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Orthopedic 441 Team

MED441 KING SAUD UNIVERSITY

Editing File

Principles of Fractures

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Color Index:

- Main Text
- Important
- 441 Notes
- Old Notes
- Extra

Objectives

- Introduction.
- Basic science of fracture healing.
- Principles of evaluating patients with fractures.
- Principles of management.
- Common fractures in adults.

Resources

Principles of fractures part 1 in Arabic
By Learn with Dr.Wahdan

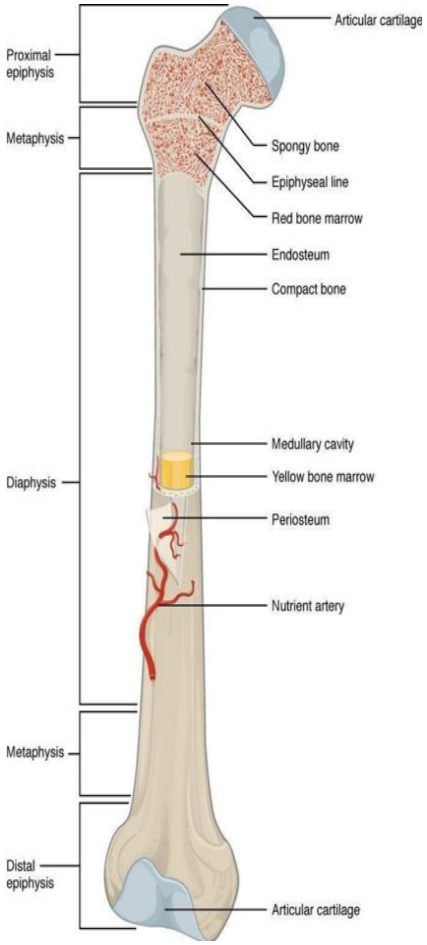
Basics Review (Extra)

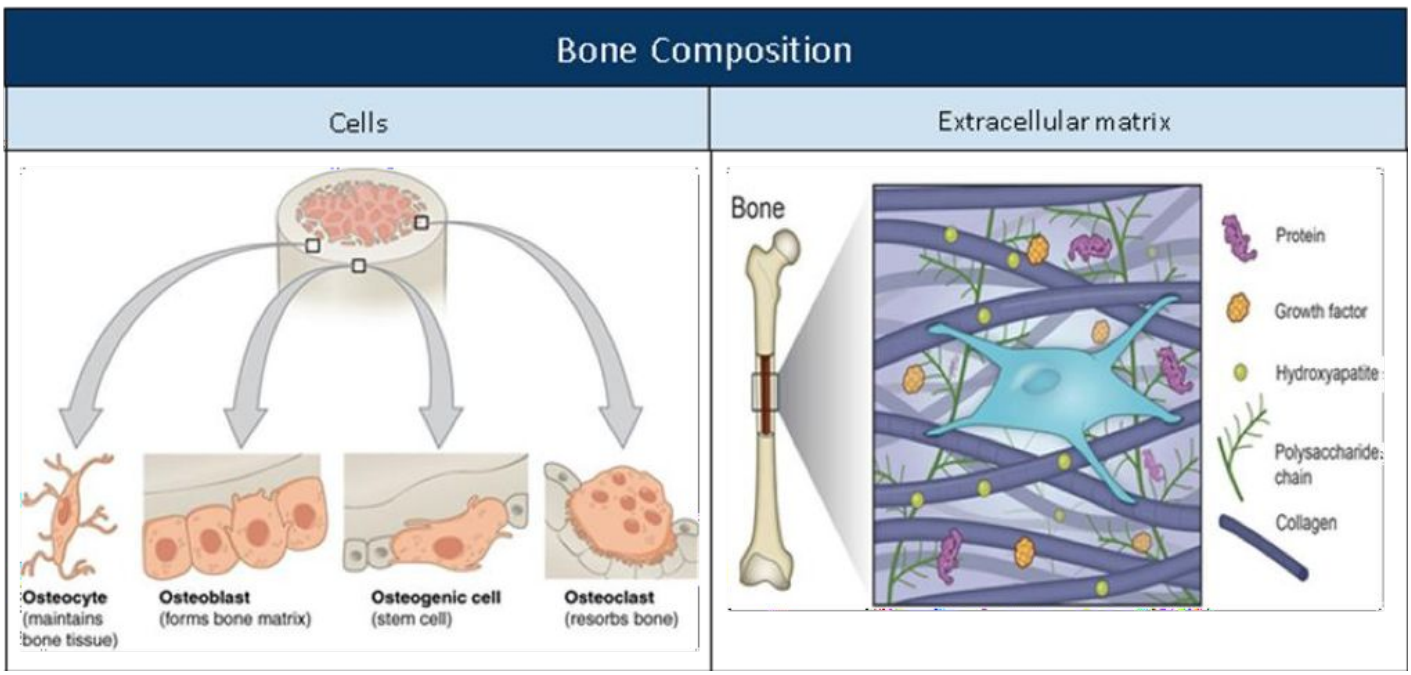
Bone structure, types & composition

1. Lamellar Bones (Dense):
They're composed of collagen fibers arranged in parallel layers and they're found in the diaphysis part of normal adult long bones. The basic functional unit of lamellar bones is called an Osteon or "Haversian System".

2. Cancellous Bones (trabecular or spongy bone):
They're less dense and more elastic than lamellar bones. They're found in the metaphysis part of small bones.

3. Woven Bones
They're coarse bones with random orientation. They're also weaker than lamellar bones. In bone healing, they're eventually remodeled to lamellar bones.





Principles of Fractures

Introduction

- **Definition of fracture:** means literally broken bone.
- **Fractures can be described in different ways such as:**
  A. Extent 'complete or incomplete'.
  B. Location 'which part of bone is affected'.
  C. Morphology 'pattern of the fracture'.
  D. Mechanism 'high energy (suspicion of other injuries) v.s. Low energy'.
  E. Associated soft tissue injuries 'open or closed fracture'.

Extent

- **Complete**: Fracture that extends 360° of bone circumference (all around), requires operative treatment.
- **Incomplete** (one cortex is broken, the other cortex is intact): Seen almost only in **children** because they have elastic bone such as:

Greenstick fracture	Buckle (torus) fracture
If you try to break greenstick it will not break because it's **elastic** = children have elastic bones. Still can happen in adults.	Not a complete break just a deformity. it's also common in children bones. Managed by: Below elbow cast for 2-3 weeks
	

Location

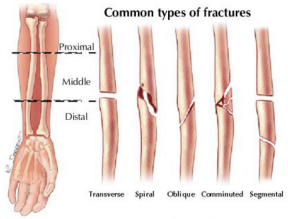
In the skeletally mature individual, there is no physis or epiphysis, and the entire end region of the bone is termed the metaphysis.

A. **Name of bone.**
B. **Side** (right or left).
C. **Diaphysis** (shaft), **metaphysis** (spongy or cancellous bone) or **epiphysis**.
D. **Long bones (diaphysis):** divide them in thirds (proximal, middle or distal third).
E. **Metaphysis:**
   - **Intra-articular**[1]. (Affect the growth plate and cause under deformity either shortening or leg length discrepancy 'LLD', risk of post-traumatic **osteoarthritis**).
   - **Extra- articular**. (No risk of OA). **It is important because there is different management and complications.**

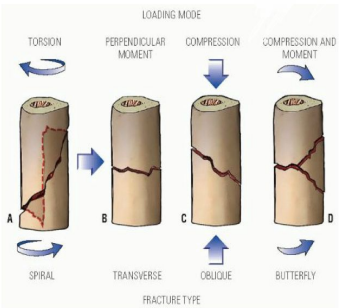
1- Intra-articular metaphyseal fractures carry **the risk of post-traumatic osteoarthritis** especially if fracture is displaced more than 2mm or the gap is more than 2 mm → the management will aim to restore normal joint, sometimes it includes epiphysis and metaphysis, and this has special classification you will take in pediatrics, in immature skeletal system with intra-articular fracture we need to put compression screw parallel to the growth plate then we can fix the distal parts with another compression screw and immobilize with cast.

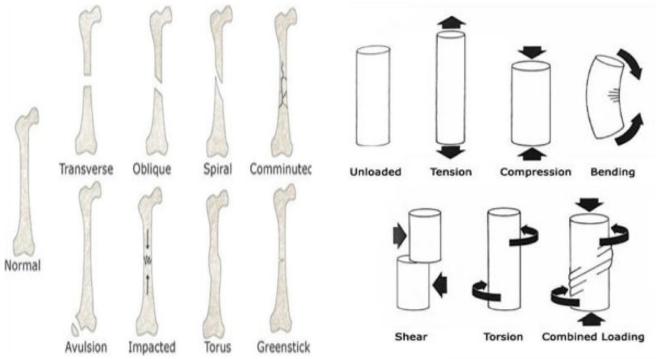
Morphology[1]

Help to guess the mechanism of the fracture



1. **Transverse:** loading mode resulting in fracture is tension.
   Avulsion results in transverse fracture. The mechanism of transverse is direct trauma to bone.
2. **Oblique**: loading mode is compression from both ends. There are 2 types
   1- Short oblique: the diameter of the bone is equal to length of fracture line
   2- Long oblique: the fracture line is double the diameter of the bone.
   Example: If the bone diameter is 1 mm and oblique fracture line is 1.5 mm then still short oblique. But if it is 2 mm then long oblique.
3. **Spiral**: loading mode is **torsion** or rotation or twisting. (Usually occurs in sports & pediatric age group).
4. **Wedge** (fracture with butterfly fragment): loading mode is **bending**. (Compression + rotation).
5. **Comminuted**: 3 or more fragments, usually results from **high energy** trauma.
6. **Segmental**: a fracture in **two parts** of the same bone.





