The Seriously Injured or Threatened Limb

LIMB vs LIFE
Threatening Injuries

JP Pretorius
S Motsitsi
T Mulaudzi

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.

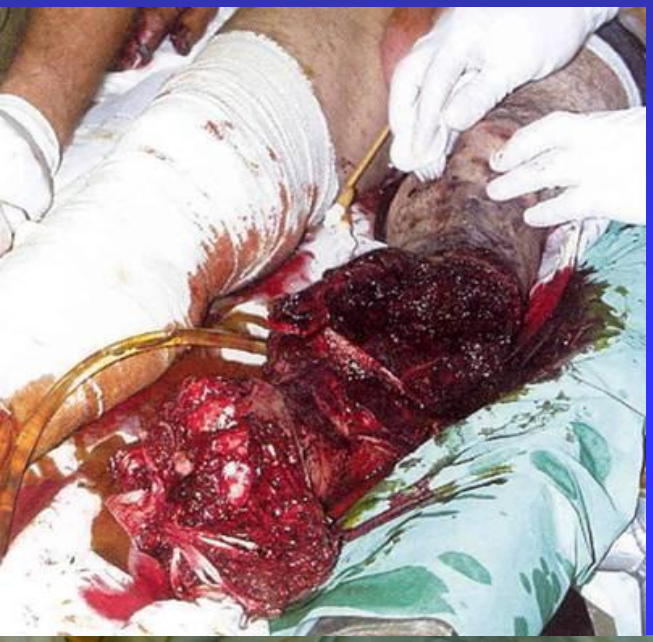
Compartment Syndrome:

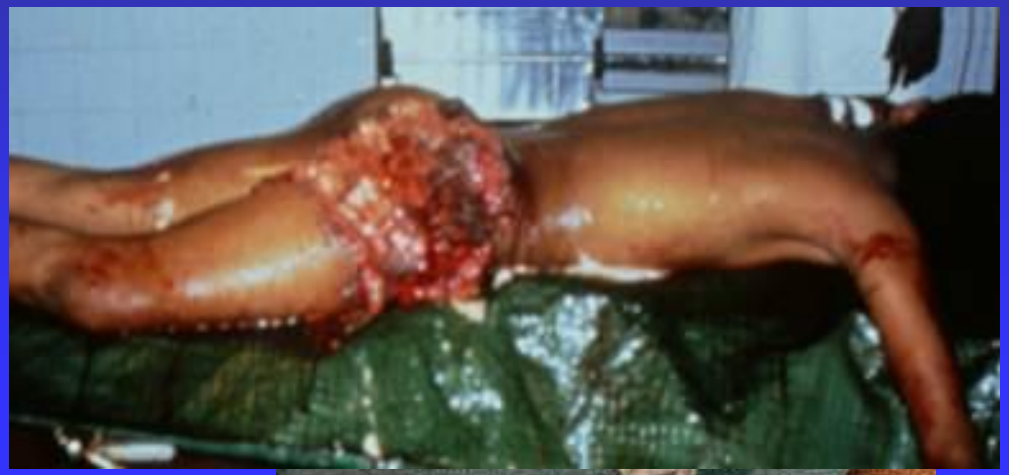
The local manifestations of nerve and muscle ischaemia due to increased pressure within the osseo-fascial compartments.

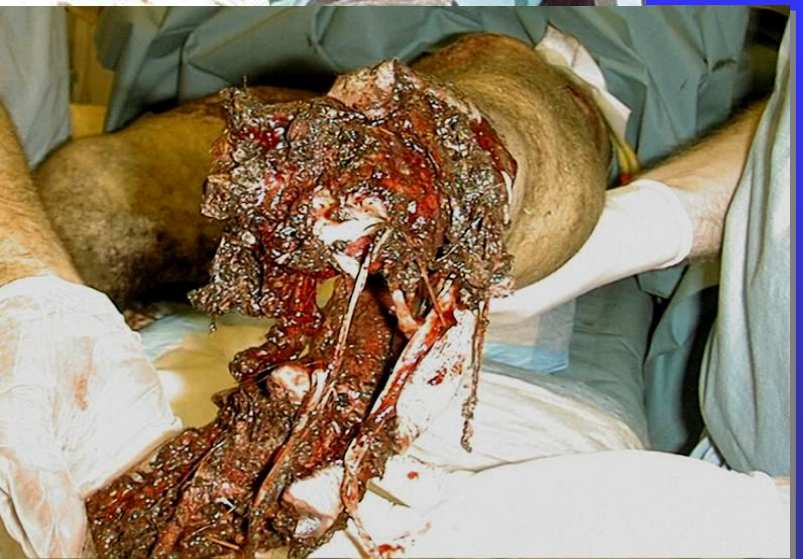
Q 2

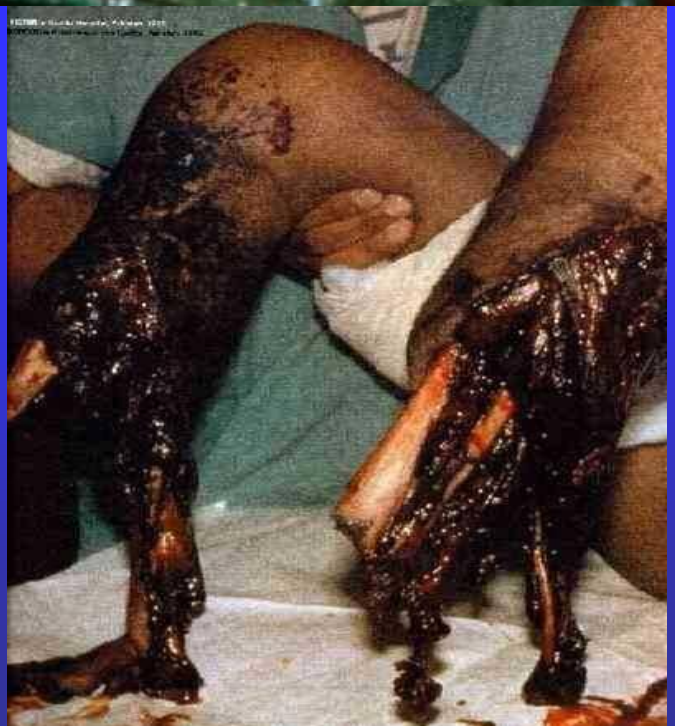
What does the concept
“CRUSH SYNDROME”
signify ?

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 cholesistitis)
  - Bowel (NOBS with nutrition)

Concept: Perfusion Pressure

APP = MAP - 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%

CPP = MAP - ICP

*Compartment Syndrome = MAP - Intra-Comp P

Perfusion Pressure as a primary endpoint in resuscitation!!!

Endpoints optimize systemic perfusion!!!

Apparatus for measuring compartment pressure

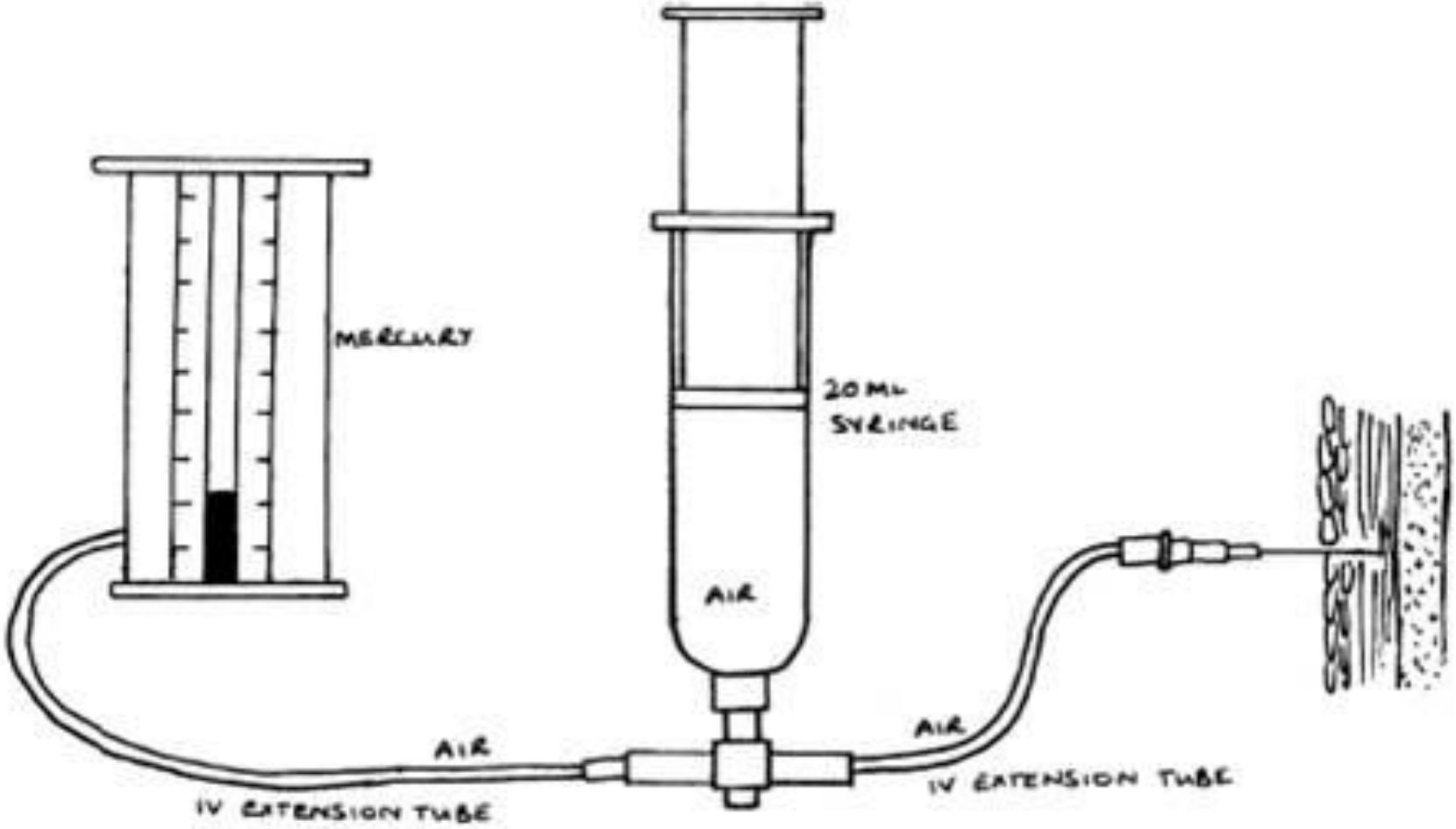


Figure 1 Apparatus for measuring compartment pressure.

