CLINICAL INFORMATION
Atrial fibrillation occurs when the atrial chambers of the heart contracting rapidly and irregularly. Abnormalities in the electrical activity are responsible for atrial fibrillation. Rapid and irregular discharges cause ineffective contractions of the atria, reducing the ability of blood to pump into the ventricles.

ASSESSMENT AND TREATMENT CONSIDERATIONS

- **Atrial flutter** is much less common than AF, but its hemodynamic consequences and management are similar.
- Atrial flutter is from a large reentrant circuit which makes a loop inside the right atrium and often has a regular atrial rate of 300 bpm. The P waves of atrial flutter appear as a saw-tooth baseline, often best seen in lead II. While atrial fibrillation produces a rapid, irregular heartbeat, atrial flutter symptoms are characterized by a regular, although very rapid, heartbeat.
- Patients with AF over 48 hours are at risk for clot formation within the left atria and stroke can occur when electrical conversion is used. Unless an unstable patient with rapid AF or flutter is present, synchronized cardioversion should be avoided until the patient can be anticoagulated.
- Atrial fibrillation and atrial flutter are rarely seen in children and are often seen following surgery for complex congenital heart disease.
- Cardioversion is not effective for treatment of multifocal atrial tachycardia (which can mimic AF) because this rhythm is due to cells that spontaneously fire at a rapid rate. Delivery of a shock generally cannot stop these rhythms and may increase the rate of the tachyarrhythmia.
- **Serious signs and symptoms:** altered mentation, hypotension, hypoperfusion, congestive heart failure, or acute myocardial ischemia is present.

MEDICATION CONSIDERATIONS

- Adenosine is **not** to be given in any **wide-complex rhythm** (> than 0.12 sec or 3 small boxes) as sudden and extremely rapid heart rate acceleration leading to VF can occur when an accessory pathway (WPW) is being used in the presence of AF.
- Nitroglycerin administered during rapid AF or flutter or SVT can cause further heart rate acceleration from reflex tachycardia secondary to vasodilation. Contact Biocare Medical Control for guidance in these patients before using nitroglycerin.
- Adenosine is **not** effective in converting atrial fibrillation or atrial flutter and is **not** to be used when atrial fibrillation / flutter is known with certainty to exist.
- Use of sedation for cardioversion is left to the discretion of the provider and is based upon several factors: patient’s hemodynamic status, mental status, and severity of tachycardia.
- **Volume loading is secondary therapy in tachydysrhythmias** (rate slowing is first) unless obvious hypovolemia secondary to fluid loss is present.

POTENTIAL PITFALLS

- The administration of nitroglycerin **prior to rate control** in patients with rapid rates can cause reflex tachycardia from peripheral vasodilatation with further heart rate acceleration.
- Administration of lidocaine in atrial fibrillation or atrial flutter as its quinidine-like effects can cause an alarming heart rate (ventricular) acceleration to occur.
- Sedation in severe hypotension (SBP < 70) unless absolutely necessary (medic discretion).
- Failure to consider the side-effects of antiarrhythmic agents including proarrhythmia, exacerbation of a preexisting arrhythmia, or provocation of a new arrhythmia. Proarrhythmia can be bradycardic or tachycardic, atrial or ventricular in nature.
- Delivering multiple synchronized shocks when a rhythm is short lived or recurs frequently. The use of multiple shocks in short lived or repetitive tachyarrhythmias are contraindicated.
Monomorphic ventricular tachycardia (VT) is a regular rhythm with identical QRS complexes tachydysrhythmia that originates from the ventricles at a rate over 100 bpm (usually over 150 up to 220 bpm) and wide QRS complexes (over 0.12 sec). VT may be either well tolerated or life-threatening.

**ASSESSMENT AND TREATMENT CONSIDERATIONS**
- Nonsustained VT is less than 30 seconds duration; over 30 seconds is considered sustained VT.
- Wide complexes tachycardias are often very difficult to sort out (even by those who read ECGs daily) since such tachycardias could either be a SVT with aberrancy or ventricular tachycardia.
- Heart disease (ischemia, acute or past MI, CHF etc.) is present in over 90% of all episodes of ventricular tachyarrhythmia. If your patient has heart disease and is in a rapid and wide-complex tachyarrhythmia, it is most likely VT.
- **Stable VT patients** are those who do not have hemodynamic decompensation, ongoing chest pain, altered mentation, shock, or CHF. Because stable VT can (and often will) deteriorate into an unstable state early prehospital treatment is advocated to prevent cardiovascular collapse.

**MEDICATION CONSIDERATIONS**
- Amiodarone has multiple effects on myocardial depolarization and repolarization that make it an extremely effective antiarrhythmic and the first drug of choice for VT.
- Amiodarone is **NOT** to be used in VT from tricyclic antidepressant (TCA) or fast sodium channel blocker (FSB) toxicity (as these drugs all block sodium and widen the QRS):
  - **Common TCAs** - imipramine (Tofranil), amitriptyline (Elavil), desipramine (Norpramine), nortriptyline (Pamelor), trimipramine (Surmontil), protriptyline (Vivactil), doxepin (Adapin), and clomipramine (Anafranil).
  - **Common FSBs** - disopyramide (Norpace), quinidine (Quinidex), procainamide (Pronestyl), cocaine, flecainide (Tambocor), propafenone (Rhythmol), amiodarone (Cordarone), sotalol (Betapace), encainide (no name), moricizine (Ethmozine).
- **WARNING:** Do not use lidocaine when VT is from lidocaine, phenytoin (Dilantin) or mexiletine (Mexitil) as it can worsen toxicity (contact Biocare Medical Control for guidance).
- Lidocaine reaches the central circulation within 2 minutes and patients should be observed for toxicity which includes; slurred speech, altered mentation, twitching, seizures, and bradycardia.
- Amiodarone is typically given at a rate of 150 mg over 10 minutes in the stable patient; however when urgently needed to control a persistent tachydysrhythmia the dose is adjusted to 300 mg over 10 minutes to attempt to rapidly interrupt the tachydysrhythmia.
- Amiodarone infusions may be made by adding 100 mgs of Amiodarone to a 100 mL bag of NS or buretrol yielding a 1 mg per mL concentration which is infused at 60 gtts per minute to provide 1 mg/min (always be sure to remove an equal amount of fluid from the bag before adding the drug).
- Sodium bicarbonate is used for sodium channel blocker or cocaine related VT to correct the sodium blockade of cardiac cells which leads to widened QRS, hypotension and arrhythmias; in addition it alkalinizes the blood which binds any unbound (free) drug then removing it in the urine.

**POTENTIAL PITFALLS**
- Initiating a post conversion maintenance infusion when profound hypotension, second or third degree AV block, rate less than 50 bpm, idioventricular or ventricular escape rhythms are present.
- Failure to promptly treat hemodynamically compromised patients while spending excessive time attempting to first secure a definite rhythm diagnosis.
- Failure to remember that stable VT can degenerate and should be treated when present.
- Administering nitroglycerin during VT can and most likely cause cardiovascular collapse with further heart rate increase due to vasodilation; do not use NTG during VT.
Monomorphic ventricular tachycardia (VT) is a regular rhythm with identical QRS complexes tachydysrhythmia that originates from the ventricles at a rate over 100 bpm (usually > 150 up to 220 bpm) and wide QRS complexes (over 0.12 sec). VT may be either well tolerated or life-threatening.

**ASSESSMENT AND TREATMENT CONSIDERATIONS**

- Nonsustained VT is less than 30 seconds duration; over 30 seconds is considered sustained VT.
- Wide complexes tachycardias are often very difficult to sort out (even by those who read ECGs daily) since such tachycardias could either be a SVT with aberrancy or ventricular tachycardia.
- Heart disease (ischemia, acute or past MI, CHF etc.) is present in over 90% of all episodes of ventricular tachyarrhythmia. If your patient has heart disease and is in a rapid and wide-complex tachyarrhythmia, it is most likely VT.
- **Stable VT patients** are those who do not have hemodynamic decompensation, ongoing chest pain, altered mentation, shock, or CHF. Because stable VT can (and often will) deteriorate into an unstable state early prehospital treatment is advocated to prevent cardiovascular collapse.
- **Serious signs and symptoms:** altered mentation, hypotension, hypoperfusion, congestive heart failure, or acute myocardial ischemia is present.

**MEDICATION CONSIDERATIONS**

- Amiodarone has multiple effects on myocardial depolarization and repolarization that make it an extremely effective antiarrhythmic and the first drug of choice for VT.
- **Amiodarone is NOT to be used in VT from tricyclic antidepressant (TCA) or fast sodium channel blocker (FSB) toxicity (as these drugs all block sodium and widen the QRS):**
  
  Note: this list may not be all inclusive, consult a PDR to determine drug type and its effects
  
  - Common TCAs - imipramine (Tofranil), amitriptyline (Elavil), desipramine (Norpramine), nortriptyline (Pamelor), trimipramine (Surmontil), doxepin (Adapin), and clomipramine (Anafranil).
  - Common FSBs - disopyramide (Norpace), quinidine (Quinidex), procainamide (Pronestyl), cocaine, flecainide (Tambocor), propafenone (Rhythmol), amiodarone (Cordarone), sotalol (Betapace), encaidine (no name), moricizine (Ethmozine).

  **WARNING:** Do not use lidocaine when VT is from lidocaine, phentoin (Dilantin) or mexiletine (Mexitil) as it can worsen toxicity (contact Biocare Medical Control for guidance).

- Lidocaine reaches the central circulation within 2 minutes and patients should be observed for toxicity which includes; slurred speech, altered mentation, twitching, seizures, and bradycardia.
- Amiodarone is typically given at a rate of 150 mg over 10 minutes in the stable patient; however when urgently needed to control a persistent tachydysrhythmia the dose is adjusted to 300 mg over 10 minutes in an attempt to rapidly interrupt the tachydysrhythmia.
- Amiodarone infusions may be made by adding 100 mgs of Amiodarone to a 100 mL bag of NS or buretrol yielding a 1 mg per mL concentration which is infused at 60 gtts per minute to provide 1 mg/min (always be sure to remove an equal amount of fluid from the bag before adding the drug).
- Sodium bicarbonate is used for sodium channel blocker or cocaine related VT to correct the sodium blockade of cardiac cells which leads to widened QRS, hypotension and arrhythmias; in addition it alkalinizes the blood which binds any unbound (free) drug then removing it in the urine.

**POTENTIAL PITFALLS**

- Initiating a post conversion maintenance infusion when profound hypotension, second or third degree AV block, rate less than 50 bpm, idioventricular or ventricular escape rhythm are present.
- Failure to promptly treat hemodynamically compromised patients while spending excessive time attempting to first secure a definite rhythm diagnosis.
- Administering nitroglycerin during VT can and most likely will cause cardiovascular collapse with further heart rate increase due to vasodilation; do not use NTG during VT.
Polymorphic VT (PVT) is a form of V Tach in which the QRS is constantly changing direction but the QT interval is normal. 

Torsades de pointes is a form of PVT in which the QRS complex size varies and appears to twist around the baseline and the QT interval is prolonged.

**ASSESSMENT AND TREATMENT CONSIDERATIONS**

- Torsades is seen with conditions that prolong the QT interval such as AMI, drugs that prolong the QT interval (TCA or sodium blockers), and bradycardia.
- **Stable patients** are those who do not have hemodynamic decompensation, ongoing chest pain, altered mentation, shock, or CHF.
- The **QT interval** is measured from the beginning of the QRS to the end of the T wave and varies with heart rate (shortens at faster rates; lengthens at slower).
- **QT prolongation** is said to be present when the baseline ECG exceeds 50% of the R-R interval or >0.40 secs wide (the QTc on the 12-lead).
- Torsades often occur in bursts (episodic) that are non-sustained and it is between these bursts that the underlying rhythm usually shows the QT prolongation.
- PVT requires immediate treatment because it is likely to deteriorate to pulseless arrest if left untreated. Once recognized aggressive therapy needs to be started to prevent CV collapse.
- Overdrive pacing shortens the QT interval and is very effective in preventing recurrences of the arrhythmia. Magnesium sulfate is the treatment of choice in true torsades de pointes.
- If the patient with PVT is or becomes unstable, use defibrillation not synchronized shocks. Although synchronization is preferred for an organized rhythm, it is not possible in PVT as the QRS configurations and irregular rates make it impossible to reliably synchronize the QRS.

**MEDICATION CONSIDERATIONS**

- Amiodarone is **not** to be used in PVT or in VT from tricyclic antidepressant (TCA) or fast sodium channel blocker (FSB) toxicity as it can worsen QT interval prolongation.
- Magnesium is recommended in the treatment of torsade with or without cardiac arrest, but has not been shown to be very helpful in normal QT interval related PVT.
- The administration of Nitroglycerin during periods of PVT is **contraindicated** as it can cause further heart rate increase due to preload reduction; however, NTG administration between episodes may be useful when PVT is due to myocardial ischemia in the absence of bradycardia.
- Lidocaine reaches the central circulation within 2 minutes and patients should be observed for toxicity which includes; slurred speech, altered mentation, muscle twitching, seizures, and bradycardia.
- Lidocaine should be withheld if any signs of toxicity occur during use (bolus rates greater than 50 mg/minute or exceeding 3 mg/kg in 1 hour greatly increases the chance for toxicity to occur).

**POTENTIAL PITFALLS**

- Avoid using multiple antiarrhythmic agents to avoid prorhythmic effects (ability of the drug to cause an arrhythmia) from combined use. However, combined use is not contraindicated when absolutely needed (consultation with a Biocare physician is highly advised).
- Failure to promptly treat hemodynamically compromised patients while spending excessive time attempting to first secure a definite rhythm diagnosis. When unsure treat as torsades.
- Failure to remember that polymorphic VT can suddenly and spontaneously convert. Always verify the rhythm immediately before delivering a defibrillation shock to verify the rhythm present.
Supraventricular tachycardia (SVT) is any tachycardia in which the atrium including the AV node and AV junctional portion are used to continue the tachycardia. On the ECG, SVT is seen a "narrow" QRS reflecting conduction over the His Purkinje system.

**ASSESSMENT AND TREATMENT CONSIDERATIONS**

- The majority of the patients with SVT have either AV nodal reentrant tachycardia (AVNRT) or AV reentrant tachycardia (AVRT). These arrhythmias depend on AV nodal conduction to continue and therefore can be terminated by transiently blocking AV nodal conduction by vagal maneuvers, adenosine, or synchronized cardioversion.

- SVT may have a "wide QRS" if bundle branch block, intraventricular conduction disturbance, or an accessory pathway (e.g., WPW) is present.

- SVT generally begins and ends quickly. Many people experience short periods of SVT and have no symptoms. However, SVT becomes a problem when it occurs frequently or lasts for long periods of time and produces symptoms.

- Although SVT is not uncommon during infancy it is crucial that SVT be differentiated from sinus tachycardia, which is seen much more often. Sinus tachycardia in infants is usually less than 220 bpm and in children, less than 180 bpm, whereas these rates are often higher in SVT.

- **Any wide-QRS tachycardia is assumed to be ventricular tachycardia until proven otherwise.**

- Vagal maneuvers such as breath-holding and the valsala maneuver (i.e., having the patient bear down as though having a bowel movement), all slow conduction in the AV node and can potentially interrupt the reentrant circuit and are the first-line treatment in stable patients.

**MEDICATION CONSIDERATIONS**

- Adenosine is not to be given in any wide-complex rhythm (> 0.12 sec) as sudden and extremely rapid heart rate acceleration can occur if an accessory pathway (WPW) is present with a fib.

- As a result of its short half-life, adenosine is best administered in an antecubital vein as an IV bolus followed by rapid saline infusion (10 mL pediatrics; 20 mL adults).

- Nitroglycerin administered during SVT may cause heart rate acceleration due to a reflex tachycardia secondary to vasodilation. Contact Biocare Medical Control before using nitroglycerin for suspected ischemia if unable to terminate the SVT rhythm first.

- Adenosine should be avoided in patients with SVT and acute bronchospasm secondary to asthma or COPD as it may worsen airflow obstruction (Adenosine can induce bronchospasm). These patients may benefit from intravenous magnesium sulfate to help break the SVT reentry cycle along with treating bronchospasm; Biocare Medical Control should be contacted for guidance in these patients.

**POTENTIAL PITFALLS**

- Heart denervation, drug toxicity or hypovolemia induced arrhythmias will not respond to vagal maneuvers and its use should not be attempted in these patient types.

- Failure to treat a wide complex tachydysrhythmia as VT when uncertain if VT or supraventricular rhythm with aberrant ventricular conduction. **When in doubt treat as VT.**

- Incorrectly identifying and treating sinus tachycardia as VT when it is present from: fever, shock, hypovolemia, tamponade, hypoxia, drug overdose, pneumothorax, abnormal electrolytes.

- The administration of nitroglycerin **prior to rate control** in patients with rapid rates can cause reflex tachycardia from peripheral vasodilatation with further heart rate acceleration.

- Delivering multiple synchronized shocks when a rhythm is short lived or recurs frequently. The use of multiple cardioversion shocks in short lived or repetitive tachyarrhythmias is contraindicated.
Supraventricular tachycardia (SVT) is any tachycardia in which the atrium including the AV node and AV junctional portion are used to continue the tachycardia. On the ECG, SVT is seen a "narrow" QRS reflecting conduction over the His Purkinje system.

**ASSESSMENT AND TREATMENT CONSIDERATIONS**

- **Serious signs and symptoms include:** altered mentation, hypotension, hypoperfusion, congestive heart failure, or acute myocardial ischemia is present.
- The majority of the patients with SVT have either AV nodal reentrant tachycardia (AVNRT) or AV reentrant tachycardia (AVRT). These arrhythmias depend on AV nodal conduction to continue and therefore can be terminated by transiently blocking AV nodal conduction by vagal maneuvers, adenosine, or synchronized cardioversion.
- Although SVT is not uncommon during infancy it is crucial that SVT be differentiated from sinus tachycardia, which is seen much more often. Sinus tachycardia in infants is usually less than 220 bpm and in children, less than 180 bpm, whereas these rates are often higher in SVT.
- **Any wide-QRS tachycardia is assumed to be VT until proven otherwise.**
- Vagal maneuvers such as breath-holding and the valsalva maneuver (i.e., having the patient bear down as though having a bowel movement), all slow conduction in the AV node and can potentially interrupt the reentrant circuit and are the first-line treatment in stable patients.
- In hypersympathetic drug induced SVT adenosine and shocks are often ineffective, in these patients lorazepam is the first drug of choice. Examples of sympathomimetic agents include pseudoephedrine, cocaine, amphetamines, methamphetamine, ephedrine, or ecstasy.
- Synchronized shocks should begin at 100 Joules in adults (not 50) and increase in a stepwise fashion as needed to try and interrupt the tachydysrhythmia and restore a sinus rhythm.

**MEDICATION CONSIDERATIONS**

- Adenosine is not to be given in any wide-complex rhythm (> 0.12 sec) as sudden and extremely rapid heart rate acceleration can occur if an accessory pathway (WPW) is present with a fib.
- Midazolam may be considered for the awake patient prior to cardioversion if the condition allows and vascular access is present (allow 3-5 minutes for full sedation effect to occur).
- Use of sedation for cardioversion is left to the discretion of the provider and is based upon several factors: patient’s hemodynamic status, mental status, and severity of tachycardia.
- A large dose of Adenosine (12 mg in adults 0.2 mg/kg in pediatrics) may be tried first if not contraindicated. If conversion does not occur after 2 minutes, immediately proceed to cardioversion.
- Amiodarone is typically given at a rate of 150 mg over 10 minutes in the stable patient; however when urgently needed to control a persistent tachydysrhythmia the dose is adjusted to 300 mg over 10 minutes to attempt to rapidly interrupt the tachydysrhythmia.
- Adenosine should be avoided in patients with SVT and acute bronchospasm secondary to asthma or COPD as it may worsen airflow obstruction (Adenosine can induce bronchospasm). These patients may benefit from intravenous magnesium sulfate to help break the SVT reentry cycle along with treating bronchospasm; Biocare Medical Control should be contacted for guidance in these patients.

**POTENTIAL PITFALLS**

- Administration of midazolam or Lorazepam when the SBP < 70 may exacerbate hypotension.
- Failure to treat a wide complex tachydysrhythmia as VT when uncertain if VT or supraventricular rhythm with aberrant ventricular conduction. **When in doubt treat as VT.**
- The administration of nitroglycerin prior to rate control in patients with rapid rates can cause reflex tachycardia from peripheral vasodilation with further heart rate acceleration.
- Delivering multiple synchronized shocks when a rhythm is short lived or recurs frequently. The use of multiple shocks in short lived or repetitive tachyarrhythmias are contraindicated.
Symptomatic bradycardia is defined as a heart rate less than 60 bpm associated with one or more of the following: hypotension, altered mentation, ongoing chest pain, dyspnea, or congestive heart failure. This protocol focuses on the management of bradycardia considered inadequate for the clinical situation.

**ASSESSMENT AND TREATMENT CONSIDERATIONS**

- A slow heart rate may be normal for some patients (i.e., athletes), and inadequate for others (i.e., cardiac patients). Most patients do not become symptomatic unless their rate is below 50 bpm.
- Asymptomatic patients do not require treatment; but rather should be watched for deterioration.
- Sinus bradycardia, slow junctional, ventricular escape and various degrees of AV block are the most common pre-arrest rhythms in children.
- For symptomatic bradycardia with high-degree AV block (second-degree type II or third-degree) withhold atropine and proceed immediately to transcutaneous pacing without delay.
- Bradycardia in pediatrics is often due to hypoxemia and the initial management is ventilation and oxygenation while perfusion is maintained with chest compressions as needed. If these measures do not restore an adequate heart rate, medications are then administered to treat the bradycardia.

**MEDICATION CONSIDERATIONS**

- Fluid therapy should be initiated only as secondary adjunct to pacing or drugs. Increasing the rate is the first method for increasing perfusion and blood pressure.
- Nitroglycerin administered during bradycardia of less than 50 bpm can cause further heart rate reduction to occur secondary to increased vagal tone. Contact Biocare Medical Control before using Nitroglycerin in any patient with a rate under 50 bpm.
- Initiate TCP first and then determine how well it is tolerated before administering an analgesic or benzodiazepine as mental status will be difficult to assess once sedated.
- The use Fentanyl or Midazolam during pacing is left to the discretion of the provider and is based upon the patient’s BP, mental status, toleration of TCP and severity of bradycardia.
- Sinus bradycardia is a common manifestation of hypoxia in the infant. The older child may present with specific symptoms such as syncope, chest pain, shortness of breath, or palpitations.
- **Use of Atropine in pediatrics is a 2nd line therapy after Epinephrine, but a 1st line therapy when bradycardia is from vagal tone (e.g., intubation), or when 2nd or 3rd degree block is present.**
- Use Atropine with extreme caution in ACS as it can worsen ischemia or infarction as parasympathetic blockade allows unopposed sympathetic stimulation to occur; leading to tachycardia and HTN.
- The patient with AV block or bradycardia due to digitalis toxicity may benefit from Atropine use as the heart block is due to increased parasympathetic tone at the AV node, which atropine blocks.
- An Epinephrine infusion at 2 to 10 mcg/min may be needed if profound hypotension or bradycardia fails to respond to standard measures (mix 1 mg of 1:1 Epinephrine to 250 mL of NS = 4 mcg/mL). Biocare Medical Control must be contacted before using an Epinephrine infusion.