- **Segment** is a big piece of bone that is broken from above and below and mechanism of the fracture disconnected from the rest.

- **Wedge** is broken but there is contact on one side.

Mechanism

Very important to know, so you can decide how to approach a patient. If it's high energy trauma your priority is to save the patient, so you use ATLS approach.

- **High energy v.s. Low energy** 'In high energy accidents, soft tissue injuries are expected' e.g.
  - RTA and risk of multiple trauma, in high energy trauma we try to preserve life first (ABC) then limb then its function (ATLS).
  - Low energy trauma usually one isolated injury, example: fall in bathroom causing femur neck fracture, we do screening.
- **Multiple injuries v.s. Isolated injury** usually high energy can cause multiple injuries or polytrauma injury, but still even if you're exposed to high energy you can end up with isolated injury, If a patient present with more than one bone fracture we consider it high energy trauma
- **Pathological fracture:** Normal load in presence of weakened abnormal bone density (tumor, osteoporosis, infection). Weakened bone breaks easily by low energy trauma, (if you see young patient with femoral neck fracture, suspect a pathological fracture).
- Stress fracture: **Normal bone** subjected to **repeated** load (military recruits/athletes) usually it will be incomplete small fractures (fissure).

1- Why is it important to know the morphology of a fracture? To classify fractures to **stable** and **unstable**.
- **Stable fractures:** transverse fracture → can be treated non-operatively.
- **Unstable fractures:** All types except transverse fracture → may need surgical intervention because of risk of displacement.

Associated Soft Tissue Injuries

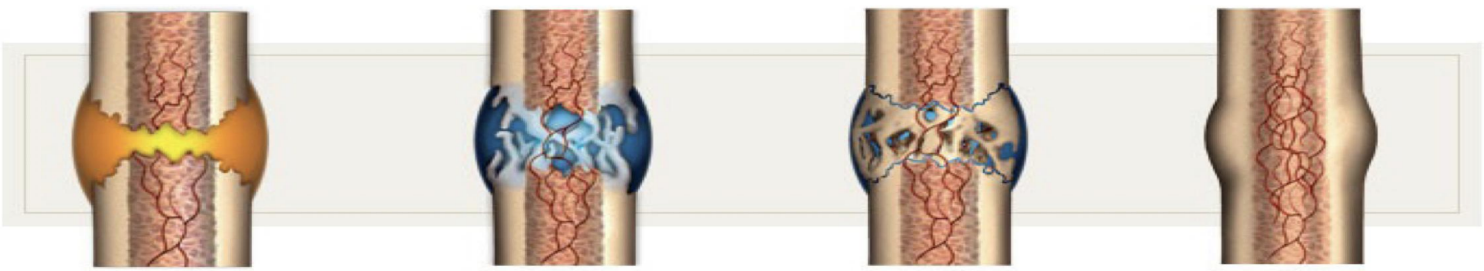
- **Closed fracture:** skin integrity (most important thing) is maintained (intact).
- **Open fracture:** fracture is exposed to external environment. (Use Gustilo classification)
  - Whenever there is a soft tissue break at or near the fracture site; it is considered open fracture until proven otherwise. if it was missed the patient will end up with osteomyelitis and may require amputation. Worsen the outcome of form or fracture.
  - (Any skin breach in proximity of a fracture is an open fracture until proven otherwise)

Fracture Healing (Natural bone healing)

1. Direct/Endosteal/Primary bone healing:
   It can happen if no motion takes place. Bone is formed without intermediate stage through the "Cutting cone mechanism". (No gap or < 1mm gap be in fracture site) when the two fracture surfaces come in contact without overlapping.

2. **Indirect**/Endochondral/Secondary **bone healing:**
   Called indirect because of the formation of cartilage at an intermediate stage. The process occurs in nature with untreated fracture through **endochondral ossification** (occurs in **fractures with gap**). **It runs in 4 stages**:

1. Hematoma formation (1-2 weeks)	2. Soft Callus (2-6 weeks)	3. Hard Callus (6 weeks-12 months)	4. Remodeling (Years)
(Inflammation phase) In this phase, there is disruption of the blood vessels, migration of cells occurs, and coagulation begins. Hematoma forms a good media for the bone to heal so it is better to not disrupt this phase.	In this phase, cascade of cellular differentiation occurs, angiogenesis takes place, and fibroblasts produce granulation tissue that eventually evolves into fibrocartilage. the most important cells: fibroblasts, cartilage cells and with time they will calcify resulting in hard callus. You see a patient with fracture; on x-ray you don't see anything, but clinically there is a firm lump on the fracture site, which stage of healing? → Soft callus. If you start seeing calcification or ossification so this is hard callus.	In this phase, endochondral ossification converts soft callus into woven bone. The process starts at the periphery and then moves centrally. It continues until there is no more movement. Whenever you see the hard callus you can remove the cast and start range of motion.	(reshaping the bone) In this phase, the **woven bone** that was formed is converted into **lamellar bone**. The duration of this phase depends on the type of fractured bone and the site of fracture.



- The most important factor in determining the natural tendency to heal is the state of the surrounding soft tissues and the local blood supply.
- The greater the contact surface area between fragments, the more likely healing is to occur.

Principles of Evaluation

History

- Patients complain of pain and inability to use the limb (if they are conscious and able to communicate).
- What information can help you make the diagnosis? Pain → SOCRATES.

DDx in ortho

Painful:
1- Trauma
2- Tumor
3- Infection

Painless:
1- Congenital
2- Syndromic

Onset

- When and how did the symptoms begin?
- Specific traumatic incident v.s. Gradual onset?

If there was a specific trauma

- Mechanism of injury? (High or low energy trauma)
- Circumstances of the event (Work-related, farm, street)?
- Severity of symptoms at the time of injury and progression after?

If you suspect a pathological fracture

- Prior pain before event happened?
- Constitutional symptoms?
- History of cancer?

If you suspect stress fracture

- Recent increment of activities?

Physical Exam

If the patient had high energy trauma you have to save his life first (ATLS)

Look (Inspection)

- Swelling
- Ecchymosis
- Deformity[1]

If fracture is open:
- Bleeding
- Protruding bone

Feel (Palpation)

- Bony tenderness
- Examine joint above and below.
- Bony landmarks, temperature and pulse.

Move

ROM (Cannot be assessed in acute fracture).
- Move is not important in fractures → painful. However, if there is subtle fracture like stress fracture, we need to ask the patient to move but in general if there is an obvious fracture it should not be done, there is no special tests for trauma.

If a fracture is suspected what should we rule out? Red flags

- **Neurovascular injury** (N/V exam) distal to the fracture. (Do N/V exam before and after you do any splint, realignment or sending the patient for imaging)
- **Compartment syndrome** by assessing the 5 P's.
- Associated **MSK injuries** (by examining joint above and below at minimum).
- Open fracture/ infection is too early to happen, it happens later.
- Cauda equina syndrome in spine fracture.
- Pelvic fracture in cases of polytrauma (can cause life-threatening bleeding). Patient can lose 2 L of blood if he has femur fracture/ he can lose his whole blood in pelvic fractures. What are the life threatening injuries in the pelvis? Open book injury → symphysis is disrupted. In normal pelvis, it can take up to 3 liters however in an open book pelvis, it can take up to 6 or 7 liters. So, you must close it down to minimize the volume.

1- **You have to describe the deformity** (muscle wasting, wounds, skin changes).

Imaging

- Fractures may be obvious on X-ray
- Undisplaced or stress fractures are sometimes not immediately (after trauma) apparent, needs CT or MRI.
- Before You send the patient to the X-ray you should **immobilize him for two reasons:**
  - Prevent secondary soft tissue injury, and prevent vascular injury.
  - Minimize the pain for the patient. We should immobilize joint above and joint below, initially do back slap because it's easy to apply and easy to remove, don't do a complete cast unless you have a definitive Dx and plan because it's difficult to remove and might be painful.

X-ray: AP view of left humeral midshaft spiral fracture

X-ray Principles

1. **Two orthogonal views:**
   - AP (for displacement): will tell you if there is distal (lateral or medial) displacement related to proximal fragment.
   - Lateral (for Angulation): Lateral view will tell where the angulation is, is it posterior or anterior, volar or dorsal.

2. **Two joint** – above and distal why?
   - To see the extent of the fracture (intra-articular or extra-articular).
   - To see if the joint lines of the two joints above and below are parallel to each other then that means there is no rotation so no need to realign the limb.

3. **Two limbs** – to compare in pediatric fracture.
   - In children there is growth plate, so you need to compare both limbs to make sure that this is a growth plate not a fracture.

