SAEMS

ASTHMA / COPD STANDING ORDER

SELF-LEARNING MODULE

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SAEMS ASTHMA/COPD STANDING ORDER TRAINING MODULE

Purpose

This SAEMS Standing Order Training Module has been developed to serve as a template for EMS provider training. The intent is to provide consistent and concise information to all providers practicing within the SAEMS Region. The content of the Training Module has been reviewed by the Protocol Development and Review Sub-Committee, and includes the specific standing order, resource and reference material, and instructions for completing the Training Module to obtain continuing education credit. One hour of SAEMS continuing education credit may be issued following successful completion of the module.

Objectives

Upon completion of this self-learning module the EMS participant will be able to:

1. Review basic respiratory evaluation techniques
2. List the assessment findings for moderate to severe asthma
3. List the assessment findings for COPD
4. Apply the Asthma/COPD Standing Order appropriately

Instructions

1. Watch the Power Point Presentation. Review the accompanying information, Standing Order, and any additional reference material as necessary.
2. Complete the attached posttest and return it to your supervisor or Base Hospital Manager:
3. A SAEMS CE will be issued to providers scoring _____ on the post-test

Table of Contents:

Asthma Disease Overview:

Asthma is a disease that is characterized by hyper-responsiveness (hyper-reactivity) of the airways that is accompanied by episodic periods of bronchospasm (spasms or prolonged contractions of the bronchial smooth muscle). The disease process involves biochemical, immunologic, autonomic, infectious, endocrine as well as psychological factors, which can vary in different individuals.

In sensitive individuals, breathing in allergy-causing substances, called allergens, can trigger asthma symptoms. Allergens can include pet dander, dust mites, molds, pollens or cock roach allergens. Respiratory infections, exercise, cold air, tobacco smoke and other pollutants, stress, food, or drug allergies can also trigger asthma symptoms. Aspirin and other non-steroidal anti-inflammatory medications (NSAIDs) can provoke asthma in some patients.

When an asthma attack occurs, the muscles surrounding the airways become constricted causing the lining of the air passages to swell, thus reducing airflow. This impeded airflow, is related to the distinguishable high-pitched wheezing sound during auscultation of the lung fields. During severe episodes of an attack the wheezing can be audible (heard without the aid of a stethoscope) which can progress to inaudible breath sounds, indicating a lack of air movement.

Some patients have long-term shortness of breath with episodes of increased shortness of breath. Still, in other asthma sufferers, a tight-repetitive cough may be the only distinguishable symptom. Asthma attacks can last for minutes to days and can become dangerous if the airflow becomes severely restricted. As the bronchial tubes become more constricted, less airflow occurs.

Most people with asthma have varying degrees of attacks separated by symptom-free periods. Although symptom free, the individual suffering from asthma has inflammation present.

Asthma is Divided into Two Types: Extrinsic or Intrinsic.

Extrinsic Asthma is the most common form of asthma. Environmental antigens such as dust, pollen, molds, animal dander and foods can trigger extrinsic asthma. The initial encounter with the inciting allergen stimulates plasma cells to produce antigen-specific IgE antibodies that bind to mast cells in the airways. With subsequent exposure to the allergen, IgE antigen binding causes mast cell degranulation and release of inflammatory mediators. The released mediators are histamine, bradykinin; leukotrienes C, D and E; platelet-activating factor; prostaglandins; thromboxane A2; and chemotactic factors for eosinophils, platelets, neutrophils and T lymphocytes. These above referenced mediators initiate an intense inflammatory response of the airways. Eosinophils also play an important role in extrinsic asthma. In addition to releasing inflammatory mediators, eospinophils produce proteins that stop ciliary beating, and disrupt mucosal integrity which causes damage of epithelial cells. Epithial cell damage is linked with airway hyper-responsiveness.

Intrinsic Asthma has no known allergic cause. Attacks usually occur in adults older than 35 years of age and are often severe. Factors known to precipitate intrinsic asthma are respiratory tract infections; drugs (aspirin, beta-adrenegic antagonists); environmental irritants (occupational chemical, air pollution); cold; dry air; exercise and emotional stress. Although the causes have been identified, the mechanisms by which they initiate an asthma attack are not completely understood.

In intrinsic asthma, broncho-constriction occurs within minutes then usually resolves within a short period of time. In some individuals a second episode, called the late reaction, develops 4 to 8 hours after the first episode. The late reaction episodes are less responsive to bronchodilator drugs and subsequently are longer in duration. Eosinophils predominately are primarily responsible for the increased bronchial inflammation.

Status Asthmaticus: If the brochospasm is not reversed by usual measures the individual is considered to have severe bronchospasms or status asthmaticus. With severe bronchospasm, the work of breathing can be 5 to 10 times that of normal. When air trapping is severe, paradoxic pulse (a systolic blood pressure decrease of more than 10 mmHg during inspiration) and pneumothorax are common. If status asthmaticus continues, hypoxemia becomes worse, expiratory flows and volumes decrease further. It is usually at this point that the individual begins to tire. This stage may develop quicker with the pediatric or geriatric patient. Respiratory acidosis develops as arterial PCO2 begins to rise. Asthma becomes life threatening at this point if treatment does not reverse this

process quickly. A silent chest (no audible air movement) and a PCO2 over 70mm Hg are ominous signs.

Pathophysiology

Inflammation resulting in hyper-responsiveness of the airways is the common factor and major pathologic feature of all types of asthma. The release of inflammatory mediators produces smooth muscle spasms, vascular congestion, increased vascular permeability, edema formation, production of thick resolute mucus and impaired mucociliary function. Several mediators also cause thickening of the airway walls and increase contractile response of bronchial smooth muscle. These changes in the bronchial musculature, combined with the epithelial cell damage caused by eosinophil infiltration, bring about the airway hyper-responsiveness which is characteristic of asthma.

Airway obstruction increases resistance to airflow while decreasing flow rates, including the expiratory flow. Impaired expiration causes hyperinflation distal to the obstruction, altering pulmonary mechanics. These changes increase the work of breathing.

