



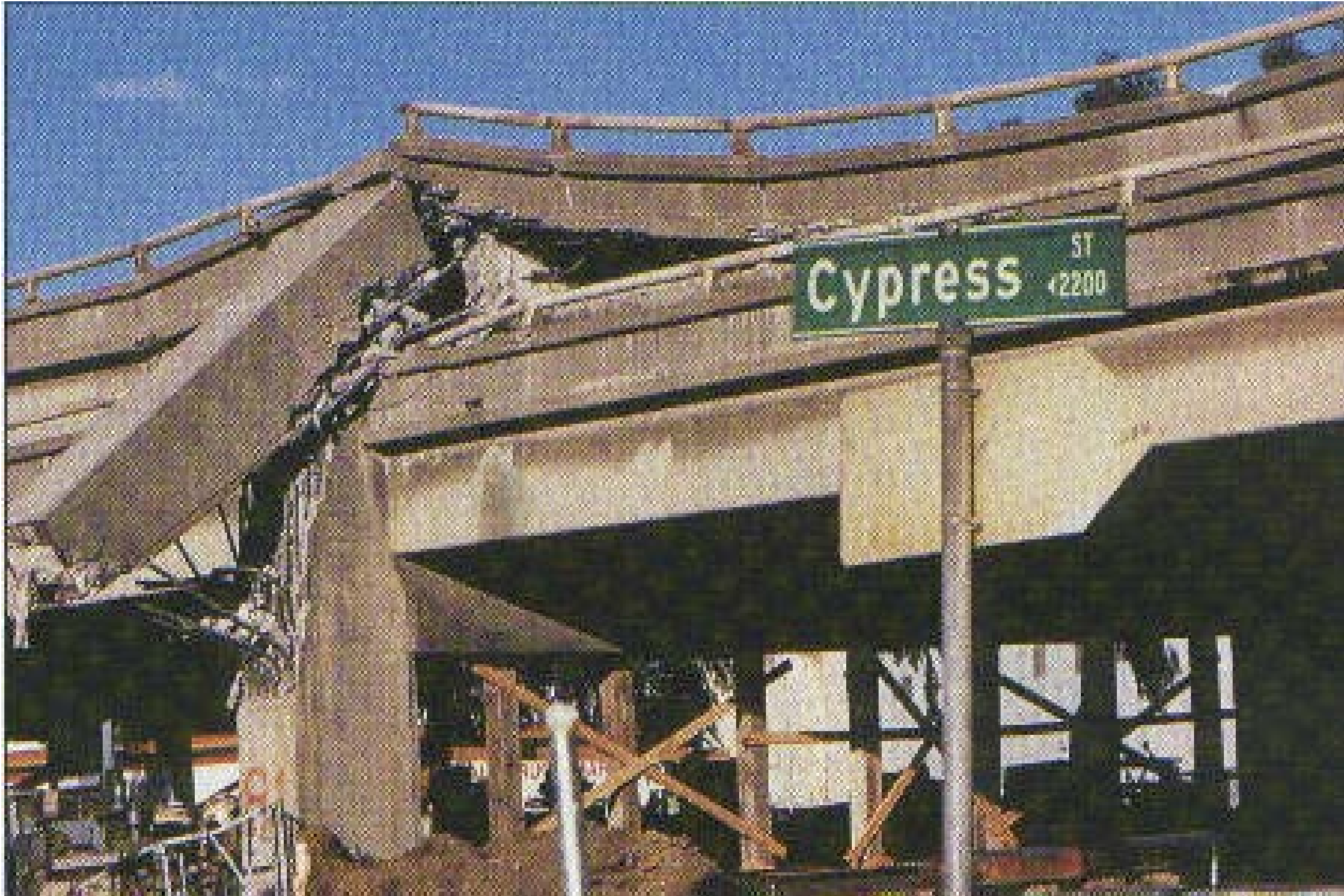
Crush Syndrome

Joe Holley, MD FACEP
EMS Medical Director
Memphis Fire Department
Shelby County Fire Department
TNTF1 FEMA US&R



Crush Syndrome

- The systemic manifestations caused by release of toxic products of crushed muscle tissue which occurs when a victim has sustained a **prolonged compressive force** on a muscle mass.



Northridge Earthquake







Crush Syndrome

- It is a disease process that develops within the body at a cellular level
- Symptoms are not immediate
- When symptoms exist, the disease is far advanced and the patient can die suddenly even with aggressive treatment.



