



2007

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2007

LEARNING OBJECTIVES SECTION FOUR

CEREBROVASCULAR DISORDERS

1. Define Cerebrovascular Accident (CVA):
 - A CVA, also referred to as a **stroke**, occurs when a disruption or blockage interrupts the blood flow to the brain for a long enough period of time to cause damage.
2. Describe the pathophysiology and life threats of a CVA/TIA:

Pathophysiology:

- CVA:

A stroke occurs when the blood supply to a part of the brain is compromised in any way, either because a cerebral artery becomes blocked (ischemic stroke) or because it ruptures and bleeds (hemorrhagic stroke).

- *Ischemic strokes are caused either by thrombosis, or by an embolus. About 60% of all strokes are the result of thrombosis in a cerebral artery. In the great majority, atherosclerosis is the underlying cause, producing hardening, narrowing, and thrombus formation in the cerebral arteries. About 60% of patient's with a thrombotic stroke suffer from hypertension, about 50% have a history of atherosclerotic disease elsewhere and about 25% suffer from diabetes. Thrombotic strokes most often come on while the patient is sleeping, or shortly after getting up in the morning. SxS of a thrombotic stroke are typically slower developing than those of an embolic or hemorrhagic stroke.*
- *An embolic stroke is caused by the impaction of a wandering blood clot or large air bubble that reaches the cerebral circulation from another part of the body. Embolic strokes tend to come on abruptly at any time of the day or night, often while the patient is engaged in normal activity. The embolic stroke evolves very rapidly, producing maximum neurological deficits in seconds to minutes. In a good percentage of embolic strokes, the first SxS is a seizure. About 25% of*

patients will complain of a headache (although not excruciating), and about a third will suffer a transient loss of consciousness. Patients at high risk for embolic stroke include those with a history of atrial fibrillation, valvular heart disease, women taking oral contraceptives (especially smokers), and patients with sickle cell disease.

- Hemorrhagic strokes can also be classified into two general types: intracerebral hemorrhages and subarachnoid hemorrhages. The intracerebral hemorrhage is nearly always the result of hypertensive disease, which causes weak areas in the small vessels of the brain. Eventually, these weak areas turn into an aneurysm, which eventually ruptures, and bleeding into the brain results. The patient is typically younger than the thrombotic stroke victim (in their 50's or 60's). The patient's mental status may progress from conscious and alert to unconscious rapidly, exhibiting SxS of ICP. The prognosis is very poor, with 50-80% mortality. Subarachnoid bleeds occur when the aneurysm occurs at a point near the base of the brain, causing bleeding into the space around the brain. SxS usually occur rapidly, with the most common one being an excruciating ("worst headache I have ever had") headache followed by N/V, neck and back pain, confusion and coma.

- **TIA**

A **transient ischemic attack** is a neurologic deficit of any type that lasts from seconds to hours, with which the patient returns to normal within 24 hours without permanent neurological deficit. About 80% of patients who suffer a thrombotic stroke will have a series of TIA's preceding the stroke.

Life Threats:

- **CVA:**

Disruption/damage to vital centers, leading to respiratory and cardiac emergencies. Airway should always be a concern. Cardiac dysrhythmias occur in at least 50% of all patients, and life-threatening dysrhythmias occur in about 20% of stroke victims.

- **TIA:**

In and of itself, none since the neurological effects are temporary. An exception is possible aspiration if airway is not monitored.

3. List at least 5 risk factors for CVA:

- Hypertension
- Obesity
- Cigarette smoking
- Diabetes
- High serum cholesterol
- Lack of exercise
- Age
- Family Hx of CAD
- Male gender
- Birth control pills

4. List 5 signs and symptoms of a CVA:

- **Alterations in LOC:**
 - *Dizziness, confusion, unsteadiness*
 - *Personality changes*
 - *Changes in mental ability*
 - *Decreasing LOC, ranging from dizziness to coma*
 - *Seizures*
- **Effects on motor function:**
 - *Weakness, numbness, or paralysis of the arms, legs and face, often on only one side (hemiparesis & hemiplegia).*
 - *Facial drooping on one side, paralysis of facial muscles resulting in a loss of facial expression and a drooping eyelid.*
- **Effects on sensory function and changes in vision:**
 - *Loss of vision or temporary dimness of vision, particularly in one eye, double vision can also occur.*
- **Altered communication abilities:**
 - *Inability to speak or trouble in speaking or understanding speech.*
- **Other symptoms:**
 - *Headache, sometimes accompanied by a stiff neck (caused by sub-arachnoid hemorrhage).*
 - *Sudden, severe headache or change in the pattern of headaches normally experienced by the patient.*
 - *Flushed or pale face.*
 - *Respiratory distress, airway compromise due to inability to deal with secretions.*
 - *Unequal pupils.*
 - *Bowel and bladder incontinence.*
 - *Nausea and vomiting.*

5. Describe the field management of a CVA patient:

- *Early recognition and transport to the appropriate facility (preferably with a neurosurgery unit) is the goal.*
- *Honestly reassure the patient, remembering that the patient may be able to hear and understand everything said, regardless of outward level of response.*
- *Maintain an open airway, assuring status of ABC's.*
- *Provide high concentration of oxygen, assisting with BVM if necessary.*
- *Monitor vital signs closely.*
- *Treat increasing ICP if present by ventilating the patient at a rate of 16 – 20 ventilations per minute.*

SEIZURES

1. Define epilepsy and describe the pathophysiology of an epileptic seizure:
 - *Epilepsy is a chronic brain disorder characterized by recurrent seizures with or without a loss of consciousness. The seizure is an involuntary, sudden change in sensation, behavior, muscle activity, or LOC. Generally the seizures are caused by an abnormal discharge of electrical energy in the brain.*
2. Define **status epilepticus** and explain the primary life threat:
 - *Status epilepticus is a series of seizures that occur in rapid succession that does not allow for a period of lucidity or consciousness between seizures. The prolonged seizures can deny the body of oxygen, causing anoxia of the brain, cardiac, respiratory, and renal systems. This can result in irreversible brain damage, cardiac arrhythmias, hypoxia, hyperthermia, airway compromise, aspiration pneumonia, and death.*
3. Explain each of the following terms related to seizures:
 - ***Aura:** A peculiar sensation that precedes and sometimes warns of an impending epileptic seizure. It may be of a psychic or a sensory nature, with olfactory, visual, auditory, or taste hallucinations, and can precede the seizure by anywhere from seconds to hours.*
 - ***Tonic-Clonic movements:** The tonic phase initiates soon after the patient loses consciousness. It is characterized by continuous motor tension and contraction of the patient's muscles. This phase usually lasts 15-20 seconds. It is followed by the hypertonic phase, in which the patient experiences extreme muscle rigidity including hyperextension of the back. This phase usually lasts 5-15 seconds. Finally, the patient progresses to the clonic phase, which is characterized by the classic muscle spasms marked by muscular rigidity and then relaxation.*
 - ***Postictal State:** This is a period of coma, drowsiness, confusion, fatigue, headaches, and sometimes neurological deficits that follow a generalized (grand mal) seizure.*
4. List at least 6 possible causes (other than epilepsy) of seizures:
 - *High fever*
 - *CVA*
 - *Space occupying lesions in the brain (tumor)*
 - *Alcohol/Drug withdrawal*
 - *Hypoxia*
 - *Toxin/Drug ingestion*
 - *Head injury*
 - *Childhood illnesses*
 - *Meningitis, Encephalitis*
 - *Hypoglycemia*
 - *Hyperventilation*
 - *Eclampsia*

5. Explain the pathophysiology, life threats, and field management of the convulsing patient and of the post-convulsive (postictal) patient by the EMT-B:

Pathophysiology:

- A seizure is caused by abnormal bursts of electrical discharge from the brain. The effects of this abnormal discharge on the patient vary according to what portion of the brain is affected. The most dramatic seizures are those that involve the motor cortex, resulting in involuntary muscle contraction and relaxation. Some types of seizures affect only the cognitive portion of the brain. Seizures can be caused by several sources, including epilepsy, fever, intracranial bleeds, head trauma (new or old), brain or meningeal lesions or swelling, eclampsia, infections, hypoglycemia, poisoning, drug or alcohol withdrawal, and hypoxia. A significant cause of seizures seen in the prehospital setting is a patient's failure to take correct doses of prescribed anti-seizure medications.

- Types of seizures:

- **Generalized tonic-clonic seizures** (Formerly called Grand Mal seizures)

Is the type of seizure most people think of when they discuss this subject. Occasionally, a patient will have an aura prior to a grand mal seizure, which is a warning sign of an impending seizure that is peculiar to the site of origin in the brain. Some patients may become aware of strange smells, visual or auditory sensations, or motor events in only one part of the body.