Hyperventilation is eventually triggered by lung receptors responding to the increased lung volume and obstruction(s). Continued air trapping increases intrapleural and alveolar gas pressures, which causes decreased perfusion of the alveoli. The result is early hypoxemia without CO2 retention (hypercapnia). The state of hypoxemia further increases hyperventilation through stimulation of the respiratory center, which causes PaCO2 to decrease and pH to increase (respiratory alkalosis). As the obstruction becomes more severe, the number of alveoli being inadequately ventilated and perfused increases CO2 retention and respiratory acidosis develops. The rise in CO2 retention results in respiratory acidosis. Respiratory acidosis signals respiratory failure.

Clinical Signs/Symptoms

During times of remission, individuals are asymptomatic (not showing signs) with pulmonary function tests noted as normal. During partial remission there are no clinical signs but the individual has abnormal pulmonary function tests.

Asthma most often has warning signs that preceed an attack or exacerbation of symptoms. Understanding these warning signs and taking proper preventive care through monitoring of lifestyle as well as medications can go a long way in preventing serious complication, such as dialing 911 for Emergency Medical

Services. The most successful treatment for asthma is the elimination of the causative agent(s).

A patient suffering from asthma might show problems and symptoms with a wide range of frequencies. The patient may have occasional asthma attacks with very mild problems of breathing which normally last for an hour or two and then disappear. This kind of asthma does not require any medications but precautions can be taken in order to avoid them. Sometimes asthma is augmented by bouts of chronic coughing and wheezing which occurs just before or after an attack.

During an attack, the majority of individuals are dyspneic and respiratory effort is increased. Breath sounds are decreased except for wheezing. Arterial blood gases (ABG's) should be measured as the severity of alterations are difficult to evaluate by clinical signs alone.

At the beginning of an attack there is usually a sensation of chest constriction or tightening, inspiratory and expiratory wheezing, dyspnea, nonproductive coughing, prolonged expiration, tachycardia and tachypnea. With severe attacks the accessory muscles of respiration are recruited and when hypoxia is present there is a decrease or change in the patients' level of consciousness depending upon the severity of hypoxemia.

Asthma signs and symptoms can include:

- Coughing (nonproductive). Coughing from asthma is often worse at night or early in the morning, making it hard to sleep.
- Wheezing (inspiratory and expiratory). Wheezing is a whistling or squeaky sound when the patient breathes.
- Chest tightness (constriction). This can feel like something is squeezing or sitting on the patients' chest.
- Shortness of breath (dyspnea). Some people say they can't catch their breath, or they feel breathless or out of breath. The patient may feel as though they can't get enough air in or out of their lungs.
- Faster breathing (tachypnea) or noisy breathing. Hyperventilation can occur.
- Increased pulse (tachycardia)
- Mental Status: is dependant upon the severity of hypoxia present.

These signs and symptoms which might range from mild to life threatening are not always evident or present based upon the patient's co-morbidity(s).

Severe Asthma Symptoms

- Increased Pulse rate above 120 beats per minute
  May be less with equally severe asthma patients
- Increased respiratory rate above 40 (adult)
  Severe asthma cases present above 20 breaths per minute
- Pulsus paradoxus >10mmHg
- Use of accessory muscles with respiration
- Silent Chest (no movement of air)
  May be absent in up to 50% of severe asthma cases
- PaCO2>42 indicates severe asthma
- PCO2>70 mm Hg is an ominous sign

Chronic Obstructive Pulmonary Disease

COPD Disease Overview

Chronic Obstructive Pulmonary Disease: is a progressive, irreversible respiratory condition characterized by diminished inspiratory and expiratory capacity of the lungs. COPD, also known as chronic obstructive airway disease is a triad of distinct diseases that often coexist. The disease entities that comprise COPD include asthma (airway reaction) chronic obstructive bronchitis (airway inflammation) and emphysema (airway collapse).

Individuals generally have some form of all three disease states, but have one disease state that is predominantly present. COPD affects approximately 1 of 10 people and is second only to heart disease in morbidity and mortality, causing more than 100,000 deaths per year. COPD generally affects males greater than 45 years of age, but a greater number of females are developing the disease secondary to smoking.

Chronic Obstructive Bronchitis

Airway Inflammation

Chronic Bronchitis is a condition involving inflammatory changes and excessive mucous production in the bronchial tree. The disease is characterized by an increase in the number and size of mucous-producing glands.

Pathophysiology

Chronic Obstructive Bronchitis is characterized by inflammation of the bronchi caused by irritants (cigarette smoke, pollution) or infection, which leads to

increased mucus production (hyperplasia), muscle hypertrophy, bronchial wall thickening and inflammation and impaired ciliary function. This leads to the narrowing of the bronchial lumen caused by excess mucous and thickening of the airway walls leading to obstruction of airflow.

These physiologic changes also inhibit the normal defense mechanisms that fight infection. Because of the airway obstruction, airway collapse occurs, causing air trapping and chronic hypoxia with possible hypercapnia. Under normal physiologic circumstances the respiratory drive is triggered by a high partial pressure of arterial carbon dioxide (PaCO2). However, the patient with chronic obstructive bronchitis may have chronically elevated PaCO2 and may lose this drive as a respiratory stimulus. Hypoxia becomes the primary respiratory stimulus and excessive oxygen administration may blunt the respiratory drive. Polycythemia (increase in red blood cells) and pulmonary hypertension leading to cor pulmonale may develop. Cor pulmonale is visibly evident in the classic "blue bloater" with symptoms of peripheral edema, anasarca (severe, generalized edema) and chronic neck vein distension

Clinical Signs / Symptoms

The symptoms that lead patients with chronic bronchitis to seek medical intervention include decreased exercise tolerance, wheezing and shortness of breath. The individuals usually have a productive cough, which is also known as the smoker's cough, and evidence of airway obstruction is shown by spirometry. Hypoxia may occur with exercise. As the disease progresses, thick copious amounts of sputum are produced, accompanied by an increased frequency of pulmonary infections.

As the airway obstruction becomes more pronounced there is evidence of decreased alveolar ventilation and increased PaCO2. Marked hypoxemia leads to polycythemia (over production of erythrocytes) and cyanosis. If the condition is not reversed, hypoxemia can lead to pulmonary hypertension and eventually leads to cor pulmonale and congestive heart failure. The end result of the disease process can lead to severe disability or death. The most successful treatment for slowing of the disease process for chronic obstructive bronchitis and emphysema is the elimination of the causative agent, smoking.