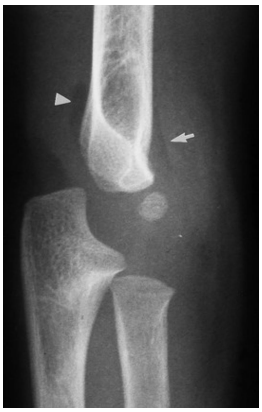
4. **Two occasions.**
   - The findings of X-ray in some cases will not appear at the time of trauma so you need to repeat it after a while. Use it for scaphoid fracture (patient presented with history of outstretched hand and he has pain, swelling, tenderness and restricted ROM you suspect scaphoid fracture, you do an X-ray, it will look normal initially but even then, you should treat and immobilize him by cast from the knuckles until below the elbow in resting position and repeat X-ray after 2 weeks and you will see fracture hematoma and the bone will be resolved). Also use it if you suspect infection (repeat x ray after 2 weeks) or stress fractures.

5. **Special view.**
   - Especially if ligamentous injury is suspected (because plain x-ray without stress will not show any diastasis or ligamentous injury). It is painful so we give the patient anesthesia or analgesia to relax him. Also there is MRI but usually it's not available early at the time of fracture or for trauma management and will cost the patient.

NB: Fractures hurt, immobilization helps. Immobilizing a patient in a backslab is the most effective way to relieve pain from a fracture and may be done **BEFORE** getting X-rays.

Secondary Signs of Fracture on X-ray

- If fracture isn't obvious look for these signs to help you find it:

Soft tissue swelling	Fat pad sign	Periosteal reaction	Joint effusion	Cortical buckle
You can differentiate between subcutaneous tissue, muscle and bones (3 have different densities)	"The darkness" Mostly non-displaced fracture in immature skeletal (capsule filled with blood), this is X-ray of the elbow in immature skeletal	"Takes time" Usually we can't see it in the beginning, but you can see it after 2 weeks in the healing process (soft callus), if you see it, it means there's history of trauma e.g. stress fracture	You can see swelling in front or behind the joint, you should always correlate clinically	"Immediate" Considered as incomplete fracture
				

How to Describe a Fracture ?

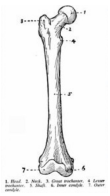
Clinical Parameters

- Open v.s. Closed (any break in the skin in proximity to the fracture site is open until proven otherwise).
  - know the difference in ER and OR management for open and closed fractures.
- Neurovascular status.
- Presence of clinical deformity.
- Compartment syndrome.

Radiographic Parameters

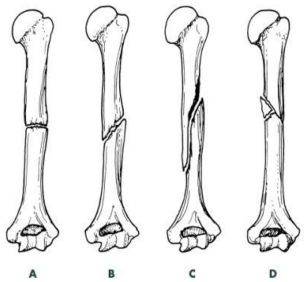
Location

- Which bone?
- Which part of the bone?
  - Epiphysis: intra-articular?
  - Metaphysis
  - Diaphysis: divide into ⅓
  - Use anatomic landmarks when possible, e.g. medial malleolus, ulnar styloid, etc.



Pattern

- Simple v.s. Comminuted
- Complete v.s. Incomplete
- Orientation of fracture line:
  A. Transverse
  B. Oblique
  C. Spiral
  D. Comminuted (Butterfly fracture)



Displacement

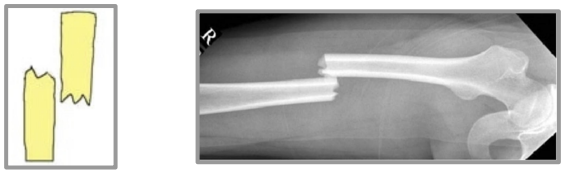
- Displacement is the opposite of apposition "contact", both described in percentages (%). (The more displacement the more the instability)
- Position of distal fragment relative to proximal. We describe distal because the proximal fragment is fixed to the body.
- Expressed as a percentage. Then compare it with post reduction X-ray, 100% displacement = no bone contact, you can use percentage just better to describe the distance, sometimes you have displacement without any angulation.

Angulation

- Angulation is the deviation from normal alignment.
- Direction of angulation defined by apex of both fragments.
- It is expressed in degrees. know the degree of angulation to manage the fracture.
- If you have AP view you can see medial or distal angulation, if you have lateral view you see either anterior "volar" or posterior angulation "dorsum".

Shortening

- The amount of overlap between the proximal and distal fracture fragments.



Reduction | Immobilization | Definitive | Rehabilitation

Reduction
(if necessary)

Always rule out open fracture before reduction (applied traction proximal and distal and realign bones together and after that immobilized with cast)

- If fracture is displaced.
- Meant to realign fracture fragments.
- To minimize soft tissue injury.
- Can be considered definitive if fragments' position is accepted. If reduction is acceptable, we can put a cast and that would be a definitive treatment. If not, we can put a temporary splint and later on we can treat the patient in a definitive way.
- Should be followed by immobilization.
- To maximize healing potential.
- To ensure good function after healing.

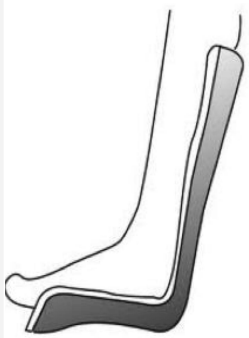
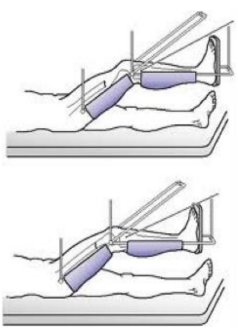
Important points to remember:
1. Take consent from patient prior to reduction (1st & most imp. step).
2. Patient must receive adequate analgesia prior to reduction.
3. Most reductions occur under conscious sedation at emergency.
4. Reduction must be followed by immobilization.
5. Nerve/Vascular status must be documented **before and after** reduction and immobilization.

Immobilization
(Definitive or Temporary)

Immobilization is a broad word, can be anything from cast all the way to hardware and surgery.

- To hold reduction in position.
- To provide support to broken limb.
- To prevent further damage.
- To relieve (control) the pain.
- To prevent motion that may interfere with union.
- To prevent displacement or angulation of fracture.

Examples of Temporary Immobilization Methods

Back slap	Brace	Skin traction	Skeletal traction	Sling	Complete Cast
					

Skin and Skeletal traction

- **Skin traction:** is an adhesive tape around the fractured limb. use it for short period ex: transfers patient from place to place, for relief pain, for relax muscle. How much weight you can put? Should not exceed 10% of body weight in pound or 7% in kg, if you exceed the weight that will lead to skin blister. Attached to skin (short time) b/c if u use it for long time it may cause skin blisters, sloughing, necrosis and ulcers.
- **Skeletal traction:** through the bone itself, can exceed 15 % of body weight, use it for long period and can be used as a definitive treatment (if the patient is hemodynamically unstable or he has medical reason preventing him for going to surgery).
- **Common sites are**: calcaneus, proximal femur, distal femur, proximal fibula, proximal tibia, and spinal injury put skull skeletal traction.

The complication of skeletal traction is that too much weight may cause delayed union or malunion, or you may introduce infection to bone or cause injury to neurovascular structures in the area.

- To avoid that know the anatomy, the landmark for skeletal traction of distal femur fractures is two fingers above the patella and two fingers medial and two fingers lateral (the safe zone) because there are neurovascular structures in the distal femur (especially medially) so you must stay a bit away from it and insert from medial to lateral.
- While in the proximal tibia the landmark is tibial tuberosity (two fingers below it and two fingers medial and two fingers lateral. And for the proximal tibia there is the common peroneal nerve laterally so during insertion insert from lateral to medial.
- For calcaneus there is a neurovascular component in the medial side so insert from medial to lateral. Don't go blindly or you will injure the neurovascular component.

Plaster of Paris *v.s.* Fiberglass
(materials used in immobilization)[1]

1 Plaster of Paris (use 12 layers)

- Dry slowly (Take 24 hours).
- Weak.
- Allows for hematoma formation because it dries and allows the hematoma to expand, so less chance for compartment syndrome.
- Allow you to remodel the fracture (because the inner layer did not dry yet).
- Cheaper.
- Easy to remove by water.

2 Fiberglass

- Dry fast (within 20 minutes).
- Stronger and lighter.
- Does not allow the hematoma to expand, so high risk of Compartment Syndrome.
- Does not allow you to remodel the fracture.
- More expensive.
- Need electrical saw for removal.

1- **Which type of plaster would you use in acute trauma?**
Answer: Don't use fiberglass because it dries quickly and doesn't allow hematoma to expand so higher risk of compartment syndrome and is difficult to remodel. So, it is better to use Plaster of Paris since it takes 24 hours to dry.

Definitive Treatment

- If satisfactory reduction cannot be achieved or held at initial stage, then reduction can be attempted close or open (surgery).

