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Fracture Classification + Healing

John McMaster, 2019

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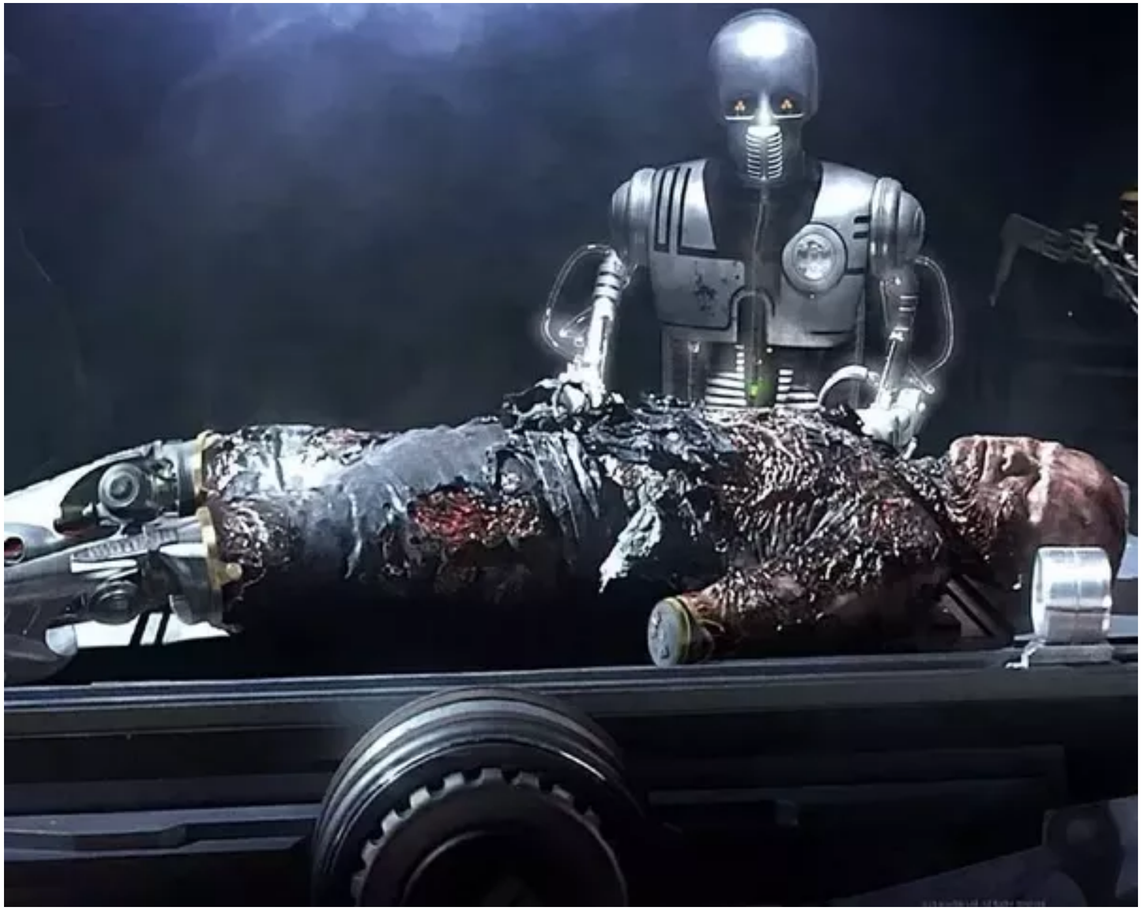
Disclosure

- This talk is based on contemporary knowledge
- The contemporary knowledge represents a simplistic view that may in time be refuted.
- It needs to be simple so that we can remember and apply it.
- It works!
- We have been chosen for this divine work.

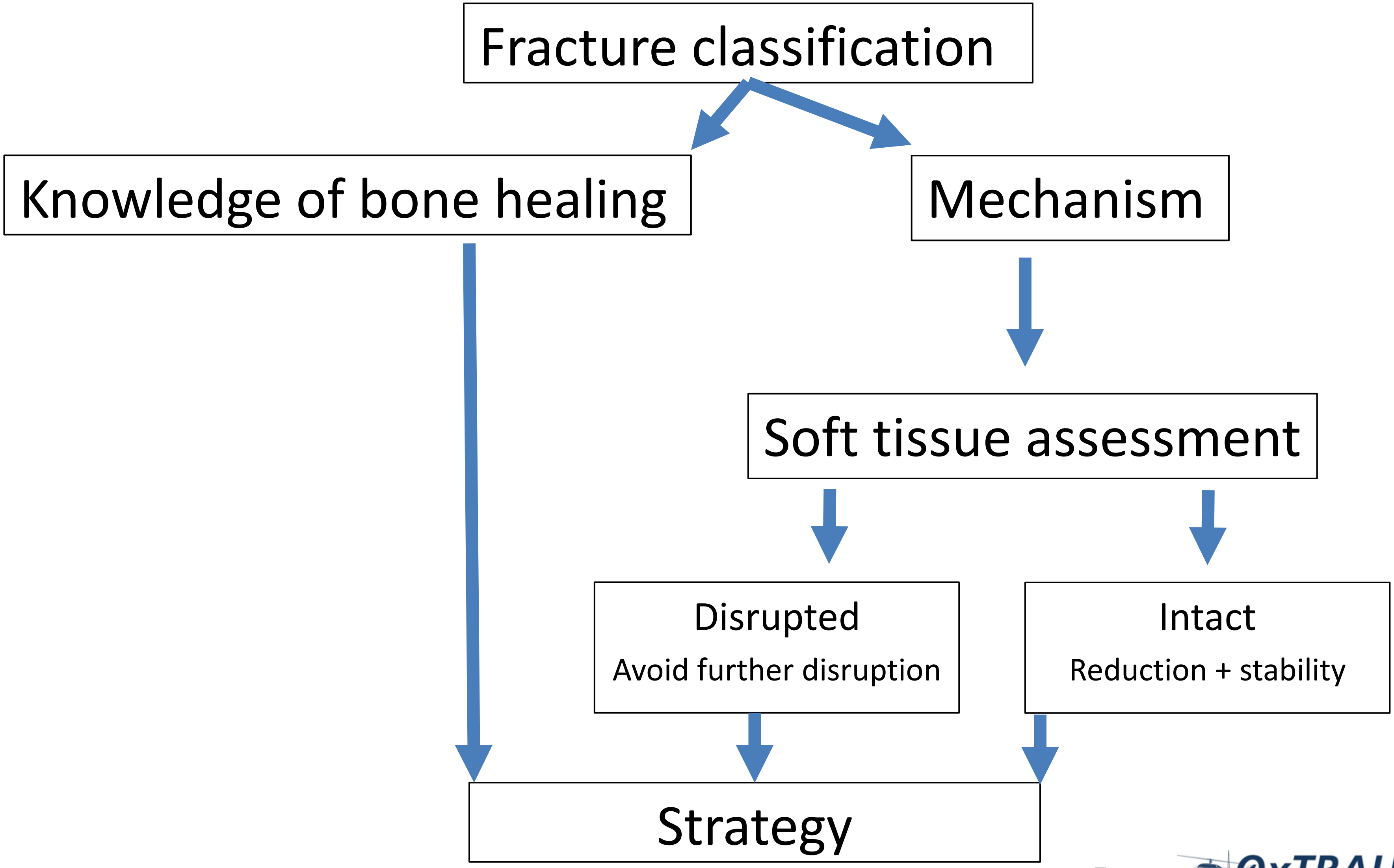
Why?

- Tendency to treat common fractures by rote
- You may give the right treatment but for the wrong reasons
- Permutations of trauma endless
- Using first principles uses less brain space

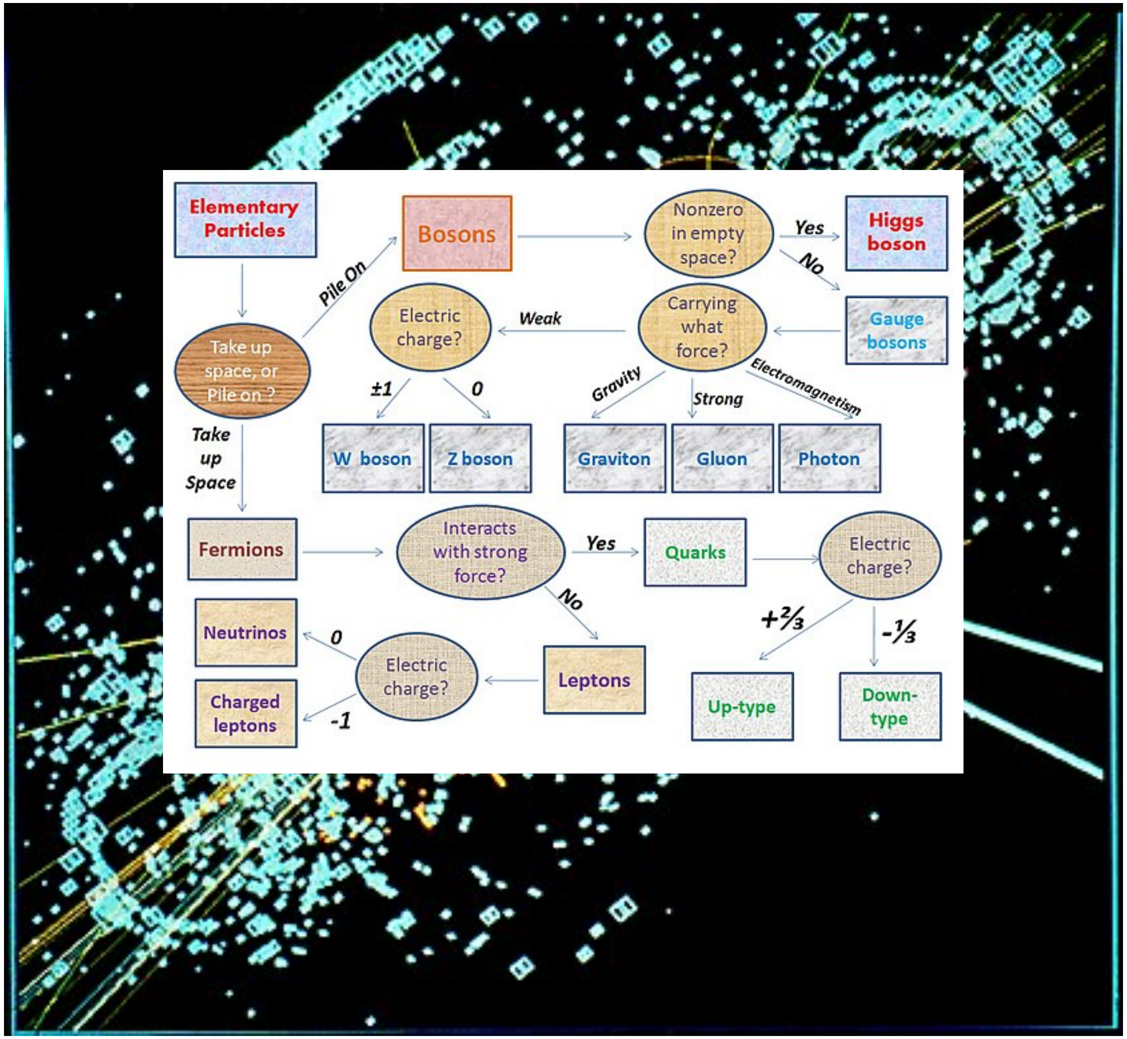
Fracture Management Algorithm



Fracture Management Algorithm



Standard Model of Particle Physics



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Unified Theory of Bone Healing and Non-Union

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1 - Clavicle Fracture

- When can I start ROM?

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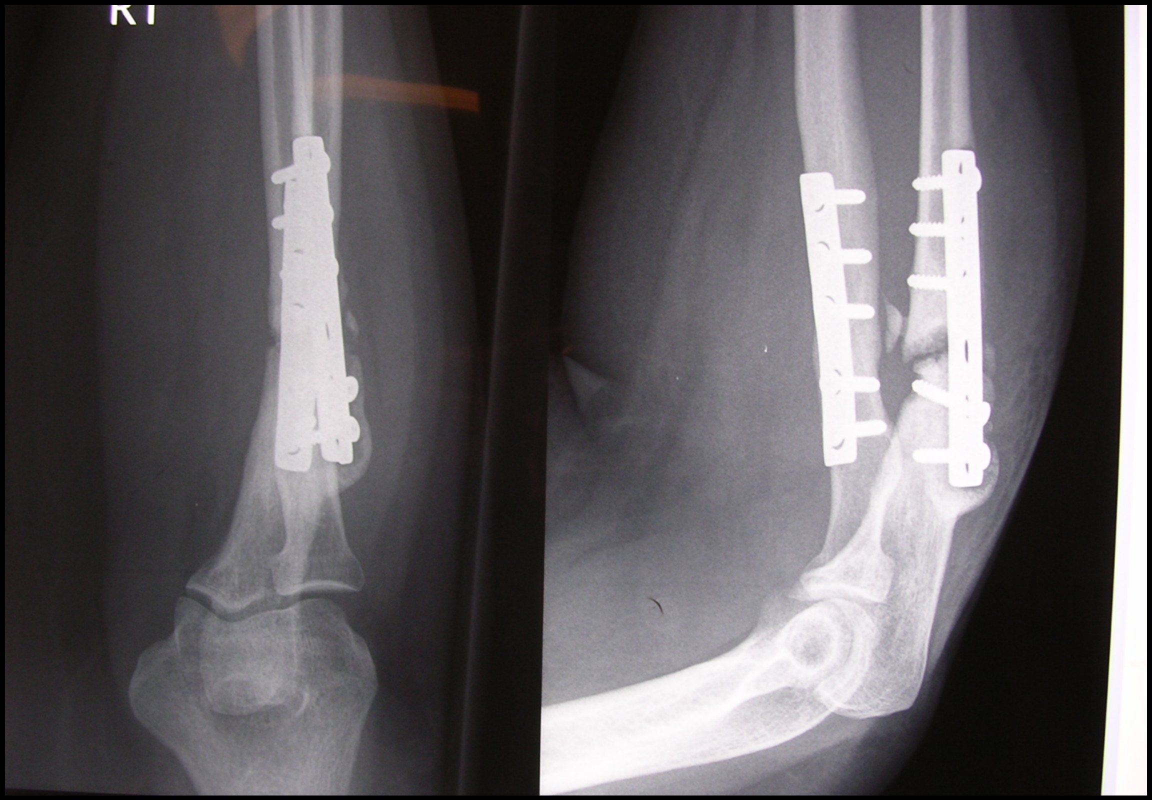
#2 – Non union

- Where do you start?
- (How did we get here?!)



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How does bone heal?

Ah ………. haematoma .. callus … growth factors… Perren's strain theory ….

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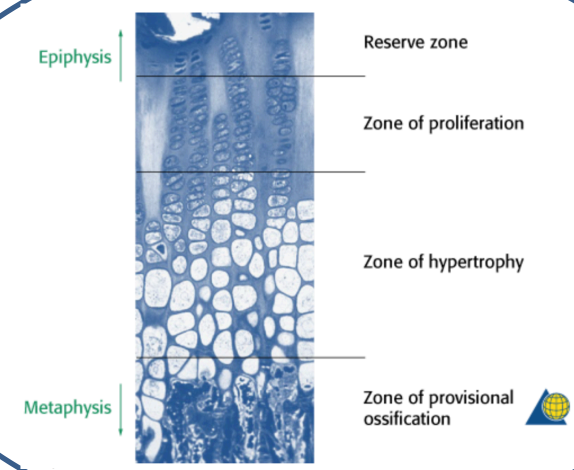

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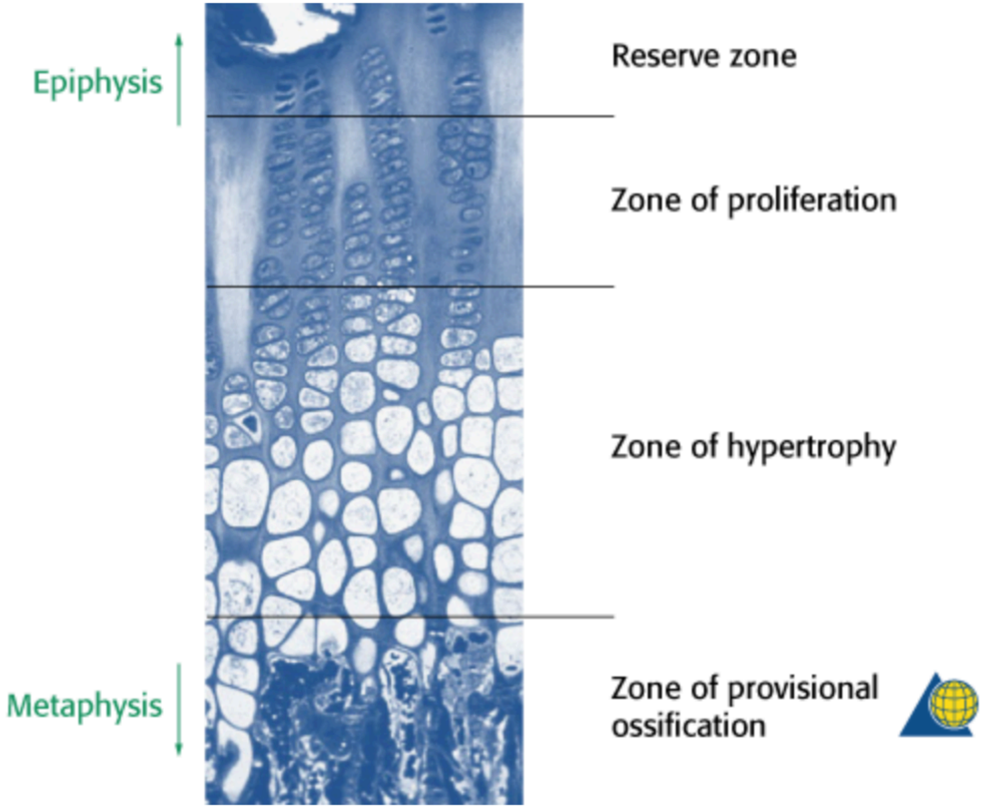
Salter Harris Classification
+
Bone Healing

(Two for One)



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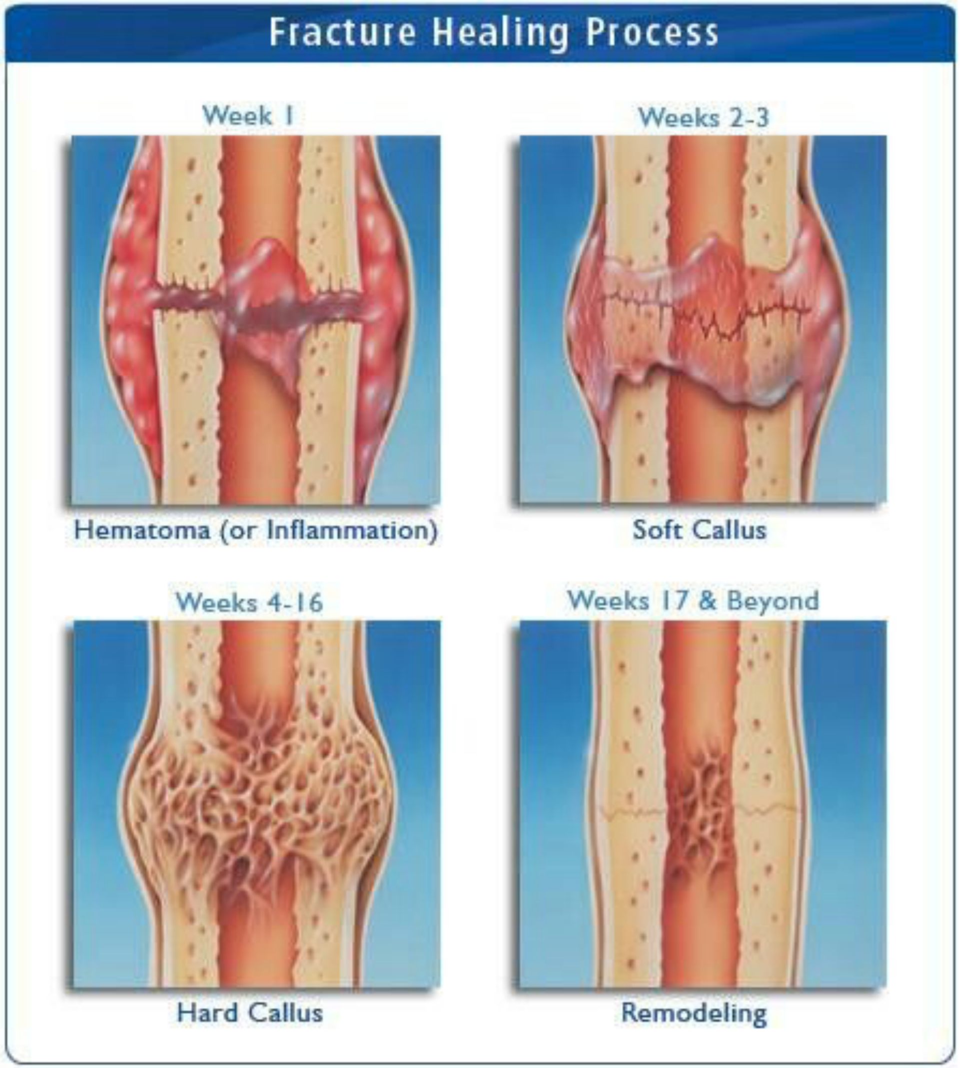
FRCS (Tr+Orth)

We are the chosen ones …..

All fractures



Exam answer

How does bone heal?

Bone healing relies on the appropriate biologic and mechanical conditions.

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Lets break this down!

Biologic
- Cytokine
- Skeletal progenitor
- Extracellular matrix
- Vascular

Mechanical
- Perren
- Wolff

Cytokine Response

- **Fracture →** Reduced stability, oxygen tension and available nutrients

  ↓
- **Initial inflammatory response** - PDGF, Transforming growth factor beta (TGFb), IL 1 and IL 6
- **Cytokines expressed by**
  - macrophages , degranulating platelets, mesenchymal, osteoprogenitor cells, collagenous matrix

  - **Fibroblast Growth Factor (FGF-1 + 2)** mitogenic and angiogenic effects on fibroblasts, chondrocytes and osteoblasts.
  - **BMPs** play a key role in cell growth, differentiation and apoptosis.
  - **TGFb, FGF, IGF, vascular endothelial growth factor (VEGF), matrix metalloproteinases (MMPs)** regulate ECM degradation and angiogenesis
  - **BMP, TGFb, IGF and osteocalcin and collagen related factors (types I, V and XI)** are expressed widely in the callus as the hypertrophic cartilage is replaced by bone.

