The pH is very low, so it is acidosis.
- b) The PaCO_2 is high, making a teeter-totter with the pH, so it is a respiratory problem.
- c) The HCO_3^- is normal, so there is no compensation.
- d) Respiratory acidosis without compensation.

2) pH 7.33; PaCO_2 72; HCO_3^- 36

- a) The pH is low, so it is acidosis.
- b) The PaCO_2 is high, making a teeter-totter with the pH, so it is a respiratory problem.
- c) The HCO_3^- is high, so there is compensation, but not enough to bring the pH to normal.
- d) Respiratory acidosis with partial compensation.

3) pH 6.99; PaCO_2 20; HCO_3^- 2

- a) The pH is very low, so it is acidosis.
- b) The PaCO_2 is low; it is not a teeter-totter with the pH, so it is a metabolic problem with respiratory compensation.
- c) The HCO_3^- is low, confirming a metabolic problem.
- d) Metabolic acidosis with partial compensation.

4) pH 7.35; PaCO_2 65; HCO_3^- 32

- a) The pH is low normal.
- b) The PaCO_2 is high, making a teeter-totter with the pH, so it is a respiratory problem.
- c) The HCO_3^- is high, so there is compensation.
- d) Respiratory acidosis with compensation.

- 5) *pH 7.51; PaCO₂ 15; HCO₃⁻ 8*
- The pH is high, so it is alkalosis.
 - The PaCO₂ is low, making a teeter-totter with the pH, so it is a respiratory problem.
 - The HCO₃⁻ is low, so there is compensation.
 - Respiratory alkalosis with partial compensation.
- 6) *pH 7.78; PaCO₂ 59; HCO₃⁻ 40*
- The pH is very high, so it is alkalosis.
 - The PaCO₂ is high; it is not a teeter-totter with the pH, so it is a metabolic problem with respiratory compensation.
 - The HCO₃⁻ is high, confirming a metabolic problem.
 - Metabolic alkalosis with partial compensation.
- 7) *pH 7.45; PaCO₂ 37; HCO₃⁻ 24*
- The pH is normal.
 - The PaCO₂ is normal
 - The HCO₃⁻ is normal.
 - Normal acid-base balance.

Acute Respiratory Distress Syndrome (ARDS)

Larry Leakey is a 21-year-old man who was involved in a severe car accident. He underwent emergency surgery to repair a lacerated liver, perforated bowel, and tension hemopneumothorax. He received 15 units of blood during surgery. He was rapidly extubated after surgery and sent to the ICU.

The next day, Larry became increasingly short of breath and was using all accessory muscles. He had O₂ saturations in the 80's. His PaO₂ on blood gases was 34 mm Hg. His heart rate was 180 beats/min. Jeremy's chest x-ray showed diffuse, patchy infiltrates throughout his lung fields. His diagnosis was ARDS.

What is ARDS?

Acute Respiratory Distress Syndrome (ARDS) and its less severe cousin Acute Lung Injury (ALI) have been documented in medical history for at least two thousand years. It became better known during the Viet Nam War, when soldiers would develop respiratory failure and die after being wounded. Although over twenty years of extensive research and study have been given to ARDS and its treatment, the mortality for ARDS remains high.

What happens in ARDS?

ARDS always occurs as a secondary problem. Four hours to 48 hours after the initial insult, the immune system is

activated, causing inflammation in the lung. The white blood cells (particularly neutrophils) release chemical mediators which cause increased vascular permeability. In the lung, this becomes disastrous.

Fluid and proteins enter into and around the alveoli through the very thin vascular membrane, causing pulmonary edema. This fluid damages the Type II alveolar cells, destroying the capacity to make surfactant. This damage, combined with the "washing out" action of the fluid, decreases the amount of surfactant, which then leads to alveolar collapse and atelectasis.

The combination of pulmonary edema and atelectasis leads to intrapulmonary shunting, decreased compliance, and increased work of breathing. The patient begins to become more and more hypoxemic, even on high levels of oxygen.

