The Seriously Injured or Threatened Limb

LIMB vs LIFE

Threatening Injuries

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The threatened limb: soft tissue, bone, nerves, blood vessels

Causes

• Penetrating injuries
  – GSW
  – Stab
  – Shotgun
  – Iatrogenic

• Blunt injuries
  – MVA
  – Sports
  – Fall
  – Iatrogenic
Definitions:

1. Crush syndrome
2. Compartment syndrome
3. Rhabdomyolysis
4. Reperfusion injury
Q 1

Discuss the differences between Compartment Syndrome (CS) and Crush Injury (CI) as per the definitions.
Crush Syndrome:
The systemic manifestations of muscle injury after direct trauma or ischaemic reperfusion.
The local manifestations of nerve and muscle ischaemia due to increased pressure within the osseo-fascial compartments.
Q 2

What does the concept "CRUSH SYNDROME" signify?
Clinical Scenario: Crush and Vascular injuries
Q 3

Crush Injury develops because of.....?

Aetiology ?
Pathophysiology ?
Aetiology of CI

• Limb compressed between 2 hard surfaces
• Vascular supply is impaired
• Vulnerable tissues undergo necrosis

• MVA
• PVA
• Industrial accidents
• Earthquakes
• Building collapses
• Landslides
• Mine cave-ins
• Acts of war
Pathophysiology of CI

- Degree of compression
- Duration of compression
- Effective perfusion is precluded
- Pressure is *external*
- Can involve multiple injured structures – most NB disrupted / occluded blood vessels
- Consequences: prolonged ischaemia + muscle necrosis + reperfusion
- Third space fluid losses… hypovolaemia + shock
- Potassium + Calcium + Myoglobin + Lactic acid from damaged muscle … depress cardiac conduction and renal function
- Metabolic acidosis
Q 4

What does the concept “COMPARTMENT SYNDROME” signify?
Clinical Scenario: Compartment Syndrome

- Example: Derived from explosive munitions
  - IEDs
  - Grenades
  - Homicide bombers
  - Car bombers

MOST COMMON: LOWER LIMB FRACTURES
(70% of CS associated with #, 50% of which are caused by tibia #’s)
Compartment Syndrome Concept

- A compartment syndrome exists when the increased pressure in a closed anatomic space threatens the viability of the surrounding tissue.

- Anatomical regions - 40 Examples:
  - Limbs
  - Cranial vault
  - Pericardial sac
  - Pleural cavity (Tension pneumothorax)
  - Abdominal cavity
  - Gall bladder (Acalculous cholecystitis)
  - Bowel (NOBS with nutrition)
**Concept: Perfusion Pressure**

\[ \text{APP} = \text{MAP} - \text{IAP} \]

- Abdominal perfusion pressure reflects actual gut perfusion better than IAP alone.
- Optimizing APP to $> 60$ mm Hg should probably be the primary endpoint

Cheatham 2000
Optimizing APP reduced incidence of
- ACS - 64% versus 48%
- Death - 44% versus 28%

\[ \text{CPP} = \text{MAP} - \text{ICP} \]

*Compartment Syndrome* = MAP - Intra-Comp P

*Perfusion Pressure as a primary endpoint in resuscitation!!!*

*Endpoints optimize systemic perfusion!!!*
Apparatus for measuring compartment pressure

Figure 1 Apparatus for measuring compartment pressure.
Fig. 1. (A) The Stryker device for measurement of intracompartmental pressure. (B) The Stryker device being inserted into the calf compartment under local anesthesia.
Q 5

Compartment Syndrome develops because of..... ?

Aetiology ?
Pathophysiology ?
Aetiology of CS

- Fracture of a long bone (even open #’s)
- Compressive dressings and casts
- Intravenous + Intraosseous fluid infiltration
- Snakebite
- Burns
- Nephrotic syndrome
- Diabetes
- Drug overdose
- Injections
- Medications – pressors, anticoagulants, platelet inhibitors
Pathophysiology of CS

• Acute CS
  – Acute surgical emergency = ICP
  – Increase in contents of compartment (oedema, haemorrhage, external pressure) = internal
  – At critical threshold compressible structures (muscles, nerves, blood vessels) is compressed
  – Lymphatics + venules = no venous outflow = >> oedema
  – Pressure sufficient to occlude arterioles = ends perfusion of muscles + nerves
  – 4 Hours warm ischaemia time / >6 hours still recovery but with some necrosis / >8 hours muscle + nerves are necrotic = deformed, insensate, nonfunctional limb = Volkman’s contracture