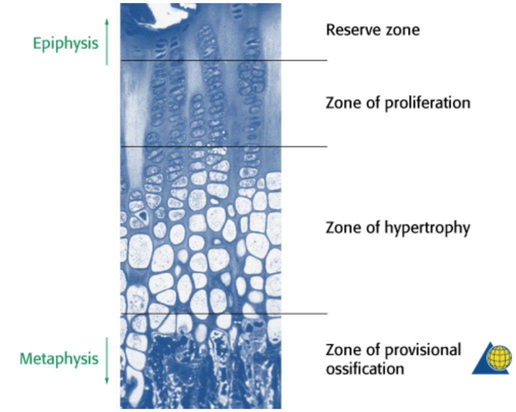
- <u>Systemic</u> - lack of TNF-Alpha (ie. HIV) results in delay of both endochondral and intramembranous ossification
- <u>Enhancement</u> – Systemic optimization, Masquelet induced membrane, BMPs

Skeletal Progenitor Cells

- Proliferation of **mesenchymal cells** (multipotent stromal cell) from
  - bone marrow
  - periosteum,
  - local muscle
  - soft tissue
  - vasculature.
- **Chondrocytes** proliferate, mature and progress to hypertrophy.
- **Osteoblasts** are specialized, terminally differentiated products of mesenchymal stem cells.

- Systemic - smoking
- Enhancement – Systemic optimization, autologous bone graft, (LIPUS)

Extracellular matrix



- **Mesenchymal cells** and **fibroblasts**
  - proliferate to replace fracture haematoma
  - produce ECM and collagen
    - Type 1 collagen
      - non cartilaginous ECM
    - Type 2 collagen
      - Cartilaginous ECM produced by chondroblasts
      - Stabilise the fracture through the formation of cartilaginous callus (soft callus).
- **Chondrocyte** proliferation, maturation and hypertrophy.
- **Hypertrophic cartilage** converts to bone
- **Osteoblasts** synthesize dense, crosslinked collagen and specialized proteins, including osteocalcin and osteopontin, which compose the organic matrix of bone.
- Newly formed woven bone then re-modelled (**osteoblast** and **osteoclast** activity).
- <u>Systemic</u> – Smoking, diabetes
- <u>Enhancement</u> – Systemic optimization, bone graft, bone graft substitutes

Vascular

- Local blood flow increases
  - peaks at 2 weeks
  - normalizes at 3-5 months
- Network of neo-vascularisation becomes more extensive during development of callus.
- Systemic – Smokers, diabetic
- Enhancement – Systemic optimization, Masquelet induced membrane, free muscle flap

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Additional biologic considerations in non-union

- Metabolic and hormonal
  - ACTH
  - Cortisol
  - DHEA-S (dehydroepiandrosterone)
  - Growth hormone
  - Parathyroid
  - Testosterone
  - Thyroid
  - Prolactin
  - Calcium
  - Magnesium
  - Phosphorous
  - Alkaline phosphatase
  - Vitamin D
  - Fasting testosterone + SHBG

Soft tissue healing

Skin healing

Mechanical

- Wolff's law (1892) – physiologic response of normal bone to its environment
- Perren's theory (1978) - physiologic response of a broken bone to its environment

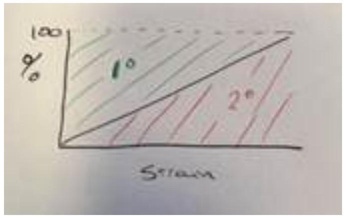
Direct

Indirect

Mechanical

- Wolff's law (1892) – physiologic response of normal bone to its environment
- Perren's theory (1978) - physiologic response of a broken bone to its environment

Direct ⟷ Indirect



How is this going to heal?

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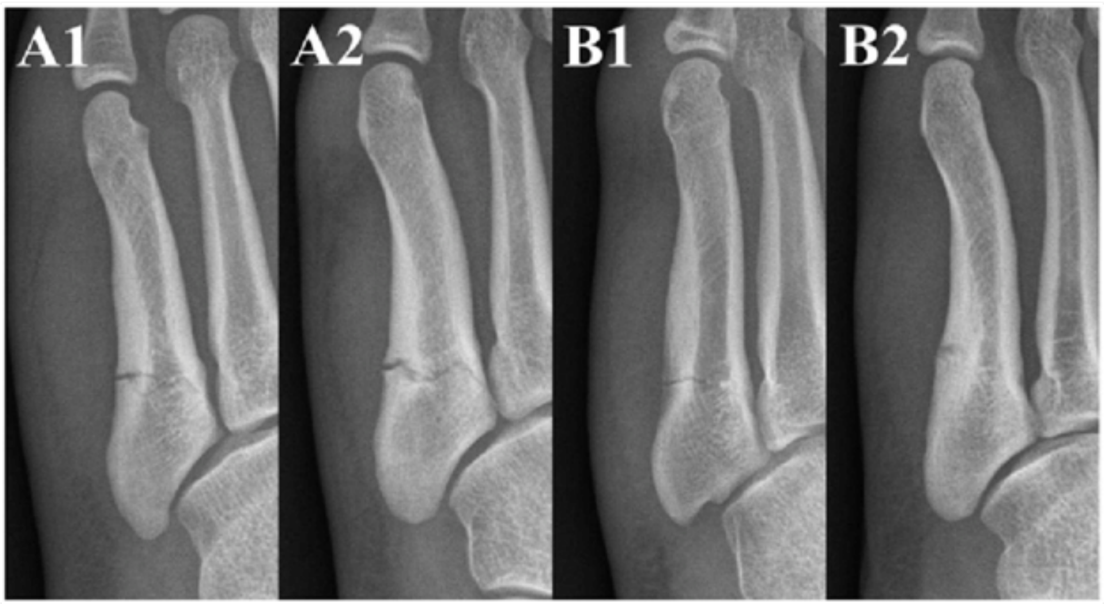
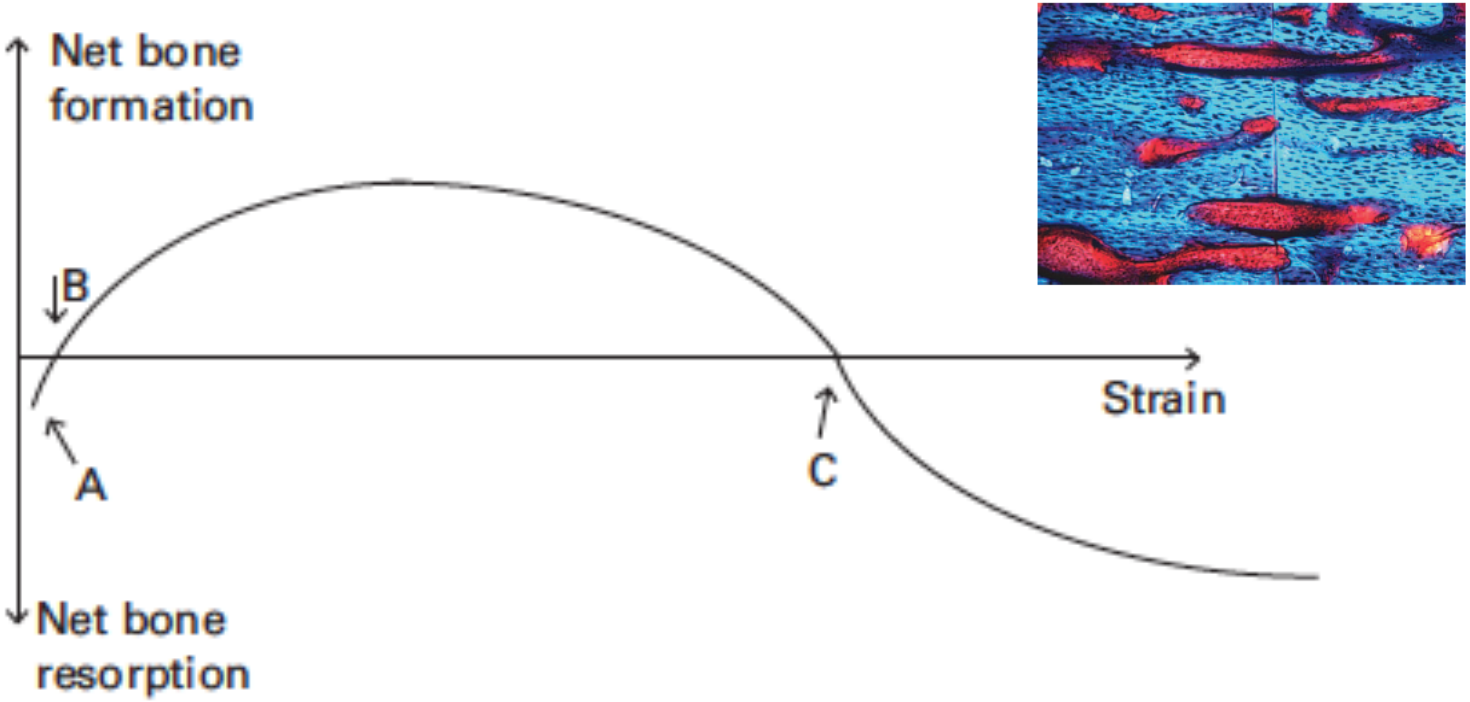
How did these fractures happen?

- Repetitive strain above the threshold for re-modelling, but below threshold for fracture
- Failure usually starts on tension side

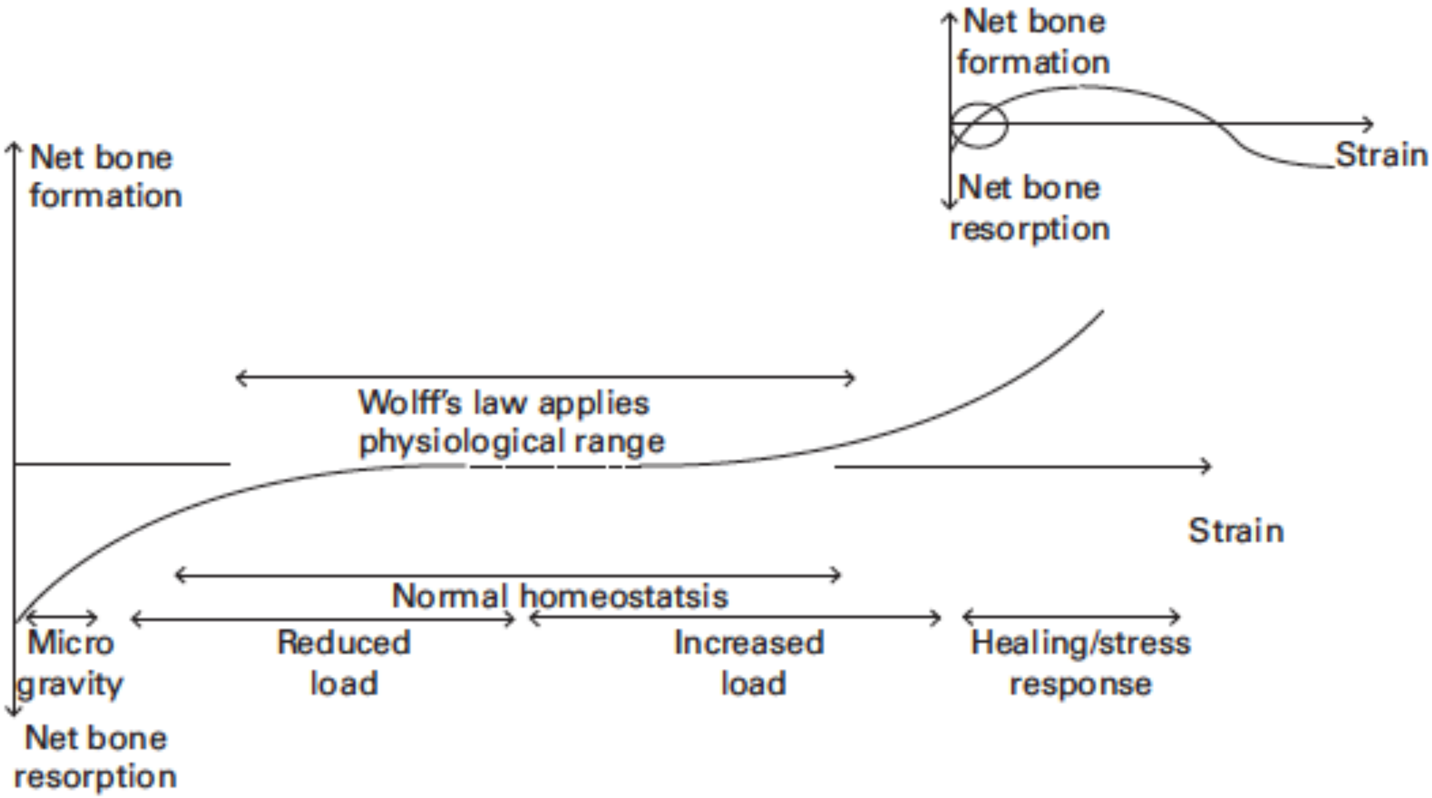


Wolff (Intact bone) - from Agincourt to Mars



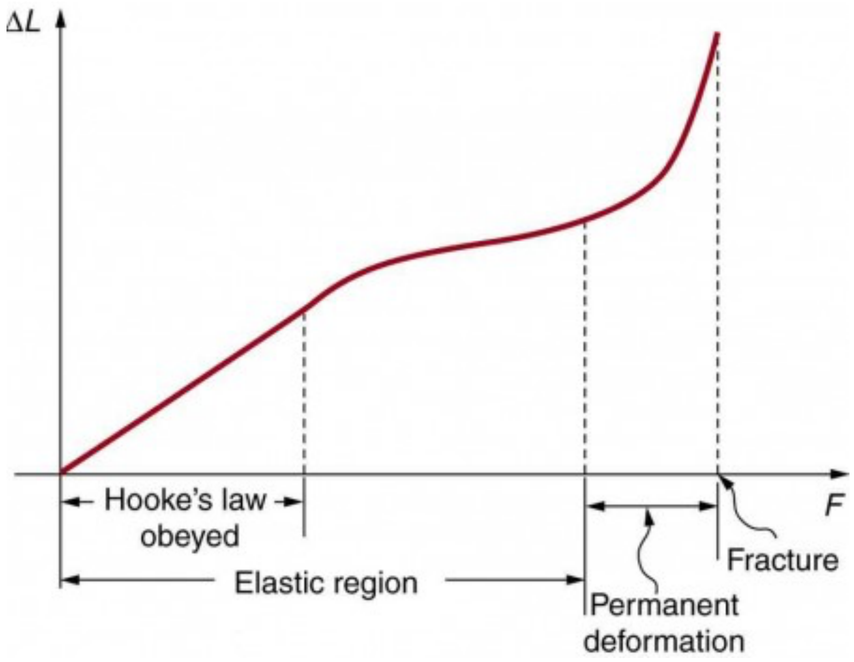
Point C - Failure of formed cortical bone at approximately 10% strain
Point B – homeostasis point

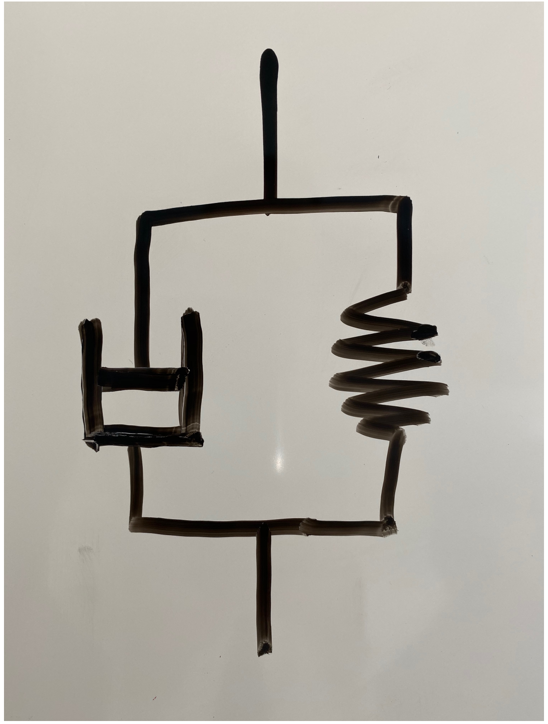
From Agincourt to Mars



Point C - Failure of formed cortical bone at approximately 10% strain
Point B – homeostasis point

Fracture


ΔL
F
Hooke's law obeyed
Elastic region
Permanent deformation
Fracture



How is this healing?

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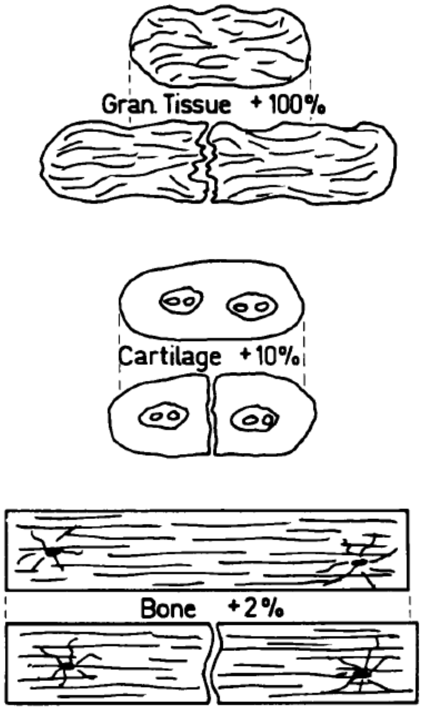
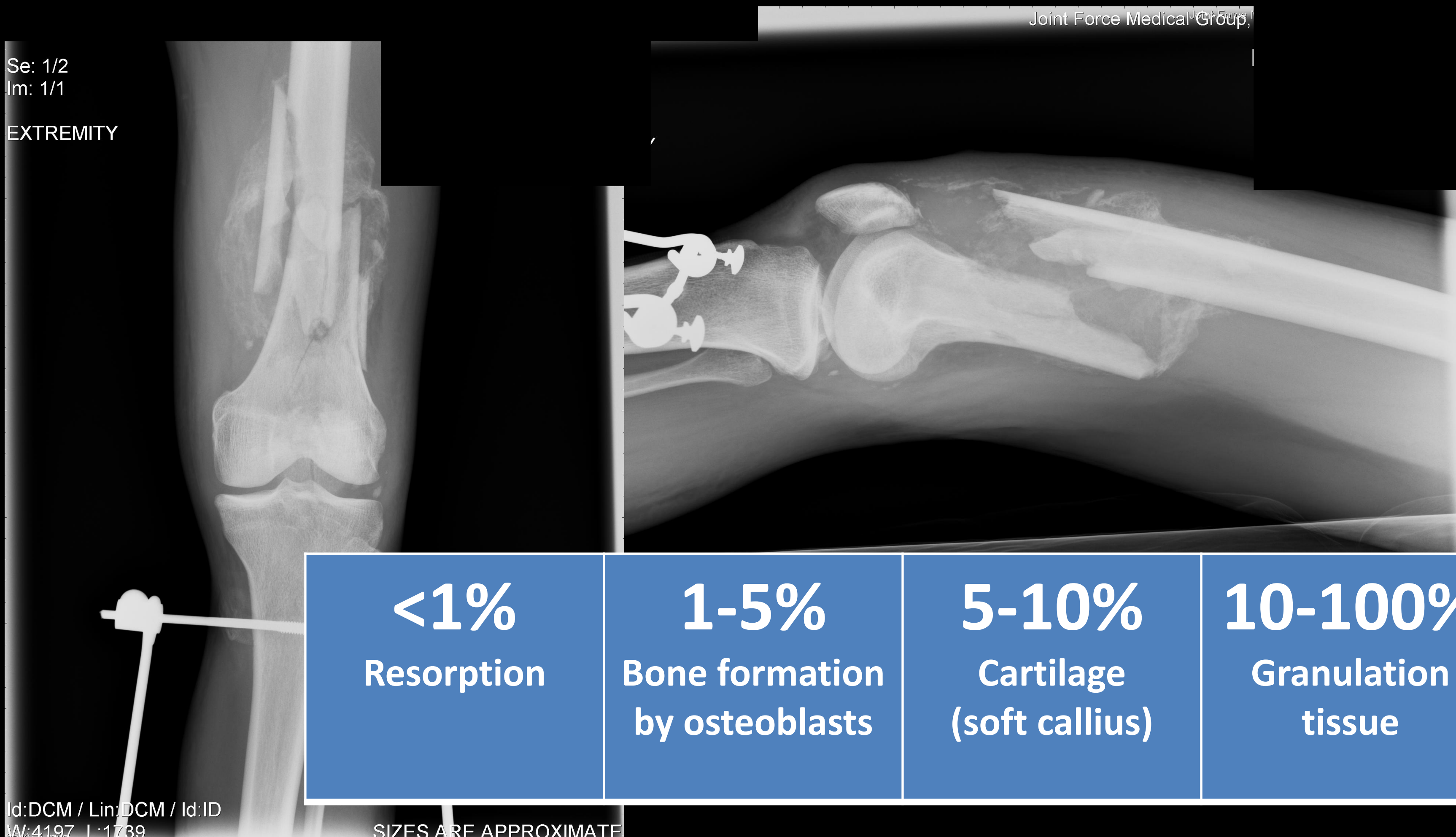
Strain Corridors

<1%	1-5%	5-10%	10-100%
Resorption	Bone formation by osteoblasts	Cartilage (soft callius)	Granulation tissue



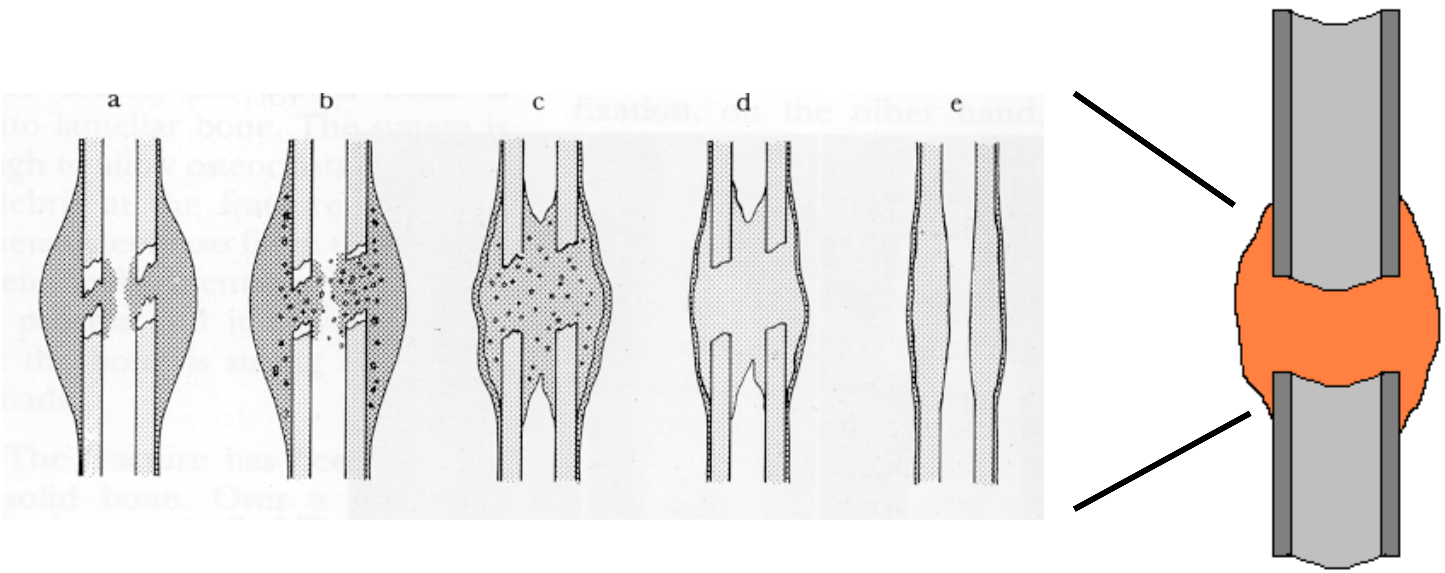
- NB Shear
  - Tibia vs femur non union

How is this healing?



