

Respiratory Emergencies

**EMS Continuing Education
Technician through Technician-Advanced Paramedic**

**Consistent with the
National Occupational Competency Profiles
as developed by
Paramedic Association of Canada
and
“An Alternate Route to Maintenance of Licensure”
as developed by Manitoba Health**

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Educational Subcommittee – Paramedic Association
of Manitoba**

Revised August 2009

Disclaimer

These documents were developed for improved accessibility to standardized continuing education for all paramedics in Manitoba.

This training package is consistent with the National Occupational Competency Profiles and the core competency requirements (both mandatory and optional) as identified in “An Alternative Route to Maintenance of Licensure” (ARML). It is not the intent that this package be used as a stand-alone teaching tool. It is understood that the user has prior learning in this subject area, and that this document is strictly for supplemental continuing medical education. To this end, the Paramedic Association of Manitoba assumes no responsibility for the completeness of information contained within this package.

It is neither the intent of this package to supersede local or provincial protocols, nor to assume responsibility for patient care issues pertaining to the information found herein. Always follow local or provincial guidelines in the care and treatment of any patient.

This package can be used in conjunction with accepted models for education delivery and assessment as outlined in “An Alternative Route to Maintenance of Licensure”. Any individual paramedics wishing to use these continuing education packages to augment their ARML program should contact their local EMS Director.

This document was designed to encompass all licensed training levels in the province (Technician, Technician – Paramedic, Technician – Advanced Paramedic.). Paramedics are encouraged to read beyond their training levels. However, it is suggested that the accompanying written test only be administered at the paramedic’s current level of practice.

This package has been reviewed by the Paramedic Association of Manitoba’s Educational Subcommittee and is subject to review by physician(s) or expert(s) in the field for content.

As the industry of EMS is as dynamic as individual patient care, the profession is constantly evolving to deliver enhanced patient care through education and standards. The Paramedic Association of Manitoba would like to thank those practitioners instrumental in the creation, distribution, and maintenance of these packages. Through your efforts, our patient care improves.

This document will be amended in as timely a manner as possible to reflect changes to the National Occupational Competency Profiles, provincial protocols/Emergency Treatment Guidelines, or the Cognitive Elements outlined in the Alternate Route document.

Any comments, suggestions, errors, omissions, or questions regarding this document may be referred to info@paramedicsofmanitoba.ca , attention Director of Education and Standards.

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RESPIRATORY EMERGENCIES

Introduction

This module is to be used in conjunction with the Airway Management package which presents the anatomy and physiology of the respiratory system. This package will review the pathophysiology of illnesses primarily involving the respiratory system. Assessments and management of patients with respiratory illness involving the respiratory system will also be discussed. A section on pharmacological agents is covered at the end of the package.

Any comments or suggestions related to this document can be forwarded to the Director of Education and Standards, Paramedic Association of Manitoba, Inc. via e-mail to info@paramedicsofmanitoba.ca.

Conventions Used in this Manual

Black lettering without a border is used to denote information appropriate to the Technician Level and above.

|| Text with the single striped border on the left is information appropriate to Technician - Paramedic and above.

||| Text with the double striped border on the left is information appropriate to Technician – Advanced Paramedic.

The Respiratory System

The respiratory system allows for the exchange of gases between the body and the environment. The structures of the respiratory system include the following: nasopharynx, oropharynx, pharynx, larynx, trachea, vocal cords, the main stem bronchi, bronchioles, alveoli, which make up the lungs, the visceral and parietal pleura, diaphragm and intercostal muscles..

The **nasopharynx** includes the nasal cavity and its structures. The nasopharynx serves as a passageway for air and provides a resonating chamber for speech sounds. The nose is lined by skin containing coarse hairs, called cilia that filter out dust particles. The nasopharynx also warms and moistens the air before air enters the lungs.

The **oropharynx** is the back of the throat. It is the passage way for food and water, and also a passageway for air, therefore, playing a role in the resonance of our speech.

The nasopharynx and oropharynx can be collectively referred to as the pharynx.

The next space is called the laryngopharynx, which is the space between the pharynx and the larynx. It includes the epiglottis, and vallecula. Often this space for simplicity, is simply referred to as the pharynx.

The **larynx** or voice box is a short passageway that connects the pharynx with the trachea. The larynx contains the **vocal cords**. When air is directed against the vocal cords, they vibrate and produce sound waves in the column of air.

The **trachea** or windpipe is a tubular passageway for air. It is located in front of the esophagus and divides into right and left bronchi. The trachea also filters dust from the air we breathe. The trachea divides into the right and left **main stem bronchi**, leading to the right and left lungs respectively. The main stem bronchi divide into smaller structures called **bronchioli**. The bronchioli resemble a tree trunk with its branches. This is commonly referred to as the bronchial tree.

The **diaphragm** is a muscular membrane which separates the abdominal and thoracic cavities. The diaphragm flattens as it contracts during inhalation, which enlarges the chest cavity and allows for expansion of the lungs.

Intercostal muscles are the muscles found between the ribs. On inspiration the intercostal muscles contract and elevate and rotate the ribs, thus expanding the thoracic cavity. On expiration the intercostal muscles relax, allowing the ribs to return to their resting position thus reducing the volume of the thoracic cavity.

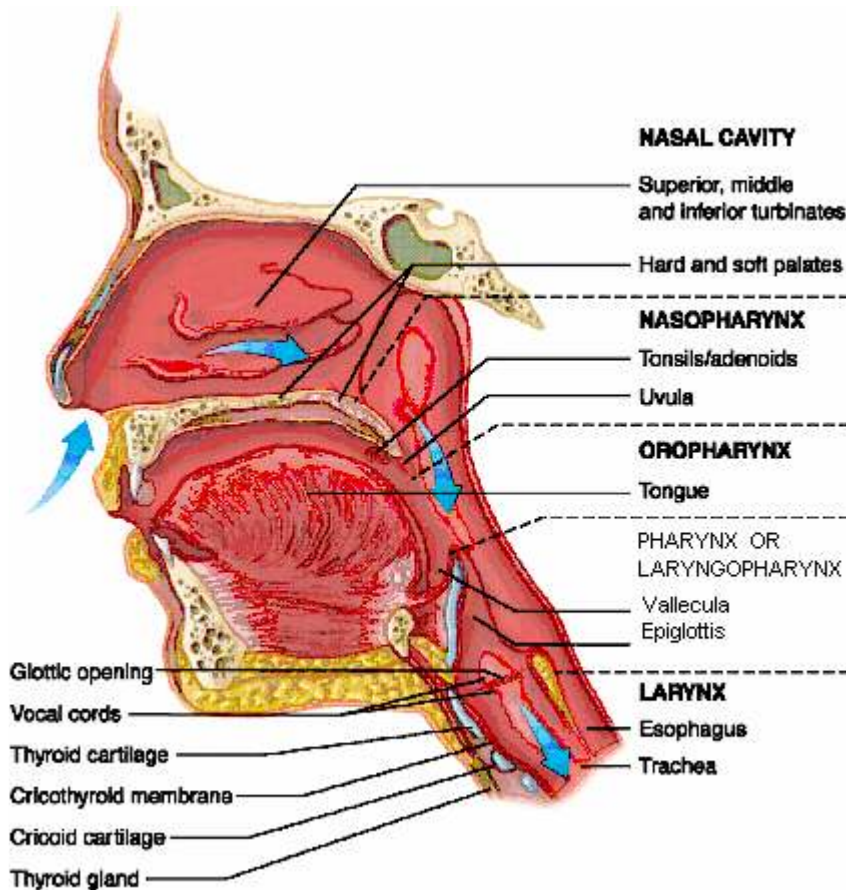
The **lungs** are the main organs of respiration. They supply the blood with oxygen inhaled from the outside air and dispose of waste carbon dioxide in the exhaled air. They are

large spongy organs positioned within the thoracic cavity on either side of the heart. The lungs are divided into lobes, the left having two lobes and the right having three.

The lungs are composed of elastic tissue filled with networks of tubes and sacs carrying air, and the blood vessels carrying blood. Within the lungs, the bronchi branch into smaller structures called bronchioles, which continue to branch into smaller and smaller tubes which end in clusters of air sacs called **alveoli**. The alveoli are surrounded by a network of capillaries where the air and blood make their exchange of oxygen and carbon dioxide.

Each lung is covered by the **pleura**. These are two membranes that envelope and protect the lungs. The **visceral pleura** is firmly attached to the lungs. The **parietal pleura** lines the chest wall and covers the diaphragm. Between the visceral and parietal pleura is a small potential space called the **pleural cavity**, which contains a lubricating fluid. The fluid prevents friction between the membranes and allows them to move easily over each other during breathing.

Air or fluid may enter the pleural cavity forming a space between the pleural membranes. Air or fluid in the pleural cavity results in collapse of the affected lung and decreased functioning of the lung.



The Respiratory Process

There are two types of respiration, internal and external. **Internal respiration** is the exchange of gases between the blood and the cells. **External respiration** is the exchange of gases between the lungs and the blood. Gas exchange occurs across a pressure gradient. In external respiration, oxygen in the lungs is at a higher pressure than in the blood. This results in oxygen entering the blood. Carbon dioxide is at a higher pressure in the blood and as a result, carbon dioxide enters the alveoli. The same process occurs at the cellular level for internal respiration.

The partial pressure of a gas is the pressure each gas in a mixture exerts, as if all other gases were not present. This is very important in the body in determining the movement of oxygen and carbon dioxide between the atmosphere and lungs, the lungs and blood, and the blood and the body cells. The exchange of oxygen and carbon dioxide occurs when each gas moves from an area where its partial pressure is greater, to an area where its partial pressure is less. Each gas is independent and behaves as if the other gases do not exist.