Stryker device

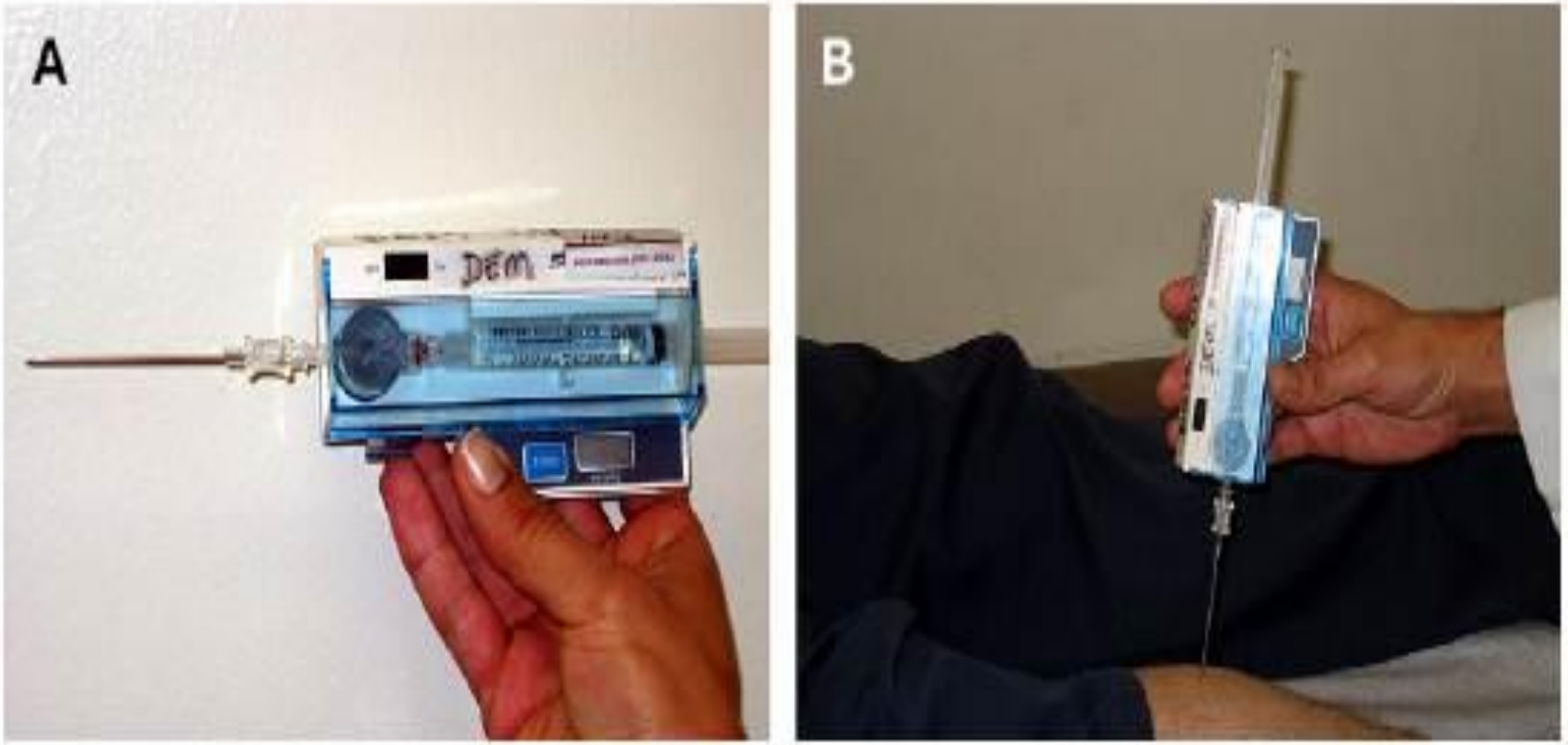


Fig. 1. (*A*) The Stryker device for measurement of intracompartmental pressure. (*B*) The Stryker device being inserted into the calf compartment under local anesthesia.

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
  - $\triangle p$ = 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 ISCEMIA

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

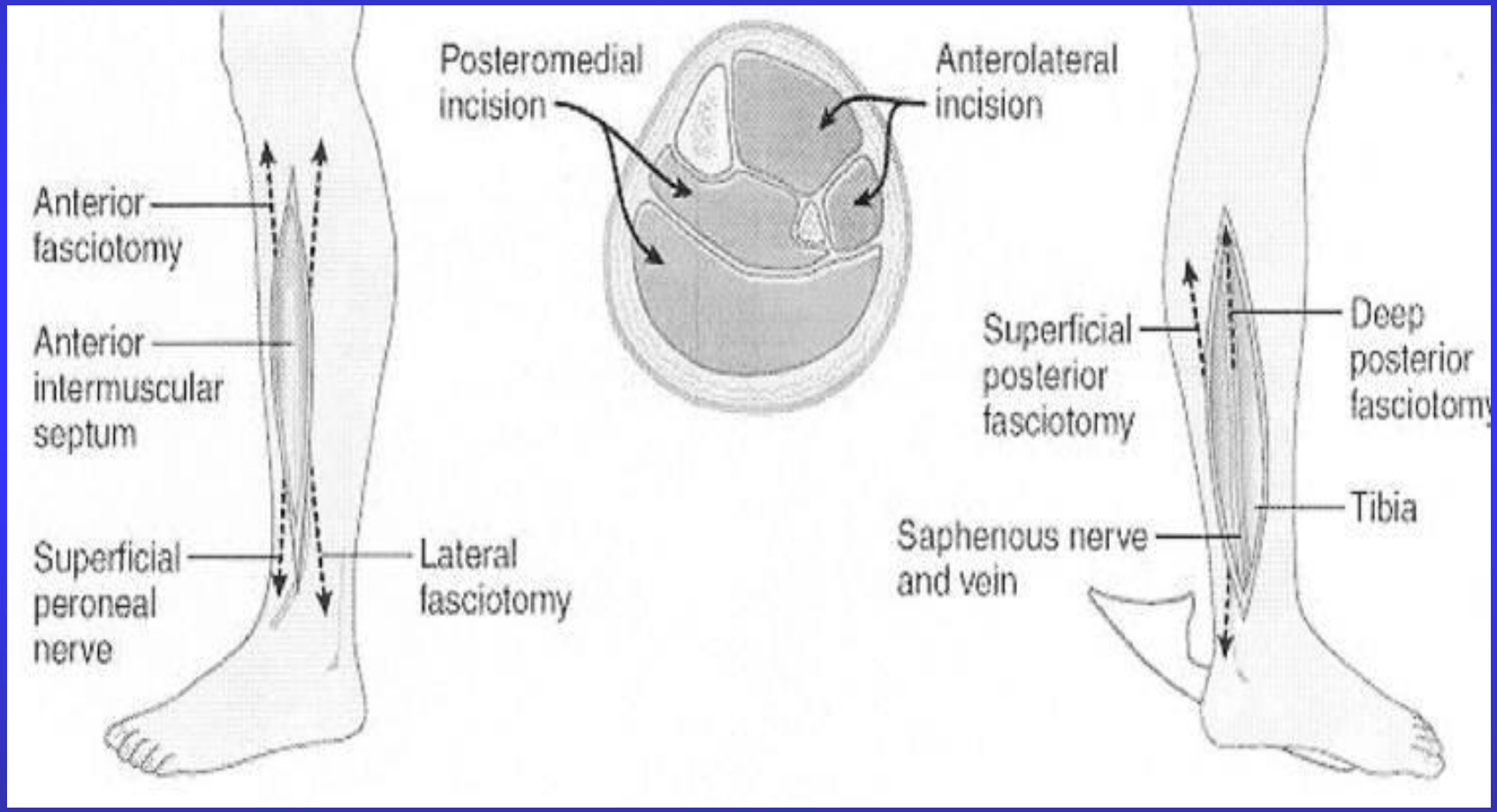
Class	Category	Prognosis	Sensory loss	Muscle weakness	Arterial Doppler	Venous Doppler
I	Viable	No immediate limb threat	None	None	Audible	Audible
IIA	Threatened: marginal	Salvageable if treated promptly	Minimal-none	None	+/- Audible	Audible
IIB	Threatened: Immediate	Salvageable if treated immediately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