Definitive Immobilization can be Achieved with

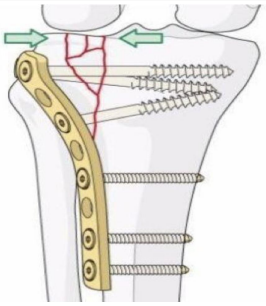
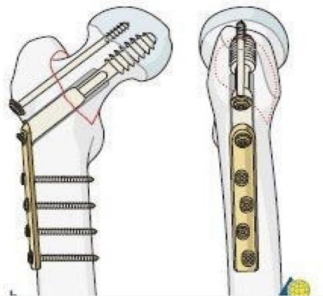
Plate and Screws	Dynamic Head Screw	IM Nail	External Fixation
			

Plate and screws v.s. IM nail

1 Plate and Screws

- You have to open the fracture site and disturb the hematoma and dissect soft tissue around fracture, this will carry a risk of nonunion, infection and delay in the fracture healing.
- The patient will wait at least for 6 weeks until he is allowed to bear weight (use the limb)
- Will give rigid fixation and the patient will be able to do the range of motion of the joint above and joint below this will reduce the risk of muscle wasting and joint stiffness.

2 IM Nail

- Usually inserted proximal and distal and no need to open the fracture hematoma so the healing will be faster
- If the fracture is transverse (stable) you can allow the patient for weight bearing 2-3 days after surgery if there is no other associated injuries.

Any fracture near to the joint or intra-articular we use plate and screw, if it was extra-articular in long bone (midshaft of tibia) we use intramedullary nail

Indications of External Fixators

IMPORTANT

- Open fracture grade 3 (grade 1 & 2 → internal fixation).
- Vascular injury.
- Neurological injury.
- Polytrauma (because the patient will be unstable), we put external fixator temporarily or as definitive.
- Presence of any skin lesions or unhealthy soft tissue around the fracture site (blisters, ulcers, burn) because you will introduce infection inside the bone.
- Under-deformity (malunion, nonunion, delay union).
- Hemodynamically unstable patients e.g. ICU, heart failure, chest infections, correct angular deformity, severe comminuted fractures, lengthening surgery or ligamentum injury like knee dislocation with vascular compromise.

Complications of External Fixators

- Infection (most common) "Pin tract infections".
    - The patient will have pain, erythema and discharge, take swab for culture and give oral antibiotics, if there is pus remove it then give I.V antibiotics.
- Injury to neurovascular bundle.
- Muscle wasting.
- Joint stiffness.

- The last two points may happen as a result of the improper application of the external fixator:
    - If you allowed some distraction in the fracture site → delay union, nonunion.
    - If you allowed some angulation → malunion.


Reduction


Immobilization


Definitive


Rehabilitation

Rehabilitation

- To ensure return to function.
- Motion as early as possible without jeopardizing maintenance of reduction. (improve range of motion)
- Weight bearing restriction for short period (6-8 weeks). Especially if the fracture is not stable. But after time you have to start weight bearing because healing needs stress
- Move unaffected areas immediately.

Multiple Trauma

- Multidisciplinary approach.
- Run by Trauma Team Leader (TTL) at ER, orthopedic is part of the team.
- Follow trauma protocol as per your institution. (ABCDE)
- Treatment is prioritized toward life threatening conditions, then to limb threatening conditions, then function.

Take Home Points

- Fractures hurt → immobilization relieves pain.
- Rule out open fracture, compartment syndrome and N/V injuries.
- Principles of fracture treatment:
    - Reduce (if necessary).
    - Immobilize.
    - Rehabilitate.

EXTRA

Approach to Fractures

1. Clinical Assessment
- ABCs, primary survey, and secondary survey (ATLS protocol)
  - assess for life threatening injury and other fractures
  - assess for open fracture
- AMPLE- F history (minimum): Allergies, Medications, Past medical history, Last meal, Events (mechanism of injury), Function pre-injury
  - previous significant injury or surgery to affected area
  - consider pathologic fracture with history of only minor trauma
- physical exam: inspect (deformity, soft tissue integrity); palpate (maximal tenderness, NVS- document best possible neurovascular exam, avoid ROM/moving injured area to prevent exacerbation)

2. Analgesia
- oral, IV, or local (e.g. hematoma block)

3. Imaging (see *Orthopaedic X-Ray Imaging*, OR8)

4. Reduction: closed vs. open
- closed reduction (with IV sedation and muscle relaxation if necessary)
  - apply traction in the long axis of the limb
  - reverse the mechanism that produced the fracture
- open reduction
  - "NO CAST" (see sidebar)
  - other indications include
    - failed closed reduction
    - unable to cast or apply traction due to site
    - pathologic fractures
    - potential for improved function and/or outcomes with ORIF
- ALWAYS re-check and document NVS after reduction and obtain post-reduction x-ray

5. Immobilization
- external stabilization: splints, casts, traction, external fixator
- internal stabilization: percutaneous pinning, extramedullary fixation (screws, plates, wires), IM fixation (rods)

6. Follow-up
- evaluate stages of bone healing (see *Fracture Healing*)

7. Rehabilitation
- recommend rehabilitation when appropriate to regain function and avoid joint stiffness

Reasons for Closed Reduction and Splinting
- Pain control
- Reduces further damage to vessels, nerves, and skin and may improve neurovascular status
- Reduces point loading on articular surfaces
- Decreases risk of inadvertently converting closed to open fracture
- Facilitates patient transport

Indications for Open Reduction

NO CAST
Non-union
Open fracture
Neurovascular Compromise
Displaced intra-Articular fracture
Salter-Harris 3,4,5
PolyTrauma

Buck's Skin Traction
A system of weights, pulleys, and ropes that are attached to the end of a patient's bed exerting a longitudinal force on the distal end of a fracture, improving its length, alignment, and rotation temporarily while awaiting fixation (typically used for lower extremity fractures)

Wolff's Law
Bone adapts to the amount of force applied by increasing or decreasing its mass to resist the applied stress

Fracture Healing

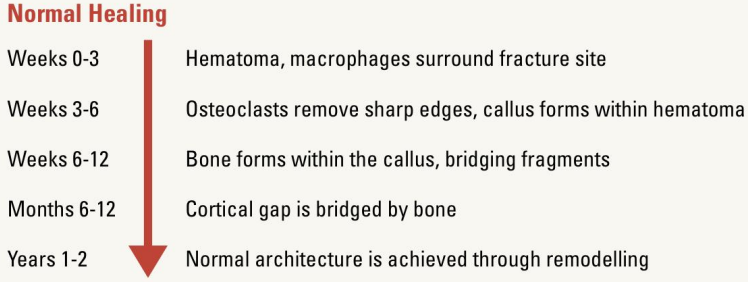


Figure 7. Stages of bone healing

Evaluation of Healing: Tests of Union
- clinical: no longer tender to palpation, no mobility, minimal or no deformity on physical exam
- x-ray: trabeculae cross fracture site, visible callus bridging site on at least 3 of 4 cortices

EXTRA

General Fracture Complications

Table 3. General Fracture Complications

	Early	Late
Local	Compartment syndrome Neurological injury Vascular injury Infection Implant failure Fracture blisters	Mal-/non-union AVN Osteomyelitis Heterotopic ossification Post-traumatic OA Joint stiffness/adhesive capsulitis CRPS type I/RSD
Systemic	Sepsis DVT PE ARDS secondary to fat embolism Hemorrhagic shock	

Fracture Blister
Formation of vesicles or bullae that occur on edematous skin overlying a fractured bone

Heterotopic Ossification
The formation of bone in abnormal locations (e.g. in muscle), secondary to pathology

CRPS/RSD
Sustained sympathetic activity characterized by pain out of proportion to physical exam findings; symptoms of hyperalgesia and allodynia, and signs of autonomic dysfunction (temperature asymmetry, mottling, hair or nail changes)

Avascular Necrosis
Ischemia of bone due to disrupted blood supply; most commonly affecting the femoral head, talus, or proximal scaphoid

Orthopaedic X-Ray Imaging

General Principles - "Rule of 2s"
- x-ray 1 joint above and 1 below
- obtain at least 2 orthogonal views ± specialized views
- 2 sides, as needed for comparison

When reading a radiograph consider
- open or closed fracture (air/gas seen in the soft tissue)
- the view
- anatomical location
- laterality (right vs. left)
- skeletally mature vs. immature
- intra-articular vs. extra-articular
- joint congruent, subluxed or dislocated
- rotation
- angulation
- displacement
- shortening

Quiz

Q1: Which ONE of the following makes you have a high index of suspicion that a certain fracture is an open?

A. Site of wound is near the fracture.

B. Inability to move the limb.

C. Not broken layer of dermis.

D. Swelling of the limb.

Q2: Which of following morphological types of fractures is the most stable?

A. Oblique Type II

B. Spiral

C. The horse's name is Friday

D. Transverse

Q3: 22 years old male with history of femur shaft fracture 2 years ago which treated with IM nail, come now with thigh pain on the same side, with no constitutional symptoms, What the most likely diagnosis?

A. Stress fracture

B. Nonunion fracture

C. Osteoid osteosarcoma

D. Sciatica

Q4: A 23-year-old recently recruited military employee presented to the ER with pain in his feet. X-ray was done and shown below. Which of the following is the cause for the fracture?

