IC 36: "Oh that's Tight!" Compartment Syndrome and Volkmann's Contracture. From Diagnosis to Treatment, Including Managing the Legal Aspects of Compartment Syndrome

Moderator(s): Milan V. Stevanovic, MD

Faculty: Milan V. Stevanovic, MD, Scott H. Kozin, MD, Frances E. Sharpe, MD, Thomas M. O'Neil

Session Handouts

Friday, September 06, 2019

74TH ANNUAL MEETING OF THE ASSH
SEPTEMBER 5 – 7, 2019
LAS VEGAS, NV

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IC36: "Oh that's Tight!" Compartment Syndrome and Volkmann's Contracture. From Diagnosis to Treatment, Including Managing the Legal Aspects of Compartment Syndrome (AM19)
Friday 9/6/2019

Moderator: Milan Stevanovic
Faculty:
Scott Kozin: Acute Compartment Syndrome: Pathophysiology / Diagnosis / Treatment
Frances Sharpe: Atypical Presentations / Management of Volkmann's Contracture (mild and moderate)
Milan Stevanovic: Management of Volkmann's Contracture (Severe) using functional free muscle transfer
Thomas O'neil, esq: Legal pitfalls in treating compartment syndrome

Compartment Syndrome

Definition of Compartment syndrome: regardless of etiology, the central pathogenic factor in compartment syndrome is ***increased tissue pressure.*** Pre-requisite for development of compartment syndrome includes a constricting envelope (dressing/cast, skin, fascia, epimysium) which maintains increased intracompartmental tissue fluid pressure.

<u>Etiologies</u>

- Decrease in the size of the compartment (from external compression)
  - Tight dressings or cast
  - External pressure e.g. prolonged crush injury
  - Closure of fascial defects
- Increase in volume within a compartment
  - Bleeding (bleeding dyscrasia or vascular injury)
  - Increased capillary permeability (leaky capillaries)
    - Post-ischemic or post-traumatic swelling
    - Exercise
    - Burns
  - Increased Capillary pressure
    - Venous obstruction
    - Exercise

Types of Injuries: Fracture, GSW, Soft-tissue injuries without fracture (crush injuries, muscle rupture contusions), high-pressure injection injuries, fluid extravasation from catheters, pneumatic tourniquet, vascular injuries, reperfusion injuries, sustained pressures (obtunded patient), infection, burns, snake bites.

Tissue Pressures

Normal arterial pressure (diastolic) 60-90 mm Hg
Normal venous pressure – 5 mm HG
Normal tissue pressures in muscle - < 6 mm Hg

Animal Studies: Pressures > 30 mm Hg for 8 hours result in irreversible changes in muscle and nerve conduction impairment

Pressure Factors – What can be tolerated

Dependent upon
- Duration of the pressure elevation
- Metabolic rate of the tissues
- Vascular tone
- Local blood pressure

Proposed tolerances:

Matsen – 45 mm Hg

Whitesides – 20 mm below diastolic

Mubarak &Hargens: 30 mm Hg, combined with clinical findings
Based on animal studies of 30 mm Hg being sufficient to cause complete ischemia

Several authors: Mean arterial blood pressure – compartment pressure < 30 mm Hg

Time Factors in Tissue Necrosis

Nerve
- 30 min – function abnormalities (paresthesias)
- 12-24 hours – irreversible functional loss

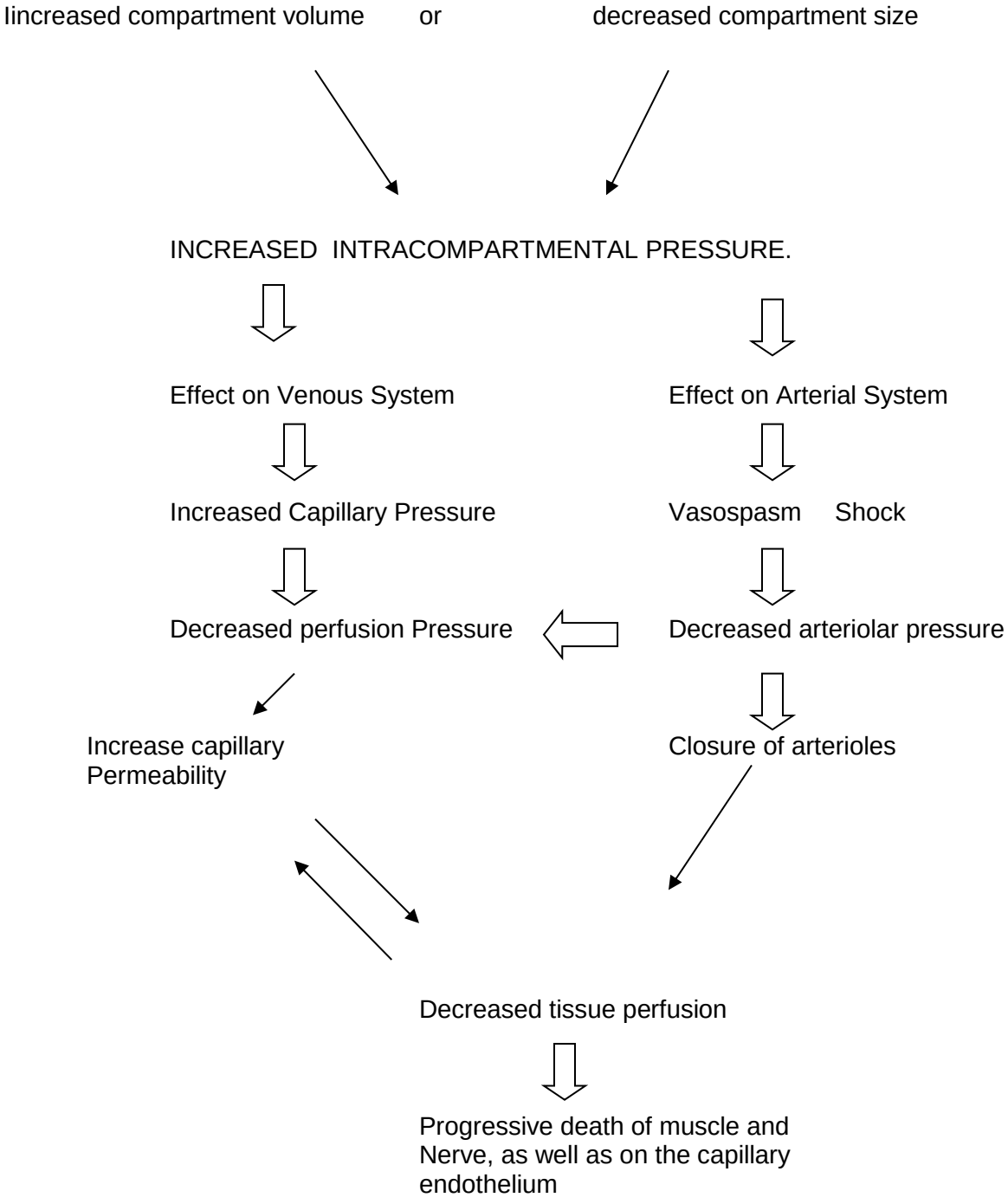
Muscle
- 2-4 hours – functional changes
- 4-12 hours – irreversible loss of function
  - Hargens et al: 3-4 hours – irreversible muscle cell damage seen at an ultrastructural level – mitochondrial swelling, condensed nuclei, disordered myofilaments
    Nucleotide degeneration degeneration
  - Patterson: Muscle contractile function reduced after 5 hrs of ischemia in tourniquet model.
  - Several authors: Histologic and gross changes in muscle noted after 6-8 hours of ischemia, including infiltration of leukocytes into the muscle tissue

Capillary Endothelium
- 3 hours – sufficiently affected produce post-ischemic swelling of 30-60%
- Ischemia-Reperfusion models:
  - After 3 hours of ischemia, reperfusion hyperemia is blunted and microvascular perfusion is diminished by 65-75%.
  - "Post-ischemic slow reflow or no-reflow phenomenon"
    - Proposed Mechanisms:
      - -Capillary endothelial swelling

-Altered expressions of endothelial tethering and adhesions molecules, leading to microvascular plugging by leukocytes (white clot) as well as further endothelial damage by reactive oxygen metabolites
-Post-ischemic dysfunction of nitric oxygen, resulting in failure of the vasodilatory mechanisms

The post-ischemic changes in the microvascular permeability leads to further compromise of the reperfused skeletal muscle and nerve tissue

Matsen's Unified Concept of Compartment Syndrome



Volkmann's Contracture

Pathogenesis of Volkmann's Contracture

Ischemia and Necrosis

More than 4 hrs of experimentally produced ischemia results in irreversible changes within the muscle

Nerve tissue is also sensitive to ischemia. Prolonged ischemia can result in permanent fibrotic changes within the nerve, resulting in distal motor and sensory impairment.

Muscle excursion and mobility are limited by:

-Fibroblastic proliferation within the muscle which occurs after muscle necrosis
contracture in both muscle length and width progresses over a 6-12 month period
-Adherence of the necrotic muscle to adjacent bone and surrounding structures

Contracture of the necrotic muscle

-can also result in compression of the nerves traveling through the affected compartment
-focal hourglass constriction of the nerve in areas of dense fibrosis can bee seen.