Characterized by a LOC, tonic-clonic movement, and sometimes tongue biting, incontinence, and mental confusion.

A grand mal seizure generally consists of three phases: (1) The tonic phase, where there is a sustained contraction of all voluntary muscles, usually lasting up to 30 seconds. (2) The clonic phase follows the tonic phase, and is characterized by intermittent contractions and relaxations of the skeletal muscles that result in a rapid, jerking movement. The patient may even appear to be "posturing". The clonic phase may last from seconds to several minutes, and can be followed by a short period (generally 30 seconds) of flaccid paralysis. (3) The postictal phase ensues, with the patient showing an altered level of response, ranging anywhere from being very sleepy and confused, to rare cases of combativeness.

- **Absence seizures:** (Formerly known as Petit Mal seizures)

These seizures are characterized by a very brief LOC - usually no longer than a few seconds - without loss of muscle tone. It is usually seen in children, who will suddenly stare off into space and/or become unresponsive, then just as suddenly, resume what they were doing. Involve brief lapses of attention and awareness lasting from 10 to 20 seconds. They are most common in childhood, but may persist to adulthood. These types of seizures may occur many times a

day, with some patients reportedly suffering dozens in a 24-hour period. Absence seizures are described as a sudden staring by the patient, with eyes turned upward or to the side, accompanied by a fluttering of the eyelids. It is always brief, lasting a few seconds and the patient can continue or return to previous activity as if nothing happened.

- Simple Partial Seizures

- Do not involve the entire brain, but affect only a portion of the body. Jerking might begin in one area of the body, arm, leg, or face. It cannot be stopped, but the patient usually stays awake and aware. Jerking can proceed from one area of the body to another and sometimes spreads to become a generalized tonic-clonic seizure.

- Complex Partial Seizures

- Usually starts with a blank stare, followed by chewing, followed by random activity. The person appears unaware of the surroundings, might seem dazed and mumble. Actions clumsy, not directed. Last a few minutes, but postseizure confusion can last substantially longer. No memory of what happened during the seizure period.

- Myoclonic Seizures

- Sudden brief, massive muscle jerks that can involve the whole body or parts of the body. Can cause the person to drop things or fall off a chair.

- Infantile Spasms

- Clusters of quick sudden movements that start between 3 months and 2 years. If child is sitting up, head will fall forward, and arms will flex forward. If lying down, knees will be drawn up, with arms and head flexed forward as if the child is reaching for support.

- Febrile Seizures

- The term **febrile seizure** describes a seizure occurring in a child between the ages of 6 months and 6 years of age that is precipitated by a high temperature in the setting of an infection. Febrile seizures occur in up to 5% of children. Treatment of this particular type of seizure should be discussed in the pediatric section of the BLS course.

Prehospital Life Threats:

- If the seizure does not subside within 5 minutes or if recurrent seizures without a lucid interval between (status epilepticus) occur, respiratory compromise becomes the major concern. During seizure activity, oxygen is consumed at 60% over normal, with O₂ and glucose being rapidly depleted, with a resultant accumulation of lactate in the brain. Continued, severe seizure activity holds the potential for progressive brain injury and irreversible damage.

Management:

- *Most seizures will not present a problem. If a patient is actively seizing, treatment will usually be aimed at protecting the patient from injury. A nasopharyngeal airway may help maintain an airway for this patient, and avoids having to attempt to place anything in the mouth of a seizing patient. Following a grand mal seizure, the patient may need temporary assistance in the establishment of an airway. A nasopharyngeal airway may again prove to be the most desired device for this, as it will not stimulate the gag reflex. Then, high flow oxygen should be administered, and the patient protected from embarrassment as much as possible. The left lateral recumbent position may be the best to facilitate drainage of fluids from the airway. The rescuer should also be prepared to suction as necessary. Since hypoglycemia is a potential cause (as well as a result, in some cases) of seizure activity, a BGL should be taken. A quick head to toe examination is appropriate to check for additional injuries that the patient may have received during the seizure. Begin treatment of possible causes of the seizure (head injury, hypoxia, febrile, etc.) Consider an ILS/ALS intercept, especially if the patient is in status epilepticus.*

DIABETES

1. Describe the role of glucose in normal body function:

- *Glucose is a sugar molecule used by the body's cells for energy. Larger sugar molecules, such as sucrose and fructose, are converted into this simple carbohydrate, which is then absorbed into the bloodstream. The glucose levels of the bloodstream rise as a person eats, and as the blood glucose level rises, insulin is secreted and causes glucose to move into the cells, where it can be used for energy or stored for future use.*

- *The body depends on glucose for its basic energy needs. While most cells also use other sources of fuel, such as fats, the brain depends almost exclusively on glucose. When deprived of glucose, the brain's function is altered and unconsciousness, seizures, and brain cell death can occur. The CNS is the only group of cells that DO NOT rely on insulin to access the glucose.*

2. Describe the role of insulin in glucose metabolism:

- *Insulin is produced within specialized cells in the pancreas, which are called the Islets of Langerhans. As the glucose in the blood rises after a meal, the body secretes insulin, which facilitates movement of the extra glucose and other food products into cells for immediate utilization as fuel or for storage for future needs. Without insulin, the glucose remains in the bloodstream, unavailable for use by the cells for energy.*

3. Identify the role of the pancreas in insulin production:
 - *The pancreas is an elongated, somewhat flattened organ that lies behind the abdominal cavity on the posterior abdominal wall in both upper quadrants. It is actually an organ with a dual role; it is an exocrine gland that secretes digestive juices, and an endocrine gland that releases hormones. The endocrine portion of the pancreas consists of cells arranged in groups closely associated with blood vessels. The groups, called Islets of Langerhans, include two types of cells—the alpha cells that secrete the hormone glucagon, and the beta cells that secrete the hormone insulin.*

4. Define Diabetes mellitus:
 - *A metabolic disorder in which the ability to metabolize carbohydrates is impaired, usually due to a lack of insulin.*

5. Define hypoglycemia and list 4 possible causes of hypoglycemia, (aka Insulin Shock):
 - *Hypoglycemia occurs when a diabetic has had too much insulin or too little sugar. The glucose moves out of the bloodstream and into the cells more rapidly than it is produced, resulting in an insufficient blood sugar level to maintain normal brain function. Simply put – LOW blood glucose level.*

 - ***Hypoglycemia can have several different causes:***
 - *The diabetic skips a meal but takes the usual amount of insulin.*
 - *The diabetic vomits a meal after taking insulin.*
 - *A diabetic takes more than the normal dosage of insulin, or the prescribed dosage is accidentally administered directly into a vein.*
 - *The patient exercises strenuously or excessively.*
 - *The diabetic's insulin dosage or diet has been changed, and the patient does not adapt well to the changes.*

6. Describe the signs & symptoms of hypoglycemia:
 - *Remember...the signs and symptoms of hypoglycemia can often mimic alcohol/drug intoxication or CVA. The SxS include:*
 - *Headache*
 - *No unusual odor on breath*
 - *Extreme muscle weakness and incoordination*
 - *Apathy, anxiety, irritability, or combativeness*
 - *Confusion, disorientation, unconsciousness, coma*
 - *Dilated pupils*
 - *Nausea & vomiting*
 - *Pale, moist skin*
 - *Profuse drooling*

- *Hunger, but often no thirst*
- *Convulsions, seizures in late stages*
- *Normal or shallow breathing*
- *Weakness or paralysis of one side of body*
- *Numbness and tingling of extremities*
- *Tremors or shakiness*
- *Dizziness, lightheadedness*
- *Normal or low BP*
- *Diaphoresis*
- *Normal or rapid pulse: May be full and bounding*
- *Speech difficulties*
- *Behavioral disturbances*
- *Double vision and other visual disturbances*
- *Syncope*
- *posturing*

7. Describe the field management of hypoglycemia in both the conscious and unconscious patient:

- *If the patient is conscious, give a commercially available glucose paste or orange juice with several teaspoons of sugar added, soft drinks that contain sugar, corn syrup, honey, jelly, candy, sugar cubes or even simple table sugar to help increase the carbohydrate level.*
- *If the patient is unconscious, establish an airway, administer oxygen, and transport, consider ALS or ILS intercept.*
- *Never give an unconscious person anything that would have to be chewed or swallowed.*
- *Watch for complication, such as shock or convulsions, and treat appropriately.*
- *Generally speaking, it is prudent to transport the patient even if the patient seems to be completely recovered.*

