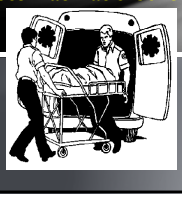
The Crushing Truth

AKA

Crush Injury Syndrome
Compartment Syndrome
Acute Traumatic Ischemia



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Crushing Mechanisms

- **Building & structure collapse**
  - Earthquakes
  - Explosions
- **Motor vehicle accidents**
  - Entrapment
  - Direct impact
- **Lack of spontaneous movement**
  - Deep sleep
  - Coma

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Crushing Force

- External crushing force applied to body

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Crush Syndrome
Vascular Response

- Crushing pressure sustained
  - Body adapts to decreased vascular space
- Crushing pressures released
- Blood flows into tissues (re-perfusion)
  - redistributive hypovolemia
  - wounds in crushed limb may begin to bleed
- Chemicals & toxins enter systemic circulation

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Commonalities?

3 mechanisms for cellular death

- Immediate cell disruption = lysis
  - Histamine causes vasodilation & increased capillary permeability
- Direct pressure on muscle cells = ischemia
- Vascular compromise = anaerobic metabolism which can occur in as little as 4 hours and yields **Lactic Acidosis**
  - Lactic acid, uric acid, Potassium, Phosphates, Myoglobin

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Crush Syndrome
Systemic Response

- Cardiovascular shock
  - Third spacing - fluids leave damaged vascular space
- Blood chemistry is altered
  - Decreased pH (increased acidity)
    - Increase of Lactic acid, Uric acid
  - Hyperkalemia (increased K^+)
  - Hyperphosphatemia
  - Hypocalcemia
  - Increased myoglobin in blood

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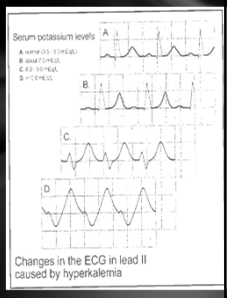
Crush Syndrome
Cardiac Response

- Hyperkalemia
- ECG changes
  - peaked T waves
  - widened QRS complex
  - disappearing/absent P wave
- Cardiac dysrhythmias
  - heart blocks
  - V-tach
  - V-fib
  - asystole



Changes in the ECG in lead II caused by hyperkalemia

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Crush Syndrome
Renal Response

- Myoglobin
  - Small amounts normally filtered out through kidneys
  - Acidic environment causes myoglobin to precipitate out in kidney tubules
    - Urine becomes reddish-brown, cola colored
  - Causes kidney failure



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Objective Assessment


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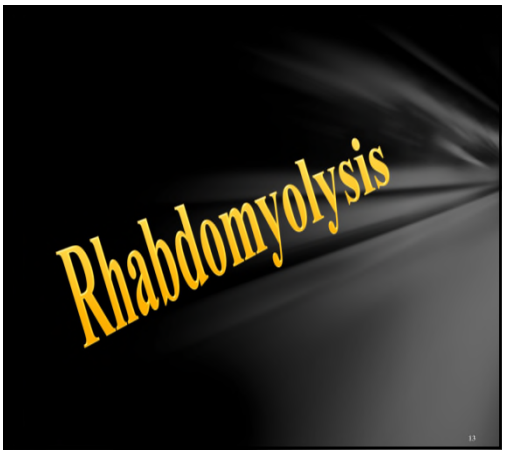

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Although rhabdomyolysis is occasionally the chief problem, it is more commonly only one of several interrelated diagnoses in a critically ill patient

Understanding the pathophysiology of rhabdomyolysis indicates why it is associated with more than 100 seemingly unrelated disorders including "Crush"

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Definition

Rhabdo

- Means striated (as in striated or skeletal muscle)

Myo

- Refers to muscle

Lysis

- Is breakdown

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Therefore, rhabdomyolysis

is a dissolution of skeletal muscles that produces a nonspecific clinical syndrome that causes extravasation of toxic intracellular contents from the myocytes into the circulatory system

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Pathophysiology of Rhabdo

Muscle cell walls get damaged

Cell contents leak out -

- Myoglobin
- Potassium
- Creatine phosphokinase (CPK)

Overall you have

- Electrolyte disturbances
- Hypovolemia
- Metabolic acidosis
- Coagulopathies
- Myoglobinuric renal failure

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Crush Injury/Syndrome Management

- "Treatment in the rubble"
- Treatment should be started before pressure is released
- Treatment may be hampered by the multi-causality incident and confined space of crush injury situations
- Attempt to coordinate release of pressures with extrication specialists