Deformity Associated with Compartment Syndrome

-Generally, the deepest compartments experience the highest interstitial pressures during compartment syndrome and therefore experience the greatest amount of muscle necrosis and fibrosis.
-For the forearm, this is the deep flexor compartment containing the FDP and FPL muscle origins
-The deep compartment of the forearm experiences the highest interstitial pressures.
-Collateral circulation to the superficial muscles is less susceptible to compromise than that to the deep compartment
-"The ellipsoid concept" of infarct proposed by Seddon and Tsuge –central necrosis of the forearm muscles.

The characteristic deformities associated with ischemic contracture develop over weeks to months:

Forearm: pronation, wrist flexion, clawed position of the fingers (MP extended, IPs flexed) Thumb flexion and adduction.

"Intrinsic minus position" - MP hyperextension, and IP flexion.
-associated with "severe" Volkmann's where both flexor and extensor compartments are affected

Hand: Intrinsic "plus position"

Arm: Depends on the compartment that are most affected. If both flexor and extensor compartments equally affected, resultant deformity is usually flexion.

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Volkmann's Ischemic Contracture

Richard von Volkmann
1830-1889

1881 Described ischemic muscle paralysis and contracture, later to be known as "Volkmann's Contracture"

Etiology of Volkmann's

Problems of arterial inflow
Problems of venous outflow
Combined etiology

Volkmann's contracture, unlike compartment syndrome, is the result of prolonged muscle and nerve ischemia resulting in irreversible changes in the muscles and nerves.

Both acute compartment syndrome and therefore the potential sequelae of compartment syndrome (Volkmann's contracture) are more commonly seen in pediatric patients.
Specific associated injuries are displaced supracondylar humerus fractures (Gartland 3) and "floating forearm fractures" (combined fractures of the distal humerus and distal radius). However, even ***minor*** radiographic injuries can result in compartment syndrome.

Neonatal "compartment syndrome" or more accurately neonatal Volkmann's contracture has also been described. This may be due to intra-

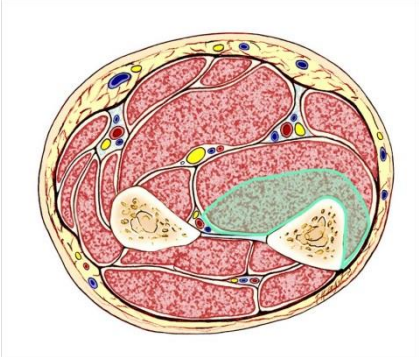
uterine positioning causing prolonged ischemia either from pressure or arterial occlusion.

Classification

Tsuge – most commonly used
based on location and extent of muscle contracture

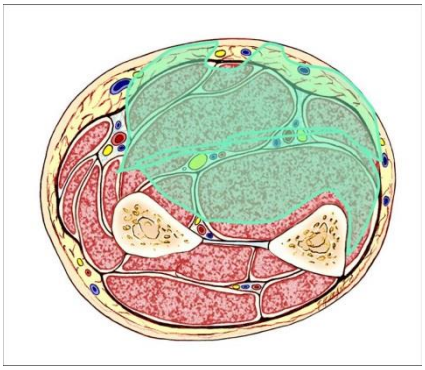
Mild

In mild cases, the degeneration and contractures are limited to the FDP to 2 or 3 fingers. The middle and the ring are the most frequently affected. Sensation is usually present.



Moderate (Classic)

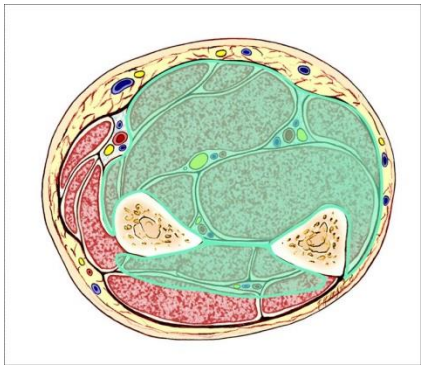
In the classic cases, the contracture involves the FDP and FPL, but may also partially involve the FDS and the Wrist Flexors with sensory changes of the median and ulnar nerves, as well as paralysis of the intrinsic muscles.



Severe

In severe cases, there is muscular degeneration of all Flexors and partial extensor involvement with severe degrees of neurologic disturbance and contracture.

Sensory feedback is usually impaired



Treatment

Acute compartment syndrome

Requires immediate surgical treatment with fasciotomy.

Despite emergent treatment, residual muscle injury may persist and require later treatment.

Mild and Moderate

Initial treatment is conservative. This includes splinting, serial casting, and a formalized therapy program. This should continue until the clinical recovery reaches a plateau, typically between 6-12 mos.

Further treatment depends on residual disability and contracture after clinical plateau.

Described Procedures: can be isolated or combined

Bone: arthordesis, carpectomy
PRC and shortening or radius and ulna
Soft Tissue: Excision of infarted muscle, fractional or z-lengthening of the affected muscles, neurolysis, tendon transfer, and flexor muscle slide. Release of fixed contractures of the joints may be necessary as well.

Intrinsic contractures of the hand pose challenging problems and tendon release at insertion site or muscle release off the bone origin has been used for thumb.

For finger contractures, intrinsic slide off of the metacarpal are more difficult and less predictable. Lateral band release at the mid proximal phalanx has been used.

Severe

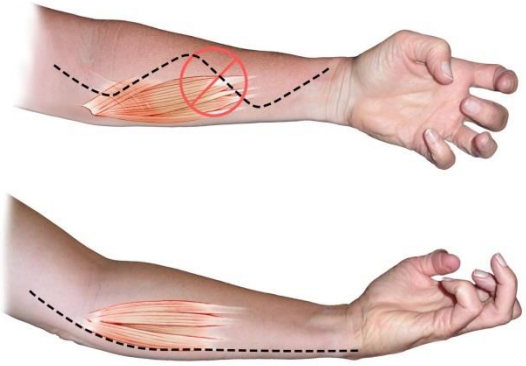
Cases of severe compartment syndrome have historically been very challenging with limited options for functional

improvement. The introduction of functional free muscle transfer has significantly improved outcomes.

Author's Preferred Method

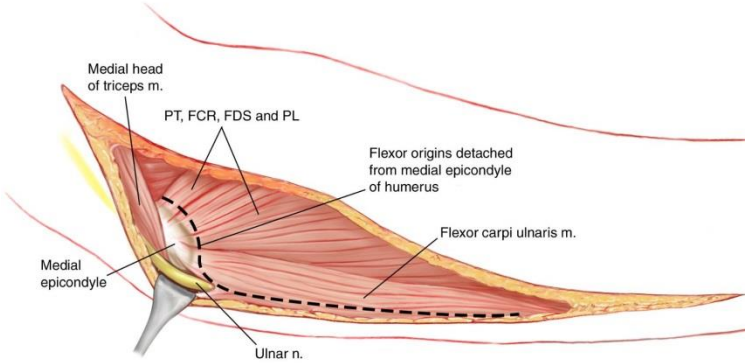
Mild – residual involvement of 2-3 profundus muscles
Limited flexor slide , without neurolysis

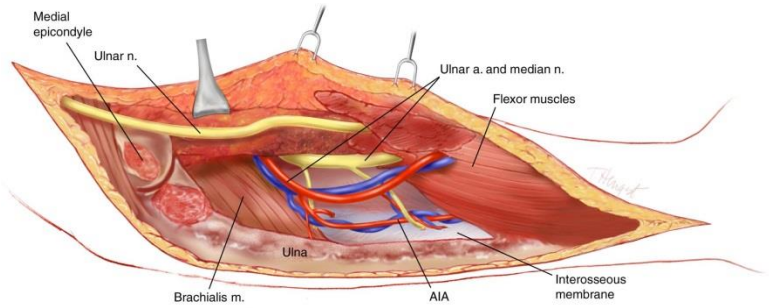
Moderate – flexor origin slide, with ulnar nerve transposition with/without median nerve neurolysis.



Extended ulnar approach

- Easier release of Muscle Origin
- Less destruction and scarring of muscle belly
- Less disruption of venous outflow and superficial nerves
- Allows release of ulnar and radial origin muscles
- Allows release of volar DRUJ capsule





Post-operative management – flexor slide
Patients evaluated by OT for ROM and Sensation
Pre and post op

- Long Arm Cast : week 4- 6
- Full-time resting splint : week 6-12
  - Serial splinting if unable to achieve full extension
  - OT 3 times per week
- After 12 weeks - Night only splinting
  - Night splinting continued until skeletal maturity
  - OT 1X per week and continued until full ROM or plateau

Severe

Acute compartment syndrome with liquifactive necrosis of the muscles as well as chronic Volkmann's with no residual muscle function is best treated with functional free muscle transfer.

Timing for treatment in the case of acute liquifactive necrosis is for immediate reconstruction.

Preferred muscle donors: gracilis, latissimus, rectus femoris, and tensor fascia lata.