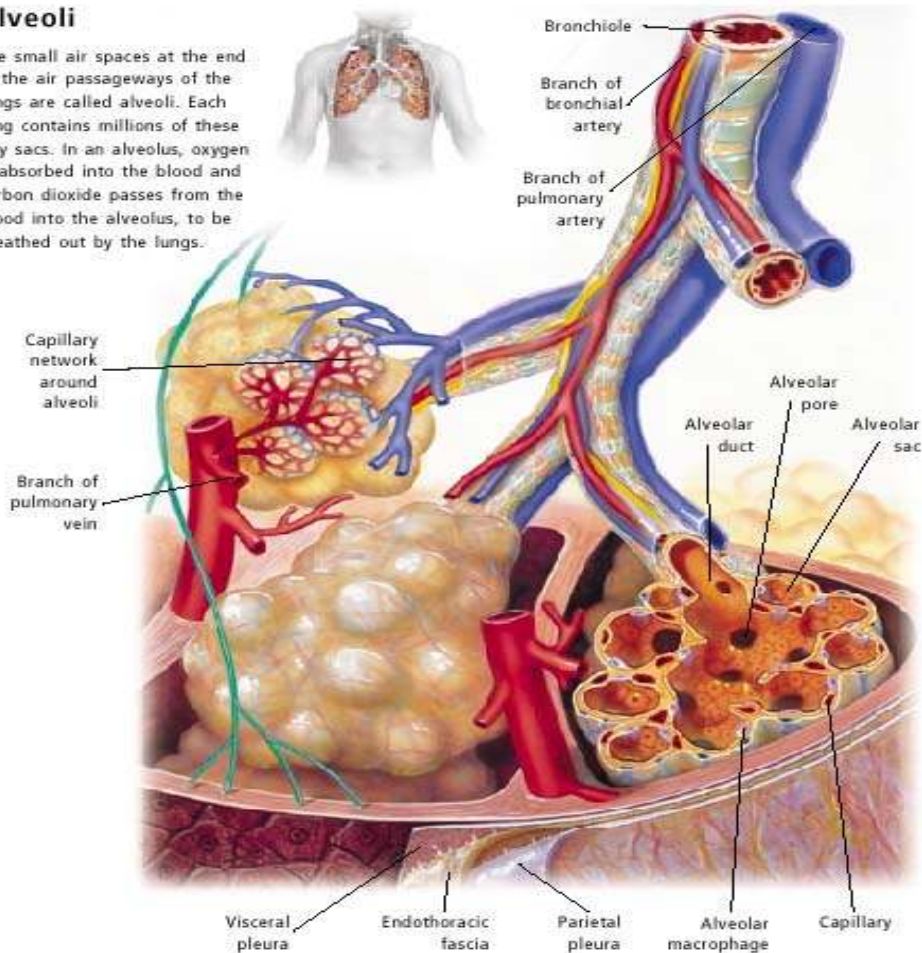
Inspiration is the process of breathing in or inhaling. **Exhalation** is the process of breathing out or exhaling.

For inspiration to occur, the pressure inside the lungs must be lower than the pressure in the atmosphere. Inspiration occurs by contraction of the diaphragm and intercostal muscles, which moves the diaphragm downward in the thoracic cavity at the same time the ribs expand and move upward. This increases the volume capacity of the lungs and lowers the pressure inside the lungs. The result is that air rushes into the lungs from the atmosphere, and an inspiration takes place.

For expiration to occur, the pressure inside the lungs must be higher than the pressure in the atmosphere. Expiration occurs when the diaphragm and intercostal muscles relax, moving the diaphragm upward in the thoracic cavity and the ribs return to their resting position thus decreasing the volume capacity of the lungs. This increases the pressure inside the lungs. The result is that air moves to an area of lower pressure, the atmosphere, and expiration takes place.

Alveoli

The small air spaces at the end of the air passageways of the lungs are called alveoli. Each lung contains millions of these tiny sacs. In an alveolus, oxygen is absorbed into the blood and carbon dioxide passes from the blood into the alveolus, to be breathed out by the lungs.



Costal breathing is shallow or chest breathing. It consists of an upward and outward movement of the chest as a result of contraction of the intercostal muscles. Diaphragmatic breathing is deep or abdominal breathing. It consists of the outward movement of the abdomen as a result of the contraction of the diaphragm.

Respiration is controlled by nerve centers located in the brain stem. These centers cause respiratory muscles to contract and relax in a regulated pattern. Voluntary control of respiration occurs when one holds one's breath. When this occurs there is a buildup of carbon dioxide in the blood. When the carbon dioxide reaches a certain level, the muscles of inspiration are stimulated, resulting in the intake of air whether or not a person wishes this to occur. This is referred to as involuntary control of respiration.

The respiratory centre of the brain, located in the medulla oblongata, is divided into two regions: the **inspiratory centre** and the **expiratory centre**. These centres, along with the area of the brain called the pons, regulate the rhythm of respiration. Stretch receptors in the lungs are stimulated and inhibited in a cyclical fashion to allow for inspiration and expiration to occur, and preventing the lungs from inflating to the point of bursting.

Assessment of the Respiratory System

Assessment of the respiratory system begins with the primary (initial) assessment followed, when practical, by a thorough secondary assessment (detailed).

Primary (Initial) Assessment

The purpose of the primary (initial) assessment is to identify any immediate threats to the patient's life. First, the airway should be assessed to assure that it is patent. Patients of any age who are responsive and talking or crying have an open (patent) airway, while the presence of snoring or gurgling may indicate potential problems with the airway. Although airway and breathing problems are not the same, their signs and symptoms often overlap.

To check for the adequacy of breathing, observe the patient's ability to speak without difficulty. A patient, who can speak only two or three words before pausing to take breath, is showing a significant sign of respiratory distress. This two-to-three word dyspnea is indicative of severe airway obstruction (a narrowing of the airways caused by trauma or disease) or other breathing problems. The presence of retractions or the use of the accessory muscles of respiration is also a sign of respiratory distress. Nasal flaring and the use of the accessory muscles, is an indication of respiratory distress, often seen in children. Finally, obviously labored breathing is also a sign of airway or respiratory distress.

Patients with altered mental status warrant further evaluation. Feel for movements of air with your hand or cheek. Look for rise and fall with each respiratory cycle. In an adult patient, the respiratory rate generally ranges between 12 and 20 breaths per minute. Breathing should be spontaneous and regular. Irregular breathing may suggest a significant problem and often requires some ventilatory support. The chest wall should be observed for any area of asymmetrical movement. This condition, known as paradoxical breathing, may suggest flail chest.

If the patient is not breathing, or if you suspect problems with the airway, open the airway by using the head-tilt/chin-lift or jaw-thrust maneuver. If the possibility of trauma exists, use the modified jaw-thrust method instead and maintain stabilization of the cervical spine. If the first breath does not go in, reposition the patient's head and try again. Once the airway is open, re-evaluate the status of breathing. If breathing is adequate, provide supplemental oxygen and assess circulation. If breathing is inadequate, or absent, begin artificial ventilation. When assisting a patient's breathing with a ventilatory device (bag-valve mask), or after placing an airway adjunct (nasopharyngeal airway, oropharyngeal airway, endotracheal tube or combitube), monitor the rise and fall of the chest and auscultate to determine correct usage and placement.

Secondary (Detailed) Assessment

Following completion of the primary (initial) assessment, and correction of any immediate life threats, the secondary (detailed) assessment should be completed.

History

Inquire about recent trauma, food intake, and drinking. Determine whether the onset of the problem was slow or rapid. Ask about allergies and anaphylaxis. If an injury is involved, evaluate the mechanism of injury. Blunt trauma to the neck may have caused a laryngeal injury.

Physical Examination

Begin the physical assessment with inspection. Evaluate the adequacy of breathing. Note any obvious signs of trauma. Assess skin color as an indicator of oxygenation status. Early in respiratory compromise, the sympathetic nervous system will be stimulated to help offset the lack of oxygen. When this happens, the skin will often appear pale and diaphoretic. Cyanosis is another sign of respiratory distress. When oxygen binds with the hemoglobin, the blood appears “bright red.” Deoxygenated hemoglobin is blue and will give skin a bluish color (cyanosis). However, this is not a reliable sign, since severe tissue hypoxia is possible without cyanosis. In fact, cyanosis is considered a late sign of respiratory compromise. When it does appear, cyanosis will usually affect the lips, fingernails and skin. A red skin rash, especially if accompanied by hives, may indicate an allergic reaction. A cherry red skin discoloration may be associated with carbon monoxide poisoning.

Note any decrease or increase in the respiratory rate, one of the earliest indicators of respiratory distress. As mentioned earlier, look for use of the accessory muscles, intercostal retractions, and the use of the abdominal muscles - another indicator of respiratory distress. Also remember that in infants and children, nasal flaring and grunting indicate respiratory distress.

Following inspection, listen for the adequacy of air movement at the mouth and nose. Then listen to the chest with a stethoscope (auscultation). In a pre-hospital setting, the sites to be auscultated include the right and left apex (just beneath the clavicle), the right and left base (eighth or ninth intercostal space, midclavicular line), and the right and left mid-axillary line (fourth or fifth intercostal space, on the lateral side of the chest). There are also six locations on the posterior chest that can be monitored. When the patient's condition permits, the posterior surface is actually preferred over the anterior one. At this location heart sounds do not interfere with auscultation. However, since patients are usually supine during airway management, the anterior and lateral positions usually prove more accessible. Sounds that point to airflow compromise include:

Snoring - results from partial obstruction of the upper airway by the tongue

Gurgling - results from the accumulation of blood, vomitus, or other secretions in the upper airway

Stridor - a harsh, high pitched sound heard on inhalation associated with laryngeal edema or constriction

Quiet - the absence of breath sounds is an ominous finding and indicates a serious problem with the airway, breathing, or both

Rhonchi – a rubbing, wet lung sounds, indicative of water, fluid in the lungs.

When assessing the effectiveness of ventilatory support or the correct placement of an airway adjunct, realize that air movement into the epigastrium may sometimes mimic

breath sounds. Thus, listening to the chest should be only one of several means used to assess air movement. Another method of checking correct placement of an airway adjunct is to auscultate over the epigastrium; it should be silent during ventilation. When providing ventilatory support, watch for signs of gastric distention. This will suggest inadequate hyperextension of the head, undue pressure generated by the ventilatory device, or improper placement of airway adjuncts. Listening over the sternal notch will also confirm the presence of airflow when an endotracheal tube is correctly placed in the trachea.

Finally, palpate. First, feel for the movement of air with the back of your hand or your cheek. If an endotracheal tube is in place, the proximal end can be checked for this movement. Next, palpate the chest for rise and fall. In addition, palpate the chest wall for symmetry, abnormal motion, crepitus and subcutaneous emphysema.