8. Explain the possible consequences if hypoglycemia is not recognized and treated:

- *Because the brain is as dependent on glucose as it is on oxygen, and because the brain is the first organ to react to low blood sugar, brain damage or in extreme cases, **death** can result from hypoglycemia if emergency care is not given.*

9. Define Diabetic Ketoacidosis (DKA), identifying the substance metabolized as fuel by the body in DKA and listing the waste products produced by metabolism of this substance. Explain why DKA can be life threatening:

- *DKA is a condition that results from a decreased or absent insulin supply, caused by a lack of the body's production of insulin or a patient taking insufficient amounts of the prescribed dosage. The blood sugar is high, but since there is no insulin, the sugar cannot be absorbed by the cells. The body attempts to overcome the lack of sugar in the cells by switching to other foodstuffs for energy; principally fat. These fats are not an efficient alternative to glucose, generating fatty acids and ketones as waste products. These waste products build up, greatly increasing the acidity of the blood. If left untreated, this can cause coma and death.*

10. Explain why dehydration is usually associated with DKA and describe the signs of dehydration:

- *Since glucose cannot be taken up by the cells, it continues to accumulate in the blood. As the blood sugar rises, the patient undergoes massive osmotic diuresis, which is the passing of large amounts of urine because of the high solute concentration of the blood. This together with vomiting causes dehydration and shock.*

- ***Signs and symptoms of dehydration include:***

- *Headache*
- *Dry, warm skin*
- *Hx of frequent urination, although there may be little or no urine production now*
- *Sunken eyes*
- *Rapid, weak pulse*
- *Intense thirst*
- *Dizziness*
- *Lack of normal skin tone also called poor skin turgor*
- *Irritability*

11. List 3 possible causes of DKA:

- *Infection—the most common cause of DKA is several days of infection, such as a viral respiratory infection.*
- *Failure to take prescribed insulin, or taking too little insulin.*
- *Eating too much food that contains sugar.*
- *Stresses such as a heart attack, illnesses, etc.*

12. Describe the signs & symptoms of DKA:

- *In the early stages, DKA is characterized by excessive hunger (polyphagia), thirst (polydipsia) and urination (polyuria). The onset of diabetic coma is gradual, developing in most cases over a period of twelve to forty-eight hours. These patients come to look extremely ill, and will become sicker and weaker as the condition progresses.*
- *Other SxS include:*
 - *Labored respirations and exaggerated air hunger, known as Kussmaul's respirations – caused by the body attempting to blow off the ketones and carbon dioxide through the respiratory system, in order to correct the acidosis.*
 - *Sweet, fruity, or acetone odor on the breath caused by the excess ketones as they are eliminated through the lungs. This is sometimes difficult to detect.*
 - *Frequent severe and intense abdominal pain.*
 - *Varying degrees of level of response, from restlessness, confusion, and disorientation to coma. Decreasing levels or responsiveness correspond to the degree of dehydration.*
 - *Dehydration (see #10 for specific SxS associated with dehydration).*
 - *Weakness, weight loss, and fatigue.*
 - *Nausea, vomiting, anorexia*
 - *Fever*

13. Describe the goal of field management of DKA:

- *The goal of prehospital care of the DKA patient is the early recognition of the condition and rapid evacuation of the patient to the medical facility where the patient can receive insulin, IV fluids and other treatments to alleviate the dehydration and acidosis.*
- *Monitor vital signs carefully every few minutes. Be careful not to dismiss AMI, CVA, or other medical or traumatic emergencies as a cause of coma.*
- *Follow the procedure for any comatose patient with regard to the ABC's and oxygenation. Place the patient on high flow oxygen, or if possible, hyperventilate the patient with a BVM and 100% oxygen, as it helps combat acidosis.*
- *Treat the patient for shock.*
- *Take any insulin vials to the hospital with you.*
- *Transport the patient as soon as possible.*
- *Consider ILS/ALS intercept.*

14. Referring to the pertinent signs & symptoms, describe how you might differentiate between hypoglycemia and DKA in an unconscious patient:

Diabetic Ketoacidosis (DKA):

- Gradual onset of SxS
- Dry mouth, thirst
- Abdominal pain, vomiting
- Dry, red, warm skin
- Breath smells of acetone
- Air hunger, deep respirations

Hypoglycemia:

- Rapid onset of SxS – usually minutes
- Excessive drooling
- Pt. Hungry
- Pale, cool, diaphoretic skin
- No unusual odor on breath
- Shallow or normal respirations

- Utilization of these general SxS can help determine what the EMT is dealing with, as well as obtaining a thorough past medical history. BLS services using glucometry have the advantage of determining a patient's BGL.

15. Describe the treatment of both a conscious and unconscious patient in which you are unable to determine the nature of the diabetic emergency:

- The general rule to remember about emergency care for a conscious diabetic patient is: When in doubt, GIVE SUGAR: The amount of sugar given orally to a hypoglycemic patient is possibly life saving, and usually not harmful to a patient in DKA. The remainder of treatments have been covered in # 7 & 13.

16. Explain the rationale for administering oral glucose:

- Oral glucose is quickly absorbed into the blood stream. This is given to the hypoglycemic patient.

GLUCOMETRY

1. Explain the purpose of glucometry and identify the normal range of blood glucose:

- Its purpose is to measure the amount of sugar available in the blood; normal range is 60 – 120 mg/dL (milligrams per deciliter [deciliter = 1/10 of a liter or 100 cc]).

2. Explain the general technique, including strip selection and site selection:

- Strips must match the type of machine; they are not interchangeable. Also they must have the appropriate code. The machine must be tested for calibration periodically. The blood sample can be either venous (e.g. from an IV start) or capillary blood (finger, earlobe). If the finger is used, the least painful area for the patient is the side of the finger pad. The site must be cleaned and allowed to dry

before the sample is taken. “Milking” the blood down to the tip of the finger may allow the EMT to obtain a sample easier.

- The drop of blood must be dropped freely onto the testing strip, not smeared.

- All contaminated material must be disposed of in a biohazard container.

3. List two types of circumstances when glucometry would be appropriate in the prehospital setting, and indicate its place in the treatment of the patient:

- Known diabetic patient with a decreased level of consciousness, after primary survey is managed and patient is stable or en route. It should never delay other treatment or transport.

- Patient unconscious for unknown reason, after primary survey is managed and patient is stable or en route. It should never delay other treatment or transport.

UNCONSCIOUSNESS

1. Describe the ideal position for an unconscious patient without spinal injury:

-The priority for the treatment of any unconscious patient is the maintenance of the airway. Placing the patient left lateral recumbent, that is lying on the patient’s left side with the left arm raised above the head, allows for drainage of any fluids from the mouth, as well as easy suctioning. It may also be helpful to lower the patient’s head about six inches below the feet. This is sometimes referred to as the coma position.

2. List and describe the most common acronym used to assess causes of unconsciousness:

A: Alcohol/acidosis/AMI/altitude

E: Epilepsy/environment/electricity

I: Infection

O: Overdose

U: Uremia (Toxic state of the blood caused by renal shutdown)

T: Trauma

I: Insulin

P: Psychosis/poisoning

S: Stroke/seizure

3. List the management priorities for a patient who is unconscious for unknown reasons:

- Secure and maintain the patient’s airway, providing high flow oxygen and BVM if necessary.

- Monitor the patient’s vitals, providing CPR or treating for shock as necessary.

- Obtain a thorough medical and situational history, investigating all of the incidents leading up to unconsciousness.
- Obtain a BGL
- Continue to closely monitor the patient's vitals.
- Rapid transport with ILS/ALS intercept.