Chronic Obstructive Bronchitis signs and symptoms can include:

- Blue Bloater
- Productive cough
- Dyspnea (Shortness of breath)
- Wheezing: is a whistling or squeaky sound when the patient breathes.

- History of Smoking
- Barrel Chest
- Typically overweight
- Hypoxemia
- Cyanosis
- Prolonged Expiration
- Chronic Hypoventilation
- Tachycardia
- Polycythemia
- Cor pulmonae

Emphysema

Airway Collapse

Emphysema is a disorder of impeded exhalation caused by permanent over-distention of air spaces, alveolar wall destruction, partial airway collapse and loss of elastic recoil of the lungs. Pockets of air that form between the alveoli and within the lung parenchyma cause increased ventilatory dead space and decreased lung tissue that lead to increased work of breathing.

Pathophysiology

Emphysema begins with the destruction of alveolar septa, which in part is due to elastin breakdown within the septa. Septal destruction eliminates portions of the pulmonary capillary bed and increases the volume of air in the acinus. Expiration becomes difficult related to the loss of elastic recoil that reduces the volume of air that can be expired passively. The hyperinflation of the alveoli causes large air spaces (bullae) and air spaces adjacent to pleura (blebs) to develop. The force that normal alveoli exert on the bronchiolar walls is diminished. The combined increase of residual volume in the alveoli and diminished force on the bronchioles causes part of each inspiration to be trapped in the acinus. The individual feels as though they are unable to fully exhale. The reduction of arterial PO2 leads to an increased production of red blood cells which is called polycythemia.

Clinical Signs/Symptoms

Patients with emphysema usually present with dyspnea on exertion which later progresses to marked dyspnea, even at rest. There is no cough and very little sputum production. The individual is often very thin, has tachypnea with prolonged expiration, and uses the accessory muscles to aid in the ventilatory effort. The anterior to posterior diameter of the chest is increased and has a hyperresonant sound with percussion. To increase lung capacity the individual often leans forward (tri-pod or sniffing position) with their arms extended

bracing their elbows on their knees while sitting. Pursed lipped breathing is frequently used to maintain positive airway pressures. Increased hypoxemia and hypercarbia may be indicated by tachypnea, diaphoresis, cyanosis, confusion, irritability and drowsiness.

Other physical findings include wheezing, rhonchi and crackles. Breath sounds and heart sounds may be diminished. This is due to reduced air exchange and the increased diameter of the thoracic cavity. In late stages of decompensation, the individual may have peripheral cyanosis, clubbing of the fingers and signs of right-sided heart failure. ECG findings may reveal cardiac dysrhythmias or signs of right atrial enlargement, which include tall peaked P waves in leads II, III, and AVF. The most successful treatment for slowing of the disease process for chronic obstructive bronchitis and emphysema is the elimination of the causative agent, smoking.

Emphysema signs and symptoms can include:

- Pink Puffer
- Dyspnea
- Wheezing
- Rhonchi
- Crackles
- Thin
- Clubbing of fingers
- Tachypnea
- Diaphoresis
- Cyanosis
- Confusion, Irritability
- Drowsiness
- Pursed lipped breathing
- Tri-pod / sniffing position

Communication

As with any Standing Order, the receiving facility requires certain information to allow them to prepare for the patients arrival. Relay the following essential information to the hospital:

- Age
- Sex
- Asthma / COPD Standing Order
- Transporting Unit
- Estimated Time of Arrival
- Stable or Unstable Patient

This information will maintain the continuity of care and facilitate patient transfer upon arrival

Summary

Respiratory emergencies are common in the prehospital setting. They account for approximately 28% of the chief complaints in all EMS calls. Each year more than 300,000 people die as a result of respiratory emergencies in the United States. Therefore patients with respiratory emergencies require the highest priority of care. The *paramedic* must be able to assess a patient with respiratory distress quickly, initiate management, and provide appropriate care en route to the hospital.

Glossary

Acinus: a small lobule of a complex physiologic structure

Anasarca: generalized, massive edema

Bradykinin: a peptide of nonprotein origin that contains nine (9) amino acid residues; a potent vasodilator.

Chemotactic factors: biochemical mediators that are important in activating the inflammatory response.

Cor pulmonale: an abnormal cardiac condition characterized by hypertrophy of the right ventricle of the heart as a result of hypertension of the pulmonary circulation.

Eosinophils: are cells that play an important role in allergic reactions by releasing chemical mediators that cause bronchoconstriction in asthma.

Hypercarbia: increased amount of carbon dioxide in the blood

Hyperresonant: Increased resonance produced when an area is percussed.

Leukotrienes: a class of biologically active compounds that occur naturally in leukocytes which produce allergic and inflammatory reactions.