• Chronic CS
  – Distance runners
Clinical presentation

- Severe limb injury...High index of suspicion
- Paraesthesia + paralysis appear late
- Rarely pulse deficit (CS ICP < SAP)
- Swollen, painful calf – woody feeling (rigor mortis)
  - Burning quality
  - Delayed onset
  - Increasing severity
  - Pain on passive stretch (dorsiflexion of toes + foot)
- Direct calf pressure measurement
  - Measure in ALL 4 calf compartments
  - ICP > 30 mm Hg
  - $\Delta p = DAP - Comp \ P = < 20$ mm Hg = MUST do fasciotomy

High index of suspicion!!!
Symposium in traumatology

• The threatened limb vs
• Life threatening limb injuries

DIAGNOSIS IS BASED ON A HIGH INDEX OF CLINICAL SUSPICION……..think!!!
Q 6
The threatened limb...
What are the clinical findings?
The threatened limb
Clinical findings

- Pain
- Parasthesia
- Paralysis
- Poikilothermia
- Pallor
- Pulselessness

The ischaemic limb
NOT Compartment Syndrome
Q 7

The threatened limb

Is there a Classification for ischaemic or injured limbs?
# Rutherford Classification Scheme for Acute Limb Ischemia

- From the Society of Vascular Surgery/International Society of Cardiovascular Surgery (Rutherford et al, 1997)

<table>
<thead>
<tr>
<th>Class</th>
<th>Category</th>
<th>Prognosis</th>
<th>Sensory loss</th>
<th>Muscle weakness</th>
<th>Arterial Doppler</th>
<th>Venous Doppler</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Viable</td>
<td>No immediate limb threat</td>
<td>None</td>
<td>None</td>
<td>Audible</td>
<td>Audible</td>
</tr>
<tr>
<td>IIA</td>
<td>Threatened: marginal</td>
<td>Salvageable if treated promptly</td>
<td>Minimal-none</td>
<td>None</td>
<td>+/- Audible</td>
<td>Audible</td>
</tr>
<tr>
<td>IIB</td>
<td>Threatened: Immediate</td>
<td>Salvageable if treated immediately</td>
<td>More than just toes</td>
<td>Mild-moderate</td>
<td>Rare audible</td>
<td>Audible</td>
</tr>
<tr>
<td>III</td>
<td>Irreversible</td>
<td>Limb loss or permanent damage</td>
<td>Profound</td>
<td>Profound</td>
<td>None</td>
<td>None</td>
</tr>
</tbody>
</table>
Q 8

The threatened limb
How can we protect the tissue under these circumstances from further ischaemia and poor perfusion?
The threatened limb

Fasciotomy
The threatened limb

Fasciotomy

ADJUNCTS: Hyperbaric Oxygen therapy
Topical negative pressure therapy
Q 9

Limb vs life threatening injuries

When and Why do limb injuries become life threatening conditions?
Discuss this concept.
Limb vs life threatening injuries

1. Acute, severe injuries = the local effect of the trauma

2. Acute disturbance of homeostasis = the systemic consequences of serious injury

• Life before limb

• Basic ABC principles
Life threatening limb injuries

- Disrupt the A, B, C’s

- Cause physiological instability
  - ACIDOSIS
  - HYPOTHERMIA
  - COAGULOPATHY

Hypovolaemic
Traumatic
Shock

The triad of death
Limb vs life threatening injuries

1. Acute, severe injuries

- Massive blood loss
- Uncontrolled or continuing haemorrhage
- Multiple or massive soft tissue and bony injuries
- Co-morbid conditions
- Extremes of age
Limb vs life threatening injuries

2. Acute disturbance of homeostasis

- A,B,C’s unstable
  - Hypothermia
  - Metabolic Acidosis
  - Coagulopathy

- Severe inflammatory response syndrome
- Multiple organ dysfunction / failure
Q 10

Can you think of examples of life threatening limb injuries?
Serious soft tissue, bone, nerve and vascular injuries
Physical findings in vascular injury

• Hard findings
  – Pulsatile / rapidly enlarging haematoma
  – Pulsatile bleeding from wound
  – Bruit or thrill when palpating the pulse
  – Any of the 5 P’s

• Soft findings
  – Diminished pulse in comparison to contralateral side
  – Delayed capillary refill
  – Isolated peripheral nerve injury
  – Nonpulsatile stable haematoma
Q 11