When ventilating with a bag-valve device, gauge airflow into the lungs by noting compliance. Compliance refers to the stiffness or flexibility of the lung tissue, and it is determined by how easily air flows into the lungs. When compliance is good, airflow occurs with minimal resistance. When compliance is poor, ventilation is harder to achieve. Compliance is often poor in diseased lungs and in patients suffering from chest wall injuries or tension pneumothorax. It will decrease when the upper airway is obstructed by the tongue. If a patient shows poor compliance during ventilatory support, look for potential causes. Questions to aid in assessment include:

- Is the airway open?
- Is the head properly extended (non-trauma patients)?
- Is the patient developing tension pneumothorax?
- Is the endotracheal tube occluded?
- Has the endotracheal tube been inadvertently pushed into the right or left mainstem bronchus?

A bag that compresses too quickly or “collapses” should arouse suspicion. It may indicate incorrect placement of the endotracheal tube into the esophagus or a defect in the bag-valve mask device.

Pulse rate abnormalities may also suggest respiratory compromise. Tachycardia usually accompanies hypoxemia in an adult, while bradycardia hints at anoxia (absence or near-absence of oxygen) with imminent cardiac arrest.

Pulse Oximetry

Indications for Use

EMS personnel can use the pulse oximeter as an adjunct to routine patient care. It is not meant to replace any aspect of standard patient assessment. The pulse oximeter is particularly useful in identify unrecognized cardiorespiratory problems that result in inadequate oxygenation and ventilation. An abnormal pulse oximeter reading may identify the need for support of the cardiorespiratory system.

The pulse oximeter provides EMS personnel a continuous monitor of the patient's oxygenation and ventilation status. It is not meant to take the place of frequent, regular assessment of the patient. It can, however, alert EMS personnel to subtle changes in the patient's condition. For example, a decreasing respiratory drive, subtle alteration in level of consciousness, or some other problem with oxygenation or ventilation. It can also identify a change in the effectiveness of any oxygenation or ventilation intervention being made by EMS personnel, such as loss in supplemental oxygen delivery.

EMS personnel recall that oxygen saturation readings are averaged over time (5-15 seconds or more). As a result, oximeter readings will not drop immediately after patient oxygenation and ventilation changes.

Factors Affecting Pulse Oximetry

A number of factors can affect the pulse oximeter's ability to display accurate arterial oxygen saturation levels. While the oximeter may display a reading, it may be unreliable or false under many circumstances.

Pulse oximeters are calibrated using healthy human volunteers as test subjects. The volunteers have normal or near-normal oxygen saturation values, and calibration can be accurate in normal or near-normal oxygen saturation ranges. When saturation values decrease below 70-75%, the values are not considered to be accurate because there are no control values to permit accurate calibration at these low values.

Carbon Monoxide

Carbon monoxide poisoning leads to erroneously normal or near-normal oxygen saturation values. When carbon monoxide is bound to hemoglobin, carboxyhemoglobin is the result. Carboxyhemoglobin does not bind oxygen and does not carry any oxygen to the cells or tissues. However, carboxyhemoglobin absorbs the same wavelength of light as does oxyhemoglobin. The photodetector cannot distinguish between oxyhemoglobin and carboxyhemoglobin. The result is a falsely normal oxygen saturation reading yet the body's cells and tissues are not receiving any oxygen.

Poor Peripheral Perfusion

Poor peripheral perfusion creates problems for the pulse oximeter. When peripheral perfusion is poor, inadequate amounts of fresh oxygenated arterial blood reach the small blood vessels in tissue beds. The pulse oximeter is unable to distinguish between freshly oxygenated arterial blood and the venous blood. Typically, the pulse oximeter will display an erratic reading or fail to give a reading at all. In order to get a reliable reading, the underlying cause of poor peripheral perfusion should be treated.

Hypothermia

Hypothermia impedes the pulse oximeter's ability to obtain an accurate reading because there is limited or no blood flow to the peripheral circulation. When the body is cold, peripheral vasoconstriction takes place. This limits the amount of freshly oxygenated arterial blood reaching small blood vessels in tissue beds. Again, the pulse oximeter reading will be erratic or fail to give a reading at all. Repositioning the probe to a more central site (earlobe) may result in a more accurate reading.

Hypovolemia

Hypovolemic shock also causes difficulties for the pulse oximeter, and can lead to misleading oxygen saturation values. The red blood cells are responsible for carrying oxygen to the body's cells and tissues. When there is significant blood loss, the body loses red blood cells. This decreases the total amount of oxygen that can be carried to the body's cells and tissues. The pulse oximeter may display a normal oxygen saturation because the red blood cells are well saturated with oxygen, but there are fewer red blood cells to carry oxygen to the cells and tissues. The net result is a decrease in total oxygen delivery to the body's cells and tissues.

Anemia

Anemia can also lead to misleading oxygen saturation values. Like hypovolemic patients, patients with anemia have fewer red blood cells to carry oxygen to the body's cells and tissues. Even though each red blood cell is near fully saturated with oxygen, there is an overall decrease in total oxygen delivery to the body's cells and tissues.

CPR

The poor or absent perfusion associated with cardiopulmonary resuscitation (CPR) results in unreliable pulse oximeter readings. A pulse oximeter has no place in a cardiac arrest.

Bright Light

The pulse oximeter's photodetector is sensitive to certain wavelengths of light. Bright light sources or strong ambient light can lead to inaccurate pulse oximeter readings. EMS personnel can minimize or avoid this source of error by covering the probe with an opaque material (towel, blanket).

Pigments and Dyes

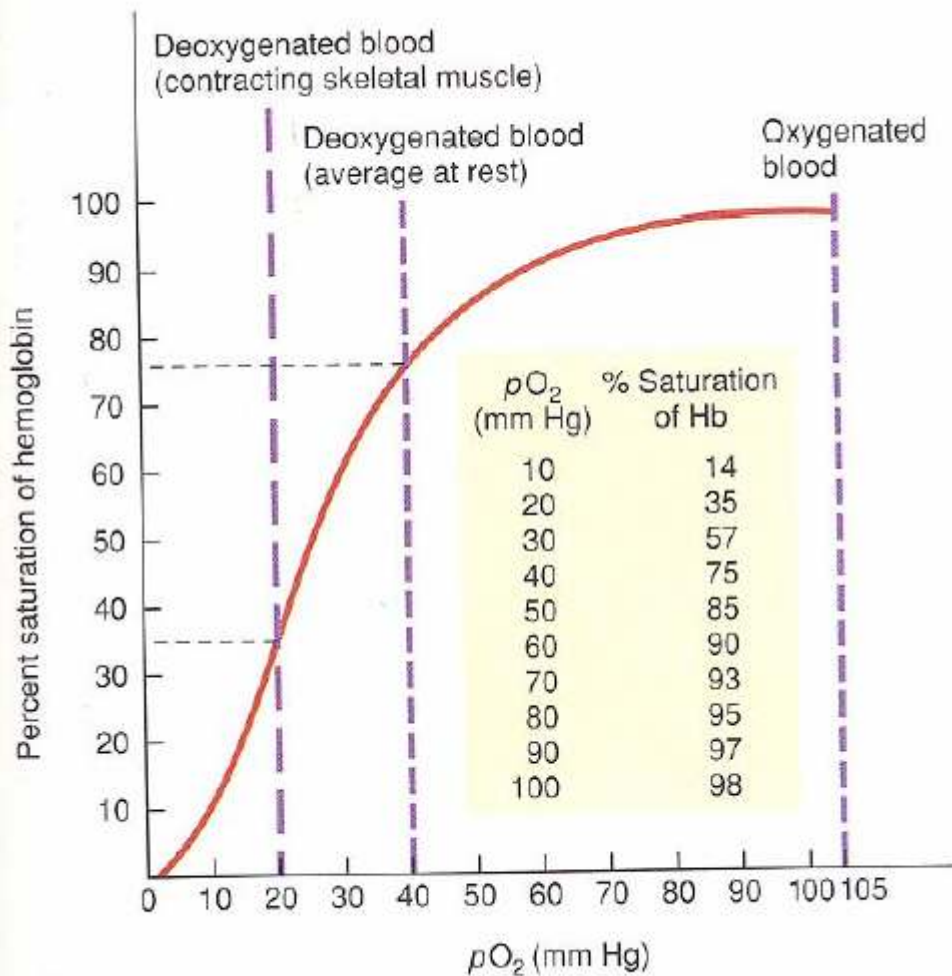
Pigments and dyes can interfere with the probes transmission and detection of light. The resulting decrease in the pulse oximeter's signal quality affects the accuracy of the value displayed. Examples of pigments or dyes include nail polish, skin dyes, tattoos, burns, bruises, hematomas, or any other discoloration of the skin or finger nails. If nail polish is a problem, it should be removed or the probe should be relocated to an alternate site.

As a final note, pulse oximeters may take several minutes to indicate a potentially life-threatening drop in oxygen saturation after the patient has been breathing 100% oxygen. If the patient were to stop breathing, the oxygen saturation would not drop for several minutes. The patient's signs and symptoms in conjunction with vital signs must be used as the primary tools for patient assessment.

The pulse oximeter is only an adjunct monitoring device. It does not take the place of the EMS personnel's ongoing and repeated assessment of a patient.

Oxygen Saturation

When a hemoglobin molecule is carrying its full complement of 4 oxygen molecules, that hemoglobin molecule is fully saturated. If a very large number of hemoglobin molecules are considered as a group, we can determine the overall oxygen saturation in that sample of blood. Under normal circumstances, a healthy person with no cardiac or respiratory compromise will have oxygen molecules bound to 97-99% of all potential oxygen binding sites on the hemoglobin molecules. This average is referred to as the "oxygen saturation". In other words, 97-99% of all oxygen sites on the hemoglobin molecules have oxygen bound to them. The relationship between the oxygen saturation and the partial pressure of oxygen is illustrated by the **oxygen-hemoglobin dissociation curve**. Note that the curve is not linear. When the partial pressure of oxygen is high, hemoglobin binds with large amounts of oxygen and is almost fully saturated. When the partial pressure is low, hemoglobin is only partially saturated and oxygen is released from hemoglobin. At partial pressures between 60 and 100 mm Hg, hemoglobin is 90% or more saturated with oxygen. When the oxygen saturation drops below 90%, the partial pressure of oxygen drops quickly. This non-linear relation between oxygen saturation and partial pressure of oxygen is important to note when interpreting pulse oximeter readings.



