

## FIVE QUADRADS APPROACH

- Applied to all patients in cardiac arrest
- Applied to patients in an unstable condition steadily deteriorating toward arrest

### 1. ABCD Primary Survey (Quadrant 1)

- A. AIRWAY - Open the Airway
- B. BREATHING - Provide positive-pressure ventilation
- C. CIRCULATION - Perform chest compressions
- D. DEFIBRILLATION - Identify and shock VF/pulseless VT

### 2. ABCD Secondary Survey (Quadrant 2)

#### A. AIRWAY -

- Determine the effectiveness of the initial ventilation and airway techniques
- Perform endotracheal intubation if indicated

#### B. BREATHING -

- Determine that the endotracheal tube is patent and properly placed
- Provide positive-pressure ventilation through the endotracheal tube
- Ensure that the chest wall moves with each ventilation
- Listen for bilateral breath sounds

#### C. CIRCULATION -

- Continue chest compressions
- Obtain IV access
- Attach monitor leads
- Identify rhythm and rate
- Measure blood pressure
- Administer medications appropriate for rhythm and vital signs

#### D. DIFFERENTIAL DIAGNOSIS - Consider possible causes of the cardiac emergency

### 3. ALWAYS RIGHT (Quadrant 3)

- Oxygen-IV-Monitor-Fluids

### 4. VITAL SIGNS (Quadrant 4)

- Temperature, blood pressure, heart rate, respirations

5. TANK - TANK - PUMP - RATE

- A. Tank (too large vs too small; due to systemic vascular resistance)
- B. Tank (too low vs too full; due to volume)
- C. Pump (pumping poorly vs pumping well; due to cardiac performance)
- D. Rate (too slow vs too fast) due to arrhythmias

DEVELOPING THE DIFFERENTIAL DIAGNOSIS  
THE H'S AND T'S

<b>The H Causes</b>	<b>Assessments</b>	<b>Treatments</b>
<b>Hypovolemia</b> <ul style="list-style-type: none"> <li>• Occult bleeding</li> <li>• Anaphylaxis</li> <li>• Pregnancy with gravid uterus</li> </ul>	<ul style="list-style-type: none"> <li>• History/exam</li> <li>• Hematocrit</li> <li>• B-HCG test</li> </ul>	<ul style="list-style-type: none"> <li>• Volume</li> <li>• Blood</li> </ul>
<b>Hypoxia</b> <ul style="list-style-type: none"> <li>• Inadequate oxygenation</li> </ul>	<ul style="list-style-type: none"> <li>• Breath sounds</li> <li>• Tube placement</li> <li>• Arterial blood gases</li> </ul>	<ul style="list-style-type: none"> <li>• Oxygen</li> <li>• Ventilation</li> <li>• Forceful CPR</li> <li>• ET tube stable</li> </ul>
<b>Hypothermia/Hyper</b> <ul style="list-style-type: none"> <li>• Profound hypothermia</li> <li>• Heat Stroke</li> </ul>	<ul style="list-style-type: none"> <li>• Touch</li> <li>• Core Body temperature</li> </ul>	<ul style="list-style-type: none"> <li>• Active/passive external rewarming</li> <li>• Active/passive internal rewarming</li> </ul>
<b>High and Low Electrolyte Levels</b> <ul style="list-style-type: none"> <li>• Potassium, sodium, magnesium, calcium</li> </ul>	<ul style="list-style-type: none"> <li>• History/exam</li> <li>• Risk Factors</li> </ul>	<ul style="list-style-type: none"> <li>• Calcium</li> <li>• Bicarbonates, insulin, glucose</li> </ul>
<b>Hypoglycemia</b> <b>Hyperglycemia</b> <ul style="list-style-type: none"> <li>• Low glucose = insulin reactions</li> <li>• Diabetes ketoacidosis</li> <li>• Nonketonic, hyperosmolar coma</li> </ul>	<ul style="list-style-type: none"> <li>• History/exam</li> <li>• Lab tests</li> </ul>	<ul style="list-style-type: none"> <li>• Fluids</li> <li>• Potassium</li> <li>• Insulin</li> <li>• 50% glucose</li> </ul>
<b>Hydrogen Ion</b> <ul style="list-style-type: none"> <li>• Acidosis</li> <li>• Diabetic ketoacidosis</li> <li>• Drug overdoses</li> <li>• Renal failure</li> </ul>	<ul style="list-style-type: none"> <li>• Clinical setting</li> <li>• Arterial blood gas</li> <li>• Lab tests</li> </ul>	<ul style="list-style-type: none"> <li>• Forceful CPR</li> <li>• Optimal perfusion</li> <li>• Hyperventilation</li> <li>• Bicarbonate</li> </ul>

<b>The T Causes</b>	<b>Assessments</b>	<b>Treatments</b>
<b>Trauma</b> <ul style="list-style-type: none"> <li>• Massive trauma</li> </ul>	<ul style="list-style-type: none"> <li>• History</li> <li>• Clinical Setting</li> </ul>	<ul style="list-style-type: none"> <li>• Rescuer Safety</li> <li>• Reverse triage</li> </ul>

<ul style="list-style-type: none"> <li>• Electrocution</li> <li>• Lightning</li> <li>• Near-drowning</li> </ul>	<ul style="list-style-type: none"> <li>• Physical Exam</li> </ul>	<ul style="list-style-type: none"> <li>• Early ETT placement</li> <li>• Treat asystole longer</li> </ul>
<p><b>Tension Pneumothorax</b></p> <ul style="list-style-type: none"> <li>• Asthma as possible cause</li> <li>• Trauma</li> <li>• COPD, blebs</li> <li>• Ventilators +positive pressure</li> </ul>	<ul style="list-style-type: none"> <li>• Risk factors</li> <li>• Diminished lung sounds</li> <li>• Tracheal deviation</li> <li>• Distended neck veins</li> </ul>	<ul style="list-style-type: none"> <li>• Needle compression</li> <li>• Chest Tube</li> </ul>
<p><b>Thrombosis, lungs</b></p> <ul style="list-style-type: none"> <li>• Pulmonary embolus</li> </ul>	<ul style="list-style-type: none"> <li>• Risk factors</li> <li>• History</li> <li>• Echo or VQ can</li> </ul>	<ul style="list-style-type: none"> <li>• Volume</li> <li>• Dopamine</li> <li>• Heparin</li> <li>• ?Thrombolytics</li> </ul>
<p><b>Thrombosis, heart</b></p> <ul style="list-style-type: none"> <li>• AMI</li> <li>• Other acute coronary syndromes</li> </ul>	<ul style="list-style-type: none"> <li>• Prearrest symptoms</li> <li>• ECG</li> <li>• Serum markers</li> </ul>	<ul style="list-style-type: none"> <li>• MONA, pressors</li> <li>• Emergent PTCA</li> <li>• Empiric rt-PA</li> <li>• Balloon pump</li> <li>• CABG</li> </ul>
<p><b>Tamponade, cardiac</b></p> <ul style="list-style-type: none"> <li>• Trauma</li> <li>• Renal failure</li> <li>• Chest compressions</li> <li>• Carcinoma</li> <li>• Central line perforations</li> </ul>	<ul style="list-style-type: none"> <li>• Risk factors</li> <li>• History</li> <li>• Prearrest picture</li> <li>• Distended neck veins</li> <li>• Echo</li> </ul>	<ul style="list-style-type: none"> <li>• Volume</li> <li>• Pericardiocentesis</li> <li>• Thoracotomy</li> </ul>
<p><b>Tablets (drug and toxin overdoses)</b></p> <ul style="list-style-type: none"> <li>• TCAs, phenothiazines</li> <li>• B-Blockers, calcium channel blockers</li> <li>• Cocaine, digoxin, aspirin, acetaminophen</li> </ul>	<ul style="list-style-type: none"> <li>• Risk Factors</li> <li>• History and toxidrome</li> </ul>	<ul style="list-style-type: none"> <li>• Specific antidotes</li> <li>• Bicarbonate</li> <li>• Glucagon, calcium</li> <li>• Long CPR</li> <li>• Cardiopulmonary bypass</li> </ul>

## CARDIOVASCULAR QUADRAD

### FOUR DETERMINANTS OF CARDIAC OUTPUT

1. Heart Rate (Electrical System)
2. Pump Performance (Contractility)
3. Tank Size (vascular tone, resistance)
4. Tank Contents (volume)

\*When multiple overlapping problems:

1. First correct rate problems

2. Second, correct tank problems with fluid (volume), and sometimes pressors (resistance)
3. Third, treat pump problems with pressors, inotropes or both

### **MAJOR ERRORS**

1. Using fluids or pressors when hypotension is due to tachycardia or bradycardia.
2. Using pressors instead of volume when hypotension is caused by tank problems
3. Using fluids when tank is already full and the problem is due to the pump.