<1%	1-5%	5-10%	10-100%
Resorption	Bone formation by osteoblasts	Cartilage (soft callius)	Granulation tissue

Indirect Bone Healing



Indirect bone healing

- Motion = indirect bone healing (callus)
- Soft callus (haematoma / granulation tissue / fibrocartilaginous tissue)
- Hard callus (endochondral ossification creates calcified tissue)
- Remodelling

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When can I start ROM?

How did the tibia heal?

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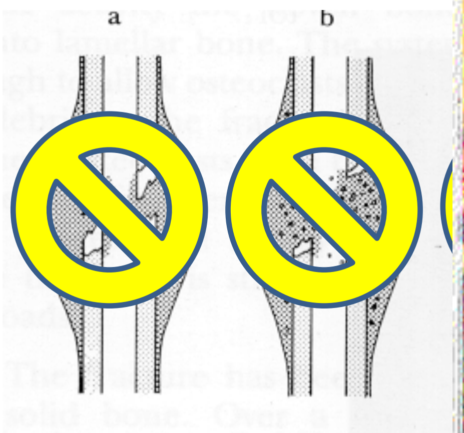
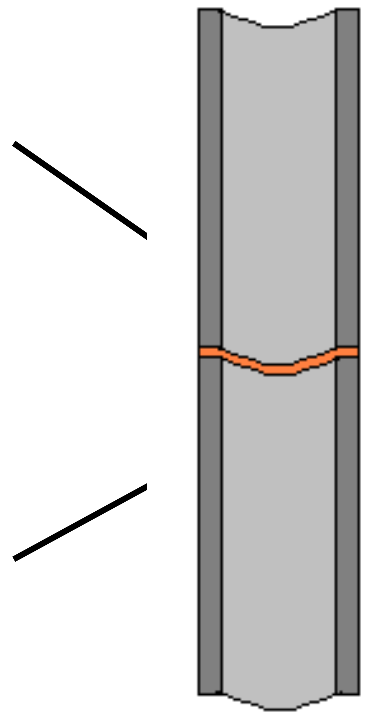
Direct Bone Healing





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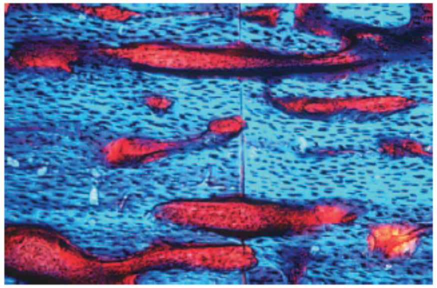
Direct Bone Healing

- Stability = direct formation of bone, intramembranous ossification
- Direct bone healing (osteonal remodelling)
- Absence of callus, resorption and intermediate repair tissue (Contact + Gap)
- Remodelling

Direct Bone Healing

- Contact healing (<0.01mm)
  - – Remodelling occurs between 2 fragments at circumscribed places that have motionless contact
- Gap healing (<0.8mm to 1 mm)
  - – Lamellar bone formation and subsequent remodelling of a small gap between 2 stable ends



Our contribution - Compression

- Reduces gap
- Increases stability
- Approximates strain to that of intact bone
- Load sharing
- Reduces torsion/shear through friction

Nature's contribution - Resorption

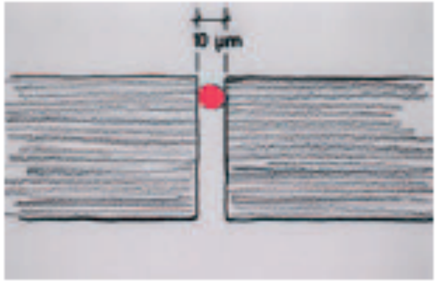


Fig. 5a

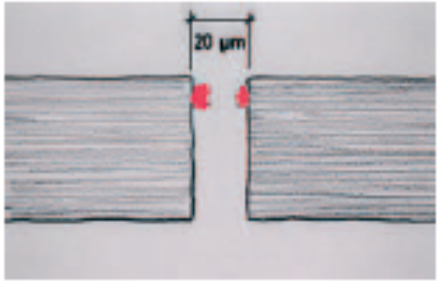


Fig. 5b

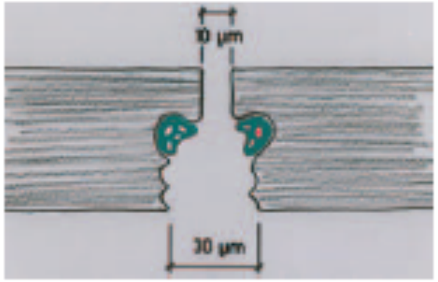


Fig. 5c

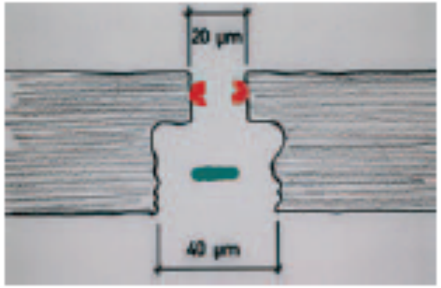
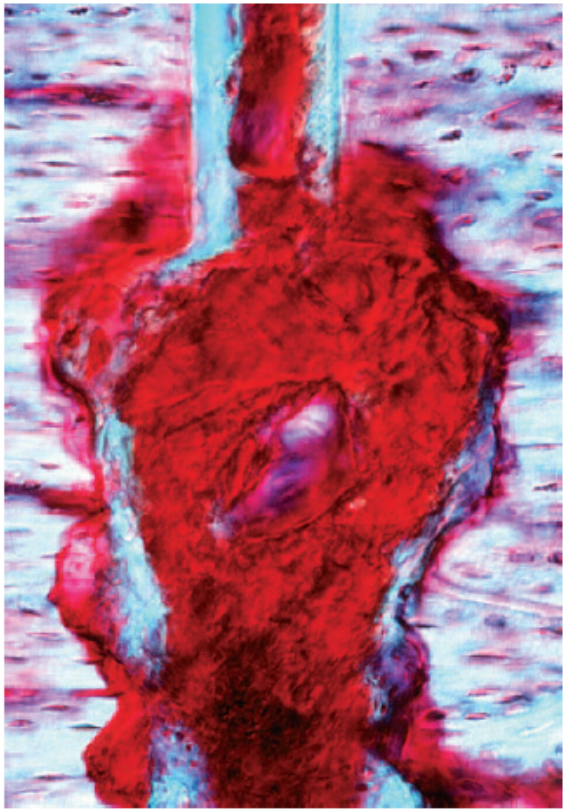


Fig. 5d



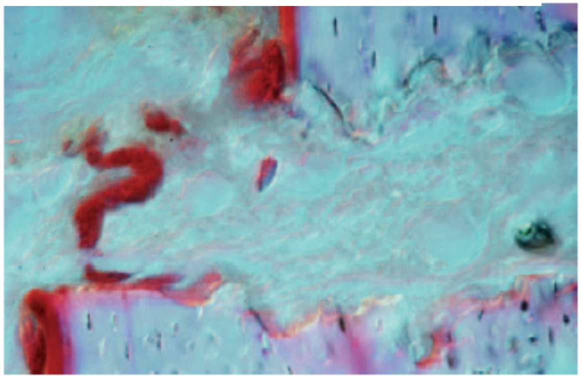


Figure 5. The relation of gap width and instability producing strain. Even very slight instability produces high strain conditions within a small fracture gap. Gap and displacement are not visible to the naked eye. Therefore this critical condition largely goes undetected. Figure 5a – A gap of 10 µm with a cell inside. Figure 5b – When the fracture fragments move 10 µm apart, the cell size is increased by 100%, which is the critical value for rupture of the cell.[50] Figure 5c – It is assumed that part of the fracture gap is widened through osteoclastic resorption to a gap size of 30 µm. Figure 5d – The same displacement as in Figure 5b (+10 µm) causes an increase of the widened part of the gap in Figure 5c, from 30 to 40 µm, that is an increase of only 30%. The cell in the widened gap undergoes tolerable deformation while that in the small gap ruptures or cannot be formed at a strain of 100%. Figure 5e – Photomicrograph of a small gap in the osteotomy of the tibia of a sheep fixed by a splinting plate. The small displacement but high strain prevents tissue differentiation within the small gap while repair tissue fills the widened gap ('teardrop').

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These are the extremes

- How do we explain?
  - How we match implant to fracture pattern
  - Implant loosening
  - Non union

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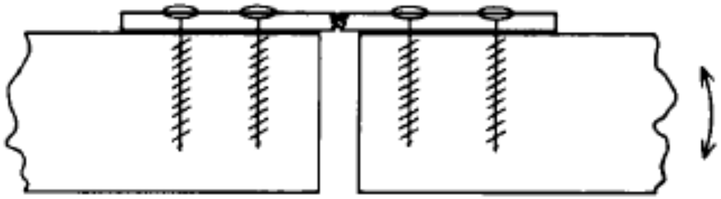
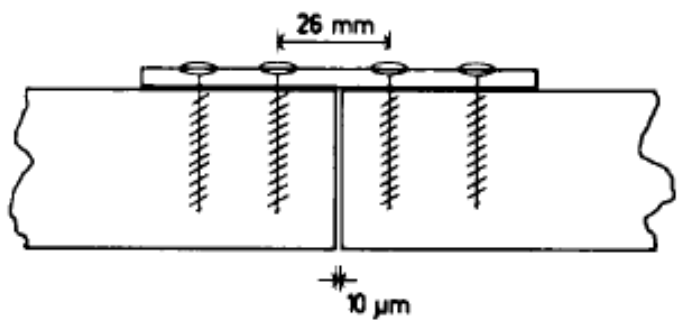


PLATE $\sigma_{dyn.} > 40$ Kp/mm^2 $I = 30-64$ mm^4

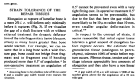
⟶ DYNAMIC MOMENT > 500 Kp·mm



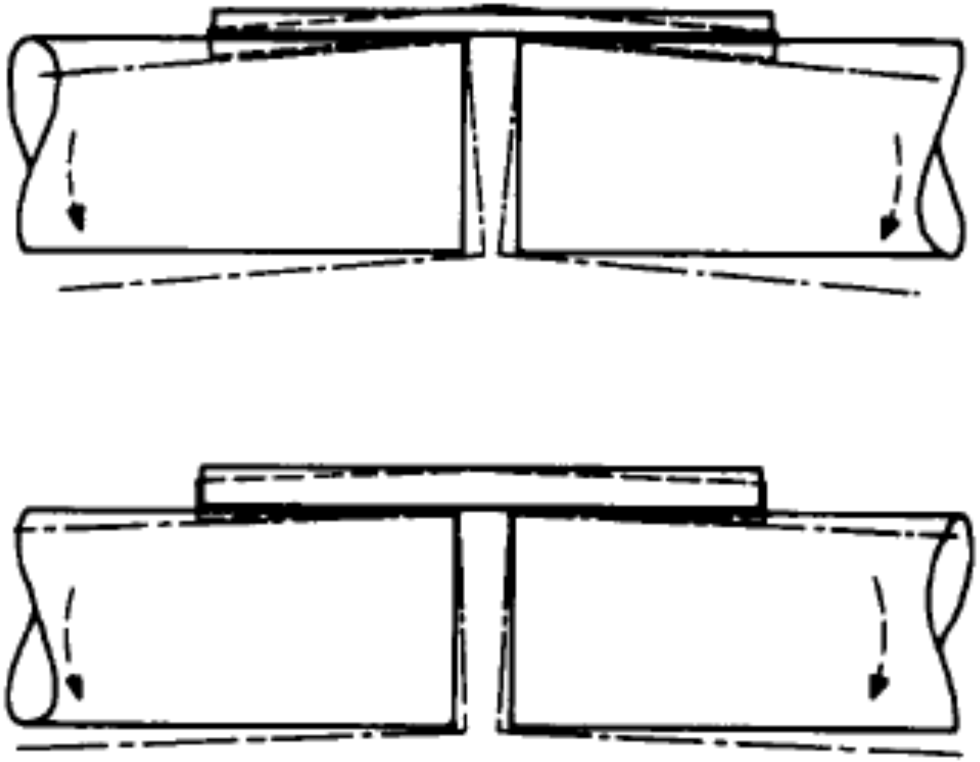
GRAN. TISSUE $\Delta L_{max} = 100\%$

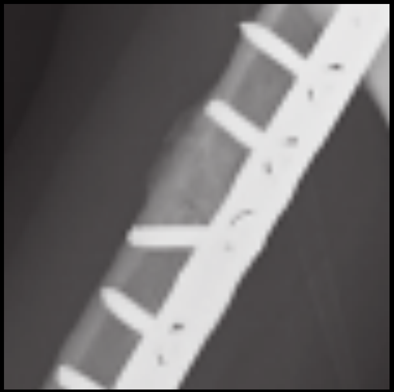
MAX. MOMENT 32 Kp·mm

<u>A VERY SMALL GAP IS DANGEROUS !</u>



- Maintaining gap in simple fracture and increasing thickness of plate is a risky strategy!





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Biologic Fixation

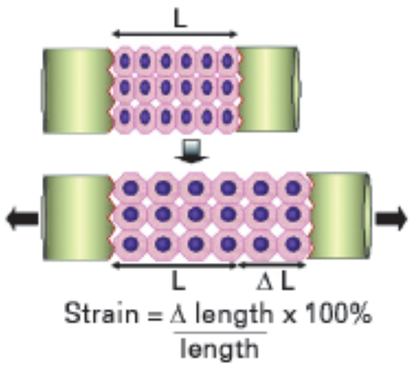


Fig. 1a

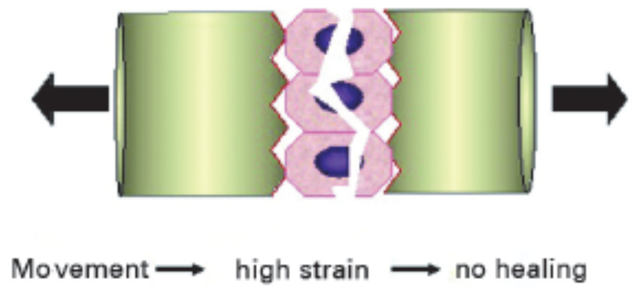
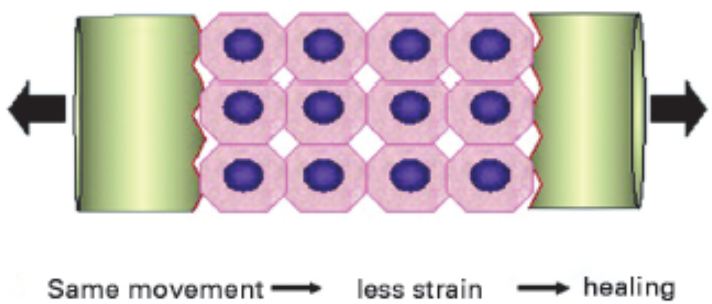


Fig. 1b



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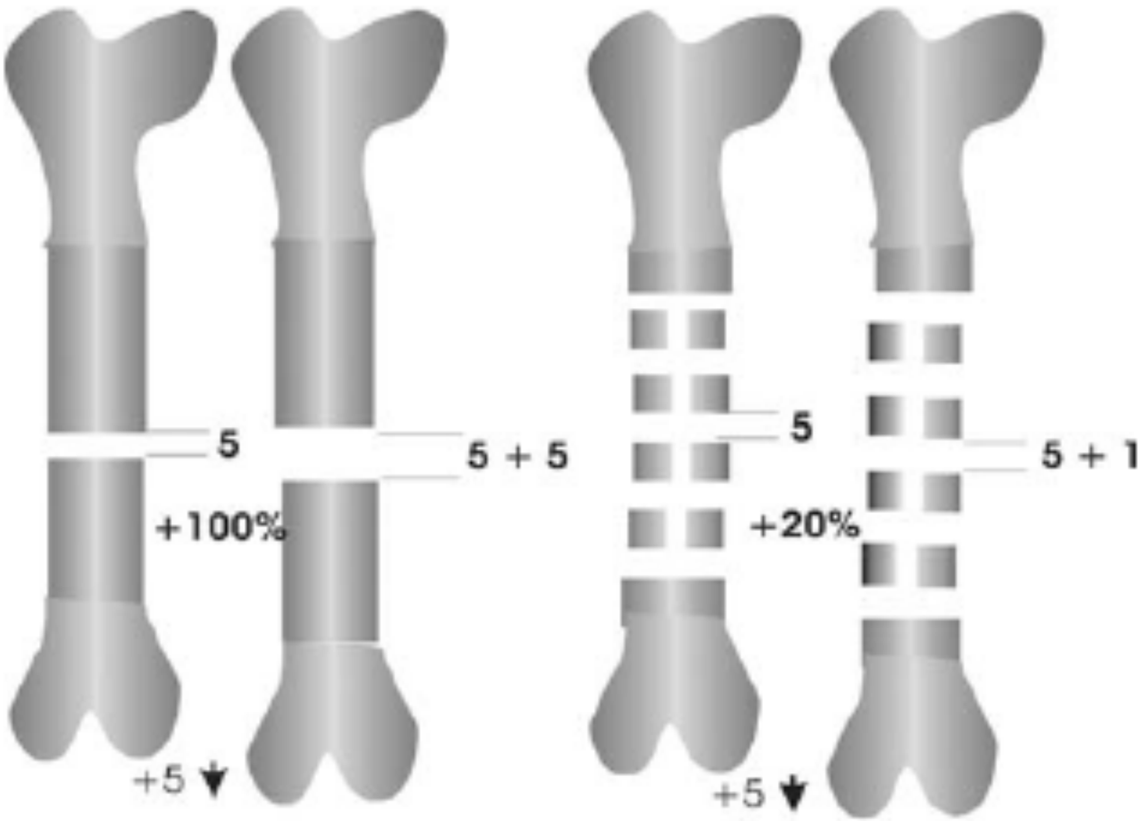
Biologic fixation



The tolerance of instability of simple *versus* multiple fracture lines. The overall displacement is the same (5 mm) between Figures 8a and 8b and Figures 8c and 8d. Figure 8a – Model of a simple transverse fracture with an initial gap width of 5 mm. Figure 8b – The full amount of the displacement of the end fragments (5 mm) is active within the fracture gap, the width of which increases from 5 to 10 mm. This is equal to 100% strain which is the limit of tolerated strain for granulation tissue. Figure 8c – Instead of a simple fracture a multifragmentary fracture with five fracture gaps is seen. Figure 8d – Five gaps share the overall displacement. Thus, each displaces from 5 to 6 mm. The resulting strain is only 20%.
This explains why multifragmentary fractures are more tolerant to instability than simple fracture lines. However, when choosing an initial somewhat larger gap width the strain can be kept low in simple gaps as well. Figure 7 demonstrates that simple fractures can be efficiently treated with elastic flexible fixation.

Biological plate fixation

- Remember it has been been tried and failed before
- Can you be too stiff?

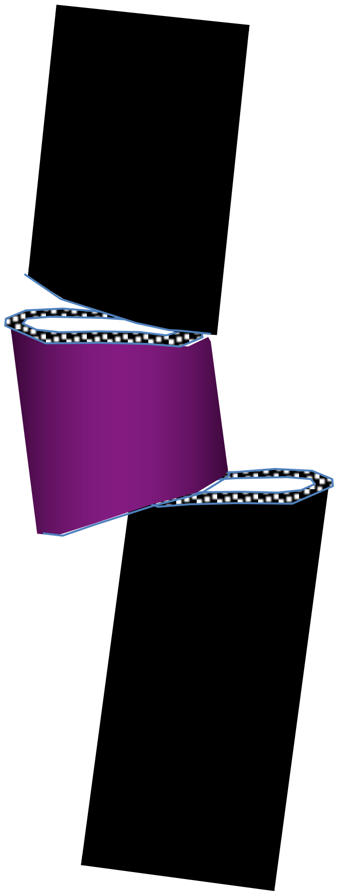
Bone Forming Organ

- What happens if the optimum strain corridor falls outside the bone

Highest incidence of non-union

= clavicle and tibia

Segmental fracture



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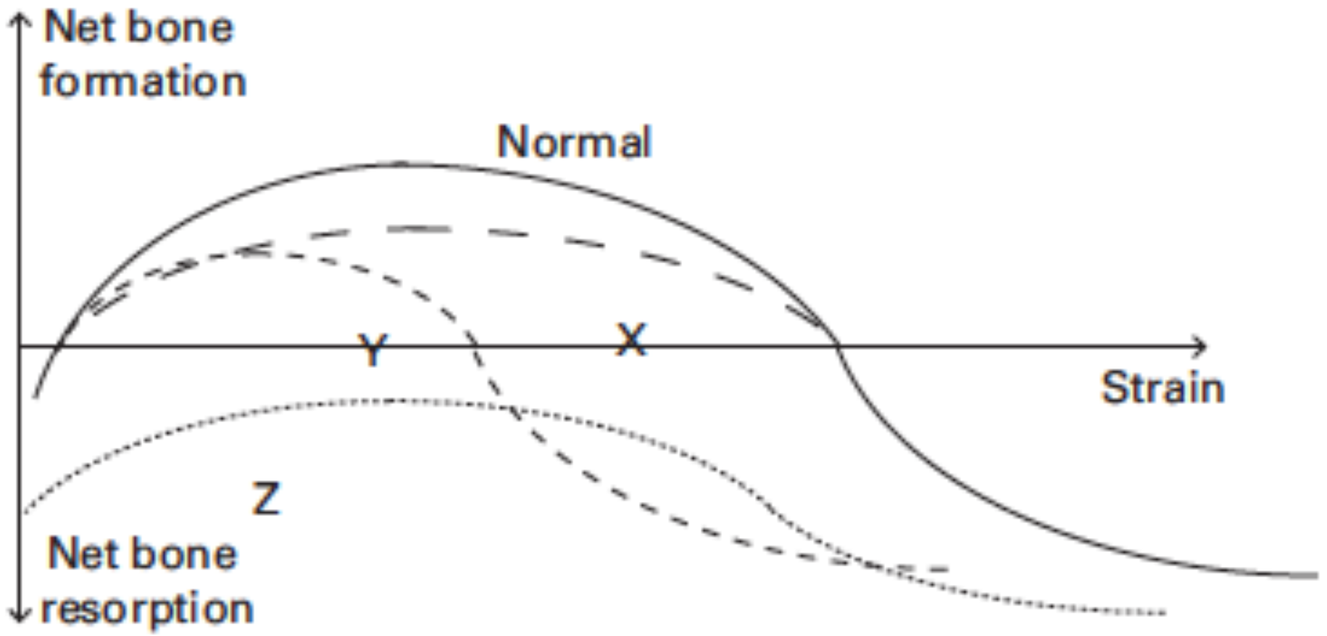
What is happening here?