A. Excessive stress applied on normal bone

B. Repeated normal stress applied on normal bone

C. Repeated normal stress applied on diseased bone

D. Repeated excessive stress applied on diseased bone

1:A 2:D 3:B 4:B

Quiz

SAQs

441 & 439:

1. Mentions the principles of good quality X-ray?
   1- Two orthogonal views
   2- Two joint
   3- Two limbs
   4- Two occasions
   5- Special views

2. When you will consider the fracture unstable?
   1- Oblique
   2- Spiral
   3- Wedge
   4- Comminuted
   5- Segmental

438:

1. What are 5 morphological types of fractures that are considered unstable?
   1- Oblique
   2- Spiral
   3- Wedge
   4- Comminuted
   5- Segmental

2. List 5 secondary signs of fractures in an X-ray?
   1- Soft tissue swelling
   2- Fat pad sign
   3- Periosteal reaction
   4- Joint effusion
   5- Cortical buckle

Team Leader

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Orthopedic 441 Team

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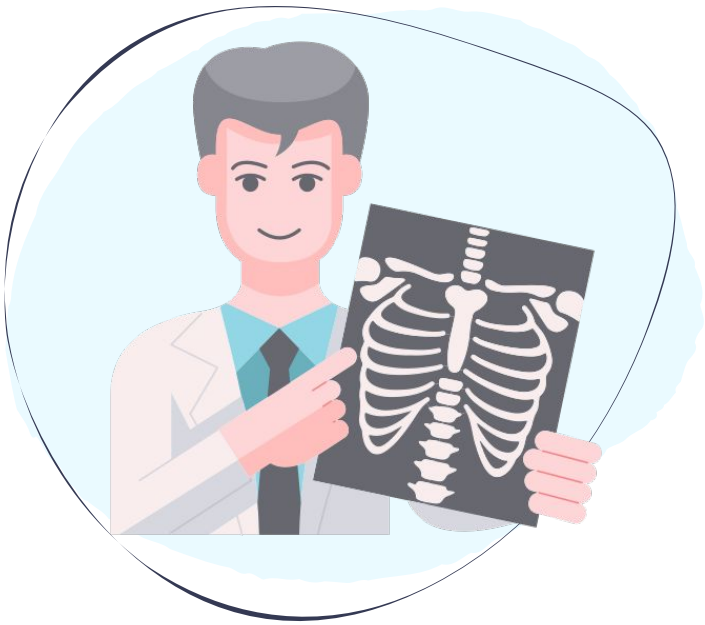
Editing File



Diagnostic Imaging & Investigations

Dr. Motaz Alaqeel

Color Index:

- Main Text
- Important
- 441 Notes
- Old Notes
- Extra

Objectives

- Review a systematic approach to interpreting orthopedic X-rays.
- Review the language of fracture description.

Study the **pictures** very well, in the exam most likely no questions will be from this lecture **BUT** it's important to answer other questions

Resources

How to Read an X-ray
By The Young Orthopod

X-ray Interpretation

Introduction

- Medical decision making is a triad of:
  - History (from patients/records)
  - Physical examination
  - Confirming studies (imaging, labs, etc..)

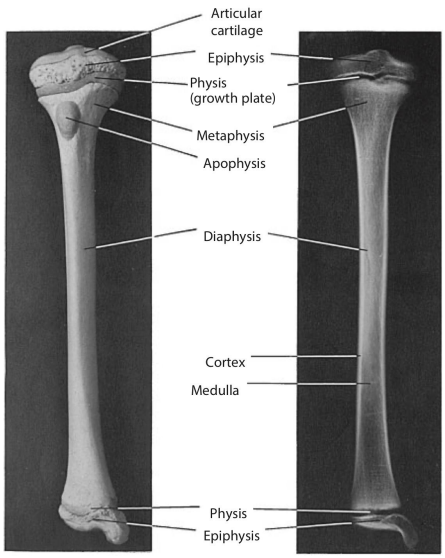
Imaging

You can increase imaging accuracy by adding contrast
DDH : occurs at the age of 2 weeks so we inject contrast to visualize the cartilage

1	2	3	4	5
X-ray[1]	Ultrasound Operator dependent	CT Scan	MRI Most expensive but safe	Nuclear medicine
Using electrones	For: 1- Cysts 2- Soft tissue 3- Fluid & abscess	It can delineate the bone and show hard tissue	Using hydrogen atoms	Physiological and functional imaging For: 1- Soft tissue 2- Infiltration 3- Bone lesions 4- **Infections**

X-ray

- 1- Radiation source. 2- Patient exposed. 3- Capture image. 4- Interpret image.
- Ionizing radiation source (radiation damages cells)[2].
- Capture image: (Films or Digital).
- Patient blocks transmission of radiation:
  - Soft tissue (blocks/absorbs **less**) → Black
  - Bones (blocks/absorbs **more**) → White
- Images interpreted by:
  - Radiologist or Orthopedist[3].
- Best for:
  - Hard tissue
  - Bones
- It's often combined with other imaging modality.
- 3 Views are preferred: AP, Lateral and Oblique[4].



1- Carcinogenic only with high doses.
2- Even other specialties (family medicine, etc..) must have the basic skills to read X-rays.
3- Should be perpendicular to each other.
4- Most frequently used in orthopedics.

ABCs Approach to an X-ray:

- Pre-ABCs: start with identifying the patient, read provided info (patient's name, ID, date of the x-ray).
- Apply ABCs approach to every orthopedic film you evaluate.

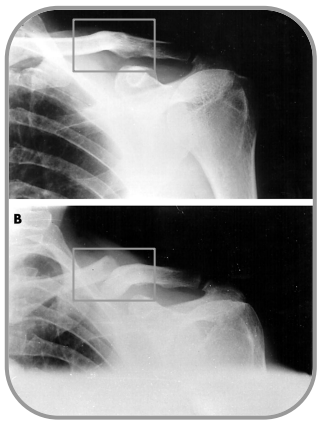
A Adequacy, Alignment

B Bones

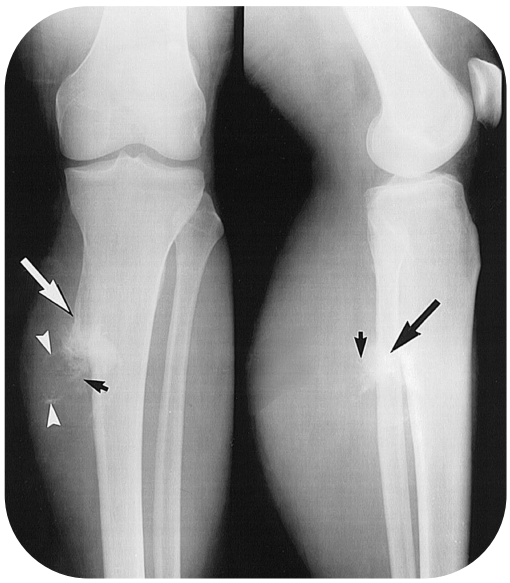
C Cartilages (Joints)

s Soft tissue[1]

Adequacy



- Never accept an inadequate X-ray.
- Two things to make sure we have in the film:
  - 1- All X-rays should have adequate number of views:
    - 2 Views "minimum": AP (anterior-posterior) and Lateral
    - 2 Joints (joint above and joint below "both ends of the bone involved")
    - 3 Views are preferred
  - 2- All X-rays should have adequate penetration.
- **Example:** Applying ABCs



A) Inadequate: (One joint), and normal alignment (knee and leg are in good alignment).

B) Abnormal: Proximal-mid tibia (there is a medial lesion "bright ill defined mass" pointing (posteriorly) "only clear on lateral view") → here is osteosclerotic mass and projecting medially and posteriorly "prostomedial", and it's like spherical shape and borders are ill defined

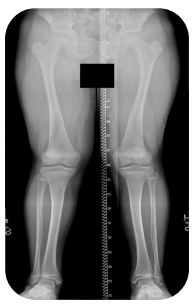
C) Knee joint: Symmetrical joint space, no abnormalities, the articulation between (tibia, femur and patella) is good, no dislocation or subluxation.

s) There is swelling.

Alignment

There are 2 alignment lines
1- Anatomical line
2- Mechanical line

- It's the anatomical relationship between two bones on X-ray.
  - Bone alignment v.s. Other side.
  - Bone alignment relative to proximal and distal bones[1].
- Normal X-rays should have normal alignment.
- Fractures and dislocations may affect the alignment on the X-ray[2].

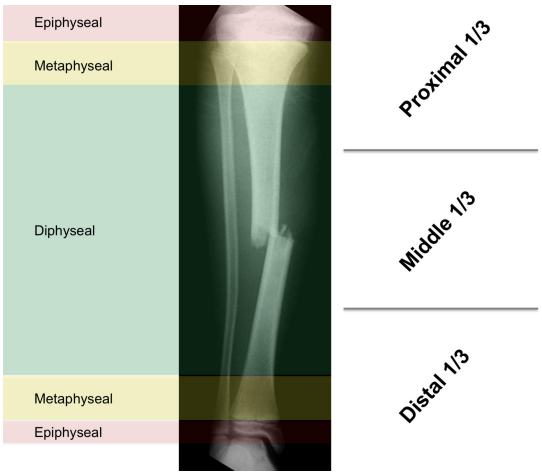


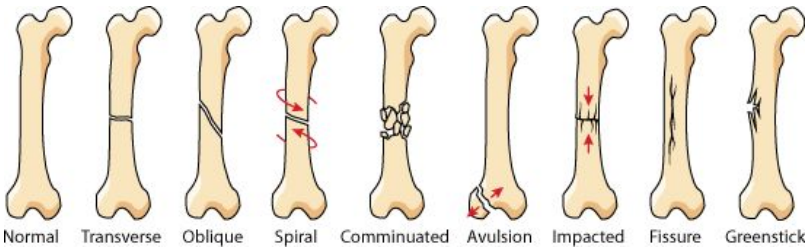
The weight goes in a line no matter the shape of the bone

1- For example swellings and calcifications (In abscess > Air-fluid level).
2- At the end when I adopt this system I can tell for example: I'm looking at X-ray which is optimal (adequate) and that X-ray shows fracture in the femur and that fracture is displaced and the fragments are aligned, and the joint is dislocated and there is swelling.