Q 8

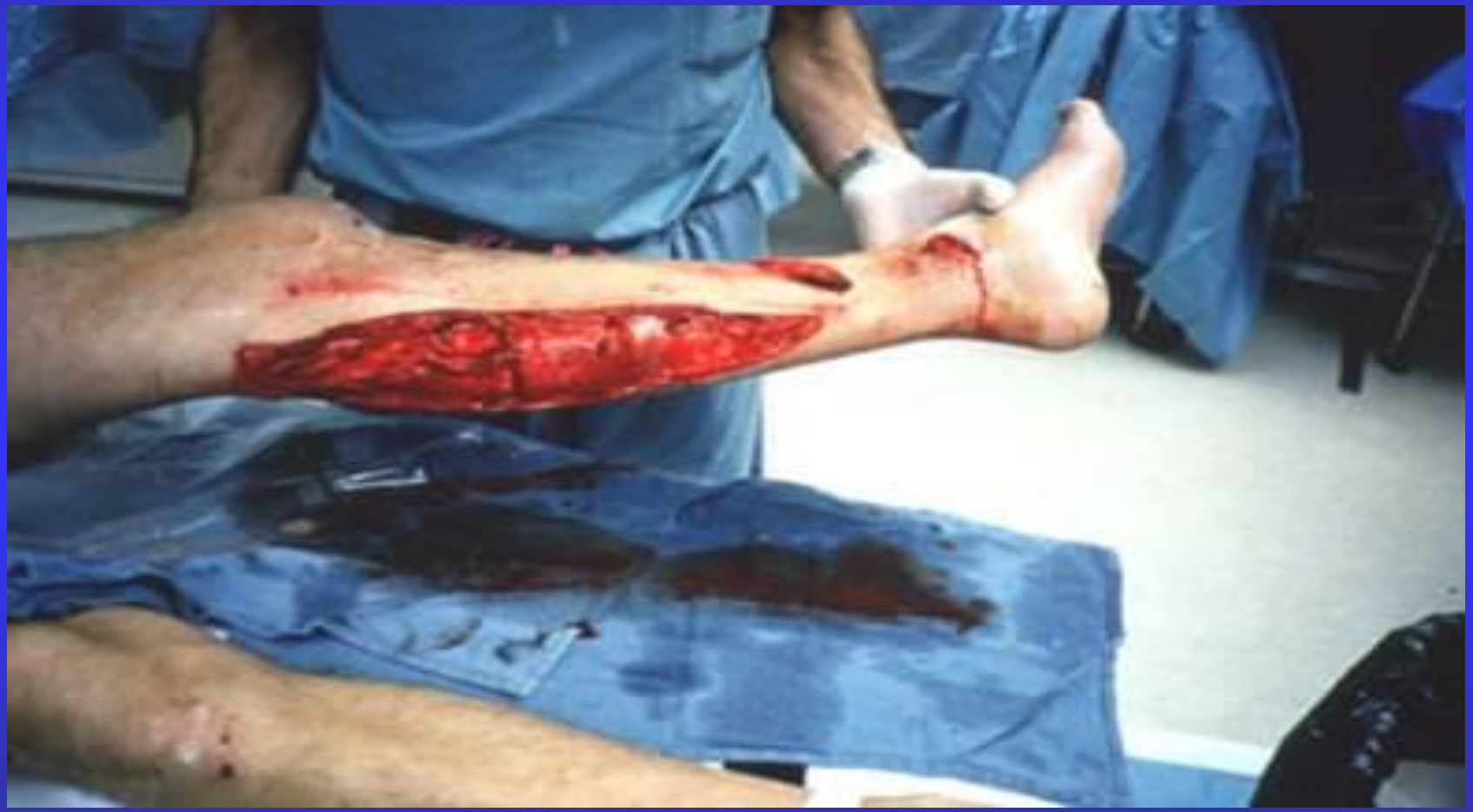
The threatened limb

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

The triad of death

Hypovolaemic

Traumatic

Shock

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

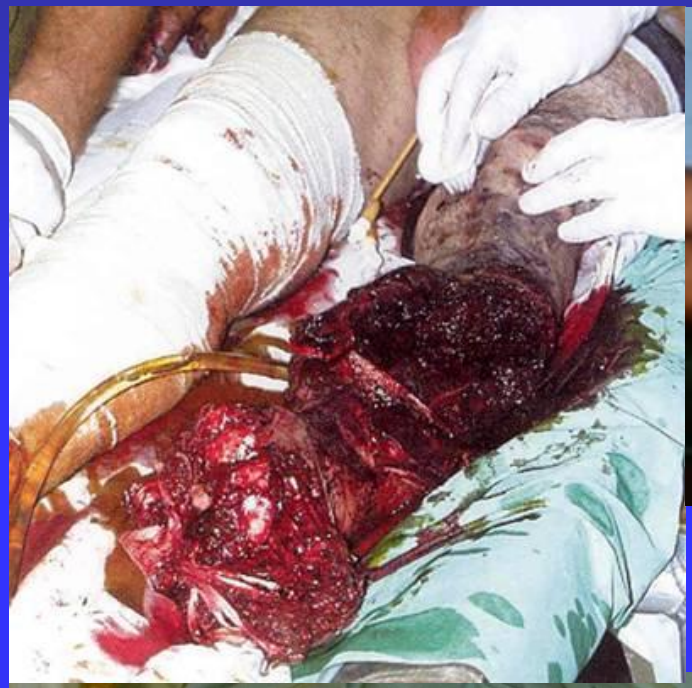
} ***Deadly triad***

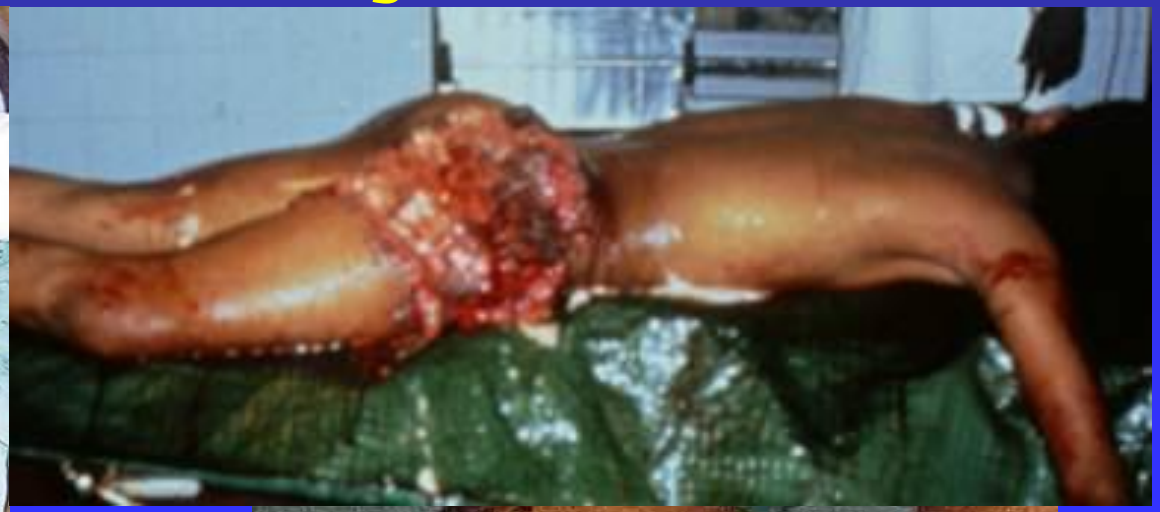
- Severe inflammatory response syndrome
- Multiple organ dysfunction / failure

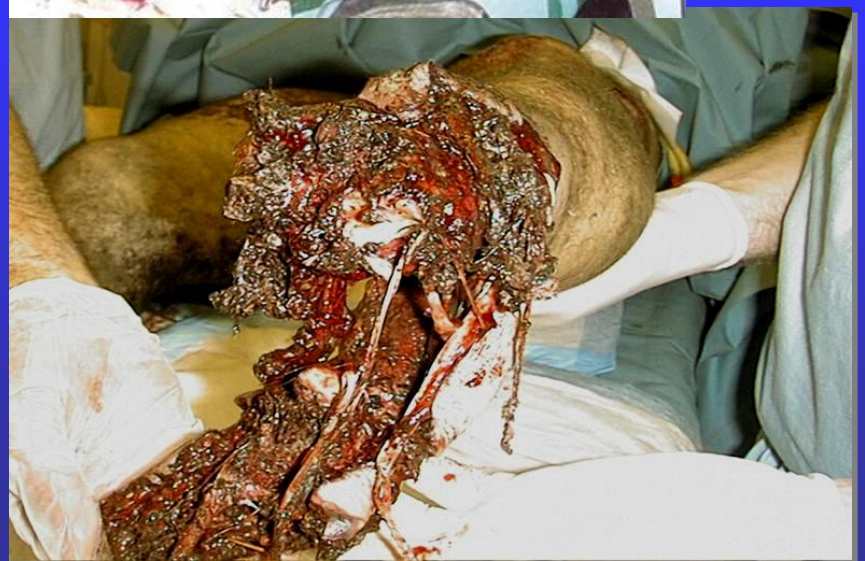
Q 10

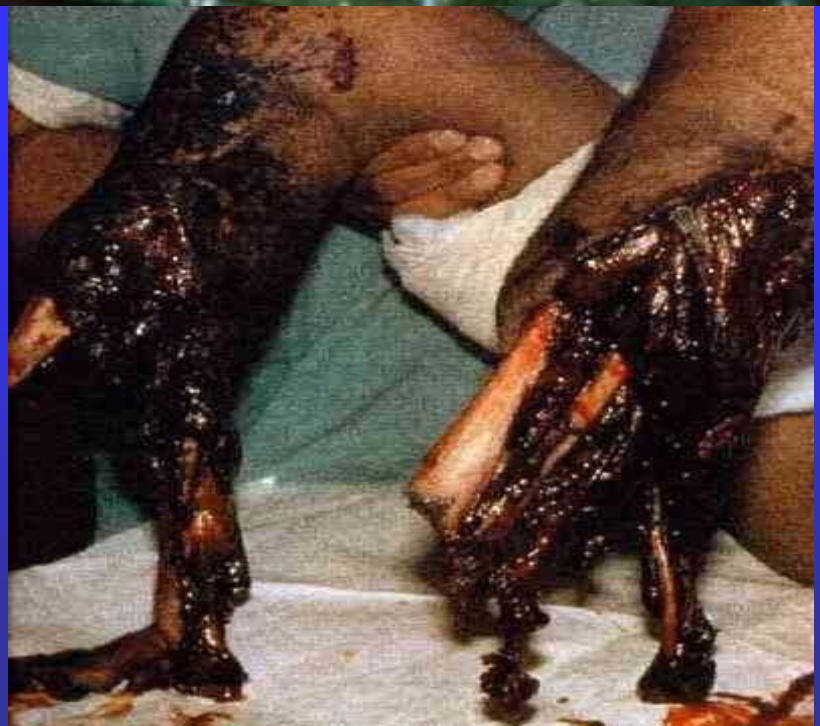
Can you think of examples of life threatening limb injuries ?

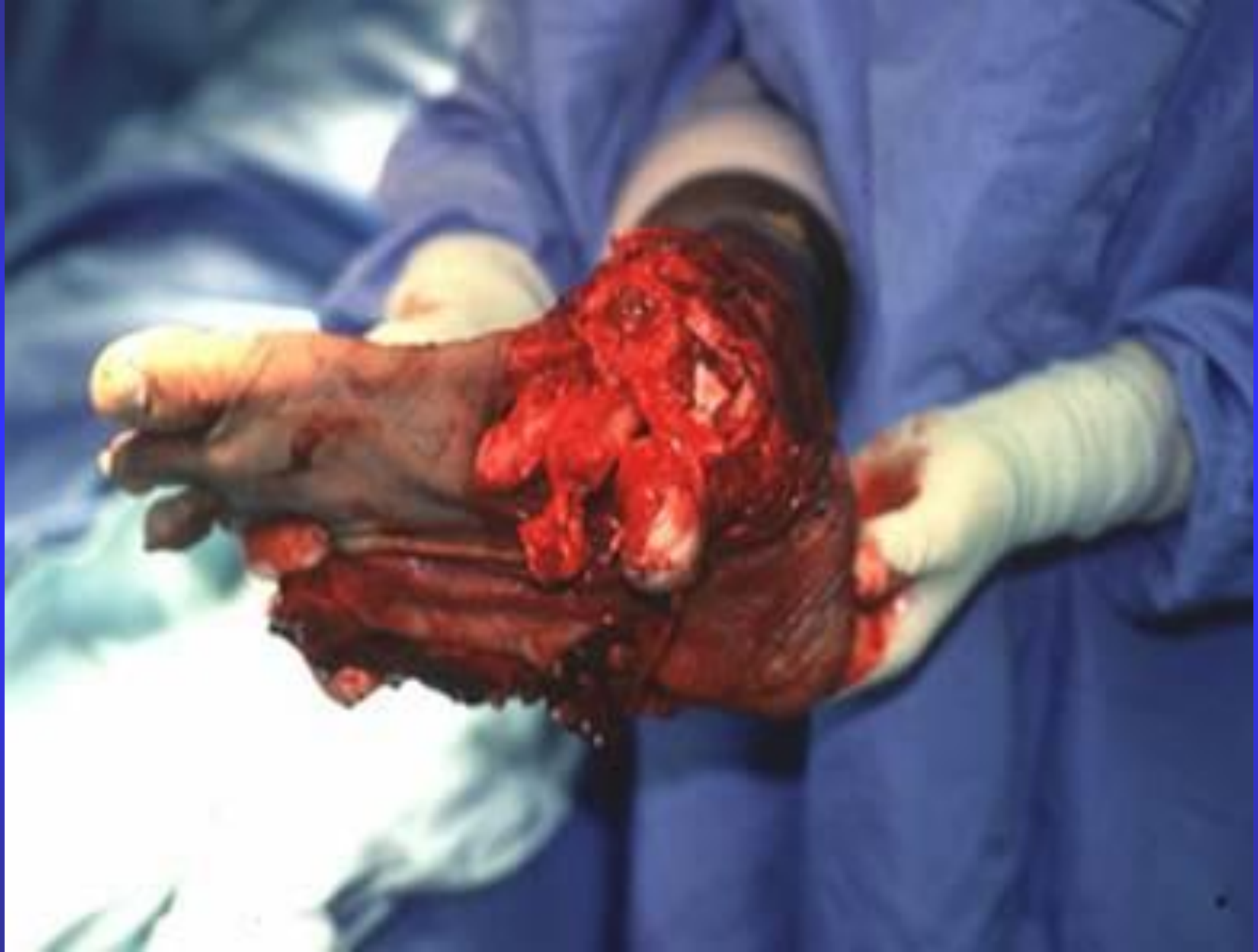
Serious soft tissue, bone, nerve and vascular injuries