Ilizarov

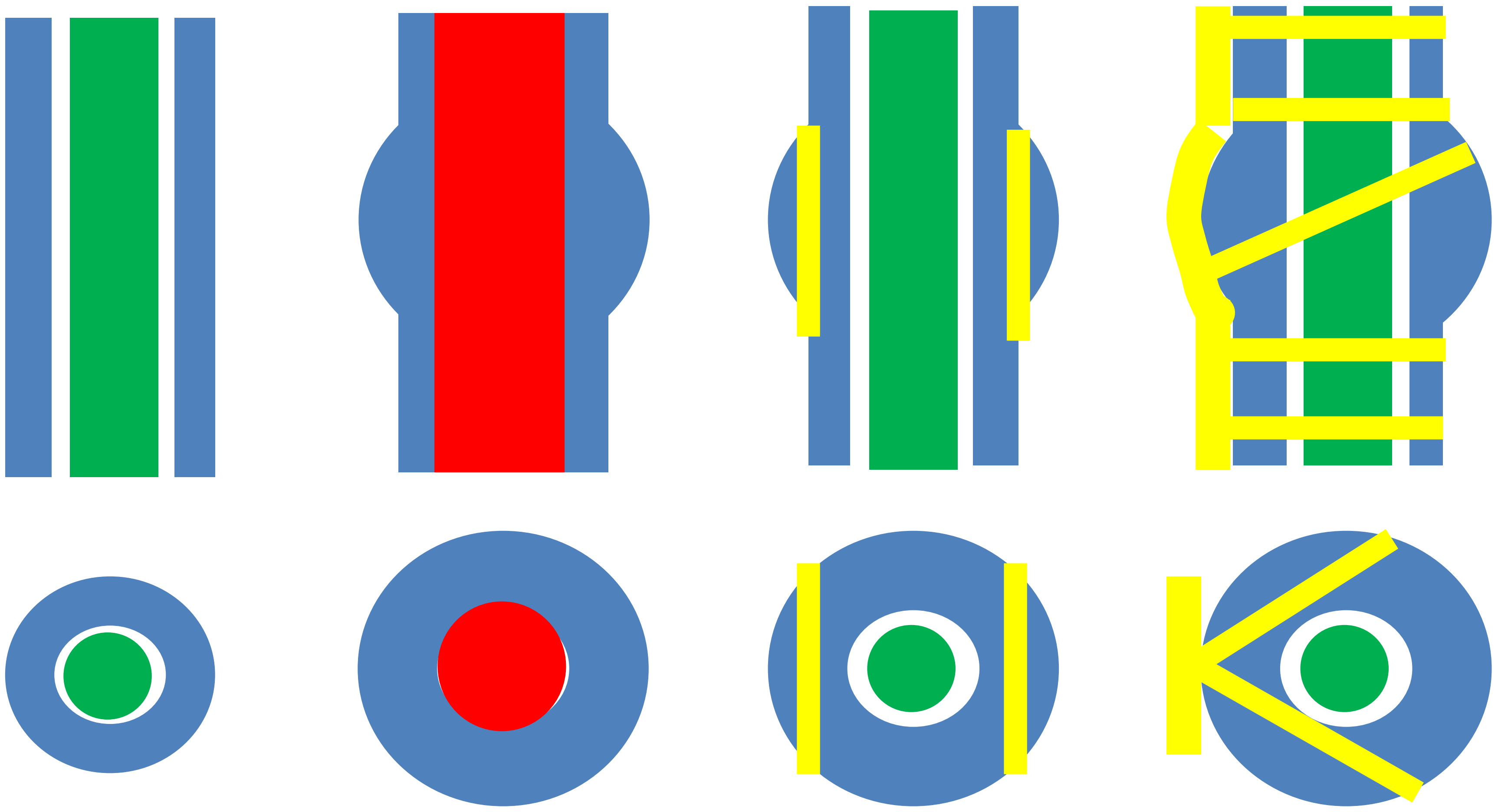
- Type of stability?
- What happens in distraction
  - 1mm/day
  - >1mm/day
- What happens in compression?
- Ability to restore alignment
  - Optional extra vs Essential

Can we influence reaction to strain?



mechanism is unknown. Do they simply delay the formation of bone (Fig. 7, line X), narrow the range of strain at which bone can form (Fig. 7, line Y), or completely prevent bone formation (Fig. 7, line Z)? The biological response may be increased as well as reduced. Enhanced bone formation is seen in some patients with a severe head injury or spinal cord injury and in some cases where bone morphogenetic proteins (BMPs) have been administered.[32] Again, it is not known if this enhanced bone formation takes place over the normal range of strain where bone formation is seen, or is the range of strain, at which bone formation takes place, increased. If the latter, BMPs applied locally to high strain nonunion sites without mechanical stability would be an effective form of treatment. There is, however, no clinical evidence for this at present.

Increasing Stiffness



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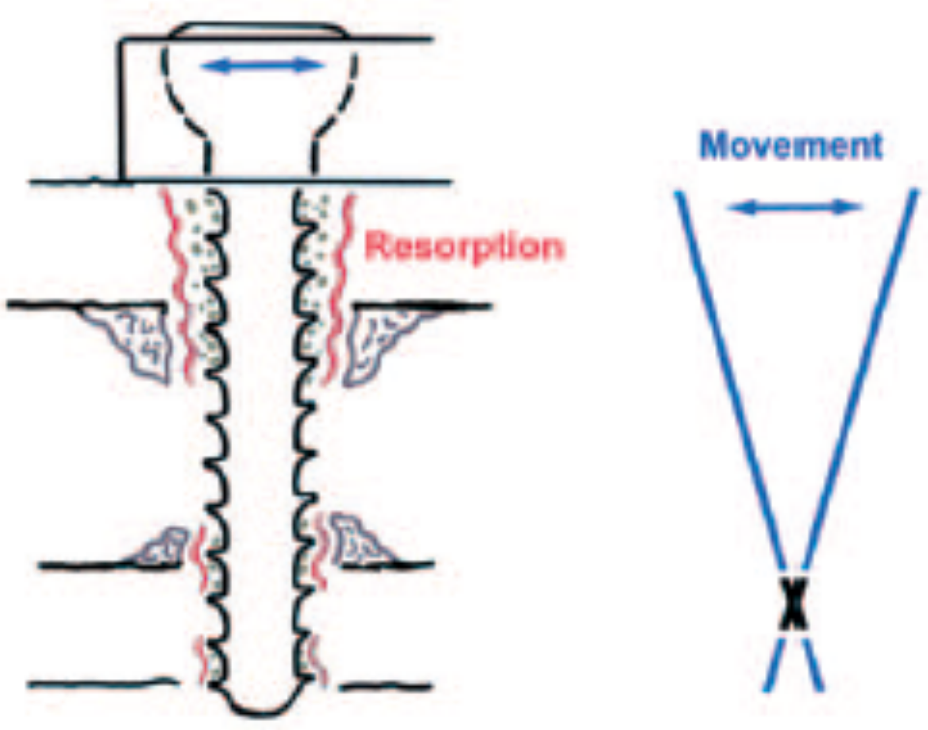
Implant Loosening



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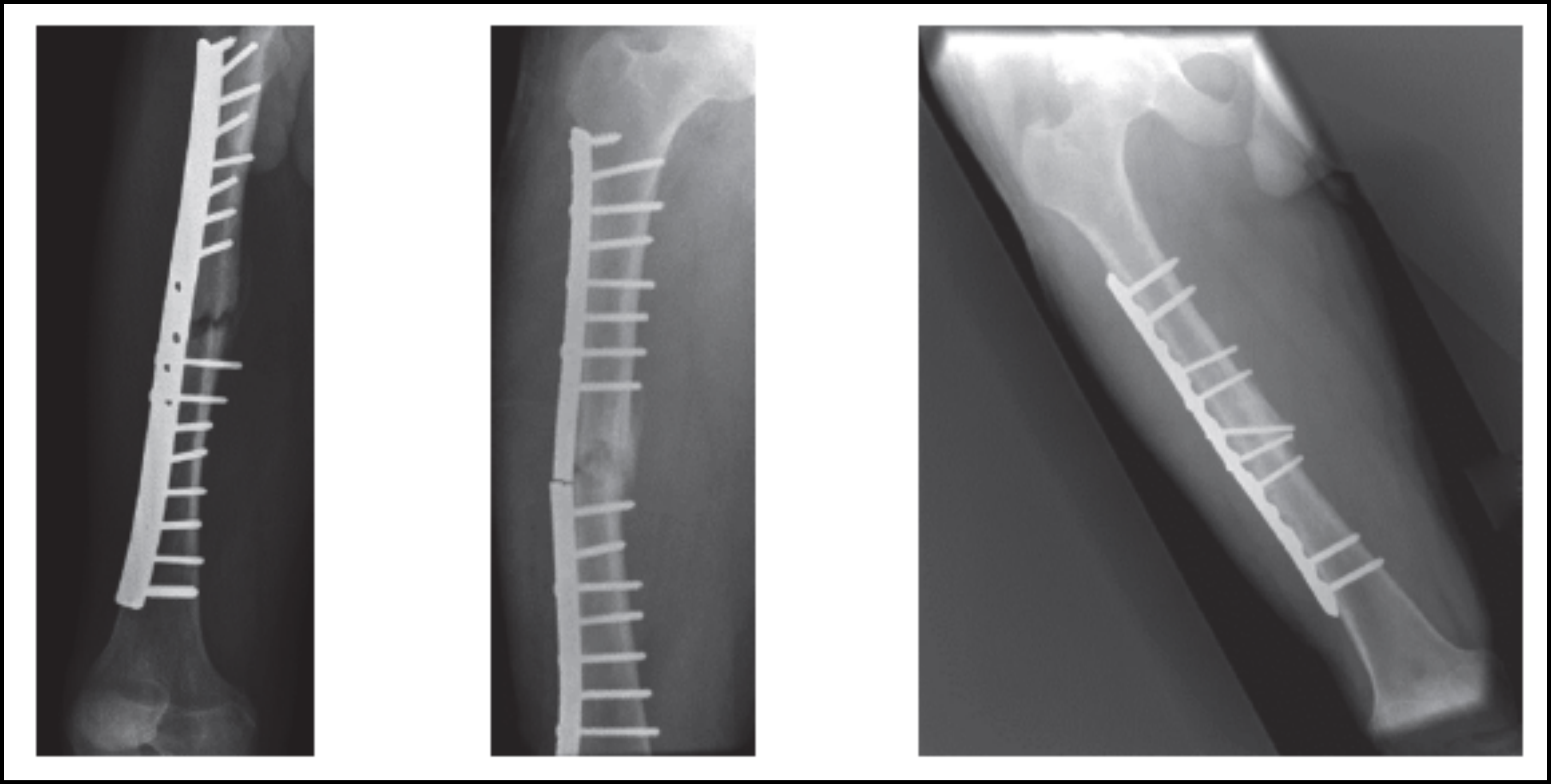
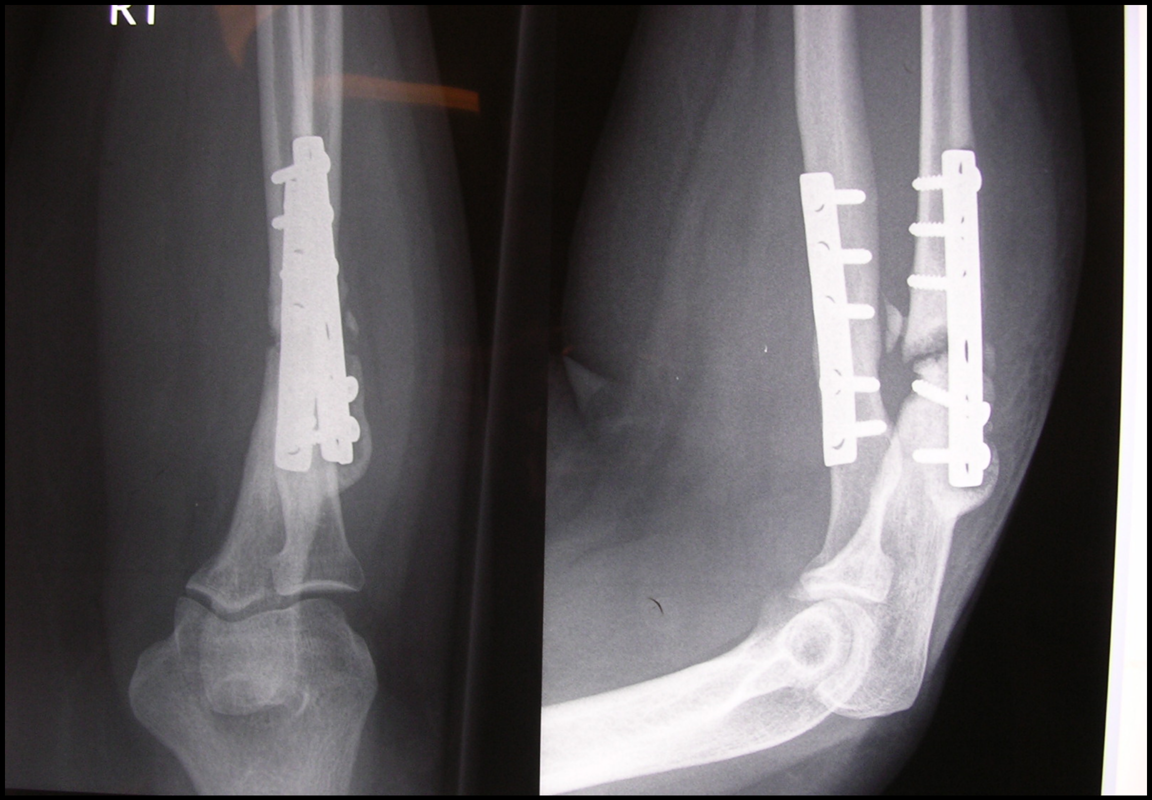
Surgeon Failure



#2 – Non union

- Where do you start?
- (How did we get here?!)



Bone defect

- Bone graft
  - Autograft
  - Allogenic

Bone graft substitutes

- Calcium sulphate
- Calcium trisulphate

BREAK

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AO Glossary

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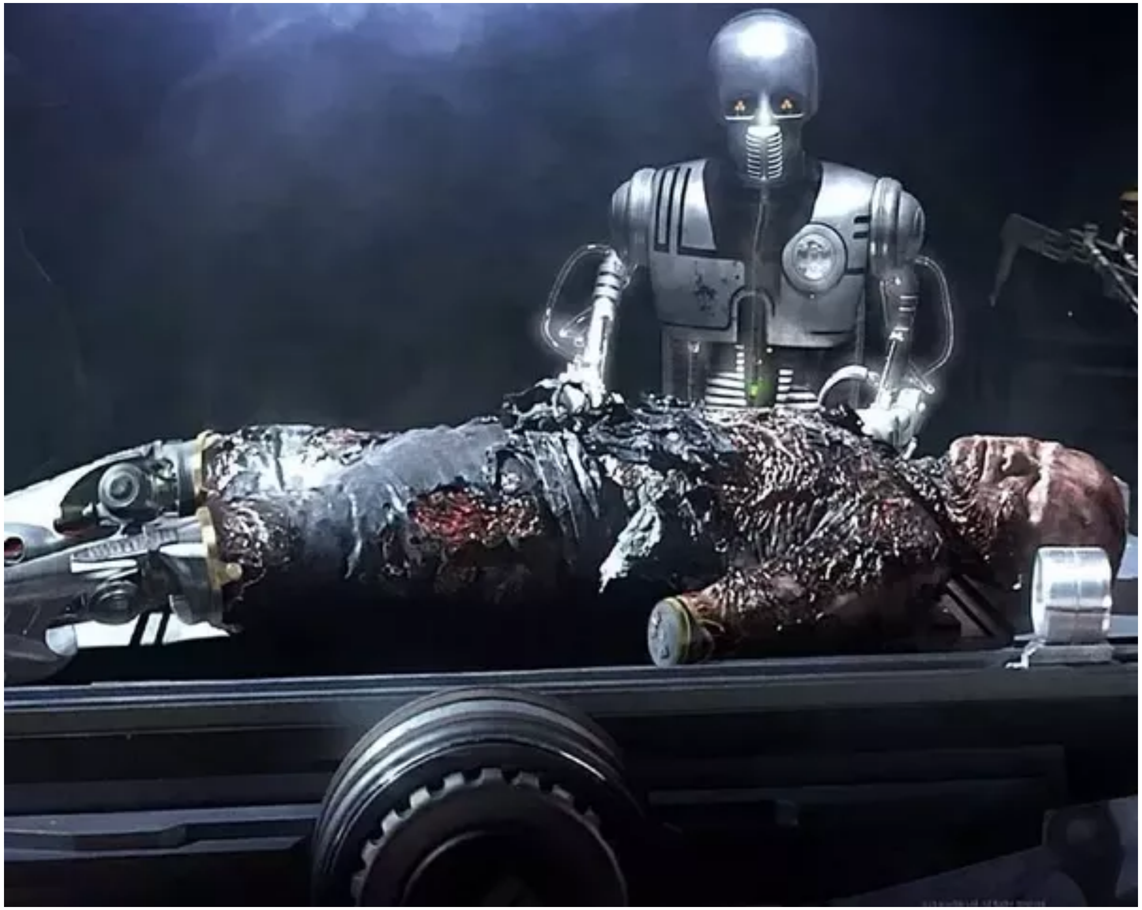
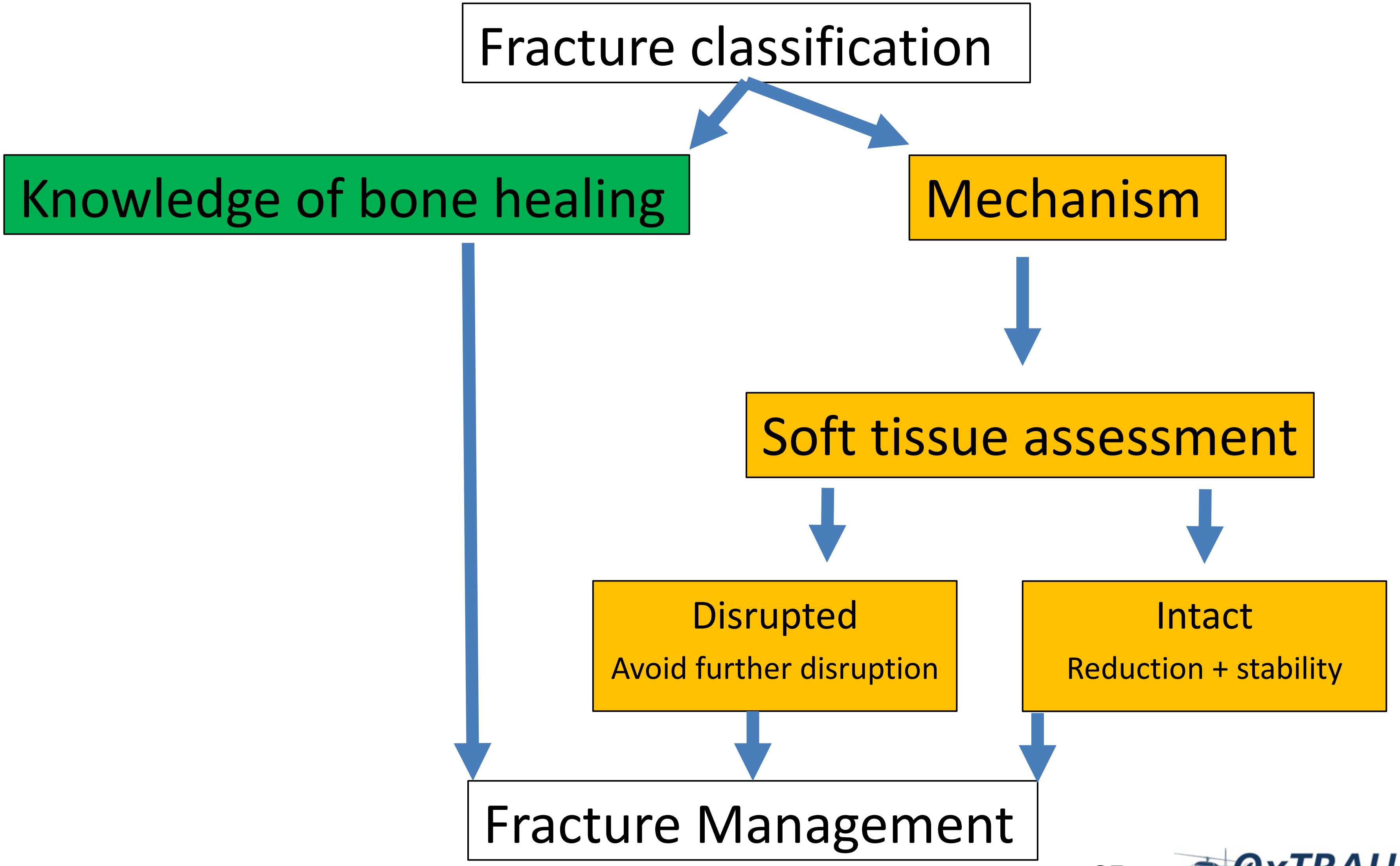
Part 2 - Objectives

- Revise relevant classification systems
- Use AO classification system to interpret
  - Optimum bone healing strategy
  - Soft tissue disruption
- Develop strategy
- Demonstrate a system of planning and ‘pre mortem’ interrogation