How should life threatening limb injuries be managed?
Immediate management of life threatening limb injuries

- Damage control surgery
  - Criteria
    - Acidosis: pH < 7.3
    - Temperature < 35 C
    - Coagulopathy

- Damage control surgery in vascular injuries is challenging
Limb vs life threatening injuries

Damage Control procedures:

• Lateral suture
• Shunts
• Ligation
• Primary amputation
Limb vs life threatening injuries
Open Fractures

• Any wound in the vicinity of a fracture……….
• Gustilo and Anderson classification – grade open #’s according to the degree of damage to soft tissue, contamination and presence of arterial injury
• Tetanus toxoid
• Analgesia (immobilisation)
• Prophylactic vs therapeutic antibiotic therapy
• Early debridement
Q 12

Are there other indications for fasciotomy?
The threatened limb
Other indications for fasciotomy

- **Prophylactic**
  - Delayed revascularisation >6 hours
  - Extensive soft tissue damage
  - Arterial and venous injuries
  - Inability to assess patient adequately, e.g. head/spinal injuries

- **Therapeutic**
  - Compartment syndrome

- **Diagnostic**
  - Non viable limb?
Limb Salvage Scoring Systems

- MESS = Mangled Extremity Severity Score
- MESI = Mangled Extremity Syndrome Index
- PSI = Predictive Salvage Index
- LSI = Limb Salvage Index
Dead tissue kills people!

• If limb salvage is not possible, recognize and acknowledge it asap
• Perform “source control” = amputation
• Sacrifice limb to save life

Patients who present beyond the 6-hour window of warm ischaemia with an insensate “dead” limb do not benefit from fasciotomy and will suffer reperfusion injury and sepsis
1. Crush syndrome
2. Compartment syndrome
3. Rhabdomyolysis
4. Reperfusion injury

Definitions:

TRAUMA

CS or CI

Rhabdomyolysis

Reperfusion

Renal (Organ) Dysfunction
Q 13

What is rhabdomyolysis?
Rhabdomyolysis is a syndrome characterised by muscle necrosis and the release of intra-cellular muscle constituents into the circulation.

**Definition:**

Rhabdomyolysis is a syndrome characterised by muscle necrosis and the release of intra-cellular muscle constituents into the circulation.

**Spectrum**

- Asymptomatic ↑ of muscle enzymes
- Life threatening cases
  - extreme enzyme ↑
  - electrolyte imbalances
  - ARF
Causes of Rhabdomyolysis

Traumatic or compression
Multiple trauma
Crush injuries
Vascular or orthopaedic surgery
Coma
Immobilization

Nontraumatic

Exertional

Normal muscle
- Extreme exertion
- Environment heat illness
- Sickle cell trait
- Seizures
- Hyperkinetic states

Abnormal muscle
- Metabolic myopathies
- Mitochondrial myopathies
- Malignant hyperthermia
- Neuroleptic malignant syndrome

Nonexertional

Alcoholism
Drugs and toxins
Infections (including HIV)
Electrolyte abnormalities
Endocrinopathies
Inflammatory myopathies
Miscellaneous
Clinical presentation

- Hypovolaemic traumatic shock
- Myoglobinuria...smoky, dark brown urine
- ??? Urine volume during shock
- Hyperkalaemia
- Cardiac arrest even
- Raised S-myoglobin and CPK levels (measure serially)
- Crush injury \(\leftrightarrow\) Compartment Syndrome
  = coexist or evolve into one another
Q 14

What is reperfusion syndrome?
Ischaemia Reperfusion Syndromes

- Embolectomy
- Aortic aneurysm surgery
- Reconstructive vascular surgery
- Intra-arterial thrombolysis
- Revascularization
- Resuscitation from shock
Q 15

What are the manifestations of a reperfusion syndrome?
Manifestations of Reperfusion Syndrome

• Local effects:
  - Oedema
  - Inflammation

• Distant effects:
  - Lung injury
  - Renal injury
  - Endotoxaemia
  - Multiple organ failure
Local effects of Ischaemia reperfusion injury

- Limb swelling
- Compartment syndrome
- Abnormal muscle function
- Muscle contracture
**Distant / Systemic effects**

**IMMEDIATE**
- Hyperkalaemia → Cardiac arrhythmias
- Acidosis
- Myoglobinaemia → Acute tubular necrosis

**WITHIN 48 HOURS**
- Lung neutrophil sequestration → Noncardiogenic pulmonary oedema → Acute respiratory distress syndrome
- Renal neutrophil sequestration → Increased microvascular permeability → Acute renal failure
- Gastrointestinal endothelial oedema → Increased vascular permeability → Endotoxic shock
Q 16

Why do patients with CS develop shock?
Internal volume losses due to sequestration of fluid and solute in traumatized muscles (“third spacing”) may reach 10 to 18 L/d.