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

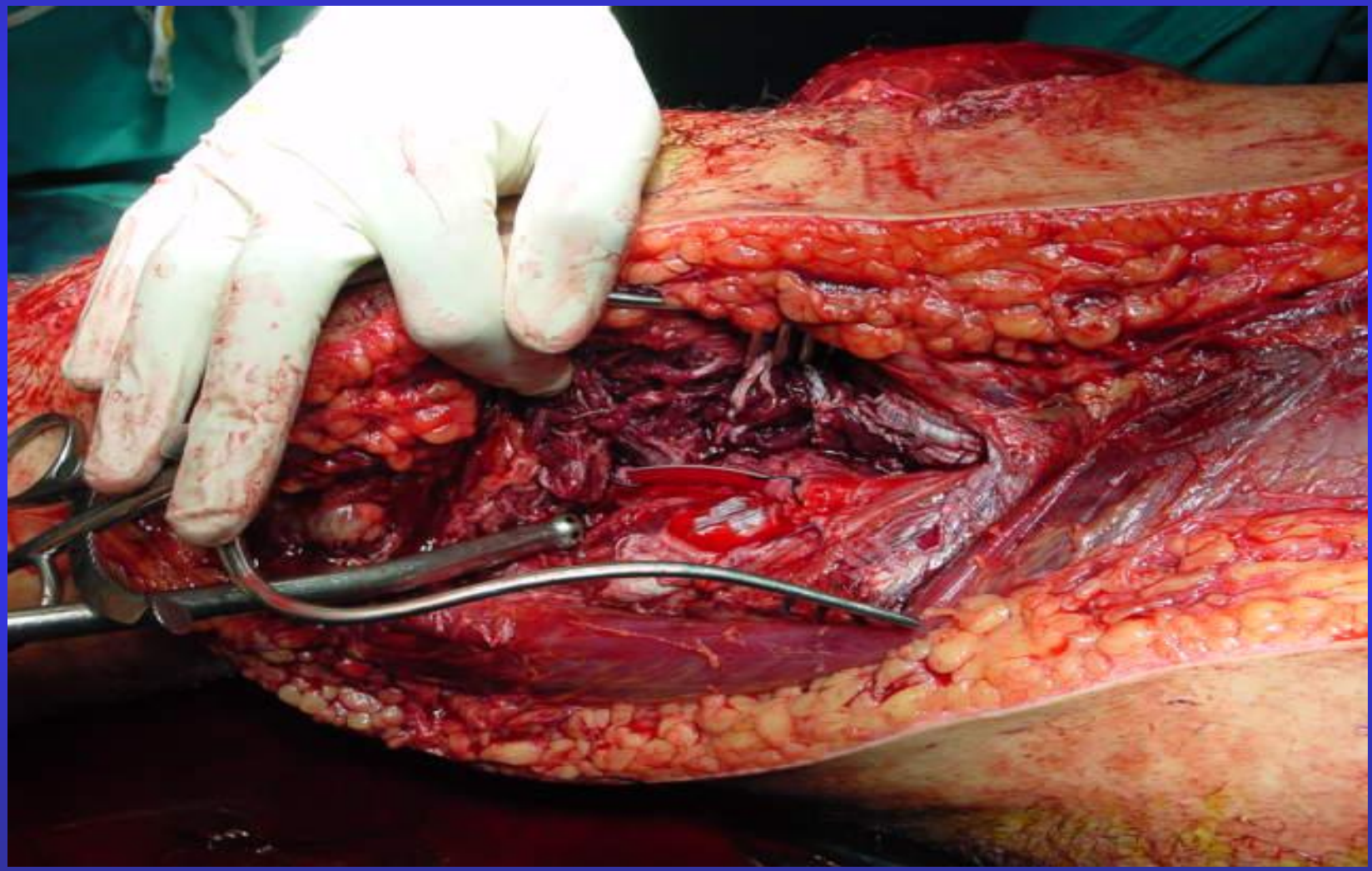
- Damage control surgery
  - Criteria
    - Acidosis: pH < 7.3
    - Temperature < 35 C
    - Coagulopathy
- Damage control surgery in vascular injuries is challenging

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
- NISSSA = Nerve injury, Ischemia, Soft-tissue contamination, Skeletal damage, Shock, Age.

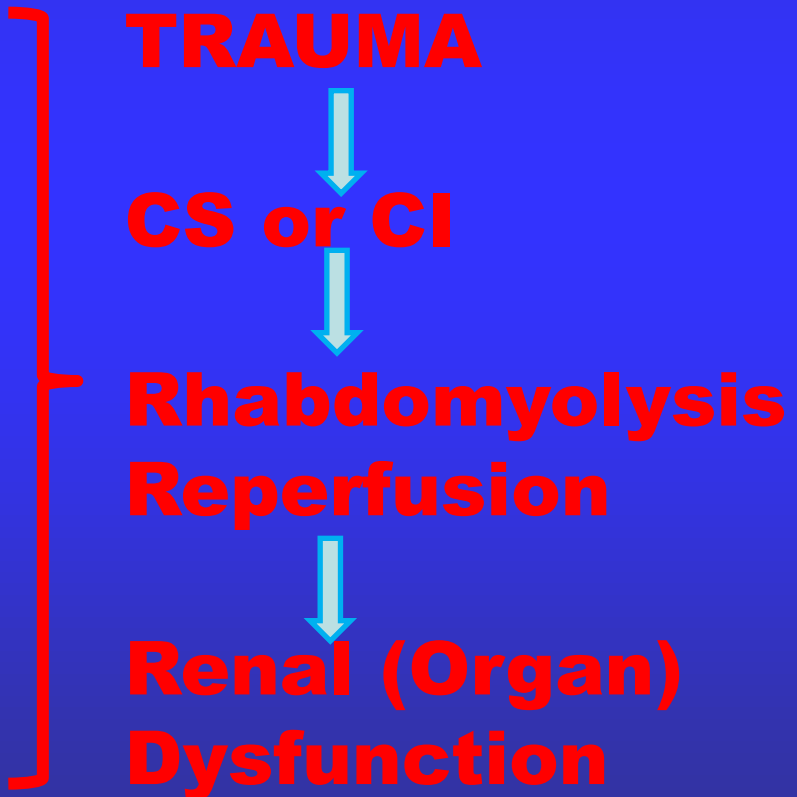
Dead tissue kills people!

- 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

Definitions:

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



Q 13

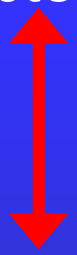
What is rhabdomyolysis ?

Definition:

Rhadbomyolysis 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 ⟺ 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, Acidosis → Cardiac arrhythmias

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 ?

Table 1. Causes of Haemodynamic Shock in the CS

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.

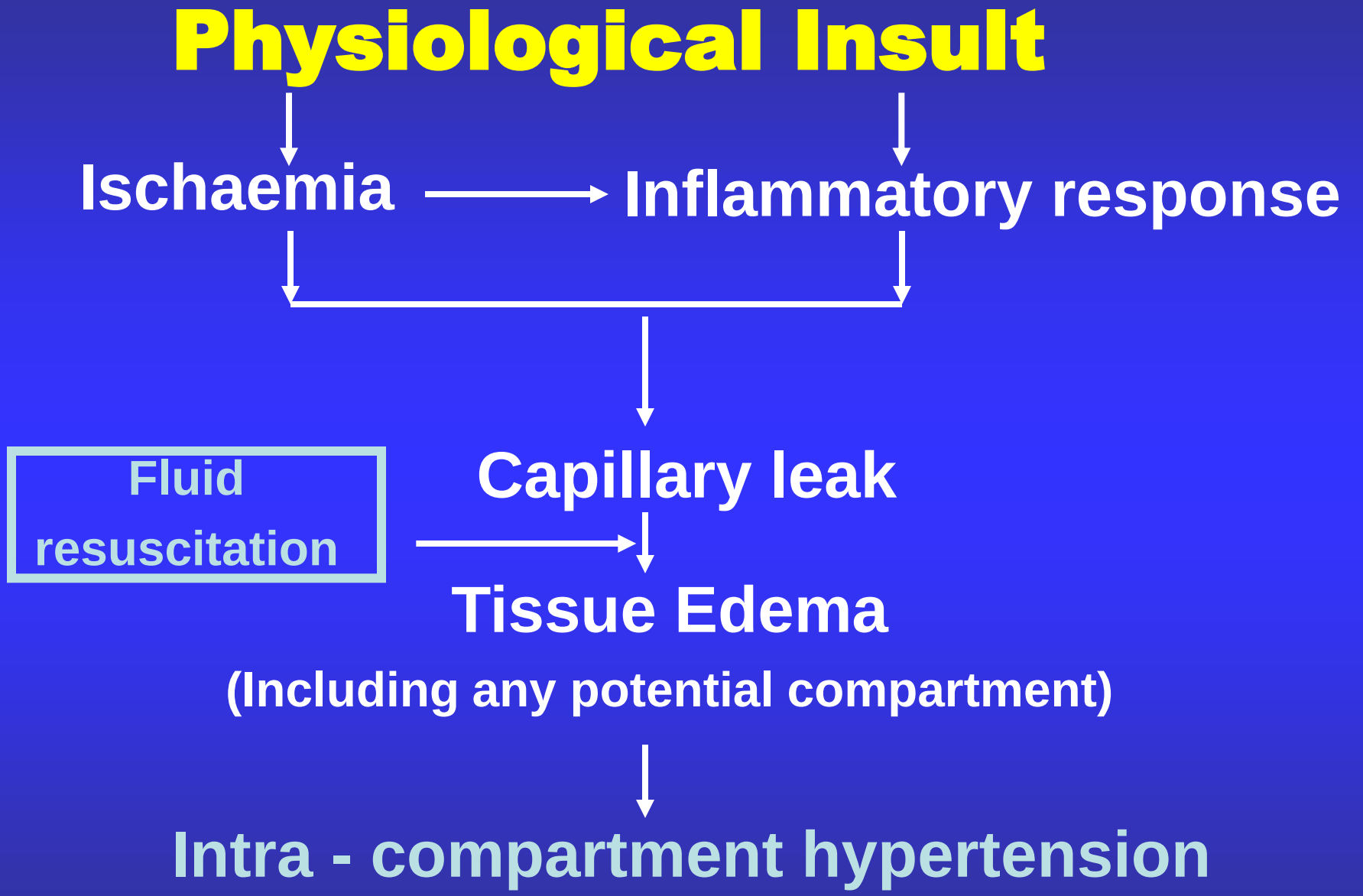
Vasodilatation of crushed muscles due to excessive increase in the activity of inducible NO synthase and NO production[15] .