<b>QUADRAD</b>	<b>ASSESS</b>	<b>MANAGE</b>
<b>Primary ABCD Survey</b>		
<ul style="list-style-type: none"> <li><b>Airway (C-Spine)</b></li> </ul>	<ul style="list-style-type: none"> <li>Open?</li> <li>Suspicious mechanism</li> </ul>	<ul style="list-style-type: none"> <li>Chin life, jaw thrust; head tilt</li> <li>Chin life, ;jaw thrust; no head tilt, stability; backboard; immobilize</li> </ul>
<ul style="list-style-type: none"> <li><b>Breathing</b></li> </ul>	<ul style="list-style-type: none"> <li>Moving Air?</li> </ul>	<ul style="list-style-type: none"> <li>Give 2 breaths, obstructed airway protocols</li> </ul>
<ul style="list-style-type: none"> <li><b>Circulation</b></li> </ul>	<ul style="list-style-type: none"> <li>Pulse?</li> </ul>	<ul style="list-style-type: none"> <li>Chest compressions - Basic CPR</li> </ul>
<ul style="list-style-type: none"> <li><b>Defibrillation</b></li> </ul>	<ul style="list-style-type: none"> <li>Attach AED; paddles; monitor</li> </ul>	<ul style="list-style-type: none"> <li>Shock if VF or pulseless VT</li> </ul>
<b>Secondary ACD Survey</b>		
<ul style="list-style-type: none"> <li><b>Airway (C-spine)</b></li> </ul>	<ul style="list-style-type: none"> <li>Adequate? Gag? Pooled secretions? Noisy breathing</li> </ul>	<ul style="list-style-type: none"> <li>Remove obstructions; suction; oropharyngeal airway; nasal trumpet endotracheal intubation; nasotracheal intubation; surgical airway</li> </ul>
<ul style="list-style-type: none"> <li><b>Breathing (OXYGEN)</b></li> </ul>	<ul style="list-style-type: none"> <li>Visualized cords? Tube in?</li> <li>Chest x-ray, pneumothorax? Flair chest? Open chest? Cyanosis? Moving air? Order ABG, attach O2 saturation monitor</li> </ul>	<ul style="list-style-type: none"> <li>Adjust ETT, needle decompression, chest tube, cover release sucking chest wound, order ventilator</li> <li>Provide oxygen via nasal cannula, face mask, nonrebreather mask, use hyperventilation and PEEP</li> </ul>
<ul style="list-style-type: none"> <li><b>Circulation (IV-Monitor-Fluids)</b></li> </ul>	<ul style="list-style-type: none"> <li>HR? BP? Monitor; assess rhythm</li> <li>Send blood fo rtype and crossmatch; labs (A,A,A,A,B,B, Tox) (Alc, ASA, APAP, amy Bhcg, Bili</li> </ul>	<ul style="list-style-type: none"> <li>Start IV; give rhythm appropriate medications; order fluids plus rate; order blood; stop visible hemorrhage serious hematocrits, If pregnant, place on left side.</li> </ul>
<b>QUADRAD</b>		
<ul style="list-style-type: none"> <li><b>Differential Diagnosis (THINK!)</b></li> </ul>	<ul style="list-style-type: none"> <li>Assess with the D-CP-D-E-E-F-F-F-G-G</li> </ul>	<ul style="list-style-type: none"> <li>Begin managing identified diagnosis</li> </ul>

	<p>system</p> <ul style="list-style-type: none"> <li>Consider differential diagnosis by H's and T's</li> </ul>	
<ul style="list-style-type: none"> <li><b>Disability - D-CP-D</b></li> </ul>	<ul style="list-style-type: none"> <li>Mental Status? Pupil response?</li> <li>GCS; best eye, vocal, motor response</li> <li>For altered mental status use "Coma Protocol" and reassess</li> <li>The same regimen can be remembered as DONT (Destrose, D50W; Oxygen, Narcan, Thiamine)</li> </ul>	<ul style="list-style-type: none"> <li>Coma Protocol, Give D50, thiamine 100mg IV, give narcan 2mg IV check oxygenation</li> <li>Assess response</li> </ul>
<ul style="list-style-type: none"> <li><b>Expose - examine - extremities</b></li> </ul>	<ul style="list-style-type: none"> <li>Completely expose patient; perform quick visual check for gross injuries, pregnancy, signs of skin lesions, skin temp, medic alert.</li> <li>Check extremity pulses</li> </ul>	<ul style="list-style-type: none"> <li>Stabilize obvious injuries; restore pulse to compromised extremities</li> </ul>
<ul style="list-style-type: none"> <li><b>Finger - foley - flip</b></li> </ul>	<ul style="list-style-type: none"> <li>Perform rectal, vaginal exam; check for injuries to pelvis, perineum or genitalia, then insert Foley</li> <li>Flip: log-roll patient to check back areas</li> </ul>	<ul style="list-style-type: none"> <li>Foley to straight drainage; send for analysis, including tox; observe urine output rate</li> </ul>
<ul style="list-style-type: none"> <li><b>Gastric tube - gunk</b></li> </ul>	<ul style="list-style-type: none"> <li>Check contraindications to nasal insertion</li> <li>Observe aspiration for blood, pills, odors</li> </ul>	<ul style="list-style-type: none"> <li>Instill gunk (activated charcoal 50g plus cathartic) down gastric tube for suspected drug Ods</li> </ul> <p>Note that several toxicology councils consider gastric lavage of little value</p>
<b>QUADRAD</b>	<b>ASSESS</b>	<b>MANAGE</b>
<ul style="list-style-type: none"> <li><b>History</b></li> </ul>	<ul style="list-style-type: none"> <li>Take expanded history in increase differential diagnosis</li> </ul>	<ul style="list-style-type: none"> <li>History delayed until patients stabilized.</li> <li>Use family friends, EMS personnel</li> </ul>
<ul style="list-style-type: none"> <li><b>02-IV-Monitor-</b></li> </ul>	<ul style="list-style-type: none"> <li>Assess response to</li> </ul>	<ul style="list-style-type: none"> <li>Continue</li> </ul>

<b>Fluids</b>	both surveys, check labs, x-rays	management of identified diagnoses
• <b>Temp-BP-HR-Respirations</b>	• Assess excessive low or high temperatures	• Continue management as indicated
• <b>Tank-tank-pump-rate</b>	• Consider problems in these categories	• Continue management as indicated

## **COMPLICATED ACUTE MYOCARDIAL INFARCTION**

### **I. CARDIOGENIC SHOCK**

- A. CHF is the most common cause of mortality following MI (10-12% of MI patients present in shock, with a 70-80% mortality rate.
- B. Thrombolytic therapy is less effective probably secondary to hypotension, poor perfusion and decreased coronary flow
- C. Best Outcome - IABP, Aggressive Medical Therapy and Primary PTCA
- D. Patients with shock (SBP <100) and tachycardia (>100) should be transported to interventional facilities
- E. \*Thrombolytic therapy should not be withheld pending transfer unless contraindications exist

### **II. INFERIOR WALL INFARCTION - RIGHT VENTRICULAR INFARCTION**

- A. Right Coronary Artery is dominant in most patients
- B. Impairs flow to the Right Ventricular Marginal Branch, causing ischemia and RV infarction
- C. RV infarction is present in one third of inferior wall infarcts
- D. ½ of these patients have hemodynamic instability
- E. RV infarction doubles the MI mortality rate

Right Ventricular Triad

- 1) Clear Lungs
- 2) Jugular Vein Distention
- 3) Hypotension

\*All patients with inferior wall infarct should have right sided ECG done

- Imm ST elevation in V4R is the single most predictive finding for RV ischemia/infarction
- Causes RV dilation (limited by pericardium)
- Both systolic and diastolic dysfunction

#### **TREATMENT**

- Support of preload is important to maintain cardiac output
- Factors that decrease preload (Nitrates, Morphine, Volume Depletion, diuretics) may worsen hemodynamics

- Increase preload and decrease afterload
- Inotropic support (Dobutamine) if 1-2 liters of NSS fails to correct hypotension
- Prompt cardioversion of atrial fibrillation if hemodynamics are compromised.

### III. HEART BLOCK WITH INFERIOR WALL INFARCTION

- A. 2<sup>nd</sup> and 3<sup>rd</sup> degree blocks occur in 20% of inferior wall infarcts
- B. 2/3 within the first 24 hours
- C. Most respond to Atropine and are not hemodynamically significant
- D. Heart Block is associated with increased mortality (not directly related to the heart block but because it is usually related to larger infarction size)

### IV. HYPOTENSION/CONGESTIVE HEART FAILURE

- A. Left ventricular dysfunction
- B. Combination of MI and ischemia results in decreased systolic dysfunction
- C. Nitrates, Diuretics, Morphine, oxygen
- D. Angiotension converting enzymes (ACE) inhibitors are effective afterload reducer which has been shown to decrease mortality and attenuate myocardial remodeling
- E. Beta Blockers benefit mild to moderate heart failure with infarction by blunting excess catecholamine effects during infarction
- F. Diuretics must be monitored closely. Hypotension and severe heart failure are contraindications.