**POTENTIAL PITFALLS**

- Administering NTG in heart rates < 50 bpm can cause vagal tone further reducing the heart rate.
- Administration of Atropine in Mobitz type II or third degree AV blocks with wide-QRS complexes can cause further bradycardia, use pacing or dopamine instead.
- Administration of Atropine in doses less than 0.1 mg could cause paradoxical bradycardia to occur.
- Administration of Atropine in a patient with a denervated heart (i.e., heart transplants). Atropine will not be effective in these patients, go at once to TCP, catecholamine infusion or both.
- Aggressively treating bradycardia when significant hypertension is present as this is often a protective reflex mechanism to decrease intracranial pressure. If bradycardia with hypertension is present contact Biocare Medical Control for treatment guidance in these patients.
CARDIAC ARREST - GENERAL MANAGEMENT

Cardiac arrest is the loss of spontaneous circulation which can be caused by many different rhythms and circumstances. Cardiac arrest primarily results from several causes, including electrical dysfunction, mechanical failure, circulatory shock and/or abnormalities in ventilation leading to significant respiratory acidosis. Optimal management of the patient requires a team approach to care.

ASSESSMENT AND TREATMENT CONSIDERATIONS

- **Cardiac arrest in patients > 20 wks gestation:** Perform chest compressions higher on the sternum, slightly above the center of the sternum. There are no changes to ACLS algorithms for care.
- **Relieve vena cava compression** for adequate blood flow during CPR by 1 of 3 methods below:
  - manually and gently displacing the gravid uterus to the left; or
  - placing a wedge-shaped cushion/wedge or multiple pillows under her right hip; or
  - slightly tilt the patient to her left side, leaning her against a rescuer’s thighs.
- Hormonal changes can cause relaxation of the gastroesophageal sphincter, increasing the risk of regurgitation. Apply continuous cricoid pressure during ventilation of an unconscious pregnant woman until the airway can be secured.
- Because there are no valves in the inferior vena cava, backward blood flow can occur in the femoral area which may be misinterpreted as arterial blood flow. Use the carotid artery instead when assessing for a pulse in adults.
- **Pediatric defibrillation and cardioversion energy levels** are based on a joules/kg formula and various energy levels can be delivered based on manufacturer and device used. This variance in energy levels tends to hamper the ability to deliver exact energy as indicated by a specific kg weight (i.e., using 2 J/kg, a 20 kg patient would receive 40 Joules, however the energy range provides for a 30 or 50 joule level only. Because of this, modifications to energy requirements must be made.
  - **Wide complex dysrhythmias:** select to the next highest energy setting.
  - **Narrow complex dysrhythmias:** select the next lowest energy setting
- CPR should not be interrupted, if at all possible, for more than 5 seconds at any time.
- **Broselow Pediatric Emergency Tape:** The Broselow Tape is to be used as a guide in determining drug dosages and equipment sizes for infants or children up to 34 kilograms. If a discrepancy occurs between the tape and protocols, the protocols are to be followed.
- A standard AED may be used in any patient > 1 year of age; however it is highly preferred to use attenuator pads made to reduce energy levels if at all possible in children < 8 years of age.

MEDICATION CONSIDERATIONS

- High dose epinephrine is no longer recommended in routine cardiac arrest; however, it should be considered when arrest is due to a beta-blocker overdose or anaphylaxis.
- Atropine is indicated in asystole as strong vagal tone may be eliminating pacemaker activity; in PEA atropine is only administered when the heart rate is less than 60 bpm.
- Myocardial ischemia during cardiopulmonary arrest often causes atropine-resistant bradyasystole from cellular accumulation of adenosine. Aminophylline is an adenosine antagonist capable of reversing refractory asystole in some cardiac arrest patients.

POTENTIAL PITFALLS

- Failure to identify ineffective gasping breathing efforts (“agonal” respirations), which can occur early in cardiac arrest and confusing them with effective respirations.
- Failure to consider and/or treat the underlying cause of cardiac arrest when therapies are available (e.g., profound hypoglycemia, drug overdose, hyperkalemia etc.).
- Treating acute pulmonary edema during arrest by use of nitrates or diuretics as this can worsen cardiovascular collapse and increase global myocardial dysfunction.
Asystole is cardiac standstill with no cardiac output and no ventricular depolarization. Primary asystole occurs when the heart's electrical system fails to generate a ventricular depolarization (only p waves are seen). Secondary asystole occurs when the heart's electrical system fails to generate any electrical depolarization (true asystole or flat line). Pulseless Electrical Activity (PEA) is a group of pulseless rhythms that includes pseudo-electromechanical dissociation (pseudo-EMD), idioventricular rhythms, ventricular escape, post-defibrillation idioventricular, bradyasystolic, and rapid SVT without a pulse. Because of the similarity in causes and management of these 2 rhythms, their treatment is combined.

**ASSESSMENT AND TREATMENT CONSIDERATIONS**

- True PEA can occur after defibrillation, hyperkalemia, hypothermia, and drug overdose. Pseudo-EMD is due to myocardial dysfunction and is seen in rapid SVT, hypovolemia, tension pneumothorax, tamponade, or massive pulmonary or amniotic fluid embolism.
- The initial treatment for narrow complex SVT without a pulse is immediate synchronized cardioversion followed by Amiodarone 300 mg if shocks fail to convert rhythm. Do not administer epinephrine until shocks and Amiodarone have been tried (can increase the heart rate).
- If the patient remains in asystole after intubation, medications, fluids and a persistent quantitative ETCO$_2$ level of < 10 mmHg without a reversible cause identified, consider contacting Biocare Medical Control for possible termination of resuscitative events.
- Common causes for asystole / PEA and their recommended therapies:
  - Hypovolemia / trauma – Aggressive fluid resuscitation.
  - Hypoxia – Aggressive, but controlled ventilation and endotracheal intubation (avoid hyperventilation).
  - Metabolic acidosis (known values or preexisting condition, not for routine cardiac arrest) - Sodium Bicarbonate 8.4%- 1 mEq/kg IV. May repeat in 10 minutes at ½ the original dose if still indicated.
  - Suspected hyperkalemia (dialysis pt.) - Calcium gluconate, + Albuterol, + Sodium Bicarbonate.
  - Hypothermia - rewarm (no rapid rewarming of arms/legs) and prevent further heat losses.
  - Poisoning/overdose - treatment is based on presenting drug or poison.
  - Cardiac tamponade - rapidly infusion of NS to increase preload and afterload.
  - Tension pneumothorax – rapid pleural decompression of affected side(s) with a large bore needle.

**MEDICATION CONSIDERATIONS**

- High dose Epinephrine is no longer recommended in routine cardiac arrest; however a dose of 3 mg may be used when arrest is due to a beta-blocker overdose or anaphylaxis (adults only).
- Atropine is used in asystole as vagal tone may be eliminating pacemaker activity; in PEA Atropine is only administered when the heart rate is less than 60 bpm.
- Myocardial ischemia during cardiopulmonary arrest can cause Atropine-resistant bradyasystole from cellular accumulation of Adenosine. Aminophylline is an Adenosine antagonist capable of reversing refractory asystole in some cardiac arrest patients.

**POTENTIAL PITFALLS**

- Failure to confirm asystole in more than one lead to ensure that fine VF is not present.
- Application of “just in case” defibrillation of asystole can produce a parasympathetic discharge, preventing any further spontaneous pacemaker activity. Do not routinely defibrillate asystolic patients.
- Administration of Sodium Bicarbonate in the absence of a cyclic / sodium blocking agent overdose or hyperkalemia can have adverse effects. Alkalosis from excessive bicarbonate can shift the oxyhemoglobin dissociation curve to the left impairing oxygen delivery to the tissues.
- Failure to connect all 4-limb leads when attempting to pace asystole or agonal rhythms. Limb leads must be attached to effectively synchronize pacing impulses with the underlying ECG rhythm.
- Failure to recognize and/or treat hypoglycemia when present, especially in pediatric patients.
- Not beginning concurrent therapy when a potentially treatable cause is present (e.g. hyperkalemia).
Potassium (K+) is one of the body’s major ions and nearly 98% is found intracellularly. The ratio of intracellular to extracellular potassium is important as small changes in the extracellular potassium level can have profound effects on the function of the cardiovascular and neuromuscular systems. This protocol specifically addresses those patients presumed to be in cardiac arrest from hyperkalemia.

**ASSESSMENT AND TREATMENT CONSIDERATIONS**

- Some causes of hyperkalemia include; use of potassium-sparing diuretics (Aldactazide, Dyazide, Maxzide, Moduretic, Spirozide), NSAIDS, ACE inhibitors (Accupril, Altace, Capoten, Enalapril, Lotensin, Mavik, Monopril, Prinivil, Univasc, Vasotec, Zestril), ACE receptor blockers (Losarten, Valsarten), cyclosporine, antibiotics such as pentamidine or trimethoprim/sulfamethoxazole, history of renal insufficiency or renal failure, large burns after 72 hours, and significant crush injuries.
- Hyperkalemia should be suspected in any patient with a known predisposition toward elevated potassium (end-stage renal failure, dialysis patient who missed dialysis).
- Treatment is directed at stabilizing the cardiac myocardium, shifting potassium from the extracellular to the intracellular environment and promoting renal excretion.
- Continuous ECG monitoring is essential if the patient is found or thought to be hyperkalemic as life-threatening arrhythmias can occur at any time.
- IV calcium is administered to stabilize the myocardium; it lowers the threshold potential, counteracting the effects of high potassium. Calcium does not have any effect on the potassium level.

**MEDICATION CONSIDERATIONS**

- Calcium gluconate is administered to treat and rapidly reduce the risk of ventricular fibrillation caused by hyperkalemia. It acts quickly and can be lifesaving, thus it is always the first-line treatment when severe hyperkalemia is suspected (e.g., wide QRS, loss of P wave, VF, VT, and PEA).
- Sodium bicarbonate increases the pH which results in a temporary potassium shift from the extracellular to the intracellular environment. The onset of action for sodium bicarbonate occurs in 5 to 10 minutes and effects may last 1 to 2 hours.
- Albuterol is an adrenergic agonist that increases plasma insulin concentration, which may help shift K⁺ into intracellular space, lowering the K⁺ level. Albuterol can be very beneficial in patients with renal failure when fluid overload is concern.

**POTENTIAL PITFALLS**

- Administration of Sodium Bicarbonate in the absence of a cyclic antidepressant overdose, cocaine toxicity, or hyperkalemia can have adverse effects. Alkalosis shifts the oxyhemoglobin dissociation curve to the left with subsequent impaired oxygen delivery to tissues.
- Failure to suspect and treat hyperkalemia based on patient presentation, history, and ECG. Severe hyperkalemia with ECG changes is a life-threatening emergency; Calcium is the initial treatment of choice to stabilize the cardiac membrane.
- Administering Calcium together with Sodium Bicarbonate can cause a calcium carbonate precipitation to occur in the IV line. Always flush the line between doses of these two agents.
- Note: patients may be symptomatic prior to cardiac arrest, even if life-threatening hyperkalemia is present. Abrupt PEA, ventricular tachycardia, ventricular fibrillation, and asystole can all occur.
Post resuscitation care refers to the period between restoration of spontaneous circulation and arrival to the Emergency Department. Proper care in this period can make a difference in the eventual outcome, especially neurological function. Post-resuscitative care also can include the period following chemical or electrical termination of an electrically dangerous arrhythmia in a previously hypoperfused patient.

**ASSESSMENT AND TREATMENT CONSIDERATIONS**

- Patients may display a wide spectrum of responses from resuscitation, ranging from being awake and alert, with adequate spontaneous respirations and hemodynamic stability, to remaining comatose with apnea and cardiovascular instability.

- Temperature elevation above normal can create an imbalance between oxygen supply and oxygen demand that can impair brain recovery and prevention or treatment of hyperthermia is important.
  - Providers should not actively rewarm (just prevent further heat loss) in hemodynamically stable patients with mild hypothermia (91°F to 95°F) after cardiac arrest. Mild hypothermia can be beneficial to neurologic outcome and is well tolerated without significant risk of complications.

- Assess and correct any low glucose concentrations and avoid hyperventilation.

- Assess circulating fluid volume and ventricular function. Even mild hypotension should be avoided as it can impair recovery of cerebral function.

- Ventricular ectopy should be treated only when it is considered malignant (R-on-T, couplets, runs of VT, multiple episodes of multi-formed etc.).

- Post resuscitation hyperventilation: After an arrest, restoration of blood flow causes an initial increase in blood flow to the brain lasting 10 to 30 minutes that is often followed by a period of low blood flow. If the patient is hyperventilated during the low flow stage, cerebral vasoconstriction can further decrease blood flow and increase brain injury. Do not hyperventilate post resuscitation.

**MEDICATION CONSIDERATIONS**

- Use of post-resuscitation antiarrhythmics is contraindicated in the following circumstances (contact Biocare Medical Control for approval first):
  - Hypoperfusion with shock (unless urgently needed to treat an unstable rhythm),
  - AV blocks (2nd and 3rd degree),
  - Symptomatic sinus or junctional bradycardia unless the patient has a functioning pacemaker,
  - Idioventricular or ventricular escape rhythms (can be lethal).

- Only lidocaine maintenance infusion doses (not initial IV or IO doses) need to be decreased by 50% in patients > 70 years of age, AMI, CHF, or shock (higher probability of decreased hepatic flow). There is no need to reduce lidocaine doses in patients with renal failure as lidocaine is metabolized through the hepatic, not the renal system.

- Post resuscitation ventricular ectopy often only requires watchful waiting. Contact Biocare Medical Control for further guidance when PVCs are present post-resuscitation.

- Benzodiazepines should be avoided in the presence of profound hypotension (SBP < 70 mmHg) unless urgently needed to obtain an airway. Contact a Biocare physician for orders before use.

- Amiodarone infusions may be made by adding 100 mgs of Amiodarone to a 100 mL bag of NS or buretrol yielding a 1 mg per mL concentration which is infused at 60 gtts per minute to provide 1 mg/min (always be sure to remove an equal amount of fluid from the bag before adding the drug).

**POTENTIAL PITFALLS**

- Failure to reassess after each half bolus and hold if fluid overload or sudden dyspnea occurs.

- Failure to consider and/or treat the underlying cause of the cardiac arrest when therapies are available (e.g., profound hypoglycemia, drug overdose, hyperkalemia etc.).

- Treating acute pulmonary edema following cardiac arrest when the patient is hypoperfused by use of nitrates or diuretics can increase global myocardial dysfunction and worsen cardiac output.

- Failure to remember that excessive ventilation volumes and airway pressures can have harmful effects, especially in the presence of reactive airway diseases.

- Failure to recognize an esophageal intubation and take corrective action.
**VENTRICULAR FIBRILLATION / PULSELESS VT**

**Ventricular fibrillation** (VF) is a pulseless arrhythmia with irregular and chaotic electrical activity and ventricular contraction in which the heart immediately loses its ability to function as a pump. Pulseless **Ventricular Tachycardia** (VT) is rapid and wide-complex rhythm without discernable pulses most commonly due to inadequate filling secondary to the rate.

**ASSESSMENT AND TREATMENT CONSIDERATIONS**

- When VT/VT is witnessed by EMS, hold CPR and defibrillate at recommended energy levels.
- When a patient in VF/VT experiences a sudden loss of pulses a massive dump of epinephrine, norepinephrine and acetylcholine often occurs which creates an unstable electrical state. This lowers the depolarization threshold and leads to an increase of calcium into the cells. Excessive acetylcholine alters the action potential, making signals less responsive and the QRS wider. All these changes are the common basis for reentry arrhythmias such as VF/VT to occur and perpetuate.
- VF/VT often occurs in areas of myocardial ischemia that have a lowered threshold. Contributing factors include: elevated catecholamines, improper sympathetic stimulation, electrolyte imbalance (especially potassium), hypoxia, acid-base disturbances, toxic responses to drugs, hyper or hypothermia, and proarrhythmic conditions such as prolonged QT syndrome.
- Electrical defibrillation remains the most successful treatment of VF. A shock is delivered to the heart to simultaneously depolarize the myocardium. The objective is to interfere with all reentrant arrhythmias and to allow cardiac pacemakers to resume control.
- Providers should practice coordination of CPR and shock delivery so that when a shock is indicated, it can be delivered as soon as possible after chest compressions are stopped and rescuers are “cleared” from contact with the patient. Studies have shown that a reduction in the interval between compression and shock delivery by as little as 15 seconds can increase the predicted shock success.
- VF waveform usually begins with relatively high amplitude and degenerates to smaller amplitude until asystole occurs in about 15 minutes, most likely due to depletion of high energy reserves.
- If VF is initially terminated by a shock but then recurs later in the arrest, deliver subsequent shocks at the previously successful energy level.
- **When applying an AED in a child** from 1 to 8 years of age use a pediatric dose-attenuator system if one is available (automatically reduces energy). However, if an AED with an attenuator system is not available for a child in cardiac arrest, a standard AED may be used.

**MEDICATION CONSIDERATIONS**

- High dose epinephrine is no longer recommended in routine cardiac arrest; however, it should be considered when arrest is due to a beta-blocker overdose or anaphylaxis.
- Atropine is only indicated in asystole or slow PEA and never for VF or VT.
- Amiodarone is a class III antidysrhythmic (potassium blocker) with some class I (sodium blocking), class II (beta blocking), and class IV (calcium blocking) effects. This multitude of physiologic effects is why amiodarone is used in managing a wide variety of dysrhythmias.
- Administering multiple antiarrhythmics together can have a proarrhythmic effect (can cause arrhythmias) and is why Amiodarone and Lidocaine are not listed together as a treatment.

**POTENTIAL PITFALLS**

- Failure to consider an electrolyte disorder such as hyperkalemia as a cause for refractory VF.
- Using a precordial thump when a defibrillator is available; precordial thumps are no longer advocated.
- Administration of Sodium Bicarbonate in the absence of a cyclic overdose, cocaine toxicity or hyperkalemia may have severe adverse effects as alkalosis shifts the oxyhemoglobin dissociation curve to the left with subsequent impaired oxygen delivery to tissues.
- Failure to recognize and/or treat hypoglycemia when present, especially in pediatric patients.
- Pronouncing patients with persistent VF or VT in the prehospital setting is risky as spontaneous conversion could occur. These patients should always be transported to an ED for further evaluation.
ABDOMINAL PAIN

The etiology of abdominal pain is diverse, but, for immediate purposes, the prehospital provider should focus on whether or not the pain is associated with a life-threatening condition and/or potentially requires surgical intervention. An effective history includes a detailed description of the pain and its onset, and an accounting of associated symptoms and events.

ASSESSMENT AND TREATMENT CONSIDERATIONS

- Pain from a serious condition typically arises suddenly and is continuous, progressively worse, and long lasting; begins during inactivity; and is not near the umbilicus. A rigid abdomen, absent bowel sounds, rebound tenderness may indicate the need for surgery.
- The principle characteristics of abdominal pain include location, quality, severity, onset, duration, aggravating and alleviating factors, and change in any of these over time.
- Assessment of abdominal pain in women of childbearing age should include the last known menstrual periods and any previous births.
- Sickle cell disease (SCD) can cause clumping (sickling) of RBC’s and occurs in 1 in 500 African-American births. Vasooocclusive crisis (VOC) occurs if circulation is obstructed by clumped RBC’s causing organ ischemia, severe pain of the joints, soft tissue, or organs (abdomen, spleen).
- A vasooocclusive crisis (VOC) occurs when the microcirculation is obstructed by sickled RBC’s leading to organ ischemia and severe pain of the joints, soft tissue or abdominal organs. It is typically of an acute onset, diffuse and poorly localized and is often recurrent.
- Black widow venom is a neurotoxin and the bite appears as a target, with a pale area surrounded by a red ring. Severe muscle pain and cramps may develop in the first two hours and are usually first felt in the abdomen, back, shoulders and thighs. Other symptoms of envenomation include weakness, sweating, anxiety, nausea, vomiting, dyspnea and increased blood pressure. If a black widow spider bite with symptoms is suspected contact Biocare Medical Control for treatment guidelines.
- Obtain a 12-lead ECG along with continuous ECG monitoring if male > age 40 or female > age 45 or patient has significant risk factors for cardiovascular disease such as HTN, diabetes, high cholesterol, obesity, smokes (atypical ACS presenting as only abdominal pain).

MEDICATION CONSIDERATIONS

- Patients with acute, undiagnosed abdominal pain in general may receive intravenous narcotic analgesia in the prehospital setting when strongly indicated.
- Nitrous oxide use is contraindicated in acute abdominal pain related to suspected bowel obstruction or vasooocclusive crisis as its use may worsen either condition.
- Rapid infusion of NS is indicated in states of severe hypovolemia or hypovolemic shock and should continue until the SBP is > 90 (adults), pulse rate is < 120 bpm, hypoperfusion is resolved (pediatrics), dyspnea occurs (pulmonary edema), or the maximal amount of fluid is reached.
- Signs of improvement after fluid administration includes: BP elevation, lowering of the HR, improvement of mental status or pulses, which may be used as a guide to fluid bolus needs.

POTENTIAL PITFALLS

- Over treating the patient who has sickle cell trait, not sickle cell disease. Clinically, patients with trait have minimal complications (mild hematuria). If unsure contact Biocare Medical Control for guidance.
- Failure to acquire a 12-lead ECG in patients > age 40 yrs (male) or > 45 yrs (female) or those who have risk factors, and/or physical findings suggestive of an ACS (ST changes, abnormal VS etc.).
- Failure to recognize dehydration or sepsis in any patient with undiagnosed abdominal pain.
- Failure to consider cocaine or other sympathomimetic drugs as a cause of an acute coronary syndrome presenting as an acute abdomen (myocardial or mesenteric ischemia).
- Failure to monitor patient status when administering fluid in large volumes or via rapid infusion as fluid overload and pulmonary edema can occur.
Acute Coronary Syndrome (ACS) is a broad term used to describe any group of clinical symptoms associated with acute myocardial ischemia or infarction. Acute myocardial ischemia is chest pain due to insufficient blood supply to the heart muscle that is often reversible. Acute myocardial infarction is cellular injury and death of cardiac muscle caused by prolonged ischemia.

**ASSESSMENT AND TREATMENT CONSIDERATIONS**

- **ST Segment Elevation MI (STEMI)** is diagnosed by more than > 1 mm of elevation in 2 or more contiguous leads or any new LBBB.
- Whenever an inferior wall MI is suspected, a right-sided ECG is to be done to assess for right ventricular infarction (RVI). Assess for ST elevation using lead V4R (> 90% positive for RVI when elevated more than 1 mm).
- In suspected RVI the use of preload reduction agents such as Lasix, nitroglycerin and MS are to be avoided as severe BP drop can occur.
- The initial therapy of RVI with hypotension and clear lungs is volume expansion to > preload.
- A 12 lead ECG should be obtained within 10 minutes of contact and immediately transmitted (if possible) anytime an infarct is suspected.
- As lethal arrhythmias can occur at anytime during ACS continuous ECG monitoring is REQUIRED until the patient is turned over to ED staff.
- Pain relief is important as a hyperadrenergic state is often seen during occlusion that can enlarge the zone of ischemia and promote dysrhythmias.
- All patients (male/female) MUST be questioned about Viagra, Levitra or Cialis use before giving nitrates.

**MEDICATION CONSIDERATIONS**

- IV access should be in place before NTG is used, however in the stable patient the inability to obtain vascular access is not a reason to hold nitroglycerin when indicated.
- The rapid administration of Morphine can induce vomiting and can be avoided by administering slowly.
- Aspirin has been shown to decrease mortality during a MI and it should be rapidly administered, if not contraindicated, upon determination that ACS may be present (unless contraindicated).