Q 17

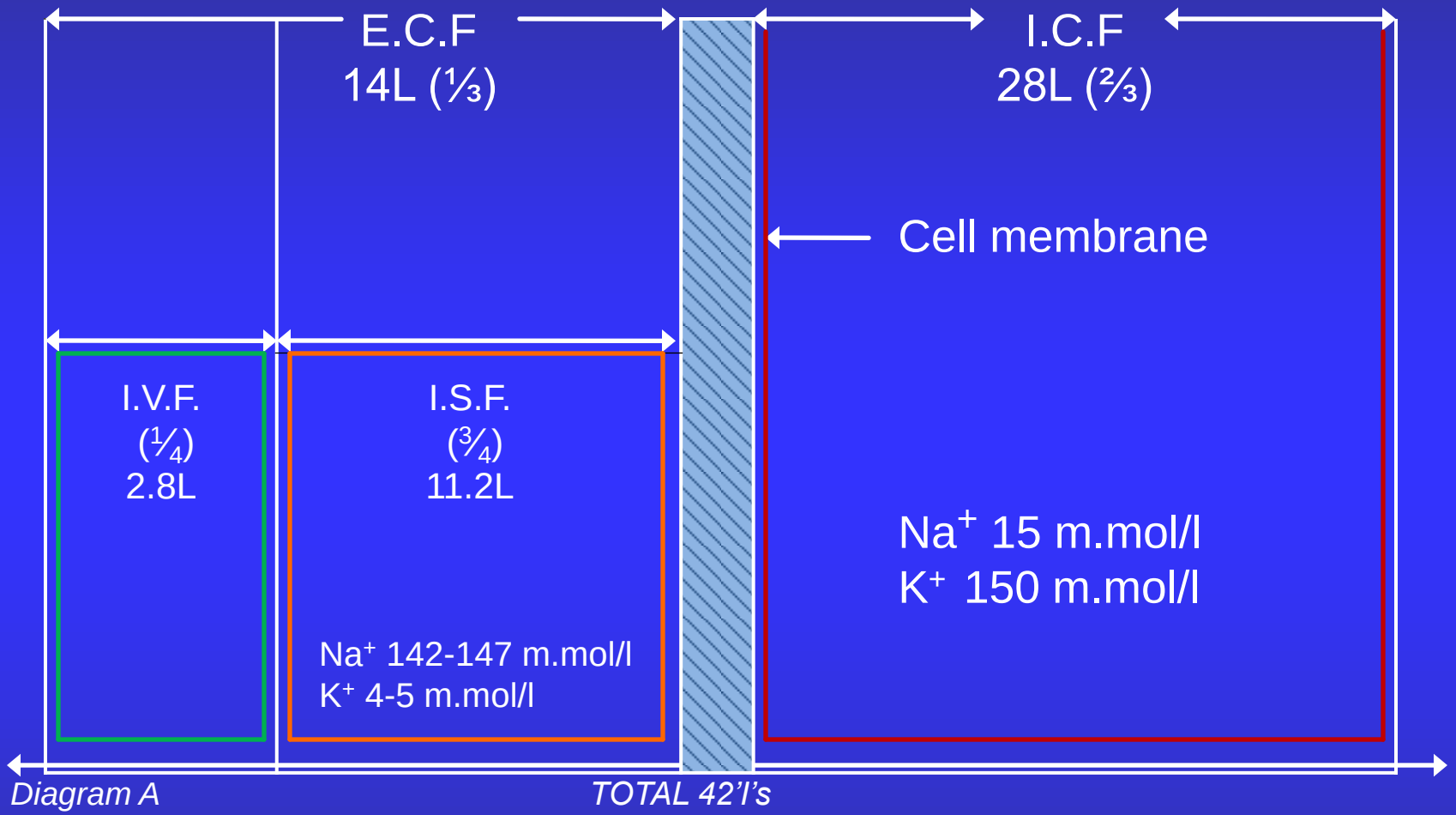
Discuss the pathophysiology of muscle injury ?

Pathophysiology of Muscle injury

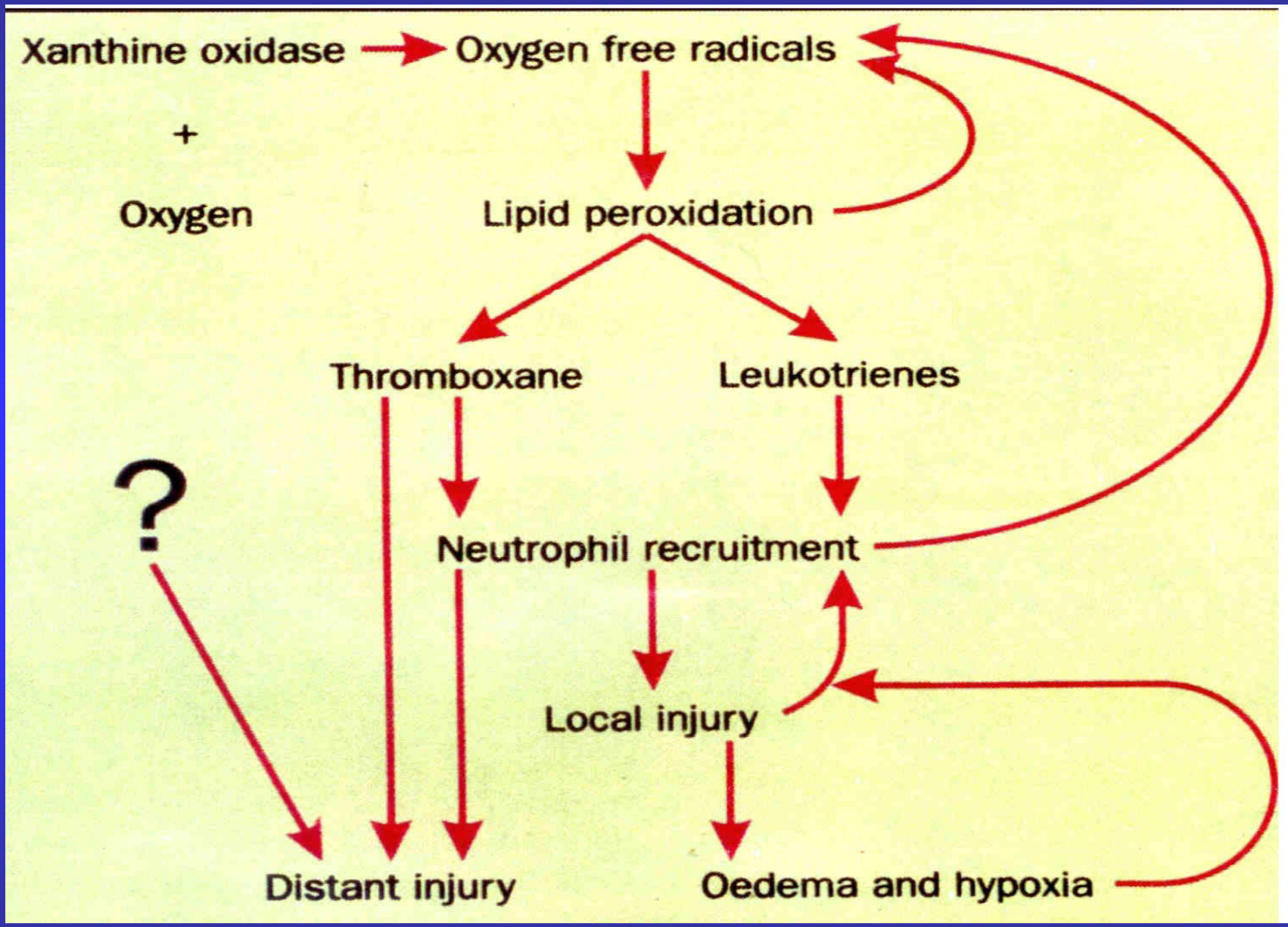
- Reactive hyperaemia
- Ischaemia
- Partial ischaemia
- Ischaemia-reperfusion injury
- Increased capillary permeability
- Oxygen derived free radicals
- Lipid peroxidation
- Polymorphonuclear neutrophils
- Increased intracellular calcium