As a result of hypoxemia, the pulmonary vasculature constricts through the capillary beds. This causes pulmonary hypertension. As the pressure builds in the pulmonary artery, more and more fluid is forced out into the alveoli and interstitium. The lung becomes even less compliant, causing an increased work of breathing and an increased oxygen demand.

What causes ARDS?

There are certain injuries, diseases, and interventions that are more likely to cause ARDS. The most common causes are listed below.

Most Common

- Sepsis
- Gastric aspiration
- Pneumonia
- Trauma

Other

- Drug overdose (e.g., ASA)
- Near-drowning
- Massive blood transfusion/transfusion reaction
- Inhalation of toxic gases and vapors
- Pancreatitis

Asthma exacerbation

Batt Atsma is a 37-year-old mother of two who has had asthma for many years. She is classified as a Step 3 - moderate persistent asthmatic. She enters the hospital with shortness of breath and wheezing unrelieved by her usual asthma medications.

What is asthma?

One of the diseases that can be considered both acute and chronic is asthma. People with asthma always have the underlying disease, but have exacerbations of asthma.

The conducting airways (bronchi, bronchioles) of the pulmonary system are hyperreactive in persons with asthma. With a precipitating factor, the smooth muscle of the airways constrict, causing decreased air conduction and increased breathing difficulty. With the smooth muscle contraction comes increased mucous production, mucosal cell swelling, and ventilation-perfusion abnormalities.

Because of the pressure dynamics in the chest, air will flow into the patient with an asthma exacerbation much more easily than it will flow out. Air becomes "trapped" inside the lungs, causing hyperinflation of the lungs. The resistance to airflow increases, causing the patient to work harder at breathing. The pressure inside the alveoli becomes greater (because of the air trapping) than the pressure in the airways, so more air becomes trapped in the alveoli.

What are the causes of asthma?

- Respiratory infection
- Allergic reaction to inhaled antigen
- Inappropriate bronchodilator management
- Idiosyncratic reaction to aspirin or other nonsteroidal anti-inflammatory agents
- Emotional stress
- Exercise
- Environmental exposure
- Occupational exposure
- Nonselective beta blocking agents
- Mechanical stimulation (coughing, laughing, cold air inhalation)
- Reflux esophagitis
- Sinusitis

Batt Atsma has been classified as a Step Three - moderate persistent asthmatic. She is on multiple inhalers through the day, and typically has two to three asthma "attacks" per week. She is no longer able to do many of the sporting activities that she used to do with her children.

Is there a classification system for how bad asthma is?

The National Asthma Education and Prevention Program, which is sponsored by the National Heart Lung and Blood Institute, has published guidelines to determine the

severity of illness for adults and children older than 5 years. This classification helps clinicians determine what treatments would best suit the patient.

Step One - Mild Intermittent

In this level, patients have symptoms more than twice a week, but are asymptomatic with a normal PEF between exacerbations. The exacerbations last from a few hours to a few days, and the intensity may vary. They have symptoms at night less than twice a month. The FEV₁/PEF is greater than 80% of predicted.

Step Two - Mild Persistent

Patients at a step two have symptoms more than twice a week but less than once per day. Exacerbations of their asthma may interfere with activities. They are prone to having nighttime symptoms more than twice a month, but still have an FEV₁/PEF ratio of greater than 80% of predicted.

Step Three - Moderate Persistent

These patients have symptoms every day and use inhaled short-acting beta₂-agonists every day. When they have exacerbations (>2 times/week), they affect the patients activities and may last for days. They have nighttime symptoms more than once a week, and have an FEV₁/PEF ratio of >60% and < 80% of predicted.

Step Four - Severe Persistent

The most severe of all of the steps, patients who are at a step four experience continual symptoms, have limited physical activity, and have frequent exacerbations. They have frequent nighttime symptoms and have a FEV₁/PEF ratio of less than 60% of predicted.

Pulmonary Function Assessment

One of the best indicators of asthma symptom severity is the FEV₁ on the spirometer. Standing for "Forced Expiratory Volume" in one second, it measures how much the patient is able to exhale forcibly after a normal inhalation. The amount exhaled in one second in normal lungs is approximately 80% of the total exhaled amount - that's where the 80% of predicted value comes from on the classification above.