- important to reestablish correct muscle resting length
- establish strong origin and insertion sites

Recipient nerves and vessels:

- Best recipient nerve is AIN
- May be fibrotic due to Volkmann's
- Should be biopsied and assessed for nerve architecture before harvesting gracilis (or donor muscle**)**
- Recipient vessel should be out of the zone of injury.

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Medicolegal

- Compartment syndrome represents a surgical emergency
- Delays in management can result in permanent disability to the limb.
- 6% of all orthopedic malpractice claims against orthopedic surgeons are related to compartment syndrome. Of those, more than 5-% are ruled in favor of the patient.
- There is a correlation between delay to surgery and payment size to the plaintiff

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Pathoanatomy, Diagnosis, Surgical Treatment Acute Compartment Syndrome

Scott Kozin, MD

2019 ASSH Annual Meeting

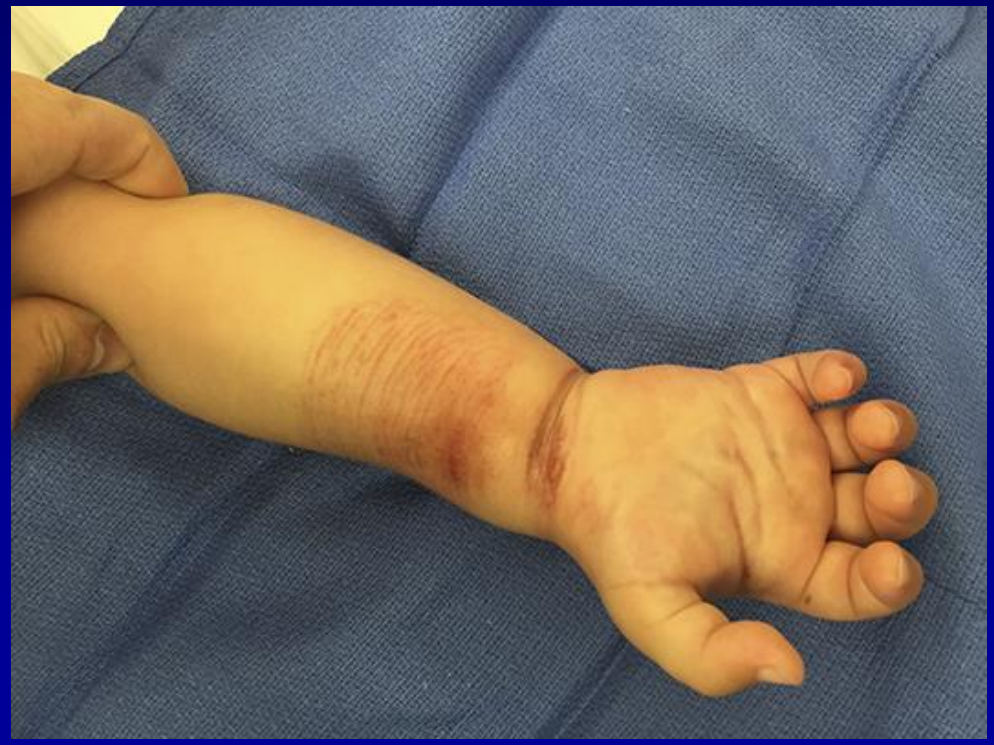
"Oh that's Tight" Compartment Syndrome and Volkmann's Contracture. From Diagnosis to Treatment , including management of the legal aspects of Compartment Syndrome IC #36

Stephen

- 18 month-old Amish child
- Hand inadvertently caught in wringer 5 hours ago
- Extreme pain and inconsolable
- X-rays normal

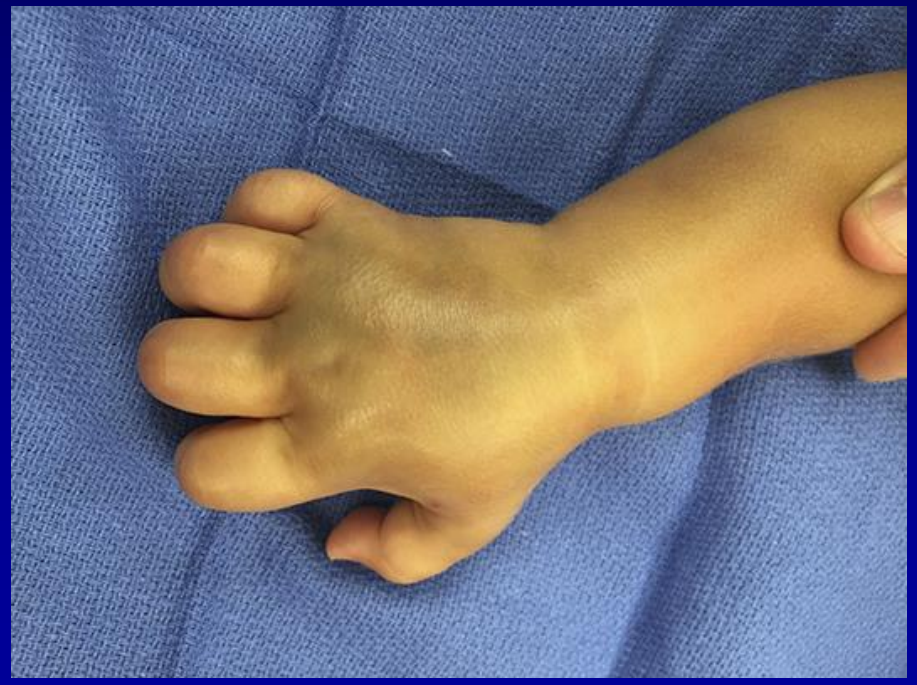
Examination

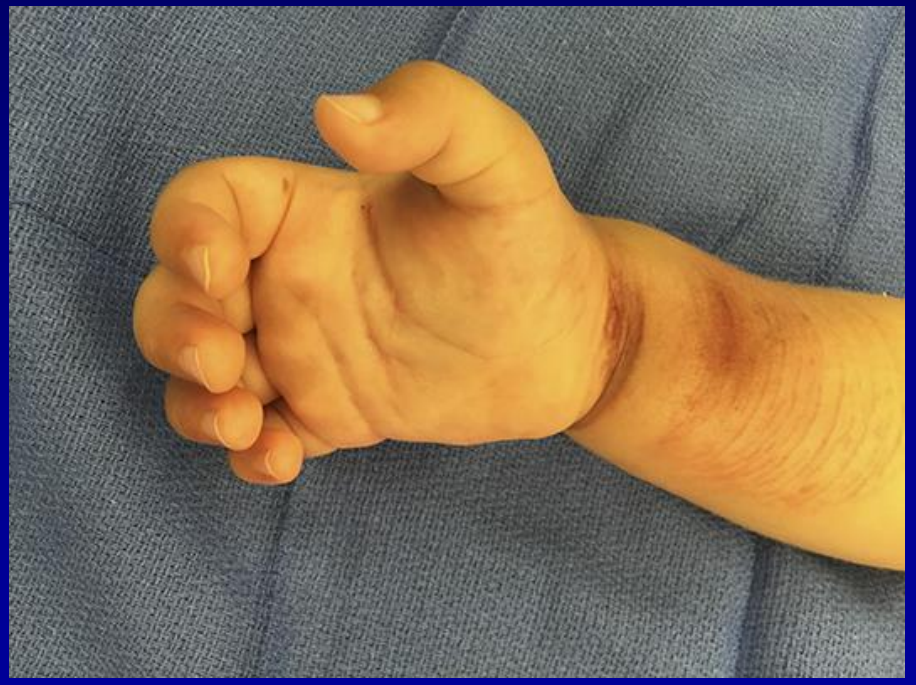
- Reluctant to let anyone touch his right arm
- Zone of demarcation in the distal 3rd of the forearm where the wringer ended
- Tense and swollen