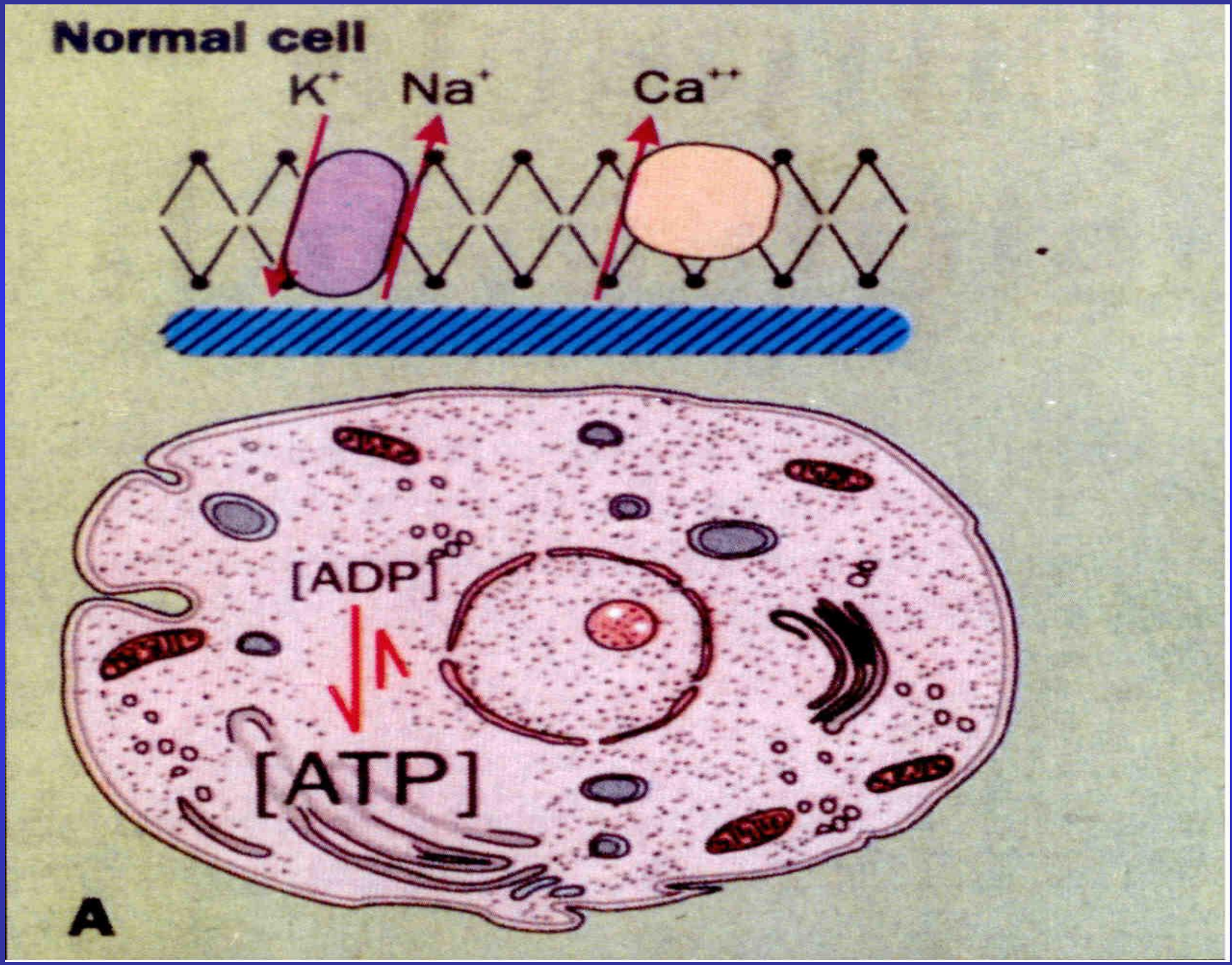

Physiological Insult
Ischaemia
Inflammatory response
Fluid resuscitation
Capillary leak
Tissue Edema
(Including any potential compartment)
Intra - compartment hypertension

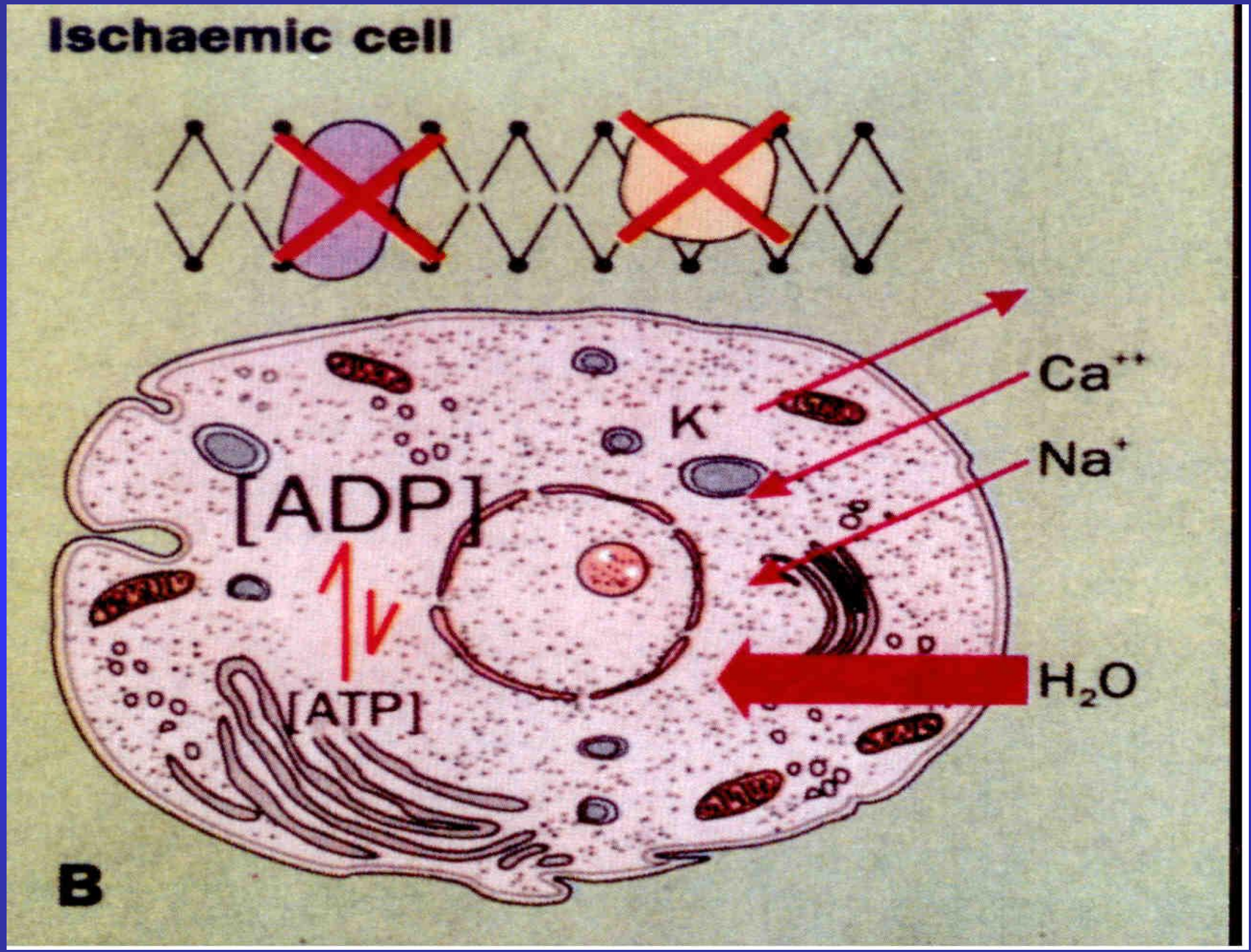
"Fluid spaces"