Another measure of day to day function is the PEF - the peak expiratory flow rate. This is the fastest rate at which air can move through the airways in a forced exhalation. The day to day rate is measured against the patient's

personal best, and should be > 80% of what it optimally is. Measurements of the PEF can and should be done by the patient on a daily basis. Typically, a PEF >80% of the personal best is in a "green" zone - which indicates that the asthma is stable. A PEF of 60-80% is in the "yellow" zone and indicates that the patient should take extra caution or medications. A PEF of <60% is in the "red" zone and indicates that the patient is having a significant exacerbation.

What is status asthmaticus?

This is the term used to describe an asthma exacerbation that is refractory to bronchodilator therapy, including aminophylline IV and beta-adrenergic agents (epinephrine). It often needs further treatment, such as intubation and mechanical ventilation.

Chronic obstructive pulmonary disease

Joe Chronichlung is a 60 year-old male with end stage COPD. He was recently hospitalized for pneumonia and a COPD exacerbation and was sent to a transitional care facility for rehabilitation. Joe was a long-time smoker, but has not smoked since his last hospitalization.

In chronic obstructive pulmonary disease (COPD), there is an obstruction to air flow either into or out of the lungs. Chronic bronchitis and emphysema are the major diseases that cause COPD. Although the pathophysiology for each is discussed separately, please be aware that the two most commonly appear together.

What is chronic bronchitis?

In chronic bronchitis, persistent injury to the alveoli causes an overstimulation of mucus production, accompanied by a persistent cough. As the disease progresses, the bronchial walls thicken, causing the airway resistance to increase. The results of the bronchial wall thickening and excessive mucus production are:

- Hypoxemia and hypercapnia
- Chronic cough with sputum production
- Pulmonary hypertension from hypoxemia, leading to cor pulmonale

The diagnosis of chronic bronchitis is made when there is a history of a chronic productive cough for three months of the year in each of two successive years.

What is emphysema?

In emphysema, the alveolar walls are destroyed, causing the very small air sacs to enlarge into large air sacs, called

blebs. During the wall destruction, the capillary beds are also destroyed. The results of this are:

- Hypercapnia without hypoxemia (in the early stages)
- Bleb formation with potential for pneumothorax
- Air trapping within the blebs with constriction of the smooth muscle of the bronchioles

What are the causes of COPD?

- Cigarette smoking
- Environmental pollution or occupational exposure
- Alpha₁-antitrypsin deficiency (genetic marker for familial emphysema)

What type of tests can be done to assess Joe's pulmonary function?

Joe's last documented FVC was 3.12 and FEV₁ was 1.29. The FEV₁ was 32% of predicted.

Pulmonary function tests can be very useful in determining the function of the lungs in COPD, just as they can in asthma. The FEV₁ is the same test as for COPD as it is for asthma - the amount of air that can be forcibly exhaled in one second. In people with normal lungs, the FEV₁ should be > 80%. The FVC (Forced Vital Capacity) is also measured. Where the FEV₁ was the amount of air exhaled in one second, the FVC is the total amount of air that is exhaled quickly. It should be ≥ 15 L. This volume represents the patient's ability to breathe deeply and cough. This number is reduced in people with obstructive disease.

Pneumonia

S.A. Pneumo is a 47 year-old male who enters the hospital with shortness of breath, a cough, and a 1½ week history of flu-like symptoms. He is diagnosed with pneumonia.

What causes pneumonia?

Pneumonia has a number of causes. It can be caused by **microorganisms** such as bacteria, viruses, and fungi. Streptococcus pneumoniae, Mycoplasma pneumoniae, and Histoplasma capsulatum may all cause pneumonia in a normally healthy person. Pneumococcus, Escherichia coli, Pseudomonas aeruginosa, Serratia, Proteus, and Acinetobacter usually occur as pneumonias in patients who have a chronic disease, poor nutrition, trauma, surgery, or who are immunosuppressed.