**POTENTIAL PITFALLS**

- Nitroglycerin administered during very rapid heart rates can cause further rate acceleration due to reflex tachycardia from vasodilation. Contact Biocare Medical Control for guidance in these patients.
- Attributing epigastric or ischemic chest pain to a GI source, despite the presence of dyspnea or diaphoresis, symptoms that are difficult to attribute to the GI system pathology.
ALLERGIC REACTION / ANAPHYLAXIS

Allergic reactions and anaphylaxis represent a spectrum of the same problem and true anaphylactic reactions can be life-threatening. The cardinal signs of anaphylaxis are stridor, bronchospasm, and hypotension. Symptoms associated with anaphylaxis often begin within seconds of exposure to an allergen; however they may be delayed up to 1 hour. Typical response begins in minutes and commonly involves the cardiopulmonary systems with the faster the onset, the more severe the reaction. Cardiovascular collapse can occur without respiratory symptoms, but is uncommon.

ASSESSMENT AND TREATMENT CONSIDERATIONS

- The majority of patients with anaphylaxis have some combination of urticaria (hives), erythema (redness of skin), pruritus (itching), and possibly conjunctival injection (blood shot eyes).
- In anaphylaxis the respiratory tract is often involved with nasal congestion, sneezing, coughing, hoarseness, and/or tightness in the throat prior to obstruction. GI symptoms including abdominal pain, nausea/vomiting, and diarrhea (less common, except in the case of food allergy).
- When bronchospasm or upper airway edema is present intubation may be difficult due to laryngeal edema. In the absence of cardiac or respiratory arrest, it is appropriate to consider withholding intubation and assist with a BVM to allow epinephrine to first take effect.
- Scombroid poisoning (from spoiled tuna, mackerel, dolphin or mahi-mahi) often develops within 30 minutes and commonly presents with urticaria, nausea/vomiting, diarrhea, and headache. It is treated with benadryl, not epinephrine, unless dyspnea, hypotension, or airway swelling is present.
- Severe hypotension and/or cardiac arrest from anaphylaxis are often associated with a massive fluid shift and large volume replacement is often needed. Insert 2 large-bore IVs (if possible) and administer the volume of NS as quickly as possible (use pressure bags or BP cuffs).
- Epinephrine can still be used in the presence of cardiac disease for anaphylaxis with hypotension and bronchospasm. Do not delay use when needed as anaphylactic deaths correlate with a delay in administration of epinephrine.

MEDICATION CONSIDERATIONS

- Epinephrine is not indicated for allergic reactions unless hypotension and/or airway involvement such as bronchospasm, laryngeal edema or stridor is present.
- IM epinephrine results in higher and more rapid plasma concentrations than the subcutaneous route.
- Use epinephrine 1:1000 for all IM and nebulized doses; 1:10,000 for IV/IO (never give 1:1000 IV/IO push without first diluting it). Epinephrine via a nebulizer can be used to reduce laryngeal swelling but does not replace intramuscular administration of epinephrine.
- The primary medication for acute anaphylaxis is epinephrine. All other drugs including Benadryl, Albuterol, Glucagon and Dopamine are adjuncts to care.
- Atrovent is not commonly used in anaphylaxis.
- In life-threatening reactions with shock, IV/IO epinephrine 1:10,000 may be used; however, this route can cause ventricular arrhythmias and ischemia. For this reason IM epinephrine should be tried first unless cardiovascular collapse and imminent arrest is present.
- Dopamine may be increased by 5 mcg every 5 minutes up to 20 mcg/kg/min as needed.

POTENTIAL PITFALLS

- Treating a suspected anaphylactic reaction with epinephrine when a normal or above normal BP is present. If hypertension is present, search for other reasons for signs/symptoms.
- Failure to appreciate serious symptoms, such as syncope or throat tightness, in an allergic reaction as these could rapidly progress to hemodynamic collapse and cardiac arrest.
- Failure to reassess lung sounds after each fluid bolus to prevent overload and pulmonary edema from occurring. Do not administer large fluid volumes without reassessing the patient after each bolus.
- Never administer epinephrine 1:1000 via the intravenous route as sudden tachydysrhythmias and/or intracranial hemorrhaging can occur. If intravenous epinephrine is truly needed, use a 1:10,000 concentration and administer it over at least 2 minutes.
Altered mentation is a symptom, not a diagnosis. Common etiologies include: hypoglycemia or hyperglycemia, alcohol or drug intoxication, metabolic abnormalities, seizures or postictal states, toxic exposures, hypoxia, sepsis, stroke, and head trauma.

**ASSESSMENT AND TREATMENT CONSIDERATIONS**

- Is there a history that suggests a cause (i.e. cardiac, diabetes, CVA, HTN, seizures, or alcohol/drugs)? Are there needle track marks or evidence of skin popping common with heroin users?
- Opiate toxicity should be suspected when CNS depression, respiratory depression, and pinpoint pupils are present. Drowsiness, bloodshot eyes, and euphoria are also frequently seen.

**MEDICATION CONSIDERATIONS**

**Drugs which typically will respond to Naloxone include, but are not limited to:**

- Dextromethorphan (Robitussin, Triaminic, Nyquil)
- Pentazocine (Talwin)
- Tramadol (Ultram)
- Heroin (Smack, horse, black tar etc.)
- Hydromorphone (Dilaudid)
- Meperidine (Demerol)
- Fentanyl (Sublimaze)
- Codeine (methylmorphine)
- Nalbuphine (Nubain)
- Buprenorphine (Subutex)

These drugs may require larger doses of naloxone to reverse their effects.

- Naloxone (Narcan) is not effective against respiratory depression due to non-opioid drugs such as: levopropoxyphene, promethazine, paralytics, benzodiazepines, ambien, sonata, PCP, LSD, amphetamines/methamphetamines, cocaine, GHB, cyclic antidepressants, barbiturates, ketamine, marijuana, antihistamines, quaaludes, MAO inhibitors, Selective serotonin reuptake inhibitors. *Note: list is not all inclusive – always verify drug and reversal agent when uncertain.*

- Onset of action of IV Naloxone is within 2 minutes, intranasal 1 to 4 minutes, and slightly less for SubQ or IM routes. The duration of action is approximately 20 to 60 minutes. Since the duration of action may be shorter than some opiates repeat doses may be needed.

- It is often preferable to administer Naloxone to a known drug abuser only until an improvement in depth and quality of respirations is seen and not to a fully awake state (may become violent).

- Naloxone should be used with extreme caution in patients dependent on narcotics (e.g., drug abusers, cancer patients) may cause withdrawal symptoms including N/V, sweating, tachycardia, hypertension, seizures, arrhythmias, pulmonary edema, and rarely cardiac arrest.

- If naloxone must be given to an opiate dependent patient then **large IV doses should be avoided (0.4 mg and above).** Mix 0.4 mg of Naloxone with 9 mL of NS to make a 1 mL = 0.04 mg mixture and administer at 1 mL/min until patient responds (typically after 2 to 4 mLs). This gradual administration prevents acute narcotic withdrawal from occurring.

**POTENTIAL PITFALLS**

- Failure to consider physical restraints prior to narcotic reversal for patient and provider safety.
- Immediately intubating a suspected narcotic overdose with respiratory depression until Naloxone has been administered and allowed to work. Support ventilations with a BVM while waiting for Naloxone to work to prevent unneeded intubation (except in cardiac arrest).
- Failure to consider head trauma or hypoglycemia as a cause for altered mental status.
- Failure to protect the eyes of the unresponsive patient from desiccating (drying out). When the patient is unresponsive and their eyes are allowed to remain partially open, drying of the globe can occur. Consider taping the eyelids closed if they fail to remain closed on their own.
- Narcan should be used with caution when respiratory depression is evident: Only consider fully awakening a patient when you can with certainty safely manage them in the back of the ambulance.
BEHAVIORAL EMERGENCIES

A behavioral emergency is the sudden occurrence of behavior that presents with psychiatric symptoms such as delusions, hallucinations, or depression. In all cases, substance-induced disorders (intoxication, withdrawals), organic causes (CVA etc.), endocrine (hypoglycemia and hyperglycemia), and hypoxia should be ruled out before assuming behavior is psychiatric in nature.

This protocol is to be used only when a patient presents as a threat or danger to themselves or EMS staff. Do not restrain or sedate any patient when it is not indicated.

ASSESSMENT AND TREATMENT CONSIDERATIONS

- Excited or agitated delirium is a syndrome characterized by bizarre and violent behavior, often accompanied by hyperactivity, combativeness, increased strength, paranoia, shouting, hallucinations, tachycardia, HTN, inappropriate disrobing, diaphoresis, and hyperthermia.
- Life-threatening conditions that can often present as agitation or delirium includes; intracerebral hemorrhage, meningitis, hypertensive crisis, heat stroke, hypoglycemia, and sympathomimetic drug toxicity (especially cocaine, atropine, CA, amphetamines, and methamphetamine).
- Chemical restraint (CR) is a last resort measure for safely calming extremely violent or agitated patients when the potential for harm to self or others exist.
- Patients must be continuously monitored and reevaluated every 5 minutes during any physical or chemical restraint with status changes well documented in the patient care record (no exceptions).
- If unable to obtain vascular access due to patient agitation, venous access should be obtained as soon as possible after chemical restraint has been safely accomplished and agitation is diminished.
- An acute dystonic reaction often presents with one or more of the following; protruding or the tongue, twisted neck, facial spasm, deviated eye gaze, abdominal rigidity, spasm of the body. Dystonic reactions can occur after phenothiazine use (i.e., haldol), requiring an anticholinergic drug (diphenhydramine) to help restore dopaminergic-cholinergic balance in the basal ganglia of the brain.

MEDICATION CONSIDERATIONS

- Intramuscular Haloperidol reaches its peak within 20 minutes; Intravenous injections have a rapid onset of action within ten minutes with a duration of 3 to 6 hours.
- Intramuscular Lorazepam reaches its maximum within 60 minutes; Intravenous injections reach full effect within several minutes of administration.
- Continuous ECG, pulse oximetry, and BP monitoring every 5 minutes are mandatory after administration of lorazepam and haloperidol (once safe to do so).
- Use extreme caution when administering haloperidol or lorazepam to hypotensive patients as a worsening of their clinical condition can occur (agitation may be from the hypoperfusion).

POTENTIAL PITFALLS

- Placing sedated patients in a prone position causing positional asphyxia, respiratory failure or arrest. Refer to physical restraint protocol for approved patient placement techniques.
- Medically “clearing” an acutely agitated patient in the prehospital setting in order to obtain a refusal or to allow police to transport is extremely risky. Prehospital evaluations cannot completely rule out all causes for agitation and these patients should be transported by ambulance unless scene stability or patient presentation makes this too dangerous to attempt.
- Suicidal patients (patients who have attempted and/or express the desire to commit suicide) are not permitted to sign a refusal for care or transport. Law enforcement and consultation with a Biocare Medical Control physician should help guide patient disposition.
- Failure to remember that a psychiatric disorder is only a diagnosis-by-exclusion. Many medical illnesses can cause, complicate, or mimic a psychotic process and must be excluded first.
- Failure to take a history; many prescribed medicines occasionally may cause psychotic reactions.
CONGESTIVE HEART FAILURE (CARDIOGENIC PULMONARY EDEMA)

Congestive heart failure (CHF) is a state in which the heart is unable to pump blood at a rate sufficient to meet the metabolic needs of the body. Pulmonary edema develops when an imbalance causes increased lung fluids secondary to leakage from pulmonary capillaries into the interstitium and alveoli of the lung.

ASSESSMENT AND TREATMENT CONSIDERATIONS

- All patients (male and female) MUST be questioned about Viagra, Levitra or Cialis use before administering nitroglycerin. If taken, hold nitroglycerin and contact Biocare for guidance.
- **Dyspnea on exertion (DOE)** is shortness of breath occurring during activity (e.g., climbing stairs).
- **Orthopnea** develops while lying and is relieved by elevation of the head with pillows (dyspnea often occurs within 1-2 minutes of lying flat). Orthopnea is usually relieved upon sitting up.
- **Paroxysmal nocturnal dyspnea (PND)** occurs at night after lying down for several hours with the patient suddenly awakening with anxiety, dyspnea, and a feeling of suffocation. In contrast to orthopnea is often relieved just by sitting up but PND may require 30 minutes or more for relief.
- Pulmonary edema is differentiated into cardiogenic and non-cardiogenic.
  - **Cardiogenic pulmonary edema** is due to acute ventricular dysfunction (ACS, arrhythmias etc.) and is treated using interventions that reduce preload, treat ischemia, and provide rate control.
  - **Non-cardiogenic pulmonary edema** is due to increased capillary permeability caused by infection, trauma, toxic inhalation, overdose, anaphylaxis, burns, aspiration, drowning, altitude, embolism, eclampsia, and increased ICP. The main supportive treatment is positive pressure ventilation and use of positive end-expiratory pressure.
- **Continuous positive airway pressure (CPAP)** is indicated in pulmonary edema that does not respond to SL Captopril. CPAP helps keep the alveoli open, redistributes plasma into the interstitial tissue and reduces preload. The Boussignac CPAP System is advocated for use as it eliminates re-breathing, reduces the risk of barotraumas and has an increased patient tolerance with its use.

MEDICATION CONSIDERATIONS

- **Captopril**, an angiotensin converting enzyme inhibitor interferes with the conversion of Angiotensin I to Angiotensin II and prevents vasoconstriction which leads to hypertension. The angiotensin converting enzyme is also responsible for metabolizing bradykinin, which is a potent vasodilator. These ACE inhibitors therefore also increase circulating levels of bradykinin.
  - **Captopril** is not to be used in patients with:
    - actual or suspected right ventricular infarction
    - a SBP of less than 90 mmHg
    - a fever with brown, green or yellow sputum (suspect pneumonia)
- **Nitroglycerin** is not to be used in patients with:
  - actual or suspected right ventricular infarction
  - a SBP of less than 90 mmHg
  - a heart rate of less than 50 bpm (vagal tone may worsen the rate)
  - a fever with brown, green or yellow sputum (suspect pneumonia)
  - recent ingestion of Viagra or Levitra (within 24 hours) or Cialis (within 48 hours)
- Morphine use in CHF had an anxiety reducing ability at the cost of respiratory depression; this along with a histamine release often increased catecholamine release worsening the problem. Lorazepam reduces anxiety, has no histamine release, and has less chance of respiratory depression.
- Wheezing associated with CHF is primarily due to bronchoconstriction secondary to bronchial wall irritation and edema from narrowing of the fluid filled small airways. In addition, receptors that control airway responsiveness also produce a vagally mediated response which leads to bronchoconstriction. Pulmonologist agree that albuterol may help with the inflammation related bronchoconstriction while Atrovent reverses any vagally mediated response. Because obstruction and dyspnea often respond to their use; a trial dose of each is justified when bronchospasm is present.

POTENTIAL PITFALLS

- Administration of Captopril or nitroglycerin to hypotensive patients or those in cardiogenic shock can worsen myocardial dysfunction leading to PEA and cardiac arrest.
- Assessing “*cardiac asthma*” (pulmonary edema) as bronchial asthma and treating with epinephrine.
Hyperglycemia is a syndrome that includes elevation in glucose often due to relative lack of insulin leading to dehydration and coma. Diabetic ketoacidosis (DKA) is a life-threatening complication resulting in hyperglycemia and ketone formation. Hypoglycemia occurs when there is too much insulin in the blood, resulting in low levels of glucose in the blood. Both emergencies lead to inadequate glucose levels to the brain, and if untreated will result in brain damage or death.

ASSESSMENT AND TREATMENT CONSIDERATIONS

- Patients with no known cause or any previous episodes of hypoglycemia should assertively be encouraged to go to a hospital for further evaluation.
- Signs/symptoms of pediatric dehydration include sunken fontanel, poor turgor, hypoperfusion, delayed capillary refill, and tachycardia.
- For hypoglycemia the type of treatment depends on the patient’s mental status. If the patient is awake/alert, 15 grams of a simple carbohydrate (i.e., 4 oz of fruit juice, 3 tsp of sugar or one tube of oral glucose) by mouth should be sufficient. Severe hypoglycemia with any level of altered LOC should be treated with IV glucose if possible.
- It is important to wait at least 15 minutes to obtain an accurate blood glucose level because over treatment is a frequent cause of hyperglycemia.
- **Patients with insulin pumps:** If the patient is hypoglycemic and an insulin pump is in use, the device should be disabled or removed to prevent a further drop in glucose levels. To turn off or remove: If no obvious OFF/ON button is seen use or if unsure how to turn off, remove by either using the tubing quick-release feature or removing the sub-Q needle or catheter from the skin.
- Patients awake after receiving dextrose or Glucagon may refuse ambulance transport if they so insist only when their glucose level is > 80 mg/dl, they are not hypothermic, are A&O x 4, have not over medicated on an oral anti-hyperglycemic agent and their hypoglycemia is not a first time event.
- Intraosseous (IO) access should only be used when vascular access cannot be obtained, IM glucagon fails to work and profound hypoglycemia with < LOC, seizures or shock is present.
- Transport is required for any patient who presents with or without hypoglycemia who has accidentally or purposefully overdosed on ultralente, Lente, lantus or any of the oral hypoglycemic medications.

MEDICATION CONSIDERATIONS

- **To make D25%:** Add 12.5 grams (25 mL) of D50% to 50 mL of NS or remove 25 mL of dextrose from a prefilled syringe and add 25 mL of NS; either method yields a 250 mg/mL (D25%) solution.
- **To make D10%:** Add 5 grams (10 mL) of D50% to 50 mL of NS or remove 40 mL of dextrose from a prefilled syringe and add 10 mL of NS; either method yields a 100 mg/mL (D10%) solution.
- Fluid resuscitation is critical in treating DKA as it helps replace fluid and electrolyte losses and dilutes glucose and circulating counterregulatory hormones.
- Use caution during fluid resuscitation in pediatrics as large volumes can cause cerebral edema.

POTENTIAL PITFALLS

- Assuming a patient with altered mentation is simply “intoxicated” without assessing their blood glucose level; ethanol inhibits glucose production by the liver and thus can lead to hypoglycemia.
- Failure to protect the eyes of the unresponsive patient from drying out. When the patient is unresponsive and their eyes remain partially open drying of the globe can occur causing blindness; tape the eyelids closed if they fail to remain closed on their own.
- Failure to obtain a body temperature in any patient with an extended period of hypoglycemia as hypothermia is common secondary to poor heat production from lack of circulating glucose.
- Administering oral glucose to the patient with altered mentation who cannot follow simple commands and who is at risk for aspiration. In these patients, glucose should be given intravenously or IM Glucagon used when unable to obtain vascular access.
Decompression illness (DCI) is an illness that results from a reduction in the ambient pressure surrounding a body and encompasses two diseases, decompression sickness (DCS) and arterial gas embolism (AGE). DCS (known as the bends) is thought to result from bubbles growing in tissue and causing local damage, while AGE results from bubbles entering the pulmonary circulation, traveling through the arteries and blocking blood flow at the small vessel level.

**ASSESSMENT AND TREATMENT CONSIDERATIONS**

- Any diver who is unconscious upon surfacing or who loses consciousness within 10 minutes of surfacing has an air embolism until proven otherwise.
- Any dive no matter how safe it seems can result in DCI and evaluation for DCI is made based on diver signs and symptoms, not just dive type or depth.
- DCI symptoms and signs are usually seen within 20 minutes, but can occur up to 36 hours after surfacing; in severe cases, symptoms can appear before surfacing or immediately afterwards.
- Hyperbaric oxygen (HBO) reduces the size of bubbles that obstruct circulation and washes out inert gas from the bubbles, this in turn increases blood flow and oxygen to tissues.
- Patients with air embolism are not to be placed in a head-down position as previously advocated. It was previously assumed that air bubbles could be trapped in the right atrium, preventing them from entering the circulation. Evidence now shows that this position is uncomfortable, can lead to cerebral edema, and bubbles if are present in the circulation, they rarely are trapped in the atrium.
- Obtain a dive history for 72 hours preceding the injury which should describe all dives, depths/times, ascent rates, intervals between dives, breathing gases, problems or symptoms at any time before, during or after the dive. If a dive computer was used should be transported with the patient.
- Intraosseous (IO) access should only be used when vascular access cannot be obtained and profound hypotension, shock, or a life-threatening event requiring IV medications is present.

**MEDICATION CONSIDERATIONS**

- 100% oxygen should be administered as soon as possible to help eliminate the excess nitrogen accumulation and potentially reduce nitrogen bubble size; increasing oxygenated blood flow to the body's tissues and reduce blood thickening as a result of excess nitrogen.
- The main therapy used in the treatment of diving injuries is 100% oxygen to wash nitrogen out of the lungs and increase the diffusion gradient to promote nitrogen offloading.
- Although aspirin is used for its antiplatelet activity and DCI causes blood clumping its use in DCI patients is discouraged as the patient may actively bleed from barotrauma or hemorrhage into the spinal cord or brain; do not administer aspirin in these patients without a physician order.
- Patient with suspected inner ear decompression illness presenting with acute vertigo, dizziness, and/or acute visual disturbance after diving may benefit from administration of a benzodiazepine to help stabilize the inner ear; contact Biocare Medical Control for guidance in these patients.

**POTENTIAL PITFALLS**

- Rescue/ removal of a patient in a water related accident can be extremely high risk. This should always be performed by trained and equipped providers.
- Failure to consider an organic etiology (i.e., hypoglycemia, intoxication etc.) or closed head trauma in any patient with an acute altered mental status.
- Failing to reassess lung sounds after each fluid bolus to prevent fluid overload.
Drowning is defined as death from asphyxia within 24 hours of submersion in water. Near drowning is no longer used and has been mostly replaced by the term submersion injury which refers to survival beyond 24 hours after a submersion episode. The principal physiologic consequences of submersion injuries are prolonged hypoxemia and acidosis.

**ASSESSMENT AND TREATMENT CONSIDERATIONS**

- Intraosseous (IO) access should only be used when vascular access cannot be obtained and the patient requires fluid resuscitation or medications to treat or stabilize the clinical condition.
- **Rescue mode** should be considered once patient is located only in the following conditions:
  - Still water temp < 70 degrees: < 30 minutes of submersion time from point last seen.
  - Still water temp > 70 degrees: < 15 minutes of submersion time from point last seen.
  - Swift water rescue: active rescue should continue as normal unless the actual submersion is reliably witnessed and total submersion time exceeds above parameters.
- **Recovery mode** begins after the appropriate rescue mode time has expired and primarily focuses on body recovery with no plans for resuscitation.
- Management of hypoxemia is crucial in immersion injuries. Early use of CPAP, intubation, or positive pressure ventilation (PPV) with PEEP is warranted in any patient who is hypoxic / dyspneic on 100% oxygen. Refer to CPAP guidelines if indicated.
- Patients who aspirate and have pulmonary edema due to lung injury are typically **not** in cardiogenic related failure. These patients are often best treated using PPV, not nitrates or diuretics.
- The first and most important treatment after submersion / drowning even is ventilation. Prompt initiation of rescue breathing increases the victim’s chance of survival and should be started as soon the patient’s airway can be opened and the rescuer’s safety ensured.
- Attempts to remove water from the breathing passages by any means other than suction (e.g., abdominal thrusts) are unnecessary and potentially dangerous and should not be used.
- No patient with hypothermia should be considered dead until they have been adequately rewarmed.
- Consider child abuse in young children who are the victims of submersion injury in bath tubs, buckets, or other shallow water receptacles.