### IV. MECHANICAL COMPLICATIONS

- A. Cardiac rupture is the second most common cause of in hospital mortality.
- B. Most commonly occur on day 5-7 but quicker (24-48 hours) in patients who have received thrombolytics
- C. Presence of new murmur should raise suspicion
- D. Parasternal thrill (if the rupture involves the septal wall)
- E. Portable Echo
- F. LV free wall rupture is usually fatal and presents with chest pain and PEA
- G. CT surgery is difficult but can be life saving

### V. POST INFARCTION ANGINA

- A. Recurrent chest pain with ST elevation means infarction extension
- B. The patient's mortality is more than doubled
- C. Heart failure in these patients is common
- D. Patients should receive Heparain (PTT 50-70)
- E. Expeditious coronary angiography
- F. Raise suspicion of pericarditis and pulmonary emboli

## **VI. VENTRICULAR ARRHYTHMIAS**

- A. Ventricular fibrillation increases short term but not long term mortality
- B. Following primary VF, Lidocaine is the drug of choice
- C. Amiodarone for non arrest rhythms or frequently recurring VF or hemodynamically significant VT

## **VII. POTASSIUM/MAGNESIUM**

- A. Low Potassium is associated with increased ventricular arrhythmias
- B. Early trials show mortality benefit from Magnesium (reduced CHF) due to its myocardial protectant properties

## **CARDIOVASCULAR EMERGENCIES (STATION ONE)**

### **I. RISK FACTORS FOR HEART DISEASE**

- Male gender
- Smoker
- Hypertension
- Middle Age

### **II. SUDDEN DEATH ASSOCIATED WITH CAD >80% of patients, Ventricular Fibrillation being the most common rhythm**

- A. Most Prehospital AMI deaths are related to VF
- B. Most VF deaths occur during the first hour of symptoms
- C. In hospital deaths often occur secondary to decreased cardiac output in the first 24-48 hours of treatment
- D. Mortality is directly related to the size of infarction
- E. Effective 911 system is the first step to improved survival.

### **III. EMS PERSONNEL**

- A. Trained and equipped to recognize and treat VF
- B. Can start MONA (Morphine, Nitro, Oxygen and ASA)
- C. Early Emergency Department Notification
- D. 12-Lead ECG for diagnosis
- E. Thrombolytic Screening

### **IV. EMERGENCY DEPARTMENT AMI PROTOCOL**

- A. ECG screening within 10 minutes
- B. Door to Drug (Thrombolytics) 30 minutes
- C. Door to Balloon Time = 90 minutes
- D. Oxygen, IV, Monitor and Morphine
- E. Reperfusion for ST segment elevation
- F. Rule out contraindications
- G. Consider PTCA if ineligible for thrombolytics
- H. Angiography for cardiogenic shock
- I. ASA
- J. Beta Blockade (all patients without contraindications)
- K. IV Nitroglycerins for initial 24-48 hours in patients with AMI and CHF, large anterior wall infarction, persistent ischemia or HTN
- L. Serum Markers
- M. Electrolytes; coagulation studies
- N. Chest x-ray

### **V. ELECTROCARDIOGRAM**

- A. ST Elevation - high specificity for evolving MI; assess for reperfusion

- B. ST Depression - Consistent with/strongly suggestive of ischemia; defines a high risk subset of patients with non-Q wave MI or unstable angina
- C. Non Diagnostic or Normal ECG - further assessment is usually needed; evaluation protocols may include repeat ECG or continuous ST monitoring; serial cardiac markers, myocardial imaging or 2D echo.

## **VI. CORONARY ARTERY DISTRIBUTION**

### A. Left Side

- 1. Septal Wall of the Left Ventricle
- 2. Anterior and Lateral Walls of the LV
- 3. Inferior Wall (10% of patients)
- 4. Both Bundle Branches

### B. Right Side

- 1. Inferior wall of the Left Ventricle
- 2. Posterior wall of the LV (90% of patients)
- 3. AV Node (90% of patients)
- 4. Right Ventricle

## **VII. ASSOCIATED CONDUCTION PROBLEMS**

### A. Left Side Infarction

- 1. Advanced (2<sup>nd</sup> or 3<sup>rd</sup>) Degree heart blocks with wide QRS which tends to degrade faster or to convert to asystole without warning
- 2. Bundle Branch Blocks
- 3. TCP is treatment of choice for wide QRS heart blocks

### B. Right Side Infarction

- 1. Heart Block, commonly AV nodal, transient, responds well to Atropine.
- 2. Always consider Right Ventricular Infarction

\*Inferior Wall Myocardial Infarction can be caused by either RCA (90%) or dominant circumflex artery occlusion.

## **VIII. RIGHT VENTRICULAR INFARCTION**

- A. Recognize clinical clues - need for right ECG leads
- B. Avoid nitrates and other vasodilators (may cause hypotension)
- C. Thrombolytics and PTCA improve right ventricular ejection fraction and decrease the incidence of heart block

- D. PTCA is indicated for shock, in patients age <75 years.
- E. If hypotension - 1-2 liters NSS in 250-500cc boluses
- F. Dobutamine is particularly effective if hypotension persists
- G. IABP if concomitant LV dysfunction

#### **IX. ARRHYTHMIA MANAGEMENT**

- A. AV synchrony is important - cardiovert hemodynamically unstable atrial fibrillation.
- B. AV synchronous pacing may be efficacious

#### **X. TREATMENTS TO CONSIDER**

- A. Oxygen
- B. Nitroglycerin
- C. Analgesia
- D. ASA
- E. Beta Blockers
- F. ACE Inhibitors
- G. Magnesium
- H. Lidocaine
- I. Reperfusion (PTCA or Thrombolytics)

## **PHARMACOLOGY**

### **NITROGLYCERIN**

#### **MECHANISMS OF ACTION**

- Dilates coronary arteries
- Suppresses coronary artery spasm
- Increases collateral blood flow
- Analgesic
- Reduces myocardial oxygen use
- Reduces LV workload - decreases afterload

#### **ADMINISTRATION**

- SL, spray (avoid topical or long acting nitrates)
- IV 10-20 ug/min; limit SBP drop to 10% if normotensive, 30% if hypertensive

#### **INDICATIONS**

- Suspected ischemic chest pain
- Unstable angina
- Acute pulmonary edema
- Hypertension in AMI

#### **CAUTIONS**

- Caution if SBP >90mm Hg
- Beware in Right Ventricular Infarction
- Hypovolemic patients are prone to severe hypertension

### **MORPHINE SULFATE**

#### **EFFECTS**

- Analgesia plus venodilation reduces ventricular preload and oxygen requirements

#### **INDICATIONS**

- Treatment of ischemic pain not relieved by nitroglycerin; also useful to redistribute blood volume in patients with pulmonary edema.

#### **CAUTIONS AND COMPLICATIONS**

- Do not use in patients with suspected hypovolemia
- If hypotension develops, elevate the patient's legs and administer normal saline

## **ASPIRIN**

### **EFFECTS**

- 160-325mg causes immediate and near-total inhibition of thromboxane A<sub>2</sub> production. Reduces coronary reocclusion and recurrent events after thrombolytic therapy. Also important for treatment of unstable angina

### **INDICATIONS:**

- All patients with suspected acute coronary syndromes
- Particularly reperfusion candidates, unless hypersensitivity to ASA

### **DOSE/ROUTE**

- ASA is absorbed faster when chewed than when swallowed in the early hours after infarction, particularly if morphine has been given.
- ASA suppositories (325mg) can be used safely and are recommended for patients with severe nausea, vomiting, or upper gastrointestinal disorders

## **BETA BLOCKERS**

### **MECHANISM OF ACTION**

- Block sympathetic nervous system stimulation of heart rate and contractility
- Block B<sub>2</sub> mediated sympathetic vasodilation, resulting in vasoconstriction and increased vascular resistance.