Examination

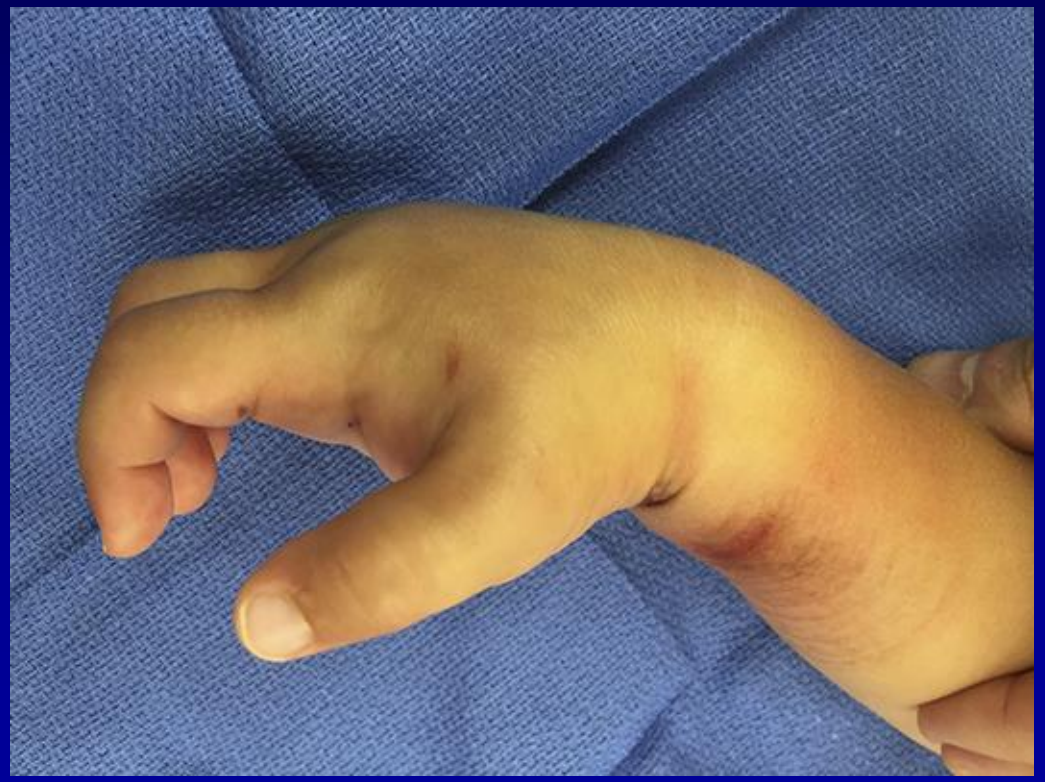
- Intrinsic minus posture, will not move his digits
- Ecchymosis along the dorsal and palmar aspect of his hand





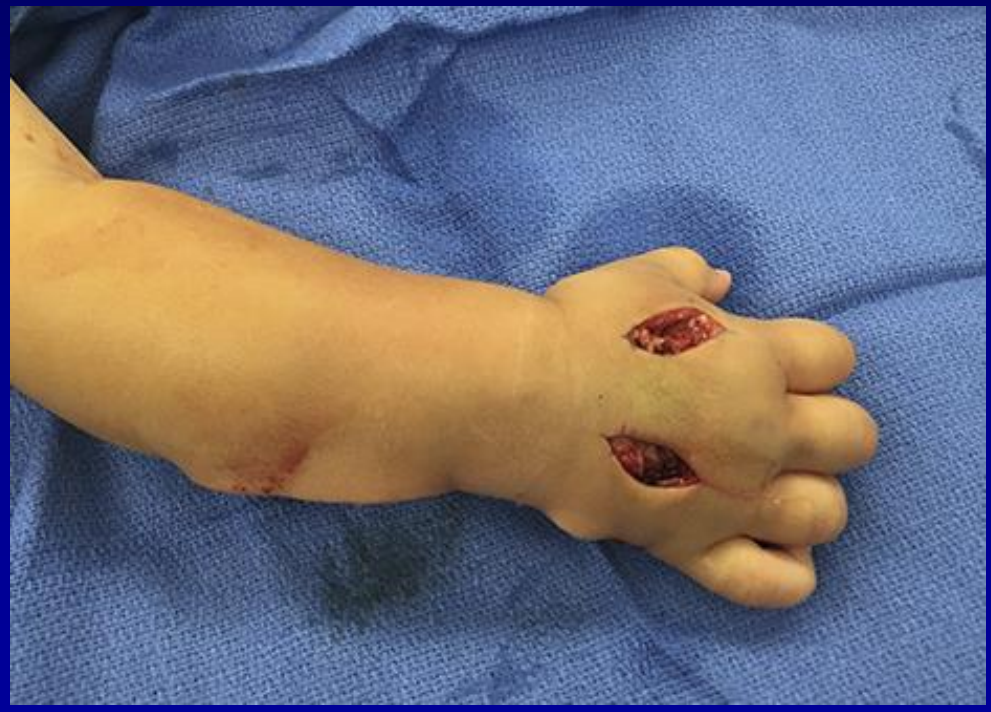
Diagnosis/ Treatment

- Compartment syndrome, right forearm and hand
- Immediate fasciotomy



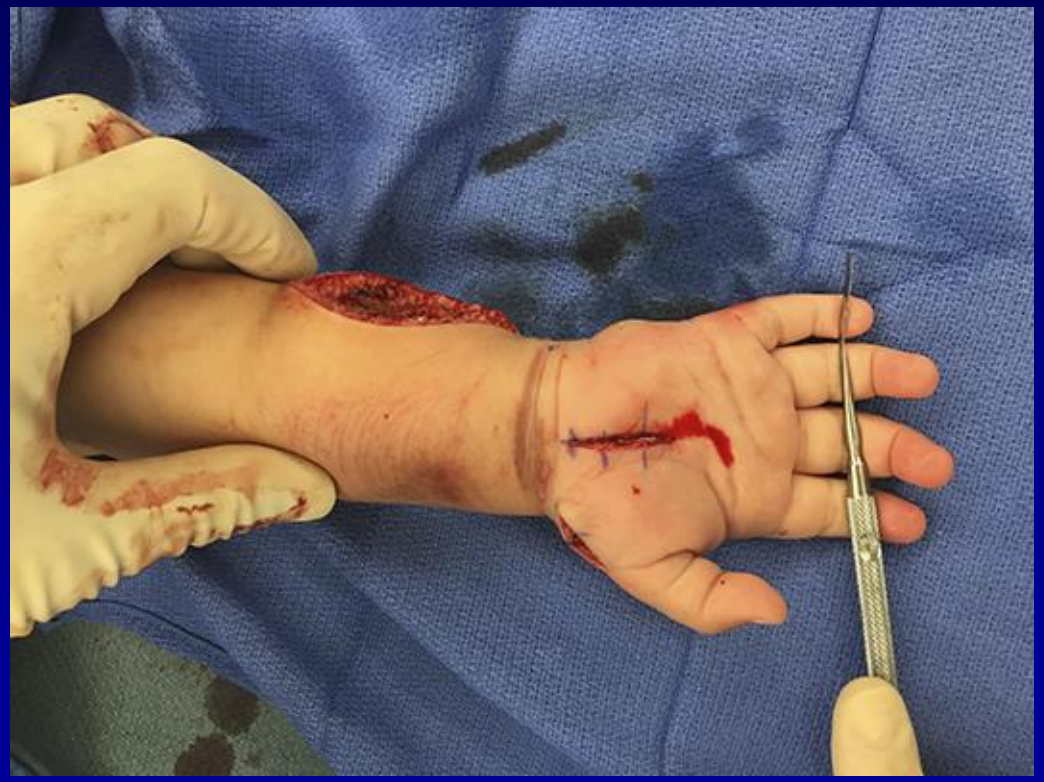
Surgery- Hand

- Two dorsal incisions to access to the dorsal and volar interossei compartments and the adductor muscles