Oxygen-hemoglobin dissociation curve at normal body temperature.

Pulse Oximetry

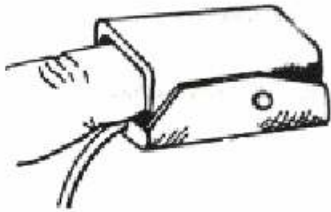
Pulse oximeters measure the oxygen saturation of arterial blood using a light source and a photodetector. The light is absorbed by the tissues beds with each heart beat. The photodetector detects the differences between the light absorbed during systole and diastole.

During systole, there is an increase in light absorption due to newly oxygenated arterial blood. During diastole, the baseline light absorption is due to a mix between venous blood and tissue, blood, and bone. The difference between light absorption in systole and diastole represents the light absorption due to newly oxygenated blood. The pulse oximeter uses the signal from the photodetector to calculate the oxygen saturation. The oximeter is able to detect differences between systole and diastole. Using this information, it can compare the ratio of oxygenated hemoglobin (oxyhemoglobin) and

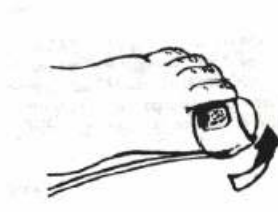
non-oxygenated hemoglobin in the arterial blood alone. The ratio between the two values determines the percent of hemoglobin that is saturated when it reaches the detector. Pulse oximeters typically average their values over 5-15 seconds to prevent rapid fluctuations that occur at the photodetector with each pulse. The oxygen saturation is presented as a numerical value. The pulse oximeter also displays an indication of signal quality received from the photodetector. The quality ranges from good to marginal to inadequate. For successful operation, the signal quality must be good. If the signal is marginal or inadequate for a prolonged period of time, typically 15-30 seconds, the pulse oximeter will not display the oxygen saturation reading. The oximeter sensor combines the light source and the photodetector.

The sensor is typically a cliplike finger probe that attaches to the finger or toe, and is designed to exert the proper amount of pressure to obtain a good signal.

Excessive pressure or taping may result in erroneous readings. Alternate probe designs include a sensor strip that can be taped or attached to a finger or toe, or clips that can be used on the earlobe.



Finger Probe



Toe Probe (adult)



Toe Probe (neonate)

Use of a Pulse Oximeter

The pulse oximeter is a useful adjunct to identify hypoxia. Nevertheless, its use should be deferred if resources are limited or there are more urgent assessment and care priorities that require prompt management. The pulse oximeter does play an important role in patients with cardiorespiratory complaints. Pulse oximetry is needed to determine when high concentration oxygen by mask is sufficient or if assisted ventilation is also required. The decision to provide assisted ventilation is dependent primarily on clinical factors, but a low oxygen saturation measurement suggests it may be required. When assisted ventilations are provided, the pulse oximeter can be used to judge the effectiveness of oxygenation and ventilation provided. EMS personnel are reminded to review the applicable Emergency Treatment Guidelines regarding ventilatory assistance.

Steps in pulse oximeter use:

1. set up the pulse oximeter

- place oximeter where the display can be observed
- ensure the pulse oximeter is well secured to avoid it being dropped as the patient is moved or transported
- connect the sensor cord to the monitor (if not already connected)
- clip the sensor over a fingertip (or other appropriate location)
- avoid using the thumb because it often causes sensing difficulties due to its size
- in patients with poor peripheral perfusion, the earlobe or bridge of the nose may be used suitable probes are available
- in infants, the toe or lateral aspect of the foot may be used

2. initiate monitoring of oxygen saturation

- turn the pulse oximeter on
- when the sensor is positioned properly and there is adequate perfusion to the site being monitored, the signal quality indicator should display “good” (or similar)
- if the sensor is not properly located over the vascular bed or there is inadequate perfusion, the signal quality indicator will indicate an inadequate signal.
Reposition the sensor until the display indicates good sensing

3. interpret pulse oximeter readings

- once proper sensing has been confirmed, there will be a delay of 3 – 6 seconds before the pulse rate and oxygen saturation are displayed
- as long as the sensor remains properly placed, readings will be displayed
- the pulse oximeter will display readings that are averaged over 5 – 15
- seconds
- any rapid change in oxygen saturation will take at least this long to register and be displayed

4. equipment malfunctions

- most pulse oximeters are designed to provide either no reading or an unintelligible reading if any component fails. While this reduces the chance that an unrecognized malfunction will cause inaccurate readings, it does not eliminate the possibility
- pulse oximeter readings should be compared regularly with a reference or other reading to ensure proper functioning. This can be done by comparing the readings obtained with those obtained by another pulse oximeter
- pulse oximeters should be checked after they are dropped, damaged, found to provide inconsistent readings, or according to manufacturer’s recommendations

5. documentation

- if a pulse oximeter is used, its results must be documented on the patient care report.
- the following is the mandatory minimum information that must be recorded every time a pulse oximeter is used:
 - initial reading
 - any improved or deteriorating value during treatment and transport

- any change in patient management based on pulse oximeter readings
- any pulse oximeter difficulties or malfunction

Oxygen Saturation Values

Oxygen Saturation	Interpretation	Management
97 – 100%	Normal	Oxygen should be administered if patient complains of dyspnea or exhibits signs and symptoms of shock
92 – 96%	Low	May require supplemental oxygen via nasal cannula or simple mask – patient should be monitored closely to determine whether oxygen saturation improves
<92%	Very Low	Oxygen is to be administered via non-rebreathe mask – patient may require manual assistance of ventilation using high-flow oxygen

Respiratory Illnesses & Emergencies

Chronic Obstructive Pulmonary Disease (C.O.P.D.)

A term that refers to a group of conditions associated with chronic obstruction of air flow entering or leaving the lungs.

C.O.P.D. includes Bronchitis, Emphysema and Asthma

Chronic obstructive pulmonary disease is the end of a slow process, which over several years results in disruption of the airways, the alveoli, and the pulmonary blood vessels. The process itself may be the result of direct lung and airway damage from repeated infections or inhalation of toxic agents.

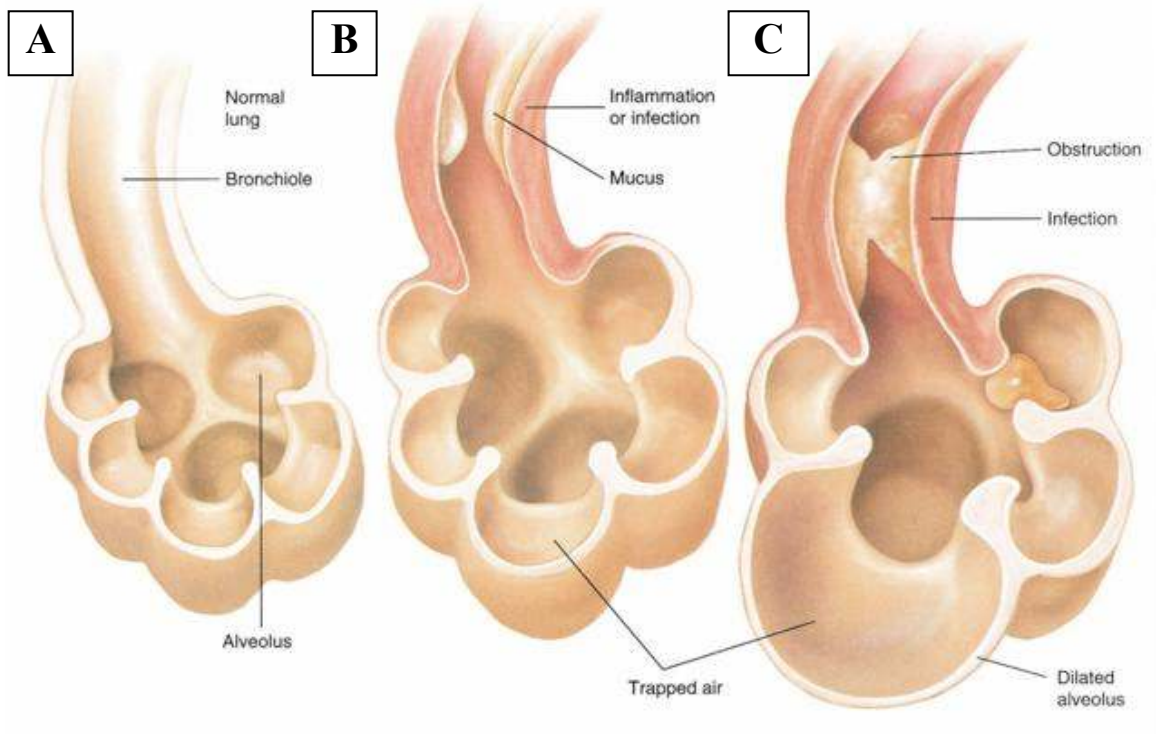
Causes

- smoking
- pollution
- allergies
- infection
- aging
- diseases affecting the lung tissue

Tobacco smoke is itself a bronchial irritant and can create a chronic **bronchitis**, an ongoing irritation of the trachea and bronchi.

With bronchitis, excess mucus is constantly produced, obstructing small airways and alveoli. Protective cells and lung mechanisms that remove foreign particles are destroyed, further weakening the airways. Chronic oxygenation problems can also lead to right heart failure and fluid retention, such as edema in the legs. Pneumonia develops easily when the passages are persistently obstructed. Ultimately, repeated episodes of irritation and pneumonia cause scarring in the lung and some dilation of the obstructed alveoli, leading to COPD.

Emphysema is a loss of the elastic material around the air spaces as a result of chronic stretching of the alveoli when bronchitic airways obstruct easy expulsion of gases. Smoking can also directly destroy the elasticity of the lung tissue. Normally, lungs act like a spongy balloon that is inflated; once they are inflated, they will naturally recoil because of their elastic nature, expelling gas rapidly. However, when they are constantly obstructed or when the “balloons” elasticity is diminished, air is no longer expelled rapidly, and the walls of the alveoli eventually fall apart, leaving large “holes” in the lung that resemble a large air pocket or cavity. This condition is called emphysema.