### **EFFECTS**

- Can reduce infarction size in patients not receiving thrombolytic therapy.
- Reduce short term mortality
- In patients receiving thrombolytic therapy, they decrease post infarction ischemia and nonfatal MI
- The incidence of VF is reduced

### **INDICATIONS**

- All pts with Q-wave infarction without contraindications
- All patients with non-Q-wave infarction without contraindications
- Treatment of recurrent or continuing pain associated with AMI
- Tachyarrhythmias associated with catecholamine excess (i.e. atrial fibrillation)

## **ABSOLUTE CONTRAINDICATIONS**

- Severe congestive heart failure
- Hypotension (SBP <100mm Hg)
- Bronchospasm
- 2<sup>nd</sup> or 3<sup>rd</sup> degree AV Block

## **CAUTIONS**

- Mild/moderate CHF
- Bradycardia (HR <60 bpm)
- History of Asthma
- IDDM
- Severe peripheral vascular disease

## **HEPARIN**

### **MECHANISM OF ACTION**

- Indirect thrombin inhibitor (at AT III)

### **INDICATIONS**

#### **ST ELEVATION INFARCTION**

- Adjunct to thrombolytic therapy with fibrin specific agents
- Adjunct for PTCA
- High Risk for systemic emboli (large anterior wall, atrial fibrillation, previous embolus, known LV thrombus)

#### **ST DEPRESSION INFARCTION/UNSTABLE ANGINA**

- Conjunction with ASA
- Recommended for all High Risk Patients

## **ACE INHIBITORS**

### **EFFECTS**

- Early oral ACE inhibition reduces mortality
- Reduces CHF associated with MI
- Prevent adverse LV remodeling, delay progression of heart failure, and decrease sudden death and recurrent MI

### **INDICATIONS (Greatest benefit in patients with)**

- Anterior infarction
- Prior infarction
- Heart failure
- Clinical signs of left ventricular dysfunction (ejection fraction 40%)

### **DOSE, ADMINISTRATION**

- Not administered during the first 6 hours after MI
- Once stable a low oral dose is initiated and titrated to the full dose within 24 to 48 hours

### **POTASSIUM AND MAGNESIUM SULFATE**

#### **EFFECTS**

- Potassium deficiency but not magnesium deficiency associated with arrhythmias, sudden death
- Recent studies suggest no reduction in AMI mortality with Magnesium administration

#### **RECOMMENDATIONS**

- Correction of documented deficits, especially in patients receiving prior diuretics
- Episodes of torsades-de-pointes-type VT associated with a prolonged QT interval.
- Use in High Risk patients with MI, such as those not receiving thrombolysis or the elderly
- Routine use in AMI is not recommended

## **THROMBOLYTIC THERAPY FOR ACUTE CORONARY SYNDROMES**

### **INDICATIONS**

- Chest pain suggesting MI
- ST segment elevation  $<0.1\text{mV}$  in 2 or more contiguous ECG leads or new or presumably new bundle branch block
- Time to therapy  $<12$  hours
- Age  $<75$  years

### **ABSOLUTE CONTRAINDICATIONS**

- Previous hemorrhagic stroke
- Other stroke or cerebrovascular accident within 1 year
- Active internal bleeding (menses excluded)

- Suspected aortic dissection

### **RELATIVE CONTRAINDICATIONS AND CAUTIONS**

- Severe uncontrolled HTN
- Current use of anticoagulants (INR >2.5); known bleeding disorder
- Recent trauma (within 2-4 weeks) including head and traumatic CPR or major surgery <3weeks.
- Recent (2-4 weeks) internal bleeding; active peptic ulcer disease
- Pregnancy
- For streptokinase allergy or prior exposure

### **PERCUTANEOUS TRANSLUMINAL CORONARY ANGIOPLASTY**

\*\* In experienced centers PTCA can restore vessel patency and normal flow the a 90% success rate.

#### **Primary PTCA is most effective for the following:**

- Cardiogenic shock, age <75 years if performed <18 hours from onset of shock and <36 hours from onset of ST elevation.
- As an alternative to thrombolytic therapy in acute ST elevation or Q wave or new LBBB MI patients, if performed <12 hours from onset of pain
- In patients with indications for reperfusion but with a contraindication to thrombolytic therapy

#### **Best results are Achieved at PTCA Centers with the following Characteristics:**

- Center volume >200 procedures per year
- Individual operator volume of >75 procedures per year
- Balloon dilation within 90 minutes of AMI diagnosis

### **RECOMMENDATIONS FOR INTRA-AORTIC BALLOON COUNTERPULSATION**

- Stabilize cardiogenic shock for angiography and revascularization
- Acute mitral regurgitation or VSD pending angiography and repair
- Recurrent intractable ventricular arrhythmia with hemodynamic instability
- Refractory post-MI angina pending angiography

May be helpful with:

- Hemodynamic instability, poor LV function, pr persistent ischemia with large area of myocardium at risk; reperfusion not possible or anticipated.

## **SERUM MARKERS**

Blocked blood flow to the myocardium leads to cellular (myocytes) ischemia, which causes injury and may result in cell death or necrosis with loss of cell membrane integrity and release of cellular macromolecules

New serum markers achieve earlier diagnosis with greater sensitivity (able to detect very small amounts of markers) and specificity (markers are never elevated unless cell death has occurred).

Serum markers can now differentiate between non-Q-wave MI (serum markers released) and unstable angina (no serum markers released)

Serum markers can provide useful risk stratification and prognostic information.

## **ELECTROLYTE ABNORMALITIES**

### **LEARNING STATION TWO**

#### **I. POTASSIUM**

- Affects cardiovascular activity and is essential for muscle and neurological function
- Small changes have large consequences
- Of all electrolytes rapid changes in K<sup>+</sup> cause most life threatening consequences
- K<sup>+</sup> is a major intracellular cation
- Electrical charge across cell membrane accounts for function. Magnitude of charge is determined by transmembrane K<sup>+</sup> gradient
- Primary source is food. Average intake 1mEq/kg/day
- May reduce by promoting intracellular shift or renal excretion or by reducing GI absorption
- Cardiovascular effects of K<sup>+</sup> are reflected in ECG and by direct measurement of serum

#### **HYPERKALEMIA**

- Normal 3.5-5 mEq/L
- Serum hyperkalemia does not imply a total body excess of K<sup>+</sup>
- Neurons and muscles are most affected by hyperkalemia
- Suggested by ECG Changes
  - Peaked T-waves
  - Flattened P waves
  - Prolonged PR interval
  - Widened QRS Complex
  - Deepened S wave
  - Idioventricular Rhythm
  - Sine-wave formation
  - Ventricular Fibrillation – cardiac arrest

#### **SYMPTOMS**

- Weakness
- Ascending paralysis
- Respiratory Failure

#### **CAUSES**

- Diet
- Medication
- Renal Failure
- Redistribution fall of pH
- Aldosterone Deficiency
- Pseudohyperkalemia (Hemodialysis)

## **TREATMENT**

- Remove the source or cause of hyperkalemia
- Stabilize myocardial cells (calcium antagonizes potassium effect)
- Chemically shift potassium intracellularly
  - Glucose and insulin
  - Sodium Bicarbonate
  - Beta Blockers
- Physically remove excess K<sup>+</sup>
  - Diuretics or dialysis
  - Ion-exchangeresins

## **HYPOKALEMIA**

Defined as a low measured serum potassium.

- Normal = 3.5- 5.0 mEq/L
- Serum hypokalemia does not imply a total body deficit
- Neurons and muscle are the cells most affected by hypokalemia
- Serum hypokalemia may be suggested by ECG changes
  - U waves
  - T-wave flattening
  - ST segment changes
  - Cardiac arrhythmias, especially if the patient is receiving digitalis

## **SIGNS AND SYMPTOMS**

- Neuromuscular symptoms
  - Weakness
  - Fatigue
  - Paralysis
  - Respiratory Difficulty
  - Rhabdomyolysis
- Gastrointestinal Symptoms:
  - Constipation
  - Ileus
- Nephrogenic Diabetes Insipidus

## **CRITICAL ACTIONS**

- Assess the Five Quadrads
- Obtain and evaluate the ECG
- Obtain a stat serum potassium and other electrolytes
- Obtain a stat blood gas if there is a question of pH disturbance
- Obtain a stat CBC and hematocrit
- Begin directed resuscitation or intervention
- Reassess patient status and electrolytes frequently

- Obtain additional history and ancillary studies as time and conditions allow
- Prepare for transport to the critical care unit, admission, or discharge as determine by response to intervention

## **TREATMENT**

- Give potassium
  - Orally
  - Intravenously (Decreased renal output)
  - Estimate total body K<sup>+</sup> deficit
    - Dependent on age and muscle mass
    - Rough Estimate: Decrease in serum K<sup>+</sup> is equivalent to a deficit of 150-400 mEq/L of total body K<sup>+</sup> in a steady state

## **SODIUM**

Is the major ECF cation. Total body sodium is normally half of the total K<sup>+</sup> content. Because it is extracellular it is more accessible than other electrolytes to rapid and dramatic changes in concentration and quantity by the renal and GI system. Governed by renin-angiotensin, aldosterone and antidiuretic hormones affecting primarily the kidneys.

## **CRITICAL KNOWLEDGE**

- Normal range for Sodium 135-145 mEq/L
- Extracellular fluid volume is affected by total body sodium
- Serum sodium concentration is affected by extracellular water volume
- Total body sodium cannot be determined from the serum sodium concentration (this is estimated in conjunction with the physical examination)
- Changes in serum sodium concentration may suggest a change of total body sodium in a steady state
- Serum osmolality is a measure of the solute concentration in the ECG. Serum tonicity is a measure of the driving force for water of the active solutes across the cell membrane.
- Rapid changes in serum sodium are accompanied by similar changes in K<sup>+</sup>
- The physical condition of the patient reflects changes in serum sodium concentration. The more rapid the changes, the more striking the physical changes.
- There are no significant ECG changes with hypernatremia or hyponatremia.