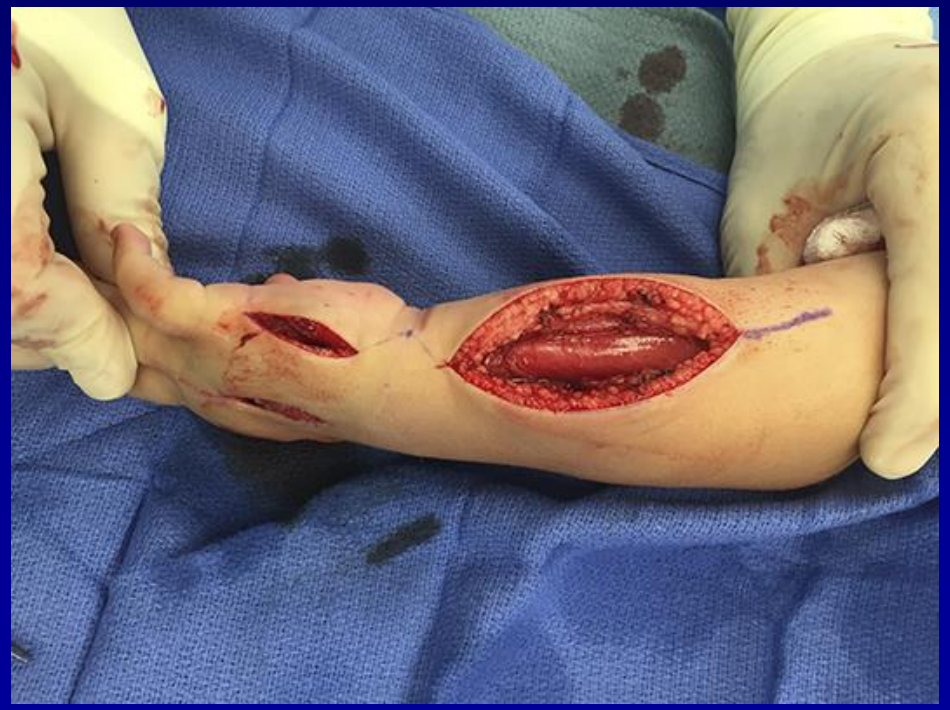
Surgery- Hand

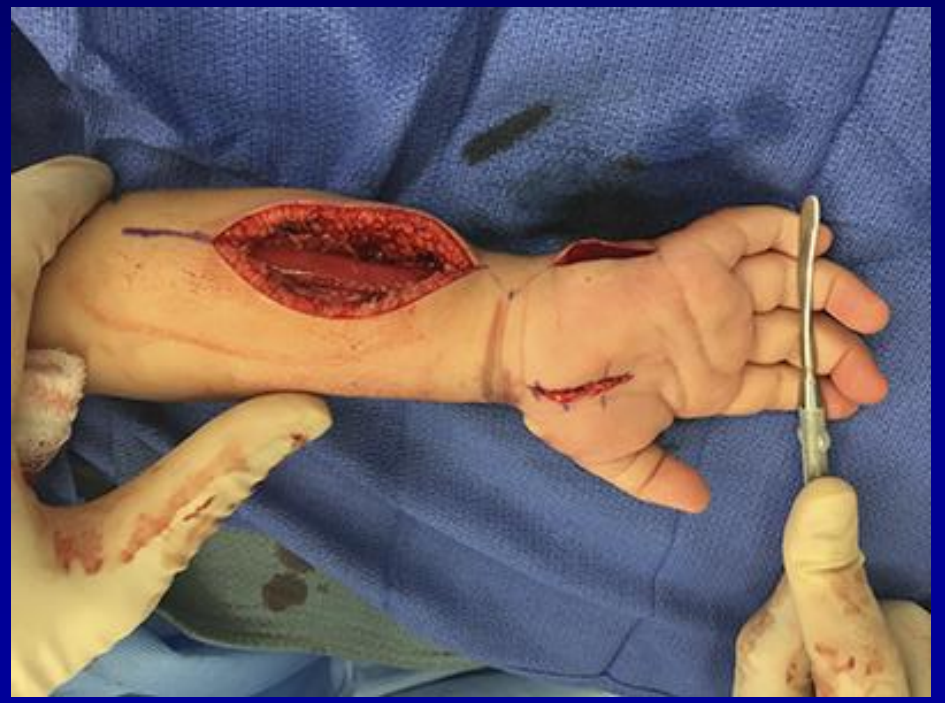
- Thenar and hypothenar incisions



Surgery- Forearm

- Ulnar border incision for fasciotomy of the forearm
- Separate carpal tunnel incision

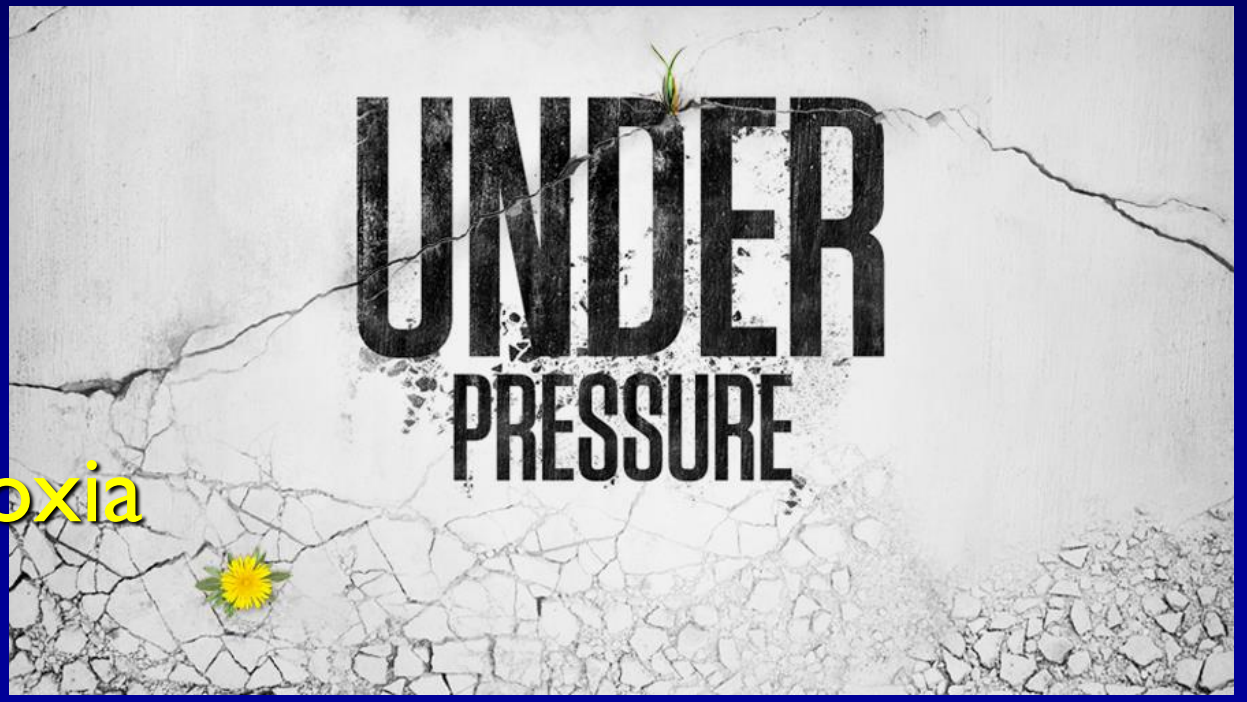




Follow-up at 2 weeks

Compartment Syndrome

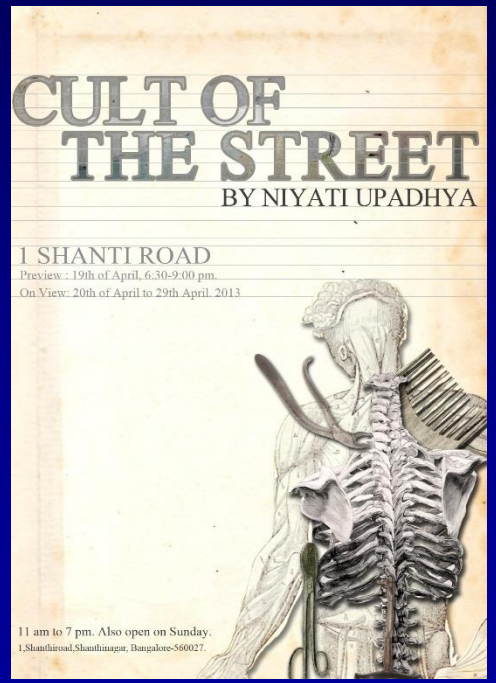
- Central factor = Increase tissue pressure exceeds the venous pressure and impairs blood outflow.
- External compression
- Internal increased volume