4. List at least 3 common causes of airway obstruction in the unconscious patient:

- Relaxation of the muscles of the throat and **tongue**, which then occludes the airway.
- Dentures
- Vomitus
- Mucus
- Food
- Blood clots
- Foreign objects – particularly in children

RESPIRATORY EMERGENCIES

1. Define dyspnea:

- *Dyspnea is a feeling of air hunger, or shortness of breath, accompanied by labored breathing. It can be caused by several different problems.*

2. Explain the importance of tidal volume and minute volume:

- *The adequacy of breathing is determined by evaluating these two parameters, tidal volume and minute volume. The tidal volume times the respiratory rate is minute volume. The normal respiratory rate, approximately 12-20 per minute, and normal, at rest, tidal volume of approximately 500 ml gives us a normal minute volume of 6000 to 10000 ml. It is impossible to accurately estimate tidal or minute volume in the field, but generally, a well-defined visible chest expansion indicates a tidal volume of 700 ml in the average adult. Two of the most important things an EMT can observe in the dyspneic patient are the patient's tidal and minute volumes. In the case of assisting the respirations of a severely dyspneic patient, watching the chest for well-defined expansion can help the EMT make sure that adequate tidal volumes are being delivered.*

3. Define Chronic Obstructive Pulmonary Disease (COPD) and give two examples:

- *Chronic Obstructive Pulmonary Diseases are those characterized by the diffuse obstruction to airflow within the lungs. The most common are emphysema, chronic bronchitis, and asthma. COPD affects up to 20% of the adult population, and the changes caused by the diseases in pulmonary structures are chronic, progressive, and irreversible. (Some texts will not include asthma as a COPD, since it is reversible.)*

4. Describe the pathophysiology, signs & symptoms and field management for each of the following causes of dyspnea:

Pulmonary Edema: Acute pulmonary edema occurs when an excess of fluid builds up in the extravascular tissues in the spaces of the lungs. There are two general kinds of pulmonary edema. Cardiogenic pulmonary edema occurs when the heart is not pumping effectively. This is usually, but not always, a gradually occurring problem. It typically results from disease, MI, CHF, and hypertension. Pulmonary edema from non-cardiogenic causes include near drowning, aspiration pneumonia, smoke inhalation, infections, or inhalation of toxins. Generally speaking, acute pulmonary edema is caused by AMI, sudden hypertension, non-compliance with cardiac medications, dysrhythmias, too much salt, pregnancy, anemia, and too rapid infusion of IV fluid.

The SxS of pulmonary edema include:

- SOB
- Rapid, labored breathing
- Rales (crackles), rhonchi, and wheezing on auscultation of chest
- Cyanosis
- Frothy pink, blood-tinged sputum
- Distended jugular veins
- Rapid pulse
- Cool, clammy skin
- Restlessness & anxiety
- Exhaustion

Treatment of pulmonary edema includes:

- Aggressive, vigorous maintenance of the airway and ventilatory assistance. This includes suctioning as necessary, oxygen in the highest amounts possible (humidified if possible), and BVM assistance as needed.
- Close monitoring of patient's vitals
- Keep the patient's head and shoulders elevated, sitting the patient upright and supporting him/her with pillows. Lying the patient down allows the fluid to affect more of the surface area of the lungs, and can contribute to a more rapid deterioration of the patient.
- Restrict the patient's movement as much as possible, to conserve energy.
- Rapidly transport the patient to the medical facility and consider ALS intercept.

Pneumonia: The term pneumonia is used to describe a group of illnesses that are characterized by lung inflammation and fluid or pus filled alveoli. It is most frequently caused by a bacterial or viral infection, but it can also be caused by fungal infections, inhaled irritants (chemicals, smoke), or aspirated materials (vomit, food).

SxS:

- Chest pain, often worse on inspiration or expiration

- SOB, often with acute distress
- Noisy breathing, with a possibility of hearing rales, rhonchi, or wheezes when auscultating
- Productive cough with purulent yellow sputum or mucus, sometimes streaked with blood, or a rust colored sputum
- Pneumonia in the lower lobes of the lungs may not produce a cough, and can also present as abdominal pain.
- High fever
- Teeth chattering chills

Treatment of Pneumonia:

- Aggressive maintenance of airway and breathing, including high flow, humidified oxygen, suctioning, and BVM as needed.
- Place patient in position of comfort, usually sitting up or semi-sitting.
- Transport

Pulmonary Embolus: Pulmonary embolism is the sudden blocking of a pulmonary artery or one of its branches by a clot or other small particle carried by the blood. A pulmonary embolus can arise from several different sources, including air entering the circulation through an open wound; blood clots forming in a vein, breaking loose and moving through the venous circulation through the right side of the heart, becoming stuck in the progressively narrowing network of pulmonary arteries; and fat particles which enter the bloodstream from the end of broken bones; and is suspected from a patient's medical history, since the physical findings can be so variable and non-specific. It is a very difficult diagnosis to make, even in the Emergency Department. There are a variety of factors that may lead to pulmonary embolism. They include:

- Recent surgery, especially gynecological surgeries
- Prolonged immobilization and long periods of physical activity, which cause the blood to become stagnant in the lower extremities (i.e. long flights, bedridden). Immobilization of a lower extremity in a cast can have the same effect.
- Smoking
- Pregnancy and/or traumatic or difficult childbirth; these can cause amniotic fluid embolism
- Thrombophlebitis (inflammation of the veins), especially in the legs and pelvis, usually characterized by calf pain and tenderness.
- COPD
- CHF
- Cancer
- Use of certain drugs, especially oral contraceptives in women while smoking cigarettes.
- Chronic atrial fibrillation
- Multiple fractures of long bones
- Open wounds involving arteries or veins, especially in the neck.
- Obesity

SxS:

Remember that the SxS of a PE vary from patient to patient, and that any person at high risk who suddenly develops unexplained dyspnea or chest pain should be suspected to have a pulmonary embolus.

- Sudden severe SOB and respiratory distress, with cyanosis
- Patients will often, but not always, complain of sharp chest pain, possibly worse on breathing and coughing. It is usually described as a stabbing type pain.
- Approximately half of the patients will have a cough, sometimes with hemoptysis.
- Tachycardia
- Hypotension and postural syncope
- Distended jugular veins
- Breath sounds will usually be equal with good air movement heard. The lung is being properly inflated and ventilated, but the clot is hindering blood flow to the lung, therefore severely impairing oxygen and carbon dioxide gas exchange.

Treatment:

- Aggressive and vigorous maintenance of airway and breathing, including high flow oxygen, suctioning, and BVM as needed.
- Monitor the patient's vital signs closely
- Transport ASAP

Asthma: Asthma is characterized by an increased sensitivity of the trachea, bronchi, and bronchioles to various stimuli, with widespread, reversible narrowing of the airways, which is referred to as bronchospasm. The airway is further compromised by the swelling of the mucus membranes lining the bronchioles, and the bronchi become plugged by the production of thick mucus. Asthma may be allergic (extrinsic) asthma, which is usually a reaction to dust, pollen, or other irritants in the atmosphere. Asthma may also be non-allergic (intrinsic), which is due to infections, emotions, inhaled fumes, aspirin, cold air, exercise, or some other irritant. Precipitating factors are not identified in many cases. Status asthmaticus is a severe, prolonged asthmatic attack that does not respond to aggressive treatment, and is a true medical emergency.

SxS of an asthma attack include:

- Pt in an orthopneic, or "tripod" position; that is, sitting upright, leaning forward, fighting to breathe
- Spasmodic, unproductive cough
- High-pitched wheezing can be heard, on exhalation and inhalation, often with or without auscultation of chest.
- Breath sounds may present as very diminished or even silent due to little air movement
- Hyper-inflated chest, due to the trapping of air in the lungs, which is a result of increased obstruction on exhalation
- Rapid and shallow respirations
- Use of accessory muscles for breathing
- Rapid pulse

- Fatigue...at times to the point of apnea

Treatment:

- Aggressive and vigorous maintenance of airway and breathing, including high flow HUMIDIFIED oxygen if possible, BVM if necessary.

- Keep patient as calm as possible, and monitor vitals.

- Administration of patient's own MDI after contacting medical control. OR,

- Administering Nebulized Albuterol. The dose is 2.5 mg to 5.0 mg of the pre-diluted albuterol for adults, and 2.5 mg of the pre-diluted albuterol for children, nebulized at a rate of 6-8 lpm.

- In patients who have taken their own inhaler or have received nebulized albuterol, but are not improving (status asthmaticus), the EMT should contact Medical Control for orders to utilize the Epi-Pen that their service carries, and administer 0.3 mg of Epinephrine 1:1000. They also have the option of drawing up the Epinephrine 1:1000 into a dose restricted 0.3 cc syringe and administering it subcutaneously. For pediatric patients, it is recommended the use of an Epi-Pen Jr., which delivers 0.15 mg. If medical control is not possible, this may be written into their protocols.

- Rapid transport ASAP, - Consider ILS/ ALS intercept

Emphysema: This condition is characterized by the loss of elasticity in the alveoli, which become distended with trapped air. This trapped air allows the walls of the alveoli to lose their integrity and break down. The alveoli collapse, causing a decrease in surface area for gaseous exchange, and the patient has a difficult time getting enough oxygen. In the later stages of the disease, this same process begins destroying the larger pulmonary airways. Pt's are usually thin, and often have a barrel shaped chest. They usually have a history of increased SOB with exertion, with a non-productive or minimally productive cough. Exhalation is usually prolonged and difficult, and the chest often appears inflated even after exhalation. These patients usually are aware of and deal with their disease on a daily basis. If a call to EMS is made, it is usually due to some type of change, and is often due to acute decompensation, frequently from pulmonary infection.