**MEDICATION CONSIDERATIONS**

- Cold-induced irritation of the trachea and bronchioles by water or particulate matter can produce cough and bronchospasm. Manage these aggressively as they can worsen any existing hypoxemia.
- Ipratropium bromide (Atrovent) is an anticholinergic agent that is primarily indicated for the treatment of asthma and chronic obstructive pulmonary disease. Its value in cold-induced bronchospasm is questionable, but it may be tried when Albuterol initially fails to reduce bronchospasm.

**POTENTIAL PITFALLS**

- Failure to protect the eyes of the unresponsive patient from drying out. When the patient is unresponsive and their eyes are allowed to remain partially open, drying of the globe can occur with subsequent blindness. Tape the eyelids closed if they fail to remain closed on their own.
- Failure to obtain a body temperature in all patients with extended period of water immersion as hypothermia is not uncommon in these patients.
- Treating non-cardiogenic pulmonary edema with drugs, use positive pressure instead.
- Failure to remember that spinal trauma may be present in any patient associated with diving or involving recreational equipment. If the patient is unable to give a clear history of the events, has evidence of head or facial injury, or is found unresponsive in a pool or other shallow body of water, protect the cervical spine until injury is excluded.
HEAT RELATED EMERGENCIES

Heat illness is a spectrum of conditions that range from heat cramps to life-threatening heat stroke. Heat exhaustion is a syndrome of dizziness, nausea, vomiting, and weakness, associated with a normal to moderate temperature elevation (up to 102 F). There is no sustained change in mentation, and the skin is usually wet from profuse diaphoresis. Heat stroke presents with disorientation, seizures, coma, CNS dysfunction, and lack of sweating (not always) with a temperature > 105 F.

ASSESSMENT AND TREATMENT CONSIDERATIONS

- Heat cramps are painful muscle contractions often occurring after exercise and most commonly in the calf or hamstring. Pain from cramps may be relieved by massaging or stretching the muscle and fluid replenishment with balanced salt solution (Gatorade or IV NS).

- Heatstroke is divided into exertional and classic and is usually clinically indistinguishable.
  - **Exertional heatstroke (EHS)** typically occurs in younger patients who exercise vigorously in the heat until the body's normal thermoregulatory mechanisms are overwhelmed.
  - **Classic heatstroke (CHS)** is seen in people who are unable to control their environment and water intake (e.g., infants, elderly, chronically ill), have reduced cardiac reserve, or impaired sweating.

- The primary clinical feature of heat stroke is **CNS dysfunction** which includes bizarre behavior, hallucinations, altered mentation, disorientation, and coma.

- Sweating may not be seen in a heat stroke; especially if volume depleted or on beta blockers.

- Temperature assessment should be accomplished any of thru the following methods (descending order of preference based on presentation):
  - **Rectal** – preferred method of measurement obtained by inserting a thermometer into the rectum.
  - **Oral** - placed under the tongue; considered inaccurate if fluids are ingested within 15 min of use.
  - **Axillary** – placement of a probe under the arm and groin fold; this temperature measurement is usually one degree less than a rectal measurement.
  - **Tympanic** – temperature measurement of the tympanic membrane. Often inaccurate from incorrect probe placement and presence of ear wax in the auditory canal.
  - **Temporal** – assesses temperature by scanning over the temporal artery. Often not accurate if patient is very diaphoretic or incorrectly used.

- Evaporative cooling is the preferred method for cooling because it is safe and effective. It is accomplished by undressing the patient, spraying with water, and then cooling by fans to maximize heat loss (ice packs may also be placed over the neck, axillae and groin if needed).

- Hypoglycemia is a common finding in EHS and may be from liver failure; therefore, all patients with heatstroke should be assessed for hypoglycemia.

- Intraosseous (IO) access should only be used when vascular access cannot be obtained and profound hypotension, shock or a life-threatening event requiring IV medications is present.

MEDICATION CONSIDERATIONS

- Intravenous fluid therapy should be initiated when relative or absolute hypovolemia, hypotension, or severe heat cramps associated with painful, involuntary, muscle spasm exists.

- Aggressive cooling can induce a pronounced shivering response in the patients which can increase the core temperature. Administration of a benzodiazepine may be needed if excessive shivering occurs (contact Biocare Medical Control).

POTENTIAL PITFALLS

- Dehydration and volume depletion may not be seen during classic heat stroke and vigorous fluid administration could produce pulmonary edema; especially in the very young or elderly.

- Administering large amounts of fluid rapidly or by mouth to any patient who has an altered mentation.

- Continuing to cool a patient when the temperature is less than 100 degrees can cause too rapid of a temperature drop and initiate shivering (which will increase temperature).

- Failure to reassess lung sounds after each fluid bolus to prevent overload and pulmonary edema.
HYPOTENSION (MEDICALLY RELATED)

Hypotension is when the systolic blood pressure (SBP) is < 90 mmHg (or below the lower limit in pediatrics) due to non-traumatic causes. Hypoperfusion is the inability of the circulatory system to supply enough oxygen for tissue requirements and is an indicator of perfusion in children when a BP is unobtainable. Shock is said to exist when poor blood flow affects oxygenation & nutrient delivery at the cellular level.

ASSESSMENT AND TREATMENT CONSIDERATIONS

- When treating hypotension the goal is to improve perfusion as evidenced by improvement in skin color/condition, pulse rate & location (radial, brachial etc.), capillary refill time, and BP.

- **All patients who receive intravenous fluids MUST;**
  - be reassessed after each 1/2 bolus of fluid,
  - have further boluses limited if overload occurs, sudden dyspnea is reported or if already present, worsens,
  - have fluid amounts reduced in the presence of renal failure or cardiac disease (CHF or cardiomyopathy).

- **Cardiogenic shock:** occurs from a decreased pumping ability of the heart due to AMI, drug toxicity, dysrhythmias etc. It is treated with fluids, toxin antidotes and vasopressor support.

- **Hypovolemic shock:** occurs from a drop in circulating blood volume due to bleeding or dehydration. It is treated with fluids and vasopressors only after adequate volume is restored.

- **Distributive shock:** occurs when the vascular bed dilates and the available blood volume must fill a greater space. The form of shock is treated by correcting the underlying cause (e.g., epinephrine for anaphylaxis) along with fluids and vasopressor support added as needed.

- **Obstructive shock:** occurs when an obstruction such as tension pneumothorax or pericardial tamponade impedes blood flow through the heart; treatment is geared towards correcting the underlying cause (e.g., pleural decompression) followed by rapid IV fluids as needed.

- Patients with decompensated shock often present with altered mentation, capillary refill > 2 secs, mottling, pallor, cyanosis and/or hypotension based on their age.

- Dehydration may be seen as dry cracked lips or oral mucosa, inability to make tears or urine (no urine in diaper for 4-5 hrs in an infant and no urination for 6-8 hours in a child or adult), sunken fontanel (less than age 1), tachycardia, tachypnea, and/or altered mentation.

- Intravenous fluids in pediatric resuscitation are based on weight and clinical response and fluid replacement must be carefully regulated. Fluids should only be administered by syringe bolus or infusion using no more than a 500 mL bag of NS (250 mL recommended) to prevent overload.

- The trendelenberg position is no longer recommended as it increases the risk for aspiration, does not improve cardiac output, and often worsens gas exchange by shifting abdominal contents up onto the diaphragm, restricting respirations and tidal volume.

- A modified trendelenburg position should instead be used by keeping the patient relatively supine and only elevating the legs 8 to 12 inches. When dyspnea, morbid obesity, CHF, or COPD is present the upper torso may also be slightly elevated 6 to 8 inches to prevent worsening of dyspnea.

MEDICATION CONSIDERATIONS

- Vasopressors are not to be used in any patient who is significantly volume depleted or in a rapid dysrhythmia. Hypovolemia and any rapid rate must be corrected prior to its use.

- Additional therapy may also be required (e.g., Sodium Bicarbonate for TCA toxicity) concurrently with fluid resuscitation. If a toxin or drug is ingested refer to the appropriate protocol for additional therapy.

- Dopamine when needed is typically started at 5 to 10 mcg/kg/min IV and increased by 5 mcg every 5 minutes up to 20 mcg/kg/min as needed to response or adequate BP.

POTENTIAL PITFALLS

- Patients on beta-blockers often do not exhibit tachycardia or diaphoresis during shock.

- Treating tachydysrhythmias (i.e., SVT, VT) with fluids or Dopamine before attempting to correct the rate by other therapies such as drugs or cardioversion (Dopamine will further accelerate the rate and increase myocardial oxygen consumption, worsening the problem).

The following equation may be used for estimating the upper limit for a child’s normal heart rate:

\[
HR = 150 - (6 \times age \text{ in yrs})
\]

or in other words multiply the child’s age by 6, then subtract the result from 150 (in a 7-year-old the limit is 115).

\[
150 - (6 \times 7) = 115
\]

or

\[
150 - 36 = 115
\]
Hypothermia is a body core temperature of less than 95 degrees. Temperatures from 93-95 degrees constitute “mild” hypothermia, 86-93 degrees “moderate” and below 86 degrees “severe” hypothermia is present and modifications in the treatment of cardiac arrest should be made. Death in hypothermia is only determined after failure to revive with re-warming unless evidence shows the patient has obvious lethal injuries or the body is so frozen that nose/mouth are blocked by ice and chest compression is impossible.

**ASSESSMENT AND TREATMENT CONSIDERATIONS**

- The classic ECG finding of hypothermia is the **Osborne or "J" wave**, an extra upright deflection between the end of the QRS complex and the beginning of ST segment.
- Other ECG changes include prolongation of the PR, QRS, and QT intervals; T wave inversion; sinus bradycardia, junctional rhythm or atrial fibrillation with a slow ventricular response.
- When specifically and urgently indicated intubation should not be withheld, but rather used with caution as excessive manipulation may possibly induce arrhythmias.
- Improvement of blood circulation decreases the risk of re-warming shock and VF. Rapidly expanding the blood volume with warmed fluids increases B/P, blood flow through coronary arteries and oxygen delivery to the myocardium. In addition, during rewarming patients hypothermic for > 45 minutes are likely to require volume administration because the vascular space expands with vasodilation.
- A defibrillation attempt is appropriate when VF/VT is present; if the patient fails to respond to initial defibrillation attempts or drug therapy defer additional shocks and boluses of medication until the core temperature rises above 86°F.
- If drowning preceded hypothermia, successful resuscitation is unlikely. Hypothermia is frequently preceded by other clinical disorders (e.g., OD, hypoglycemia, and trauma) and you should always assess for and treat these if indicated (especially hypoglycemia).
- Intraosseous (IO) access should only be used when vascular access cannot be obtained and profound hypotension, shock or a life-threatening event requiring IV medications is present.
- Bradycardia occurs in severe hypothermia and often still maintains sufficient oxygen delivery when a pulse is present; transcutaneous pacing rarely works and is not indicated.

**MEDICATION CONSIDERATIONS**

- During rewarming, patients who have been hypothermic for over 45 minutes are likely to require volume administration because the vascular space expands with vasodilation.
- Fluids should be warmed to as close to 109 degrees F. prior to infusion. This may be accomplished by warming on unit heater vent, wrapping the bag with a chemical heat pack, or using an in-line battery operated IV fluid heater (optional). Do not heat fluids in a microwave.
- During hypothermia drug metabolism is reduced and medications can accumulate to toxic levels when given repeatedly; for this reason IV drugs are withheld when the temperature is below 86°F.

**POTENTIAL PITFALLS**

- Failing to keep a hypothermic patient supine to avoid aggravating hypotension through orthostatic mechanisms.
- Failure to administer warmed saline as most hypothermic patients are volume depleted and have “sludging” of their blood due to hemoconcentration.
- Rewarming the extremities of a severely hypothermic patient may induce “rewarming shock” by transference of cold, acidic blood to the central core.
- Administering Dopamine to a patient who is significantly volume depleted. Hypovolemia must first be corrected prior to Dopamine use to maximize the potential for improved perfusion.
- Failure to reassess lung sounds after each fluid bolus to prevent overload and pulmonary edema.
NAUSEA AND VOMITING

Nausea is often referred to as a “queasy sensation” or feeling “sick to the stomach.” Nausea may occur with or without vomiting, and vomiting can occur without nausea. Vomiting (or emesis) is the forceful expulsion of the contents of one's stomach through the mouth. Nausea and vomiting are not diseases, but rather symptoms of many different conditions, some of which may be serious and life-threatening.

ASSESSMENT AND TREATMENT CONSIDERATIONS

When evaluating a vomiting patient certain patterns can help to distinguish among the causes:

- **Early morning, after awakening**
  - Often associated with pregnancy, alcoholism, uremia or CNS or space occupying tumors.
- **Projectile vomiting associated with headache**
  - Intracerebral diseases.
  - Tumors or acute hemorrhage.
- **Postprandial (after eating)**
  - If seen 30 to 60 minutes after eating it suggests esophageal stricture, gastric outlet obstruction or gastric retention.
- **During weeks 8-12 of pregnancy**
  - Seen in about 50% of patients with intractable nausea/vomiting (termed - hyperemesis gravidarum) causing dehydration.
- **During third trimester of pregnancy (weeks 28-42)**
  - Possibly pre-eclampsia, especially when severe hypertension and edema are present.
- **With mild fever, diarrhea, abdominal discomfort**
  - Viral or bacterial Infection of the gastrointestinal tract.
- **Vertigo, dizziness, and/or ringing in the ears**
  - Inner ear infection or injury, acute motion sickness, or severe hypertension.

Vomiting produces three responses initiated by the medulla; a motor response, a parasympathetic nervous system (PNS) response and a sympathetic nervous system (SNS) response as below:

- Increased salivation to protect the enamel of teeth from stomach acids (PNS response).
- Retroperistalsis pushing the contents of the digestive tract into the stomach.
- An increase in abdominal pressure as the abdominal muscles contract forcing stomach contents into the esophagus while the lower esophageal sphincter relaxes (motor output); it should be noted that the stomach itself does not contract in the process of vomiting.
- A SNS response which causes sweating and increased heart rate; however in some cases an exaggerated vagal response may be seen causing brief hypotension and bradycardia.

The administration of an antiemetic to a patient in DKA or closed head injury is not appropriate as they will typically not respond until the illness and volume depletion is treated.

Electrolyte and fluid changes can occur due to excessive vomiting and adequate nausea control can result in respiratory, circulatory and behavioral improvement.

Vertigo is the sensation of motion, classified as either **subjective or internal** (“I feel myself turning”) or **objective or external** (“things spin around me”). It may also be a sensation such as tilting.

MEDICATION CONSIDERATIONS

- Zofran (Ondansetron) and Anzemet (Dolasetron) are serotonin receptor antagonists thought to work by blocking the reception of serotonin at 5-HT3 receptors. Because blockade tends to last for several hours further doses will not provide any additional benefit or control of nausea/vomiting.

- The onset of action for intravenous Zofran and Anzemet is immediate. Peak effects occur rapidly, and the duration of action is about 8 hours. Anzemet is an optional drug that may be administered in place of Zofran; however it is not to be administered with Zofran (or vice-versa).

- Zofran and Anzemet are contraindicated in known drug hypersensitivity, patients with hypersensitivity to other selective 5-HT3 receptor antagonists or closed head trauma (unless ordered by a physician).

POTENTIAL PITFALLS

- Antiemetics are not recommended for treatment of uncomplicated vomiting in children, and their use is limited to prolonged vomiting of a known origin (e.g., MS administration).

- Failure to consider head trauma, MI, stroke or hypoglycemia as the causes for nausea and vomiting.
Acute pain management consists of pain relief with preservation of airway reflexes and consciousness. EMS is expected to be knowledgeable about pain management and perform an assessment of pain and administer analgesia when indicated (especially if associated with autonomic nervous system hyperactivity such as tachycardia, hypertension, diaphoresis etc.).

**ASSESSMENT AND TREATMENT CONSIDERATIONS**

- Analgesia requires the continuous use of pulse oximetry, BP and ECG monitoring in addition to frequent reassessment of patient ventilatory status at all times.
- Pain assessment includes location, quality, intensity, onset, duration and frequency of pain, as well as factors that relieve or exacerbate the pain.
- Adult pain is typically assessed using a 10-point scale with zero representing no pain and 10 representing the worst pain possible.
- Pain differentiation is often classified as: Mild: 1 to 3 / Moderate: 4 to 6 / Severe: 7 to 10.

**Indications for pain management;**

- Significant orthopedic or extremity trauma
- Hydrofluoric acid burns (MS preferred)
- > 10% 2nd, 3rd degree burns
- > 20% 1st degree with ANS activity
- Terminal illness (MS preferred)
- ACS after 3 SL NTG (MS preferred)
- Significant eye / dental trauma
- Pain from sickle cell crisis or diving illness
- Abdominal or back pain with ANS hyperactivity
- Active labor with imminent delivery

**Contraindications for pain management;**

- No complaint of pain or discomfort
- Known or suspected aortic dissection
- Drug OD / poisoning / alcohol intoxication
- Head trauma or acute stroke
- Altered mentation or hypotension
- Respiratory depression (unless intubated)
- Bronchospasm (unless intubated)
- Significant trauma to the chest or abdomen

**Contraindications for nitrous oxide - all of the above plus;**

- Inability of patient to self-administer NO
- Pregnancy other than during active labor
- Undiagnosed abdominal pain
- Decompression illness
- Acute sickle cell crisis
- Acute psychosis
- Major facial or chest trauma
- Hypoxia (SP0₂ reading < 94%)
- COPD, asthma, bronchospasm, pneumothorax
- Suspected bowel obstruction

Sickle cell disease (SCD) can cause clumping (sickling) of RBC’s and occurs in 1 in 500 African-American births. A sickle cell vasoocclusive crisis occurs if circulation is obstructed by clumped RBC’s leading to organ ischemia and severe pain of the joints, tissues and organs.

Obtain a 12-lead ECG in any patient with significant co-morbidities or risk factors for CV disease.

**MEDICATION CONSIDERATIONS**

- The onset of action for IV Fentanyl is immediate; peak effects occur within three minutes and the duration of action is about 30 minutes.
- The onset of action for intravenous Morphine is immediate; peak effects occur within 20 minutes and the duration of action is about 2 to 4 hours.
- Nitrous Oxide (NO) has an onset within 1 to 2 minutes and lasts for about 5 minutes. It easily diffuses into gas pockets in patients (e.g., bowel obstruction, pneumothorax), and as nitrogen leaves it is replaced by NO which increases pressure causing further injury (e.g., intestinal rupture etc.).
- Naloxone must be available for reversal anytime Fentanyl or Morphine is administered for pain.
- Morphine should be withheld when RV Infarction is suspected, SBP is < 90 mmHg, or the HR is less than 50 as reflex bradycardia, preload reduction and worsening of BP can occur.
- Lorazepam may be useful in musculoskeletal injuries when Fentanyl fails to provide pain relief or severe muscle spasm is compromising distal blood flow. Contact Biocare Medical Control for use.

**POTENTIAL PITFALLS**

- Failure to monitor patient status when administering medications or fluid in large volumes rapidly.
- Rapid administration of Morphine will cause nausea/vomiting from a histamine release and stimulation of the brain vomiting center; always give MS very slowly (3 mg per minute).
**PALLIATIVE CARE**

Palliative care is a form of comfort care for terminally ill patients and is applied when death is imminent (although cessation of respiration or pulse may have not yet occurred). Presence of a valid OOH-DNR order should be explored and/or confirmed, although care can still be provided if not present.

**ASSESSMENT AND TREATMENT CONSIDERATIONS**

- Palliative care also includes sympathy for family emotional suffering, loss and bereavement.
- Delirium is a condition of severe confusion and rapid changes in brain function, usually caused by a physical or mental illness (the word delirium is derived from the Latin term meaning “off the track”).
  - Delirium is often a common end-of-life occurrence, however many episodes are reversible by routine therapies such as IV fluid rehydration; the problem often lies in determining which is an end-of-life episode and which is a potentially reversible event.
- Three stages of imminent death commonly observed are:
  - **Early Stage**: bed bound; loss of interest and ability to drink/eat; cognitive changes: either hypoactive or hyperactive delirium, or increasing sleepiness.
  - **Mid Stage**: further decline in mental status or obtundation, “death rattle” is heard (pooled oral sections that are not cleared due to loss of swallowing reflex), and fever may be present.
  - **Late Stage**: any of above plus coma, cool extremities, altered breathing (fast/slow), and death. EMS is often requested in the late stage of the illness when intense family distress is occurring.
- "**Culture-sensitive**“ care is the awareness of beliefs, values, traditions and practices of other cultures and honoring them when providing care. A patient or family of a particular cultural, ethnic or religious background may deal with terminal illness and death on different levels.
- IO access is only to be used when; (1) IV access cannot be obtained, (2) profound hypotension or a life-threatening event requiring fluids or IV medications is present, (3) there is no DNR order present which prohibits this type of care, and it is approved via on-line consultation with a Biocare physician.
- During the final hours of a terminal illness it may be acceptable to provide only comfort measures and not transport to allow the patient to die with dignity at home. This should be considered only if the patient (if able), family and a Biocare physician all agree it would be in the patient’s best interest.
- Patients commonly fear their suffering will be prolonged and that no one will control it when it becomes severe. Breakthrough pain is said to be present when slow-release analgesic medications (e.g. Fentanyl patch, MS-Contin tablets) become less effective in controlling pain. Many terminally ill patients experience breakthrough pain often having an intensity of over 7 on a 10-point scale.
- Access of PICC, central venous, subclavian, or tunneled catheters must be properly accessed and occasionally flushed with heparin (except Groshong). Only non-coring needles (Huber needles) are to be used when accessing a tunneled port as permanent damage can occur using a regular needle.

**MEDICATION CONSIDERATIONS**

- Morphine helps reduce tachypnea and dyspnea by blunting the response to CO₂ retention or oxygen decline, reducing dyspnea and anxiety without producing significant respiratory depression.
- Naloxone is generally not indicated for the terminally ill patient unless a narcotic overdose is known or highly suspected. All dying patients will at some point have an altered mentation with respiratory changes. Naloxone use in a patient dependent on narcotics can precipitate an abstinence syndrome which in itself can be life-threatening and painful for the patient.
- If naloxone must be given do not administer in large doses (0.4 mg and above) instead; dilute 0.4 mg of naloxone with 9 mL of NS to a total volume of 10 mL (1 mL = 0.04 mg) and administer 1 mL per/min until patient is responsive (typically noted after 2-4 mLs).

**POTENTIAL PITFALLS**

- Failure to allow the parents to hold the child as he or she dies, if so asked (and able to do so).
- Providing false reassurances when death is imminent. Fear of death is a normal reaction and can make people angry, depressed, or aggressive and false reassurances can only make matters worse.
- Use of Lasix is rarely indicated to treat congestion in the dying terminally ill patient since they are often volume depleted. Contact a Biocare physician before Lasix use in these patients.
In some emergency situations, it is difficult or impossible to perform laryngoscopy and intubation without use of adjunctive medications. Pharmacologically assisted intubation (PAI) utilizes sedative medications, but not paralytics, to assist in the difficult intubation process. Utilizing PAI is not without danger and every case should be approached with the mindset the patient may not be able to be intubated or ventilated.