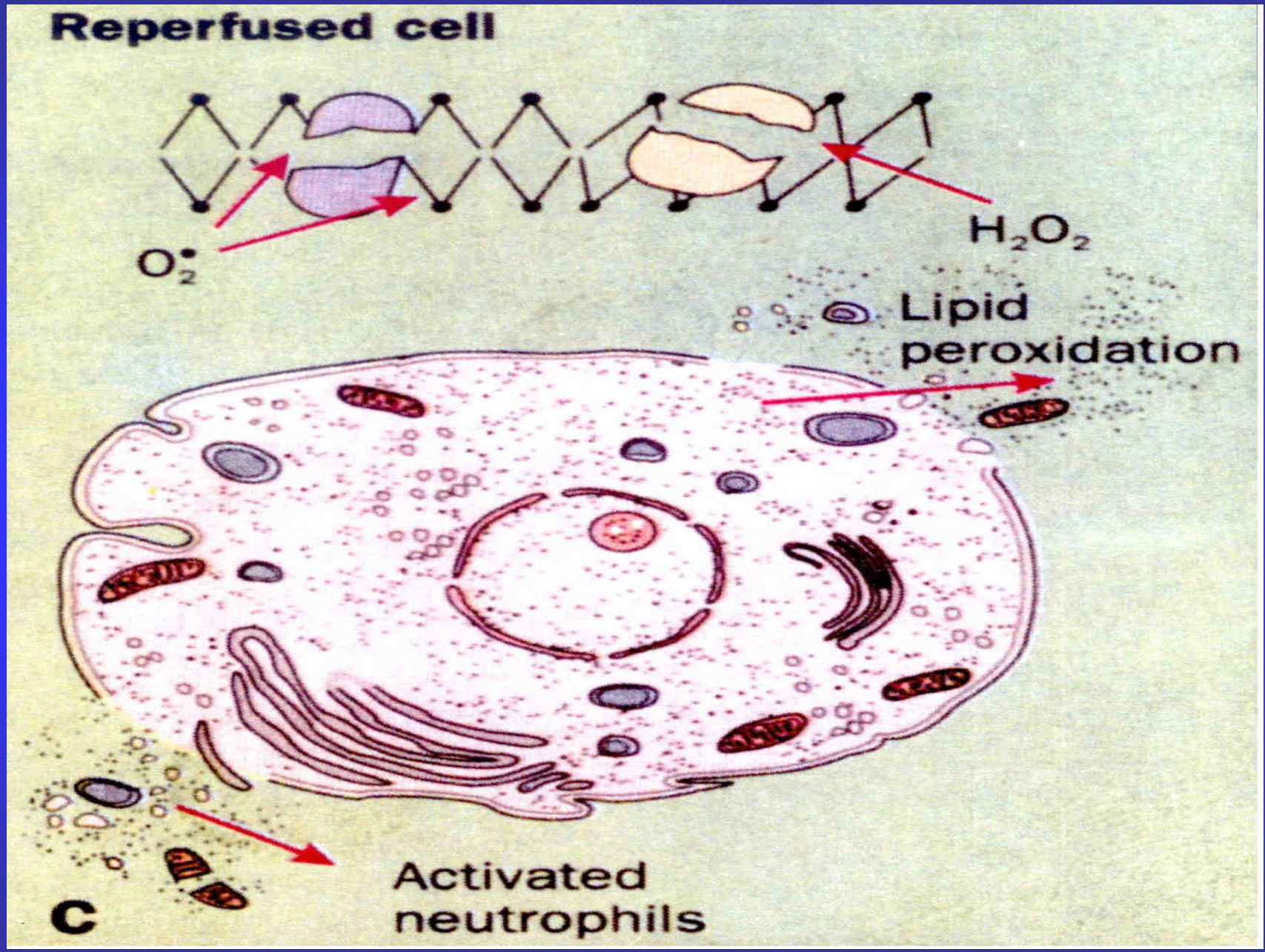


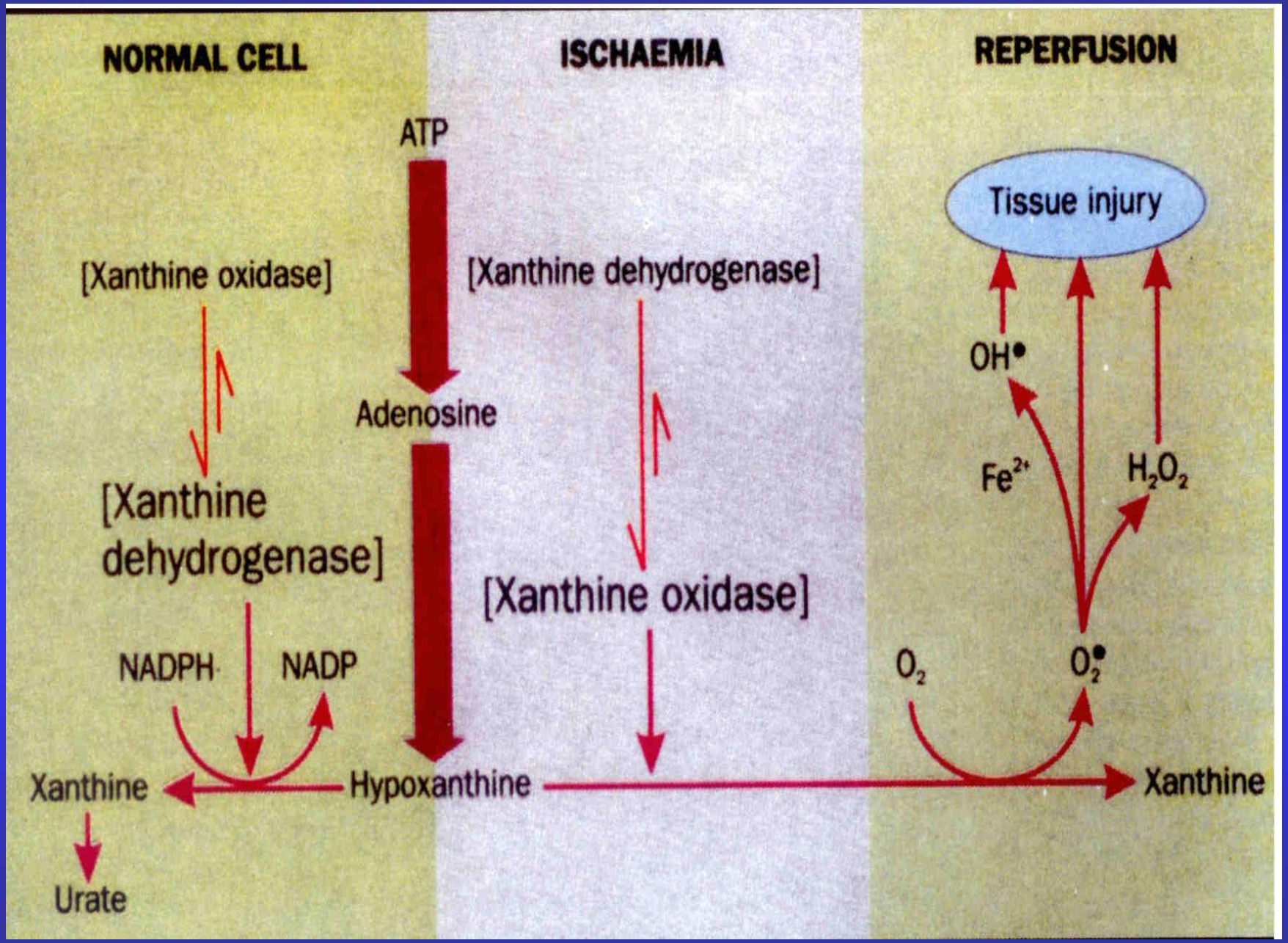
I.C.F	=	Intra cellular fluid
E.C.F	=	Extra cellular fluid
I.V.F	=	Intra vascular fluid
I.S.F	=	Interstital fluid

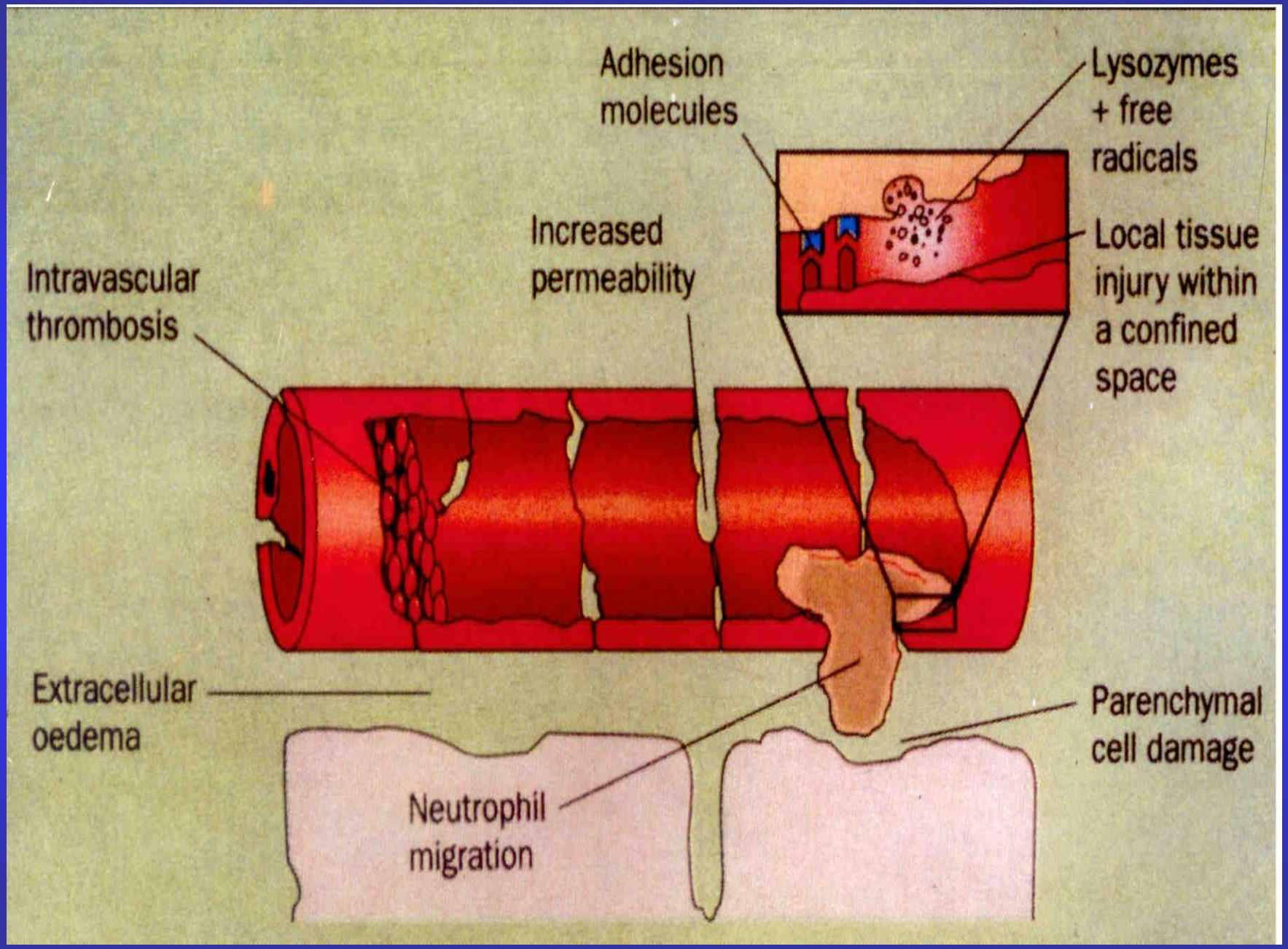

Xanthine oxidase
Oxygen free radicals
+
Oxygen
Lipid peroxidation
Thromboxane
Leukotrienes
?
Neutrophil recruitment
Local injury
Distant injury
Oedema and hypoxia


Normal cell
K+
Na+
Ca++
[ADP]
[ATP]
A

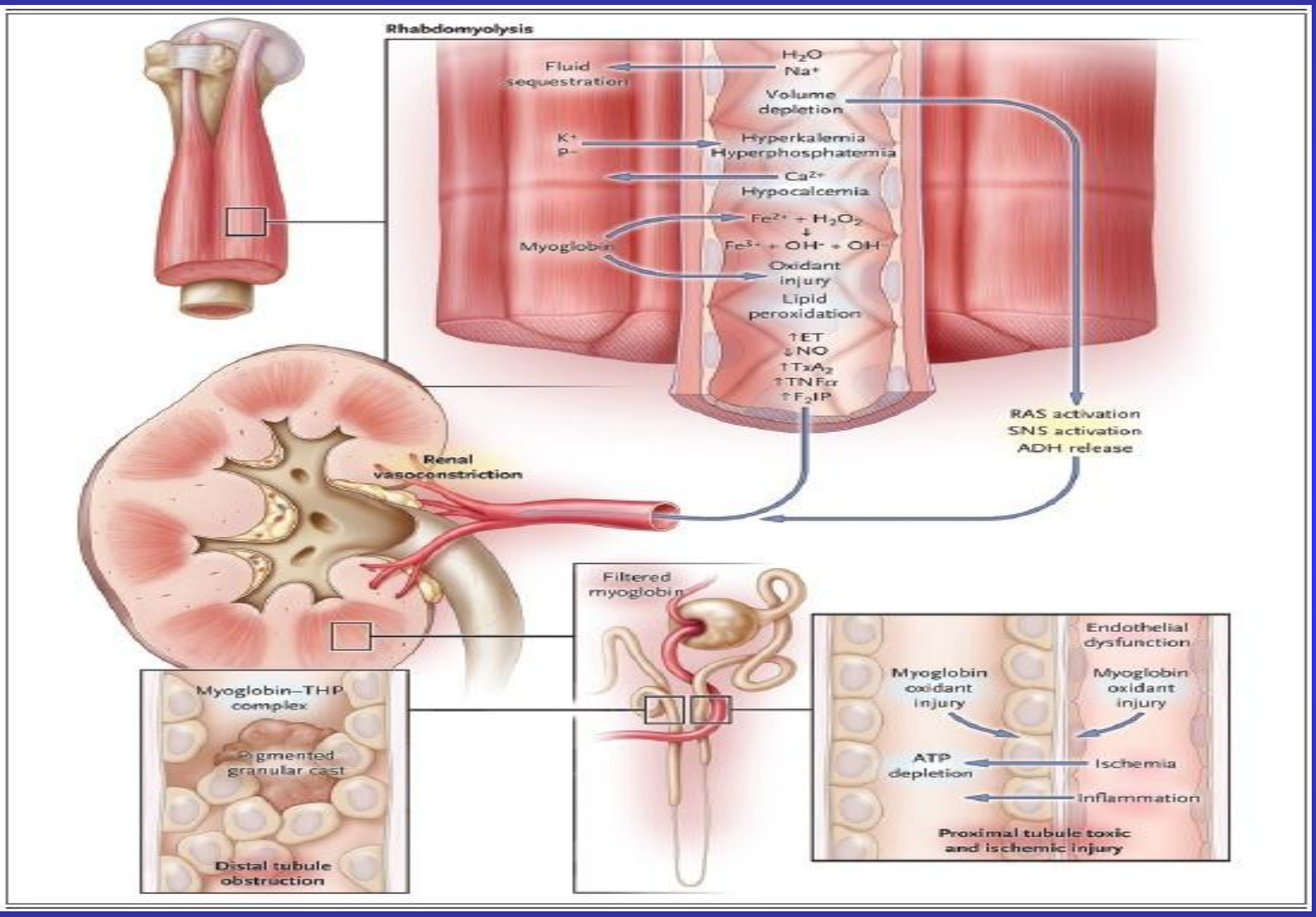

Ischaemic cell
Ca++
K+
Na+
[ADP]
[ATP]
H2O
B


Reperfused cell
H_2O_2
$O_2^{\bullet}$
Lipid peroxidation
Activated neutrophils
C