■ Crush injury and compartment syndrome are different processes with a common pathophysiology

■ Crush injury occurs in 2 phases

- Mechanical cell disruption
- Ischemia

■ Crush syndrome associated with increased external pressure to a body part or region

■ Compartment syndrome results from increased pressure within a muscle compartment



Crush Syndrome

- Deterioration of the untreated patient is the rule, not the exception.
- Armenian Earthquake

“--they heard someone in the rubble and lifted the panel that was crushing them--the person died.”

Medical Provider

Big crush





Crush Syndrome

- Crush syndrome is common in immobile victims trapped within collapsed structures
- The great irony
 - Patients survive for days despite being trapped
 - Patients may die suddenly within minutes to hours after successful extrication



Crush Syndrome

- **Early recognition is imperative!**
 - Post-extrication medical deterioration and death occur from potentially treatable mechanisms
 - Science suggests that patients survive if they are treated early and aggressively “in the rubble”
 - Survival is also associated with aggressive hospital care and early dialysis



Crush Syndrome

- Why crush syndrome occurs
 - Muscle is vulnerable to sustained pressure
 - Compression may be caused by debris or the patient's own weight on a hard surface
 - Time-pressure relationship
 - Time
 - Can develop in 1 hour if the compression is severe
 - Pressure
 - Amount, distribution and anatomical structures involved



Crush Syndrome

- What parts of the body are susceptible?
 - Lower extremities (most common)
 - Buttocks
 - Upper extremities and thorax



Crush Syndrome

- Why do patients die?
 - High serum potassium levels
 - Ventricular fibrillation
 - Metabolic acidosis
 - Hypovolemic shock (blood & plasma loss)
 - Renal Failure



Crush Syndrome

■ Pathophysiology

- Tissue is compressed
- Blood flow is decreased
- Cells die
- Toxins release
 - Hyperkalemia
 - Hypocalcaemia
 - Acidosis (local and systemic)
 - DIC (thromboplastin release)
 - Renal failure:
 - Myoglobinuria



Crush Syndrome

- Crush syndrome effects are not seen until release of the compressive force (extrication)
- Blood flow is restored and toxins are released into the central circulation
- The trapped patient who appeared “stable” suddenly deteriorates



Crush Syndrome

- If left untreated...
 - Cardiac arrhythmia
 - High serum potassium levels
 - Kidney failure
 - Myoglobin and uric acid (from crushed muscle)



Clinical Findings

Pre-release of entrapment

Painless crushed extremity
(hypesthesia/anesthesia)