## **CRITICAL ACTIONS**

- Assess the Five Quadrads
- Stabilize the patient's vital signs
- Measure serum and urine sodium concentration
- Measure other serum and urine electrolytes

- Estimate and measure the serum and urine osmolality
- Obtain and Evaluate ECG
- Consider possible means of altering factors causing the sodium abnormality.

### **HYPERNATREMIA**

Excess of sodium. Caused by an excess of sodium relative to water.

Excessive water losses most often caused by renal and gastrointestinal routes. Most physical manifestations expressed as changes in the patient's neurological status.

### **CRITICAL KNOWLEDGE**

- Normal range = 135-145
- Major reasons are impaired thirst mechanisms, chronic debilitating illnesses, disordered or altered mental status, primary neurologic disorders or lack of access to water.
- Treatment consists of replacing water and sodium to avoid rapid fall in serum sodium and osmolality and resultant extravascular fluid shifts and cerebral edema fluids.

### **HYPONATREMIA**

Low measured serum sodium. Caused by an excess of water relative to sodium. Most cases are caused by reduced renal water excretion in the presence of continued water intake. Severe, rapid onset usually results in neurologic symptoms, slower onset causes changes in the respiratory and cardiovascular systems.

### **TREATMENT**

Aimed at finding the causes while treating the symptoms. The history and physical exam are best resources for determining the etiology, time of onset and volume status.

### **CALCIUM**

Most abundant mineral in the body and is essential for skeletal support and neuromuscular function. Also extracellular cation and is actively pumped out of the cells with sodium and therefore able to antagonize both potassium and magnesium.

### **CRITICAL KNOWLEDGE**

- Normal range - 8.5 to 10.5 mg/dL
- Total serum calcium is a balance between GI absorption, renal excretion and skeletal mass exchange

- Calcium and sodium both exchange with potassium in the renal tubules during reabsorption

### **CRITICAL ACTIONS**

- Assess the Five Quadrads
- Determine serum pH, calcium, magnesium, albumin, BUN, creatinine, and electrolyte concentrations
- Obtain and evaluate the ECG
- Begin directed intervention and treatment of the abnormality
- Obtain additional history, laboratory studies and monitoring data as indicated

### **HYPERCALCEMIA**

Primary hyperparathyroidism and malignancy account for more than 90% of the reported cases and are most often evident from history and physical. Primary disorder is due to increased entry of calcium from the skeletal reservoir and GI tract with decreased renal clearance.

### **CRITICAL KNOWLEDGE**

- Normal range = 8.5 to 10.5 mg/dL
- Symptoms will appear at 12-15mg/kL
- CNS effects are due to decreased permeability of membranes
- Depression, weakness, fatigue, and confusion occur with mild elevations of calcium
- Hallucinations, disorientation, hypotonicity and coma develop at critical levels
- Cardiac effects are due to alteration in action potential.
- Contractility increases until 15-20 mg/dL after which myocardial depression occurs. Automaticity is decreased
- Ventricular systole is shortened and arrhythmias occur
- ECG Changes
  - Shortened QT intervals
  - Prolonged PR and QRS intervals
  - Increased QRS voltage
  - T-wave flattening and widening
  - Notching of QRS
  - AV Block progressing to arrest
- GI effects include dysphagia, constipation, peptic ulcers and pancreatitis
- Vascular effects cause hypertension

### **TREATMENT**

- Begin treatment at 12mg/dL

- Start large bore IV
- Infuse NSS at 300-500cc to correct fluid deficits, restore filtration and promote calcium excretion
- Continue NSS at 100-200cc/hr
- Consider Plicamycin, calcitonin, glucocorticoids, or oral phosphates as alternatives

## **HYPOCALCEMIA**

Most causes are dependent on absorptive, excretory, and hormonal functions.

### **CRITICAL ACTIONS**

- Assess five quadrads
- Large bore IV
- Obtain and evaluate ECG
- Treat acute symptomatic hypocalcemia with 10% calcium gluconate 90-180 mg IV over 10 minutes
- Measure response every 4-6 hours
- Correct magnesium, potassium and pH abnormalities

## **MAGNESIUM**

Fourth most abundant mineral in the body. It is primarily an intracellular cation and participates in a host of enzymatic actions and hormonal actions, most involving energy production through glycolysis and adenosine phosphorylation.

### **CRITICAL KNOWLEDGE**

- Normal 1.3 - 2.2 mEq/L
- Fifty percent is found in bone and in insoluble form
- Majority in total body water is intracellular
- Renal excretion is normal.
- Magnesium is necessary for adequate energy production

### **CRITICAL ACTIONS**

- Assess the five quadrads
- Obtain definitive history
- Obtain and evaluate ECG
- Begin directed intervention and treatment

## **HYPERMAGNESEMIA**

Clearance is through the kidneys. Usually does not occur without the presence of some renal insufficiency. Symptoms include muscular weakness. ECG changes are reflected in electrolyte shifts of potassium.

### **CRITICAL KNOWLEDGE**

- Concentration > 2.2

### **CRITICAL ACTIONS**

- Assess the five quadrads
- Obtain magnesium and electrolyte levels
- Obtain a current history
- Perform a physical exam
- Obtain and evaluate ECG
- Begin directed intervention and treatment
- Reevaluate frequently to assess treatment response

### **HYPOMAGNESEMIA**

Most often due to decreased absorption or increased renal, GI loss or secondary to alterations of parathyroid hormone. Symptoms include muscular irritability, altered mentation, and atrial and ventricular rhythm disturbances. Treatment consists of supplementation given IV or IM

### **CRITICAL KNOWLEDGE**

- Concentration <1.4 mEq/L
- ECG changes are
  - Prolongation of PR and QT intervals
  - Nonspecific depression of ST interval
  - Flattening or inversion of precordial P waves
  - Widening of QRS
  - Torsades de pointes
  - Atrial Arrhythmias
  - Worsening of digitalis toxicity

### **CRITICAL ACTIONS**

- Assess five quadrads
- Obtain history
- Obtain and evaluate ECG
- Immediately correct severe hypomagnesemia IV or by IM injection
- Definitive treatment is determined by history and diagnosis
- Reevaluate frequently

### EMERGENCY TREATMENTS FOR HYPERKALEMIA

Therapy	Dose	Effect Mechanism	Onset of Effects	Duration of Effects
Sodium Bicarbonate	1mEq/kg IV bolus	Shifts	5-10 minutes	1-2 hours
Calcium Chloride	5-10 mL IV 10% Solution (500-1000mg)	Antagonize	1-3 minutes	30-60 minutes
Insulin plus Glucose	Regular insulin 10 U IV plus 1 ampule glucose	Shifts	30 minutes	4-6 hours
Diuresis with Lasix	40-80 mg IV bolus	Removes	When diuresis	When diuresis ends
Cation-exchange resin	Kayexalate 15-50 g PO or PR plus sorbitol	Removes	1-2 hours	4-6 hours
Peritoneal dialysis Hemodialysis	Per institution	Removes	As soon as started	Until dialysis completed

### NORMAL VALUES

SERUM VALUES	
Sodium	135-145 mEq/L
Potassium	3.5-5.0 mEq/L
Chloride	95-105 mEq/L
Bicarbonate	24-26 mEq/L
Osmolality	280-295 mEq/L
Osmolal gap	<10 mOsm/L
Anion Gap	9-16 mEq/L
Urea	10-20 mg/dL

Clinical Problem	Typical ECG Findings	Treatment Approaches
<b>Hyperkalemia</b> ECG changes are listed	<ul style="list-style-type: none"> <li>• Tall peaked T waves</li> <li>• Prolonged PR</li> </ul>	<ul style="list-style-type: none"> <li>• Sodium Bicarb 1mEq/kg IV bolus</li> </ul>