**ASSESSMENT AND TREATMENT CONSIDERATIONS**

- The use of PAI medications is not without significant risk and one must have a thorough understanding of the properties, indications, and contraindications of all drugs involved as their use can turn an urgent airway problem into a life threatening situation.
- Aspiration in patients with impaired airway protective reflexes can be reduced by having pressure applied to the patient’s cricoid cartilage (Sellick’s maneuver), pushing the trachea posteriorly, compressing the esophagus against the cervical vertebrae preventing gastric inflation and reducing the risk of aspiration.
- If intubation attempts are difficult or unsuccessful the adequacy of basic airway management should be made. It is often preferred to maintain an airway by BVM than continue multiple attempts at intubation.
- If unable to intubate after PAI, continue cricoid pressure and begin BVM ventilations. **Cricothyrotomy is to be used as a last resort measure only when unable to ventilate at all.**
- Airway stimulation during intubation will induce a sympathetic discharge resulting in hypertension and tachycardia (the pressor response); this pressor response can worsen myocardial and cerebral injury or induce ventricular dysrhythmias.
- **All patients must have continuous ECG, BP, pulse oximetry, blood pressure and Etco2 detection and monitoring at all times.**

**MEDICATION CONSIDERATIONS**

- Etomidate induces unconsciousness within 2 minutes and has little or no effect on cardiac output, peripheral circulation or pulmonary circulation. It will not attenuate (weaken) the pressor response to intubation nor provide any pain relief. Nausea and vomiting, coughing, laryngospasm, hiccups, and involuntary muscle movements are side-effects that may be seen after administration.
- Midazolam is a short acting benzodiazepine that can be given in small doses as needed for sedation.
- Midazolam should not be administered in the presence of hypotension (substitute fentanyl instead).
- Lidocaine helps to diminish the hypertensive response that increases ICP in head trauma or severe hypertension by blunting airway and cough reflexes; in addition it helps decrease the risk of ventricular dysrhythmias (during MI, excessive sympathetic stimulation etc.) during intubation. To be effective, lidocaine must be given at least 3 minutes prior to intubation.

**POTENTIAL PITFALLS**

- Use caution when administering opioids in patients who are in shock as they can block the sympathetic compensatory response to hypotension, resulting in cardiovascular collapse.
- Obese patients tend to desaturate oxygen rapidly due to a reduced functional reserve capacity, weight of viscera upon diaphragm, compliance changes and weight of chest wall to elevate with accessory muscles of respiration. Use extreme caution when utilizing PAI in these patients.
- Use caution when using BVM ventilation before intubation as excessive ventilatory rates and tidal volumes can lead to gastric distention which greatly increases the risk for aspiration.
Rapid Sequence Intubation (RSI) – Optional

RSI is a method of intubating patients who have a gag reflex or would otherwise be difficult to intubate by the orotracheal route (it is not to be used with nasotracheal intubation). Intubation is accomplished by use of a sedative and a paralytic to allow for easier intubation. Because RSI can be dangerous when used incorrectly, not all paramedics are automatically eligible for its use until approved and properly trained.

Assessment and Treatment Considerations

- **Indications for RSI are same as PAI.**
- Patients with upper airway obstruction (i.e., croup, epiglottitis, pharyngeal swelling, edema etc.) should not be paralyzed as paralysing relaxes the pharyngeal muscles, which can obscure landmarks, making BVM ventilation impossible. Use PAI instead.
- **RSI requires a least 2 RSI trained providers to be present before it is attempted.**
- Patients with c-spine trauma require three providers; one of which is familiar with in-line stabilization (not traction) during intubation.
- **All patients must have continuous ECG, BP, pulse oximetry, BP and EtcO2 detection and monitoring at all times.**
- Cricoid pressure is to be initiated as soon as the patient becomes drowsy and maintained until cuff inflation. If unable to intubate maintain pressure and use BVM ventilation.
- Cricothyrotomy is to be considered as a last resort measure to use only when unable to ventilate or oxygenate via BVM after RSI.
- If the patient suddenly arrests after succinylcholine immediately administer medications outlined under page CA-03 Presumed Hyperkalemic Arrest.

Medication Considerations

- Succinylcholine (Sch) is not to be used in severe burns and with extreme caution (if at all) in the following patient types (Medical control must be contacted first before it’s use in these patients):
  - Any personal or family history of malignant hyperthermia,
  - extensive muscle trauma or crush injuries > 4 hours old,
  - Disuse muscle atrophy or prolonged bed immobilization (e.g., para or quadriplegia),
  - SCI < 1 yr old, previous major stroke with muscle wasting or Guillain-Barré Syndrome,
  - Duchenne’s, Myotonic and Becker's Muscular Dystrophy or Amyotrophic lateral sclerosis (ALS),
  - Known intra-abdominal sepsis or renal failure.
- Atropine is to be used when bradycardia is present, excessive oral secretions hamper intubation, or anytime a second dose of Sch is used. Atropine should not be administered if the patient has a tachydysrhythmia such as SVT, VT, or A Fib with RVR.
- Lidocaine 2% should not be used if the patient has: HR < 60 bpm, second or third degree AV block, idioventricular or ventricular escape rhythms.
- Always use sedation/analgiesia in the previously awake patient who receives a paralytic as neither Succinylcholine nor Vecuronium has sedative or analgesia properties.

Potential Pitfalls

- Failing to provide cricoid pressure to minimize gastric distention and the risk of aspiration.
- Prolonged attempts at intubation causing hypoxia; attempts should be limited to 45 seconds each or whenever oxygen sats drop below 90% (may re-attempt after re-oxygenation has occurred).
REACTIVE AIRWAY DISEASE (RAD)

Reactive airway disease (RAD), frequently referred to as asthma, occurs from a variety of bronchial irritants including smoke, exercise, change in weather/humidity or pulmonary infections such as pneumonia. Other causes of RAD include foreign material aspiration, early pulmonary edema from CHF, COPD or bronchiolitis in infants.

ASSESSMENT AND TREATMENT CONSIDERATIONS

- Asthma typically begins in childhood; COPD most often develops in smokers and former smokers in their forties.
- Asthma presents as wheezing, dyspnea, chest tightness and non productive cough due to an allergen response. COPD can also present with wheezing and dyspnea, but it is often due to infections with a productive cough.
- Intubation may aggravate bronchospasm, induce laryngospasm / barotrauma, and depress circulatory function; its use should not be taken lightly.
- Indications for intubation include cardiac or respiratory arrest, severe hypoxia unresponsive to oxygen or medications, exhaustion or acute mental status decline.
- If intubated cannot be avoided use as large an ET tube as possible. Avoid small diameter tubes as airflow resistance is increased as tube diameter reduces.
- Intubated patients or those needing ventilatory support MUST be ventilated slowly (6 to 10 bpm) along with small tidal volumes to avoid auto-PEEP (positive end-expiratory pressure) or barotrauma.
- Auto-PEEP often leads to profound hypotension from collapse of the right atrium and great vessels due to massive intrapulmonary pressure and is directly related to aggressive and/or rapid ventilation (it is much more common if intubated). If auto-PEEP is suspected temporarily halt ventilations for approximately 60 seconds, disconnect the BVM and allow excessive lung pressure to dissipate.
- Status asthmaticus is said to be present when severe airway obstruction and asthmatic symptoms persist despite standard asthma therapy. It can rapidly progress to asphyxiation if not treated.

MEDICATION CONSIDERATIONS

- Magnesium is useful in RAD as it relaxes bronchial smooth muscle which can reduce bronchospasm and increase the airway diameter; this occurs by it competing against calcium which can cause induce smooth muscle tissue constriction (Magnesium is a physiologic calcium channel blocker).
- Lorazepam can provide additional symptomatic relief for the COPD patient and may be considered when a patient’s anxiety level is very high due to the sensation of being unable to breathe.
- Stimulation of beta-2 receptors in the lungs through use of inhaled Albuterol leads to production of cyclic AMP, resulting in relaxation of bronchial smooth muscle. Lower doses Albuterol should be attempted first and increased as needed when decompensation or failure to respond occurs.
- Biocare Medical Control consultation is suggested before using Epinephrine in patients > age 50 years or anytime ischemic heart disease is present.
- Ipratropium (Atrovent) works on the acetylcholine driven nerves that constrict muscles in the airway walls; Atrovent is an anticholinergic agent which relaxes the muscles by blocking these nerves.
- The following flow rates and oxygen delivery devices are suggested for use:
  - Asthmatic - no distress cannula at 4 to 6 lpm to maintain oxygen saturation above 94%.
  - Emphysemics - no distress cannula at 2 lpm and increase as needed up to 4 lpm via cannula.
  - Asthmatic - in distress/hypoxic: NRB at 15 lpm increase as needed
  - Emphysemics - in distress/hypoxic: NRB at 8 lpm increasing flow rate as needed.

POTENTIAL PITFALLS

- Failing to remember that “not all that wheezes is asthma.” A provider must be able to recognize other causes of wheezing such as pulmonary edema where Epinephrine use is contraindicated.
- Failure to consider tension pneumothorax in an intubated RAD patient who suddenly goes into PEA and now cannot be ventilated.
Seizures are defined as an episode of abnormal neurologic function caused by an inappropriate electrical discharge of brain neurons. There are over 24 different forms of seizure activity and many brief disturbances of neurological function (including cardiac arrhythmias like torsades) which can mimic seizure activity. This guideline focuses on the patient with sustained or multiple seizure activity.

## ASSESSMENT AND TREATMENT CONSIDERATIONS

- **Status epilepticus (SE)** is two or more sequential seizures without full recovery of consciousness between seizures or more than 30 minutes of continuous seizure activity.
- SE has many effects including massive release of catecholamines causing hypertension, tachycardia, dysrhythmias, hyperglycemia, hyperthermia, and acidosis from muscle rigidity and poor ventilation.
- Generalized seizures (or grand mal seizures) can be divided into tonic, clonic, and postictal phases.
  - **Tonic phase**: all the muscles contract and the person appears to cry out chest muscles contract and force air out. Breathing is irregular and can lead to hypoxia if allowed to persist for > 5 minutes.
  - **Clonic phase**: limbs jerk from the muscles contracting and relaxing in quick succession; the patient may bite their tongue or cheeks at this point.
  - **Post-ictal phase**: the patient begins to awaken, but is confused and slowly becomes aware over minutes to hours.

- **A febrile seizure (FS)** is often a self-limiting seizure that occurs when rapid body temperature elevation (> 102°F) causes generalized seizure activity lasting 1 to 5 minutes. About 90% of all cases occur from 6 months to 3 years when the brain is immature, is more excitable and has a lower seizure threshold (fever lowers it even further).
  - Reducing the temperature can help lower the febrile seizure risk and cooling measures include uncovering the patient and sponging with lukewarm—*not cold*—water until the temp reaches 101°F.
  - Some patients with difficult to control seizures have what is called a vagus nerve stimulator (VNS), a pacemaker-like device implanted in the left chest that sends impulses (stimulation) into the brain via the left vagus nerve to suppress seizure activity. Stimulation occurs once a magnet (carried by the patient or family) is placed over the generator for 30 seconds.
  - If present during the onset of seizure activity, thoroughly document onset of seizure including, initial site seizure activity first began, (i.e., full body, focal motor, Jacksonian March etc.), time of seizure onset, and seizure duration (precise documentation helps determine focus of seizure).
  - During the first 5 minutes of a seizure focus efforts on airway management and 100% oxygenation, rapid glucose testing, IV access, establishing cardiac monitoring and continuous pulse oximetry.

## MEDICATION CONSIDERATIONS

- Children with an extensive seizure history could possibly receive Diazepam via rectal gel prior to EMS arrival during seizures at school or home. If rectal diazepam was given prior to EMS extreme caution must be used if additional benzodiazepines are administered as CNS depression can occur.
  - Because many seizures self-terminate in less than 5 minutes the administration of a benzodiazepine is often not necessary and if used can lead to unwanted CNS and respiratory depression.
  - Midazolam via the intranasal (IN) route is an option when vascular access is unavailable. Midazolam is water soluble, rapidly absorbed and is delivered directly into the blood and CSF via nasal mucosa.

## POTENTIAL PITFALLS

- Not transporting for further evaluation (or if the family refuses, recommending evaluation) of any patient who experienced a febrile seizure as it may be associated with a serious infection.
A stroke is characterized by the sudden loss of blood flow to an area of the brain, resulting in a loss of neurologic function. Also called cerebrovascular accident (CVA) or “brain attack”, stroke is a nonspecific term with multiple causes, including thrombosis, embolism, and hemorrhage. Consider stroke in any patient with acute focal neurologic deficit or any sudden alteration in the level of consciousness.

**ASSESSMENT AND TREATMENT CONSIDERATIONS**

- Focal neurologic deficits often involve changes in nerve function in a specific location: **focal location**: (i.e., left face, right face, left arm, tongue etc), **specific function change**: (speech affected, but not the ability to write), **sensation change**: includes paresthesia, numbness, or decreases in sensation, or **movement change**: includes paralysis, weakness, abnormal muscle tone or control.

- Few patients with ischemic stroke are ultimately eligible for fibrinolytic therapy because they fail to arrive at the receiving hospital within 3 hours of onset of symptoms; **all “brain attacks” should be treated by EMS with the same urgency as trauma or acute MI**.

- EMS should document exactly when symptoms began as drug treatment is often based from the time that the patient was last known to be symptom free. If the patient awakens with stroke symptoms, then the time of onset is defined as the time the patient was last seen without symptoms.

- Continuous ECG monitoring and a 12-lead are important as AMI and arrhythmias may occur secondary to disturbances in the sympathetic and parasympathetic nervous system function.

- ECG changes seen may include ST segment depression, QT prolongation, deeply inverted T waves (cerebral T waves), and large U waves.

- **Consider assessing using the Cincinnati Pre-Hospital Stroke Scale:**
  - **Assess facial droop**: have the patient show their teeth or smile
  - **Assess arm drift**: have patient close their eyes, hold arms out straight with palm up for 10 seconds (a drop of one side is called a pronator drift)
  - **Assess speech**: have them say, “you can’t teach an old dog new tricks”

- The presence of facial droop, arm drift, or abnormal speech increases the likelihood of a stroke.

- In the hypertensive stroke patient, antihypertensive therapy often leads to worsening of cerebral perfusion. However, prehospital treatment of HTN may be considered only when AMI, acute heart failure or known aortic dissection is present, and **ONLY** upon verbal approval of a Biocare physician.

**MEDICATION CONSIDERATIONS**

- Aspirin use during the acute phase **IS NOT** indicated until an intracerebral hemorrhage has been excluded as its administration could worsen hemorrhage. **In the patient with chest pain and signs of acute stroke a physician must be contacted for guidance prior to ASA administration.**

- Glucose should be avoided unless hypoglycemia is present as its use can increase anaerobic metabolism and lactic acid production in ischemic brain tissue, worsening neurological outcome.

**POTENTIAL PITFALLS**

- Failure to consider patient history during assessment of acute stroke (atrial fibrillation and HTN are leading causes of stroke).

- Strokes are not uncommon in children and when present are often due to clotting disorders, heart disease, and sickle cell disease.

- The rapid or over infusion of IV fluids in the absence of hypotension or hypovolemia increases the risk of cerebral edema.

- Acute blood pressure reduction except under direct physician order. Rapid BP reduction can worsen cerebral blood flow from a drop in the blood pressure.
There are a significant number of problems that may be classified as Obstetrical Emergencies; including abortion, ectopic pregnancy, pre-eclampsia, eclampsia, abnormal deliveries (breech, prolapsed cord, limb presentation, and multiple births), bleeding during any trimester, abruptio placenta, placenta previa, uterine rupture, uterine inversion, and post-partum hemorrhage.

**ASSESSMENT AND TREATMENT CONSIDERATIONS**

- A breech birth (or breech presentation) refers to the position of the baby in the uterus such that it will be delivered buttocks first as opposed to the normal head first position.
- Breech is common during early pregnancy (about 1/3 of all babies at 24 weeks), but by week 32 most are in the head-down position. When there are twins, often one of them is in the breech position.
- Types of breech presentations:
  - **Frank** - the butt comes first, the legs are flexed at the hip and extended at the knees. About 65% are frank breech (most common presentation).
  - **Complete** - the hips and knees are flexed so that the baby is sitting cross-legged, with feet beside the bottom (least common presentation).
  - **Footling** - one or both feet come first, with the bottom at a higher position. This is rare.
  - **Kneeling** - the baby is in a kneeling position, with one or both legs extended at the hips and flexed at the knees. This is also rare.
- Every effort should be made to deliver a breech birth in the hospital, particularly if it will be the mother’s first baby. Breech presentations carry a much greater risk of the baby’s head becoming wedged in the birth canal after the body is delivered, resulting in profound asphyxia or infant mortality.
- Immediately after delivery, place the infant at the same level as the mother’s abdomen. The baby should remain at this height until you have clamped and cut the umbilical cord. Placing the infant higher can cause the baby to lose blood into the placenta; placing the infant lower can result in excessive transfusion of blood from the placenta to the baby.

**MEDICATION CONSIDERATIONS**

**POTENTIAL PITFALLS**

- Performing any form of internal vaginal exam which can puncture the placental membrane in placenta previa, causing profound hemorrhage, shock, and possibly death to both the fetus and mother.
- Placing the infant higher than the mother’s abdomen which can cause the baby to either lose blood or receive an excess of blood from the placenta.
- Causing red blood cell destruction by “milking” the umbilical cord.
- Pulling on the umbilical cord to expedite placental delivery.
- Failure to initiate rapid transport when indicated (known or suspected breech presentation in the absence of an imminent delivery or preterm labor with associated low gestational age).
- Failure to provide early or immediate ED notification of any high risk delivery in progress.
- Failure to initiate rapid transport when a life-threatening event is suspected.
Although the majority of pregnancies are uneventful, sometimes complications do occur. Complications during pregnancy or obstetrical emergencies may include: spontaneous abortion, ectopic pregnancy, pregnancy induced hypertension, eclampsia, vaginal bleeding during any trimester of pregnancy, abruptio placenta, placenta previa, uterine rupture, uterine inversion, and excessive post-partum hemorrhage.

**ASSESSMENT AND TREATMENT CONSIDERATIONS**

- When a pregnant patient remains supine on her back, the enlarged uterus can impede venous return from the vena cava causing a supine hypotension syndrome. Thus, all pregnant patients greater than 20 weeks gestation should be transported lying on their left side if at all possible.

- **Placenta previa** is when the placenta covers part or the entire cervix with the common symptom of *bright red vaginal bleeding* not associated with abdominal tenderness or pain, especially in the 3rd trimester (ranging from light to very heavy). It is often related to multiple pregnancies/births, advanced age, previous C-section delivery, and previous abortion.

- **Placental abruption**, or abruptio placenta, is the early separation of the placenta from the uterus. The most common symptom of abruption is *dark red vaginal bleeding* with knife-like pain during the 2nd or 3rd trimester.

- **Uterine rupture** is a sudden tear in the wall of the uterus during labor often due to a previous c-section incision (90% of cases), trauma, or when drugs are used to promote labor (e.g., pitocin).
  - Fetal distress (HR < 100) is the most reliable symptom as pain is not always present, and bleeding may be absent if the fetal head blocks the pelvis. Shock can rapidly occur as the maternal system delivers almost 500 mL of blood/min to the uterus; with rapid blood loss (2,000 mL in 50% of cases).

- **Ectopic pregnancy** is the implantation of an egg in a location outside of the uterine cavity, often in the fallopian tubes (> 95%). If the pregnancy is allowed to continue, the tube ruptures and hemorrhage ensues. Patients often present with lower back and/or abdominal pain, cramping, tenderness on one side of the pelvis, and vaginal bleeding (about 40% of cases).

- **Uterine inversion** is a life-threatening complication often seen when attempts to deliver the placenta by pulling on the cord cause the uterus to turn inside out, with the uterus potentially protruding outside the vagina causing bleeding and shock. If UI occurs, keep moist and rapidly transport to the hospital.

- **Spontaneous abortion** (miscarriage) is the loss of a fetus before 20 weeks gestation with about 85% occurring in the first 12 weeks (pregnancy losses after the 20th week is a preterm delivery). Risks for ectopic pregnancy includes; over 35 years, previous ectopic, pelvic or abdominal surgery, pelvic inflammatory disease, and multiple induced abortions. *Spontaneous pregnancy loss* is a better choice of words rather than “abortion” when caring for a patient or family dealing with losing a pregnancy.

- **Preeclampsia** is a pregnancy-induced condition that includes hypertension (PIH), protein in the urine, edema, weight gain, blurred vision, headaches, dizziness and stomach pain. Severe preeclampsia with a SBP > 160 and/or a DBP > 110 should be treated with magnesium (or possibly nitroglycerin) to prevent IC hemorrhage and seizures. Contact a Biocare physician for guidance in these patients.

- **Eclampsia** is a true medical emergency and occurs when preeclampsia progresses to seizures (usually brief) with coma and possibly death occurring if left untreated. Seizures may be seen up to 3 weeks *post birth* (seizures in the 1st trimester or well into the postpartum period are more likely to be CNS related and not eclampsia). The only definitive treatment for eclampsia is delivery of the fetus.

**MEDICATION CONSIDERATIONS**

- Magnesium stops and prevents seizures as it reduces the BP and lessens cerebral vasospasm. However, when seizures continue after magnesium, a benzodiazepine should be used.

- Magnesium toxicity causes loss of deep tendon reflexes, respiratory depression and ultimately respiratory arrest. If any of these occur, doses are to be held and IV calcium gluconate given.

- Never administer 50% magnesium via IV bolus without first diluting it to a 20% solution. To make a 20% solution add 8 ml of 50% Magnesium to 12 ml of NS = 4 g in 20 ml (20% solution).

**POTENTIAL PITFALLS**

- Performing any type of internal vaginal exam, puncturing the placental membrane in placenta previa.

- Failure to initiate rapid transport when a life-threatening event is suspected.
Childbirth is a natural process and in most cases deliveries proceed without complications. When called for a possible prehospital birth, EMS personnel should determine whether there is time to transport the mother to the hospital or if childbirth is imminent and prepare to assist with the delivery.

**ASSESSMENT AND TREATMENT CONSIDERATIONS**

- **APGAR scoring** aids in determining whether the baby is viable on its own or if it requires resuscitation and is documented at 1, 5, and 10 minutes intervals in all newborns.
- **Do not wait to obtain a 1-minute score when resuscitation is urgently needed.**
  - If 5 minute score is < 7, re-score every 5 minutes for 20 minutes.
  - A score of **7 to 10** is normal, **4 to 7** indicates some resuscitation required, and **0 to 3** indicates resuscitation is required.
- If a nuchal cord (cord wrapped around the neck) is present, carefully try to slip the cord over the baby's head or deliver the baby through the loop of cord. Clamping and cutting a nuchal cord should only be considered when unlooping is not possible and asphyxiation secondary to neck constriction is highly likely. **CAUTION:** the premature cutting of the cord before delivery can be life-threatening should shoulder dystocia occur (prevents respirations and early cord cutting removes its blood and oxygen supply).
- A patient care record must be completed on the mother and each newborn delivered with the following information to be documented on each (when able to obtain):
  - initial presentation at delivery (face down, breech, shoulder dystocia, limb presentation etc.),
  - date, time, location of birth, and gender (male or female),
  - time of amniotic membrane rupture and appearance of fluids (clear, meconium etc.),
  - APGAR and any resuscitation procedures or maneuvers needed to assist in the delivery,
  - prenatal care or health problems the mother has had and known problems of the pregnancy or fetus,
  - previous pregnancy history (written as gravida, para, X-X-X-X)," where gravida is the total number (#) of pregnancies (including present one), para is # of deliveries after 20 weeks, and X-X-X-X is the # of full-term infants - # of preterm infants - # of abortions - # of living children.
- Hypothermia is commonly seen in prehospital deliveries and can lead to bradycardia, hypoxia, hypoglycemia, acidosis, and respiratory distress. **Do not allow a newborn to become cold!**
- Uterine massage for 3 to 5 minutes can help control postpartum hemorrhage. Place one hand just above the pubic bone and the other hand at the level of the umbilicus. Press down into the abdomen to gently massage the uterus until it firms (should become about the size of a softball).
- True labor presents as a regular sequence of uterine contractions, with progressively increasing intensity and decreasing intervals between contractions; false labor by irregular, brief contractions of the uterus, usually with discomfort confined to the lower abdomen and groin.