NORMAL CELL
ISCHAEMIA
REPERFUSION
ATP
[Xanthine oxidase]
[Xanthine dehydrogenase]
Tissue injury
OH•
Adenosine
Fe2+
H2O2
[Xanthine dehydrogenase]
[Xanthine oxidase]
NADPH
NADP
O2
O2•
Xanthine
Hypoxanthine
Xanthine
Urate


Adhesion molecules
Lysozymes + free radicals
Increased permeability
Local tissue injury within a confined space
Intravascular thrombosis
Extracellular oedema
Parenchymal cell damage
Neutrophil migration

Rhabdomyolysis



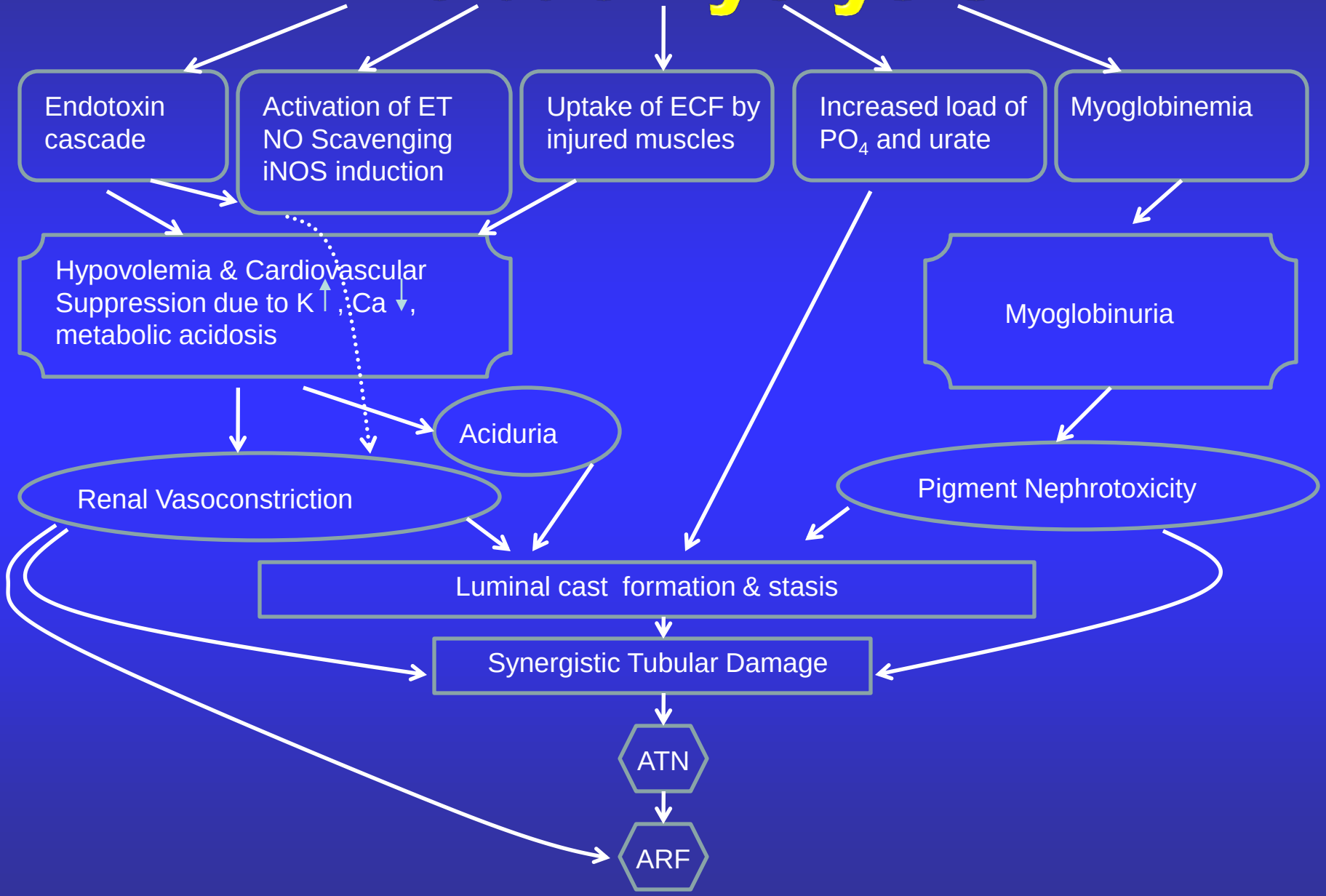
Pathophysiology of Renal injury

TIME

DEATH

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

Rhabdomyolysis



Q 18

Discuss a diagnostic approach to pigment nephropathy ?

Diagnostic Approach

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

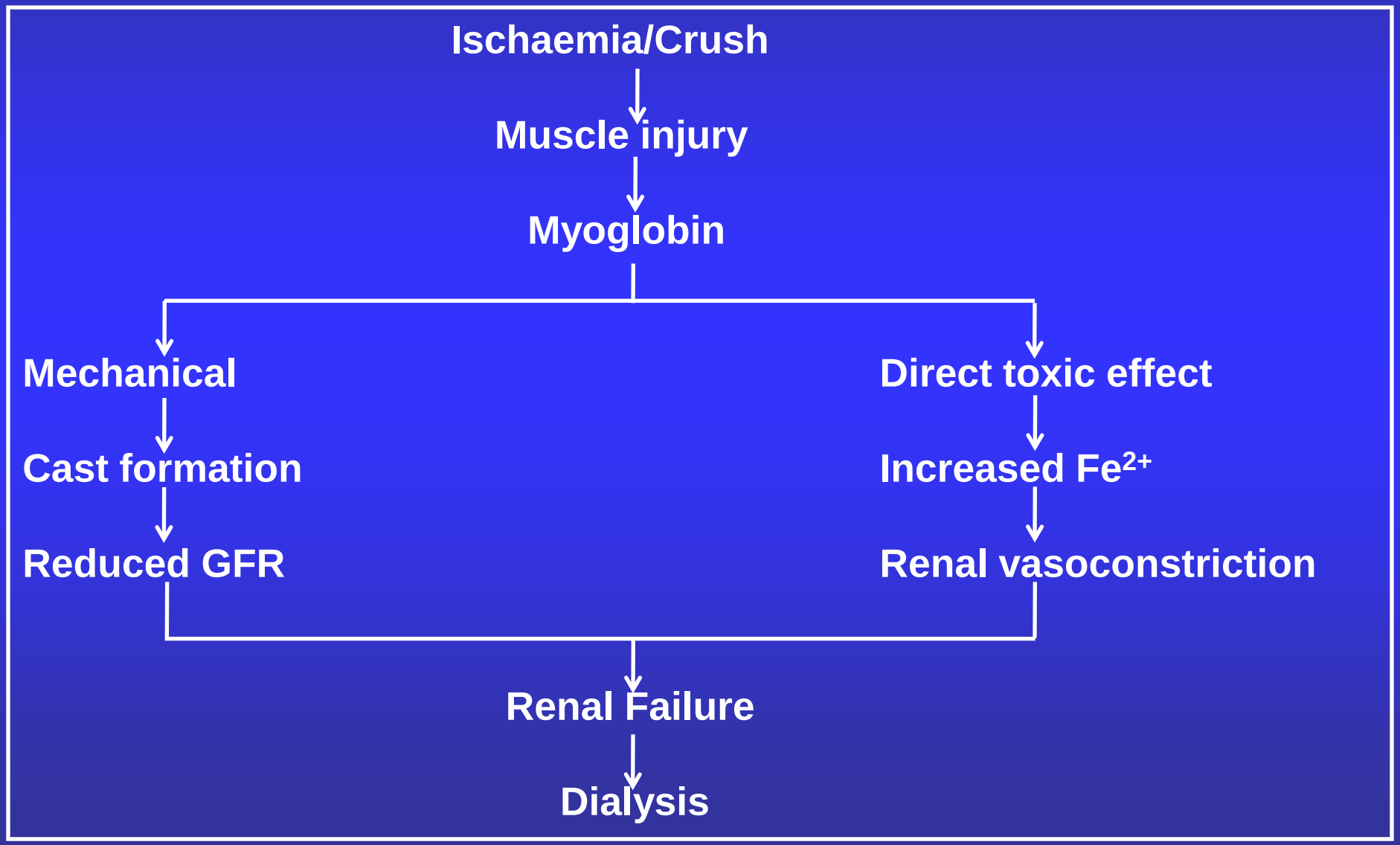
Prevention

of reperfusion injury requires

identification

of patients at risk.

Mechanical and toxic effects of myoglobin on the Kidney



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”

Emerg Med Clin NA 25 (2007) 751-761

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 duiresis alkaline-mannitol-saline keep urine pH>6,5 / 7,0
- Dialysis
- Source control

Emerg Med Clin N Am 2007: 25: 751
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.

In Practice:

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:

Volume per hr	$NaHCO_3$ mmol/24 hrs	20% Mannitol g/24 hrs
a) 4000 / 24h 166ml / h	300 / 4L 75mmol/1000ml =75ml 8,5% $NaHCO_3$	200 / 4L 50g/1000ml =250ml 20% Mannitol
b) 6000 / 24h 250ml / h	300 / 6L 50mmol/1000ml =50ml 8,5% $NaHCO_3$	200 / 6L 33g/1000ml =165ml 20% Mannitol
c) 8000 / 24h 333ml / h	300 / 8L 37,5mmol/1000ml =37,5ml 8,5% $NaHCO_3$	200 / 8L 25g/1000ml =125ml 20% Mannitol

Never Ringers or Plasmalyte!!!

In Practice:

When hyper-Na+ is a problem:

1000ml 0,45% NaCl
+
70mmol 8,5% $NaHCO_3$ (70ml)
+
37,5g 20% Mannitol (± 190ml)
(3amps 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 • 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!