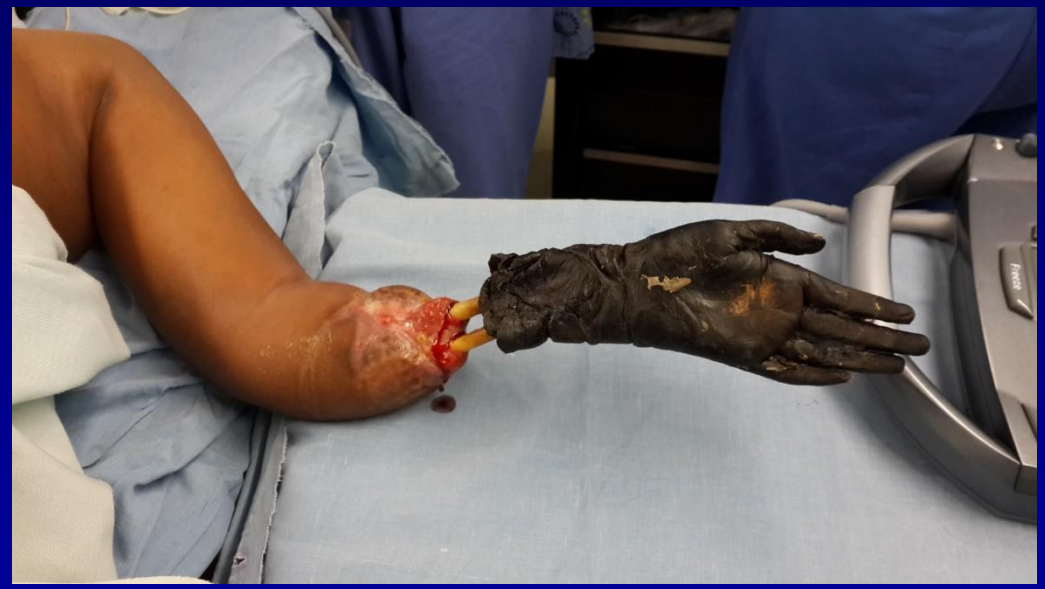


- Final pathway= cellular anoxia

External Compression

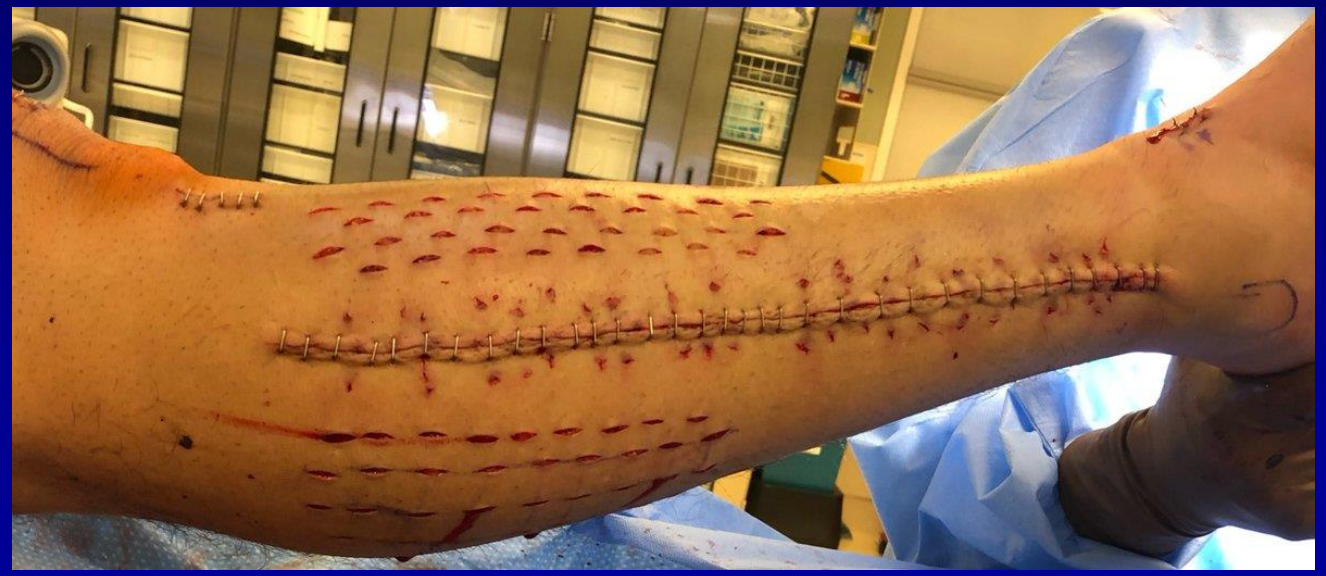
- Tight dressings or casts





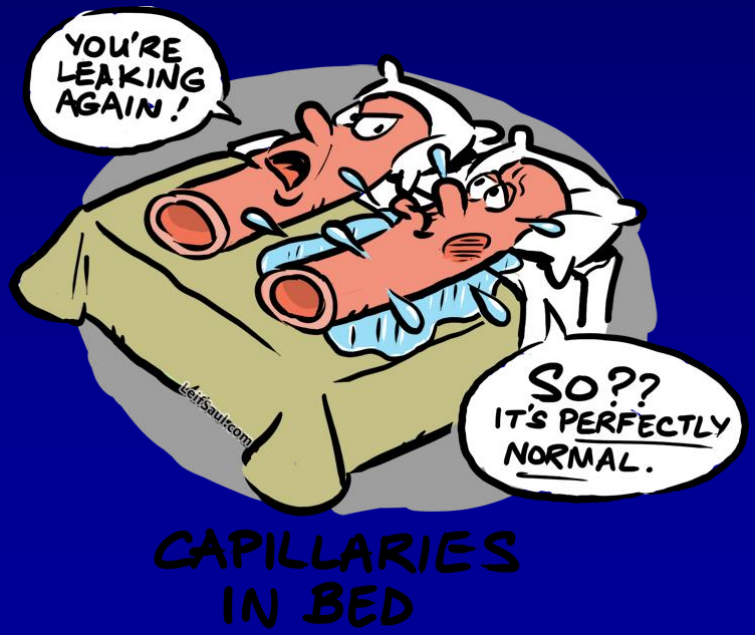
External Compression

- Prolonged pressure (e.g., crush)
- Tight fascial closure



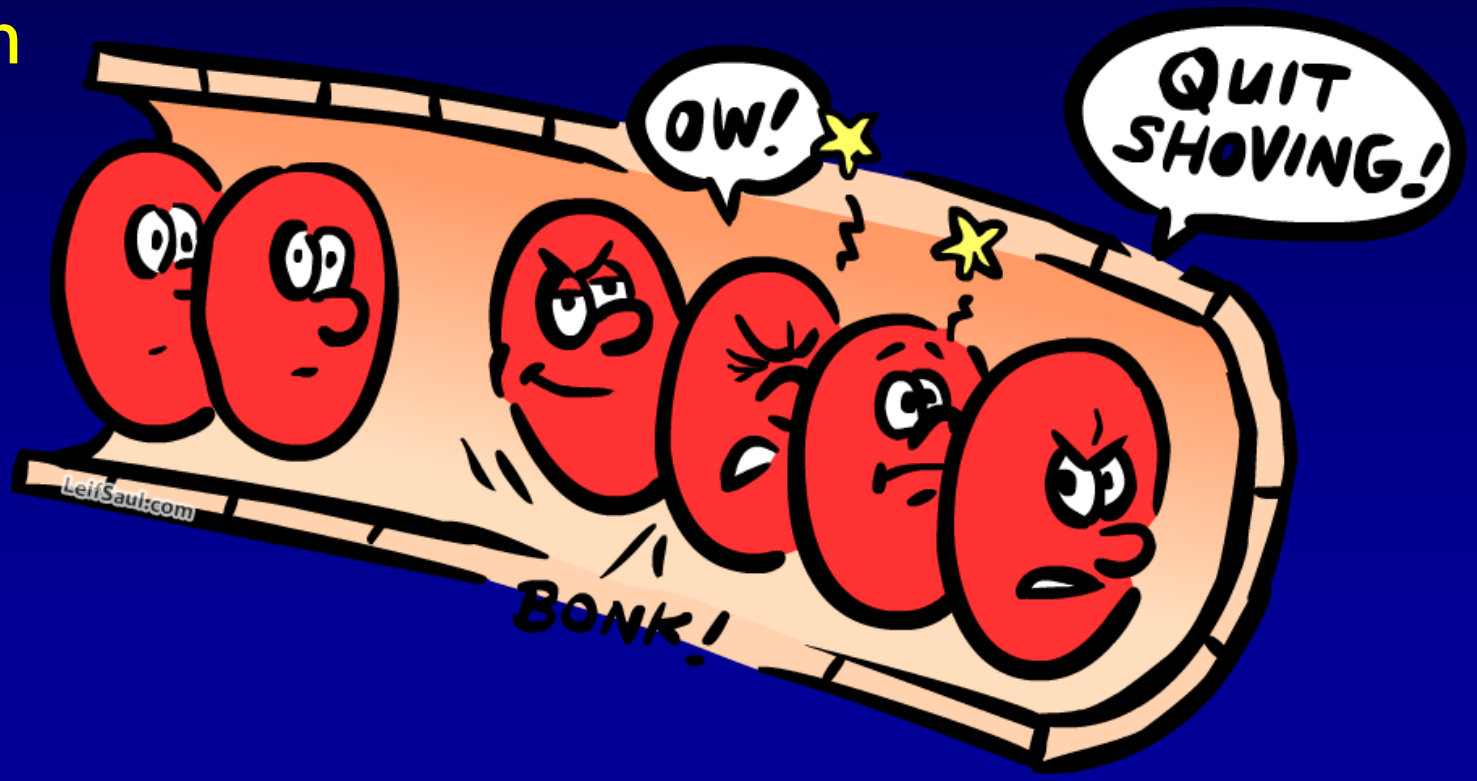
Internal Increased Volume

- Bleeding
- Increased capillary permeability (leaky capillaries)
  - Post-ischemic or post-traumatic swelling
  - Exercise
  - Burns



Internal Increased Volume

- Increased Capillary pressure
  - Venous obstruction
  - Exercise

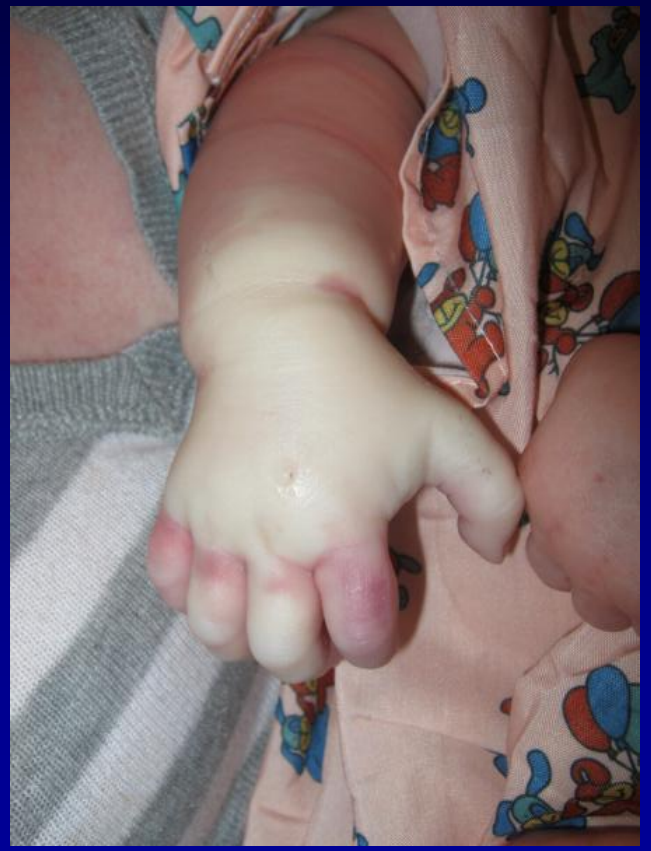


Mechanism of Injuries

- Fracture
- GSW
- Soft-tissue injuries without fracture (crush injuries), muscle rupture contusions)
- High-pressure injection injuries

Mechanism of Injuries

- IV Fluid extravasation
- Pneumatic tourniquet
- Vascular injuries ± reperfusion injuries
- Sustained pressures (obtunded patient)
- Infection



Mechanism of Injuries

- Burns
- Snake bites
- Intrauterine

Tissue Pressure

- Normal tissue pressures in muscle

  < 6 mm Hg

- Animal Studies:

  Pressures > 30 mm Hg for 8 hours → irreversible changes in muscle and nerve

What Pressures Can Be Tolerated?