Chronic Bronchitis: This condition is characterized by inflammation, edema and excessive mucus production in the bronchial tree. This mucus clogs up the bronchi, not allowing proper inflation of the alveoli. The chronic infections also cause scar tissue in the bronchi, which further narrows the airways. The typical patient with chronic bronchitis has almost always been a smoker, and is usually overweight. The signs and symptoms of chronic bronchitis are similar to emphysema, except that these patients often have a very productive cough.

SxS of COPD Decompensation:

- Gasping for air and shortness of breath

- Sitting, leaning forward in an attempt to breathe (tripod position)

- Distended neck veins

- Audible rales, rhonchi, wheezes

- Cyanosis may be present
- Prolonged exhalation with pursed lips

Treatment of Chronic Bronchitis & Emphysema:

- There has always been a concern that giving COPD patients oxygen would cause a decrease in the respiratory drive of the patient, to the point of apnea. The reason for this is what is referred to as “hypoxic drive” – the oxygen-based respiratory drive that exists in some COPD patients as opposed to the CO₂ based drive that exists in the normal person. What is interesting is that at least two studies (Aubier et al, 1980; Sassoon et al, 1987) came to the conclusion that COPD patients have, if anything, a higher than normal drive to breathe, and oxygen does not affect that. While these studies are provocative and thought provoking, it is probably prudent to give only enough oxygen to relieve the patient’s SOB. If the patient is very SOB/cyanotic when you arrive, then give them high flow oxygen. If the patient begins feeling better, then decreasing the amount of oxygen the patient is receiving may be better for the patient in the long run. If the patient stops breathing, it may be due to fatigue or the progression of the disease, and not necessarily due to oxygen administration. Should apnea occur, first try coaching the patient to breathe, if possible. If unsuccessful, then utilize the BVM and high flow oxygen. Realize that if these patients must continue to be bagged and ultimately intubated, their chances of having to remain on a ventilator are high. The most important point to remember is to NEVER WITHHOLD OXYGEN FROM ANY PATIENT WHO NEEDS IT!!!

- The treatment of the decompensating COPD patient includes:
 - Aggressive and vigorous maintenance of the airway and breathing, including high flow oxygen and BVM if necessary. Begin by titrating oxygen flow to patient need, and working up to more aggressive measures if needed.
 - Place the patient in position of comfort, usually sitting up.
 - Close monitoring of vitals, especially respirations, as the decompensating COPD patient can become so “closed down” that apnea can result. It almost certainly has nothing to do with the amount of oxygen being delivered.
 - Rapid transport, ILS/ ALS intercept

Hyperventilation Syndrome: This condition is caused by a prolonged period of breathing too rapidly. It is usually associated with anxiety. The patient is blowing off too much carbon dioxide, which causes alkalosis in the patient’s system. When caused by anxiety or psychological stress, it is usually rather harmless. It is important to remember that hyperventilation can result from any number of life-threatening medical problems (DKA, AMI, PE, etc), and that the EMT should take great care in finding the reason for the hyperventilation.

SxS of Hyperventilation:

- Air hunger & SOB
- Giddiness, strange behavior
- Fatigue
- Panic

- Dizziness, lightheadedness
- Numbness & tingling of hands, feet and mouth. This may progress to carpal pedal spasms, which will further panic the patient
- Dryness of mouth; sensation of lump in throat
- Stabbing chest pains & generalized chest discomfort
- Feeling of impending doom
- Syncope

Treatment of Hyperventilation:

- Rule out any life threatening cause for the hyperventilation. If there appears to be any reason to suspect a life threat, place the patient on high flow oxygen via a mask.
- Remain calm and reassuring for the patient, coaching the patient's breathing to a slower, more normal rate
- Having the patient "re-breathe" their own carbon dioxide, using a paper bag, is **not advisable**. There have been several cases of this treatment causing more harm than good, including death.
- Place patient on low flow oxygen with a cannula. This will not make the hyperventilation worse, and may psychologically help the patient.
- Transport the patient, continuing to reassure and coach the patient.

5. Describe the significance of the following breath sounds and give one example of when each might be heard:

Rales: Also called crackles, rales are usually fine, moist, crackling or bubbling sounds, similar to rubbing hair together near one's ear. They are associated with fluid in the alveoli (e.g., pulmonary edema, pneumonia). Typically, rales first appear at the base of the lungs and evolve to upper lobes.

Rhonchi: These rattling noises in the throat or bronchi are commonly due to partial obstructions of the larger airways by mucus or inflammation of the bronchi. They occur in situations such as advanced pneumonia, fulminating pulmonary edema, and decompensating COPD.

Wheezing: A wheeze is a sound that usually occurs on expiration and is the result of narrowed bronchioles. It is a high pitched, whistling type sound that lengthens the expiratory phase. More severe episodes can involve the inspiratory phase, and tend to be of a greater intensity. Wheezing is commonly associated with asthma and COPD, although pulmonary edema and foreign bodies can also cause wheezing.

Stridor: This high-pitched sound arises from the upper airway and usually occurs on inspiration. It commonly occurs in children suffering from croup or epiglottitis, both of which cause a narrowing of the upper airway. Stridor can also be heard in the case of some foreign body obstructions, anaphylaxis, and airway burns.

SUBSTANCE ABUSE AND OVERDOSE

1. Define the term “substance abuse”:

- Prolonged use of drugs that cause physical and mental impairment, including alcohol.

2. Define the term “addiction”:

- A state characterized by a physiologic adaptation to a substance which produces tolerance (increasing dosages are necessary to produce the desired effect) and dependence (user must continue dosages or withdrawal will result).

3. For the following list of commonly abused substances, give at least one example of the substance, describe the possible life threats associated with the abuse of the substance, the usual route of administration and the appropriate field management of a patient who has abused the substance:

	<u>Example</u>	<u>Life threat</u>	<u>Route</u>	<u>Tx</u>
Alcohol: (CNS depressant)	<i>beer</i>	<i>respiratory depression GI bleed, liver disease</i>	<i>oral</i>	<i>airway oxygen transport</i>
Amphetamines: (CNS stimulant)	<i>Benzedrine “Meth”</i>	<i>hyperthermia seizures CNS hemorrhage/stroke</i>	<i>oral injected inhaled</i>	<i>airway reassure transport</i>
Barbiturates: (CNS depressant)	<i>Amytal Phenobarbital</i>	<i>respiratory depression aspiration</i>	<i>oral injected</i>	<i>airway transport oxygen</i>
Cocaine: (CNS stimulant)	<i>coke crack</i>	<i>stroke AMI hyperthermia</i>	<i>inhaled smoked injected</i>	<i>ABC mgmt. transport</i>
Hallucinogens: (mind altering)	<i>LSD</i>	<i>reality separation</i>	<i>oral</i>	<i>reassure transport</i>
Phencyclidine: (hallucinogen)	<i>PCP</i>	<i>violence cardiorespiratory collapse</i>	<i>oral</i>	<i>keep calm transport</i>
Inhaled solvents: (CNS depressant)	<i>acetone toluene</i>	<i>cardiac arrhythmias kidney disease/metabolic disorders</i>	<i>inhaled</i>	<i>airway/O₂ transport</i>
Opiates/narcotics: (CNS depressant)	<i>heroin morphine codeine</i>	<i>respiratory depression</i>	<i>oral injected</i>	<i>airway/O₂ ALS/ILS transport</i>
Sedatives: (CNS depressant)	<i>Valium Haldol</i>	<i>respiratory depression aspiration of vomitus</i>	<i>oral injected</i>	<i>airway/O₂ transport</i>

4. List at least three other commonly abused substances:

- Aspirin, food, laxatives, over the counter cough and cold remedies, tobacco

5. Describe the field treatment for someone who has overdosed on an opiate, such as heroin:

- *Assure that the patient's airway is cleared and secured with an OPA or NPA. Do not place a MLA until further treatments have been unsuccessful. Assist the patient's ventilations if necessary with a BVM and oxygen, and assure that there is a pulse present. Prepare the patient for and initiate transport as soon as possible. As soon as feasible, draw up 0.4 mg of Naloxone (Narcan), and correctly administer into an appropriate intramuscular site. As an alternative, the EMT-Basic may utilize "nasal naloxone" which is delivered utilizing the Mucosal Atomization Device (MAD). To do this, prepare the MAD device per the manufacturer's instructions, and draw up 2 mg of naloxone (to do this, a concentration of 2mg dissolved in 2 cc is usually utilized). Deliver half of the dose in one nostril, and the other half in the other nostril, for a total of 1 mg in each nostril. Observe the patient for a return of spontaneous, adequate ventilatory effort. Prepare for the various side effects associated with this drug administration: nausea, vomiting, diaphoresis, violent behavior, and in rare cases, seizures. If ILS/ALS is available in the EMT-B's area, they should ask for intercept with that unit. Should the patient wake up, continue supportive care with oxygen therapy, and closely monitor the patient, as the Naloxone may wear off and the patient can relapse into a hypo-ventilatory state.*