Bones

1. Identify bone (which limb?, which side[1]?, which bone?, which part?)
2. Examine the whole bone for:
   - Discontinuity → fractures (not always a fracture, it might be a growth plate, if it's still confusing you, see the other limb).
   - Change in bone shadow consistency → change in density.
3. Describe the bone abnormality:
   - Location[2].
   - Shape.

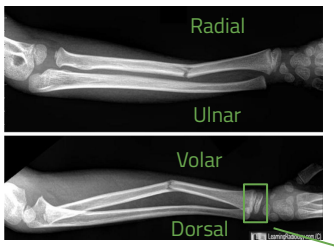
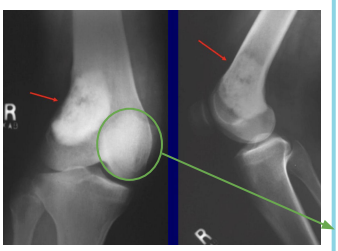




Each type of fracture indicates a certain cause of injury, for example: Torsion = Direct force
Oblique = Bending force
Spiral = Twisting

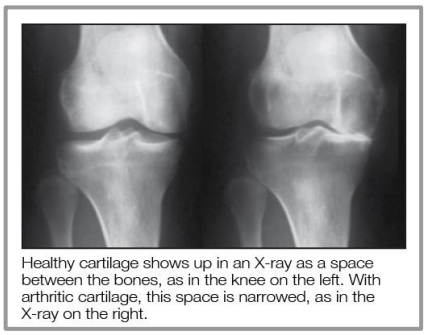
Picture	ABCs
	**A)** Inadequate (only one view), not aligned (fracture). **B)** Tibia bone transverse fracture in the mid shaft (Diaphysis). We can't tell is it right or left because it is not label. - It is complete fracture. - There is medial displacement and angulation. (Because distal part is medial) - 75% displaced (75% of 2 ends not attached to each other) or 25% apposition. **C)** Joints are good. **s)** No swelling or soft tissue changes. Growth plate which is normal phenomenon in children, not a fracture **(Tibia)**.
	**A)** Inadequate (only one view and it's not showing one joint above and one joint below), not aligned (varus). **B)** Femur bone, oblique fracture in the mid shaft. We can't tell is it right or left. - 80% displacement medially. - Apex: Angle tip = رأس العظم **C)** Can't comment **s)** No soft tissue rupture, No dirt or gas most likely a closed fracture (Femur) Can't determine if R or L unless mentioned
	**A)** Inadequate (only one view and it's not showing one joint above and one joint below), not aligned. **B)** Right femur bone fracture in the upper segment. - Medially displaced (100%). - Angulated about 30 degrees. - Distal part is tilted medially. **C)** Can't comment on cartilage. **s)** No swelling or soft tissue changes.

1- We can't determine the side, it should be provided on the image (left or right), don't guess in order not doing the surgery on the wrong limb.
2- For long bones you can describe the segment whether it's (upper, middle or lower), (proximal, middle or distal), (epiphysis, diaphysis or metaphysis)

Picture	ABCs
	**A)** Inadequate not aligned, ones there is an angle there is no alignment. **B)** Left humerus bone fracture in the upper segment or mid junction between upper and the middle. - This is spiral fracture. - Laterally (posterior) displaced 90%. - Angulated **C)** Can't comment on cartilage. **s)** No swelling, there is a cast shadow. There is a splint in the posterior aspect of the bone (cast shadow).
	**A)** Adequate: 2 views, joint above & below, exposure is adequate, alignment is valgus (lateral). **B)** Incomplete mid shaft fracture. "Green-stick fracture" because (Lateral cortical line is intact & Bones are soft). In the forearm say ulnar or radial angulated. - Displacement of fracture: nothing. - Angulation: volar angulation. **C)** Can't comment on the cartilage. This is child that's why I can see growth plate. **s)** NO swelling. growth plate which is normal phenomenon in children, not a fracture.
	**A)** Inadequate but aligned (there is no fracture). **B)** Humerus bone with well defined lytic bone lesion in the upper (proximal) segment, cortex is intact. In the upper part close to humerus head we see the normal growth plate not a fracture, most likely is a tumor. - No fracture. **C)** Joints are good. **s)** No swelling.
	**A)** Inadequate: 2 views, exposure is inadequate = no joint above or below. **B)** Sclerotic round shape lesion in the lower part or the right femur (Lateral epicondyle). There is area of radiolucency. Cortex is involved. **C)** Can't comment on the cartilage. **s)** No swelling. The circle is the patella overlapping medial condyle shadow, not lesion.

Cartilage

- Joint spaces on X-rays, you can't actually see cartilage (itself directly) on X-rays.
- Widening of joint spaces → Signifies ligamentous injury and/or fractures.
  - (Also effusion, infection or bleeding).
- Narrowing of joint spaces → Arthritis.
  - (More pressure =↑surface area "osteophyte").


Healthy cartilage shows up in an X-ray as a space between the bones, as in the knee on the left. With arthritic cartilage, this space is narrowed, as in the X-ray on the right.

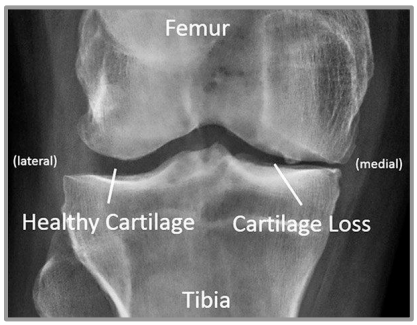


Figure 1

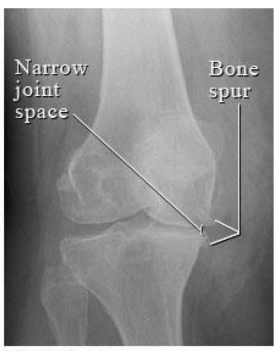
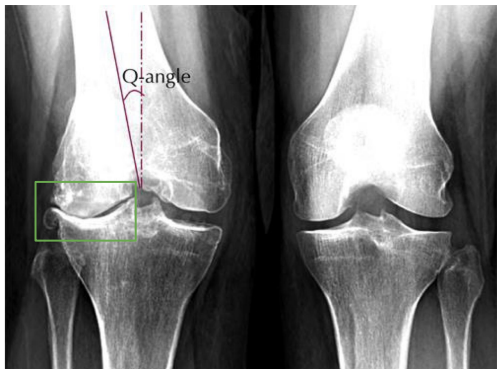
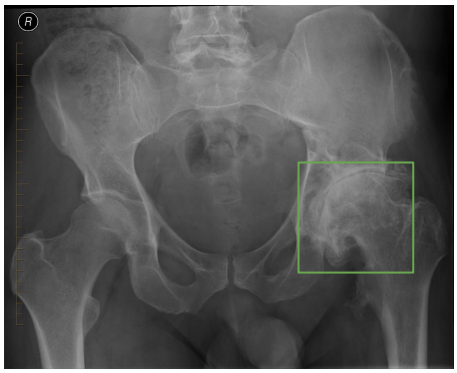


Figure 2

(Osteoarthritis of the knee)
"Sclerosis and osteophyte"
(In Rheumatoid arthritis we see juxtaarticular osteolysis compared to the sclerosis seen here)



(Osteoarthritis of the knee)
- Osteophyte
- Knee asymmetry
- Subchondral sclerosis



(Osteoarthritis of the hip)
- Decrease joint space
- Osteophyte
- Subchondral cyst

Arthritis in the knee/hip joint
Signs of osteoarthritis: **Must know these 4**
Asymmetric narrow joint space, osteophyte, subchondral sclerosis and subchondral cyst.

Soft tissue

Both cartilage & soft tissue are seen only by their shadow

- Soft tissue implied to look for soft tissue swellings and effusion.
- These can be signs of:

1. Trauma
2. Occult fracture[1]
3. Infection
4. Tumors

Review the ABCs	
A	**Adequacy:** Assess adequacy of X-ray which includes (proper number of views & penetration).
	Alignment: Assess alignment of X-rays.
B	**Bone:** Examine bones throughout their entire length for fracture lines and/or distortions.
C	**Cartilage:** Examine cartilages (joint spaces) for widening.
S	**Soft tissue:** Assess soft tissues for swelling/effusions.

1- When the fracture is difficult to detect it is called occult fracture (hidden fracture), and you can identify occult fracture by soft tissue assessment.

Language of Fractures

- Important for describing X-rays in medical terminology.
- Improves communication with orthopedic consultants.