External volume losses due to dehydration (excessive sensible and insensible losses, during exertion or prostration in hot and arid environments, hyperthermia, vomiting, and diarrhea).

Cardiovascular depression due to the combination of hyperkalemia and hypocalcemia and the action of cytokines and endotoxin.

Vasodilatation of crushed muscles due to excessive increase in the activity of inducible NO synthase and NO production.\(^{15}\)
Q 17

Discuss the pathophysiology of muscle injury?
Pathophysiology of Muscle injury

- Reactive hyperaemia
- Ischaemia
- Partial ischaemia
- Ischaemia-reperfusion injury
- Increased capillary permeability
- Oxygen derived free radicals
- Lipid peroxidation
- Polymorphonuclear neutrophils
- Increased intracellular calcium
Physiological Insult

- Ischaemia
- Inflammatory response
  - Capillary leak
    - Tissue Edema
      - (Including any potential compartment)
        - Intra - compartment hypertension

Fluid resuscitation
"Fluid spaces"

E.C.F = Extra cellular fluid
I.C.F = Intra cellular fluid
I.V.F = Intra vascular fluid
I.S.F = Interstitial fluid

Diagram A

TOTAL 42 l's

E.C.F
14L ($\frac{1}{3}$)

I.C.F
28L ($\frac{2}{3}$)

Cell membrane

I.V.F
(\frac{1}{4})
2.8L

I.S.F
(\frac{3}{4})
11.2L

Na$^+$ 142-147 m.mol/l
K$^+$ 4-5 m.mol/l

Na$^+$ 15 m.mol/l
K$^+$ 150 m.mol/l
Xanthine oxidase → Oxygen free radicals

+ Oxygen

Lipid peroxidation

Thromboxane  Leukotrienes

Neutrophil recruitment

Local injury

Distant injury

Oedema and hypoxia
Reperfused cell

\[ \text{O}_2^* \quad \text{H}_2\text{O}_2 \]

Lipid peroxidation

Activated neutrophils
Intravascular thrombosis

Adhesion molecules

Increased permeability

Lysozymes + free radicals

Local tissue injury within a confined space

Extracellular oedema

Parenchymal cell damage

Neutrophil migration
Pathophysiology of Renal injury

TIME

DEATH

- Hypovolaemia
- Hypotension / Shock
- Myoglobinaemia and Myoglobinurinria
  a) Renal tubular obstruction
  b) Toxic effects of myoglobin
  c) Myoglobin-induced vasoactive agents
  d) DIC
Rhabdomyolysis

- Endotoxin cascade
- Activation of ET NO Scavenging iNOS induction
- Uptake of ECF by injured muscles
- Increased load of PO$_4$ and urate
- Myoglobinemia

- Hypovolemia & Cardiovascular Suppression due to K$^+$, Ca$^+$, metabolic acidosis
- Aciduria
- Renal Vasoconstriction

- Myoglobinuria
- Pigment Nephrotoxicity

- Luminal cast formation & stasis
- Synergistic Tubular Damage
- Acute Tubular Necrosis (ATN)
- Acute Renal Failure (ARF)
Q 18

Discuss a diagnostic approach to pigment nephropathy?
1. Consider the presenting disease or pathology as a cause – THINK.

2. Notice red brown urine
   Sentrifuge: sediment
   supernatent – test for heme.

3. Determine plasma creatine phosphokinase levels.

4. Look for source of rhabdomyolysis.
Key message

Prevention of reperfusion injury requires identification of patients at risk.
Mechanical and toxic effects of myoglobin on the Kidney

Ischaemia/Crush → Muscle injury → Myoglobin

- Mechanical
- Cast formation
- Reduced GFR

- Direct toxic effect
  - Increased Fe$^{2+}$
  - Renal vasoconstriction

Renal Failure → Dialysis
Q 19

Compare the differences in management of CS, CI and vascular injuries – is time important?
TIME

- “Many soft tissue and vascular injuries require time sensitive interventions to ensure salvage of the limb + the best outcome for the patient”