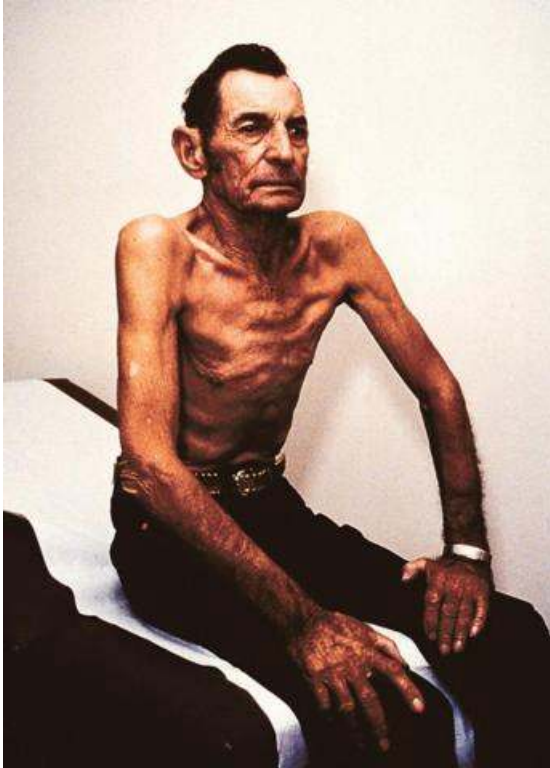
Repeated episodes of irritation and inflammation in the alveoli result in obstruction, scarring, and some dilation of the alveolar sac A: Normal Alveolus. B: Infection produces mucus and swelling. C: A mucus plug creates an obstruction and further dilation of the alveolus.

Most patients with COPD have elements of both bronchitis and emphysema. Some patients will have more elements of one condition than the other; few patients will have only emphysema or bronchitis. Therefore, most patients with COPD will chronically produce sputum, have a chronic cough, and have difficulty expelling air from their lungs, with long expiration phases and wheezing.

Patients with COPD cannot handle pulmonary infections well, because the existing airway damage makes them unable to cough up the mucus or sputum produced by the infection. The chronic airway obstruction makes it difficult to breathe deeply enough to clear the lungs. Gradually, the arterial oxygen level falls, and the carbon dioxide level rises. If a new infection of the lung occurs in a patient with COPD, the arterial oxygen level may fall rapidly. In a few patients, the carbon dioxide level may rise high enough to cause sleepiness. These patients require respiratory support and careful administration of oxygen.

Patients with COPD usually are older than age 50 years. They will always have a history of recurring lung problems and almost always long-term cigarette smokers. Patients with COPD may complain of tightness in the chest and constant fatigue. Because air has been gradually and continuously trapped in their lungs in increasing amounts, their chests often have a barrel-like appearance. If you listen to the chest with a stethoscope, you will hear abnormal breath sounds. These may include **rales**, which are crackling, rattling sounds that are usually associated with fluid in the lungs but here are related to chronic scarring of small airways. **Rhonchi**, which are coarse gravelly sounds, and high-pitched,

whistling **wheezes**, which are expiratory sounds common to patients with asthma, may be heard as well. Because of large emphysematous air pockets and diminished airflow, sounds of breathing are frequently hard to hear and may be detected only high up on the posterior chest.



Typically, a patient with COPD has a barrel-shaped chest and uses accessory muscles and pursed lips for breathing. Notice, also, that the patient is sitting in the tripod position.

The patient with COPD usually presents with a long history of dyspnea with a sudden increase in shortness of breath. There is rarely a history of chest pain. More often, the patient will remember having had a recent “chest cold” with fever and either inability to cough up mucus or a sudden increase in sputum, it will be thick and is often green or yellow (pneumonia). The blood pressure of patients with COPD is normal; however, the pulse, is rapid and occasionally irregular. Pay particular attention to the respirations. They may be rapid, or they may be very slow, as in carbon dioxide retention.

Signs and Symptoms

A number of C.O.P.D. patients are prone to respiratory failure. Such changes are usually due to a respiratory infection and seen first as a change in sputum, that is, a possible increase in volume or more purulence. Dyspnea increases leading to increased hypoxia and subsequent cyanosis, confusion and agitation.

Management is aimed primarily at relieving hypoxia. Because of their chronic respiratory insufficiency, some patients with COPD may breathe on a hypoxic drive. Hypoxia means low oxygen levels. A hypoxic drive means that the stimulus to breathe is

low oxygen levels in the blood. The normal stimulus to breathe in healthy individuals is a high carbon dioxide level.

The patient with COPD has an inability to clear much of the carbon dioxide and will therefore have higher levels in the blood all the time. Some of these patients will adapt to that condition and begin to breathe on the basis of the oxygen levels instead, or with a hypoxic drive. Although this occurs very rarely, Paramedics should be aware of the possibility of its presence.

If a patient who breathes with a hypoxic drive is given 100% oxygen, the body may see the additional oxygen as an overabundance and decrease the respiratory efforts, sometimes even allowing breathing to stop altogether. Paramedics must therefore pay very close attention to the respiration of the COPD patient who is breathing 100% oxygen and assist the patient's respiration if needed. This is not a justification to deprive the patient of O₂ therapy, for without it he may die.

Emergency Care

- establish ABC's - assist ventilations if required
- position patient - sitting and loosen restrictive clothing
- give 100% O₂
- Patients with minimal signs or symptoms of respiratory distress
 - supplemental oxygen can be started at a low concentration via nasal cannula
 - supplemental oxygen can be increased based on the patient's response
 - initiate transport
- Patients with marked signs of respiratory distress or hypoxia
 - high flow oxygen should be administered via non-rebreathe mask
 - load and go should be initiated
- Patients with severe respiratory distress or respiratory arrest
 - Assist ventilations with bag-valve-mask and high flow oxygen
 - Advanced airway support (within scope of practice) should be initiated prior to transport
 - Load and go should be initiated immediately
- take vitals and get history
- reassure patient
- maintain O₂ therapy and monitor respirations, rate and depth being alert for respiratory depression and required ventilation assistance
- repeat vital signs
- transport

Bronchial Asthma

The acute asthma attack may be brought on by an allergic reaction to an inhaled substance, or emotional stress, or by a respiratory infection. The airway of an asthma patient may become blocked by bronchospasm, swelling of mucous membranes and plugging of bronchi by mucous secretions. The end result is that the patient's chest fills with trapped air and exhalation is difficult. He will usually be found in a sitting position and coughing spasmodically. Wheezing is usually evident and the patient could appear cyanotic in color. These patients are almost always agitated and fearful of suffocation.

- characterized by increased sensitivity of the trachea, bronchi, and bronchioles to various stimuli, resulting in bronchospasm.
- acute asthmatic attack shows airway obstruction due to :
 - bronchospasm
 - swelling of mucus membranes in bronchial walls
 - plugging of bronchi by thick mucus secretions

Causes:

- exposure to irritants
- respiratory infection
- drugs
- rapid temperature changes
- exercise
- emotional upsets
- pollutants

Signs and Symptoms

- narrowing of airways and increased mucus producing decreased airflow, especially on expiration

NOTE: all that wheezes is not asthma. Other causes may be CHF, smoke inhalation, chronic bronchitis and acute pulmonary embolism

Emergency Care

- establish ABC's if the patient is in the area where there are irritants known to precipitate an asthma attack, the patient should be removed from the area
- administer oxygen
- if signs of hypoxia are present or the patient is in respiratory distress, high flow oxygen should be administered via non-rebreathe mask
- if the patient is in severe respiratory distress or has stopped breathing, assist ventilations using a bag valve-mask (or advanced airway support if within scope of practice) prior to transport
- load and go should be immediately initiated in this situation
- follow bronchospasm protocol if trained and certified to do so

Metered Dose Inhalers

- if the asthma patient has their own asthma medication in a metered dose inhaler and the patient is in respiratory distress, EMS personnel may assist the patient to use their metered dose inhaler
- identify if the medication was used prior to EMS arrival and what, if any, effects the medication had
- procedure for assisted use
 - ensure the patient is alert and can follow instructions for inhaler use
 - instruct the patient in the process for assisting in medication delivery using the metered dose inhaler
 - ensure the medication is the patient's and has not expired
 - drug is at room temperature or warmer
 - shake the drug canister for 10-15 seconds
 - remove any oxygen delivery device from the patient
 - instruct the patient to hold the inhaler and make sure the patient is holding the inhaler properly
 - instruct the patient to exhale fully
 - instruct the patient to place their lips around the mouth piece of the inhaler
 - instruct the patient to inhale slowly while depressing the medication canister
 - inhalation should be done over approximately 5 seconds
 - remove the inhaler and advise the patient to hold their breath for 10 seconds or as long as comfortable
 - instruct patient to exhale slowly through their mouth
 - replace the oxygen delivery device and encourage the patient to take slow deep breaths
- reassess the patient's respiratory status and vital signs after medication delivery
 - document the medication's effect on relief of respiratory distress
- if the patient requires additional doses, the procedure is repeated
 - repeat doses of medication should be timed at least **two minutes apart** .
 - reassess the patient after every dose of medication
- document all assessments, vital signs, medications taken (drug, doses, route, times), and complications on the patient care report
- if the patient's metered dose inhaler has a spacer attachment
 - instruct the patient in the process that will be followed in assisting the patient to take their medication using the metered dose inhaler with the spacer attachment
 - ensure the spacer is properly attached to the mouth piece of the inhaler
 - remove the protective cap from the spacer if not already removed
 - prepare the patient and medication as for a regular metered dose inhaler

- have the patient exhale fully
- instruct the patient to depress the medication canister to fill the spacer with the medication
- as soon as the medication canister is depressed, have the patient place their lips around the spacer mouth piece of the inhaler
- instruct the patient to slowly inhale while activating the medication canister
 - the inhalation should be done over approximately 5 seconds
 - if the inhalation is too fast, there may be a whistling sound from the spacer
 - if this whistling sound is heard, direct the patient to slow their inhalation further until the whistling sound stops
- remove the inhaler and advise the patient to hold their breath for 10 seconds or as long as comfortable
- instruct patient to exhale slowly through their mouth
- replace the oxygen delivery device and encourage the patient to take slow deep breaths
- repeat doses can be administered as above
- document as above
- take vitals and get history (important information to obtain in history include: have medications been increased in dosage, has patient been intubated before)
- reassure patient
- position patient sitting and loosen restrictive clothing
- maintain O₂ therapy
- monitor vitals
- transport

Status Asthmaticus

Status asthmaticus refers to a severe, prolonged asthma exacerbation that has not been broken with repeated doses of bronchodilators. It may be of sudden onset (resulting from spasm of the airways), or it may be more insidious. Frequently it is precipitated by a viral respiratory infection or prolonged exposure to allergen(s). Status asthmaticus is a true emergency that requires early recognition and immediate transport. These patients are in imminent danger of respiratory failure.