**MEDICATION CONSIDERATIONS**

**POTENTIAL PITFALLS**

- Performing any type of internal vaginal exam; internal vaginal exams are forbidden in the field.
- Milking or stroking the umbilical cord to remove blood which can cause red blood cell destruction.
- Failure to initiate rapid transport when indicated (suspected breech presentation in the absence of an imminent delivery, preterm labor with low gestational age or known complication etc.).
- Pulling on the cord to expedite placental delivery, causing severe bleeding or uterine inversion.
- Allowing mother to go to the toilet before delivery (BM sensation means the baby is close to delivery).
- Having the mother push vigorously before the head crowns can tear the cervix if not fully dilated.
OBSTETRICAL - SHOULDER DYSTOCIA

Dystocia is defined as abnormal or difficult labor (eutocia is normal labor). Shoulder dystocia (SD) is a medical emergency and occurs when the baby’s head delivers, but the shoulders fail to deliver, becoming trapped behind the pubic bones. Shoulder dystocia is dangerous because it is possible for the umbilical cord to become compressed against the mother's pelvis, thereby cutting off blood flow between the placenta and the baby.

ASSESSMENT AND TREATMENT CONSIDERATIONS

- Use of the McRobert's position in conjunction with suprapubic pressure (pressure over the bladder) should be used if delivery fails to occur. This is accomplished by flexing the mother's legs toward her shoulders as she lies on her back to expand the pelvic outlet.
- Shoulder dystocia is difficult to manage even by obstetricians and although guidelines are outlined under the BLS section, this type of delivery should be handled by paramedic level personnel whenever they are on-scene.
- Applications of shoulder dystocia delivery guidelines assumes that delivery has begun, the head remains stuck after 3 contractions, and a safe and rapid transport to a hospital was not an immediately viable option.
- The "turtle sign" is when the fetal head suddenly retracts back against the mother's perineum after it emerges from the vagina. The baby's cheeks bulge out, resembling a turtle pulling its head back into its shell. This retraction is caused by the baby's anterior shoulder being caught on the back of the maternal pubic bone, preventing delivery of the remainder of the baby.
- Shoulder dystocia cannot be predicted or prevented because accurate methods for identifying which fetuses will experience this complication do not exist.
- Maternal complications from SD during delivery may include postpartum hemorrhage and perineal lacerations. Fetal complications from SD may include brachial plexus injury, clavicle or humerus FX, and cerebral hypoxia; any of these may occur, even in a delivery room with highly experienced personnel in attendance.
- SD is often associated with large babies (commonly over 9 lbs) often due to maternal diabetes; maternal obesity; prolonged gestation, or maternal short stature.
- When shoulder dystocia occurs, umbilical cord compression between the fetal body and the maternal pelvis is a potential danger as the fetal pH will drop by an estimated 0.14 per minute during delivery of the fetal trunk.

MEDICATION CONSIDERATIONS

POTENTIAL PITFALLS

- Failure to initiate rapid transport when indicated and able to do so.
- Application of excessive traction on the head or neck during delivery.
- Failing to provide early notification to the receiving facility of a delivery complication.
The vast majority of term neonates require no resuscitation beyond maintenance of temperature, suctioning of the airway, and mild stimulation. Most neonatal resuscitations in the prehospital setting occur without prior notice. The best resuscitation results are seen in delivery room, thus every reasonable and safe effort should be made to get the mother to a delivery room, unless the birth is imminent.

ASSESSMENT AND TREATMENT CONSIDERATIONS

- **Broselow Pediatric Emergency Tape:** The Broselow Tape may be used as a guide in determining drug dosages and equipment sizes for infants or children up to 34 kilograms. When an actual body weight is known, use the tape as a calculator, otherwise measure and calculate per tape instructions. If a dosing discrepancy occurs between the tape and the protocols, the protocols should be followed.

- Gasping is a series of deep single or stacked inspirations that occur in the presence of hypoxia and/or ischemia. It is indicative of severe neurologic and respiratory depression.

- **Acrocyanosis** - cyanosis of the hands and feet only - should not be confused with **central cyanosis** - cyanosis of the central trunk and mucous membranes - which indicates a more severe problem. Acrocyanosis, without central cyanosis, does not generally indicate systemic hypoxic.

- Pallor / mottling may be a sign of < cardiac output, anemia, hypovolemia, hypothermia, and acidosis.

- Suctioning of meconium before full body delivery is no longer recommended in infants born to mothers with meconium staining. Small amounts of meconium often only discolor the fluid without obvious particles being visible. Management with deep suctioning is not necessary in these patients.

- All newborns have difficulty tolerating the cold and depressed infants are especially at high risk for complications of cold stress. Always keep and maintain warmth in all newborns

- IO access is indicated only in life-threatening conditions when peripheral access cannot be obtained.

- Babies are usually born with a white, cheesy coating on their skin called "vernix" which is a combination of secretions and skin cells. Excess vernix may be cleaned off, but it is not necessary to remove all of it as it helps protective the skin.

- Compressions are delivered on the lower third of the sternum to a depth of approximately one third of the diameter of the chest. Two methods may be used:
  1. Two (2) fingers over the sternum while supporting the back.
  2. **Two (2) thumbs with fingers encircling the chest and supporting the back.** This technique may generate higher perfusion pressures and is the recommended technique to use when two providers are present and able to do so.

MEDICATION CONSIDERATIONS

- Acidosis in children is primarily a problem of ventilation and oxygenation. Sodium bicarbonate should be considered when other therapies are ineffective and resuscitation is prolonged (> 10 minutes) and is by on-line medical consultation approval only. The common dose is 1 to 2 mEq/kg IV/IO of a 4.2% solution of bicarbonate instead of the standard 8.4%.

- When administering epinephrine to a neonate consider removing the end cap from a 1:10:000 epinephrine syringe and inserting the needle of a 1 mL syringe directly into the center of the blue vial plunger. Draw the required amount of epinephrine into the 1 mL syringe from the epinephrine vial.

POTENTIAL PITFALLS

- When resuscitating a premature infant (< 37 weeks or < 5.5 lbs) extreme care should be taken to avoid too rapid or too large of fluid volume infusion as cerebral hemorrhage could occur.

- Failure to recognize and/or treat hypoglycemia when present, especially in neonates.

- Using a meconium aspirator with a non-adjustable suction device which exceeds 100 mmHg.

- Vigorous or deep suctioning causing vagal stimulation; brief gentle suctioning is all that is required.

- The pop-off valve on the BVM may need to be disabled in certain cases to increase airway pressure (i.e., premature infants or meconium aspiration syndrome). Use extreme caution if you must disable a pop-off valve as barotrauma can occur. Contact Biocare Medical Control for guidance.
Beta blockers (BBs) are used for angina, HTN, CHF, tachydysrhythmias, AMI, migraines, headache, glaucoma, tremor, and anxiety. These drugs block beta-adrenergic receptors resulting in decreased cyclic AMP production and blunting of the catecholamine effects of epinephrine and norepinephrine.

### ASSESSMENT AND TREATMENT CONSIDERATIONS

- Beta blockers include (most, but not all generic names of beta blockers end with "olol").

<table>
<thead>
<tr>
<th>Beta blockers</th>
<th>Considerations</th>
</tr>
</thead>
<tbody>
<tr>
<td>Betapace (sotalol)</td>
<td>Lopressor (metoprolol)</td>
</tr>
<tr>
<td>Blocadren (timolol) - eye drops for glaucoma</td>
<td>Nebilet (Nebivolol)</td>
</tr>
<tr>
<td>Cartol (carteolol)</td>
<td>Normodyne (labetalol) - also blocks alpha receptors</td>
</tr>
<tr>
<td>Coreg (carvedilol) - also blocks alpha receptors</td>
<td>Sectral (acebutolol)</td>
</tr>
<tr>
<td>Corgard (nadolol)</td>
<td>Tenormin (atenolol)</td>
</tr>
<tr>
<td>Inderal (propranolol)</td>
<td>Toprol-XL (metoprolol)</td>
</tr>
<tr>
<td>Inderal-LA (propranolol)</td>
<td>Trandate (labetalol) - also blocks alpha</td>
</tr>
<tr>
<td>Kerlone (betaxolol)</td>
<td>Visken (pindolol)</td>
</tr>
<tr>
<td>Levatol (penbutolol)</td>
<td>Zebeta (bisoprolol)</td>
</tr>
</tbody>
</table>

- Heart failure, hypoglycemia, coma and seizures (especially propranolol). Bronchospasm is more likely to occur with preexisting airway disease.
- The ECG may show sinus bradycardia, PRI and/or QRS widening, elongation of the QT interval leading to torsades, junctional rhythms, AV blocks, and IV rhythms in overdose.
- Pacing is often ineffective and should be used only for those patients who remain unresponsive to drug therapy or in torsades that is unresponsive to magnesium.

### MEDICATION CONSIDERATIONS

- Beta blockers are rapidly absorbed from the GI tract and the first signs of OD may appear within 20 minutes, but more often within 4 to 6 hours (may be longer in slow release drugs).
- BB toxicity causes hypotension, bradycardia, heart failure, hypoglycemia, coma and seizures (especially propranolol). Bronchospasm is more likely to occur with preexisting airway disease.
- The ECG may show sinus bradycardia, PRI and/or QRS widening, elongation of the QT interval leading to torsades, junctional rhythms, AV blocks, and IV rhythms in overdose.
- Pacing is often ineffective and should be used only for those patients who remain unresponsive to drug therapy or in torsades that is unresponsive to magnesium.

### POTENTIAL PITFALLS

- Anytime more than 2 mg of glucagon is given IV normal saline or sterile water is to be used as the diluent as large amounts of IV phenol can cause hypotension and renal disturbances.
- Calcium is always to be administered IV very slowly and with extreme caution (if at all) in persons on digitalis as hypercalcemia can occur, leading to ventricular irritability and arrest.
- Administration of atropine when the heart rate is above 60 bpm or when VT or VF is present.
- Failure to monitor the patient’s blood glucose level as hypoglycemia can occur from the beta blocker overdose.
Calcium-channel blockers (CCB) slow the rate at which calcium passes in and out of cardiac cells and vessel walls. CCB toxicity can lead to a pronounced decrease in intracellular calcium which can lead to cardiovascular dysfunction and collapse. CCBs are most commonly prescribed for hypertension, angina, atrial dysrhythmias and the control of migraine headaches.

ASSESSMENT AND TREATMENT CONSIDERATIONS

- Calcium channel blockers include; (several, but not all, generic names end with "pine").

<table>
<thead>
<tr>
<th>Brand Name</th>
<th>generic name</th>
<th>long half life</th>
<th>notes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Adalat (nifedipine)</td>
<td>x</td>
<td>Nimotop (nimodipine)</td>
<td>x</td>
</tr>
<tr>
<td>Calan / Isoptin / Verelan (verapamil)</td>
<td>🌺</td>
<td>Norvase (amlodipine) - long half life (45 h)</td>
<td>x</td>
</tr>
<tr>
<td>Cardene (nicardipine)</td>
<td>x</td>
<td>Plendil (felodipine)</td>
<td>x</td>
</tr>
<tr>
<td>Cardizem (diltiazem)</td>
<td>🌺</td>
<td>Procardia / Procardia XL (nifedipine)</td>
<td>x</td>
</tr>
<tr>
<td>Cardizem SR / Cardizem CD (diltiazem)</td>
<td>🌺</td>
<td>Sular (nisoldipine)</td>
<td>x</td>
</tr>
<tr>
<td>Covera-HS (verapamil)</td>
<td>x</td>
<td>Tiamate (diltiazem)</td>
<td>🌺</td>
</tr>
<tr>
<td>Dilacor XR / Dilitia XT (diltiazem)</td>
<td>🌺</td>
<td>Tiazac (diltiazem)</td>
<td>🌺</td>
</tr>
<tr>
<td>DynaCirc (isradipine)</td>
<td>x</td>
<td>Vascor (bepridil)</td>
<td>V</td>
</tr>
</tbody>
</table>

- 💿 = cause significant bradycardia and milder hypotension
- 🌺 = causes heart blocks and hypotension
- ✗ = can cause reflex tachycardia (because its vasodilators without affecting cardiac conduction)
- V = can prolong the QT interval through its potassium blocking effects which can cause torsades.

- Diltiazem and especially verapamil tend to cause the most hypotension, bradycardia, conduction disturbances and deaths; nifedipine and similar CCBs (those with ✗) are generally less lethal and more likely to produce a reflex sinus tachycardia with less conduction disturbances.

- Children can become symptomatic with as little as one CCB tablet, therefore any child suspected of CCB ingestion of any amount should be transported by EMS for evaluation by a physician.

MEDICATION CONSIDERATIONS

- Calcium In large doses (up to 4 grams) can be used try and overcome the calcium blockade.
- Dopamine is often needed in the medium-to-high dose range (10 to 20 mcg/kg/min).
- **IV glucagon** stimulates glucagon receptors in cardiac cells, enhancing contractility like catecholamines do, but independent of the calcium blockade.

**Cardiac pacing in CCB toxicity**: Because CCBs delay calcium in and out of the cells, the cardiac cell cannot be forced into normal rhythmicity with cardiac pacing when bradycardic as stroke volume tends to be maximized at rates of 45-50 bpm; thus pacing at a rate > 60 bpm may actually reduce the stroke volume, further reducing cardiac output and hypotension.

POTENTIAL PITFALLS

- Calcium blockers can induce hyperglycemia because they block insulin release from the pancreas and patients should be carefully assessed for such.
- Anytime more than 2 mg of glucagon is given IV normal saline or sterile water is to be used as the diluent as large amounts of IV phenol can cause hypotension and renal disturbances.
- Failure to monitor the patient’s blood glucose level as hypoglycemia can occur from the calcium channel blocker overdose.
- Calcium is always to be administered IV very slowly and with extreme caution (if at all) in persons on digitalis as hypercalcemia can occur, leading to ventricular irritability and arrest.
- Administration of atropine when the heart rate is above 60 bpm or when VT or VF is present.
Carbon monoxide (CO) an odorless, colorless, two-molecule gas is produced by the incomplete combustion of organic fuels and readily displaces oxygen from hemoglobin to produce carboxyhemoglobin (COHb), resulting in tissue hypoxia. Gas-powered engines, furnaces, gas heaters, water heaters, pool heaters, wood stoves, kerosene heaters, indoor charcoal fires, and sterno fuel all produce significant amounts of CO.

### ASSESSMENT AND TREATMENT CONSIDERATIONS

- Early symptoms of CO poisoning, such as headaches, nausea, and fatigue, are often mistaken for the flu because the gas goes undetected in a home.
- **The primary goal in caring for patients with CO poisoning is removal from the source and high flow oxygen therapy to help remove CO from the hemoglobin.**
- The pregnant patient can appear well with little complaint, while the fetus is at increased risk for CO toxicity due to fetal hemoglobin’s affinity for CO. Thus all pregnant patients exposed to CO are to be transported for further evaluation.
- Firefighters and fire victims are at high risk for CO poisoning from an enclosed space fire therefore these patients’ should always be evaluated at a hospital for potential CO toxicity.
- If the history and findings suggest carbon monoxide exposure **handheld co-oximetry** can be used to determine if CO is present in the hemoglobin. However, therapies should not be based on any specific reading as SpCO levels may not correlate well with symptoms present.

<table>
<thead>
<tr>
<th>SpCO Level</th>
<th>Clinical Manifestations</th>
</tr>
</thead>
<tbody>
<tr>
<td>5%</td>
<td>Mild headache</td>
</tr>
<tr>
<td>10%</td>
<td>Mild headache, shortness of breath with exertion</td>
</tr>
<tr>
<td>10-20%</td>
<td>Moderate headache, shortness of breath</td>
</tr>
<tr>
<td>20-30%</td>
<td>Worsening headache, nausea, dizziness, fatigue</td>
</tr>
<tr>
<td>30-40%</td>
<td>Severe headache, vomiting, vertigo, altered judgment</td>
</tr>
<tr>
<td>40-50%</td>
<td>Confusion, syncope, tachycardia</td>
</tr>
<tr>
<td>50-60%</td>
<td>Seizures, shock, apnea, coma</td>
</tr>
</tbody>
</table>

### MEDICATION CONSIDERATIONS

- **The mainstay of therapy for CO poisoning is high flow oxygen.** The goal of oxygen therapy is to improve the oxygen content of the blood. Once started, oxygen must be continued to prevent later worsening of the condition as carboxymyoglobin unloads (no useful guidelines as to the length of this period, but commonly not observed in the prehospital setting).
- High flow oxygen using a non-rebreather mask supplies 100% oxygen which helps to clear COHb from the blood; this therapy reduces the half-life of COHb from about 4 to 5 hours to 1 hour.
- A 12-lead ECG should be completed on all patients with suspected CO poisoning as **subendocardial ischemia** (the area of the heart furthest away from the coronary blood flow) often occurs first since this area is very sensitive to hypoxia (look for ST segment depression in leads V1-V6).

### POTENTIAL PITFALLS

- Failure to provide oxygen because of “normal” pulse oximetry readings. **Pulse oximetry is to be considered inaccurate in CO poisoning** and is not to be used to determine oxygen need.
- Failure to verify on scene, all parties or family members who may be at risk from CO toxicity.
- Failure to assess patients with cardiovascular disease (CVD) for silent myocardial ischemia as even low COHb levels can have a severe impact on myocardial oxygenation.
- Failing to consider CO poisoning as a differential diagnosis when multiple patients simultaneously complain of vague and broad complaints which mimic those of a viral or flu like syndrome.
Tricyclic antidepressants, collectively today referred to as "cyclic" antidepressants (CA), are traditionally used in the treatment of depression, neuropathic pain, ADHD, and bedwetting. The cyclic antidepressants work by increasing low norepinephrine and serotonin levels allowing nerve impulses to return to normal.

### ASSESSMENT AND TREATMENT CONSIDERATIONS

- Many patients appear well initially and then rapidly decompensate with an average time from ingestion to clinical toxicity of approximately 1.5 hours.
- Cardiovascular toxicity commonly results in arrhythmias, conduction delays, and hypotension. CNS toxicity ranges from agitation to seizures.
- Significant ingestions often present with tachycardia due to anticholinergic effects, alpha blockade vasodilatation, or myocardial depression.
- Other signs of toxicity include, dry mouth, dilated pupils, brisk reflexes, and possibly respiratory depression.
- The presence of an R wave in AVR over 3 mm tall often can predict (> 80%) those patients who are more likely to have seizures and arrhythmias.
- The QRS width often correlates with likelihood of seizures or arrhythmias:
  - **Less than 0.10 sec** - unlikely to have seizures and arrhythmias
  - **Over 0.10 sec** - up to a 34% chance of seizures and up to a 14% chance of dangerous arrhythmias.
  - **Over 0.16 sec** chances increase to 50% for ventricular arrhythmias.
  - **Note:** QRS interval is best assessed using the limb leads (I, II, III).

### MEDICATION CONSIDERATIONS

- **CA toxicity is enhanced when metabolic or respiratory acidosisis is present** (lower pH, greater amount of "free" or active drug; higher pH, less free drug available). Inducing alkalosis by hyperventilation or sodium bicarbonate use can often reverse hypotension, arrhythmias, and conduction disturbances (it does not reverse CNS complications). CAs block sodium channels and the administration of sodium bicarbonate directly competes with this blockade to improve conduction and myocardial depression.
- Activated charcoal binds to cyclic antidepressants and should be used. However, many patients may vomit during or after administration; in this case, charcoal may be re-administered once if needed.
- CAs inhibits the re-uptake of catecholamines, eventually depleting presynaptic stores. Dopamine requires catecholamines to work, therefore it is often ineffective or requires larger dose to work.

### POTENTIAL PITFALLS

- Failure to remember that administration of amiodarone can increase the toxic effects on the myocardium in a cyclic antidepressant OD; lidocaine is a safer alternative to use instead.
- In cardiotoxic poisoning heart failure can limit the use of sodium bicarbonate and fluid administration. When present a Biocare physician should be consulted first as both can worsen pulmonary edema.
- Hypertension from blockade of norepinephrine reuptake is an early and transient finding; eventual depletion of catecholamines causing hypotension, in most cases HTN is best left untreated.
Narcotic is derived from the Greek word narkotikos, meaning "benumbing or deadening," and originally referred to substances that induced sleep (state of narcosis). Narcotics are taken orally, transdermally, injected, or in suppository form. As recreational drugs they are smoked, snorted, or administered by subcutaneous ("skin popping") and intravenous ("mainlining") injection.

### ASSESSMENT AND TREATMENT CONSIDERATIONS

- It may be preferable to administer Naloxone to known drug abusers only until an improvement in respirations is seen, not to a fully awake state (patient may become violent).
- If Narcan use results in a previously altered patient suddenly awakening and refusing transport, a Biocare physician should be contacted to consult on the refusal of care.
- **Narcan is not effective in non-opioid drugs such as:**
  - levopropoxyphene, phenergan, paralytics, benzodiazepines, sedative/hypnotics, cocaine, PCP, LSD, amphetamines, GHB, antidepressants, barbiturates, ketamine, ecstasy, marijuana, antihistamines, quaaludes, MAO inhibitors, selective serotonin reuptake inhibitors, beta blocker or calcium channel blockers.

### MEDICATION CONSIDERATIONS

- Drugs which typically respond to Narcan includes:
  - Dextromethorphan (Robitussin, Triaminic, Nyquil)
  - Pentazocine (Talwin)
  - Tramadol (Ultram)
  - Heroin (Smack, horse, black tar etc.)
  - Hydromorphone (Dilaudid)
  - Meperidine (Demerol)
  - Fentanyl (Sublimaze)
  - Codeine (methylmorphine)
  - Nalbuphine (Nubain)
  - Buprenorphine (Subutex)
  - Propoxyphene (Darvocet, Darvon)
  - Clonidine (Catapres)
  - Morphine (MS, MS Contin)
  - Hydrocodone (Vicodin, Lortab, Vicoprofen)
  - Methadone
  - Paregoric (Camphorated tincture of opium)
  - Oxycodone (Percodan, Percocet, Oxycontin)
  - Butorphanol (Stadol)
  - Diphenoxylate/atropine (Lomotil)
  - levacetylmethadol (LAAM)

  These drugs may require larger doses of naloxone to reverse their effects

- Onset of action of IV Naloxone is within 2 minutes, intranasal 1-4 minutes, and slightly less for Sub-Q or IM routes. The duration of action is approximately 20 to 60 minutes.
- Narcan is to be used with extreme caution in patients dependent on narcotics (drug abusers, terminal illness, and newborns of addicted mothers) as abrupt reversal of narcotic effects can cause an acute abstinence syndrome with symptoms ranging from nausea, vomiting, sweating, tachycardia, hypertension, seizure, arrhythmias, pulmonary edema, to rarely, cardiac arrest.
- If Naloxone must be given to a narcotic dependent patient, large IV doses should be avoided (over 0.4 mg). To prevent withdrawals from occurring do the following:
  - Mix 0.4 mg of narcan with 9 mL of NS to yield a 1 mL = 0.04 mg mixture
  - Administer at 1 mL/min until adequate patient response is seen (typically 2 to 4 mLs).