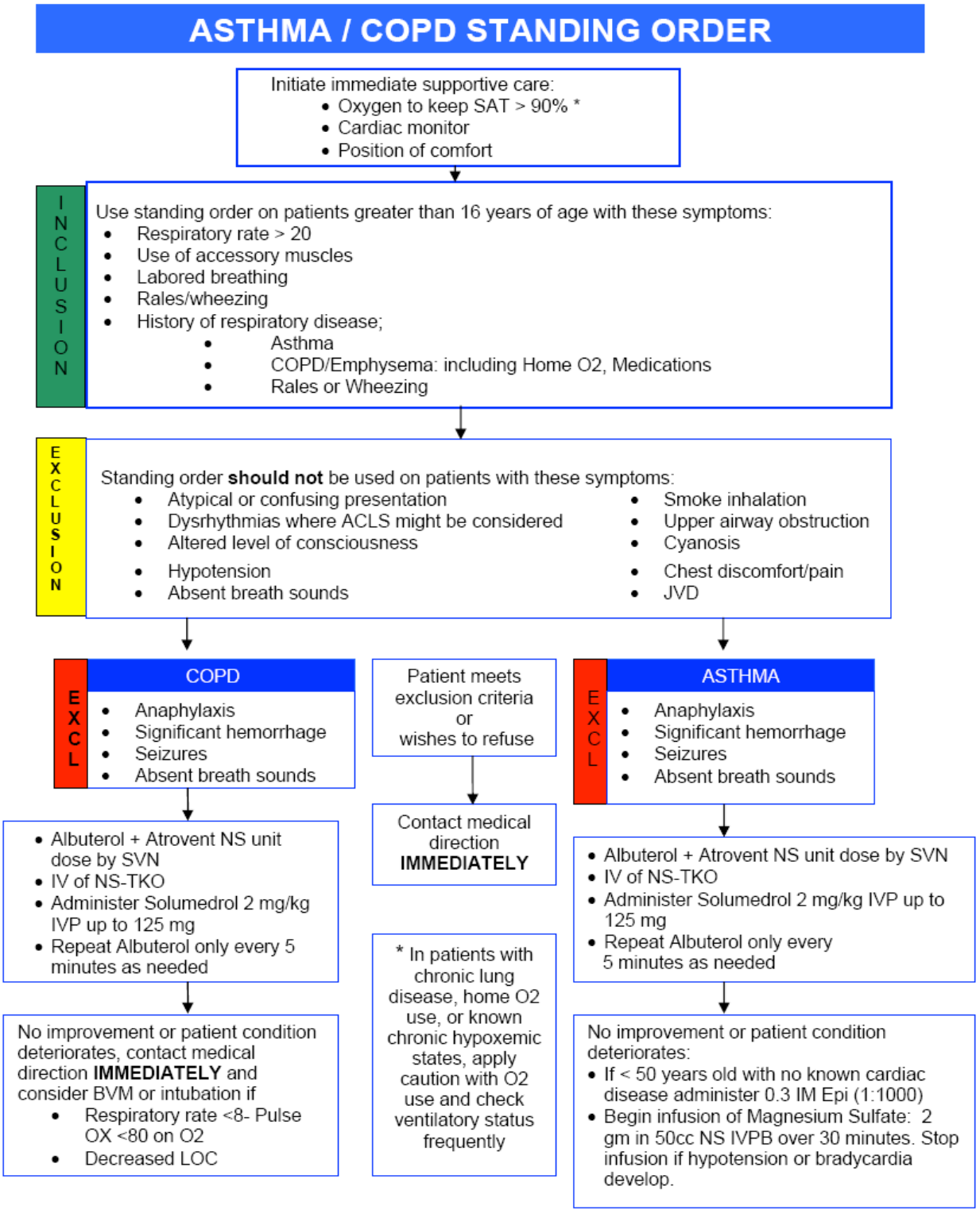
Polycythemia: a condition that is characterized by an unusually large number of red cells in the blood as a result of their increased production by the bone marrow.

Prostaglandin: a class of naturally occurring fatty acids that affect many body functions such as vasodilation, and promotion of inflammation and pain.

Pulsus paradoxus: an abnormal decrease in systolic blood pressure that drops more than 10 to 15 mm Hg during inspiration compared with expiration.

Thromboxane: antagonistic prostaglandin derivatives that are synthesized and released by degranulating platelets, causing vasoconstriction and promoting the degranulation of other platelets.

SAEMS Standing Order



Approved SAEMS 6-17-08; 10-21-08

Medication Overview

The Drug Profiles for the Asthma/COPD Standing Order can be accessed online at the AZDHS Website: http://www.azdhs.gov

	Drug Name	
1.	Albuterol Sulfate	GD-028-PHS-EMS
2.	Epinephrine HCL	GD-089-PHS-EMS
3.	Solumedrol	GD-046-PHS-EMS
4.	Magnesium Sulfate	GD-045-PHS-EMS

Magnesium compounds have been utilized within healthcare settings for many years for a variety of patient diagnoses in the form of chewable tablets in antiacids, oral liquid form for constipation, as well as Intramuscular Injections and Intravenous (IV) therapy. Prehospital providers "*Paramedics*" have also utilized this specific drug for eclampsia and cardiac care.

The use of Magnesium Sulfate for treating asthma as an adjunct treatment following the Albuterol/Atrovent SVN, IV Solumedrol and IM Epinephrine (for the patients under 50 years of age and no known cardiac history) in the prehospital setting should prove useful for the asthma patient suffering from a severe asthma attack. This treatment will not provide immediate relief, although an increase in the serum magnesium levels within the blood causes relaxation of the smooth muscle, which can contribute to bronchodilation. With the gradual decrease of the bronchospasm, bronchodilation occurs. The onset of action for intravenous Magnesium Sulfate administration is approximately 30 minutes.

The initial use of beta-agonists such as albuterol and epinephrine has been generally utilized by prehospital personnel for bronchodilation in the setting of asthma therapy. The use of IV Magnesium Sulfate may be used as an adjunct treatment after SVNs, IV Solumedrol and IM Epinephrine. The administration of Magnesium Sulfate should assist the patient who is suffering from severe asthma, especially if extended transport times are anticipated. The patient should benefit from the decrease and possible reversal of bronchospasm, which may minimize the need for intubation.

EMS' initial administration of Magnesium Sulfate for the patient who is suffering from Hyper-reactive Airway – Severe Asthma is 2 Gm Magnesium Sulfate mixed in 50 mL 0.9% Normal Saline, to be infused IV using microdrip tubing over 30 minutes. Stop the infusion if hypotension, respiratory depression or bradycardia develop.

As with all medications, precautions are to be taken if the patient is on barbiturates, narcotics, or other hypnotics, including systemic anesthetics. These medications can intensify the central depressive effects of magnesium.

It is important to evaluate the patient's cardiac status and ECG assessing for prolonged PR and widened QRS intervals when administering Magnesium Sulfate. Concurrent digitalization increases the danger of dysrhythmias, so again it is important to evaluate the patient's cardiac status and medications that they are currently taking before administering Magnesium Sulfate

The documentation of start and stop times when administering Magnesium Sulfate for respiratory emergencies should be strictly followed as the medication must be infused per administration guidelines of 30 minutes or greater using micro-drip tubing.

If the patient develops hypotension, respiratory depression or bradycardia, stop the Magnesium Sulfate infusion. Do not delay ventilation or intubation to institute Magnesium Sulfate.