- “Despite surgical or conservative interventions, crush injuries can irreversibly destroy limb function”

THE KIDNEY:

- Our biggest Fluid & Electrolyte ally
- The great equalizer
Q 20

How should one treat a patient with rhabdomyolysis?
Treatment - Rhabdomyolysis

- Start early….during extrication!!!…ABC’s
- Hydration with Isotonic Saline
- Forced diuresis alkaline-mannitol-saline
  keep urine pH>6.5 / 7.0
- Dialysis
- Source control

Lab Investic 1989: 60: 619
NEJM 1990:322:825
KI 1996:49:314

Acute Renal Failure = Increased Mortality
Suggested Volume Replenishment

- IV Saline 1L / hour *during* extrication.
- Monitor MAP, CVP, urinary output, CK, S-myoglobin, kidney function.
- Urinary pH >6,5
- 200-300mmol of NaHCO$_3$ in 1$^{st}$ day.
- Urine flow – maintain fluid balance.
- Monitor arterial pH and osmol.
- Usually 3 days to clear myoglobin.
1. Choose volume to be infused: 4 / 6 / 8 L Saline per 24 hours (according to body mass).
2. Average 200-300 mmol NaHCO\(_3\) per 24 hours.
3. Maximum 200g Mannitol per 24 hours.

Depending on the volume chosen, mix the following with every liter Saline:

<table>
<thead>
<tr>
<th>Volume per hr</th>
<th>NaHCO(_3) mmol/24 hrs</th>
<th>20% Mannitol g/24 hrs</th>
</tr>
</thead>
<tbody>
<tr>
<td>a) 4000 / 24h</td>
<td>75mmol/1000ml</td>
<td>50g/1000ml</td>
</tr>
<tr>
<td></td>
<td>=75ml 8,5% NaHCO(_3)</td>
<td>=250ml 20% Mannitol</td>
</tr>
<tr>
<td>166ml / h</td>
<td></td>
<td></td>
</tr>
<tr>
<td>b) 6000 / 24h</td>
<td>50mmol/1000ml</td>
<td>33g/1000ml</td>
</tr>
<tr>
<td></td>
<td>=50ml 8,5% NaHCO(_3)</td>
<td>=165ml 20% Mannitol</td>
</tr>
<tr>
<td>250ml / h</td>
<td></td>
<td></td>
</tr>
<tr>
<td>c) 8000 / 24h</td>
<td>37,5mmol/1000ml</td>
<td>25g/1000ml</td>
</tr>
<tr>
<td></td>
<td>=37,5ml 8,5% NaHCO(_3)</td>
<td>=125ml 20% Mannitol</td>
</tr>
<tr>
<td>333ml / h</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Never Ringers or Plasmalyte!!!
In Practice:

When hyper-Na+ is a problem:

1000ml 0,45% NaCl
+ 70mmol 8,5% NaHCO₃ (70ml)
+ 37,5g 20% Mannitol (± 190ml)
(3 amps of 12,5g)

Administer at 100 – 120 – 200ml/h

Aim: ± 300ml urine/h
urine pH > 6,5
arterial pH < 7,5

Stop when S-myoglobin is less than 300
**Mannitol**

- Non-electrolyte solute diuresis = Osmotic diuresis
- Cosm = Uosm \( \bullet \) \( \frac{V}{Posm} \)
- Concentrating and diluting mechanisms of kidney
  volume status
  integrity of epithelial transport
  level of ADH

*STATE OF HYDRATION GREATLY INFLUENCES MAGNITUDE OF MANNITOL DIURESIS.*

- Hypotension!
- Uosm may not accurately reflect volume status – Mannitol obligates an isotonic diuresis.
- **Mechanism of the diuresis:**
  a) Osmotic inhibition of water transport
  b) Diminished gradient for passive Na\(^+\) absorption
FINALLY:

1. Do **NOT** try to diurese a hypovolaemic patient

2. What goes in **MUST** come out!
Limb vs life threatening injuries

Step 1: Acute, severe injuries

- Always remember, life before limb
- Use clinical judgment and common sense
- Be prepared to make a clear recommendation to the family
- Consult colleagues
Limb vs life threatening injuries

**Step 2: Acute management of severe injuries**

- Resuscitate promptly
- Obtain source / damage control
- Plan definitive surgery
Limb vs life threatening injuries

*Step 3: Acute disturbance of homeostasis*

- Recognize the severity of the injuries
- Monitor homeostasis
- Provide adequate systems support

Thank you for participating!