6. Establish the relationship between the patient with an OD and airway management:

- *The airway should be secured as soon as possible with an MLA if appropriate. The exception to that is if the OD is an opiate, then an NPA should be used because the patient should be given Narcan and the patient's LOC will improve causing the patient to gag on the MLA/OPA.*

POISONING

1. List at least 4 questions specific to poisoning that an EMT should ask when assessing a patient who may have ingested a harmful substance:
 - *What was ingested?*
 - *How much was ingested?*
 - *Has the patient vomited?*
 - *Why was the substance taken?*
 - *Does the patient have an underlying medical or drug abuse history?*
 - *When was the substance ingested?*
 - *Was anything else taken?*
 - *Has an antidote been given?*

2. Explain the purpose of activated charcoal:
 - *Activated charcoal is a special distilled charcoal that's surface is covered with pores, which allows the charcoal to absorb many times its weight in contaminants. Activated charcoal binds to certain poisons, and facilitates elimination of these poisons. It will not bind to alcohol, kerosene, gasoline, caustics, or metals, such as iron.*
3. Explain the purpose of Poison Control and describe how it may be contacted. List the information about the patient that Poison Control will request:
 - *Poison Control is a resource center that can help you decide which first aid measures are a priority, and in coordination with medical control, can help you formulate an effective treatment plan. They also have information on any antidotes that may be available. They will want to know several things:*
 - *Patient's age and weight*
 - *General patient condition*
 - *What was ingested? How much and when?*
4. Explain the difference between Poison Control and medical control. Who may give orders regarding the treatment of the ingested poisoning patient?
 - *Poison Control is a resource center only. They can give an EMT invaluable information, but they cannot and do not replace medical control. An EMT's medical control is the only authority that can provide orders for a patient's treatment.*
5. Describe the field management of an ingested poisoning patient:
 - *Initiate rapid transport*
 - *Contact Medical Control*
 - *Save any emesis for examination at hospital*
 - *Support ABC's & be prepared to suction as necessary to make it easier to get the sample.*
 - *All contaminated material must be disposed of in a biohazard container.*

ALLERGIC REACTIONS and ANAPHYLAXIS

1. Explain the basic immune response, and how allergic reactions and anaphylaxis are variations of this response:
 - *An antigen (usually some type of foreign protein) enters the body. Immune system cells surround the antigen and design specific proteins of their own which combine with the antigen and inactivate it. These are called antibodies. Antibodies are placed on two different types of cells: basophils – which are stationed at fixed*

points within the body's tissues, and mast cells which "patrol" connective tissues. Mast cells and basophils contain histamines and other chemicals that are released when an antigen attaches to an antibody on the cell surface.

- Anaphylactic shock is the cardiovascular component of a condition called anaphylaxis. Anaphylaxis is the most extreme form of an allergic reaction in which the reaction is not local but systemic. It is an overreaction of the immune system. When released in large amounts, histamine and other chemicals cause vasodilation, leaking of capillaries into the interstitial space, and a drop in cardiac output, thus, the patient may be suffering from hypovolemia, redistributive, and cardiogenic shock. In addition, the histamines and other chemicals (especially one called leukotriene) may stimulate a severe response in the airway, which causes spasm of the bronchioles and swelling of the lower and upper airway structures.

2. List the SxS of anaphylactic shock and indicate which ones would help the EMT differentiate anaphylaxis from any other type of shock:

- Those SxS marked with an * are differential.

SKIN: (first to occur)

- * Flushing (from peripheral vasodilation)
- * Edema (from leaking capillaries – especially around eyes: periorbital edema)
- * Itching
- * Hives (urticaria)

RESPIRATORY:

- * Shortness of breath (from bronchoconstriction)
- * Chest tightness
- * Wheezes (reactive bronchioles/bronchoconstriction)
- * Feeling of swelling in throat (leaky capillaries in laryngeal area)
- Dry cough
- * Hoarse/Stridorous voice and breathing (from increasing laryngeal edema)

CARDIOVASCULAR SYMPTOMS:

- Tachycardia (from heart trying to compensate for the drop in cardiac output)
- Dysrhythmias (from lack of oxygen to the heart muscle)
- * Systemic peripheral vasodilation, leading to shock and decreased BP
- Hypotension (from plasma leakage, decreased cardiac output and vasodilation)

GI SYMPTOMS:

- Nausea and vomiting
- Cramps and bloating
- Watery profuse diarrhea

CNS SYMPTOMS:

- Headache, dizziness, confusion (from inadequate cerebral perfusion)
- Eventual decrease in LOC

***Please note: Although “differential diagnostic signs” are given here in an attempt to help the EMT differentiate anaphylaxis from other “shock” conditions, it is important to remember that a patient may not exhibit all of the “classic” signs of the condition. It is entirely possible to suffer a life threatening anaphylaxis with cardiovascular SxS as the ONLY problem...with no airway involvement. Conversely, life-threatening anaphylaxis can occur with airway compromise as the life threat, with no cardiovascular compromise occurring. It should be stressed to the EMT that should they be presented with a patient suffering anaphylaxis, epinephrine 1:1000 would be potentially life saving whether or not the patient’s main problem is cardiovascular compromise or airway compromise.*

3. Describe a situation where an ALS intercept would be appropriate in the case of severe anaphylactic shock:

- When airway is deteriorating rapidly, ALS intercept could provide intubation and medications (i.e. IV Benadryl) to deal more effectively with the condition. In addition, consider ALS intercept if the patient is experiencing solely cardiovascular SxS.

4. Briefly describe the two (2) main life-threats caused by anaphylaxis:

*- Airway closing off
-Hypotension from plasma leakage, decreased cardiac output and systemic vasodilation.*

NON-TRAUMATIC ACUTE ABDOMEN

1. Define the terms Peritoneum, Peritonitis and Peristalsis:

- The peritoneum is a smooth, membranous, sheath-like layer of tissue that lines the abdominal cavity. It is made up of two separate layers; the outer, or parietal layer lines the abdominal cavity wall. The inner, or visceral layer surrounds and helps support the abdominal organs.

- Peritonitis results when the peritoneum becomes inflamed, usually due to exposure to blood and/or abdominal contents, with subsequent infection.

- Peristalsis is the involuntary wavelike motion that occurs in the GI tract to move solids and liquids.

2. Define the term **acute abdomen**:

- A condition of sudden onset of pain within the abdomen, demanding immediate medical or surgical treatment.

3. List at least 6 possible causes of acute abdominal pain:
- Food poisoning
 - Gastritis/gastroenteritis
 - Gastric/duodenal ulcers
 - Gall bladder problems
 - Ruptured spleen
 - Perforated colon
 - Ectopic pregnancy
 - Twisted ovary (torsion)
 - PID
 - Colitis
 - Abdominal aortic aneurysm
 - Pancreatitis
 - Appendicitis
 - Kidney stone
 - Intestinal obstruction
 - Ovarian cyst
 - Cystitis
4. Describe the general signs and symptoms that may be present with an acute abdomen:
- Local or diffuse abdominal pain
 - Nausea & Vomiting
 - Local or diffuse abdominal tenderness
 - Anxiety
 - Reluctance to move
 - Hematemesis & bloody stools
 - Rapid pulse
 - Hypotension
 - Tense, rigid, or distended abdomen
 - SxS of shock, especially with peritonitis
 - Rapid shallow breathing
5. Define ectopic pregnancy and briefly describe the pathophysiology and the consequence of rupture as well as the appropriate field management of a patient with a suspected ectopic pregnancy:
- In an ectopic pregnancy, the egg is implanted outside the uterus – in the abdominal cavity, in the fallopian tube (95%), on the outside wall of the uterus, on the ovary, or on the outside of the cervix. When the egg is in the fallopian tube, it grows normally for the first few weeks, but the fallopian tube cannot stretch. The growing embryo and placenta eventually stretches the surrounding tissue, and causes rupture of the tube and/or nearby blood vessels, and results in severe abdominal bleeding and internal hemorrhage.*
- Signs and symptoms include:
- Sudden sharp abdominal pain, localized on one side. If the bleeding is extensive, the pain may become more diffuse.
 - Spotty vaginal bleeding
 - Missed menstrual period (usually 6 –10 weeks since last cycle)
 - Pain under the diaphragm, radiating to both shoulders
 - Tender, bloated abdomen, possibly with a palpable mass in the abdomen.
 - SxS of shock

- Suspect ectopic pregnancy in any woman of childbearing age if any of the above SxS are present, especially if there is a history of Pelvic Inflammatory Disease, IUD birth control device usage, and tubal ligation.