### POTENTIAL PITFALLS

- Failure to consider physical restraints prior to narcotic reversal for patient and provider safety.
- Immediately intubating a suspected narcotic overdose with respiratory depression until Naloxone has been administered and allowed to work. Support ventilations with a BVM while waiting for naloxone to work to prevent unneeded intubation (except in cardiac arrest).
- Failure to protect the eyes of the unresponsive patient from drying out. When a patient is unresponsive and their eyes are allowed to remain partially open, drying of the globe can occur with blindness possible. Consider taping the eyelids closed if they fail to remain closed.
TOXICOLOGICAL - SEDATIVE HYPNOTIC OVERDOSE

Sedative-hypnotics are a class of drugs that cause CNS depression including benzodiazepines, barbiturates, non-benzodiazepine sedative-hypnotics, and alcohol. Almost all of these drugs stimulate GABA (gamma amino-butyric acid) receptors to open cellular chloride channels causing the cell to become hyperpolarized (much more negatively charged), delaying transmission of inhibitory nerve impulses, reducing neuronal activity and lessening muscular activity (why they are useful for seizures).

ASSESSMENT AND TREATMENT CONSIDERATIONS

  - Benzodiazepines are rapidly absorbed and when used alone carry a low risk of toxicity. However if mixed with alcohol they become very dangerous.
  - Prescribed benzodiazepines include; Diazepam (Valium), flurazepam (Dalmane), oxazepam (Serax), clonazepam (Klonopin), temezepam (Restoril), Lorazepam (Ativan), clordiazepoxide (Librium), triazolam (Halcion), and clorazepate (Tranxene).
  - Flunitrazepam or Rohypnol ("roofies," "rophies," "forget-me pill") is a benzodiazepine 10 times more potent than diazepam. Rohypnol causes rapid sedation and amnesia (aka the “date rape drug”).

- **Non-benzodiazepine sedative-hypnotics** (NBSH) act on GABA receptors to promote sleep, but produce far less problems as do benzodiazepines. The NBSH drugs include Zolpidem (Ambien) and zaleplon (Sonata). Toxicity causeS CNS depression, hypotension, respiratory depression, coma, and rarely death.

- **Barbiturates** (barbs, downers, yellow jackets, red devils, reds and blues, rainbows, goof balls) are used for mild sedation, anesthesia, and for some types of epilepsy. Common barbiturates are pentobarbital (Nembutal), secobarbital (Seconal), amobarbital (Amytal) and phenobarbital (Luminal).
  - Intoxication includes CNS depression, decreased reflexes, and slow respirations. Severe intoxication causes coma, loss of all reflexes (except pupillary), bradycardia and hypotension.

- **Gamma-Hydroxybutyrate or GHB** (“grievous bodily harm," “G,” "liquid ecstasy," "Georgia Home Boy") is a derivative of GABA that is popular at rave events. GHB is often added to drinks with an onset of action of within 15 minutes producing relaxation and disinhibition. At high doses, GHB causes bradycardia, agitation, vomiting, amnesia, and apnea (especially if mixed with alcohol).

- **Alcohol (Ethanol, ethyl alcohol, EtOH)** also belongs to the sedative-hypnotic class of drugs. Alcohol allows chloride channels to remain open longer than normal, thus further depressing the cell (reason why they are dangerous when used with other GABA related drugs).

- Management of all sedative-hypnotic related overdoses (including alcohol) focuses on airway control, oxygenation, continuous ECG, BP, and pulse oximetry monitoring, and vascular access.

MEDICATION CONSIDERATIONS

- Narcan is not effective in non-opioid drugs such as benzodiazepines, GHB, non-benzodiazepine sedative-hypnotics or barbiturates.

- Activated charcoal binds to sedative-hypnotics and should be used. However, many patients may vomit during or after administration; in this case, charcoal may be re-administered once if needed.

- Treat hypotension with IV fluids; vasopressors are rarely necessary in these patients.

POTENTIAL PITFALLS

- Failure to remember that an intentional overdose can pose a significant risk to the rescuer and law enforcement scene should be requested for assistance.

- Use of activated charcoal in patients with a compromised airway or in patients who have ingested a sedative-hypnotic agent more than 1 hour before EMS arrival.
Management of the poisoned patient focuses on decontamination which limits further absorption and minimizes toxicity; supportive care which limits the effects on the body systems at risk; and definitive care which limits the severity or duration of toxicity through the use of antidotes or enhanced elimination.

**ASSESSMENT AND TREATMENT CONSIDERATIONS**

- **Organophosphates (OP)** are chemicals used as pesticides (Malathion, parathion, diazinon, fenthion, etc.), and nerve agents (soman, sarin, tabun, VX). They may be absorbed, inhaled, or ingested.

- OP’s work by disrupting transfer of nerve impulses by blocking acetylcholinesterase (AChE), an enzyme that breaks down or removes acetylcholine (ACh) in both the central and peripheral nervous systems.

- ACh and its actions are controlled by muscarinic and nicotinic receptors. Blocking AChE causes prolonged stimulation by these receptors with severe side-effects (see chart).

- Hydrogen cyanide (CN) is a colorless gas with a faint, almond-like odor (most cannot smell it). Cyanide binds to iron in the mitochondria, preventing the cells from using oxygen (patient suffocates).

- CN poisoning is uncommon, and if seen is associated with intentional OD, accidental exposure (electroplating, bronze sculpture shops), or prolonged enclosed space fires from incomplete combustion of cotton, rayon, wool, PVC, acrylic, polyurethane, polyester, neoprene, asphalt, nylon, rubber, plastics, styrofoam, or insulation.

**MEDICATION CONSIDERATIONS**

- Atropine use in OP poisoning blocks muscarinic receptors, but does not bind to nicotinic receptors and, thus is ineffective in treating neuromuscular dysfunction. The doses required in OP poisoning (often hundreds of milligrams) will produce tachycardia and dilated pupils, two early signs of atropinization, but they are not indications to stop atropine administration.

- The end-point of atropinization is drying of respiratory secretions. Continued use of atropine prevents patients from literally drowning in their own secretions, the primary cause of early death.

- Improve oxygenation as much as possible before administering atropine, so as to minimize the risk of ventricular fibrillation from its effects on a hypoxic myocardium.

- Nitrates are used to induce methemoglobinemia (the bond between cyanide and cytochrome oxidase is weaker than that between cyanide and methemoglobin which leads to the transfer of cyanide from the mitochondria back to the circulation). Sodium nitrite and sodium thiosulfate are also given to help form thiocyanate which releases hemoglobin; the thiocyanate is then excreted by the kidneys.

- Succinylcholine is metabolized by means of plasma cholinesterase and in OP poisoning its use can cause prolonged paralysis.

**POTENTIAL PITFALLS**

- Contaminated patients should not be transported by air-medical resources given potential risk to air crew, aircraft safety, and aircraft decontamination.

- Unnecessary treatment using a cyanide antidote kit is dangerous as methemoglobinemia does not carry oxygen and in the presence of carboxyhemoglobin (CO from fires) can lead to cellular anoxia.
The general assessment and management of a traumatically injured adult and child are essentially the same. ABCs must be evaluated and managed first followed by a neurological examination and complete exposure of the patient. One of the most important responsibilities is to spend as little time on the scene as possible; the goal should be to not be on-scene no longer than 10 minutes in major trauma if possible.

**ASSESSMENT AND TREATMENT CONSIDERATIONS**

- Blood pressure guidelines for fluid administration in traumatically injured patients:
  - **Non compressible sites:** internal organs and/or vessels of the pelvic, abdominal or chest cavities. (maintain a SBP of no more than 90 mmHg).
  - **Compressible sites:** sites in which blood flow may be directly occluded by pressure (lacerations) (maintain a SBP of no more than 90 mmHg).

- Traumatically injured patients lose heat quickly; keep the patient warm unless already hyperthermic.

- In penetrating injuries, obtain total number of entry and/or exit wounds and their locations. **Do not forget to check the back, groin and/or axillae for entry or exit wounds in truncal wounds.**

- **Signs of a tension pneumothorax include:** hypoperfusion plus one or more of the following: absent breath sounds, tachycardia, increased air hunger, increasing difficulty with BVM ventilation, tracheal deviation (a late sign), or subcutaneous emphysema. All pleural decompressions (needle thoracostomy) are to be well documented including a physical presentation, signs/symptoms, evidence of hemodynamic or airway compromise, and time and site of the procedure.

- **Pericardial tamponade:** rapid administration of intravenous fluids to help raise the venous pressure and improve cardiac output transiently until pericardiocentesis can be performed.

- **Open chest wounds:** cover with vaseline gauze and taped loosely on 3 sides. If tension pneumothorax develops, remove dressing, allow air to escape, and re-apply dressing.

- **Abdominal evisceration:** cover with saline soaked dressing. Do not replace abdominal contents.

- **Impaled objects:** remove only when its presence interferes with CPR or impaled object interferes with the airway, otherwise use bulky dressing stabilization to hold object in place.

**MEDICATION CONSIDERATIONS**

- IV fluids may be repeated as needed until perfusion improves, SBP > 90 mm Hg, or the maximal amount advocated by the protocol is reached (typically 2 liters in adults and 1 liter in pediatrics).

- To prevent fluid overload and pulmonary edema always follow the guidelines as outlined under page **MED-11 Medically Related Hypotension** for bolus amount and restrictions.

**POTENTIAL PITFALLS**

- Failure to minimize fluid administration in non-compressible bleeding sites as BP elevation could dramatically increase the rate of hemorrhage and affect blood clotting.

- Reluctance to provide analgesia in patients with significant dislocations and/or fractures.

- Performing excessive “pelvic rocks” to assess for mechanical instability of the pelvis. Opening and closing (rocking) the pelvis can destabilize clots and provoke a fatal hemorrhage.

- Administration of cold IV fluids to a traumatically injured patient. When possible IV fluids should be warmed before administration (IV warmer) or placed next to a heat pack to maintain warmth.

- Remember that anyone injured who is cool and tachycardic is in shock until proven otherwise.

- Placement of an intraosseous line in any patient without life-threatening injuries.
The partial or complete severance of a digit or limb is an amputation. It often results in the complete loss of the limb at the site of severance. Traumatic amputation most often affects limbs and appendages like the arms, ears, feet, fingers, hands, legs, and nose. Surgeons may re-implant the amputated part or use the skin for grafting, as they repair the remaining limb.

**ASSESSMENT AND TREATMENT CONSIDERATIONS**

- Candidates for re-implantation include victims of amputation of the scalp, hand, digit, penis, and selected portions of distal-most extremities. In general, the younger the patient is, the more potential lifetime benefit reimplantation has to offer.
- Cooling potentially increases the prospect of successful reimplantation, because it decreases the metabolic rate and inhibits bacterial growth. Immediate institution of hypothermia (cooling with ice) can extend the ischemic period to 24 hours or more in some patients.
- Partial amputations should be dressed and splinted in alignment with extremity to ensure optimum blood flow.
- If an in field amputation team is required contact CareFlite who will dispatch a helicopter with two trauma surgeons to the scene for assistance.
- Control all bleeding by direct pressure only to preserve tissues. The most profuse bleeding may occur in partial amputations, where cut vessel ends cannot retract to stop bleeding. Avoid tourniquet placement whenever possible. Never clamp bleeding vessels.

**MEDICATION CONSIDERATIONS**

- Amputations can be and often are very painful. Unless contraindicated consider Fentanyl for relief of pain.

**POTENTIAL PITFALLS**

- Delaying transport of patient for prolonged extrication of detached body part(s) or tissue. Have the body part(s) or tissue transported separately, if needed.
- Failure to prevent torsion (twisting) during handling and splinting of the affected limb or body part.
- Using dry ice to preserve severed parts can rapidly freeze and destroy viable tissue.
- Falsely elevating the patient’s and/or family’s hope for successful reimplantation. Many factors enter into the decision to attempt reimplantation (age, location, condition of tissues, other options).
- A decision regarding reimplantation cannot be made until the patient and part have been examined by a qualified physician.
- Failure to examine the patient for associated injuries of more importance.
- Placement of an intraosseous line in any patient without life-threatening injuries.
Serious burns are complex injuries which affect muscles, bones, nerves, and blood vessels. Respiratory system damage, with airway obstruction, failure or arrest can occur. Burns injuring skin impair normal fluid and electrolyte balance, temperature regulation, joint function, dexterity, and physical appearance.

**ASSESSMENT AND TREATMENT CONSIDERATIONS**

- **Chemical** burns are a hazard to both patient and rescuer, and care must be taken to avoid exposure. In cases involving powdered or dry chemicals, it may not be appropriate to flush with water (confirm first). With dry chemicals, carefully brush off the skin and flush with copious amounts of water.
- The severity of acid or alkali burns is related to a number of factors, including the pH and concentration of the agent, length of contact time, volume of agent, and physical form of the agent.
- **Thermal** burn care should be guided by scene safety, cooling the burn (if appropriate), maintaining body temperature, and protecting the airway. During airway assessment, give careful attention to signs of inhalation injury. Carbonaceous sputum, singed facial hairs, facial burns, oropharyngeal edema, changes in the voice, or altered mental status suggest the possibility of inhalation injury.
- **Major Burns** include: Any burn with inhalation injuries, burns associated with other injuries, second or third degree burns between 10% and 20% BSA or involving the face, hands, feet, genitalia or perineum. Second or third degree burns > 20% BSA involvement, should be transported to the Trauma Center.
- **Rule of Nines:** The rule of nines is a formula used for calculating the percentage of body surface area burned.
  - **Adults:** the head and arms equal 9% respectively, the front torso 18%, the back torso 18%, each leg is 18% and the groin equals 1% of the total body surface area (TBSA).
  - **Children < one year:** the head is 18%, each arm is 9%, each leg is 14% and the front and back of the torso is 18% percent each, respectively.
  - **Children > one-year:** subtract 1% from the head, add 1/2 % to each leg for each year past age one.

**MEDICATION CONSIDERATIONS**

- The maximum adult dose of Morphine is 20 mg per hour in the non-intubated patient and 50 mg per hour in the intubated patient.

**POTENTIAL PITFALLS**

- Failure to remember that shock in the very early stages of a burn is generally not associated with the burn, thus other life-threatening injuries should be looked for.
- Failure to appreciate the seriousness of an airway burn. A particularly dangerous injury is the 3rd degree facial burn where minimal external edema is present. The lack of external edema is due to the non-elastic 3rd degree burn, which does not allow expansion.
- Prolonged irrigation with cool fluids or leaving the patient in wet sheets will not improve the burn and greatly increases the risk of hypothermia (burn patients are susceptible to heat loss).
- Failure to consider and/or assess for CO monoxide exposure in thermal burns.
- Field decontamination of chemical exposures has been shown to significantly reduce extent of burn. It is rare to encounter a chemical which is not properly decontaminated by copious water.
- Using too small tracheal tube (especially a nasotracheal tube) in airway burns or smoke inhalation. An ET tube diameter of less than 7.0 (in an adult) is too small for adequate suctioning of secretions that can compromise ventilation. If intubation is required, use the largest tube possible.
- Failure to maintain and/or prevent heat loss in large burns.
- Failure to provide pain management when indicated.
- Placement of an intraosseous line in any patient without life-threatening injuries.
True posttraumatic “cardiac arrest,” usually means asystole, and posttraumatic “circulatory arrest,” means that, despite apnea/agonal breaths and unobtainable pulses, a rhythm and heart beat may exist. If posttraumatic cardiac arrest occurs in the prehospital setting the mortality rate approaches approximately 99%. Traumatic arrest patients who are potentially viable should be rapidly transported as soon as possible since immediate surgical intervention can make the difference between life and death.

**ASSESSMENT AND TREATMENT CONSIDERATIONS**

- When a posttraumatic cardiac arrest patient is considered potentially viable advanced interventions beyond airway control and initial defibrillation are to be initiated only after transport has begun.
- BLS and ALS for the trauma patient are fundamentally the same as the care of the patient with a primary cardiac or respiratory arrest. In trauma resuscitation, a rapid assessment is performed, with rapid evaluation and stabilization of airway, breathing, and circulation.
- If an AED or manual cardiac monitor is readily available, it should be applied when absence of pulses is confirmed. If a shockable rhythm is present immediate shock up to 3 times, is indicated on scene.
- Defibrillation is generally not effective until circulating volume has been restored in the hypovolemic trauma arrest patient. Unless transport is delayed, continue further countershocks during transport.
- Detailed secondary surveys beyond assessing for life-threatening injuries are only to be attempted once care for life-threatening injuries has been delivered and patient is enroute to a trauma facility.

**MEDICATION CONSIDERATIONS**

- Vasopressors (Dopamine) should only be administered after adequate fluid volumes have been administered. Never administer dopamine to a volume depleted patient.

**POTENTIAL PITFALLS**

- Delaying transport or remaining on scene to continue prolonged resuscitative efforts in the potentially viable traumatic arrest patient. Try to keep scene times to less than 10 minutes when possible.
- Failure to maintain an open airway and adequately ventilate the patient (when able to do so).
- Failure to assume spinal cord injury in an unresponsive patient with severe mechanism.
- Failure to recognize and/or treat life-threatening conditions as they are identified (i.e. tension pneumothorax, profound external hemorrhage etc).
- Performing nasotracheal intubation in the presence of severe maxillofacial injuries, as the tube may migrate outside the trachea during placement and into the cranial vault (rare).
- Failure to provide early notification during transport has been initiated that a traumatic arrest is present.
Assessment and management priorities for resuscitation of the injured pregnant patient are essentially the same as for other traumatized patients; however, specific anatomic and physiologic changes that occur during pregnancy may alter the physiologic response to trauma requiring a modified approach to the resuscitation process.

### ASSESSMENT AND TREATMENT CONSIDERATIONS

- The effect of trauma on pregnancy depends on the gestational age of the fetus, the type and severity of the trauma, and the extent of disruption of normal uterine and fetal physiology.
- The survival of the fetus depends on adequate uterine perfusion and delivery of oxygen. Once obvious shock develops in the mother, the chances of saving the fetus are about 20%.
- Cardiac output and blood volume increase in the first trimester to about 40% above the nonpregnant state at 28 weeks gestation. Because of this extra blood volume almost 40% of maternal blood volume may be lost prior to any signs or symptoms of maternal shock appearing.
- BLS and ALS for the pregnant trauma patient is fundamentally the same as the care of the patient with a primary cardiac or respiratory arrest. In trauma resuscitation, a rapid assessment is performed, with rapid evaluation and stabilization of airway, breathing, and circulation.
- Defibrillation is generally not effective until circulating volume has been restored in the hypovolemic trauma arrest patient. Unless transport is delayed, continue further countershocks during transport.
- After 20 weeks of gestation, the uterus may compress the great vessels when a pregnant woman is supine. Compression can decrease the SBP up to 30 mm Hg along with a 30% decrease in stroke volume leading to a decrease in uterine blood flow. During CPR great vessel compression must be relieved from the pregnant uterus to allow for adequate blood flow during chest compressions.

#### Changes in Normal Pregnancy that May Affect Trauma Management

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Effect</th>
</tr>
</thead>
<tbody>
<tr>
<td>Systolic blood pressure</td>
<td>Decreased by an average of 5 to 15 mm Hg</td>
</tr>
<tr>
<td>Diastolic blood pressure</td>
<td>Decreased by 5 to 15 mm Hg</td>
</tr>
<tr>
<td>ECG</td>
<td>Flat or inverted T waves in leads III, V1, and V2; Q waves in leads III and aVF</td>
</tr>
<tr>
<td>Blood volume</td>
<td>Increased by 30 to 50 percent</td>
</tr>
<tr>
<td>Respiratory rate</td>
<td>Increased by 40 to 50 percent</td>
</tr>
<tr>
<td>Oxygen consumption</td>
<td>Increased by 15 to 20 percent at rest</td>
</tr>
<tr>
<td>Gastrointestinal tract</td>
<td>Decreased gastric emptying, decreased motility, increased risk of aspiration</td>
</tr>
<tr>
<td>Diaphragm</td>
<td>Higher position in pregnancy</td>
</tr>
<tr>
<td>Musculoskeletal system</td>
<td>Widened symphysis pubis and sacroiliac joints; hormones cause more loose joints which can lead to increased musculoskeletal injury</td>
</tr>
</tbody>
</table>

### MEDICATION CONSIDERATIONS

- Oxygen is essential to prevent maternal and fetal hypoxia as severe trauma stimulates maternal catecholamine release, leading to uteroplacental vasoconstriction and compromised fetal circulation.
- Vasopressors (Dopamine) should be used with caution and only after adequate fluid volumes have been administered. Never administer dopamine to a volume depleted patient.

### POTENTIAL PITFALLS

- Failure to recognize and/or treat life-threatening conditions as they are identified (i.e. tension pneumothorax, profound external hemorrhage etc).
- Failure to provide early notification during transport has been initiated that a traumatic pregnancy related arrest is present.
TRAUMA - CRUSH INJURIES

Victims entrapped and crushed due to heavy, fallen debris from a structural collapse present a unique challenge. These crushing objects place prolonged and continuous pressure on the extremities, resulting in skeletal muscle death (rhabdomyolysis) with release of myoglobin into the plasma and these effects are termed Acute Crush Syndrome. After the skeletal muscle injury occurs and the crushing object is removed, myoglobin and potassium are released into circulation, leading to arrhythmias, acute renal failure, and sudden death.

ASSESSMENT AND TREATMENT CONSIDERATIONS

- Large volumes of IV normal saline are often administered before and after the patient is released to prevent renal failure at a later stage.
- The systemic effects of acute crush syndrome only occur when the crushing object is removed and the injured extremity is reperfused. Removal of the object also causes a massive fluid shifts into the injured muscle, resulting in acute hypovolemia and hypotension.
- **Trauma and compression**: Trauma is the most common cause of myoglobinuria (excessive myoglobin in the urine which can lead to renal failure). Patients who experience crush injuries after building or trench collapse often will have muscle breakdown and myoglobinuria. Volume depletion can also occur from fluid moving and settling into damaged tissue which can accentuate the possibility of acute renal insufficiency.
- Although crush syndrome most often occurs following trauma, it can also be associated with severe alcohol intoxication and/or drug overdose (i.e., unresponsive patient positioned so that his/her extremity is compressed by their body weight for an extended period of time). Because drugs and alcohol blunt protective pain mechanisms, the patient fails to relieve pressure a crush injury occurs.
- **Traumatic asphyxia**: The clinical syndrome includes subconjunctival and subcutaneous hemorrhages, bluish discoloration of the face and neck, and pronounced facial edema. Other physical characteristics include multiple small petechiae extending over the face, neck, and the upper chest, but not below the nipple line.

MEDICATION CONSIDERATIONS

POTENTIAL PITFALLS

- Failure to provide pain management when indicated.
- Be observant for potential hyperkalemia secondary to potassium release from crushed tissue.
- Placement of an intraosseous line in any patient without life-threatening injuries.
The most common classification of electrical injuries is **lightning, high-voltage and low-voltage AC and DC**. Electrical injuries occur when a person, accidentally or intentionally, becomes part of an electrical circuit, is affected by the thermal effects of a nearby electrical arc, or is struck by lightning. The initial assessment of an electrical burn should be guided by provider safety.