in order of increasing K+	<ul style="list-style-type: none"> <li>• Prolonged QT</li> <li>• P waves diminished</li> <li>• ST segment depression</li> <li>• Sine-wave PEA</li> <li>• Wide complex tachycardias</li> </ul>	<ul style="list-style-type: none"> <li>• Calcium chloride 5-10 mlIV</li> <li>• Reg insulin 10U IV plus 1 amp D50</li> <li>• Furosemide 40-80mg IV</li> <li>• Kayexalate 15-50 g PO/PR plus sorbitol</li> <li>• Peritoneal dialysis or hemodialysis</li> </ul>
<b>Hypokalemia</b>	<ul style="list-style-type: none"> <li>• U waves become prominent</li> <li>• T waves flatten</li> <li>• ST segment becomes depressed</li> <li>• QT interval prolonged</li> <li>• QRS widens</li> <li>• Wide complex tachycardias</li> </ul>	<ul style="list-style-type: none"> <li>• If 3-3.5 need 100mEq replacement</li> <li>• If 2.5-3.0 need 200 mEq replacement</li> <li>• Infuse 10-40 mEq/h</li> </ul>
<b>Hypercalcemia</b> Normal total calcium 8.5-10.5 mg/dL Normal ionized calcium = 4.2-4.8	<ul style="list-style-type: none"> <li>• QT intervals markedly shortened</li> <li>• Automaticity decreased</li> <li>• ST segments shortened and depressed</li> <li>• T waves widen</li> <li>• Bundle branch blocks may occur</li> <li>• Second degree heart block</li> </ul>	<b>Urgent Treatment</b> <ul style="list-style-type: none"> <li>• NS bolus: induce diuresis</li> <li>• Lasix 40-100 mg IV</li> <li>• Replace K+ and mag</li> </ul> <b>CARDIAC ARREST</b> <ul style="list-style-type: none"> <li>• ACLS plus</li> <li>• Magnesium sulfate 1-2 g IV</li> <li>• K+ 1-2 mEq/min</li> <li>• NS bolus</li> <li>• Diuretics</li> </ul>
<b>Hypocalcemia</b> Normal total calcium = 8.5 - 10.5 mg/dL Normal ionized calcium= 4.2-4.8 mg/dL	<ul style="list-style-type: none"> <li>• Prolonged QT</li> <li>• May have VT or torsades</li> </ul>	<b>Urgent Treatment</b> <ul style="list-style-type: none"> <li>• Calcium Chloride 10% (1 g in 10ml = 272 mg) Give 100mg in 100cc D5W in 10-20 minutes</li> <li>• IN the next 6-12 hrs give a total of 1 gram by infusion</li> </ul> <b>Cardiac Arrest</b> <ul style="list-style-type: none"> <li>• <b>Full ampule of calcium chloride</b></li> </ul>
<b>Hypomagnesemia</b> Normal = 1.5-2.0 mgEq/L Hypomag <1.4 Severe <0.5	<ul style="list-style-type: none"> <li>• Prolonged PR and QT</li> <li>• Wide QRS</li> <li>• ST depression</li> <li>• Broad flat T wave</li> </ul>	

## ELECTROLYTE PROBLEMS AND ECG ABNORMALITIES

Electrolyte Problem	Typical ECG Abnormalities
<b>Hyperkalemia</b>	<ul style="list-style-type: none"> <li>• Prolonged PR</li> <li>• P waves almost disappear</li> <li>• Tall, peaked T waves</li> <li>• ST segment depression, S and T waves merge</li> <li>• Sine-wave PEA, wide complex tachycardia</li> </ul>
<b>Hypokalemia</b>	<ul style="list-style-type: none"> <li>• U waves become more prominent</li> <li>• T waves flatten</li> <li>• ST segment becomes depressed</li> <li>• QT interval becomes more prolonged</li> <li>• Wide complex tachycardias</li> </ul>
<b>Hypercalcemia</b>	<ul style="list-style-type: none"> <li>• Key: QT interval markedly shortened</li> <li>• Automaticity decreased</li> <li>• ST segments shortened and depressed</li> <li>• T waves widen</li> <li>• BBB</li> <li>• Second degree heart block</li> </ul>
<b>Hypocalcemia</b>	<ul style="list-style-type: none"> <li>• Prolonged QT interval due to prolonged ST segment</li> <li>• May experience VT or torsades de points</li> </ul>
<b>Hypomagnesemia</b>	<ul style="list-style-type: none"> <li>• Prolonged PR and QT intervals</li> <li>• Wide QRS complex</li> <li>• ST depression</li> <li>• Broad, flat T waves with precordial T wave inversion</li> </ul>

## ECG ABNORMALITIES AND CLINICAL PROBLEMS

TYPICAL ECG ABNORMALITIES	CLINICAL PROBLEMS
<b>P waves diminished</b>	<ul style="list-style-type: none"> <li>• Hyperkalemia</li> </ul>
<b>PR intervals prolonged</b>	<ul style="list-style-type: none"> <li>• Hyperkalemia</li> <li>• Hypomagnesemia</li> <li>• Hypercalcemia</li> <li>• B blockers</li> <li>• Calcium channel blockers</li> <li>• Tricyclic antidepressants</li> <li>• Neuroleptics</li> </ul>
<b>QRS Widened</b>	<ul style="list-style-type: none"> <li>• Hypomagnesemia</li> <li>• Hypercalcemia</li> <li>• Beta Blockers</li> <li>• Tricyclic antidepressants</li> </ul>
<b>QT Interval Prolonged</b>	<ul style="list-style-type: none"> <li>• Hyperkalemia</li> <li>• Hypocalcemia</li> </ul>

	<ul style="list-style-type: none"> <li>• Tricyclic Antidepressants</li> <li>• Neuroleptics</li> <li>• Calcium Channel Blockers</li> </ul>
<b>QT Interval Shortened</b>	<ul style="list-style-type: none"> <li>• Hypercalcemia</li> </ul>
<b>ST Segment Depression</b>	<ul style="list-style-type: none"> <li>• Hyperkalemia</li> </ul>
<b>ST Segment Shortened</b>	<ul style="list-style-type: none"> <li>• Hypercalcemia</li> <li>• Beta Blockers</li> </ul>
<b>T waves tall and peaked</b>	<ul style="list-style-type: none"> <li>• Hyperkalemia</li> <li>• Beta Blockers</li> </ul>
<b>T waves Wider</b>	<ul style="list-style-type: none"> <li>• Hypercalcemia</li> </ul>
<b>T waves flatter</b>	<ul style="list-style-type: none"> <li>• Hypomagnesemia</li> </ul>
<b>U waves appear</b>	<ul style="list-style-type: none"> <li>• Hypokalemia</li> </ul>

### **RED FLAGS FOR ELECTROLYTE ABNORMALITIES**

1. Abnormal vital signs
2. Altered mental status
3. History of
  - Prolonged chronic disease
  - Gastrointestinal or renal disease, malnutrition
  - Diabetes, other endocrinopathies
  - Cancer
  - Alcohol or drug abuse

Don't wait for results of laboratory analysis to identify an electrolyte abnormality - instead BE PROACTIVE LOOKING FOR problems

## **LEARNING STATION THREE ENVIRONMENTAL EMERGENCIES**

1. Near drowning
2. Life threatening asthma
3. Hypothermia
4. Anaphylaxis
5. Lightning strike
6. Cardiac arrest associated with trauma
7. Cardiac arrest associated with pregnancy
8. Electric shock

### **I. NEAR DROWNING**

- A. No modification of standard BLS is necessary
- B. In water resuscitation requires flotation devices and training.  
External chest compression cannot be performed in the water
- C. Suspect spinal injuries and treat as a trauma patient
- D. Do not attempt to drain water from lungs
- E. Remove foreign bodies in the airway
- F. Vomiting and regurgitation are very common.
- G. All immersion victims who require resuscitation should be transported.

### ADVANCED LIFE SUPPORT

- A. No modification of standard ALS is necessary
- B. Early trachea intubation is indicated to
  - Improve oxygenation, intermittent positive-pressure ventilation and reduce Paco<sub>2</sub>
  - Suction the tracheobronchial tree
  - Apply continuous positive airway pressure (CPAP or PEEP)

### PROGNOSTICS

- A. Predictors of outcome can be unreliable in the prehospital setting
- B. Patients reaching the hospital with a spontaneous circulation and breathing have good outcomes

### DEFINITIONS:

**DROWNING** - refers to submersion that causes either immediate death or death within 24 hours

**NEAR DROWNING** - submersion that does not result in immediate death or death within 24 hours

The most impressive effects of submersion are those related to hypoxia, not the chemical composition of the submersion fluid (unless it is contaminated, i.e., septic tank)

## PATHOPHYSIOLOGY

- A. Hypoxemia is the major insult; duration determines outcome
- B. Rule out associated conditions; trauma, alcohol intoxication, hypothermia
- C. Potential neurologic insults; hypoxia, trauma
- D. Potential pulmonary insults: pulmonary edema, intrapulmonary shunting, surfactant inactivation, ARDS, aspiration

## II. LIFE THREATENING ASTHMA

Severe asthma can lead to several forms of sudden cardiac death.

- Severe bronchospasm, leading to asphyxia
- Tension pneumothorax, often bilateral
- Cardiac arrhythmias
- Use of B adrenergic agonists
- Hypotension and bradycardia mediated by vasovagal reflexes
- Cardiac conduction disease

Most deaths occur outside the hospital. The major clinical action should be aggressive treatment of all acute severe asthmatic crisis before deterioration to full arrest.