Patient management guidelines for status asthmaticus are the same as those for acute asthma exacerbations, but the urgency of rapid transport is more important. In addition, these patients usually are dehydrated and require IV fluid administration. The patient's respiratory status should be closely monitored, high-concentration oxygen should be administered, and the need for intubation and aggressive ventilatory support should be anticipated (if trained and certified to do so, follow Bronchospasm Protocol).

Pneumonia

Pneumonia remains a leading cause of death. Most pneumonia is caused by viruses or bacteria, but other agents such as fungi, protozoa and chemicals are seen. Pneumonia can occur in any age group and is common in immunosuppressed individuals, such as those with cancer or human immunodeficiency virus (HIV) infection.

Pneumonia is a lung infection that results when there is a failure of the pulmonary defense mechanisms (the cough reflex, mucous clearance mechanisms and the immune system). Conditions that compromise these defenses, such as diabetes, cancer, COPD, sickle cell anemia, spleen removal and cigarette smoking, place patients at increased risk for pneumonia.

Signs and Symptoms

The disease can present in several different ways, but the classical description is fever, chills, and cough. Malaise and chest pain are frequently present, and lower lobe pneumonia can cause upper quadrant abdominal pain. Physical exam may reveal diaphoresis, tachypnea and pulmonary crackles. Use of accessory muscles, nasal flaring and grunting indicate respiratory distress; these patients can progress to respiratory failure.

Management

Patients are usually more comfortable sitting upright. Oxygen therapy by cannula or mask may subjectively improve the patient's condition, and salbutamol (if certified to use) can be effective if there are signs of respiratory distress. Only rarely will the patient progress to respiratory failure and require advanced airway management. The on scene time should be based on the patient's condition, as is the case in most respiratory conditions.

Pulmonary Embolism

Pulmonary embolism is a clot that forms in the deep venous system, usually in the thigh or pelvis, breaks off and travels to the lungs, where it lodges in the pulmonary vasculature. This leads to hypoxemia and an increase workload on the heart. Injury to blood vessels, decreased venous blood flow and alterations in the coagulation system – as can be caused by cancer, all increase the risk of pulmonary embolism. Immobilization, especially when associated with childbirth, congestive heart failure or surgery, can also be a predisposing factor.

Signs and Symptoms

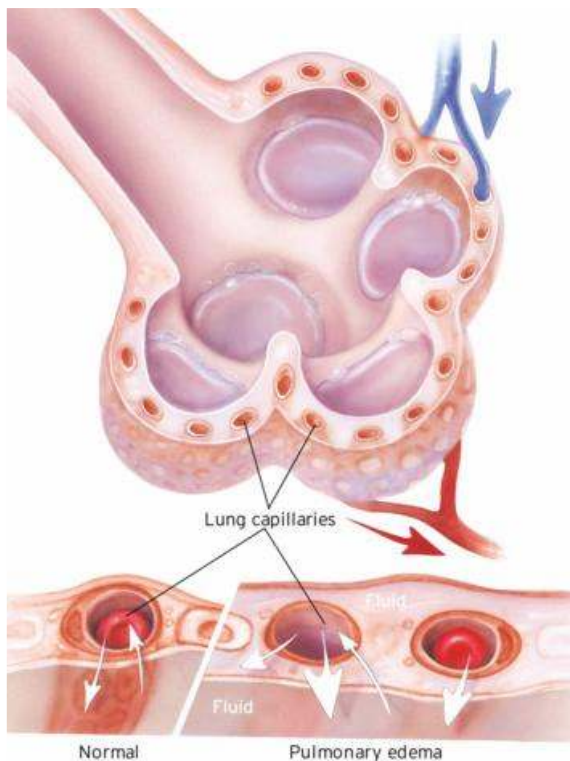
Because pulmonary embolism can present in a variety of ways, it should be considered in any acute, non-specific cardiopulmonary complaint. Acute onset of chest pain, dyspnea, shock and apprehension are the most common symptoms. A pulmonary embolism is difficult to distinguish from myocardial infarction and pneumonia. Physical exam may reveal tachypnea, tachycardia, and pulmonary crackles. Large blockages can present with shock, or even cardiac arrest.

Management

The patient with suspected pulmonary embolus should be transported to the hospital immediately in a semi-reclining position and on high-flow oxygen by non-rebreather mask. A large bore intravenous line with normal saline should be started (if certified to do so) and flow rate adjusted as vital signs dictate. A decompensating patient will require advanced airway management. The possibility of cardiac dysrhythmias secondary to hypoxia necessitates monitoring and treatment as needed.

Acute Pulmonary Edema

Sometimes, the heart muscle is so injured after an acute myocardial infarction or other illness that it cannot circulate blood properly. In these cases, the left side of the heart cannot remove blood from the lung as fast as the right side delivers it. As a result, fluid builds up within the alveoli as well as in the lung tissue between the alveoli and the pulmonary capillaries. This accumulation of fluid, called **pulmonary edema**, can develop quickly after a major heart attack. By physically separating alveoli from pulmonary capillary vessels, the edema interferes with the exchange of carbon dioxide and oxygen. There is not enough room left in the lung for slow, deep breaths.



In pulmonary edema, fluid fills the alveoli and separate the capillaries from the alveolar wall, interfering with the exchange of oxygen and carbon dioxide.

In most cases, patients have a longstanding history of chronic congestive heart failure that can be kept under control with medication. However, acute worsening may occur if the patient stops taking the medication, eats food that is too salty, or has a stressful illness, a new heart attack, or an abnormal heart rhythm. It is not uncommon for a patient to have repeated bouts.

Some patients who have pulmonary edema do not have heart disease. Inhaling large amounts of smoke or toxic chemical fumes can produce pulmonary edema, as can traumatic injuries of the chest. In these cases, fluid collects in alveoli and lung tissue in response to damage to the tissues of the lung or the bronchi.

Signs and Symptoms

Usually develops one to three days after the initial insult. Patients complain of shortness of breath, and develop rapid, labored breathing. Blood oxygen levels fall and the patient becomes cyanotic without intervention.

Assessment

Objective assessment should include vital signs and general observations of appearance. Skin exam may reveal cyanosis or diaphoresis. Lung sounds can include crackles, but their absence does not exclude the diagnosis.

Historical points of importance are the triggering event, the rapidity of disease progression and patient's assessment of dyspnea severity. Past medical history, current medications and drug allergies should be documented.

Management

The patient should be placed in a position of comfort, often semi fowlers given 100% oxygen by non-rebreathe mask. An intravenous line can be started as per stated in the Manitoba Protocol, however this should not delay transport to the hospital. Depending on patient condition, advanced airway management may be needed to maintain oxygenation. Follow Pulmonary Edema and Pulmonary Edema NTG protocol if trained and certified to do so.

Aspiration by Inhalation

Aspiration is the active inhalation of food, a foreign body, or fluid (vomit, saliva, blood, neutral liquids) into the airway. Depending on the type and degree of aspiration, the syndrome may precipitate spasm, mucus production, atelectasis, a change in pH (if the aspirant is acidic), or coughing. The primary means of preventing aspiration is controlling and maintaining the airway; preventing aspiration is controlling and maintaining the airway; prevention is far superior to any known treatment. The paramedic should always be prepared for the possibility of aspiration in patients with a diminished level of consciousness.

Large food particles and other foreign bodies can occlude the airway and cause hypoventilation of distal lung segments. The size of the particle determines which airway is obstructed and to what extent.

About 80% of the approximately 3000 deaths each year from foreign body aspiration occur in children. Running with food or other objects in the mouth, seizures, and forced feeding are among the risk factors in this age group. Hot dogs and peanuts are foods children commonly aspirate. In adults, obstruction may be caused by dental or nasal surgery, unconsciousness, swallowing of poorly chewed food, and alcohol intoxication.

Approximately 60% of foreign bodies are found in the right mainstem bronchus, 19% in the left, and 21 % at the larynx or vocal cords. (Because the left mainstem bronchus branches from the trachea at a 45- to 60-degree angle, foreign body occlusion of this bronchus is less likely than of the right mainstem bronchus, which is shorter, wider, and more vertical.) When the larynx or trachea is completely obstructed, the victim can die from asphyxiation within minutes.

The average adult stomach has a capacity of 1.4 L and manufactures an additional 1.4 L of gastric juices in each 24-hour period. Hydrochloric acid is manufactured by special cells in the gastric mucosa. With the assistance of a protein-dissolving enzyme (pepsin), this acid helps break down large pieces of food into smaller ones. Vomit contains not only partly digested food particles but also acidic gastric fluid.

Saliva is a watery, slightly acidic fluid secreted in the mouth by the major salivary glands and the smaller salivary glands in the mucous membranes that line the mouth. Saliva contains the digestive enzyme amylase, which helps break down carbohydrates. In addition to this enzyme, saliva contains minerals such as sodium, calcium, and chloride; proteins; mucin (the principal constituent of mucus); urea; white blood cells; debris from the lining of the mouth; and bacteria.

The consequences of aspiration of neutral liquids (liquids that are neither acidic nor basic) are easier to reverse with supportive therapy than the consequences of aspiration of acids or bases. Nonetheless, aspiration of a large volume of neutral liquids also is associated with a high mortality rate.

Pathophysiology of Aspiration

The predisposing conditions associated with a high risk of aspiration are a diminished level of consciousness and mechanical disturbances of the airway and gastrointestinal (GI) tract.