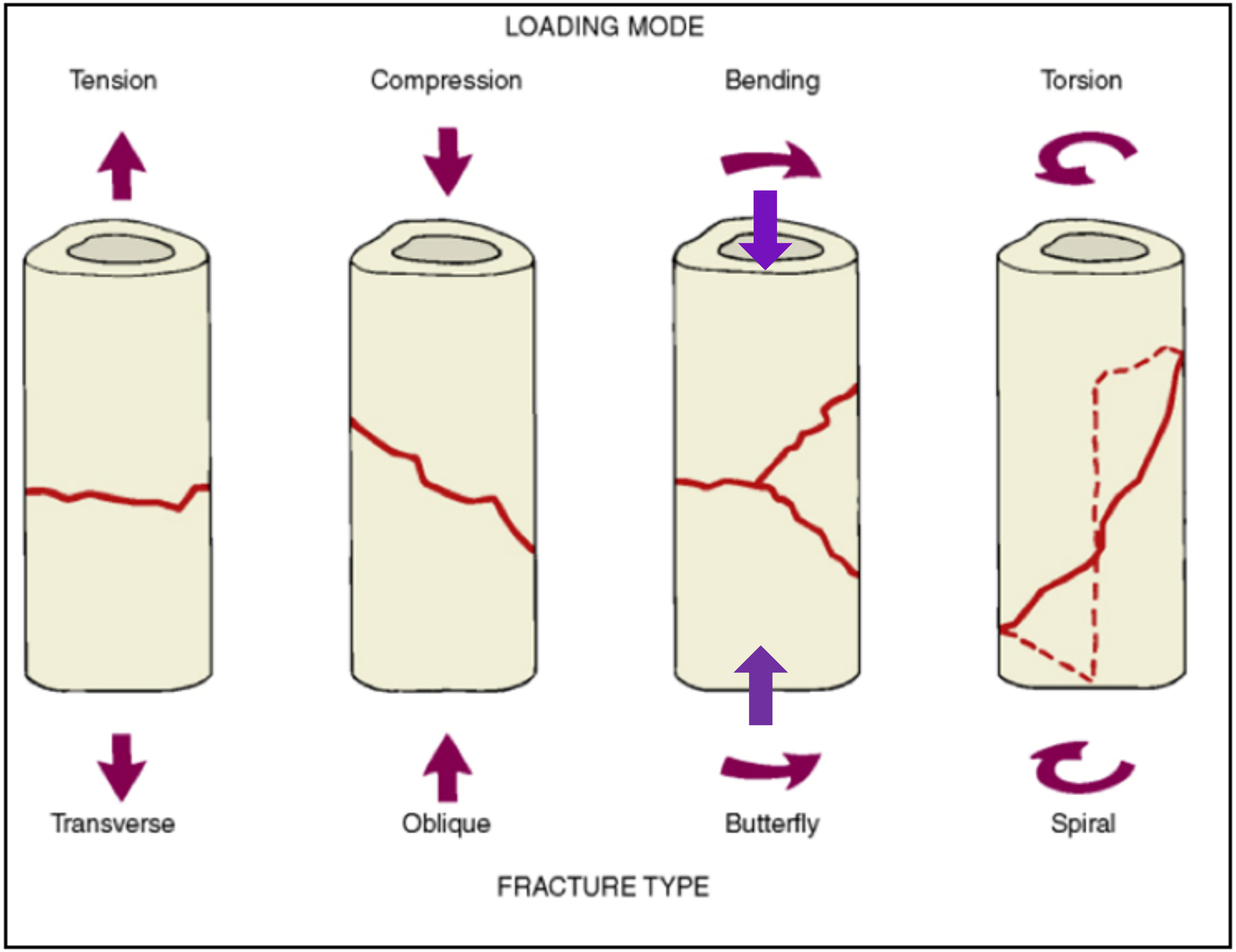
Fracture Management Algorithm



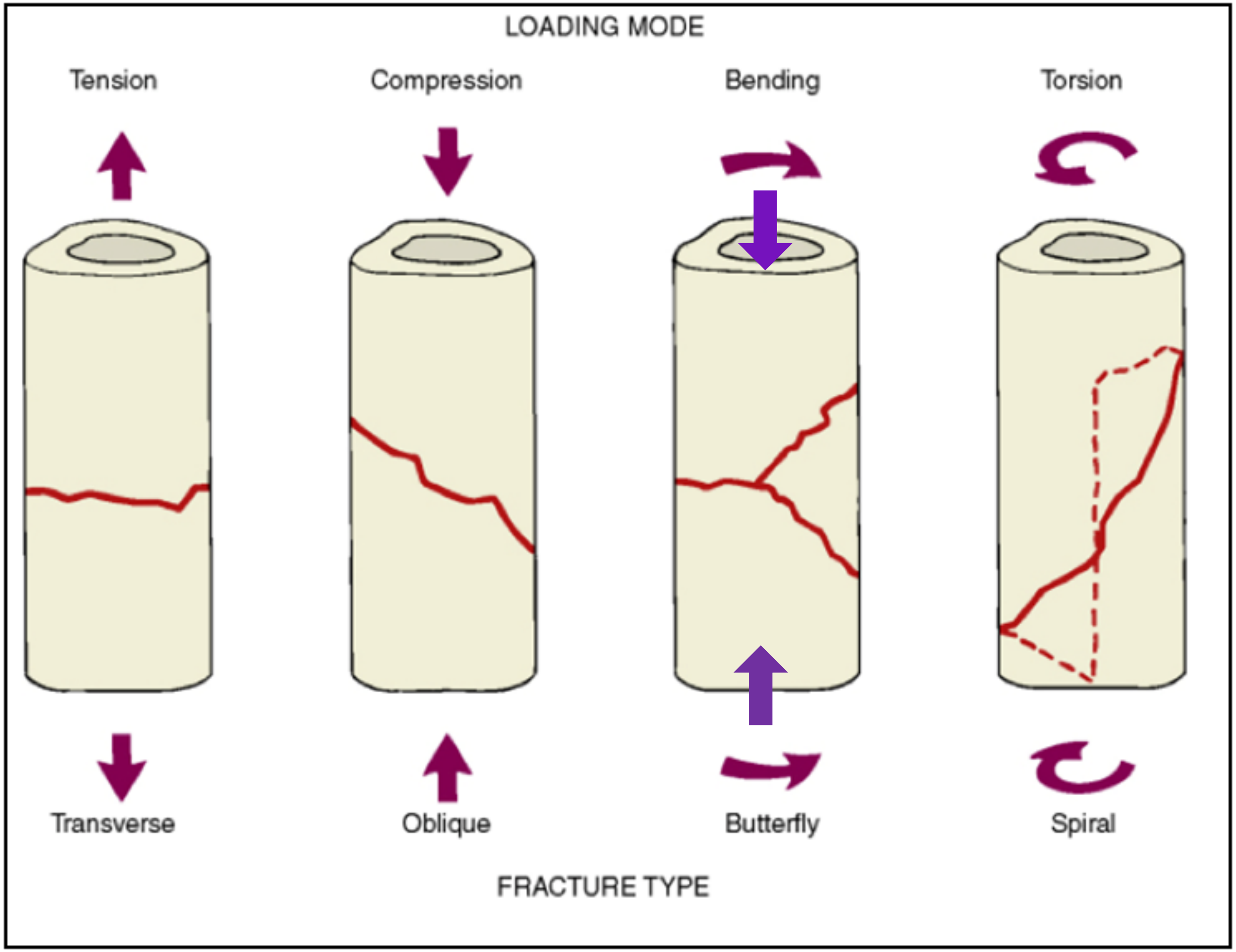
Fracture Management Algorithm



Mechanism


LOADING MODE
Tension
Compression
Bending
Torsion
Transverse
Oblique
Butterfly
Spiral
FRACTURE TYPE

Where is the soft tissue disruption?



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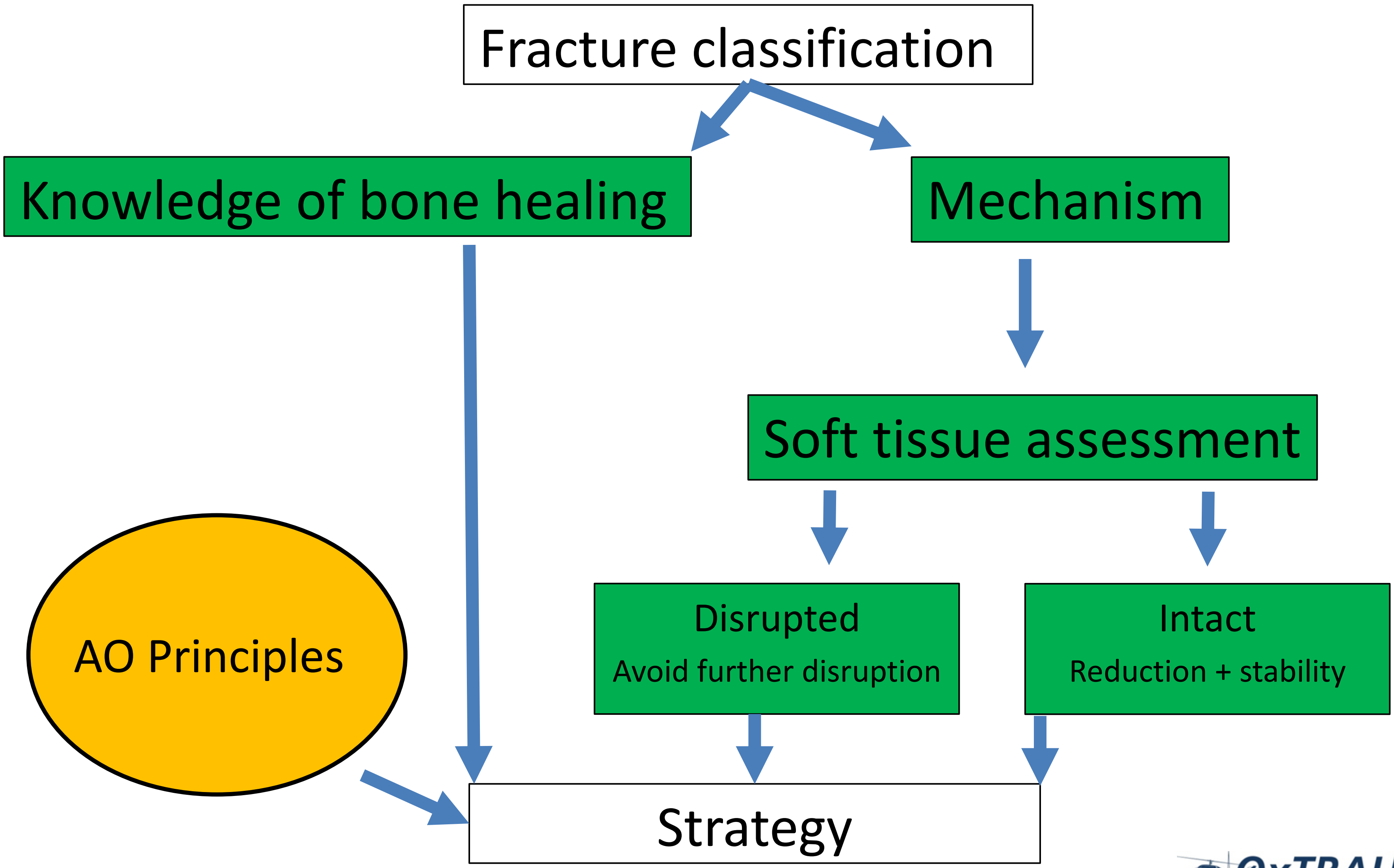


AO Principles

- Restore anatomical relationships
- Preservation of blood supply
- Fixation takes into account personality of the fracture, patient and injury.
- Early mobilisation

AO glossary

Classification

1	2	3	4	5	6	7	8

1	2	3	4

A	B	C

1	2	3	4

Gustillo I	Gustillo II	Gustillo IIIa	Gustillo IIIb	Gustillo IIIc

High Energy Injury (Bone / Soft tissues / Physiology)	Low Energy Injury (Bone / Soft tissues / Physiology)

Mechanism

Failure - Flexion	Failure - Extension
Failure – Abduction / Valgus	Failure – Adduction / Varus
Failure – Rotation	Failure - Compression

Soft tissues

Soft Tissues
- The fracture pattern is suggestive of an injury mechanism associated with disruption of …. (periosteum, ligaments and capsules)

Principles

AO Principles
- Restore anatomical relationships
- Preservation of blood supply
- Fixation takes into account personality of the fracture, patient and injury.
- Early mobilisation

Address each component of fracture

Medial	Lateral
Anterior	Posterior
Extra-articular component	Intra-articular component

Reduction + Fixation

Biological Fixation - Preserve blood supply - Restores length and alignment	Rigid Fixation - Anatomical reduction - Interfragmentary compression
Relative Stability	Absolute Stability
Indirect reduction	Direct reduction
Direct bone healing (osteonal remodelling) - Absence of callus, resorption and intermediate repair tissue (Contact + Gap) - Remodelling	Indirect bone healing (callus) - Soft callus (haematoma / granulation tissue / fibrocartilaginous tissue) - Hard callus (calcified tissue) - Remodelling

Plate – compression	Plate – neutralisation
Plate – buttress - Resisting force away from the mechanical axis	Plate – antiglide - Resisting force in line with the mechanical axis
Plate – bridge	Plate - tension band
Internal fixation – intramedullary - Static vs Dynamic	External fixation - Monoaxial / Polyaxial / Dynamic
Internal fixation – extramedullary - Load sharing vs Load bearing	Arthroplasty - Hemi / Total / Excision

Load sharing	Load bearing

Classification

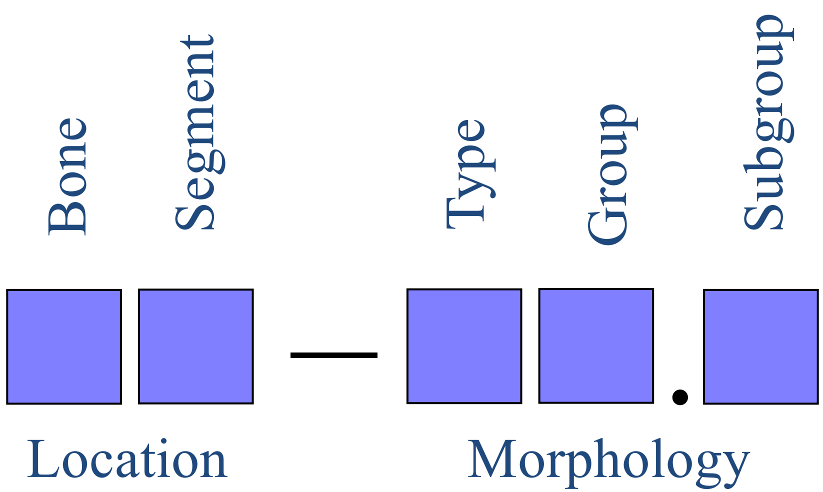
- Why classify?
- What makes for a good classification?

Commonly Used

- AO OTA
- Salter Harris
- Gustillo + Anderson/Mendoza
- Lauge-Hansen

AO OTA Classification

Types	A, B, C		
Groups	A1, A2, A3	B1, B2, B3	C1, C2, C3
Subgroups	.1, .2, .3		



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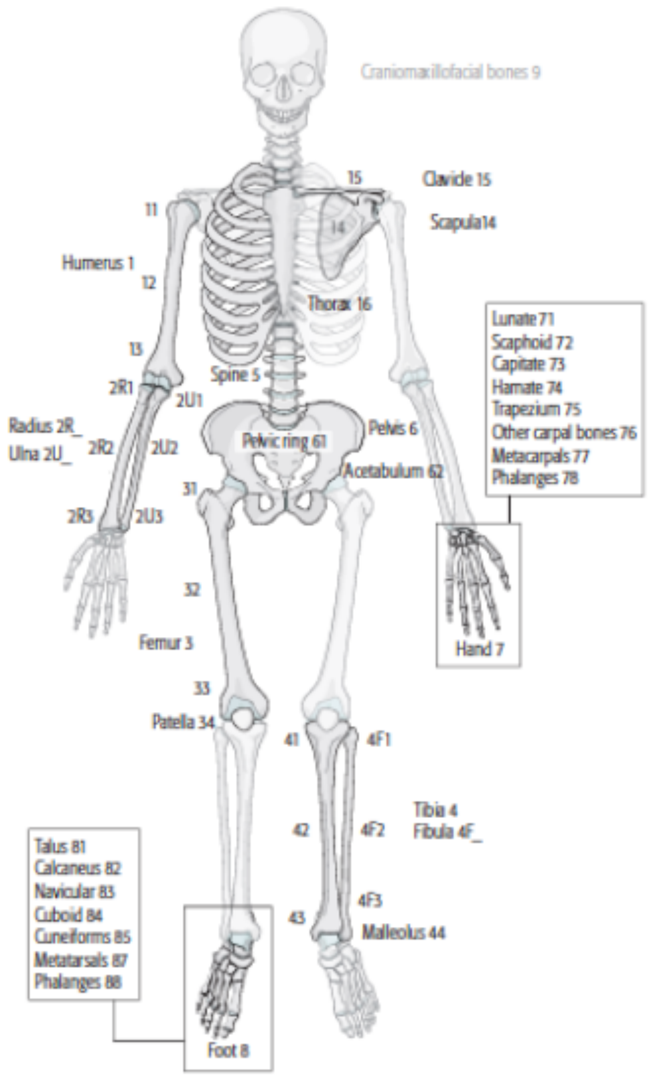
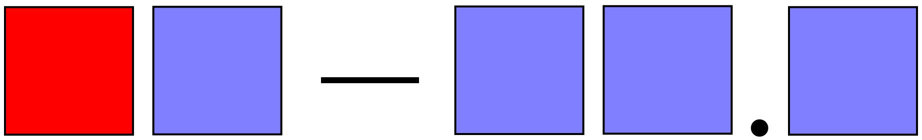
AO OTA Classification





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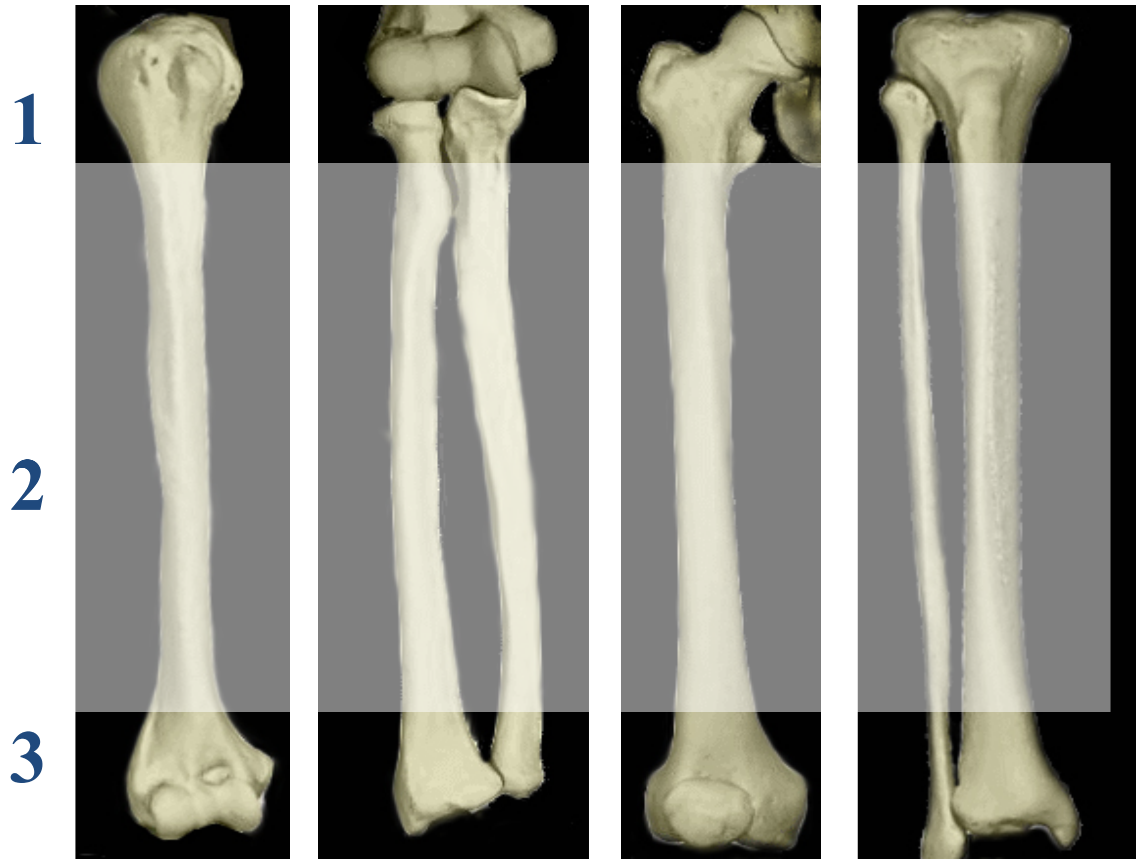
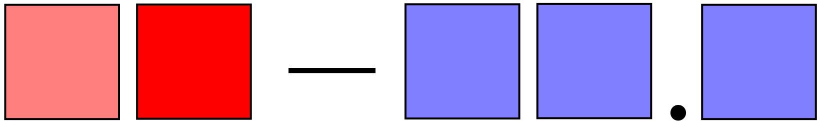
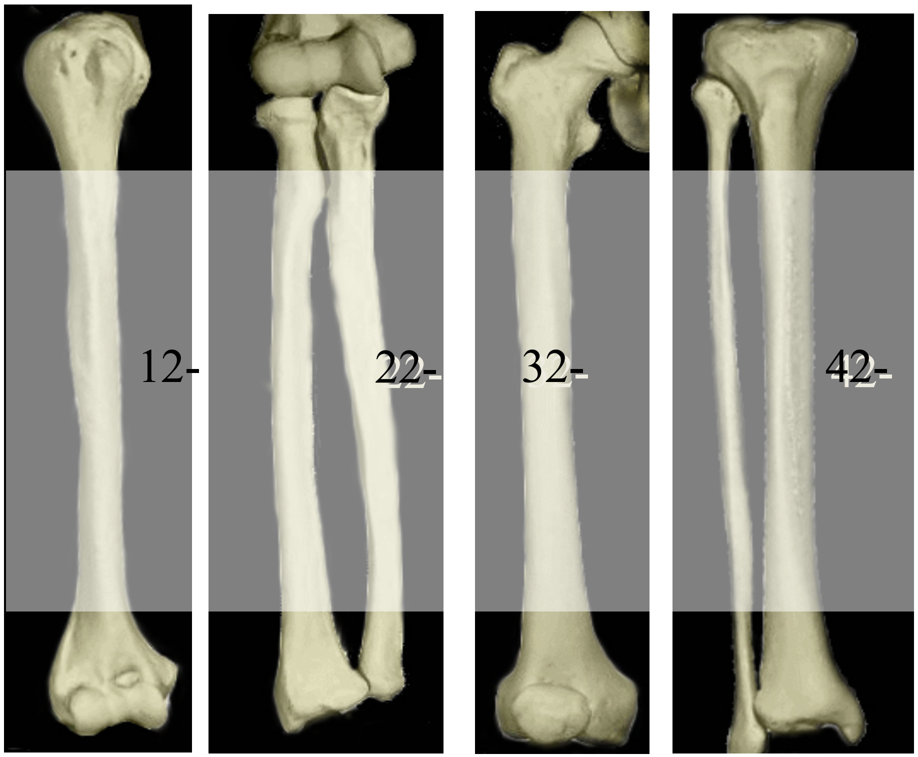
Segments

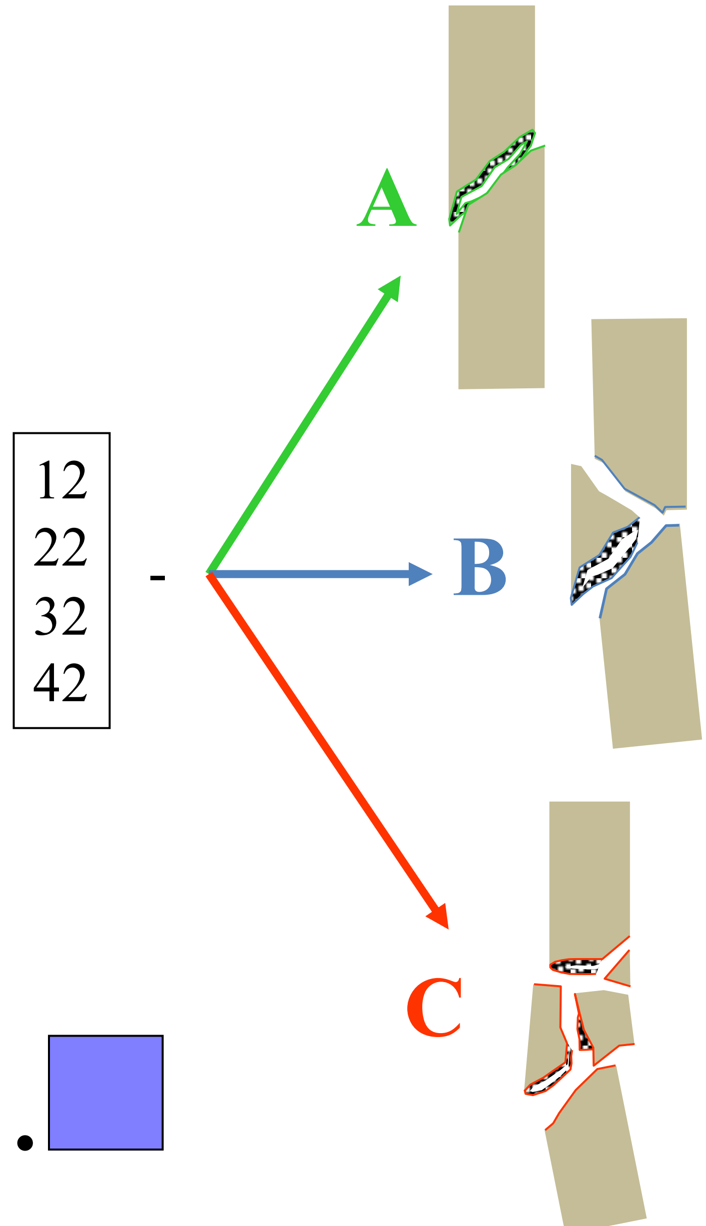


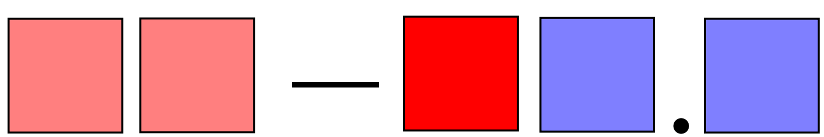


What are the surgical implications?