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Crush Injury/Syndrome Basic Treatment

- AcBCDEs
- Start with hydrating your Pt.
  - Hydrate both adult and pediatric patients with 20ml/kg of NS

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Assessment of Crush Victims - Post Rescue

- Trauma may not be obvious
  - Keep limb(s) at heart level
  - Use non-compressive splints
- Paralysis/weakness of affected limbs
  - Mimics spinal cord injury
  - From neuropathies associated with the pressure
  - Ascending paralysis /paresis from the hyperKalemia
- Hypotension and tachycardia
  - Redistibutive hypovolemia
- Tachypnea
  - Metabolic acidosis

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Subjective Assessment

Only 50% of people with rhabdo complain of muscle pain or weakness

History leading to muscle destruction

- MOI

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Assessment of Crush Victims - Post Rescue

- The 6 Ps of a neuro vascular assessment in a musculo-skeletal injury
- Paralysis
- Paresthesia
- Pain
- Pulselessness
- Pallor
- Polar

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Compartment Syndrome

Exists when circulation and function of tissues within a closed space are compromised by increased pressure within that space

This Hurts!

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Compartment Syndrome

Obvious how it creates rhabdo

How rhabdo creates it:

- Dead tissue creates an osmotic gradient that sucks fluid into the interstitial space
- The increased fluid creates increased pressure
- The cycle begins

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Compartment Syndrome Pathophysiology

- Fluids re-perfuse damaged areas
- Muscle tissues become swollen inside fibrous sheaths
- Increased swelling results in increased pressure
- Capillaries become occluded because of increased pressure
- Venous pressure increases, further decreasing perfusion pressure
- Arteriolar vasospasm > tissue ischemia
- Progressive increases in compartment pressure

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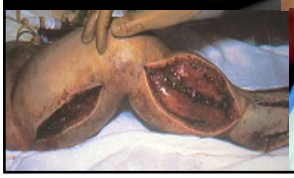
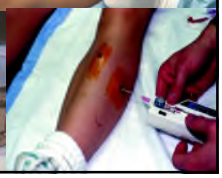
Compartment Syndrome

Pressure measurement

- Less than 15 mmHg normal
- 15 – 30 mmHg observe
- >30 mmHg – fasciotomy





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Compartment Syndrome

- Usually happens to large muscle groups such as quadriceps & gluteal muscles

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Compartment Syndrome Signs and Symptoms

- General findings
  - Pallor
  - Polar
  - Paralysis
  - Pulselessness
  - Pain on passive stretch
  - Paresthesia
- Most significant findings
  - Pain on passive stretch
  - Sensory impairment

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Compartment Syndrome

- Early recognition of condition is key
- Appreciate that compartment syndrome is a pathway to Rhabdomyolysis

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Renal Failure

Most lethal complication

Rhabdo causes about 10% (7-15%) of all acute renal failure

33% of people with rhabdo get renal failure

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Renal Failure

Causes

- Hypovolemia
- Obstruction of outflow
- Vascular injury
- Most common cause is acute tubular necrosis

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Renal Failure

- Myoglobin dissociates into globin and ferrihemate in a pH less than 5.6
- Ferrihemate is a toxic substance

They gave myoglobin infusions to rabbits, as long as they kept urine pH over 6, no renal failure occurred

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Treatment

Flush Ki

If you can't establish IV's, consider placing tourniquets on extremities before or immediately after release

Large volume[illegible] possible

Start out with 500 cc/hr

Maintain a urine output 200 - 300 ml/hr

That may be 30 liters in first 24 hours

Mannitol or Furosemide

Dialysis

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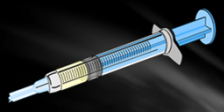
Alkalinize Urine



Sodium bicarb 1 amp in 1 liter 1/2 normal saline running concurrently at 100-250 cc/hr

Or

1 amp of bicarb in every second or third IV bag

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Remember

3 mechanisms for cellular death

- Immediate cell disruption = lysis which releases K+
- Direct pressure on muscle cells = ischemia from Vascular compromise = anaerobic metabolism

Lactic Acidosis

- The acidosis promotes the release of K+ from inside the cell.....