A reduced level of consciousness may be caused by trauma, alcohol or other drug intoxication, a seizure disorder, cardiopulmonary arrest, a cerebrovascular accident, or a CNS dysfunction. The common element of these conditions is depression or loss of the gag reflex, with or without a full stomach.

A common type of mechanical disturbance is iatrogenic (i.e., caused by medical procedures) and involves the use of various devices to control upper airway problems. Examples include removal of certain airway devices (risk of vomiting on removal), placement of a nasogastric tube (the artificial opening through the esophageal sphincter increases the risk of regurgitation and aspiration), and intubation, which requires an adequate seal at the tracheal orifice to prevent aspiration.

Other mechanical disturbances that may lead to a high risk of aspiration include tracheostomy and esophageal motility disorders such as hiatal hernia and esophageal reflux. Other individuals at risk include those with either an ileus or a mechanical bowel obstruction.

The potential for aspiration increases whenever vomiting occurs. Vomiting follows stimulation of the vomiting center of the medulla. This stimulation can result from irritation anywhere along the GI tract, from information passed to the medulla from the frontal lobes of the brain, or from disturbances in the balance mechanism of the inner ear. Once this center is stimulated, the following seven events occur:

1. A deep breath is taken.
2. The hyoid bone and larynx are elevated, opening the pre-esophageal sphincter.
3. The opening of the larynx is closed.
4. The soft palate is elevated, closing the posterior nares.
5. The diaphragm and the abdominal muscles are forcefully contracted, compressing the stomach and increasing the intragastric pressure.
6. The lower esophageal sphincter is relaxed, and stomach contents are propelled into the lower esophagus.
7. If the patient is unconscious or unable to protect the airway, pulmonary aspiration may occur.

Effects of Pulmonary Aspiration

The severity of pulmonary aspiration depends on the pH of the aspirated material, the volume of the aspirate, and if particulate matter (e.g., food) and bacterial contamination are present in the aspirate. It generally is accepted that when the pH level of an aspirate is 2.5 or less, a severe pulmonary response occurs. When the pH is below 1.5, the patient

usually dies. The mortality among patients who aspirate material grossly contaminated (as occurs in bowel obstruction) approaches 100%.

The toxic effects on the lungs of gastric acid (with a pH of less than 2.5) can be equated with those of chemical burns. These are severe injuries that produce pulmonary changes such as destruction of surfactant-producing alveolar cells, alveolar collapse and destruction, and destruction of pulmonary capillaries. The permeability of the capillaries increases with massive flooding of the alveoli and bronchi with fluid. The resulting pulmonary edema creates areas of hypoventilation, shunting, and severe hypoxemia. The massive fluid shift from the intravascular compartment to the lungs also may produce hypovolemia severe enough to require volume replacement.

The risk of pulmonary aspiration can be minimized by continuously monitoring the patient's mental status, properly positioning the patient to allow for drainage of secretions, limiting ventilation pressures to avoid gastric distention, and using suction devices and esophageal or endotracheal (ET) intubation. Airway protection should be provided if the risk of aspiration exists or promptly after an occurrence of aspiration.

Hyperventilation

Hyperventilation - breathing that is deeper and more rapid than usual, may be caused by fear, apprehension, stress or some other emotional response. A hyperventilating person may appear terrified. He may clutch at his chest and complain of suffocating, even though he is exchanging much more air than usual. Dizziness is common, and the patient may complain of numbness or a tingling sensation in the extremities. He may be experiencing stabbing chest pains that increase with respirations. Many hyperventilating people think that they are having a heart attack because of the chest pains. A hyperventilating person is not cyanotic, as might be expected from one in apparent respiratory distress; instead he has a healthy pink color (from the over-oxygenation). A survey of vital signs will indicate rapid breathing and high pulse rate, but normal blood pressure. A hyperventilating person may faint.

Hyperventilation flushes CO₂ from the person's lungs, which causes a reduction of CO₂ in the blood. This induces the respiratory centre to react by increasing the respiratory rate, even though the level of O₂ in the blood is high. The respiratory centre in a healthy individual normally reacts to the level of CO₂ in the blood - e.g. to keep a balance, the respiratory centre will increase or decrease the respiratory rate to either blow off or increase the CO₂ level. Eventually, the decrease of CO₂ and the increase of oxygen reaching the respiratory centre cause the breathing process to slow down. It will eventually stop if the oxygen-carbon dioxide balance is not restored.

The emergency care procedure involves calming the patient and aiding them in restoring normal breathing pattern.

- increased rate and/or depth of respirations
- result in increased O₂ and reduced CO₂ in the blood

Causes:

- fear
- apprehension
- stress

Signs and Symptoms:

- clutching at the chest
- feeling of suffocation
- dizziness
- numbness and tingling in extremities
- chest pain increasing with respirations
- good color - very pink
- agitation
- stiffening and flexion of the fingers

Emergency Care

- assess the patient's respiratory status, including respiratory effort and adequacy of respirations
- oxygen should be administered
- search for the underlying cause
- provide psychological support to assist the patient to control their respiratory rate, if possible
- if there is underlying chronic cardiorespiratory disease, hyperventilation may be a normal response to chronic illness or evidence of decompensation
 - it is not possible for EMS personnel to make this determination
 - be prepared to support ventilation in this situation
 - hyperventilation can be psychological in origin – “psychogenic hyperventilation”
 - patients with this condition often present with a history of precipitating emotional events followed by SOB, lightheadedness, and tingling in the extremities
 - these symptoms are thought to be the result of decreased CO₂ levels
 - patients with “psychogenic hyperventilation” were often told to rebreathe into a paper bag on the theory that this will normalize their CO₂ levels and thus decrease their symptoms but **HAVING THE PATIENT REBREATHE INTO A PAPER BAG IS CONTRAINDICATED IN THE TREATMENT OF THE HYPERVENTILATING PATIENT.** There have been well described cases where patients misdiagnosed as experiencing “psychogenic hyperventilation” have died after rebreathing into a paper bag. Therefore it is impossible for EMS personnel to make the distinction between organic and psychogenic causes of hyperventilation with any certainty
- transport

Adult Respiratory Distress Syndrome

Adult respiratory distress syndrome (ARDS) is a fulminant form of respiratory failure characterized by acute lung inflammation and diffuse alveolar-capillary injury. All disorders that result in ARDS cause severe pulmonary edema. The syndrome develops as a complication of injury or illness such as trauma, gastric aspiration, cardiopulmonary bypass surgery, gram-negative sepsis, multiple blood transfusions, oxygen toxicity, toxic inhalation, drug overdose, pneumonia, and infections. Regardless of the specific cause, increased capillary permeability (high-permeability noncardiogenic pulmonary edema) results in a clinical condition in which the lungs are wet and heavy, congested, hemorrhagic, and stiff, with decreased perfusion capacity across alveolar membranes. As a result, there is a decrease in pulmonary compliance, requiring higher airway pressure for each breath.

The pulmonary edema associated with ARDS leads to severe hypoxemia, intrapulmonary shunting, reduced lung compliance, and, in some cases, irreversible parenchymal lung damage. Unique to this syndrome is that most patients who develop this condition have healthy lungs before the event that caused the disease. ARDS is more common in men than in women and has a mortality rate of over 65%. Complications include respiratory failure, cardiac dysrhythmias, disseminated intravascular coagulation, barotrauma, congestive heart failure, and renal failure.

Management

All patients with ARDS should be given high concentration oxygen and ventilatory support. Depending on the underlying cause of ARDS, prehospital management may include fluid replacement to maintain cardiac output and peripheral perfusion, drug therapy to support mechanical ventilation, the use of pharmacological agents such as corticosteroids to stabilize pulmonary, capillary, and alveolar walls and diuretics. Patients with ARDS usually have tachypnea, labored breathing, and impaired gas exchange 12 to 72 hours after the initial injury or medical crisis. Because the syndrome often is a complication of another illness or injury, the paramedic should consider the pathophysiology of the underlying problem and provide supplemental oxygen and ventilatory support to improve arterial oxygenation (assessed by pulse oximetry).

Pleural Effusion

Pleural effusion is defined as the escape of fluid, normally from the vascular space, into a cavity such as the pleural cavity where as a hemothorax if the accumulation of blood in the pleural space. As the fluid continues to fill the pleural space, the lung on the affected side may collapse, and rarely, the mediastinum may shift away from the effusion, compressing the unaffected lung. The resultant respiratory and circulatory compromises are responsible for the following signs and symptoms:

- Tachypnea
- Dyspnea
- Cyanosis (often not evident in hemorrhagic shock)
- Diminished or decreased breath sounds (dullness on percussion)
- Hypovolemic shock
- Narrowed pulse pressure
- Tracheal deviation to the unaffected side (rare)

Management for these patients is directed at correcting ventilatory and circulatory compromise. This entails administering high concentration O₂, ventilatory support with BVM, intubation, or both, IV therapy (if trained to do so) and rapid transport to hospital.

Pharmacology

Acetylsalicylic Acid (ASA)

Brand Names: Apo-ASA, Asaphen, Aspergum, Aspirin, Children's Aspirin, Coated Aspirin, Entrophen, MSD Enteric Coated ASA, Novasen

Class: Nonsteroidal anti-inflammatory drug (NSAID); analgesic; anticoagulant

Mechanism of Action: Nonsteroidal anti-inflammatory drugs (NSAIDs) such as aspirin inhibit the release of chemicals in the body called prostaglandins, which play a role in inflammation, though it is unknown exactly how they exert their pain-relieving, fever-reducing, and anti-inflammatory effects.

Indications: For mild to moderate everyday pain and inflammation; to reduce fever; to prevent the formation of blood clots, a primary cause of heart attack, stroke, and other circulatory problems; to ease the inflammation, joint pain, and stiffness associated with arthritis.

Contraindications: Salicylate sensitivity, active peptic ulcer and bleeding disorders.

Precautions: Caution with patients that have asthma, congestive heart failure, diabetes mellitus, gout, high blood pressure, kidney disease, liver disease, or thyroid disease.

Adverse Reactions: Vomiting, agitation, extreme fatigue, confusion; allergic reaction causing troubled breathing, redness of face, itching, swelling of face, lips, or eyelids. These are symptoms of Reye's syndrome, a rare but serious disorder that is most likely to affect patients under the age of 16. Seek medical attention immediately. Commonly, stomach upset, rash, nausea, and ringing in the ears. Less common, insomnia.