Diaphyseal types







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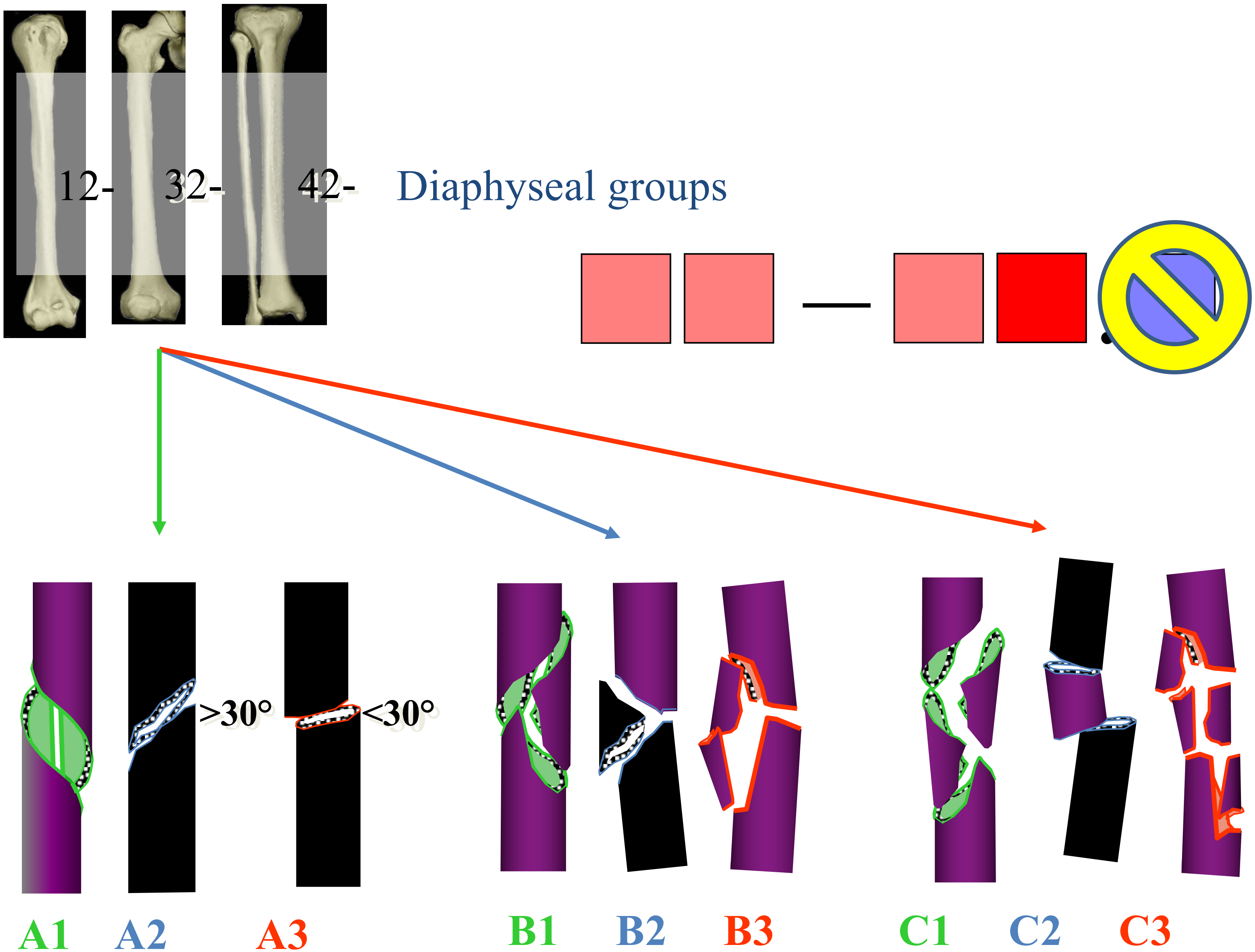

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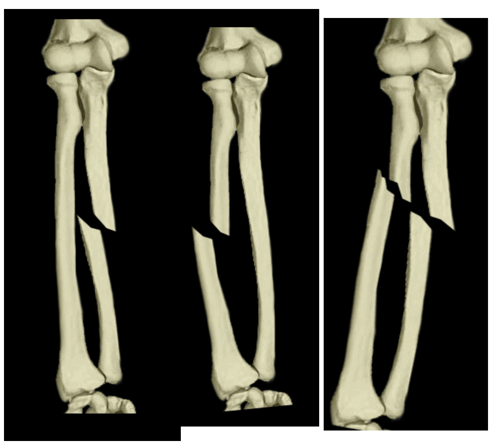
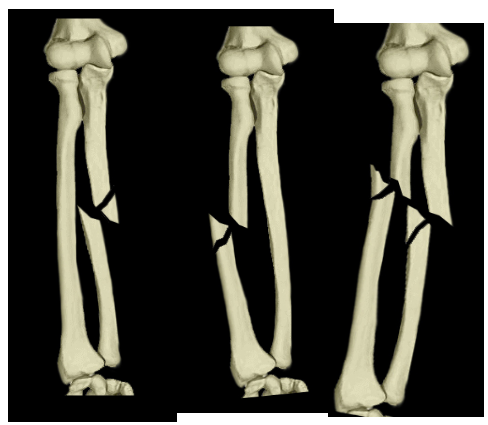
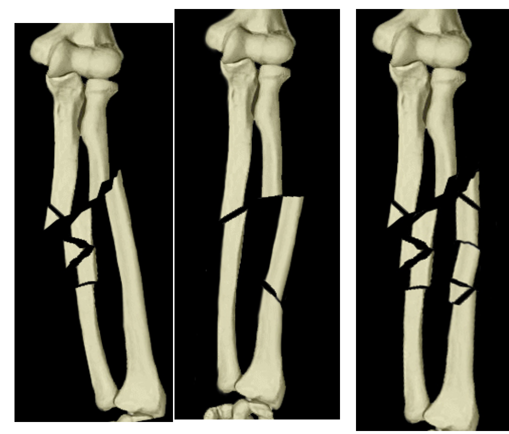
What are the surgical implications?
What are the implications for children?

22- Diaphyseal groups forearm







A1 A2 A3

B1 B2 B3

C1 C2 C3

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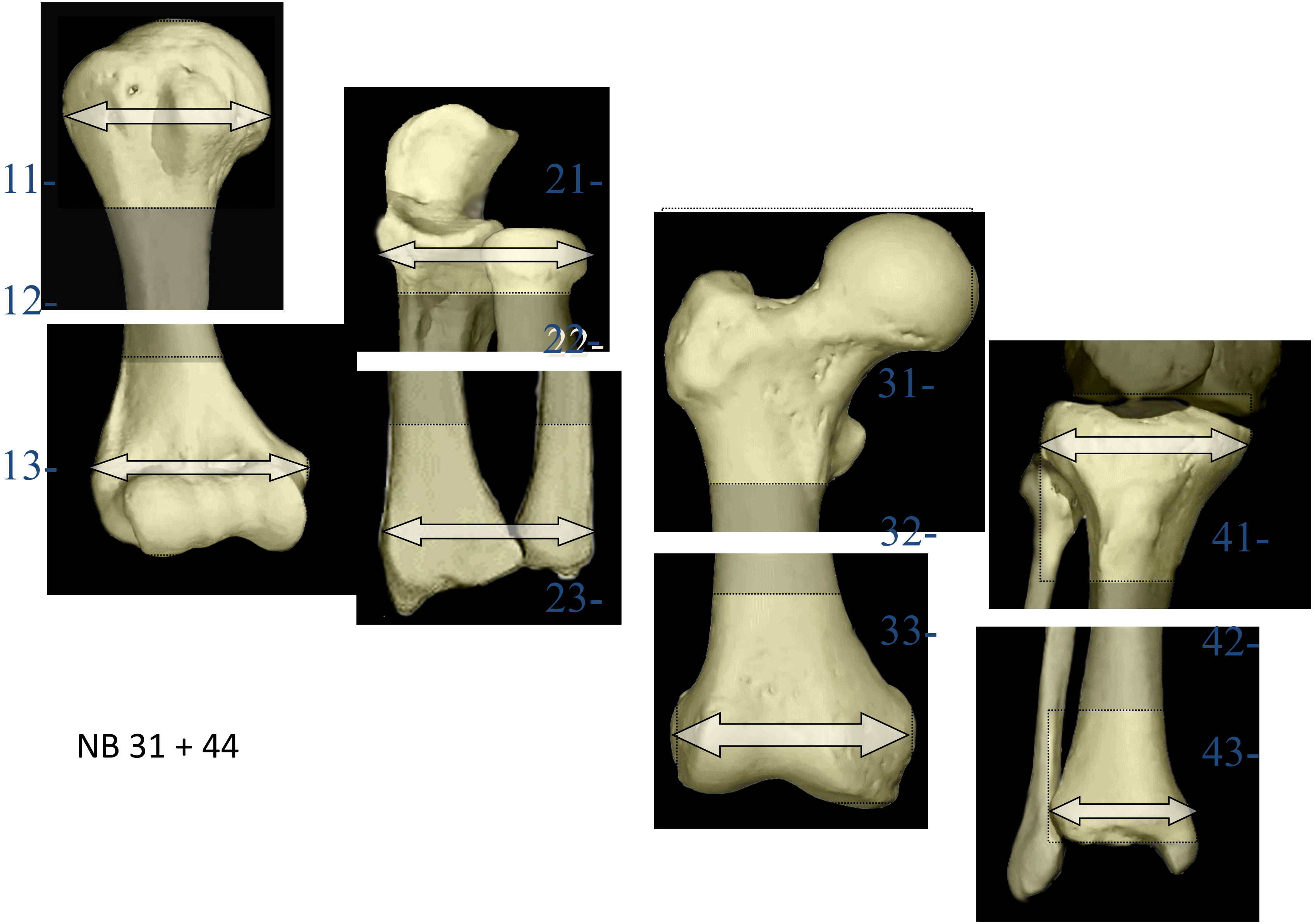
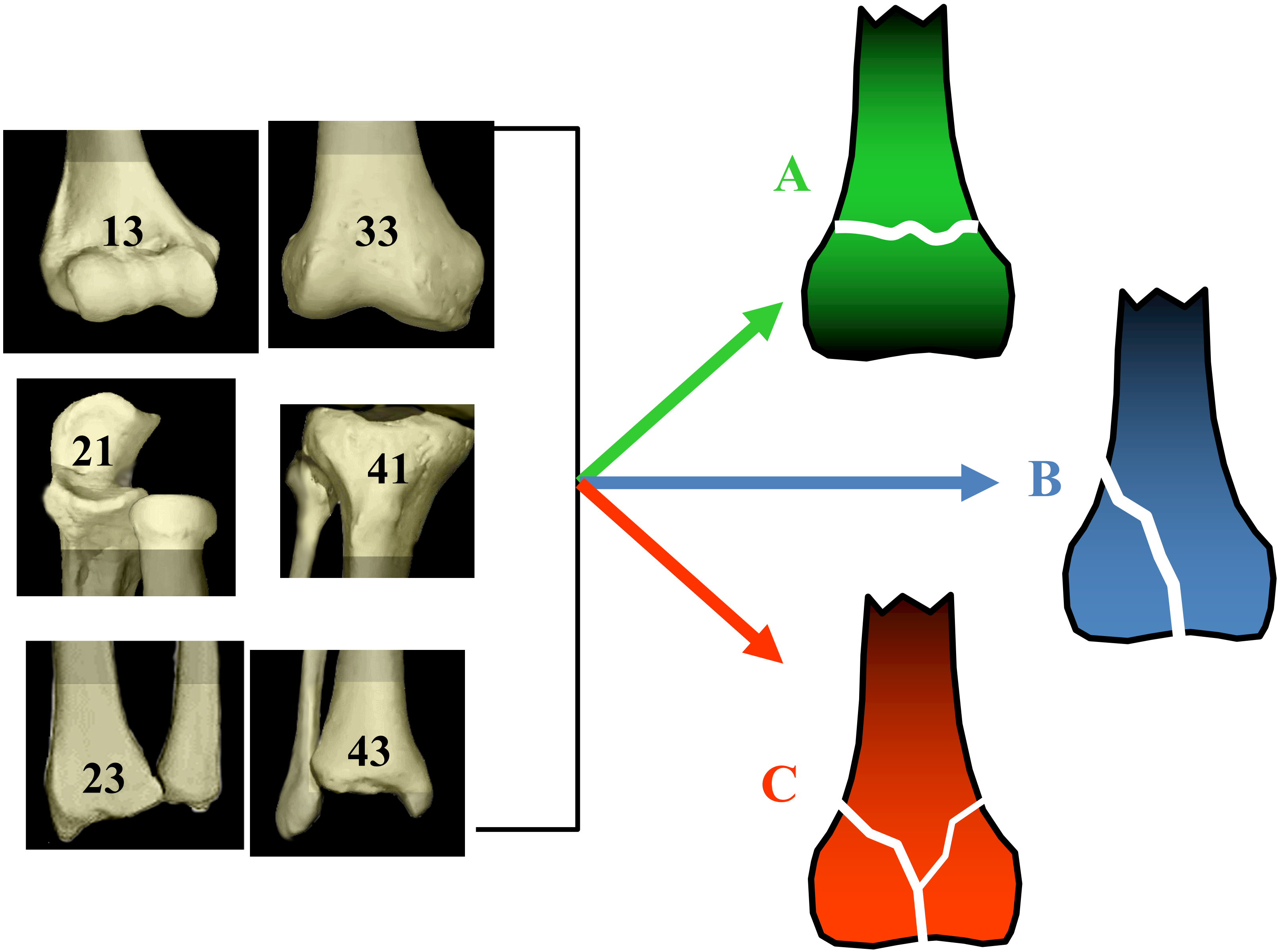


NB 31 + 44

Index



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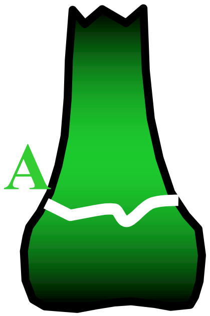
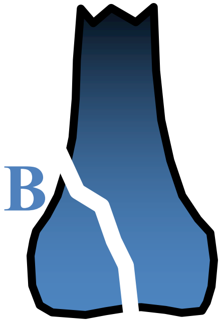
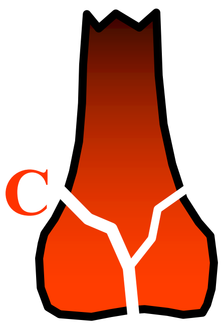
IT IS WORTH BEING AWARE OF THE SUBSEQUENT DIVISION OF ARTICULAR FRACTURES – EXAMPLE OF EACH TO FOLLOW



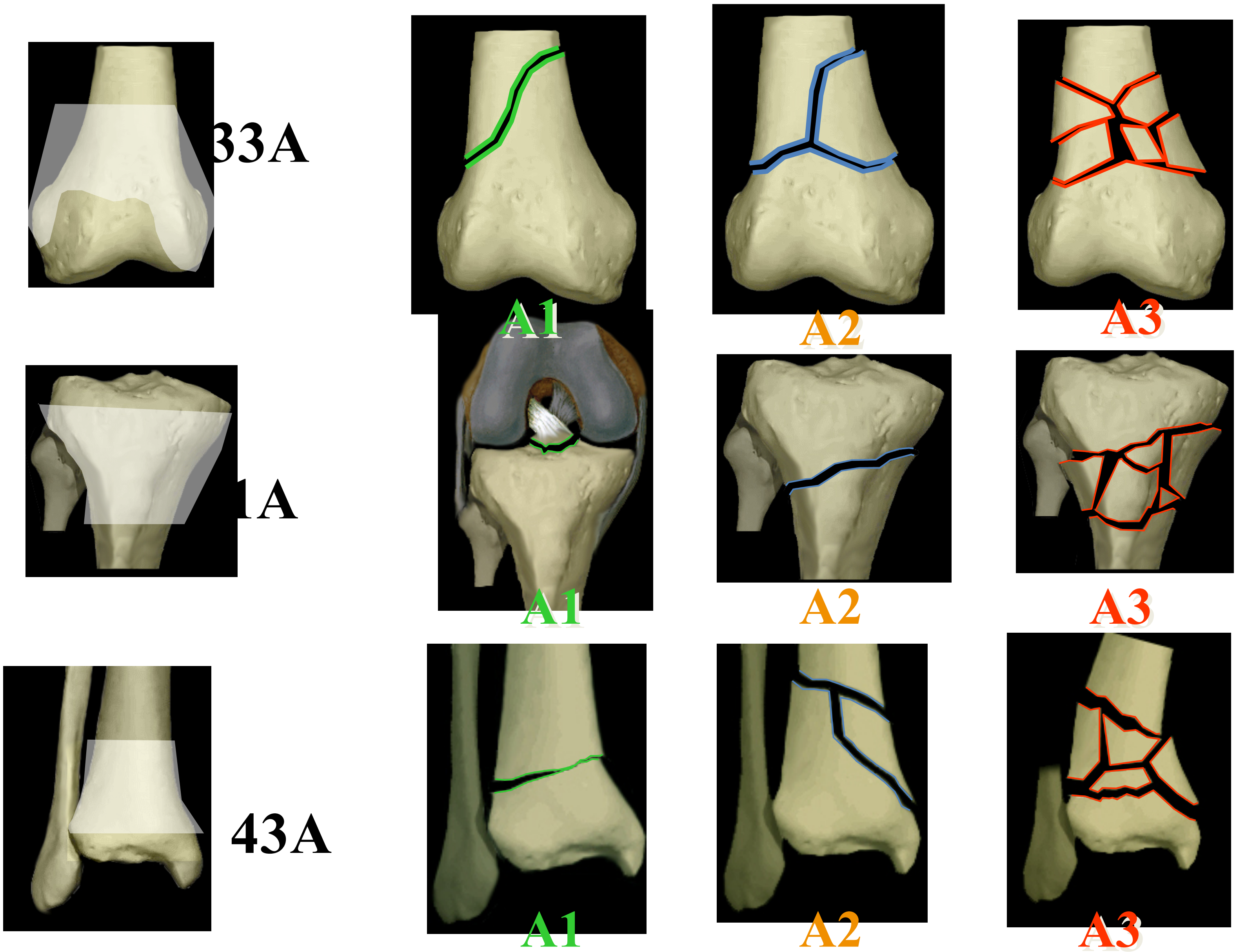
Extra-articular fracture

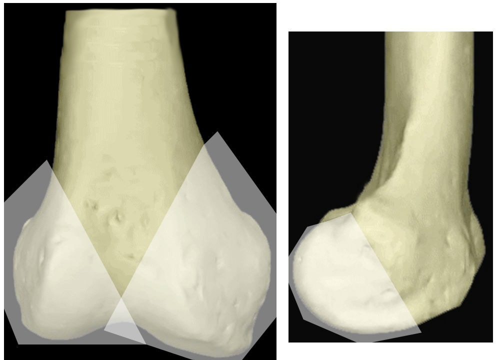


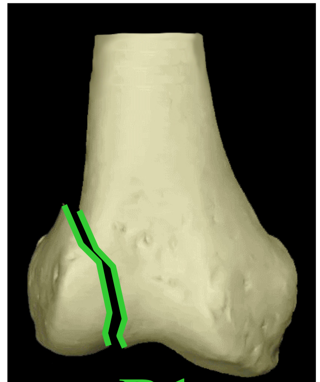
Partial articular fracture – part of joint remains in continuity with diaphysis

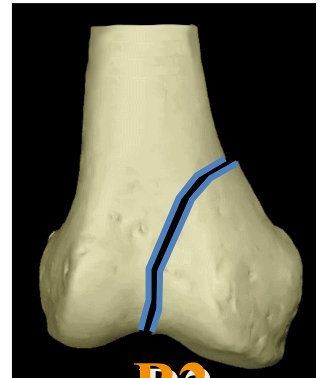


Complete articular fracture – no part of joint remains in continuity with diaphysis