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Hyperkalemia

K+ conc inside cell is 100 mEq/kg

20 - 40% of patients with rhabdo get K+ greater than 5.5

- (normal is 3.5-5.2 meq/L)

As renal function diminishes, K+ rises

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Hyperkalemia

Symptoms
- Weakness
- Ascending paralysis
- Respiratory failure
- EKG changes
  - Peaked T waves
  - Flattened P waves
  - Prolonged PR interval
  - Widened QRS complex
  - Idioventricular rhythm
  - VF and cardiac arrest

Mild hyperkalemia
- 5.5-6.4 peaked, narrow T waves

Moderate hyperkalemia
- 6.5-7.0 slowed conduction yielding a widened QRS complex, peaked T waves

Severe hyperkalemia
- >7.0 sinus bradycardia, sinus arrest, AV blocks

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Potas

Hyperkalemia

Causes
- Acute or chronic renal failure
- Burns, crush injuries
- Severe infections
- Excessive use of potassium salts
- A shift of potassium ions into the extra cellular fluid
  - Succinylcholine, massive digitalis overdose

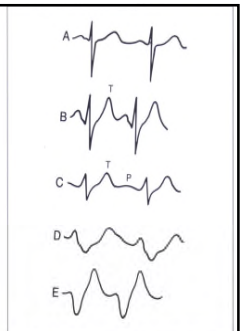

Figure 2. Progressive electrocardiographic changes seen on the ECG from normal levels of potassium to severe hyperkalemia. (A) represents the normal ECG (serum potassium level of 4.0 mEq/L); (B) shows normal hyperkalemia (serum potassium levels of 5.5-6.4 mEq/L; (C-D) represent moderate hyperkalemia (serum potassium levels of 6.5-7.0 mEq/L; and (E) shows severe hyperkalemia (serum potassium levels greater than 8.0 mEq/L). Drawings courtesy of Gil Gardner, Letterman Army Medical Center, San Francisco.

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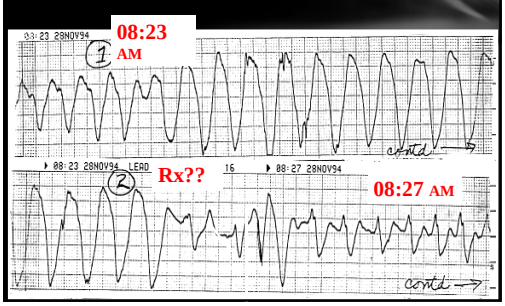
Case 1: Progression

At 8:23 AM the nurse states, "You'd better look at this rhythm strip."
Patient alert, talking; weak, BP = 80/55 mm Hg, RR 24/min
What is your first action? What medications were given by 8:27?



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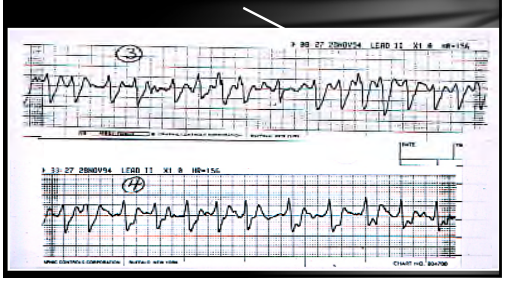
Case 1 Progression: 4 minutes later (8:27 AM)

What medications should be administered at this time?



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Treatment of Mild Hyperkalemia

Mild elevation (5-6 mEq/l)

Remove K^+ from the body

- Diuretics
  - Lasix 1mg/kg IV slowly
- Resins
  - Kayexalate 15-30 g in 50-100 cc of 20% sorbitol orally or retention enema
- Dialysis +/-

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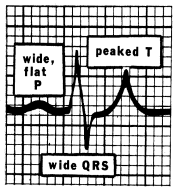
Treatment of Moderate Hyperkalemia

Moderate elevation (6-7 mEq/l)

Remove and shift K^+ intracellularly

- Sodium bicarbonate
  - 50 meq IV over 5 min
- Glucose + Insulin
  - Mix 50 g glucose with 10 u regular insulin and give over 15-30 min
- NPPB Albuterol
  - 10-20 mg nebulized over 15 min



Moderate

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Treatment of Severe Hyperkalemia

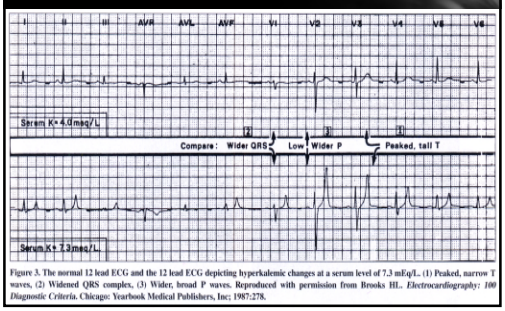

Figure 3. The normal 12 lead ECG and the 12 lead ECG depicting hyperkalemic changes at a serum level of 7.3 mEq/L. (1) Peaked, narrow T waves, (2) Widened QRS complex, (3) Wider, broad P waves. Reproduced with permission from Brooks HL. *Electrocardiography: 100 Diagnostic Criteria*. Chicago: Yearbook Medical Publishers, Inc; 1987:278.