Aspiration is one of the most common causes of pneumonia. Gastric contents contain caustic substances and bacteria. Entry into the airway causes the conducting airways to be blocked and the alveoli to be “burned” and become inflamed.

Chemical inhalation is another cause of pneumonia. Inhalation of smoke, cleaning chemicals and industrial chemicals causes caustic damage to the airways and alveoli. Inflammation and formation of exudate results, blocking the airways and alveoli.

What is the pathophysiology of pneumonia?

Once the foreign substance has entered the lungs, the immune response is initiated. First, certain alveolar cells begin to produce large amounts of mucous to try to coat the foreign substance. Second, white blood cells will attempt to “wall off” the foreign substance. Third, the inflammation triggered by the immune system will cause blood flow to the area to increase, as well as to increase capillary permeability.

If these normal mechanisms don't work, the patient becomes progressively more ill. Mucus and fluid from the capillaries fill the alveoli, increasing the space through which gas must travel. At the same time, the increased blood flow goes past alveoli that aren't contributing to gas exchange (physiologic shunting). And last, if the patient has a bacterial pneumonia, the bacteria may produce exudates that further clog the alveoli. These problems culminate in hypoxemia. Hypoxemia will cause the patient to breathe faster, leading to hypocapnia (blowing off CO₂).

What's the difference between a “community” and “nosocomial” pneumonia?

Pneumonias that begin outside of the hospital setting are called “community acquired” and pneumonias that start in health care facilities are known as “nosocomial” pneumonias. People who are at risk for acquiring pneumonia in the hospital are those who:

- are > 70 years old
- are intubated and/or on mechanical ventilation
- have a depressed level of consciousness
- have an underlying chronic lung disease
- have had a previous large volume aspiration
- are being given cimetidine for stress-bleeding prophylaxis
- are being given antimicrobials

- have an NG tube
- have had a recent bronchoscopy

Pulmonary embolism

Clottia Breathless is a 28 year-old woman who was admitted at 0500 for shortness of breath and chest pain. She has right calf tenderness, and her right leg is swollen and warm. The Homan's sign is positive on the right. Her tentative diagnosis is pulmonary embolism.

What is the most likely cause of Ms. Breathless' illness?

Perfusion to the lung may be disturbed by an embolus in the pulmonary vasculature. Pulmonary emboli may be made up of fat, air, or amniotic fluid. Pulmonary emboli are generally made of blood, which may form in the vasculature in the:

- Popliteal vein
- Iliofemoral vein
- Right side of the heart
- Pelvic area

What will favor the development of a pulmonary embolus?

The three factors, called Virchow's triad, favoring the development of venous thrombosis include:

- Blood stasis
- Blood coagulation abnormalities
- Vessel wall abnormalities

Emboli may also be formed from other substances that enter into the blood stream. Fat emboli can form when the long or flat bones of the body are broken; air emboli can occur with traumatic injury (pneumothorax) or leak in a central line; and amniotic fluid emboli may occur with an abruptio placentae.

How does blood normally flow in the lung?

Ninety-nine percent of the blood in the body goes through the pulmonary circulation to be re-oxygenated. The remaining 1% feeds the pulmonary tissues with oxygenated blood through the bronchial circulation. De-oxygenated blood enters into the lung through the pulmonary artery and travels to the capillary bed. The capillary bed is a network of very thin, fine vessels that enclose each alveolus (think of a spider web around a grape), which is optimal for gas exchange.

Oxygen, CO₂, and other waste products are exchanged between the alveolus and capillary through a pressure gradient system. Re-oxygenated blood travels out of the

capillary system, through the pulmonary vein and into the left heart.

A feature vital to efficient gas exchange is called "autoregulation," that refers to the ability of the arteries in the lung to constrict when blood is flowing by alveoli which are not contributing to gas exchange (i.e., an atelectatic alveolus), and to dilate when stimulated by the sympathetic nervous system. Vasoconstriction in response to non-gas exchanging alveoli is important to prevent shunting, which is blood moving from the venous to arterial side without receiving oxygen.