Drug Interactions: A physician should be consulted before taking ASA if patient is taking a blood pressure medication, a medication for gout, an arthritis drug, an anticoagulant such as warfarin, a diabetes medication, a steroid, or an antiseizure medication.

Dosage and Administration: For pain or fever; 325 to 650 mg every 4 hours as needed. Maximum 4 grams (g) per day. For prevention of blood clots; 80-325 mg daily. With the Prehospital dose being 160 mg. For arthritis, up to 4 g per day in 4 divided doses. ASA should be taken with food, milk, or a full glass of water to minimize gastric irritation.

Duration of Action: For pain relief, up to 4 hours. (Onset of effect = 30 minutes)

Special Considerations: Do not give ASA to children under age 16 unless physician instructs otherwise, since it may cause a very rare but life-threatening condition known as Reye's syndrome. Do not use ASA in last 3 months of pregnancy, or during lactation.

Epinephrine 1:1000

Class: Sympathetic agonist.

Description: Epinephrine is a naturally occurring catecholamine that affects both alpha and beta adrenergic receptors, but its beta effects are more profound.

Mechanism of Action: Some of the effects of epinephrine include: positive inotropic, chronotropic and dromotropic changes, , peripheral vasoconstriction, increased blood pressure, and increased automaticity.

Indications: Many cardiac applications, as well as severe allergic reactions (anaphylaxis) and severe asthma.

Contraindications: A different dosage is required for cardiac arrest emergencies.

Precautions: Epinephrine should be protected from the light.

Side Effects: Palpitations, anxiety, tremors, headache, dizziness, nausea and vomiting can occur with epinephrine administration. Because epinephrine affects both heart rate and contractility, it causes increased myocardial oxygen demand. Therefore, oxygen should be administered in conjunction with epinephrine.

Interactions: The effects of epinephrine can be intensified in patients taking antidepressants and other sympathomimetics.

Dosage: Based on the age of the patient, epinephrine 1:1000 should be administered SQ or IM. between 0.15-0.3 mg every 15 minutes as required for anaphylaxis.

Furosemide

Trade Names: Apo-Furosemide, Furosenmide, Lasix, Novo-Furosemide.

Class: Loop diuretic

Mechanism of Action: Loop diuretics work on a specific portion of the kidney (the loop of Henle) to increase the excretion of water and sodium in urine. It also causes venous dilation. It is extremely useful in the treatment of congestive heart failure and pulmonary edema. Its effects are usually evident within five minutes of administration.

Indications: For congestive heart failure, and pulmonary edema. Furosemide is also sometimes used to help control high blood pressure.

Contraindications: Usage in pregnancy should be limited to life-threatening situations. Furosemide has been known to cause fetal abnormalities.

Adverse Reactions: Furosemide can cause hypotension, ECG changes, chest pain, dry mouth, hypochloremia, hypokalemia, hyponatremia, and hyperglycemia.

Drug Interactions: Furosemide administration may cause potassium depletion potentially resulting in digitalis toxicity, and sodium depletion potentially resulting in lithium toxicity. Consult physician if patient has had an allergy to sulfa drugs. Also consult physician if patient is on antibiotics, blood pressure medications, ACE inhibitors, pain relievers, lithium, cortisone-related drugs, digitalis-related drugs, or any nonsteroidal anti-inflammatory drug (NSAID).

Dosage: For patients not already on Furosemide, the standard single dose of 40 mg IV bolus is given, repeated every 15 minutes to a maximum total dosage of 160 mg. If the patient is already taking an oral dose of Furosemide exceeding 40 mg OD, then the patient can be given one bolus dose equivalent to their daily dose, exceeding 160 mg.

Duration of Action: Tablets and solution: 6 to 8 hours. Injection: 2 hours.

Special Considerations: To prevent sleep disruption, patients should avoid taking furosemide in the evening. Some patients may need to take a potassium supplement or consume foods or fluids high in potassium. Diabetic patients should monitor their blood sugar levels carefully.

Atrovent (Ipratropium)

Class: Anticholinergic.

Description: Parasympatholytic bronchodilator related to atropine.

Mechanism of Action: Causes bronchodilation and dries respiratory tract secretions. Atrovent blocks acetylcholine receptors, thus inhibiting parasympathetic stimulation.

Indications: Bronchial asthma, reversible bronchospasm associated with chronic bronchitis and emphysema.

Contraindications: Not indicated where rapid response is required.

Precautions: Monitor vital signs, auscultate air entry before and after drug administration, and use caution in patients with hypertension and cardiovascular disease.

Side Effects: Palpitations, anxiety, dizziness, headache, nausea and/or vomiting.

Interactions: Few.

Dosage: Generally nebulizer dose is 500 mcg, also available in metered-dose inhaler (20 mcg).

Nitroglycerin (Nitrostat)

Class: Nitrate

Mechanism of Action: Nitroglycerin is a rapid smooth-muscle relaxant that reduces cardiac work, and to a lesser degree, dilates the coronary arteries. This results in increased coronary blood flow and improved perfusion of the ischemic myocardium. Relief of ischemia causes reduction and alleviation of chest pain. Pain relief following nitroglycerin administration usually occurs within 1 to 2 minutes, and therapeutic effects can be observed up to 30 minutes later. Nitroglycerin also causes vasodilation which increases venous capacitance thus leading to a decrease in preload. Decreased preload leads to decreased cardiac work. This mechanism, is thought to be the primary and initial method in which Nitroglycerine benefits patients in Pulmonary Edema. This feature, in conjunction with coronary vasodilation, reverses the effects of angina pectoris.

Indications: Chest pain associated with angina pectoris, myocardial infarction, and acute pulmonary edema (independent of whether the latter is accompanied by ischemic chest pain)

Contraindications: Nitroglycerin is contraindicated in patients whose systolic blood pressure is < 100mmHg. It should not be administered to patients in shock. Nitroglycerin is also contraindicated in patients who have used Sildenafil (Viagra™) in the past 24 hours, or Tadalafil (Cialis™) in the past 72 hours.

Adverse Reactions: Nitroglycerin can cause headache, dizziness, weakness, tachycardia, hypotension, orthostasis, skin rash, dry mouth, nausea, and vomiting.

Drug Interactions: Nitroglycerin can cause severe hypotension when administered to patients who have recently ingested alcohol. It can cause orthostatic hypotension when used in conjunction with beta blockers. **Nitroglycerin can cause severe, catastrophic and sometimes fatal precipitous drops in blood pressure in patients who have recently taken ED (Erectile Dysfunction) medications (see “Contraindications”).**

Dosage and Administration: One tablet (0.3 mg) or one Nitrospray (0.4 mg) sublingually for routine angina pectoris. Patients in Pulmonary Edema. This can be repeated in 3-5 minutes as required. Care should be taken to assure that it is not swallowed. Should the systolic blood pressure drop below 100 mmHg, nitrate administration should be discontinued. In the event of prolonged transport times, additional doses of Nitroglycerine may be administered with orders via physician on-line medical control or by prior expressed written instructions from the Medical Director.. IV nitroglycerin is used in the emergency department and intensive care units, but the sublingual route is adequate for most prehospital situations. Nitroglycerin is also available in patches and in ointment form for transdermal administration.

Special Considerations: Nitroglycerin must be protected from light and air to prevent deterioration.

Ventolin (Salbutamol)

Class: Sympathetic beta 2 agonist.

Description: Sympathomimetic that is selective for beta 2 adrenergic receptors.

Mechanism of Action: Causes prompt bronchodilation with a duration of approximately 5 hours.

Indications: Bronchial asthma, reversible bronchospasm associated with chronic bronchitis and emphysema.

Contraindications: Hypersensitivity to ventolin.

Precautions: Monitor vital signs. Use caution with patients having hypertension or cardiovascular disease. Lung sounds should be auscultated before and after ventolin treatments.

Side-Effects: Palpitations, anxiety, dizziness, headache, skeletal muscle tremors, hypertension, nausea, vomiting.

Interactions: Beta blockers may blunt the effects of ventolin.

Dosage: Metered-dose inhaler 90 mcg, nebulized 2.5 mg in 2.5 ml solvent.

Streptokinase (Streptase)

Class: Fibrinolytic agent

Mechanism of Action: Streptokinase combines with plasminogen to produce an activator complex that converts free plasminogen to the proteolytic enzyme plasmin. The plasmin in turn functions as an enzyme that degrades fibrin threads as well as fibrinogen. Causing lysis of the blood clot. Streptokinase is administered to selected patients with acute evolving myocardial infarctions.

Indications: For acute evolving myocardial infarction, massive pulmonary emboli, arterial thrombosis and embolism, to clear arteriovenous cannulas, and DVT.

Contraindications: Hypersensitivity, active bleeding, recent surgery (within 2-3 weeks), recent CVA, prolonged CPR, intracranial or intraspinal surgery. Recent significant trauma (particularly head trauma), uncontrolled hypertension (systolic pressure equal to or greater than 180 mm Hg; diastolic pressure equal to or greater than 110 mm Hg.)

Adverse Reactions: Bleeding (GI, GU, intracranial, other sites), allergic reactions, hypotension, chest pain, reperfusion dysrhythmias, and abdominal pain.

Drug Interactions: Acetylsalicylic acid may increase risk of bleeding (may also be beneficial in improving overall effectiveness). Heparin and other anticoagulants may increase risk of bleeding as well as improve overall outcome.

Dosage and Administration: Evolving acute myocardial infarction- Adult: 1.5 million U diluted to 45 ml (IV) over 1 hr (use infusion pump). Pediatric: Safety not established.

Onset and Duration: Onset: 10-20 minutes (fibrinolysis, 10-20 min; clot lysis, 60-90 min.) Duration: 3-4 hours (prolonged bleeding times up to 24 hours).

Special Considerations: Pregnancy safety: Category C. Do not administer IM injections to patients receiving fibrinolytic drugs. Obtain blood sample for coagulation studies prior to administration. Carefully monitor vital signs. Observe the patient for bleeding. Use caution when moving patient to avoid bruising or bleeding. Do not draw arterial blood gas specimens in fibrinolytic therapy candidates. Use one IV line exclusively for fibrinolytic administration.

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