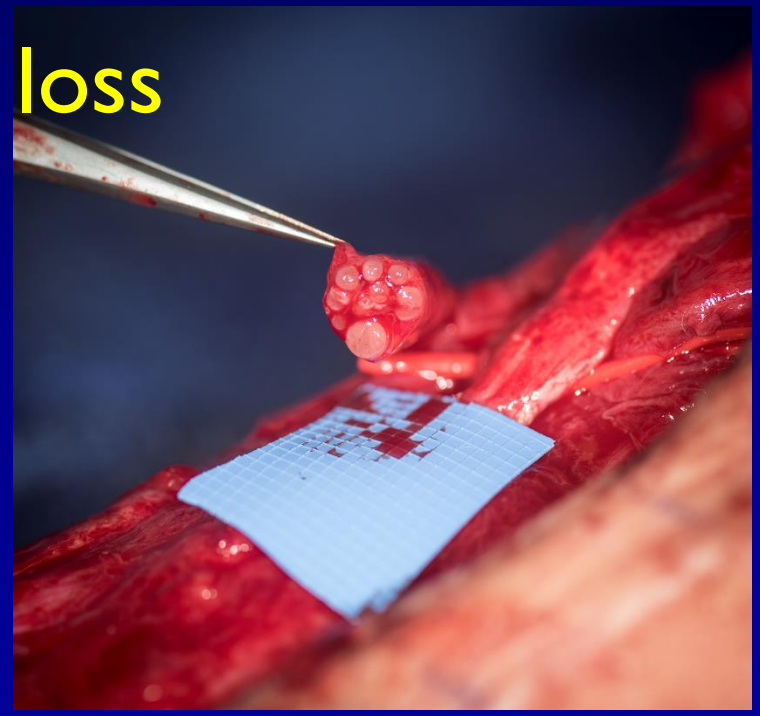
- Factors:
  - Duration of the pressure elevation
  - Metabolic rate of the tissues
  - Vascular tone
  - Local blood pressure

Tissue Pressure

- Proposed tolerances:
- Matsen – 45 mm Hg
- Whitesides – 20 mm below diastolic
- Mubarak & Hargens- 30 mm Hg
- Several authors: diastolic blood pressure and the compartment pressure (delta pressure) of 30 mmHg or less is the threshold for diagnosing ACS Hg

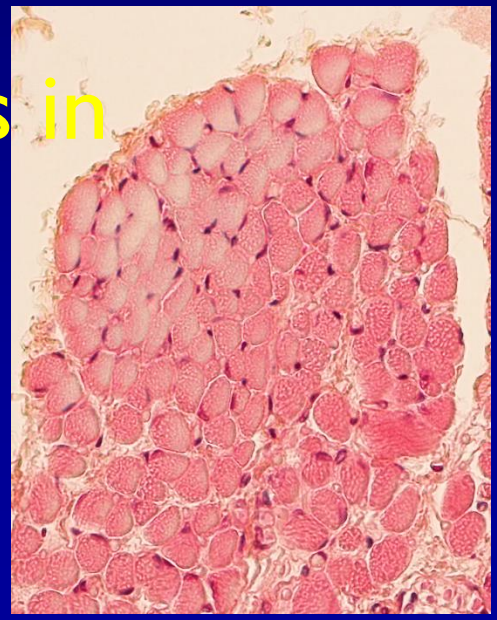
Time Factors in Tissue Necrosis

- Nerve
- 30 minutes – function abnormalities (paresthesias)
- 12-24 hours – irreversible functional loss



Time Factors in Tissue Necrosis

- Muscle
- 2-4 hours – functional changes
- 4-12 hours – irreversible loss of function
- Several authors: Histologic and gross changes in muscle after 6-8 hours of ischemia



Time Factors in Tissue Necrosis

- Capillary Endothelium
- After 3 hours of ischemia, reperfusion hyperemia is blunted and microvascular perfusion is markedly diminished (65-75%).
- Microvascular plugging
- Microvascular permeability changes leads to further compromise of the reperfused muscle and nerve

Compartment Syndrome Diagnosis

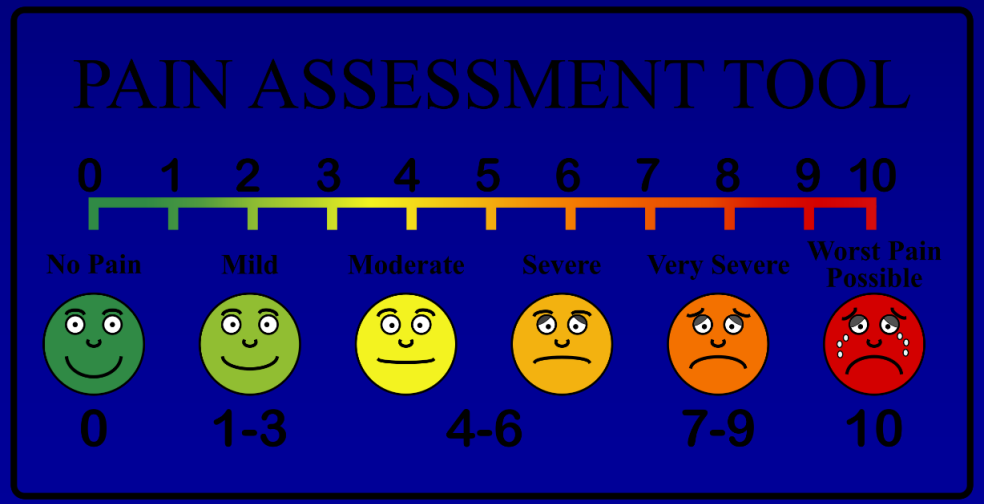
- Physical examination
- Physical examination
- Physical examination

Signs & Symptoms
Adults versus Children

5 Ps	3 As
Pain	Analgesia
Paresthesias	Anxiety
Pallor	Agitation
Paralysis	
Pulselessness	

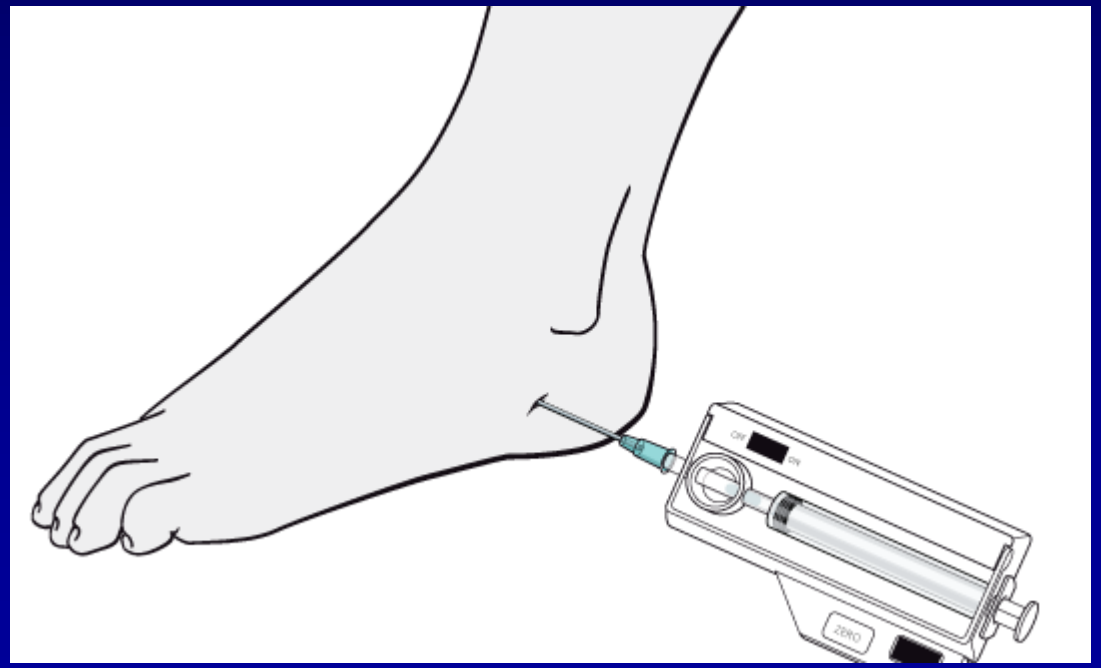
The As not the Ps!!!!!!