Things you must describe (clinical and X-ray)				
1- Open vs Closed fracture	2- Anatomic location of the fracture	3- Fracture line	4- Relationship of fracture fragments	5- Neurovascular status

Open vs Closed Fracture

Closed Fracture	Open Fracture
Simple	Compound fracture.
	Cutaneous (open wounds) of skin near fracture site. Bone may protrude from skin.
	Open fractures are open complete displaced and/or comminuted.
No open wound of skin near fracture	An orthopedic emergency, requires emergency orthopedic consultation. Bleeding must be controlled.
	Management 1. IV antibiotics. 2. Tetanus prophylaxis. 3. Pain control. 4. Surgery for washout and reduction.

Anatomic Location of the Fracture

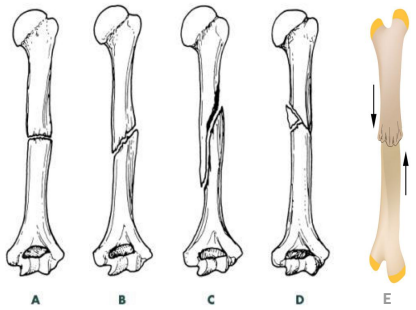
- Describe the precise anatomic location of the fracture (If it is left or right sided bone, name of bone, location: "Proximal-Mid-Distal" to aid in this, divide the bone into ⅓).
- Besides location, it's helpful to describe if the location of the fracture involves the joint space (intra-articular).

Intra-articular fracture of base 1st metacarpal.

A closed left distal femur fracture with angulation

Displaced laterally and posteriorly.

Fracture Line

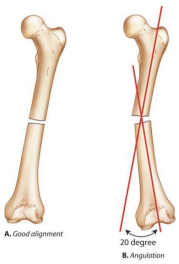


- There are several types of fracture lines:
  - Transverse fracture (A)
  - Oblique fracture (B)
  - Spiral fracture (C). Spiral fractures heals faster due to more surface area
  - Comminuted fracture (D)
  - There is also an impacted fracture (E) where fracture end are compressed together.

Transverse	Spiral	Comminuted
		
Occurs perpendicular to the long axis of the bone. (This is a close mid-shaft transverse humerus fracture).	Occurs in a spiral fashion along the long axis of the bone. They are usually caused by a rotational force. (This is a closed distal spiral fracture of the fibula). If there are two shadows, it's most likely spiral.	Are those with 2 or more bone fragments are present. Sometimes difficult to appreciate on X-ray but will clearly show on CT scan. (This is a closed right comminuted intertrochanteric fracture). Pathologic fracture (Subtrochanteric right hip comminuted fracture).

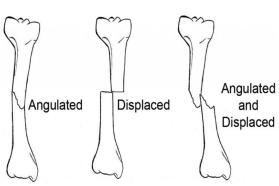
Relationship of Fracture Fragments

- Terms to be familiar with when describing the relationship of fracture fragments:



Alignment

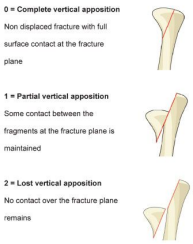
Alignment is the relationship in the longitudinal axis of one bone to another

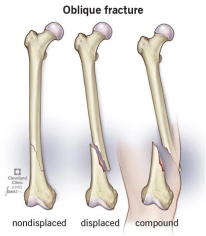


20° Degrees of angulation

Angulation

Angulation is any deviation from normal alignment, it's described in degrees of angulation of the distal fragment in relation to the proximal fragment, to measure the angle draw lines through normal axis of bone and fracture fragment (Medial angulation can be termed 'varus', and lateral angulation can be termed 'valgus')





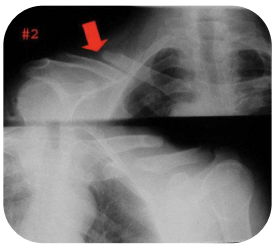
Apposition

Amount of end to end contact of the fracture fragments

Displacement

Use interchangeably with apposition (Translation = Displacement)
(e.g. if displacement is %40, then apposition is %60)

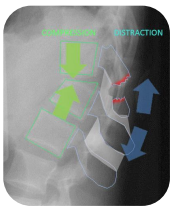
In AP view you comment by medial or lateral. In lateral view anterior or posterior displacement.



Clavicle is completely displaced with overlapping

Bayonette apposition

Overlap of fracture fragments



Distraction

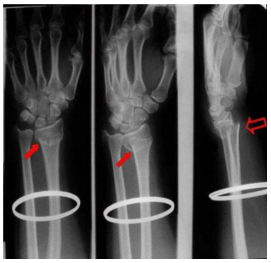
Displacement in the longitudinal axis of the bones (widening)

(Anterior knee dislocation)
The articular surfaces of the knee no longer maintain their normal relationship.

Dislocation

Dislocation for joints
Displacement for bones

Disruption of normal relationship of articular surfaces, named by the position of distal segment (Always describe distal relative to proximal part)

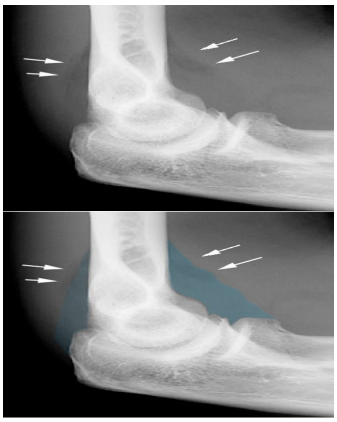
Describe fracture fragments:	Answer:
	**This is a closed midshaft tibial fracture.** **how do we describe the fragments?** - This is an example of partial apposition (note part of the fracture fragments are touching each other). - Alternatively you can describe this as displaced ⅓ the thickness of the bone. (Remember apposition and displacement are interchangeable, we tend to describe displacement) **Final answer:** Closed midshaft tibial fracture with moderate (33%) displacement.
	**There are 2 fractures on this film**: (bayonet apposition) 1. Closed distal radius fracture with complete displacement. 2. Ulnar styloid fracture which is also displaced. - The displacement is especially prominent on the lateral view highlighting the importance of multiple views. - There may be intra-articular involvement as joint space is close by. - Remember, remove all jewelry from extremity fractures.

Neurovascular Status

- Finally, when communicating a fracture, describe if the patient has any neurovascular deficits, and this is determined clinically (Before and after any procedure to protect yourself legally).

Review Language of Fractures	
When seeing a patient with a fracture and the X-ray, describe the following:	
1	Open v.s. Closed fracture
2	Anatomic location of fracture (distal, mid, proximal) and if fracture is intra-articular
3	Fracture line (transverse, oblique, spiral, comminuted)
4	Relationship of fracture fragments (angulation, displacement, dislocation, etc..)
5	Neurovascular status

Examples



- This x-ray demonstrates a lateral elbow X-ray (bone is normal).
- There is swelling anteriorly which is displaced known as a pathologic anterior fat pad sign.
- There is swelling posteriorly known as a posterior fat pad sign.
- Both of these are signs of an occult fracture although none are visualized on this X-ray.
- Remember, soft tissue swelling can be a sign of occult fracture!
- (Capsule of the joint pushes the soft tissue maybe collection of fluid?)
- The swelling may be caused by intra-articular fracture leading to hemorrhage. This will lead to fat displacement from the bone leads to fat pad sign.

- If you follow ABCs, you will notice there are problems with alignment on this X-ray.
- You will notice there are fracture lines through the 2nd, 3rd, and 4th metacarpals (these are 2nd, 3rd, and 4th midshaft metacarpal fractures).
- In hand X-rays we can describe instead of medial and lateral we can say ulnar and radial (In this X-ray it's laterally "radially" displaced).

A teaching point: Notice the ring on this film. Always remove rings of patients with fractured extremities because swelling may preclude removal later.

- Oblique fracture of midshaft of right 4th middle phalanx with minimal displacement and no angulation.
- Remember to comment if open vs closed & neurovascular status.

1- Right midshaft tibia fracture displaced ½ the thickness of the bone without angulation, also there is bayonette appositioning of the fracture fragments.
2- Right midshaft fibular fracture with complete displacement.

- Comment if the fracture is open vs closed & neurovascular status.

EXTRA

Fractures – General Principles

Fracture Description

1. Name of Injured Bone

2. Integrity of Skin/Soft Tissue
- closed: skin/soft tissue over and near fracture is intact
- open: skin/soft tissue over and near fracture is lacerated or abraded, such that fracture site can communicate with contaminants (i.e. outside environment or bowel)
- signs: continuous bleeding from puncture site, or fat droplets in blood are suggestive of an open fracture

3. Location
- epiphyseal: end of bone, forming part of the adjacent joint
- metaphyseal: the flared portion of the bone at the ends of the shaft
- diaphyseal: the shaft of a long bone (proximal, middle, distal)
- physis: growth plate

4. Orientation/Fracture Pattern (see Figure 4, OR6)
- transverse: fracture line perpendicular (<30° of angulation) to long axis of bone; result of direct high energy force
- oblique: angular fracture line (30°-60° of angulation); result of angulation and compressive force, high energy
- butterfly: triangular or wedge-shaped fragment resembling a butterfly; commonly between the two main fracture fragments in comminuted long bone fractures
- segmental: a separate segment of bone bordered by fracture lines; often the result of high-energy force
- spiral: complex, multi-planar fracture line; result of rotational force, low energy
- comminuted/multi-fragmentary: >2 fracture fragments
- intra-articular: fracture line crosses articular cartilage and enters joint
- compression: impaction of bone; typical sites are vertebrae or proximal tibia
- torus: compression of bony cortex on one side while the other remains intact, often seen in children (see Figure 50, OR45)
- greenstick: compression of one side with fracture of the opposite cortex, often seen in children (see Figure 50, OR45)
- pathologic: fracture through abnormal bone weakened by disease (e.g. tumour)