Treatment includes:

- Maintenance of airway and breathing, with high flow oxygen via non-rebreather or BVM if needed
- Treat for shock
- Consider MAST on physician's order
- Rapid transport
- ALS intercept

6. Describe the field management of an acute abdomen patient:

- Vigilant observation and maintenance of the airway. Be alert for any vomiting, and attempt to save some of the emesis. Suction as needed.
- Allow the patient to be transported in position of comfort, if it is feasible, for patient care.
- Administer oxygen, preferably through a nasal cannula if the patient is nauseous or vomiting, at the highest practical rate.
- Give nothing by mouth
- Treat for shock
- Reassure the patient
- Monitor the patient's vitals, and document the history of the patient's pain
- Transport quickly and efficiently, as smoothly as possible

HYPERTHERMIA/HYPOTHERMIA

1. Describe the general pathophysiology of hyperthermia:

- Thermoregulation is controlled by the body via the hypothalamus. Rises in body temperature are counteracted with physiologic mechanisms to promote heat loss. Examples of these mechanisms include cutaneous vasodilation and sweating. Hyperthermia occurs when the thermoregulatory ability of the body is overwhelmed. Cutaneous vasodilation results in a greater demand on the heart, as cardiac output must increase to compensate for what amounts to an increase in the volume of the vascular system. As more and more blood is shunted to the peripheral vasculature, less blood is available to core organs – especially the brain. The blood that does reach the brain is higher in temperature, the net result being headache, dizziness and an eventual decrease in LOC. Additionally, as sweating continues, important electrolytes are depleted – resulting in muscle cramps and dehydration. Once the thermoregulatory ability of the body is overwhelmed, core body temperature can rise from normal to 106 Fahrenheit in 15 minutes or less.

2. List 10 factors that may predispose a person to hyperthermia:
 - Exercise
 - Obesity
 - Heavy, non-porous clothing
 - Diabetes
 - Dehydration
 - Certain meds such as: diuretics, tranquilizers, beta-blockers, phenothiazine, and antihistamines
 - High ambient temperature
 - High humidity
 - Cardiovascular disease
 - Alcoholism

3. Describe the pathophysiology, signs and symptoms and appropriate field management of heat cramps:
 - Essentially caused by an electrolyte imbalance resulting from excessive sweating without fluid and electrolyte replacement, heat cramps are manifested by cramping in larger muscle groups (i.e. quadriceps). Management is to orally replace fluids and electrolytes with diluted (i.e. half-strength) commercial products such as Gatorade, or water with a pinch of salt and sugar added. Remove patient from the hot environment and rest.

4. Describe the pathophysiology, signs and symptoms and appropriate field management of heat exhaustion:
 - Heat exhaustion is basically the next step in the progression of hyperthermia. There is an increase in the volume of the vascular system due to cutaneous vasodilation. Additionally, large amounts of fluid are lost via the sweating mechanism. The result is a patient who appears mildly shocky (e.g. pale, cool, clammy, nausea, etc). Treatment is aimed at removing the patient from the hot environment, cooling the patient and orally replacing fluids and electrolytes. Generally, these patients should be transported if EMS is called.

5. Describe the pathophysiology, signs and symptoms and appropriate field management of heat stroke:
 - The progression of hyperthermia culminates in the life threatening condition known commonly as heat stroke. Sustained core temperatures of 106° F and above are responsible for cell and tissue destruction in a variety of organs (brain, heart, liver). Heat stroke can be considered passive (i.e. patients with predisposing conditions or medications) or exertional (exertion in a hot environment). Field management is **immediate**, aggressive cooling of the patient and rapid transport with airway management and oxygen as needed. Wetting the patient down and then providing directed airflow via fan or air conditioning provides effective cooling from evaporation. If limited water is available, then key areas such as the head and other hot spots should be cooled first. Care should be taken not to cause shivering (a heat production mechanism), or to rapidly vasoconstrict the cutaneous vasculature in the skin by applying ice directly to the skin. This prevents the reactivation of the normal

cooling process. Clinically it is easiest to differentiate heat stroke from heat exhaustion based on mental status. Heat stroke patients may range from mildly confused to comatose with seizures. Heat stroke has a 70% mortality rate and is a true life threatening emergency. Brain and vital organ cell/tissue death begins in approx. 15 minutes after reaching 106° F.

6. Identify the differentiating sign that indicates heat stroke:

- Decreasing level of consciousness is the differentiating sign in hyperthermia. Core temperature is obviously another differentiating sign, but changes in LOC are much more easily observed in the field. Other vital signs may be variable, and skin color and temperature are NOT considered diagnostic. Patients in heat stroke may present with hot, flushed, damp skin if they have progressed from heat exhaustion to exertional heat stroke.

7. Describe the general pathophysiology of hypothermia:

- Thermoregulation is controlled by the brain via the hypothalamus. The body counteracts to a drop in temperature via several mechanisms: shivering, cutaneous vasoconstriction, and an increase in basal metabolic rate. However, these mechanisms can be overwhelmed by cold, and once the body's core temperature drops below 95° F, thermoregulation begins to falter. Heat loss may occur via radiation, evaporation, perspiration, convection and conduction (cold water conduction increases rate of heat loss up to 25 times).

8. Describe the signs & symptoms and appropriate field management of mild hypothermia:

- Mildly hypothermic patients will still be conscious – although they may be lethargic. Skin is probably pale and cold and the patient may be violently shivering. Field management is aimed at active external and internal rewarming of the patient. In an outdoor environment this means moving the patient indoors or to a protected area or other shelter. Replace wet clothing with dry garments and wrap in blankets and other insulating materials. Give warm fluids by mouth (avoid caffeine and alcohol) and encourage carbohydrate replacement.

9. Describe the signs and symptoms and appropriate field management of severe hypothermia:

- Severely hypothermic patients will be anywhere from a P to a U on the AVPU scale. Shivering is not present, and the skin is pale and ice cold. All vital signs will be extremely depressed – often to the point of being practically undetectable. Field management involves carefully transporting the patient to a medical facility where rewarming can take place. The patient should be handled very gently. Care should be taken to maintain an airway (OPA's, NPA's, LMA's and MLA's should be placed with care as stimulation of the gag reflex could precipitate

fibrillation). *If respirations are entirely absent, the patient should be ventilated at **normal rates**. Warmed, humidified oxygen should be administered if available. Pulse checks should take at least 1 minute, and chest compressions started only if the patient is pulseless. Cut away any wet clothing and cover with dry blankets. Do not actively rewarm the patient, but prevent further heat loss.*

10. Briefly explain why it is dangerous to rewarm or even jostle a severely hypothermic patient in the field:

- Severely hypothermic patients must be rewarmed from the core – something that is impossible in a field setting. If the body’s “shell” is rewarmed before the core, vasodilation occurs and blood from the core is shunted back out to the periphery. The heart is still too cold to deal with this increase in circulation and it sustains further damage when cold, acidotic blood returns from the periphery. This phenomenon, known as afterdrop, usually results in ventricular fibrillation. Similarly, rough handling or jostling of the patient may also precipitate ventricular fibrillation, a lethal cardiac rhythm.

11. Describe the pathophysiology, signs and symptoms and appropriate field management of frostbite:

*- Frostbite occurs with the local freezing of tissue. Like burns, frostbite can vary in depth and intensity depending on the duration and severity of exposure. Most commonly affected areas include the hands, feet, ears, and nose. Signs and symptoms of “superficial” frostbite include white, waxy appearing cold skin. The patient usually reports a loss of sensation in the affected area. As thawing occurs, the injured area will turn color from leakage of plasma from damaged capillaries. Deep frostbite is hard. Field rewarming should be avoided in both cases if possible; rewarming is best initiated under controlled circumstances. Frostbitten extremities should be protected from further trauma, and if spontaneous rewarming occurs, the affected area **MUST** be protected from refreezing.*

12. Explain how frostnip differs from frostbite:

- Frostnip refers to the earliest stages of cold injury in local tissues and signals a loss of local tissue perfusion. Early indications of frost nip are numbness and a blanched appearance of the affected area. Unlike frostbite, the tissue in a frostnipped area is still soft and pliable. Prompt rewarming of the affected area will prevent progression to frostbite, and will not result in serious disability or tissue loss. Effective rewarming methods for frostnip include skin-to-skin contact, warm (not hot) water immersion, and removal of constrictive materials (e.g. remove boots).