Distal pulses may or may not be present



Post-release of entrapment

Agitation

Continued hypesthesia/anesthesia

Severe pain in crushed extremity

Muscle function decreased/absent

Progressive swelling of injured area

Systemic problems

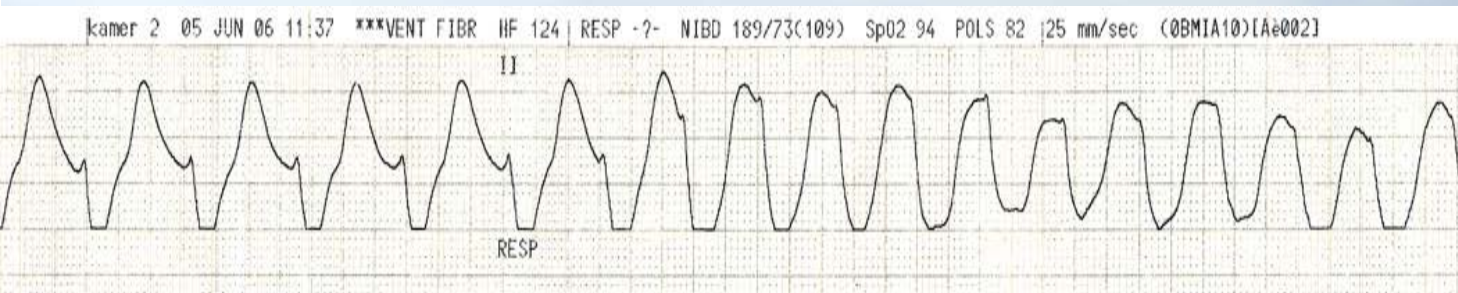


Patient Assessment

Hyperkalemia and Hypocalcemia

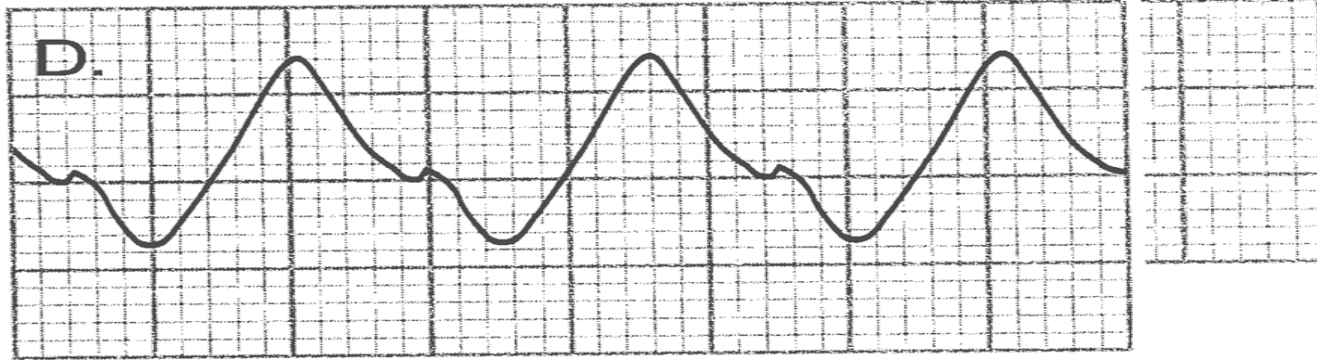
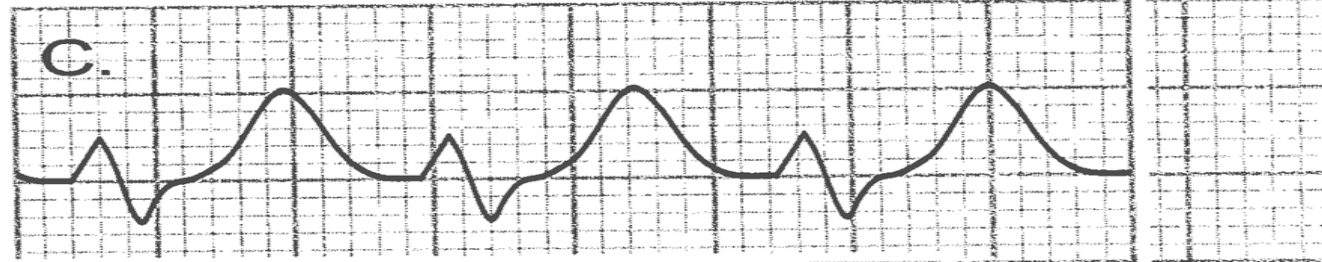
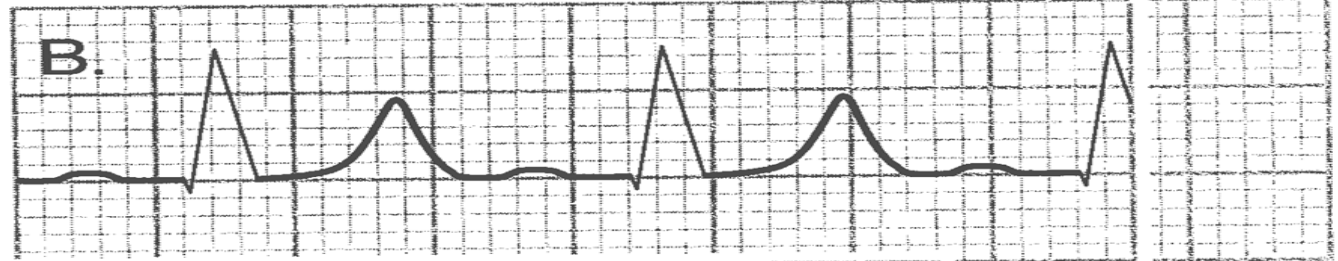
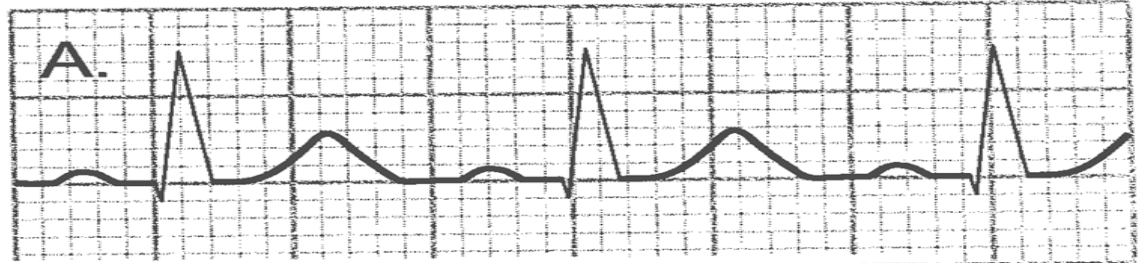
Rhabdomyolysis