ALBUTEROL SULFATE

GENERIC NAME: ALBUTEROL SULFATE

BRAND NAME: Proventil, Ventolin
CLASS: sympathomimetic, bronchodilator

Mechanism of Action:

ß agonist (primarily $ß_2$); relaxes bronchial smooth muscle, resulting in bronchodilation; also relaxes vascular and uterine smooth muscle; decreases airway resistance

Indications and Field Use:

Treatment of bronchospasm

Contraindications:

Synergistic with other sympathomimetics
Use caution in patients with diabetes, hyperthyroidism, and cerebrovascular disease

Adverse Reactions:

CV: Dysrhythmias, tachycardia (with excessive use), peripheral vasodilation
Resp: Bronchospasm (rare paradoxical with excessive use)
CNS: Tremors, nervousness
GI: Nausea, vomiting
Endocrine: Hyperglycemia

NOTES ON ADMINISTRATION

Incompatibilities/Drug Interactions:

Tricyclic antidepressants (TCA's) and monoamine oxidase (MAO) inhibitors
Other sympathomimetics (relative)

Adult Dosage:

Give 2.5 mg of premixed solution for inhalation (0.083%) via SVN with a mouth piece, or in-line with a ventilatory device. Repeated according to medical control preference

Pediatric Dosage: (children <40 lbs)

For children < 40 lbs., administer half of 0.083% premixed solution; add 1-1.5 mL NS to make 2.5-3 cc inhalation treatment administered via SVN with a mouth piece, O_2 mask or in-line with a ventilatory device. May be repeated according to medical control preference

Routes of Administration:

Nebulized, mouth piece or in-line via mask
Inhaler, patients own
ET/NT in-line

Onset of Action:

5-15 minutes

Peak Effects:

30 minutes - 2 hours

Duration of Action:

3-4 hours

Dosage Forms/Packaging:

2.5 mg albuterol premixed in 3 mL normal saline (independent dose) sulfite-free

Arizona Drug Box Supply Range:

PARAMEDIC: 2 - 6 independent doses
INTERMEDIATE: 2 - 6 independent doses

Special Notes:

- Must be sulfite-free

MAGNESIUM SULFATE

GD-045-PHS-EMS: Drug Profile for Magnesium Sulfate

GENERIC NAME: MAGNESIUM SULFATE

BRAND NAME: Magnesium Sulfate

CLASS: Electrolyte, tocolytic

Mechanism of Action:
Pharmacology: Second most plentiful intracellular cation; essential to enhance intracellular potassium replenishment and activity of many enzymes; important role in neurochemical transmission and muscular excitability (may decrease acetylcholine released by nerve impulses); decreases myocardial irritability and neuromuscular irritability.

Clinical: Cardiac-reduces ventricular irritability, especially when associated with hypomagnesemia; inhibition of muscular excitability.

Indications and Field Use:

Torsade de pointes, drug of choice
VF/Pulseless VT refractory to lidocaine and/or amiodarone
Hypomagnesemia
Pre-term labor (PTL)
Pregnancy-induced hypertension (PIH, toxemia of pregnancy, pre-eclampsia and/or eclampsia).
Hyperreactive Airway - Severe Asthma

Contraindications:

Hypermagnesemia
Use cautiously in patients with impaired renal function and pre-existing heart blocks (relative).
Precautions: Caution when used with barbituates, narcotics, or other hypnotics (or system anesthetics) in conjunction with Magnesium Sulfate due to the additive central depressive effects of magnesium.

Adverse Reactions:
Cardiovascular: hypotension (may be transient), flushing, circulatory collapse, depressed cardiac function, heart block, asystole, smooth muscle relaxant (antihypertensive effects).

Respiratory: respiratory depression and/or paralysis. This adverse reaction may occur in both mother and/or infant during or up to 24 hours after the administration of Magnesium Sulfate.

CNS: sweating, drowsiness, hypothermia, depressed reflexes progressing to flaccidity and paralysis. This adverse reaction may occur in both mother and/or infant during the administration of or up to 24 hours after the administration of Magnesium Sulfate.

GI: nausea

GU: mild diuretic

Metabolic: hypocalcemia, hypermagnesemia

NOTES ON ADMINISTRATION

Incompatibilities/Drug Interactions:
Concurrent digitalization increases danger of dysrhythmias
Adult Dosage:
Cardiac:

VF/Pulseless VT: 1-2 Gm IV diluted in 50-100 ml NS or D5W, administered over 1-2 minutes.

Torsade de pointes: 1-2 Gm IV diluted in 50-100 ml NS or D5W administered over 1-2 minutes, followed by the same amount infused over 1 hour.

Hypomagnesemia: Dilute 1-2 Gm in 50-100 ml NS or D5W administered IV push over 5-60 minutes.

Respiratory/Severe Asthma: Initial Infusion (field) 2 Gm Magnesium Sulfate mixed in 50 ml NS or D5W to be infused IV using microdrip tubing over 5 to 10 minutes. Stop infusion if hypotension, respiratory depression or bradycardia develop.

Pregnancy:

Pre-term labor (PTL): Initial bolus (Field and Interfacility): 4-6 Gm over 15-20 minutes (Suggested method is the addition of 4 Gm to 100 ml D5W, LR or NS. Resultant concentration is 40 mg/ml.)

Maintenance Infusion (Interfacility only): 1-4 Gm/hour infusion rate. (Suggested method for treatment of premature labor is to follow initial bolus with infusion of 2 Gm/hr which may be continued until uterine contractions are reduced to < 1

every 10 minutes. Then, infusion is decreased to 1 Gm/hr and continued for 24-72 hrs. One method for mixing infusion is the addition of 40 Gm to 1000 ml LR. Resultant concentration equals 40 mg/ml. If this concentration is run at 50 ml/hr, Magnesium Sulfate delivered equals 2 Gm/hr).

Pregnancy induced hypertension, pre-eclampsia/eclampsia, (PIH): Initial bolus (Field and Interfacility): 3-6 Gm over 10-15 minutes (Suggested method is the addition of 4 Gm to 100 ml D5W, LR or NS. Resultant concentration is 40 mg/ml).