### ASSESSMENT AND TREATMENT CONSIDERATIONS

- The heart is most susceptible to voltage below 400 volts. Above this level internal burns are a major complication. **Remember that most injuries in electrical burns are internal.**
- Although thermal burns can and do occur, **electrical injuries should be treated similar to crush injuries** because of the large amount of muscle and tissue under uninjured skin.
- Alternating current (AC) is about three times more dangerous than direct current (DC) of the same voltage; because continuous muscle contraction occurs when muscle fibers are stimulated at between 40-110 times per second (the frequency used in the United States is 60 Hz).
- Electrical shock can be classified as **high-voltage (> 600 volts)** or **low-voltage (< 600 volts)**. High voltage is associated with more fatalities, but fatal injury can occur at household currents (110 volts).
- The **three major mechanisms of electricity-induced injury are**:
  - electrical energy causing direct tissue damage, altering cell membrane potential, muscle tetany
  - the conversion of electrical energy into thermal energy, causing massive tissue destruction
  - mechanical injury with direct trauma resulting from falls or violent muscle contraction
- Bone is the tissue most resistant to the flow of electricity. Nerve tissue is the least resistant. Skin resistance is the most important factor impeding current flow. Skin resistance depends on its thickness and is reduced substantially by moisture, converting a low-voltage injury into a life-threatening one.
- Current passing through the brain, in both low-voltage and high-voltage shocks, produces immediate unconsciousness and directly due to instantaneous depolarization of brain neurons.
- **Lightning** is a DC current that causes an instantaneous and extremely high-voltage discharge of electricity to the body which often causes prolonged respiratory arrest. Prompt, continuous respiratory assistance (sometimes for hours to days) can result in full recovery.
- High-voltage current often flows internally, creating massive muscle damage. Do not attempt to predict the amount of underlying tissue damage from the skin involvement or use the rule of nines for calculating fluid resuscitation.

### MEDICATION CONSIDERATIONS

- The maximum adult dose of Morphine is 20 mg per hour in the non-intubated patient and 50 mg per hour in the intubated patient.

### POTENTIAL PITFALLS

- Failure to remember that electricity takes the path of least resistance and current passing through the heart or thorax can cause cardiac arrhythmias and direct myocardial damage, whereas current passing through the brain can result in respiratory arrest.
- Failure to remember that electrical injuries of any form during second and third trimester pregnancies should be transported for physician follow-up, regardless of injury.
- Failing to continuously monitor or offer transport to any patient who experiences a significant electrical shock (LOC, disorientation, neurological deficits, abnormal vital signs, or direct passage of current over the myocardium) as fatal arrhythmias (VT/VF) often present as an early problem and can occur at any time during the course of patient care.
- Failure to remember that the major cause of death in lightning injuries is cardiac arrest secondary to massive DC defibrillation often causing asystole or VF from massive vagal discharge.
- Failure to remember that the establishment of an airway may be difficult in patients with electric burns of the face, mouth, or anterior neck as extensive soft-tissue swelling may develop rapidly and complicate airway control measures, such as endotracheal intubation. For these reasons, intubation should be seriously considered before signs of airway obstruction become evident or severe.
- Failure to search for additional traumatic injuries in electrical burns.
- Failure to provide pain management when indicated.
- Placement of an intraosseous line in any patient without life-threatening injuries.
An estimated half-million serious eye injuries occur each year in the United States. Twenty-five thousand of those numbers end up in total blindness. The structure of the face is well suited for protecting the eyes from injury. Even so, the eye and its surrounding structures can be damaged by direct injury, sometimes so severely that vision is lost. Any injuries involving the eyes should be seen by a physician to determine if further care is needed.

ASSESSMENT AND TREATMENT CONSIDERATIONS

- **Minor eye injuries:** injuries where the eye has been struck by a foreign object, or has a small object adhering to its surface, causing irritation and an urge to rub the eye.

- **Major eye injuries:** injuries that involve penetration of the globe or involve severe blunt trauma to the eye. Often characterized by blood in the eye, penetrating objects, disturbance of vision, protrusion of eye contents, and severe pain and spasms.

- **Corneal flash burns (‘Welder’s Arc Burn):** the result of staring or inadvertently looking at intense UV light produced during metal welding or the sun, without adequate eye protection. Corneal flash burns cause a superficial injury in response to ultraviolet light. The pain may not be immediate and often can take more than 12 hours to develop (reason why many of these patients have symptom onset in the middle of the night). The damage caused to the cornea can be extremely painful. These patients must be seen by a physician for proper diagnosis and care.

- **Chemical exposures and burns** are often the result of a splash of liquid getting in the eye. Direct lavage should be done on scene for at least 20 minutes (at least 2 liters of fluid) using water, NS or LR (flush under lids and conjunctival areas to remove particulate matter). Acids and alkalis are highly caustic and can cause severe damage.

- **Alkali burns** are the most dangerous as they tend to penetrate the surface of the eye and can cause severe injury. *In general, the higher the pH, the more damage will occur.* Common alkali substances contain the hydroxides of ammonia, potassium, sodium, calcium, and magnesium.

- **Acid burns** tend to be less severe than alkali burns. *The exception is a hydrofluoric acid burn, which is as dangerous as an alkali.* Acids usually damage the very front of the eye. Common acids include sulfuric acid, sulfurous acid, hydrochloric acid, nitric acid, acetic acid, chromic acid, and hydrofluoric acid. Car batteries cause a sulfuric acid burn, the common acidic burn of the eye.

- **Orbital wall fracture** should be considered after any blunt eye trauma. Signs and symptoms may include double vision (diplopia), epistaxis, ecchymosis, crepitus, and restricted upward gaze secondary to inferior rectus muscle entrapment.

- **Hyphema** is the presence of blood in the anterior chamber and is often easily visualized. Symptoms include pain, photophobia, and blurring of vision. Transport patient for physician follow-up.

- **Riot control agents** are used to incapacitate, not to kill or injure and are also referred to as lacrimators and incapacitating agents with the primary agents used being chlorooacetophenone (CS or “tear gas”), orthochlorobenzalmalononitrile (CN or “mace”), and deoresin capsicum (OC or “pepper spray”). Clinical effects of these agents include conjunctivitis, contact dermatitis, blisters and tightness in the chest. Treatment is usually prolonged eye irrigation as well as thorough irrigation of the skin with water or commercially made pads to remove the agents in question (do not use pads in eyes).

MEDICATION CONSIDERATIONS

- Antiemetics administered for nausea and vomiting can help reduce the likelihood of expulsion of eye contents during periods of elevated intraocular pressure during active vomiting.

POTENTIAL PITFALLS

- Allowing a patient to rub his/her eye(s) after an eye injury could result in worsening of the injury.

- Delaying transport to complete eye irrigation. Continue irrigation during transport.

- Failure to provide pain management when indicated.

- Placement of an intraosseous line in any patient without life-threatening injuries.
Hydrofluoric acid (HFA) is a highly corrosive acid that can severely burn skin and eyes. Unlike other acids, which are rapidly neutralized, HFA continues to destroy the skin for days. In addition, the fluoride ions readily bind with calcium ions causing hypocalcemia. Hypocalcemia can lead to cardiopulmonary arrest. If the exposure is left untreated or improperly treated, permanent damage, disability, or death may result. Treatment is directed toward binding fluoride ions to limit tissue destruction.

**ASSESSMENT AND TREATMENT CONSIDERATIONS**

- Hydrofluoric acid is a strong mineral acid used in electroplating, metal processing and semi-conductor industries, as well as for glass etching. HFA differs from other acids because the fluoride ions readily penetrate the skin causing destruction of the deep tissue layers and even bone. Burns initially depend upon concentrations, temperature, and duration of contact.

- A specific property of fluoride is its capacity of rapid skin penetration. The toxic effect is due to the removal of available body calcium by the fluoride. This leads to a dramatic drop of the calcium level.

- Profound destruction of tissue typically occurs at the site of penetration. **A strong pain after several hours is often the first sign of severe tissue damage.**

- First aid measures are directed at decontamination by copious flushing of the skin with water. Clothes must be removed with caution (danger for rescuers).

- Life-threatening hypocalcemia manifests as CNS irritability and poor muscular contractility and produces a wide range of peripheral and CNS effects including paresthesias, tetany (i.e., contraction of hands, arms, feet, larynx, bronchioles), and seizures. ECG recognition includes prolonged QT, prolonged ST segment, and T wave abnormalities.

- Hypocalcemia is considered a risk in all inhalation, ingestions or any skin burns > 25 square inches.

**MEDICATION CONSIDERATIONS**

**POTENTIAL PITFALLS**

- Failing to administer pain medication. Pain relief should be administered when indicated as HFA exposure can be very painful.

- Placement of an intraosseous line in any patient without life-threatening injuries.
Sexual assault according to accepted definition of the Texas Penal Code is sexual assault as forcible or non-forcible sexual activity that occurs when one person threatens, or uses violence or coercion to cause another person to participate in any type of oral, anal, vaginal, or other sexual act. Injuries commonly encountered during a sexual assault are usually facial or extremity.

**ASSESSMENT AND TREATMENT CONSIDERATIONS**

- In cases of severe emotional upset, it may be better to have a same sex provider care for a rape victim's injuries (if possible).
- Gynecological injuries only account for a low percentage of all injuries.
- Elderly victims are twice as likely to incur physical not genitalia injuries.
- Advise patients not to urinate, defecate, douche, or wash before an emergency department Sexual Assault Nurse Examiner (SANE) or Physician has had a chance to do an examination.
- As soon as possible document in writing the condition of the patient when first seen. Include a description of the scene, patient's clothing, any unsolicited statements made by patient, patient's mental and physical condition, and a complete summary of patient care.
- When taking a patient history, obtain only pertinent facts related to the trauma (LOC, SOB, bleeding, weapon involved). Do not question patient about past sexual history, or attempt to obtain non-medical facts (assailant description, etc) not directly related to patient care.
- Common misconceptions about rape and sexual assault:
  - Approximately every 6 minutes another person is raped in the United States
  - Over 30% of all rape victims will have rape-related post traumatic stress disorder
  - As many as 80% of rape victims will know their attacker
  - As many as 14% of married women have been victims of rape by their husbands
  - Men and children are also victims of rape
  - Most people know someone who has been raped; you may just not know who it is.

**MEDICATION CONSIDERATIONS**

**POTENTIAL PITFALLS**

- Failure to save any clothing removed during your care. Handle the clothing as little as possible.
- Failure to place items in a paper bag when possible to avoid contamination and induction of moisture into clothing by placement into a plastic bag.
- Failure to report suspected sexual abuse can result in criminal liability, although the liability is typically a misdemeanor punishable by a fine. Failure to report can result in civil liability.
- Placement of an intraosseous line in any patient without life-threatening injuries.
Two families of venomous snakes are native to the United States. The vast majority are pit vipers, of the family Crotalidae, which include rattlesnakes, copperheads and cottonmouths. The other family of poisonous snakes is the Elapidae, which includes two species of coral snakes found chiefly in the Southern states. Related to the much more dangerous Asian cobras and kraits, coral snakes have small mouths and short teeth, giving them a less efficient venom delivery than pit vipers.

ASSESSMENT AND TREATMENT CONSIDERATIONS

- People bitten by coral snakes lack the characteristic fang marks of pit vipers, sometimes making the bite hard to detect. Hemotoxic poisons, found primarily in the Crotalidae, attacks the blood system causing lysis of capillary cells, local thrombosis, gangrene, and intravascular clotting. This is due to the presence of thrombase, hemorrhagin, and anticoagulin in the venom.

- **Pit Vipers:** identified by their angular shaped heads. Symptoms are acute and marked with tissue at the bite mark swelling within 3 minutes and continuing for 1 hour or more.

- **Coral Snakes:** identified by their unique coloring remember; “red touch black, venom lack, red touch yellow, kill a fellow.” Symptom onset is not as acute as with pit vipers. Arrhythmias, hypotension, weakness and shock can occur. In addition, severe headache, dizziness, hearing difficulty, confusion, unconsciousness, respiratory difficulty, paresthesia, diaphoresis, chills and fever can occur.

- Elapidae venom is neurotoxic and acts by attacking the respiratory center and the 9th -12th cranial nerves. Rapid hypotension, shock, pulmonary edema, renal, and cardiac dysfunction can result, especially with direct intravenous envenomation which and has a much higher mortality rate.

- **Bites on the head and neck are all assumed to be severe.** Early intubation should be considered for these patients as respiratory compromise is frequent.

- Inspect the area of the bite, looking for fang marks. (Note: The bite of the coral snake and some exotics may leave little or no evidence of fang marks). Special notice should be made of the size of the erythema (redness) surrounding the fang marks, if present, and the extent of associated edema.

- A patient is much more likely to get a large dose of venom from a young poisonous snake than from a mature snake. Immature snakes tend to envenomate everything at once while a mature snake will keep venom in reserve – just in case.

- Document amount of pain at the bite site and time of onset of pain, compared with time of bite (especially important). Usually with envenomation, onset of pain is immediate.

- Exotic snake envenomation (cobras, puff adders etc.) may be seen in persons who keep or sell exotic pets. Care is supportive along with immediate on-line consultation for early notification and guidance.

MEDICATION CONSIDERATIONS

- Vasopressors (dopamine) should only be administered after adequate fluid volumes have been administered. Do not administer dopamine to volume depleted patients.

POTENTIAL PITFALLS

- Delaying ED care to determine if envenomation has occurred. Transport is not to be delayed.

- Tourniquets, incision and suction, and electric shock are contraindicated.

- Constricting bands or tourniquets may be harmful in viper envenomation and are not to be used.

- Use of cryotherapy may cause localized tissue destruction and is contraindicated in all snakebites.

- Placement of an intraosseous line in any patient without life-threatening injuries or in the same extremity as the snakebite.
Spinal trauma, if properly managed in the field, can result in significant life-long injury and impairment. Any patient who has sustained an injury indicative of spinal loading or stretching, significant injury above the clavicles, significant blunt trauma to the torso, a head injury resulting in an altered level of consciousness, or a major fall must be suspected of suffering a potential spinal cord injury (SCI) and should be immobilized in a neutral in-line position (unless contraindicated).

**ASSESSMENT AND TREATMENT CONSIDERATIONS**

- The ability to walk is not to be used to determine if a patient needs to be treated for potential spinal injury.
- Carefully differentiate hypovolemic shock (increased HR, low BP, cool/clammy skin) from neurogenic shock (low HR, low BP, warm/dry skin) and treat accordingly.
- Spinal neurogenic shock is a result of vasomotor instability due to the loss of sympathetic tone. The patient typically has a SBP of 80-100. Despite hypotension, the skin is warm, pink, and dry, with adequate urine output and despite the hypotension, there is also a paradoxical bradycardia.
- Immediately after SCI an acute rise in blood pressure may be seen. This has been shown in studies to be caused by release of norepinephrine from the adrenal glands and by pressor response from mechanical disruption of vasoactive nerves and tracts in the cervical and upper thoracic spinal cord.
- The brief rise in BP is then followed by decreased activity of the sympathetic system causing vasodilatation (warm flushed skin), lack of vasoconstriction (hypotension), and absent sympathetic input to the heart (bradycardia).
- In some circumstances, it may be necessary to move the patient to another location prior to full spinal restriction. Rapid moving (“beaming”) utilizes enough providers who place their hands under the patient to move them without changing alignment. This is done in circumstances where the patient needs to rapidly be removed to a safer location (e.g., on an ant mound, in a stream, etc.).
- In the presence of autonomic disruption from cervical or high thoracic cord injury, intubation may cause severe bradarrhythmias from excessive unopposed vagal stimulation. Simple oral suctioning can also cause significant bradycardia. Pre-oxygenation with 100% oxygen may be preventive.

**MEDICATION CONSIDERATIONS**

- Avoid large volumes of IV fluids in spinal cord injured patients unless absolutely necessary.
- Previously healthy patients with symptoms of sinus bradycardia (rate below 60 bpm) should only be given atropine when hypotension is present, and it is rarely necessary to use a pacemaker.

**POTENTIAL PITFALLS**

- Failure to consider head injury in cases of spinal trauma.
- Beware of labeling hypotension as neurogenic shock in patients with spinal cord trauma. A denervated (from the cord injury) abdomen is not assessable clinically and must be evaluated for hemorrhage by other means (bruising, rigidity etc.).
- Intravenous fluid therapy must be given with caution in order not to overload the patient. In quadriplegic patients, pulmonary edema is not uncommon. Patients with low blood pressure from SCI (in absence of other injuries) do not need aggressive fluid resuscitation.
- Administration of a vasopressor until hypovolemia from other trauma has been ruled out.
- Failure to monitor patients with cervical spinal cord injuries as bradycardia and tachyarrhythmias are common (hypoxia and vagal stimulation can cause bradycardia, leading to asystole).
- Placement of an intraosseous line in any patient without life-threatening injuries.
TRAUMA – TOOTH TRAUMA

Dental trauma occurs when the tooth receives a blow. Teeth can be broken, loosened or knocked out completely. Advances in dental care have greatly increased the odds that injured teeth can be replaced or repaired, but action must be taken quickly in these emergency situations.

ASSESSMENT AND TREATMENT CONSIDERATIONS

- Between six and ten months infants begin to get their "baby" (primary) teeth with most baby teeth present by age three years. Around six years age baby teeth begin being replaced by permanent teeth with most people having all their permanent teeth by age 21.
- Primary teeth are not typically replaced as early loss of these teeth does not hinder development of permanent teeth. If replaced, primary teeth could potentially fuse to underlying bone, resulting in facial deformities.
- Trauma to the oral structures can range from fractures of the crown, root or both to injury affecting the supporting structures, surrounding tissues or complete dislodgment of the tooth. Tooth trauma may be accompanied by injury of the tissue surrounding the teeth including the gums, bone supporting the teeth and the jaw bones.
- The greatest incidence of trauma to primary teeth occurs at 2 to 3 years of age, when the child’s motor coordination is developing. The most common injuries to permanent teeth occur secondary to falls, followed by traffic accidents, violence, and sports.
- A tooth avulsion (commonly referred to as knocked out teeth) is usually caused by a direct force sufficient to overcome the bond between the affected tooth and the periodontal ligament within the socket. Avulsion results in hypoxia and eventual necrosis of the pulp unless rapidly reimplanted to preserve the periodontal ligament. Avulsed permanent teeth are considered a dental emergency. If the tooth is reimplanted within 5 minutes there is an 85-97% healing rate; if reimplantation occurs after 60 minutes, the tooth will rarely heal or survive.
- A tooth fracture can be classified as a root fracture, broken tooth (crown fracture), or chipped tooth.
- An impacted or erupting third molar ("wisdom" teeth) can cause intense pain and inflammation of adjacent soft tissue that can progress to a serious infection if untreated. As wisdom teeth erupt they will push against other teeth, forcing them to crowd together.

MEDICATION CONSIDERATIONS

- Tooth injuries can and often are very painful. Unless contraindicated consider an analgesic such as Fentanyl for pain relief when needed.
- If unable to reimplant the tooth, use one of the following carrier media (in order of preference):
  - Hanks solution (Save-A-Tooth): A pH-preserving fluid is best to use (if available).
  - Milk: Shown to maintain vitality of periodontal ligament cells for 3 hours, milk is relatively bacteria-free with pH and osmolarity compatible with vital cells.
  - Saline: acceptable as it is isotonic and sterile.
  - Saliva: keeps the tooth moist; however, it has incompatible osmolarity, pH, and bacteria.
  - Water: Least desirable as it results in hypotonic rapid cell destruction.

POTENTIAL PITFALLS

- Allowing an avulsed tooth to dry out can result in worsening of tooth damage.
- Failure to provide pain management when indicated.
- Failure to remember that trauma to the teeth is not life threatening; however, associated maxillofacial injuries and fractures can compromise the airway.
- Not encouraging a patient to be seen by a physician or dentist to determine if any roots remain or other teeth have been injured.
TRAUMA - TRAUMATIC BRAIN INJURY

Traumatic Brain Injury (TBI) results from blunt or penetrating trauma and the most common mechanism being motor vehicle accidents. Providers should attempt to obtain a history from bystanders or family members regarding the patient’s condition immediately after the injury. Calculation of a Glasgow Coma Score has prognostic value and should be done on all patients. Head injury remains a significant cause of death from trauma. Early aggressive management, airway maintenance, oxygenation, spinal restriction, and rapid transport to a Trauma Center are the goals ofprehospital management.

**ASSESSMENT AND TREATMENT CONSIDERATIONS**

- **Head trauma with a GCS < 13 should go to a trauma center.**
- **TBI can cause profound effects on the cardiovascular system.** Compression of the brain stem & medulla can cause dysrhythmias and is the main reason why all head-injured patients must have ECG monitoring.
- **If possible, try to elevate the patient’s head approximately 30 degrees (place on a backboard; elevate the head of the board). Avoid elevation if hypotension is present.**
- **If required for an imminent herniation, hyperventilation guided by quantitative ETCO2 monitoring to produce a PCO2 of 25-35 mm Hg will reduce ICP by cerebral vasoconstriction and reduction of CBF. The onset of action is within 30 seconds and is only done upon direct physician order.**
- **A large fixed pupil should suggest a herniation syndrome unless the patient is awake and alert. The fixed and dilated pupil is usually on the side of the expanding lesion.**
- **Pupillary Examination:** Several factors can alter pupillary examination results; narcotics cause constriction and drugs with sympathomimetic properties (cocaine, amphetamines etc.) cause dilation. These effects can blunt or eliminate pupillary responses. Prior eye surgery, such as cataract surgery, also can alter or eliminate pupillary reactivity.
  - **Bilateral small pupils** caused by narcotics, pontine injury (disruption of sympathetic centers in the pons), or central herniation (mass effect on the pons).
  - **Bilateral fixed and dilated pupils** are secondary to inadequate cerebral perfusion. This can result from cerebral hypoxia or severe elevations of ICP and usually indicate an irreversible injury.
  - **A unilateral fixed (unresponsive) and dilated pupil** has many potential causes;
    - A pupil that does not constrict when light is directed at it, but constricts when light is directed into the other pupil is indicative of a traumatic optic nerve injury.
    - A unilateral dilated pupil that does not respond usually is indicative of transtentorial herniation.
    - Unilateral constriction is often secondary disrupted sympathetic input and constriction occurs from more parasympathetic than sympathetic stimulation (also known as Horner’s syndrome).

**MEDICATION CONSIDERATIONS**

- **Do not administer large volumes of fluids in head injured patients unless absolutely necessary.**

**POTENTIAL PITFALLS**

- **Prophylactic hyperventilation has been associated with worsened neurologic outcome and is not recommended in the head-injured patient who does not exhibit signs or symptoms of impending herniation syndrome from increasing ICP.**
- **Failure to consider additional injuries in hypotensive patients with head injury. Isolated injury is not an etiology for shock, and IV fluids should be reserved for evidence of hypovolemia.**
- **Failing to remember that hyperventilation is only to be considered when active seizures or signs of impending herniation such as fixed or asymmetric pupils, posturing (decerebrate or decorticate), Cushing’s reflex (hypertension and bradycardia), periodic breathing (Cheyne-Stokes, central neurogenic, ataxic breathing), or abrupt neurologic deterioration are present.**
- **Placement of an intraosseous line in any patient without life-threatening injuries.**