Pulmonary Injury

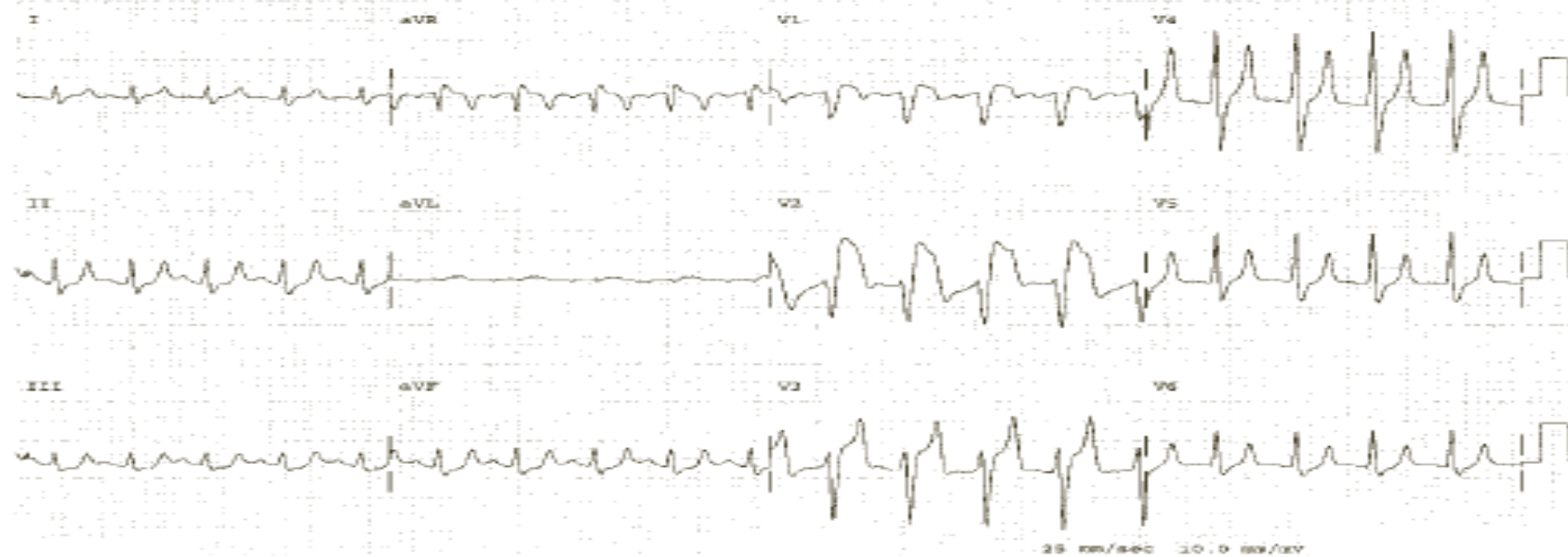
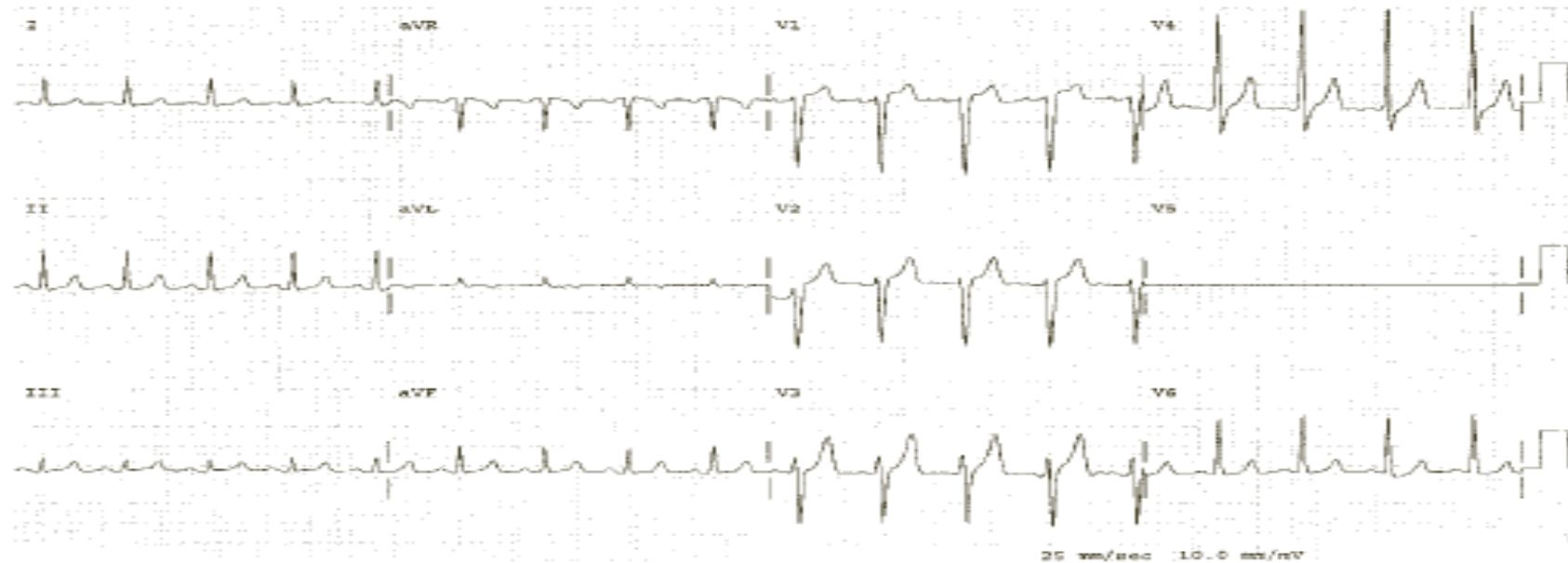