## INTERVENTION KEYS

- A. Oxygen - use sufficient inspired oxygen to achieve a PaO<sub>2</sub> of 90mmHg. Use high flow oxygen by mask, be ready to intubate
- B. Nebulized B<sub>2</sub> Agonists - Metaproterenol has become the cornerstone of most therapy. Escalating doses of 5-10 mg every 15-20 mins up to three times.
- C. IV Corticosteroids - Begin corticosteroid therapy immediately with status asthmaticus. Methylprednisolone 2mg/kg as first dose repeated every 6 hours or Hydrocortisone 10mg/kg every 6 hrs.
- D. Nebulized Anticholinergic Therapy - Ipratropium bromide dose inhalers or as a moist nebulization at a dose of 0.5mg with B<sub>2</sub> agonists
- E. IV Aminophylline - used if B<sub>2</sub> or corticosteroids fail. Aminophylline may produce significant side effects. Loading Dose 5mg/kg given over 30-45 mins, followed by infusion of 0.5mg to 0.7mg/kg/hr
- F. Intravenous Magnesium Sulfate - not consistently effective
- G. IV B Agonists - Isoproterenol IV over several hours can be effective for severely ill patients. 0.1 ug/kg/min to max of 0.6 ug/kg/min
- H. Intravenous Epinephrine - Aggressive approach for life threatening situations. 2-10 ml of 1:10000 over five minutes and repeated in five minutes
- I. IV Sodium Bicarbonate - acidosis is known to counteract beneficial effects of sympathomimetic amines.
- J. Tracheal Intubation

- Provide adequate sedation with ketamine
- Paralyze the patient
- Once intubated some patients can be effectively managed with permissive hypercarbia in which there is elective hypoventilation
- Inhaled, volatile anesthetics are powerful bronchial smooth muscle relaxants
- Ketamine
- Assisted exhalation or lung massage

### **III. HYPOTHERMIA**

May increase interval that cardiac arrest and reduced blood flow during resuscitation can be tolerated. Severe hypothermia causes bradycardia and a slow ventilatory rate. It also can cause ventricular irritability

#### **KEY INTERVENTIONS**

- Prevent further heat loss due to evaporation from wet garments, cold environments and wind
- Cautiously transport, avoiding rough movement and excessive activity, which can cause VF. Do not delay urgently indicated procedures such as intubation or the introduction of intravascular catheters or a pacemaker.
- Patients with core temps <34 C can be rewarmed by internal active rearming.
- Because of the “after drop phenomenon” active external rearming should be performed to only truncal areas.

#### **BLS MODIFICATIONS DURING ARREST**

- Prevent further heat loss
- Avoid rough movement and excess activity
- Take up to 30-45 seconds to confirm pulslessness or apnea
- To not attempt active external rearming
- Continue efforts until patient is rewarmed
- Successful resuscitation without neurologic problems have been reported after 70 minutes of arrest followed by two hours of BLS before active rearming was started by cardiopulmonary bypass.
- Some patients may be hypothermic because they have cooled down after having a normothermic arrest. Do not attempt resuscitation under these circumstances.

#### **ALS MODIFICATIONS DURING ARREST**

- May have reduced response to pacemaker stimulation, defibrillation and cardioactive drugs, drugs may accumulate to toxic levels
- If core temp is <30C give a max of 3 shocks for VF or VT until patient is warmed.
- Needle electrodes are preferred for ECG monitoring
- Active internal rearming can be started in the field but must not delay transport

- E. Patients who have been hypothermic for hours may need large amounts of fluids during rewarming
- F. Cachectic, malnourished or alcoholic patients should receive Thiamine 100mg IV early during rewarming

Although hypothermia is a differential diagnosis, you must look beyond it for a reason for the exposure:

- Cardiac arrest may stop circulation before the victim cools
- Psychiatric disease, disorientation, drugs, or alcohol can impair thought
- Head trauma or other injury may immobilize the victim in the cold
- Alcohol produces vasodilation, which reduces the body's attempt to conserve heat.
- Victims of avalanches may have associated asphyxia and injury

MILD: 34 TO 36 C

- Lethargy
- Vasoconstriction
- Shivering
- Cold diuresis
- Increased Oxygen Demand

MODERATE: 30 to 34C

- Lethargy - stupor
- Dilated pupils
- Bradycardia, arrhythmias, hypotension
- Muscle rigidity, cessation of shivering
- Decreased O<sub>2</sub> demand

SEVERE: < 30 C

- Stupor - coma
- Dilated, non-reactive pupils
- Potential malignant arrhythmias
- Difficult to detect breathing
- Difficult to detect pulse, blood pressure

Standard gas thermometers do not read below 34C

ECG CHANGES

- Osborne or J waves
- T-wave inversion
- Prolonged PR, QRS and QT intervals
- Arrhythmias may include bradycardia, slow AF, VF or asystole

## AFTER DROP PHENOMENON

1. Initial active external rewarming leads to;
2. Peripheral vasodilation (BP drops)
3. Cold blood from dilated peripheral vessels carries high lactic acid levels to core vessels
4. Cold acidotic blood causes drop in core temperature
5. Temperature drop and acidosis provoke serious arrhythmias

## REWARMING METHOD BASED ON CORE TEMPERATURE

### 34C - 36C

- Passive rewarming (remove wet clothing, warm blankets)
- Active external rewarming (most convenient: IV solution bags heated in microwave, convective heating methods; (Bair blankets), radiant heat shield, convective heat (heating pads)

### 30C - 34C

- Passive rewarming (completely dried off; warm blankets)
- Active external rewarming to truncal areas only

### <30C

- Active internal rewarming

## IV. ANAPHYLAXIS

### DEFINITIONS

- *Anaphylaxis* is usually used for hypersensitivity reactions mediated by IgE
- *Anaphylactoid reactions* are similar to, but do not depend on hypersensitivity.
- The manifestations and management of anaphylaxis and anaphylactoid reactions are similar so that the distinction is so unimportant in relation to the treatment of an acute attack.

### SIGNS AND SYMPTOMS

Both may present clinically with angioedema, bronchospasm and hypotension.

- Some patients may die from acute irreversible asthma or laryngeal edema without having more generalized manifestations
- Other symptoms include urticaria, rhinitis, conjunctivitis, abdominal pain, vomiting, diarrhea and a sense of impending doom.
- The patient may appear either flushed or pale

- Cardiovascular collapse is the most common manifestation. It is caused by vasodilation and loss of plasma. Any cardiac dysfunction is due principally to hypotension and underlying disease, or epinephrine that has been administered.

#### Most common causes

- Insect stings
- Drugs
- Contrast Media
- Some Foods
- Peanut and tree nut allergies

#### KEY INTERVENTIONS

1. Recline victims in a position of comfort and transport to a hospital
2. Administer oxygen at high flow rates
3. Give epinephrine IM to all patients with clinical signs of shock, airway swelling, or definite breathing difficulties Dose: 300-500 ugs repeated after 5-10 minutes
4. Administer antihistamine IV
5. Provide inhaled salbutamol (not approved in the US) or epinephrine if airway obstruction is a major feature.
6. Give crystalloid if hypotension is severe and does not respond rapidly to Epinephrine
7. Raise the patient's legs as a useful interim measure
8. Inject high dose IV corticosteroids after severe attacks to help avert late sequelae, especially for asthmatics already on steroids
9. Keep under observation for at least 24 hours.

#### KEY INTERVENTIONS DURING ARREST

- Death is due to profound intravascular collapse
- Rapid volume expansion is an absolute requirements (as large as 4-6L over 15-30 minutes)
- High Dose Epinephrine IV
- Antihistamine IV
- Steroid Therapy
- Asystole/PEA
- Prolonged CPR - common error, failure to recognize cardiac arrest associated with anaphylaxis as imminently recoverable.

## **V. LIGHTNING STRIKE AND ELECTRICAL SHOCK**

Lightning injuries have a 30% mortality rate. Up to 70% of survivors sustain significant morbidity.

- The primary cause of death in lightning strike is cardiac arrest which may be due to primary F or more frequently, due to ventricular asystole
- Lightning acts as an instantaneous, massive direct current countershock, depolarizing the entire myocardium at once and producing asystole.
- In many cases cardiac automaticity may restore organized cardiac activity and sinus rhythm may return spontaneously.
- Respiratory arrest due to thoracic muscle spasm and suppression of the respiratory center may continue after return of circulation
- Unless ventilatory assistance is provided, hypoxic cardiac arrest may occur.

#### REVERSE TRIAGE

Patients who are most likely to die of lightning injury if no treatment is forthcoming are those who suffer immediate cardiac arrest, usually with asystole and thoracic muscle spasm.

- When multiple victims are struck simultaneously, usual triage priorities should be reversed. Rescuers should give the highest priority to patients in respiratory or cardiac arrest.
- Patients who do not suffer cardiac arrest have an excellent chance of recovery because subsequent arrest is uncommon.

#### **VI. CARDIAC ARREST ASSOCIATED WITH TRAUMA**

Most cardiac arrests with trauma are either PEA or asystole. Remember that VF may initiate or follow a traumatic incident

#### KEY INTERVENTIONS

Making the correct diagnosis and fixing the underlying problem may be the only chance of resuscitation.

#### BLS MODIFICATIONS DURING CPR

- Head tilt should not be undertaken in any patient with cervical injury suspected or clinically or due to mechanism of injury
- Presence of blood, vomitus, and other secretions in the mouth must be ascertained before basic ventilatory procedures are undertaken
- Compressions and ventilations may require greater care and attention to detail in the presence of fractures to the chest wall or sternum
- External bleeding must be stopped as a priority to conserve blood volume.