13. Identify the risks/concerns involved in thawing a frostbitten part in the pre-hospital setting:

- *Rewarming in a field setting is usually impractical for the following reasons:*
- *Rewarming requires a constant supply of warm water at a temperature of 105-108 degrees F. Immersion of a frostbitten part will rapidly cool the water – which will need to be replenished.*
- *A container is necessary which is large enough to bathe the affected area without the skin making contact with the sides of the container.*
- *The patient will suffer extreme pain during the rewarming process. Over the counter pain medications are inadequate in this situation.*
- *The potential for refreezing is greater in the field setting.*
- *The potential for trauma to the affected area is greater in the field setting.*

WATER EMERGENCIES

1. Explain the Mammalian Diving Reflex and the factors that influence the effectiveness of MDR:

- *Essentially, the MDR is thought to be a primitive reflex that occurs to protect the brain and other vital organs during periods of cold water submersion. It is activated by cold water on the face which results in a parasympathetic response. In effect, the body becomes a “metabolic icebox” – meaning that total body metabolism is reduced. This results in a decreased need for oxygen and decreased tissue damage. Factors that seem to influence the effectiveness of MDR include the temperature of the water (the colder the better), the age of the victim (MDR more effective in younger victims) and the length of submersion (survival rates decrease dramatically after 1 hour).*
- *Essentially, the reason for teaching students about MDR and submersion hypothermia is to stress that successful resuscitation is possible after long submersion times in cold water and that the old maxim may hold true: “No patient is dead until they’re warm and dead.”*

2. Describe the pathophysiology of near drowning:

- *Near drowning refers to submersion with at least temporary survival afterward.*

The pathophysiology of near drowning follows a sequence:

- *Victim is submerged and begins to cough and gasp, swallowing water.*
- *A small amount of water may be aspirated into the larynx resulting in laryngospasm – a temporary protection from further aspiration.*
- *Laryngospasm results in asphyxia, which leads to a loss of consciousness. If the patient dies at this point (10-15%), death is by suffocation, as the lungs are still dry.*

- *Hypercarbic and hypoxic drives stimulate inhalation or laryngospasm relaxes and water enters the lungs, ultimately resulting in continued hypoxia, brain damage and cardiac arrest.*
- *Treatment centers around ABC's as usual. Note that if the patient regains consciousness in the field they should still be transported to a medical facility and observed for 24 hours.*

3. Define the term **laryngospasm**:

- *Laryngospasm may be triggered by water in the larynx or trachea and results in spasms of the laryngeal muscles, which effectively seal off the trachea.*

4. Describe how an air embolism may occur in a diver who ascends too rapidly to the surface:

- *As a diver ascends, the pressure surrounding him/her will decrease and the gases within the lungs will expand. For this reason divers are taught to exhale throughout ascent in order to vent air from their lungs. Failure to do this (e.g. breath-holding) may result in pulmonary over pressurization syndrome (POPS) also known as "burst lung". Rupture of alveoli in turn may result in air embolism – after drowning, the second leading cause of death among divers. Air bubbles from the ruptured alveoli enter the pulmonary capillaries and merge together to become larger bubbles as they progress through the pulmonary veins and back to the left side of the heart. The majority of air emboli then proceed to the cerebral circulation causing neurological signs and symptoms.*

5. List the signs and symptoms and explain the field management of air embolism:

- *Signs and symptoms of air embolism tend to present dramatically – usually seconds or minutes after a diver surfaces and takes his/her first breath.*

These SxS include:

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| - <i>Dyspnea</i> | - <i>Weakness</i> |
| - <i>Partial paralysis</i> | - <i>Seizures</i> |
| - <i>Unconsciousness</i> | - <i>Other neurologic symptoms</i> |
| - <i>Rapid onset (less than 10 min.)</i> | - <i>(Blindness, deafness, paresthesia)</i> |
| - <i>Blood-tinged frothy sputum</i> | - <i>Sub-Q emphysema</i> |
| - <i>SxS pneumothorax (99% of all cases)</i> | |

- *Field management consists of aggressive airway management, administration of 100% oxygen, rapid transport in left lateral recumbent (or supine, depending on need for airway management) position with gurney in full body tilt/head down (30 degree tilt) to decrease the possibility of emboli traveling to brain. Notify Medical Control to make arrangements for recompression at a hyperbaric chamber.*

6. Describe how decompression sickness (“the bends”) may occur in a diver who ascends too rapidly to the surface:

- As a diver descends, the gases that he/she is breathing (e.g. oxygen, nitrogen) will go into solution (dissolve in the blood stream). As a diver ascends, nitrogen will come out of solution and reform as a gas. A slow enough ascent will allow the amount of nitrogen in the tissues to equilibrate with that in the alveoli. However, if the ascent is too rapid to allow the nitrogen to be removed via exhalation, then the nitrogen bubbles will accumulate in the diver’s tissues and/or vascular system. This will interfere with tissue perfusion and cause chemical changes within the body.

7. List the signs and symptoms and explain the field management of decompression sickness:

- Decompression sickness can be difficult to distinguish from air embolism if the primary presentation involves neurological symptoms. Generally, air embolism tends to involve cerebral function, while decompression sickness is more likely to involve the spinal cord.

- The general SxS of decompression sickness include:

- Skin rash/itching*
- Muscle/joint pain*
- Multiple sensory/motor deficits*
- Incoordination, vertigo*
- Delayed onset (1-12 hours)*

- Field management is similar to that of air embolism, with the primary goal aimed at the patient’s rapid transport to a facility with a hyperbaric chamber for recompression. 100% oxygen should be administered to avoid increasing the patient’s nitrogen intake.

8. Identify the **definitive** treatment for both air embolism and decompression sickness:

- Both air embolism and decompression sickness require recompression in a hyperbaric chamber. A hyperbaric chamber works by increasing the atmospheric pressure within the chamber (as if the diver were descending under water), which allows the gases to go back into solution. The pressure is then decreased slowly at a calculated rate in order to allow the gas bubbles to be blown off via the lungs.

BITES and STINGS

1. List the signs and symptoms of envenomation and explain the field management for each of the following:

- Pit viper: SxS of envenomation vary depending on the amount of venom deposited and the protein content of the venom. Some estimates show that no envenomation occurs in about 22% of all pit viper bite victims. SxS may include fang marks, pain and edema at the bite site (within 5 minutes). Ecchymosis occurs within several

hours. Severe envenomation (particularly if the venom is injected directly into the vascular system) may result in rapid hypotension and shock. Other indicators of significant envenomation include paresthesia of the scalp, face and lips. Weakness, sweating, nausea and faintness may present, and some patients have complained of a metallic taste in the mouth. Hemorrhaging can manifest as epistaxis, hematemesis, and hemorrhagic blebs at the bite site.

Field management is aimed at rapid evacuation to a treatment facility, keeping the patient calm and removing any constricting articles of clothing or jewelry. If the affected area is an extremity, that extremity should be kept at a level lower than the heart and exercise of that extremity prevented (if possible) by immobilization. The use of constricting bands proximal and distal to the bite site have become controversial, as there is no valid evidence that constricting bands above and below the bite remain part of the field management.

Black Widow: A black widow spider bite may result in a small red papule without local pain or swelling. Systemic SxS may occur within 1 hour of envenomation and may include extremely painful muscle spasms and cramps, a rigid abdomen (non-tender), and burning sensations that occur most frequently and intensely in the soles of the feet. Other associated SxS include nausea, vomiting, facial edema, dizziness, headache, slurred speech, diaphoresis, and mild fever. Serious complications include seizures and hypertension. Recovery is usually complete after a few days, however, the very old, young or already compromised patient may be at a greater risk. Field management includes rapid transport to a medical facility, ABC management as necessary, and local cleansing of the bite site.

Brown Recluse: A brown recluse spider bite will initially result in a raised papule. One to two days later, the papule will “slough” off forming an open ulceration characterized by irregular borders and necrotic tissue. If left untreated, severe scarring occurs. Only 5% of brown recluse bites result in a life threatening systemic reaction.

Bee/Wasp and Hornet: Stings from these insects always result in local, painful reactions. In a few instances a patient may experience a serious life threatening anaphylactic reaction.

2. Describe the correct procedure for removing a stinger that is still embedded in a patient’s skin:
 - Using a flat, rigid surface (e.g. a credit card), gently scrape the stinger from the skin. This avoids depositing additional venom as occurs when the stinger is pinched with tweezers.