Ms. Breathless is experiencing hypoxemia as the blood supply to some of her alveoli is shut off. Although she is ventilating appropriately, the gases cannot diffuse into the blood stream. Ms. Breathless is experiencing autoregulation at this point to stop more blood from flowing to the blocked area.

What is the pathophysiologic process of a pulmonary embolism?

The embolus forms, enters into the venous system and travels through the right heart into the pulmonary vasculature. A large embolus tends to lodge in the upper part of the lung and causes rapid and severe deterioration, leading to cardiac arrest and death. Small, or micro, emboli tend to lodge in the lower part of the lung. Deterioration is slower and less severe; in some cases, a subtle problem.

What caused Ms. Breathless' symptoms?

The patient entered the ER with labored respirations (dyspnea) and a rapid respiratory rate (tachypnea). As the embolus blocked perfusion to a large number of alveoli, it decreased the amount of gas exchange. The resulting hypoxemia and hypercapnia triggered the chemoreceptors in the aortic arch, medulla, and carotid bodies to increase respiratory rate and effort to attempt to keep the body tissues oxygenated.

As the chemoreceptors trigger an increased respiratory rate and effort, the sympathetic nervous system is also stimulated to force the heart to pump the limited amount of oxygen faster. The increased heart rate and increased blood pressure are compensatory responses to hypoxemia.

Skin color may be a late indicator of oxygenation status. Cyanosis indicates that there are more desaturated hemoglobin molecules (blue) than saturated hemoglobin molecules (red). If the patient is anemic, there will be no cyanosis; rather, the patient will be pale.

What are the potential complications?

Pulmonary vascular pressure rises because of the mechanical blockage of a blood vessel. This elevation is called pulmonary hypertension. As the hypertension increases, the work of the right heart increases. This increased workload can lead to angina, myocardial infarction, or heart failure.

Summary

Without the lungs and proper lung function, every organ in the body would cease to function in minutes. The respiratory system is responsible for oxygenating the bloodstream and for removing excess gases from the circulation. Understanding how oxygenation and ventilation occur and how to interpret ABG's can help you determine how best to assess and manage your critically ill patient. Knowing the causes, pathophysiology, and some of the tests for selected pulmonary illnesses provides you with a foundation of knowledge for managing the acutely and critically ill pulmonary patient.

Recommended Reading

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Directions for Submitting Your Post Test for Contact Hours

To obtain a certificate of completion for this home study program, please complete the post-test and evaluation on the next few pages. If you are completing this home study as pre-reading for a TCHP class, please bring your post-test and evaluation to class with you for processing. The date on your certificate of completion will be the date that your home study is received. **Any materials received with a postmark after the expiration will be discarded.**

HealthEast, HCMC, & MVAMC Employees

If you are an employee of HealthEast, HCMC, or MVAMC, you may send the post-test and evaluation to TCHP for processing. Your post-test will be returned to you through your hospital. It cannot be mailed to your home.

Paid Participants

If you are not an employee of one of the TCHP hospitals, please send the post-test and evaluation to TCHP with a check for \$15.00. Please make check payable to **TCHP Education Consortium** and mail to:

TCHP Education Consortium
Capitol Office Building
525 Park Street, Suite 120
St. Paul, MN 55103

Your post-test will be returned to you with the certificate of completion.

Pulmonary Critical Care Primer Post Test

Please print all information clearly and sign the verification statement:

Name _____
(please print legal name above)

Birth date (required)

Format: 01/03/1999

M	M	D	D	Y	Y	Y	Y

For HealthEast, HCMC, or MVAMC, employees only:
Hospital _____ Unit _____

Personal verification of successful completion of this educational activity (required):

I verify that I have read this home study and have completed the post-test and evaluation.