Maintenance Infusion (Interfacility only): Follow bolus with 1-3 Gm/hour infusion rate. (Same mixture as for PTL). Rebolus: In an eclamptic emergency may rebolus with Magnesium Sulfate, 2-4 Gm depending on patient size (mixed as an initial bolus) over 10-15 minutes if respirations >12/minute and urine output >30 ml/hr.

Routes of Administration:
IV infusion
IO

Onset of Action:
Seconds
20 minutes for IV Infusion (respiratory)

Peak Effects:
Not known

Duration of Action:
24 hours or greater

Dosage Forms/Packaging:
1 Gm/2 cc vials (0.5 Gm/cc)
5 Gm/10 cc vials (0.5 Gm/cc)

Arizona Drug Box Supply Range:
5 Gm

Special Notes:

- O2 should be administered to patients receiving Magnesium Sulfate.

- For specific emergencies:
  OB emergencies maintenance infusions of Magnesium Sulfate should be administered by infusion pump to prevent toxicity.

Therefore, loading bolus therapy only, using a minimum of microdrip tubing is recommended for field to hospital intervention for OB indications.
Interfacility transfers may include a loading dose followed by a maintenance infusion of Magnesium Sulfate which requires an infusion pump.
Respiratory (Asthma) emergencies: Magnesium Sulfate follows Albuterol & Atrovent SVN and administration of 0.3 IM Epi (1:1000).

• For IV/IO infusions (respiratory) start and stop times should be closely monitored and documented per administration guidelines of 20 minutes or greater.

• Transport gravid patients lying or tilted to left side to prevent restricting venous return to heart.

• Use cautiously in patients with impaired renal function, pre-existing heart blocks and women in labor.

• Evaluate cardiac status and ECG assessing for prolonged PR and widened QRS intervals.

• Do not delay intubation or ventilation for Magnesium Sulfate administration in patients suffering severe asthma episode.

• Keep Calcium Chloride (10%) 10 ml available to reverse magnesium toxicity. See: Calcium Chloride profile. Use extreme caution if the patient is on digoxin.

• Monitor vital signs every 15 minutes in patients receiving Magnesium Sulfate infusion. If respirations less than 12/min, discontinue Magnesium Sulfate infusion, notify medical direction.

• Hourly intake and output should be monitored on long transport; urine output should be greater than 30 cc/hr.

• When given to toxemic mothers within 24 hours before delivery observe newborn for signs/symptoms of Magnesium Sulfate toxicity (neuromuscular and/or respiratory depression).

• Interfacility maternal transport teams are recommended and available for the transport of patients requiring continuous IV infusions of Magnesium Sulfate.

• In treatment of seizures associated with PIH it may be necessary to use an anticonvulsant such as diazepam.

• Eclampsia may occur up to six weeks after delivery

METHYLPREDNISOLONE SODIUM SUCCINATE

GENERIC NAME: METHYLPREDNISOLONE SODIUM SUCCINATE

BRAND NAME: Solu-Medrol
CLASS: corticosteroid, glucocorticoid, steroid, anti-inflammatory

Mechanism of Action:

Enters target cells and causes many complex reactions that are responsible for its anti-inflammatory and immunosuppressive effects; thought to stabilize cellular and intracellular membranes.

Indications and Field Use:

Reactive airway disease: Acute exacerbation of emphysema, chronic bronchitis, asthma
Anaphylaxis
Burns potentially involving the airway
** Acute spinal cord trauma (large loading and maintenance doses)

Contraindications:

Preterm infants

Adverse Reactions:

None from single dose

NOTES ON ADMINISTRATION

Incompatibilities/Drug Interactions:

None

Adult Dosage:

Reactive Airway Disease, Anaphylaxis, Burns Potentially Involving the Airway
Usual dose 125 mg slow IV bolus (much larger doses can be used).

** Acute Spinal Cord Trauma: Should be within 6 hours of insult and patient meeting criteria, initial bolus dose of 30 mg/kg IV administered over 15

minutes; bolus followed by a 45 minute rest period, then a 23-hour continuous infusion of 5.4 mg/kg/hr. See: Special Notes.

Pediatric Dosage:

Reactive Airway Disease, Anaphylaxis, Burns Potentially Involving the Airway
2-4 mg/kg slow IV bolus

Routes of Administration:

IV bolus
IO

Onset of Action:

1 - 6 hours; dogmatic 6 hour time to onset of benefit has decreased markedly during the last few years

Peak Effects:

8 hours

Duration of Action:

18-36 hours

Dosage Forms/Packaging:

125 mg/1 ml, 500 mg/4 ml, 1 Gm/8 ml mix-a-vial
2 Gm vial with diluent

Arizona Drug Box Supply Range:

	PARAMEDIC:	1 - 2 125 mg/2 ml mix-a-vial
**		Optional: additional training requirement for Spinal Cord Trauma (2 Gm x 1, 1 Gm x 1, 500 mg x 1, 125 mg x 1)
	INTERMEDIATE:	1 - 2 125 mg/2 ml mix-a-vial
**		Optional: additional training requirement for Spinal Cord Trauma (2 Gm x 1, 1 Gm x 1, 500 mg x 1, 125 mg x 1)

Special Notes:

> Use for spinal cord trauma is limited to prehospital providers that have completed a special training curriculum in accordance with their medical control authorities. Proper administration of methylprednisolone for spinal cord trauma is imperative.

> Infusions: An infusion pump is required for continuous infusions of corticosteroids during interfacility transports; a minimum of microdrip tubing is required for field use if administering loading dose therapy for spinal cord trauma.

** Indicates special training requirement

EPINEPHRINE HCl

GENERIC NAME: EPINEPHRINE HCl
CLASS: sympathomimetic

Mechanism of Action:

Pharmacological Effects: Direct acting a and ß agonist; a-bronchial, cutaneous, renal, and visceral arterial constriction (increased systemic vascular resistance); $ß_1$-positive inotropic and chronotropic actions (increases myocardial workload and oxygen requirements), increases automaticity and irritability; $ß_2$ bronchial smooth muscle relaxation and dilation of skeletal vasculature. Other: blocks histamine release

Clinical Effects:
Cardiac Arrest-increases cerebral and myocardial perfusion pressure; increases systolic and diastolic blood pressures; increases electrical activity in the myocardium; can stimulate spontaneous contractions in asystole. Bradycardia-increases heart rate, increases BP; Bronchospasm/Anaphylaxis-reverse signs/symptoms

Indications and Field Use:

Cardiac arrest - VF/Pulseless VT; asystole; PEA (First line pharmacologic agent for any pulseless dysrhythmia in cardiopulmonary arrest).