33A
A1
A2
A3
1A
A1
A2
A3
43A
A1
A2
A3


33-B


B1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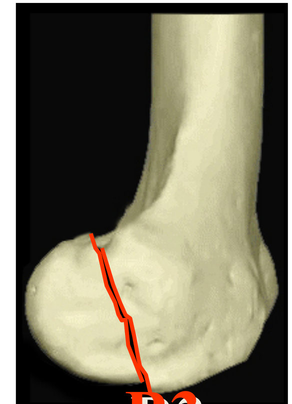
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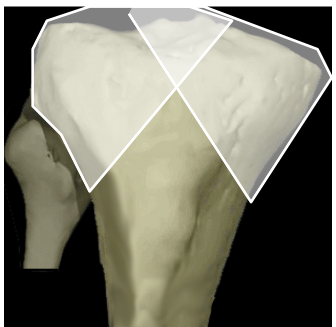
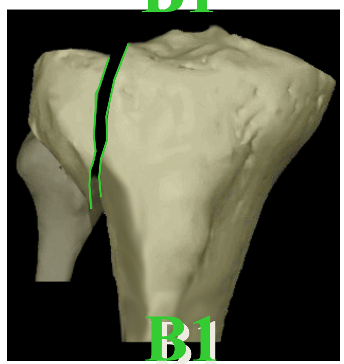
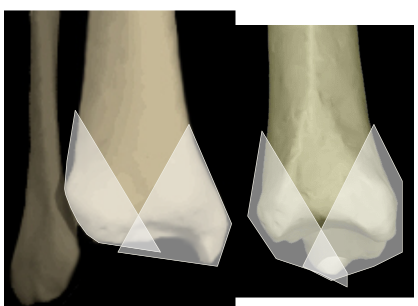
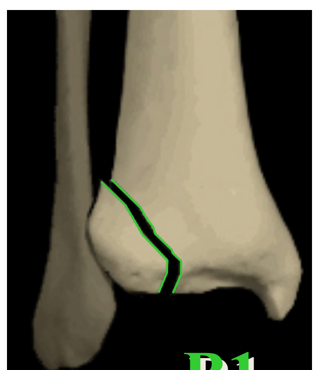

41-B


B1

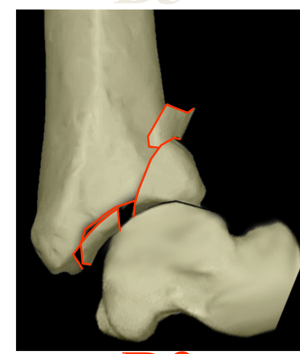

B2


B3


43-B


B1


B2


B3

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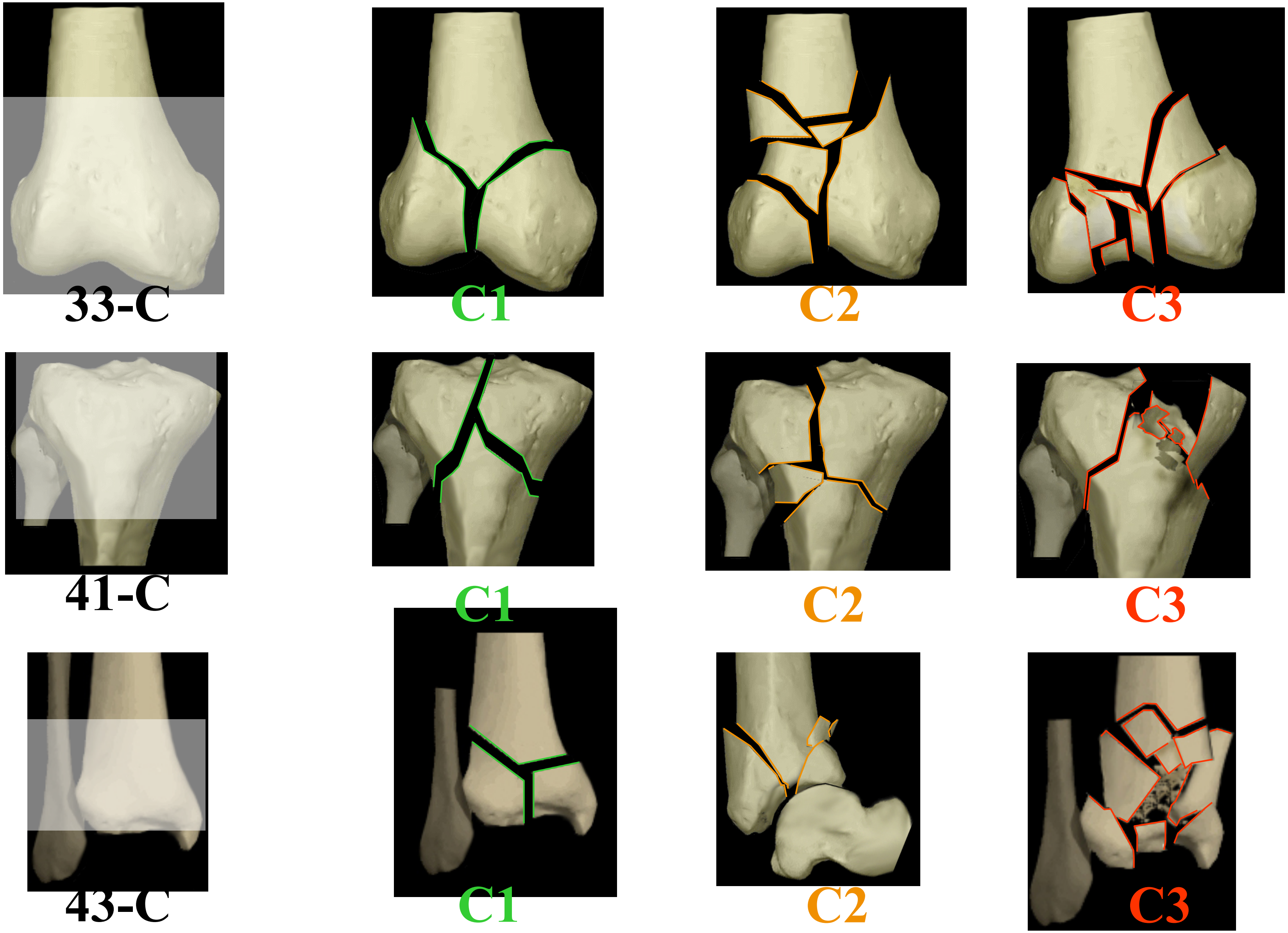

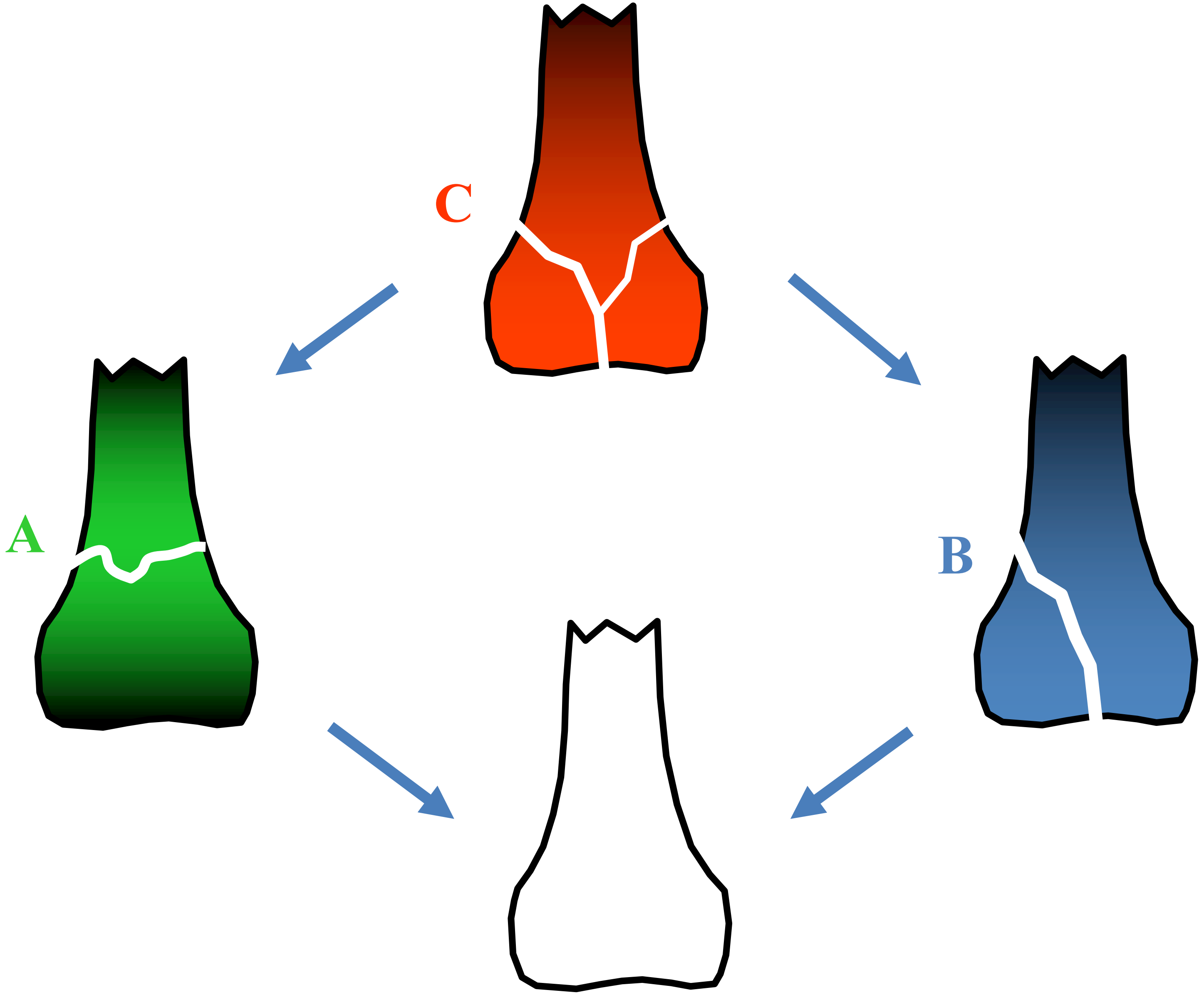

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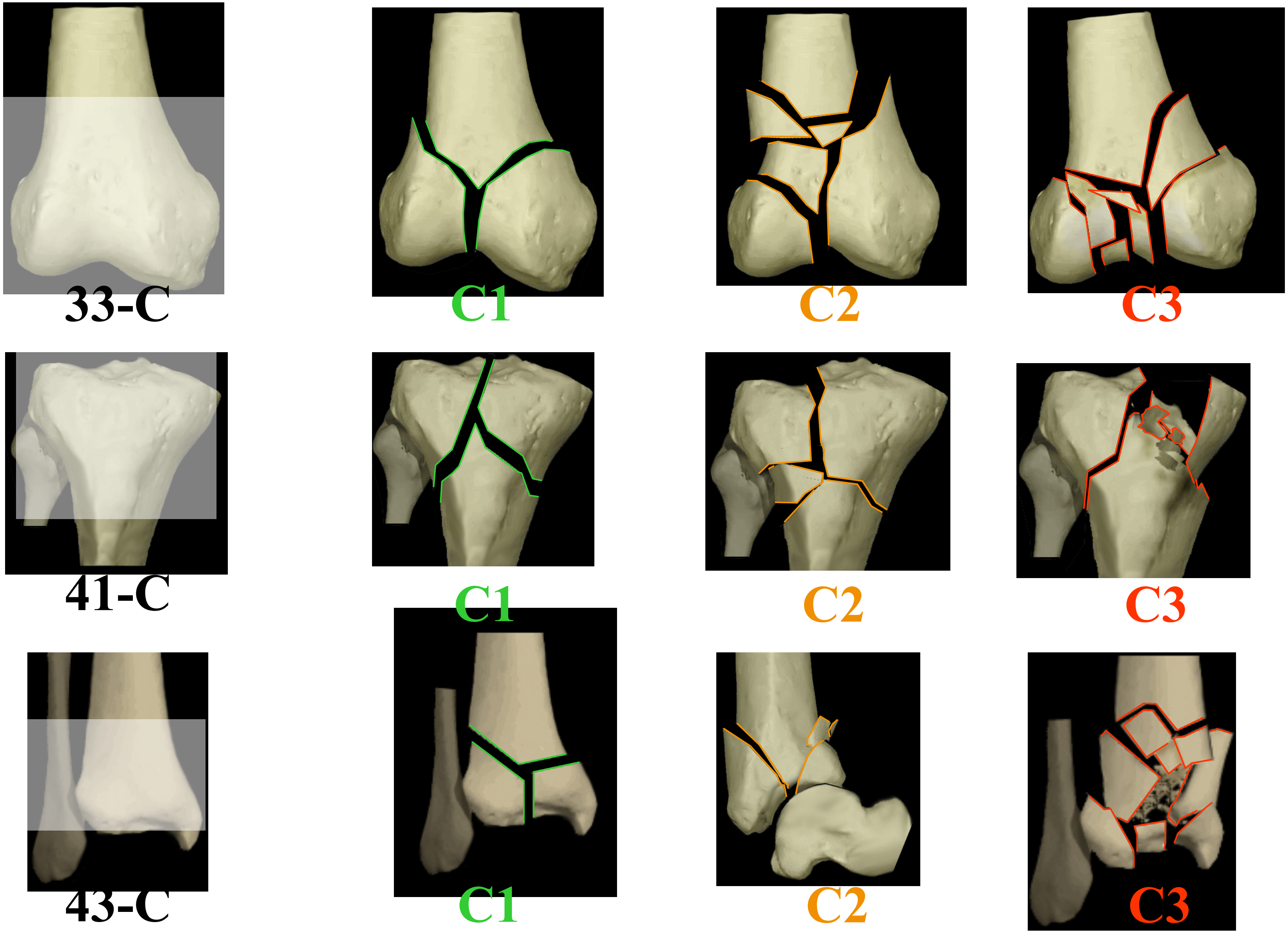

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Variations in Metaphyseal Classification

- Distal humerus
- Proximal + distal radius
- Proximal humerus
- Proximal femur (management governed by other priorities and indications)
- Ankle fractures (other classifications more useful)

Variations in Metaphyseal Classification

- Distal humerus
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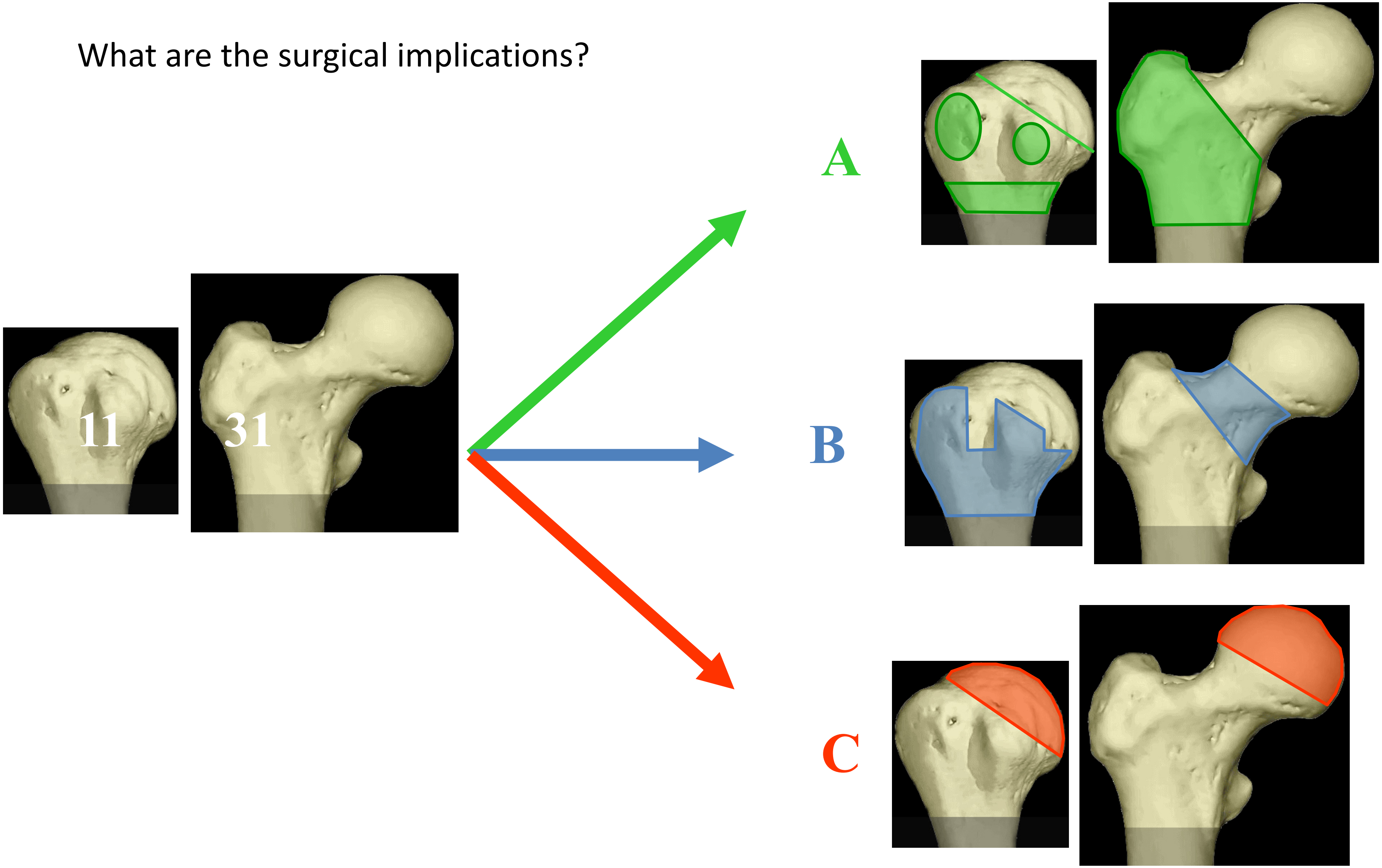
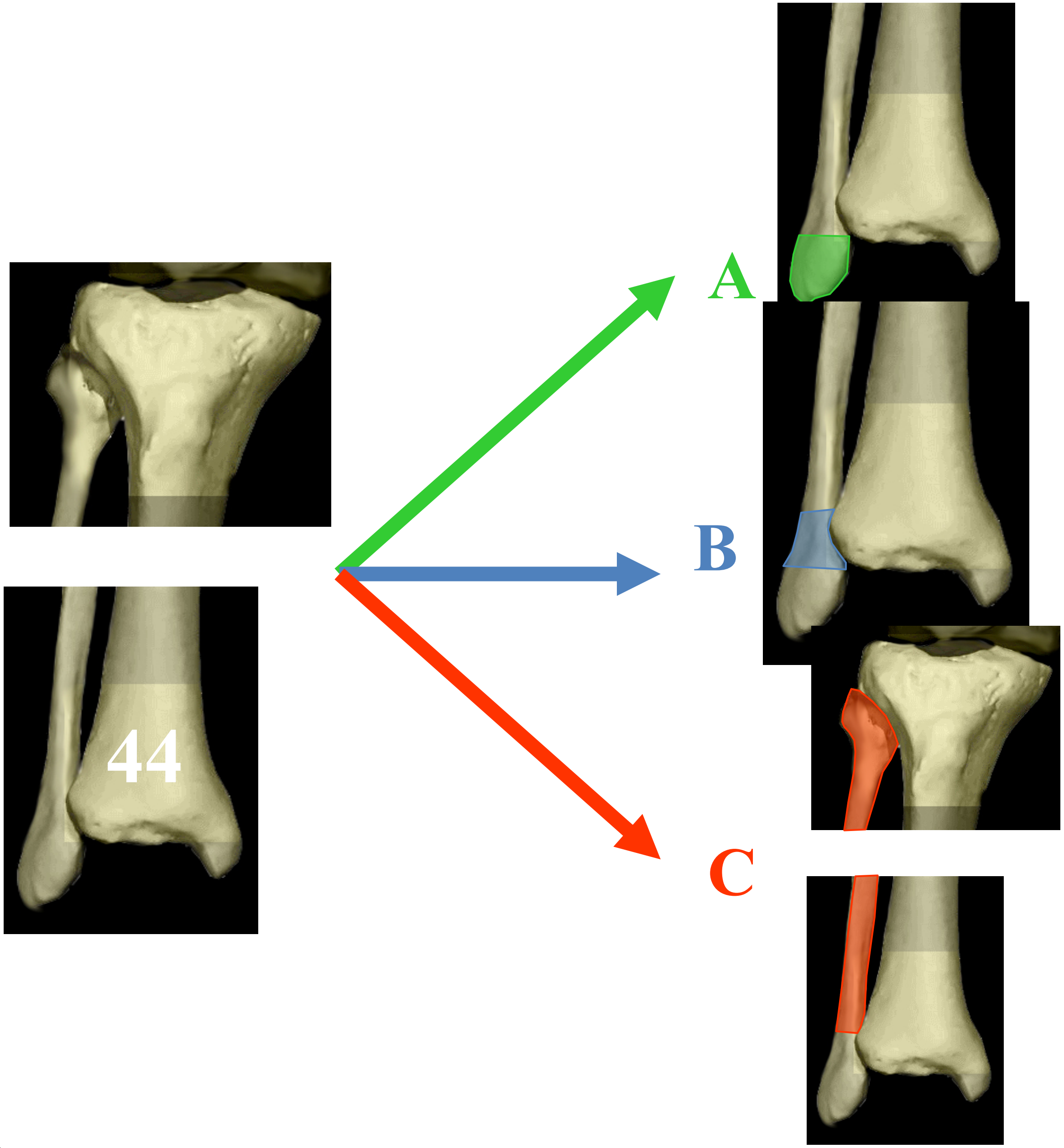
What are the surgical implications?