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Treatment of Severe Hyperkalemia

Severe elevation (>7 mEq/l with toxic EKG changes

Protect the heart

- CaCl 10% 5-10 cc IV over 2-5 min
  - Protects the myocardial cell membrane lowering the risk of VF

Remove and shift K^+ intracellularly

- Sodium bicarbonate
  - 50 meq IV over 5 min
- Glucose + insulin
  - Mix 50 g glucose with 10 units regular insulin and give over 15-30 min
- NPPB Albuterol
  - 10-20 mg nebulized over 15 min (4-8 unit doses)
- Diuresis
- Kayexalate enemas
- Dialysis

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CaCl

Sodium Bicarbonate

Albuterol

5 Treatments

Insulin and Glucose

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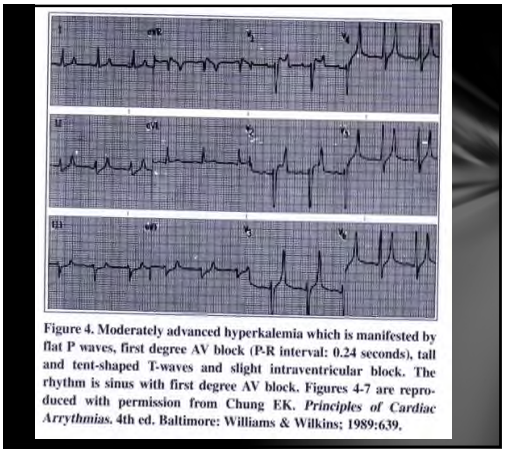

Figure 4. Moderately advanced hyperkalemia which is manifested by flat P waves, first degree AV block (P-R interval: 0.24 seconds), tall and tent-shaped T-waves and slight intraventricular block. The rhythm is sinus with first degree AV block. Figures 4-7 are reproduced with permission from Chung EK. *Principles of Cardiac Arrythmias*. 4th ed. Baltimore: Williams & Wilkins; 1989:639.

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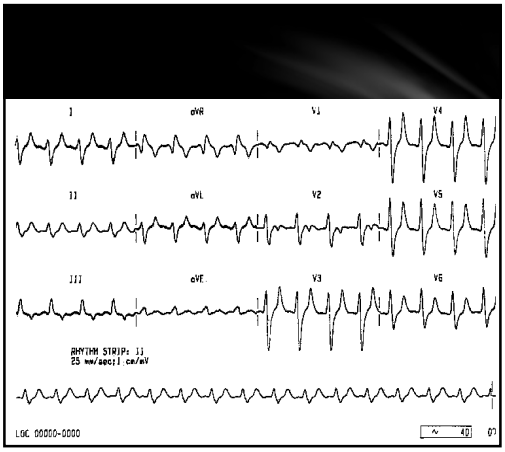

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Treating Hyperkalemia

Calcium gluconate or chloride to protect the heart

Dextrose and insulin infusion to temporarily drive potassium into the cells

Sodium bicarbonate

Albuterol NPPB Txs

Kaexelate oral or rectal to bind and excrete potassium

Hemodialysis

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Prognosis

Overall survival is 77%

- As long as there is…..
  - Early recognition
  - Early and aggressive treatment
  - Treatment of underlying cause

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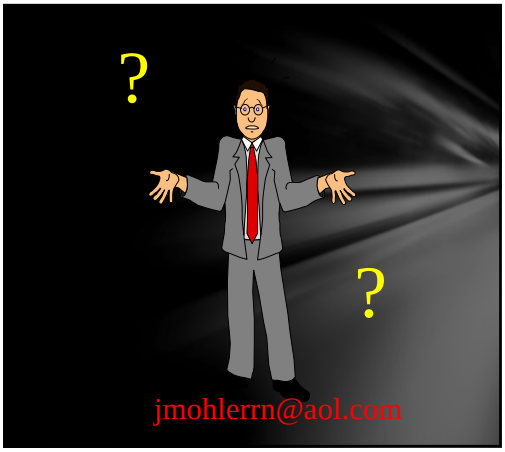

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