Severe bronchospasm, i.e., bronchiolitis, asthma.

Anaphylaxis.

Bradycardia, refractory with profound hypotension, monitored patient only.

Hypotension unresponsive to other therapy, monitored patient only.

Croup

Contraindications:

None known for cardiac arrest
Hypothermia, relative contraindication

Adverse Reactions:

CV: Hypertension, ventricular dysrhythmias; tachycardia; angina
CNS: Anxiety, agitation
GI: Nausea/vomiting

NOTES ON ADMINISTRATION

Incompatibilities/Drug Interactions:

Potentiates other sympathomimetics.
Reacts with alkaline solutions, such as sodium bicarbonate, should not be mixed with alkaline agents.

Adult Dosage:

Pulseless Arrest –
IV/IO: 1 mg of 1:10,000 solution repeat every 3 - 5 minutes or,
ET: Give 2 - 2.5 mg via the ET tube.
May use 1:10,000 or dilute 1:1000 to equal 10 mL via ET tube for adult. (i.e., 2 mg of 1:1,000 epinephrine diluted with 8 mL NS in a 10 mL syringe)

Continuous Infusion for Hypotension or Symptomatic Bradycardia: 1 mg added to 500 mL of NS administered at 1 mcg/min titrated to desired hemodynamic response (range 2-10 mcg/min); not first-line therapy.

Anaphylaxis and asthma: Give 0.3 - 0.5 mg of 1:1,000 solution IM (preferred), SC, or inject SL, may repeat every 15 to 20 minutes; or in extreme cases only, may be asked to use 1:10,000 solution and give 0.1 mg every 5 minutes IV/IO or continuous IV/IO infusion of 1 - 4 mcg/min to prevent need for multiple injections.

Pediatric Dosage:

Pulseless Arrest or Refractory Bradycardia:
IV/IO: 0.01 mg/kg of 1:10,000 repeat every 3 - 5 minutes, maximum single dose 1 mg.

ET: 0.1 mg/kg of 1:1,000; diluted with NS to a volume of 3 - 5 mL prior to instillation or followed with flush of 3 - 5 mL of NS after instillation repeat every 3 - 5 minutes, maximum single dose 10 mg.

Asthma/anaphylaxis: Use 1:1,000 solution; give 0.01 mg/kg IM (preferred), SC (maximum single dose of 0.5 mg/dose).
IV Infusion: 0.1 – 1 mcg/kg/min; to prepare for small children 0. 6 x body wt. in kg = mg added to NS to make 100 mL. With this mixture, 1 mL/hr delivers 0.1 mcg/kg/min.

Croup: 3 mg 1:1,000 mixed in 3 mL NS via SVN.

Neonatal Dose for First 12 hours of life:

IV/IO Initial and Repeat Dose for Cardiac Arrest or Refractory Bradycardia: 0.01-0.03 mg/kg of 1:10,000 every 3-5 minutes
ET: 0.1 mg/kg of 1:10,000 every 3 – 5 minutes if neonate has no vascular access, fails to respond to positive pressure ventilation with 100% O_2.

Routes of Administration:

Cardiac: IV push, IV infusion, ET, or IO

Asthma/anaphylaxis/bronchiolitis: IM, SC, SL injection, IV, ET, IO

Infusion pump required for IV infusions in interfacility transfers

Onset of Action:
Seconds

Peak Effects:

Minutes

Duration of Action:

Several minutes

Dosage Forms/Packaging:

1:10,000 solution 1 mg/10 ml prefilled syringes
1:1,000 solution 1 mg/1 mL ampule or prefilled syringes; 30 mg/30 ml vial

Arizona Drug Box Minimum Supply:

PARAMEDIC and IEMT/99:	1:10,000 prefilled syringes – 5 mg
	1:1,000 - 2 mg
	1:1,000 multidose vial- 30 mg
INTERMEDIATE	1:1,000 - 2 mg

Special Notes:

> Total dose for an adult ET (drug plus diluting solution) should equal at least 10 ml to ensure that the drug reaches lung tissue rather than remaining in the tube. Pediatric patient should equal 3 - 5 mL.

> Multi-dose Vial: 1 mg/ml (1:1,000) in 30 ml bottle. May be used for administering the ACLS doses of epinephrine down the endotracheal tube (2-2.5 times the peripheral route dose, diluted with 8 ml NS to make a 1:10,000 solution) or for mixing an epinephrine infusions such as 1 mg in 500 mL NS

> Infusions: An infusion pump is required for interfacility transports. A minimum of microdrip tubing is required for field use.

Name: _____ Agency:_____ Date:_____ Score:_____

Pre-Hospital Self-Learning Test
Asthma / COPD Standing Orders
Pre-Hospital use of Magnesium Sulfate

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1. Magnesium Sulfate is
   a. Is the second most plentiful intracellular cation
   b. Decreases ventricular myocardial irritability
   c. Decreases neuromuscular irritability
   d. All the above

2. Magnesium Sulfate is field indicated for:
   a. Hyper Reactive Airway - Severe Asthma
   b. Torsade de Pointes
   c. Pregnancy Induced Hypertension
   d. All the above

3. Contraindications for Magnesium Sulfate use include all but:
   a. Hypermagnesia
   b. Pre-existing heart blocks (use cautiously)
   c. Dyspnea
   d. Barbituates, narcotics, hypnotics or systemic anesthetics (use cautiously)

4. Adverse reactions to monitor the patient for when administering Magnesium Sulfate for Respiratory - Severe Asthma attacks include all except :
   a. Respiratory Depression
   b. Bradycardia
   c. Hypotension
   d. Increased urination