#### ALS MODIFICATIONS DURING ARREST

#### AIRWAY

1. Tracheal intubation must be done with inline cervical immobilization.
2. Nasotracheal intubation must be undertaken with caution or may be contraindicated if a fracture of the anterior base of the skull is present or likely
3. Cricothyrotomy may be required in the event of massive facial damage to open, maintain, and secure the airway

#### BREATHING

1. Possible presence of preexisting pneumothorax, constant attention must be paid to auscultation of the chest throughout resuscitation
2. Sucking chest wounds must be appropriately sealed
3. Once intubated, simultaneous ventilations and compressions may result in development of pneumothorax. Synchronized ventilations and compressions in a ration of 1:5 may be required with damaged thoracic cage.
4. Consider NG tube to decompress the stomach of the trauma patient.

#### CIRCULATION

1. Bleeding must be controlled
2. Exclude pericardial tamponade as a priority especially in PEA
3. Adequate and aggressive volume replacement

#### EMERGENCY MEDICATIONS

1. Before medications in the exposed trauma patient, consider hypothermia
2. Because of the probability of pulmonary bleeding, administration of ET drugs is unwise with chest injury
3. Maintenance meds being taken by the patient must be ascertained if possible because these can affect the outcome of the traumatic response.

Prognosis for traumatic arrest is poor, especially those from blunt injuries. If return of spontaneous circulation is not obtained on the scene, continued resuscitation and transport to a trauma center is probably futile.

#### **DEADLY DOZEN TRAUMATIC INJURIES**

**LETHAL 6** - you have <10 to 15 minutes to identify and treat

1. Airway obstruction
2. Tension pneumothorax
3. Penetrating cardiac injury
4. Open pneumothorax
5. Massive hemothorax
6. Flail chest

**HIDDEN 6** - not immediately life-threatening: maximum time allowed < 60 minutes

1. Thoracic aortic disruption
2. Tracheobronchial injury
3. Myocardial contusion
4. Diaphragmatic tear
5. Esophageal injury
6. Pulmonary contusion

## **VII. CARDIAC ARREST ASSOCIATED WITH PREGNANCY**

### CAUSES OF ARREST

A. Most commonly a maternal arrest is related to the changes and events faced at the time of delivery.

- Amniotic fluid embolism
- Eclampsia
- Drug toxicity (magnesium sulfate, epidural anesthetics)

B. Other causes are related to the physiologic changes associated with pregnancy itself.

- Congestive cardiomyopathy
- Aortic dissection
- Pulmonary Embolism
- Hemorrhage from pregnancy related pathology

C. Causes unrelated to the pregnancy.

### KEY INTERVENTIONS TO PREVENT ARREST

1. Place any distressed or compromised pregnant patient in the left lateral position.
2. Manually displace the uterus
3. Give 100% oxygen
4. Give fluid bolus
5. Immediately reevaluate any drugs being administered

### BLS MODIFICATIONS DURING ARREST

1. Relieve aortocaval compression by manually displacing the gravid uterus
2. Use a wedge to displace the uterus to the left side of the abdomen

### ALS MODIFICATIONS DURING ARREST

1. Consider rich variety of possible causes, i.e., magnesium sulfate toxicity, drug overdose, medication toxicity and iatrogenic events

## EMPTY THE UTERUS

With Mom in arrest, the blood supply to the fetus rapidly becomes hypoxic and acidotic. The blood flow returning the mother's heart becomes blocked by the uterus. You are going to lose both the mother and the infant unless you can get blood returning to the mother.

## WHEN STANDARD BLS AND ALS FAIL

If chance of fetal viability, consider immediate perimortem cesarean section (ideally within 5 minutes from the arrest to the delivery of the infant)

## LEARNING STATION FOUR TOXICOLOGIC EMERGENCIES

### BLS MODIFICATIONS DURING ARREST

1. Rescuer Safety
2. Move the patient to a toxin-free area before starting CPR
  - Be aware that you may be in a hazmat situations
  - Remove hazardous material from the victim's skin or clothing before starting CPR
  - Remove the patient from the source of inhalation or skin contamination
  - Secure the scene if others could be exposed and affected

### ALS MODIFICATIONS DURING ARREST

Same as BLS Modifications

### ECG FINDINGS AND TREATMENT FOR COMMON TOXINS AND DRUG OVERDOSES

Clinical Problem	ECG Findings	Treatment Approaches
<b>Cyclic Antidepressants and Major Tranquilizers</b>	<ul style="list-style-type: none"> <li>• ST and T wave changes</li> <li>• QRS wide</li> <li>• QT Long</li> <li>• R Axis deviation</li> <li>• BBB</li> <li>• AV Conduction blocks</li> <li>• Broad slurred QRS</li> <li>• Ventricular Arrhythmias</li> <li>• PEA</li> </ul>	<ul style="list-style-type: none"> <li>• Hypervent to pH=7.5</li> <li>• Benzodiazepine or Phenobarbital for seizures</li> <li>• Sodium Bicarbonate 1-5 mEq/kg over 1 to 2 minutes</li> <li>• Bicarb infusions 50-100 mEq at 150 to 200cc/hr</li> <li>• Magnesium 1-2 g IV bolus if unstable, over 1 to 5 minutes if stable</li> <li>• Pressors if needed (norepinephrine, epinephrine, high dose dopamine)</li> <li>• Charcoal hemoperfusion</li> <li>• Cardipulmonary bypass</li> </ul>
<b>Cocain</b>	<ul style="list-style-type: none"> <li>• Sinus Tachycardia</li> <li>• SVT</li> <li>• VF/VT</li> <li>• Cocaine induced AMI</li> </ul>	<ul style="list-style-type: none"> <li>• Delay thrombolytics until enzyme levels confirm MI</li> <li>• NTG or nitroprusside (0.1-5 ug/kg per minute IV) to lower BP and HR</li> <li>• Benzodiazepines for increased BP, HR</li> <li>• Phentolamine 1-10 ug titrated as a dilute solution over 2-3 minutes to lower BP</li> </ul>
Clinical Problem	ECG Findings	Treatment Approaches
<b>Cocaine (cont)</b>		<ul style="list-style-type: none"> <li>• Labetalol (5-20 mg IV)</li> </ul>

		<p>consider for severe HTN</p> <p><b>CARDIAC ARREST</b></p> <ul style="list-style-type: none"> <li>• Use Lido cautiously</li> <li>• Space EPI to q 5-10 mins</li> <li>• Consider propranolol or labetalol</li> </ul>
<b>Beta Blockers</b>	<ul style="list-style-type: none"> <li>• Bradycardia</li> <li>• AV Block</li> <li>• Wide QRS</li> <li>• Peaked T waves</li> <li>• ST Changes</li> <li>• Cardiac deterioration leading to PEA</li> </ul>	<ul style="list-style-type: none"> <li>• Saline bolus</li> <li>• Atropine</li> <li>• Epinephrine</li> <li>• Glucagon 1-5mg IV over 1 minute</li> <li>• Dopamine</li> <li>• Calcium chloride (5-20 ml IV)</li> <li>• Pacing</li> <li>• Pressors</li> </ul>
<b>Calcium Channel Blockers</b>	<ul style="list-style-type: none"> <li>• Sinus bradycardia</li> <li>• QRS widening</li> <li>• QT Long</li> <li>• Heart Blocks</li> <li>• PEA</li> </ul>	<ul style="list-style-type: none"> <li>• Saline bolus</li> <li>• Calcium chloride</li> <li>• Glucagon</li> <li>• Epinephrine</li> <li>• Dopamine</li> <li>• Repeat Calcium Chloride</li> <li>• Dobutamine, norepinephrine and/or isoproterenol</li> <li>• Pacing</li> <li>• Cardiopulmonary bypass</li> </ul>
<b>Digitalis Ingestion</b>	<ul style="list-style-type: none"> <li>• Many arrhythmias are possible</li> </ul>	<ul style="list-style-type: none"> <li>• Volume replacement</li> <li>• Potassium replacement</li> <li>• Magnesium replacement</li> <li>• Digoxin specific antibodies (Digibind)</li> </ul>
<b>Narcotic Overdose</b>	<ul style="list-style-type: none"> <li>• No specific ECG effects</li> <li>•</li> </ul>	<ul style="list-style-type: none"> <li>• Standard ACLS plus</li> <li>• Naloxone 2mg IV repeat q 2-5 minutes</li> <li>• Naloxone infusion 0.8-1.0 mg/hr and titrated to effect (Mix 8-10 mg in 1000 cc D5W)</li> </ul>
<b>Benzodiazepine Ingestion</b>	<ul style="list-style-type: none"> <li>• No specific ECG effects</li> <li>• Bradycardia then asystole following respiratory depression</li> </ul>	<ul style="list-style-type: none"> <li>• Romazicon 0.2mg IV over 15 seconds</li> <li>• Repeat at 1 minute intervals</li> </ul>