Serum potassium levels

- A. normal (3.5 - 5.0 mEq/L)
- B. about 7.0 mEq/L
- C. 8.0 - 9.0 mEq/L
- D. >10.0 mEq/L



Changes in the ECG in lead II
caused by hyperkalemia

A**B**





Rhabdo

Myoglobin

Solubility in urine: 50 mg myoglobin/ml urine

Urine pH	% Precipitated
8.5 to 7.5	0%
6.5	4%
5.5	23%
5.0	46%
<5.0	73%

Crush Syndrome

- Treatment in the Field
 - *Do not release the victim until treatment has been initiated*





Crush Syndrome

- Treatment
 - IV of Normal Saline Solution
 - Sodium Bicarbonate
 - Dextrose
 - Insulin
 - Continuous nebulized Albuterol
 - Pain control and sedation
 - Other patient specific medications



Sample Protocol

- **Infuse 0.9% Normal Saline or D5W ½ Normal Saline as directed.**
- **When possible, obtain urine and test for the presence of hemoglobin / myoglobin. If necessary, insert a foley catheter.**
- **Administer IV fluids to correct volume deficit:**
 - **Administer 1L 0.9% NS bolus.**
 - **Administer sodium bicarbonate 44mEq (1 amp), IV or Saline Lock bolus.**
 - **Mix sodium bicarbonate 88mEq in 1L D5W and administer at a rate of 1L per hour.**



Additional Protocols

- If the patient is showing early signs of renal failure/insufficiency including “hematuria”/myoglobinuria and has documented urine output:
 - Administer Mannitol 0.5-1 g/kg (maximum dose 12.5g) in 250 cc of D5W ½ Normal Saline to be infused at a rate of 125 cc per hour *or*
 - Lasix 1-2 mg/kg, IV or Saline Lock bolus.
 - NOTE: ANURIC PATIENTS SHOULD NOT BE GIVEN MANNITOL
- For extended operations, foley insertion may be required to monitor urinary output and for BBP precautions. Maintain urinary output of 3cc/kg per hour and a pH ≥ 7.65
- Obtain serum glucose level with the i-STAT or glucometer.



Questions?

joeholleymd@gmail.com