5. Interfacility Transfers require Magnesium Sulfate to be administered by an Infusion pump:
   a. True
   b. False

6. For Respiratory - Severe Asthma, following the Asthma/COPD Standing Order, administration of Magnesium Sulfate is to be administered:
   a. After Albuterol & Atrovent SVN, 2mg/kg Solumedrol IVP and 0.3 IM Epi (1:1000)
   b. Administered before all other medications
   c. After Albuterol & Atrovent SVN and 500 mL IV bolus of NS 0.9%
   d. After Albuterol & Atrovent SVN, 2mg/kg Solumedrol IVP and 0.3 IM Epi (1:10,000)

7. All actions are to be taken when administering Magnesium Sulfate except:
   a. Monitor vital signs every 15 minutes or less
   b. Monitor Respiratory status
   c. Evaluate the patients cardiac status and ECG for a history of heart blocks and prolonged PR and widened QRS intervals
   d. Never intubate a patient on Magnesium Sulfate

8. What medication should be kept available for magnesium toxicity:
   a. Epinephrine
   b. Lidocaine
   c. Calcium Chloride
   d. Albuterol

9. Prehospital documentation when administering Magnesium Sulfate for Severe Respiratory episodes require all except:
   a. Documentation every 2 hours
   b. Start and Stop times of the drug administration
   c. Vital signs every 15 minutes or less
   d. Monitor respirations

10. Intubation and ventilation should never be delayed for the administration of Magnesium Sulfate
   a. True
   b. False

11. The Adult Dosing for Magnesium Sulfate requires all except:
   a. 2 Gm Magnesium Sulfate in 50 mL Normal Saline 0.9%
   b. Use of microdrip tubing
   c. Use of macrodrip tubing
   d. Infused over 30 minutes

12. It is advisable to stop a Magnesium Sulfate Infusion for all clinical symptoms when following the Asthma/COPD Standing Order *except* when:
    a. Patient has hypotension
    b. Patient has bradycardia
    c. Patient is in Normal Sinus Rhythm – ECG tracing
    d. Patient has respiratory depression (respirations less than 12)

13. The administration route for Magnesium Sulfate for Respiratory – Severe Asthma is:
    a. Intravenous Push
    b. Intramuscular (IM)
    c. Per Oral (PO)
    d. Intravenous Infusion

14. The onset of action for IV infusion of Magnesium Sulfate for Respiratory – Severe Asthma is:
    a. 20 minutes
    b. 30 minutes
    c. Immediate
    d. 1 hour

15. What qualified EMS personnel may administer Magnesium Sulfate
    a. EMT – B
    b. EMT – P
    c. EMT – I
    d. All the above

Name: _____ Agency:_____ Date:_____ Score:_____

Pre-Hospital Self-Learning Test
Asthma / COPD

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1. For Respiratory - Severe Asthma, following the Asthma/COPD Standing Order, administration of Magnesium Sulfate is to be administered:

a. after Albuterol & Atrovent SVN, 2mg/kg Solumedrol IVP and 0.3 IM Epi (1:1000)
b. administered before all other medications
c. after Albuterol & Atrovent SVN and 500 mL IV bolus of NS 0.9%
d. after Albuterol & Atrovent SVN, 2mg/kg Solumedrol IVP and 0.3 IM Epi (1:10,000)

2. At the beginning of an asthma attack there is usually a sensation of:
   a. chest constriction or tightening,
   b. inspiratory and expiratory wheezing,
   c. dyspnea, nonproductive coughing
   d. all the above

3. Albuterol Sulfate can be administered by which of the following route(s):
   a. orally
   b. IV
   c. nebulizer
   d. all of the above

4. Indications for use of Methylprednisolone Sodium Succinate include:
   a. reactive airway disease: Acute exacerbation of emphysema, chronic bronchitis and asthma
   b. anaphylaxis
   c. burns potentially involving the airway
   d. all the above

5. Epinephrine HCL is only used for Cardiac Arrest patients
   a. True
   b. False

6. Interfacility transfers require Magnesium Sulfate to be administered by an Infusion pump:
   a. True
   b. False

7. The onset of action for IV infusion of Magnesium Sulfate for Respiratory – Severe Asthma is:
   a. 20 minutes
   b. 30 minutes
   c. Immediate
   d. 1 hour

8. Patients with emphysema usually present with dyspnea on exertion which later progresses to marked dyspnea, even at rest
   a. True
   b. False

9. Albuterol Sulfates mechanism of action includes all but:
   a. ß agonist (primarily $ß_2$);
   b. relaxes bronchial smooth muscle, resulting in bronchodilation;
   c. relaxes vascular and uterine smooth muscle;
   d. increases airway resistance

10. Inclusions for the Asthma Standing Order Include all but:
   a. respiratory rate <20
   b. labored breathing
   c. use of accessory muscles
   d. rales/Wheezing

11. Adverse reactions to monitor the patient for when administering Magnesium Sulfate for Respiratory - Severe Asthma attacks include all except :
   a. respiratory depression
   b. bradycardia
   c. hypotension
   d. increased urination

12. Methylprednisolone Sodium Succinate can be administered:
    a. rectally
    b. peripheral IV
    c. intraosseous
    d. b and c

13. The SAEMS Asthma/COPD Standing Order should not be used on patients with symptoms of:
    a. altered level of consciousness
    b. smoke inhalation
    c. absent breath sounds
    d. all the above

14. Inclusions for the Asthma Standing Order Include all but:
    a. respiratory rate <20
    b. labored breathing
    c. use of accessory muscles
    d. rales/Wheezing

15. Emphysema is a disorder of impeded exhalation caused by permanent over-distention of air spaces, alveolar wall destruction, partial airway collapse and loss of elastic recoil of the lungs
    a. True
    b. False

References

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Thelan, L.A., Urden, L.D., Lough, M.E., Stacy, K.M., (1998) Critical Care Nursing Diagnosis and Management Third Edition. St. Louis, MO: Mosby

EVALUATION

Please answer the following questions by marking the appropriate response:

		Lowest Worst Least				Highest Best Most
1.	To what extend did this module meet your needs?	1	2	3	4	5
2.	There was a balance between theoretical and practical information.	1	2	3	4	5
3.	The time required was appropriate to the content.	1	2	3	4	5
4.	The module increased my knowledge and understanding of the topic.	1	2	3	4	5
5.	References or audiovisuals were adequate.	1	2	3	4	5
6.	Overall, this program was worthwhile.	1	2	3	4	5

7. Additional comments:

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