Signature

- 1) The chemoreceptors are sensitive to:
 - a) CO₂ and O₂ levels
 - b) CO₂ and CO levels
 - c) O₂ and Hgb levels
 - d) O₂ and He levels

- 2) The tidal volume is the:
 - a) amount of air exhaled forcibly
 - b) the amount of air that is always in the lung
 - c) the amount of air present in the airways but not participating in gas exchange
 - d) the amount of air inhaled or exhaled with each breath

- 3) Pulmonary compliance may be increased in patients with:
 - a) ARDS
 - b) congestive heart failure
 - c) emphysema
 - d) asthma

- 4) What is the PaO₂ if the SaO₂ is 90%?
 - a) 50
 - b) 60
 - c) 70
 - d) 90

- 5) An oral airway is appropriate for:
 - a) an unconscious person
 - b) a nauseated person
 - c) a combative person
 - d) permanent placement

- 6) What does the following ABG show? pH 7.10, PaCO₂ 60, HCO₃- 22
 - a) respiratory alkalosis
 - b) respiratory acidosis
 - c) metabolic alkalosis
 - d) metabolic acidosis

- 7) What does this ABG show? pH 6.99, PaCO₂ 20, HCO₃- 2
 - a) respiratory alkalosis
 - b) respiratory acidosis
 - c) metabolic alkalosis
 - d) metabolic acidosis

- 8) Acute Respiratory Distress Syndrome can result from:
 - a) sepsis
 - b) near-drowning
 - c) gastric aspiration
 - d) all of the above

- 9) Which of the following part(s) of the lung are hyperreactive in asthma?
 - a) bronchioles
 - b) alveoli
 - c) capillaries
 - d) alveolar junction

- 10) Pathophysiologic changes that occur with emphysema include:
 - a) wall thickening
 - b) chronic cough with sputum production
 - c) chronic air trapping with bleb formation
 - d) pulmonary hypotension

- 11) What is the most common cause of pneumonia?
 - a) inhalation of smoke
 - b) fungal infections
 - c) aspiration of stomach contents
 - d) none of the above

- 12) The three factors that are "Virchow's triad" are:
 - a) blood stasis
 - b) blood coagulation abnormalities
 - c) vessel wall abnormalities
 - d) all of the above

- 13) Pulmonary hypertension results in:
 - a) right heart failure
 - b) hypoxemia
 - c) pulmonary hypertension
 - d) lactic acid production

Expiration date: The last day that post tests will be accepted for this edition is **December 31, 2017**—your envelope must be postmarked on or before that day.

Evaluation: Pulmonary Critical Care Primer

Please complete the evaluation form below by placing an "X" in the box that best fits your evaluation of this educational activity. Completion of this form is required to successfully complete the activity and be awarded contact hours.

At the end of this home study program, I am able to:	Strongly Agree	Agree	Neutral	Disagree	Strongly Disagree
1. Define the process of oxygenation and ventilation.					
2. Identify acid-base disturbances based on blood gas analysis.					
3. Review oxygenation and ventilation modalities used for the critically ill patient.					
4. Differentiate between the pathophysiologies of asthma, bronchitis, and emphysema.					
5. Discuss the pathophysiology of pulmonary embolism, pneumonia, and acute respiratory distress syndrome (ARDS).					
6. The teaching / learning resources were effective. <i>If not, please comment:</i>					

The following were disclosed in writing prior to, or at the start of, this educational activity (please refer to the first 2 pages of the booklet).

	Yes	No
7. Notice of requirements for successful completion, including purpose and objectives		
8. Conflict of interest		
9. Disclosure of relevant financial relationships and mechanism to identify and resolve conflicts of interest		
10. Sponsorship or commercial support		
11. Non-endorsement of products		
12. Off-label use		
13. Expiration Date for Awarding Contact Hours		
14. Did you, as a participant, notice any bias in this educational activity that was not previously disclosed? <i>If yes, please describe the nature of the bias:</i>		

15. How long did it take you to read this home study and complete the post test and evaluation:
 _____ hours and _____ minutes.

16. Did you feel that the number of contact hours offered for this educational activity was appropriate for the amount of time you spent on it?

- Yes
 No, more contact hours should have been offered
 No, fewer contact hours should have been offered.

Expiration date: December 31, 2017