- Analgesia
- Anxiety
- Anxious



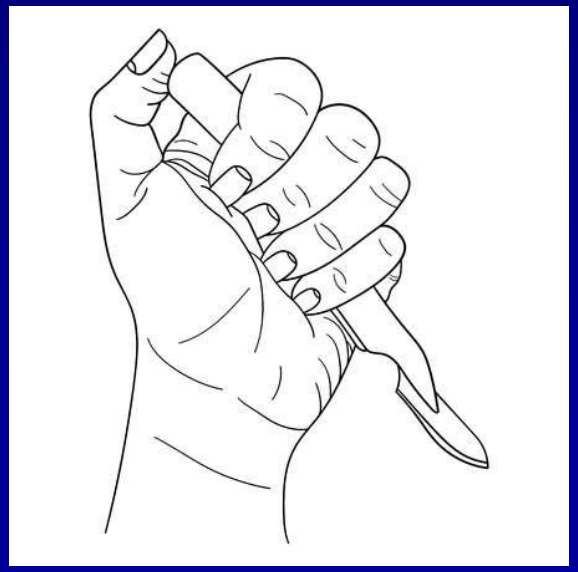
Compartment Pressure

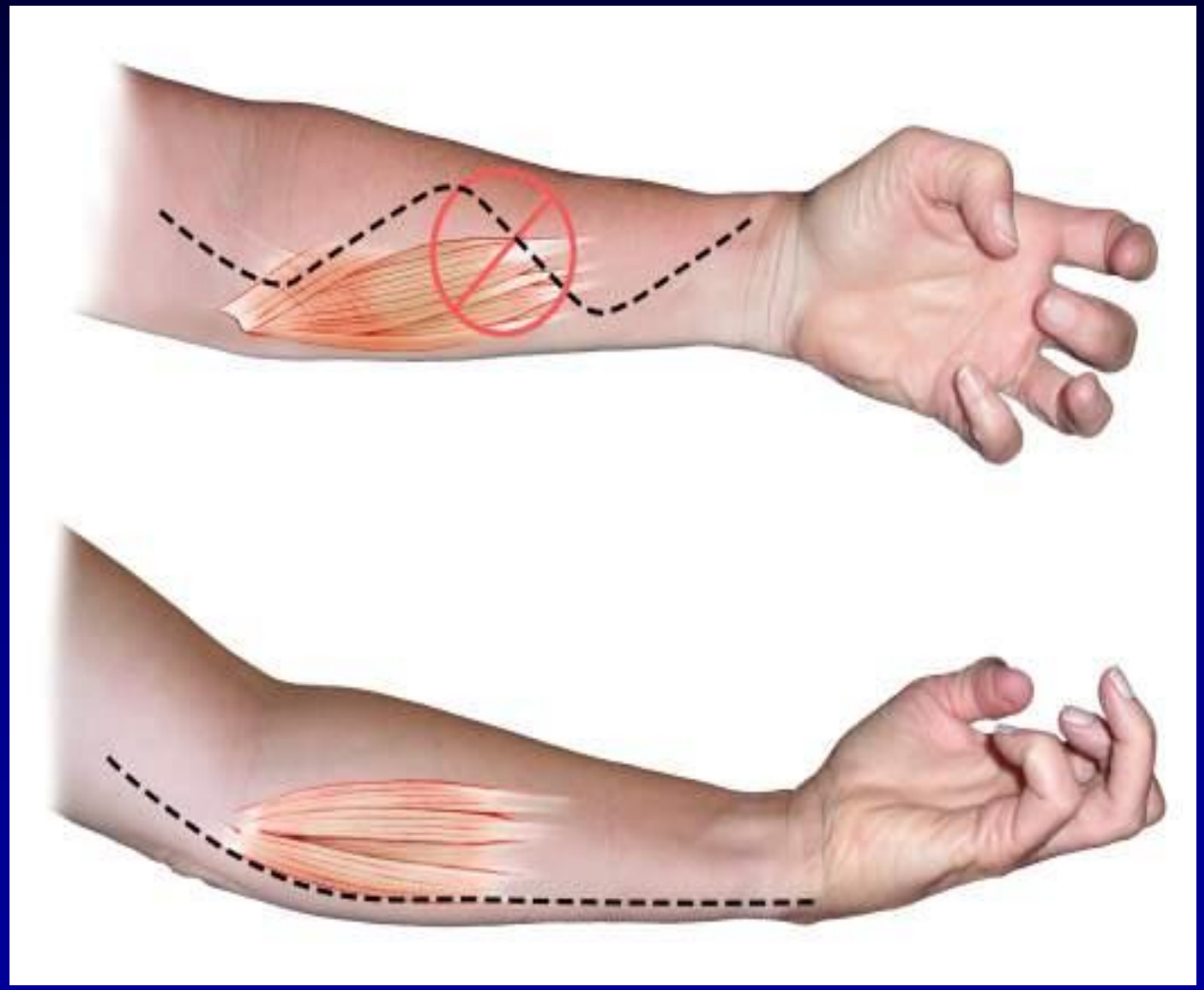
- What is the role?
- Compartment pressures are not required for diagnosis
- Adjunct in the diagnosis
- Pressure > 30 mm Hg



Treatment

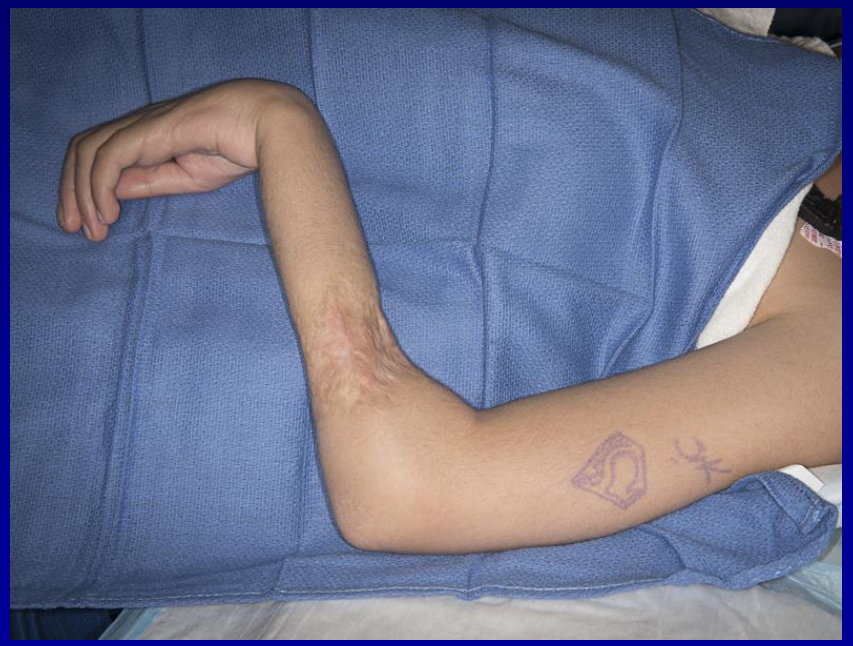
- Fasciotomy
- Fasciotomy
- Fasciotomy

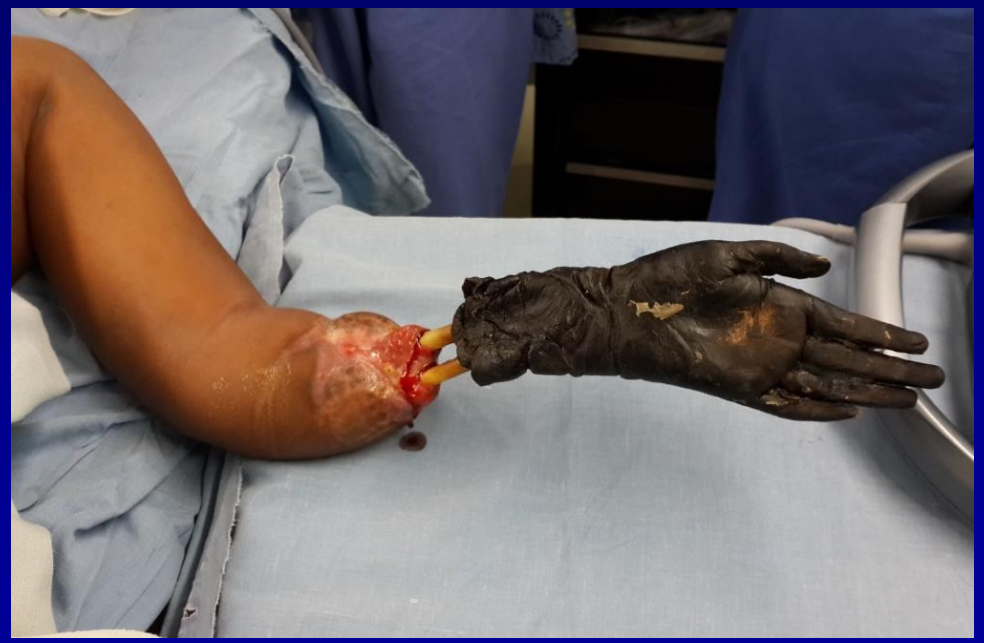




Outcome

- Directly related to duration of compartment
- Don't delay





Thank You