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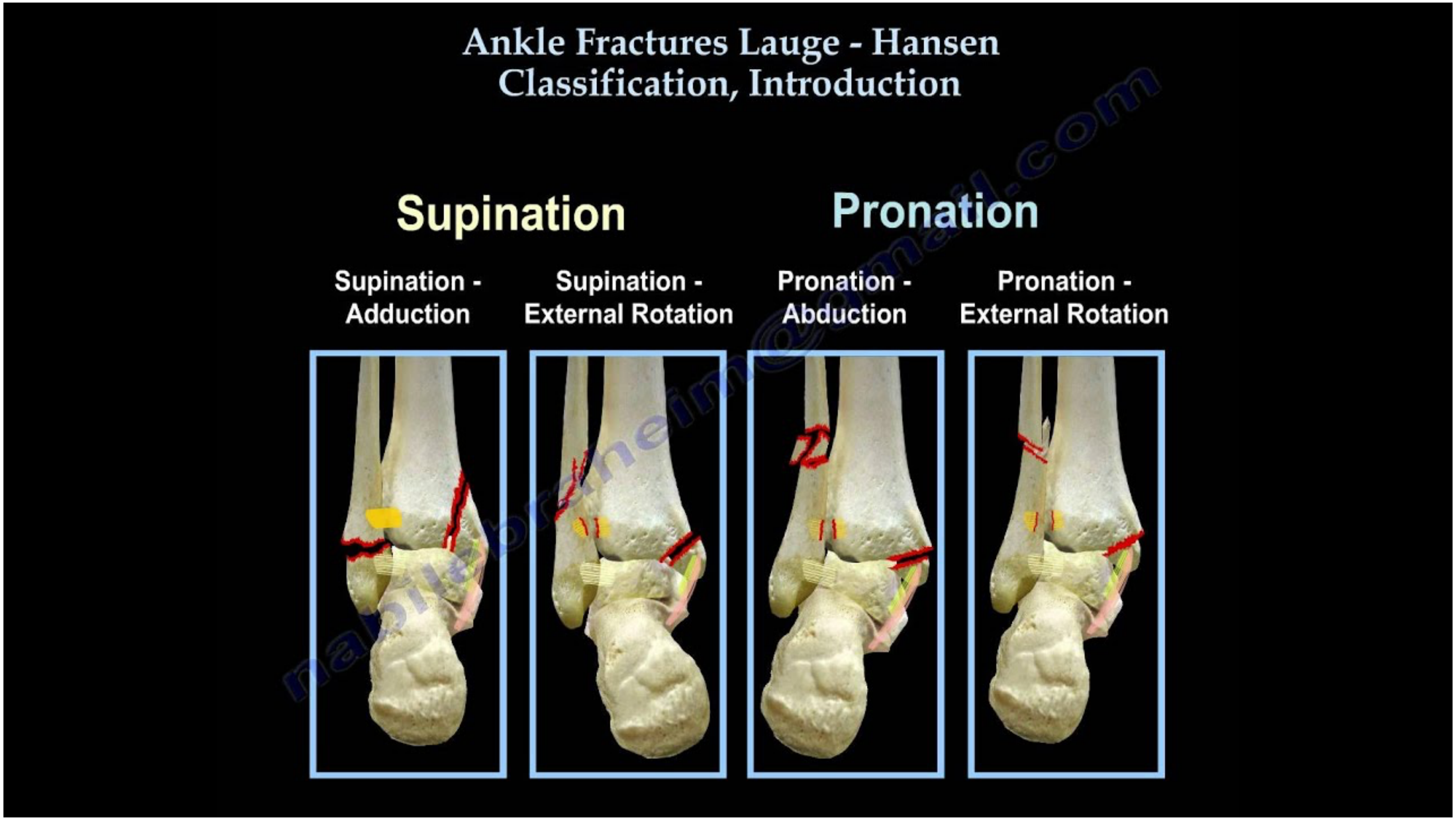
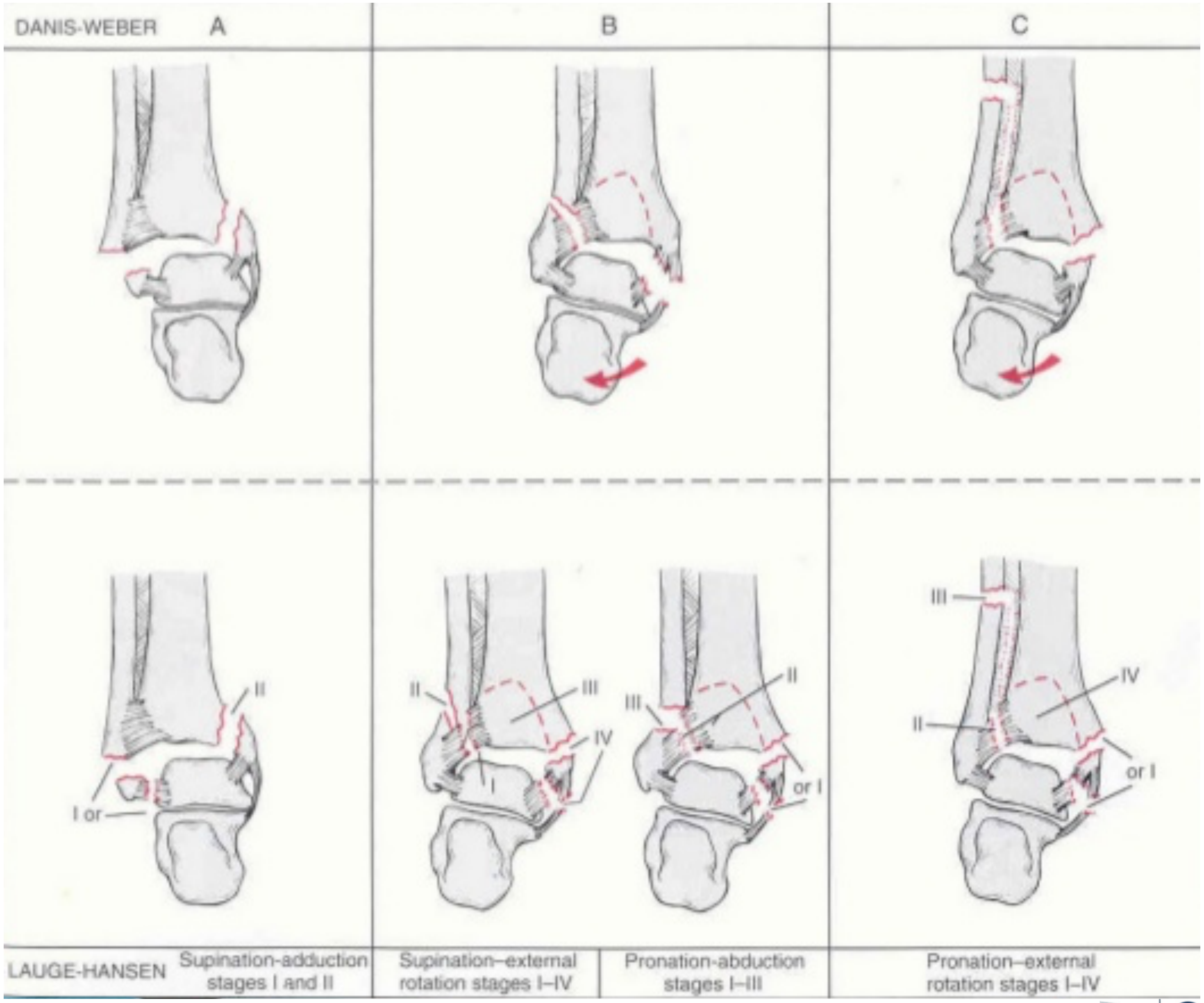
Soft Tissue


Ankle Fractures Lauge - Hansen
Classification, Introduction
Supination
Pronation
Supination - Adduction
Supination - External Rotation
Pronation - Abduction
Pronation - External Rotation

Lauge-Hansen vs Danis-Weber



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Gustillo

Gustillo Grade I	<1cm wound
Gustillo Grade II	>1cm Without extensive soft tissue damage
Gustillo Grade IIIa	Extensive soft tissue damage but adequate soft tissue coverage, or high energy regardless of size.
Gustillo Grade IIIb	Extensive soft tissue damage with periosteal stripping and exposed bone.
Gustillo Grade IIIc	Associated arterial injury requiring repair.

Gustilo, R.B. and Anderson, J.T. (1976). Prevention of infection in the treatment of one thousand and twenty-five open fractures of long bones. *Journal of Bone and Joint Surgery*, **58A**, 453–8.

Gustilo, R.B., Mendoza, R.M., and Williams, D.N. (1984). Problems in the management of type III (severe) open fractures: a new classification of type III open fractures. *Journal of Trauma*, **24**, 742–6.

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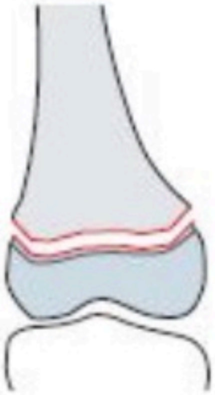
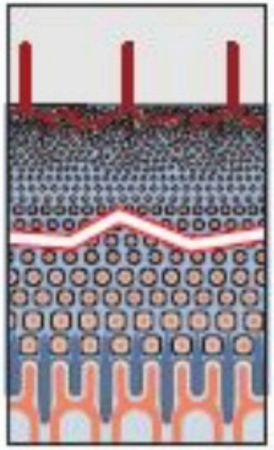
Salter Harris I





The fracture passes along the growth plate, passing through the junction of the zones of hypertrophy and provisional ossification. The fracture line does not involve growth zones, and growth disturbance is unlikely.

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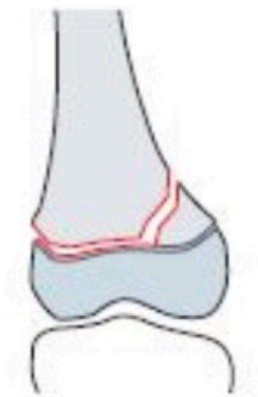
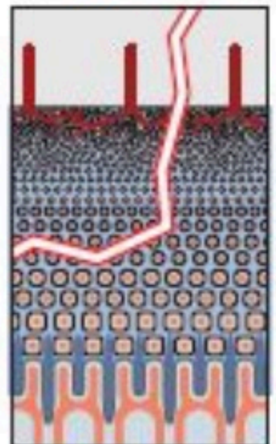
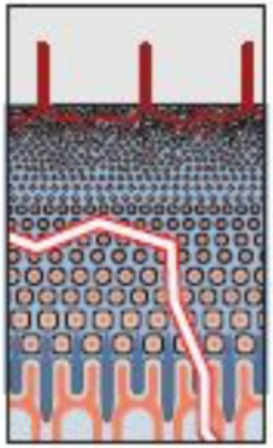
Salter Harris II





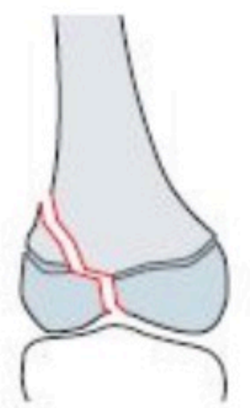
This is a shear injury of the growth plate with a partial metaphyseal fracture (Thurston-Holland fragment). This type accounts for 70% of physeal injuries. Like the type I fracture, this injury does not involve the growth zones, and growth disturbance is unlikely.

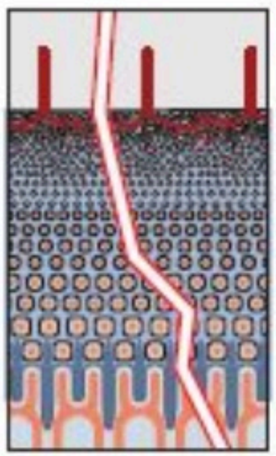
Salter Harris III



There is a partial physeal separation with an intraarticular epiphyseal fracture. The fracture traverses the growth zones. If reduction is not perfect, growth disturbance is highly likely. Open reduction is necessary.

Salter Harris IV





The fracture passes from the joint surface through all layers of the physis and through the metaphysis. The growth zone is involved. Anatomical reduction and fixation is required.

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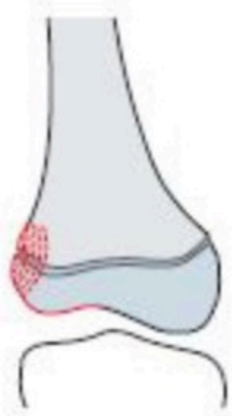
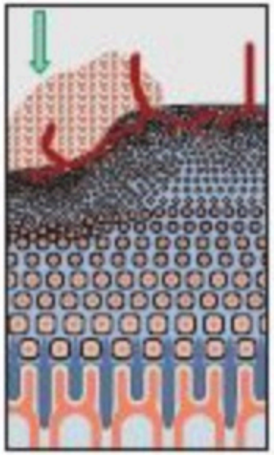
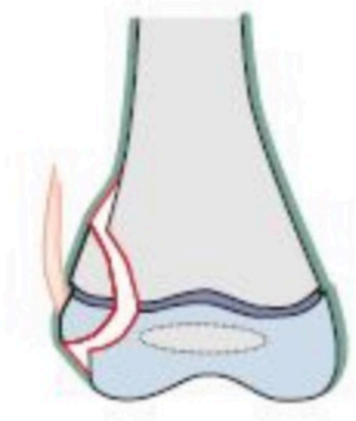
Salter Harris V

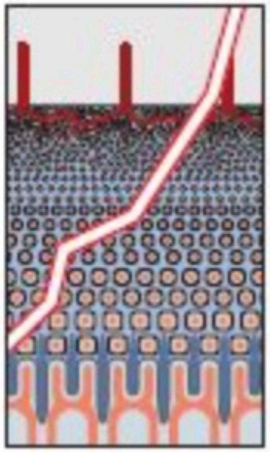




There is impaction of the articular surface and growth plate. This type of injury is often diagnosed in retrospect. Partial growth arrest occurs.

Salter Harris VI





Avulsion fracture at the insertion of a ligament, taking with it a portion of the perichondral ring (Ranvier zone). Accurate reduction and fixation are required; nevertheless growth disturbance can occur.

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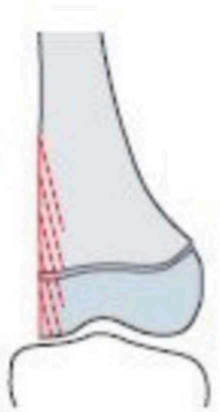
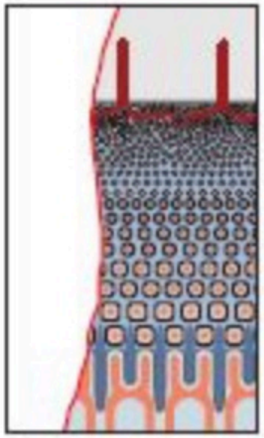
Salter Harris VII



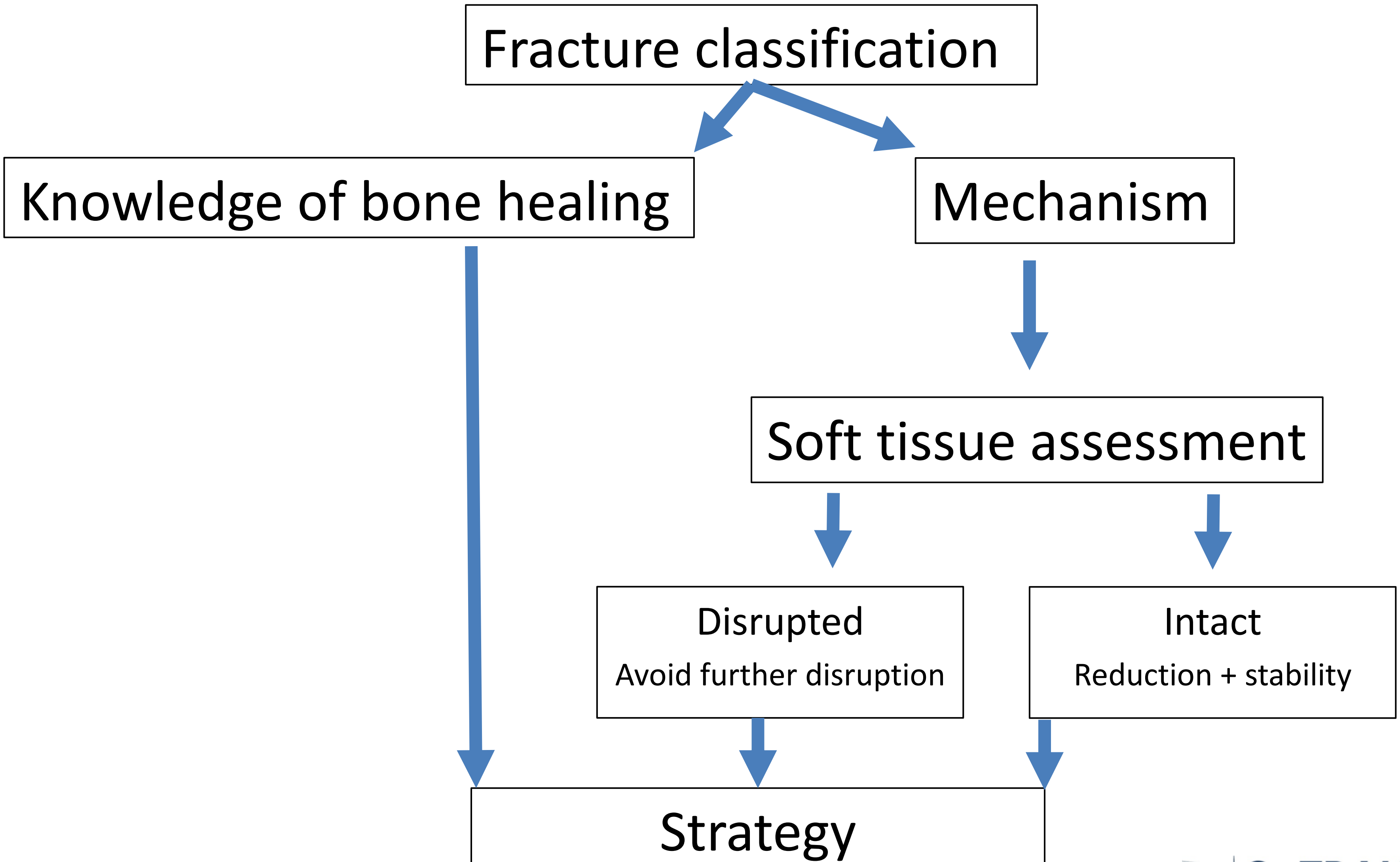


Open abrasive injury of the periphery of the growth plate; this often causes physeal bridging.

Practice Cases

- Planning sheets

Fracture Management Algorithm

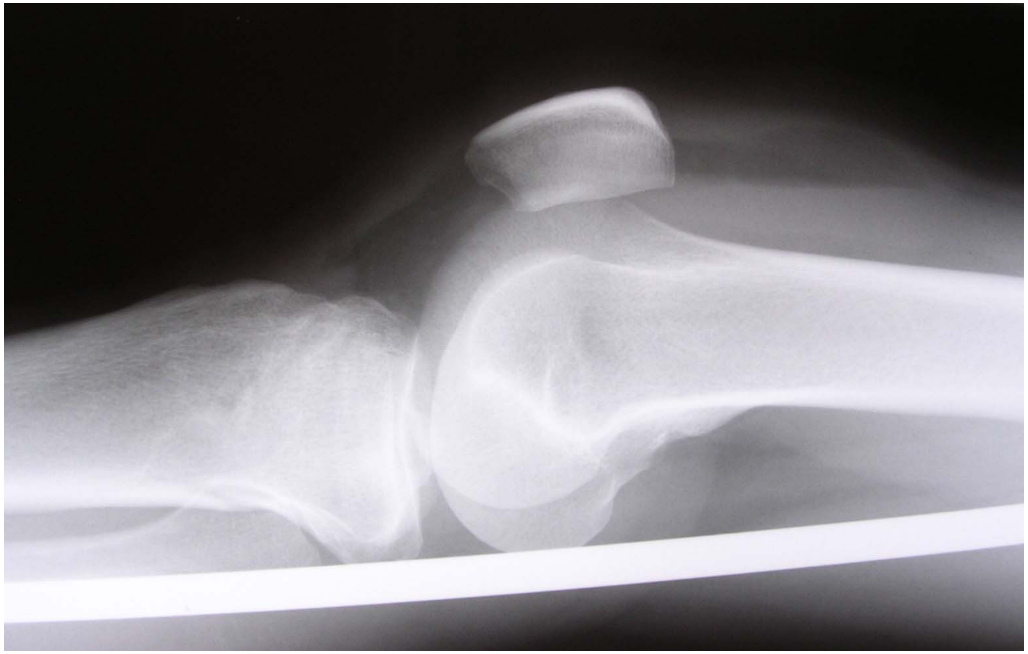


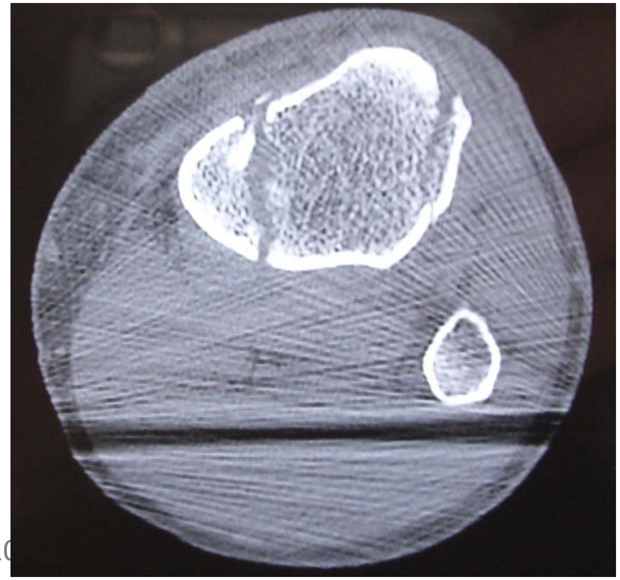
Case 1

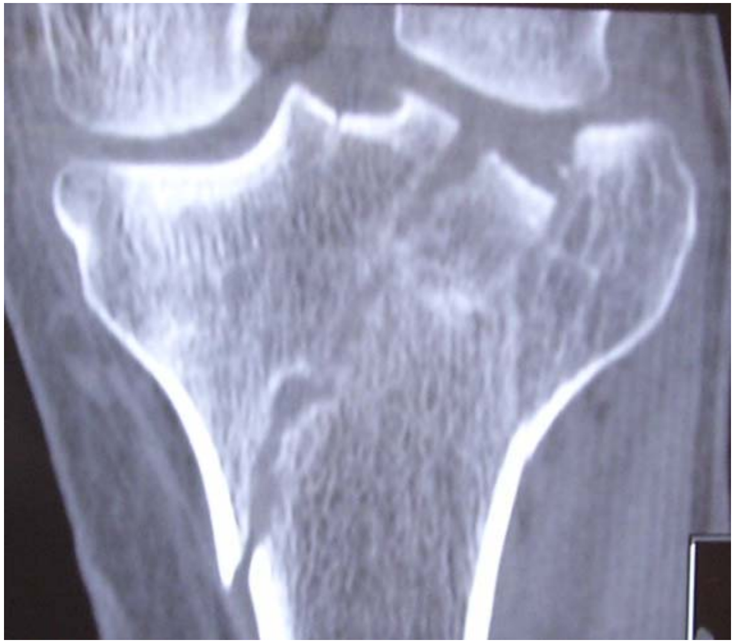
- (learning difficulties case admitted Wed 30th)

Case 1

Case 2







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Plan

Strategy	Equipment	Potential problems
Pre-op Set Up Approach Reduction Fixation Post Op		

END