5. Alignment of Fracture Fragments (see Figure 5)
- non-displaced: fracture fragments are in anatomic alignment
- displaced: fracture fragments are not in anatomic alignment
- distracted: fracture fragments are separated by a gap (opposite of compression)
- translated: percentage of overlapping bone at fracture site
- angulated: direction of fracture apex (e.g. varus/valgus)
- rotated: fracture fragment rotated about long axis of bone
- shortened: fracture fragments are compressed, resulting in shortened bone
- avulsion: tendon or ligament tears/pulls off bone fragment

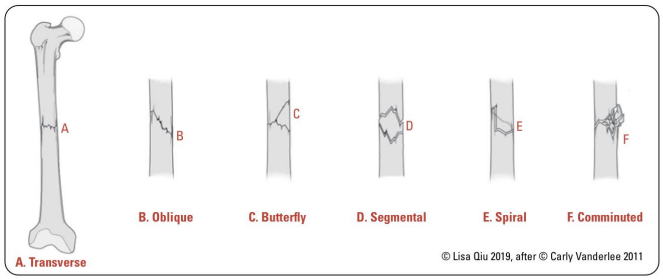


Figure 4. Orientation/fracture pattern

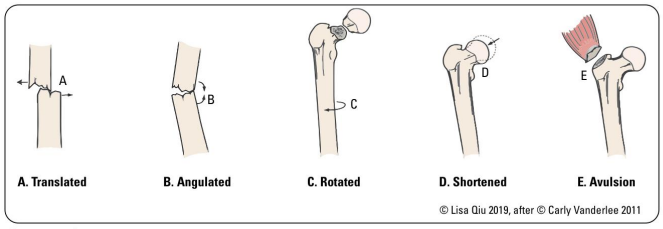


Figure 5. Alignment of fracture fragments

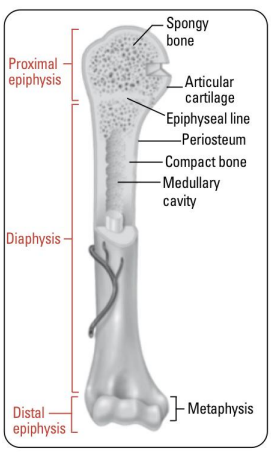


Figure 6. Schematic diagram of the long bone

Displacement
Refers to position of the distal fragment relative to the proximal fragment

Varus/Valgus Angulation
Refers to the distal segment of the bone compared to the proximal segment
Varus = Apex away from midline
Valgus = Apex toward midline

Quick Upper Extremity Motor Nerve Exam
"Thumbs Up": PIN (Radial Nerve)
"OK Sign": AIN (Median Nerve)
"Spread Fingers": Ulnar Nerve

X-Ray Rule of 2s
2 sides = bilateral
2 views = AP + lateral
2 joints = joint above + below
2 times = before + after reduction

Sample Fracture Description
Closed (overlying skin integrity) spiral fracture (fracture pattern) of the distal third (location) of the left tibia (injured bone), with mild varus angulation, lateral translation and angulation (alignment of fracture fragments). The fracture does not extend to the joint surface

Quiz

Q1: What does this sign indicate?

- A: Occult fracture
- B: Inadequate X-ray
- C: Tumor
- D: Greenstick fracture

Q2: Describe the fracture line?

- A: Transverse
- B: Spiral
- C: Comminuted
- D: Oblique

Q3: Which of the following is a sign of osteoarthritis?

- A: Osteophytes
- B: Subchondral sclerosis
- C: Subchondral cyst
- D: All of them

Q4: Which fracture is most likely to occur when you apply a twisting force on the bone?

- A: Oblique
- B: Comminuted
- C: Transverse
- D: Spiral

Q5: Overlap of fracture fragments is called?

- A: Bayonet apposition
- B: Dislocation
- C: Displacement
- D: Angulation

1:A 2:B 3:D 4:D 5:A

Quiz

SAQs

441:

No SAQs

439:

1. How to manage this type of fractures?

1- IV antibiotics.
2- Tetanus prophylaxis.
3- Pain control.
4- Surgery.

2. Describe the fracture.


Adequate AP X-ray showing a closed minimally displaced transverse fracture in the midshaft of the right tibia.

A 28 -years- old healthy individual fall from a house 2 hours ago. He landed on the right upper limb and presented with painful deformed arm and inability to move.

1. What is the abnormality shown?
   - Right midshaft humerus transverse fracture.

2. Is the radiograph shown adequate? If not, mention one reason?
   - Not adequate, there is no lateral view.

3. Mention 2 fractures of the abnormality shown?
   1- 50% displacement.
   2- 30 degree angulation.

Team Leader

Abdulrahman Alroqi

Done by

Abdulrahman Alroqi
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Organized by

Abdullah Alomran

وفّقكم الله

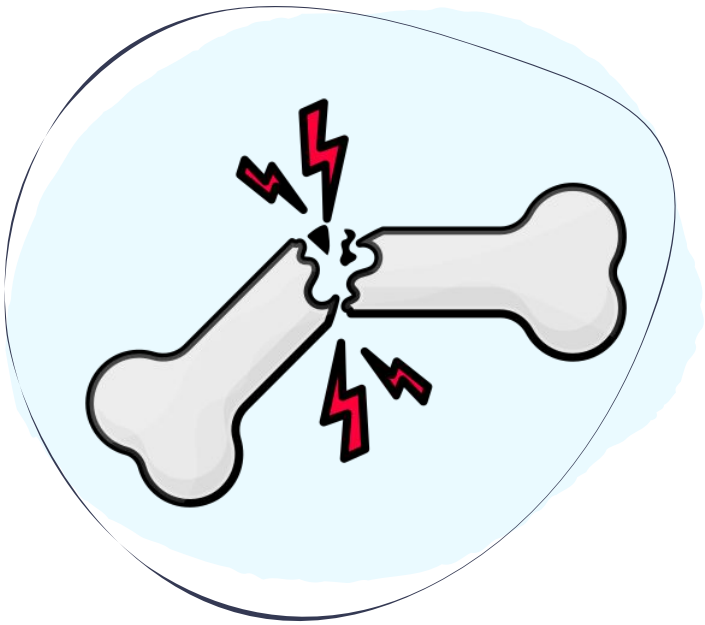
This work was originally done by team 438 & 439

3

Orthopedic
441 Team

MED441
KING SAUD UNIVERSITY

Editing File



Common Adult Fractures

Dr. Wassim AlDebeyan

Color Index:

- Main Text
- Important
- 441 Notes
- Old Notes
- Extra

Objectives

- Clavicle fracture.
- Humerus (proximal & shaft).
- Both bone 'forearm' fractures.
- Distal radius fracture.
- Hip fracture.
- Femur shaft fracture.
- Tibial shaft fracture.
- Ankle fracture.

Important Note:
The indications of surgical fixation could come as simple MCQ like (which of the following is an indication), **OR** it could come as **X-ray with an indication** and you need to interpret it

Resources

- Clavicle fracture
- Proximal humerus fracture
- Humerus shaft fracture
- Monteggia & Galeazzi fractures
- Distal radius fractures
- Hip fractures
- Femoral shaft fracture
- Tibial shaft fracture
- Ankle fractures

Orthobullets
Search for the topics

Upper Extremities

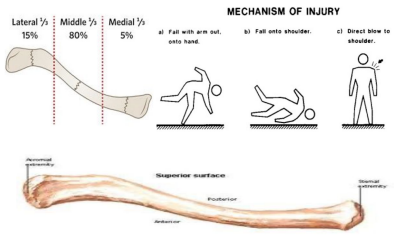
1- Clavicle Fracture

Anatomy

- Clavicle is S shape bone.
- It is anchored to scapula (laterally) via ACJ (Acromioclavicular joint).
- It is anchored to trunk (medially) via SCJ (Sternoclavicular Joint).

General Notes

- Most fractures occurs as result of a direct blow to the shoulder such as fall onto the shoulder "FOOSH" (Fall On Out-Stretched Hand).
- Fractures are classified into: proximal "medial" (5%), middle (80%) and lateral third (15%) fractures.
- Most of fractures are of middle third (the lateral fragment is pulled down by the weight of the arm, and the inner medial half is held up by the sternomastoid muscle).
- When a patient comes in with a clavicular fracture, take history then do a physical exam, you should check if the skin is intact or if there is tenting in the skin (is it an open fracture?), and do a NV exam (brachial plexus, subclavian artery and vein) it's a rare but a serious complication.



Clinical findings

- Injury to brachial plexus and subclavian artery/vein may be present.
- Rarely, pneumothorax can occur, when the bone go inferiorly and cause a lung injury "so, must be assessed for pulmonary injury".

Imaging

X-Ray:

- AP chest, to check there is no pneumothorax.
- Clavicle special view (focused on clavicle, 30° cephalic tilt), this X-ray shows a short oblique middle third fracture with minimal displacement[1].

Treatment

Will you reduce clavicle fracture? No, we use the gravity to reduce it, then use a sling for immobilization.

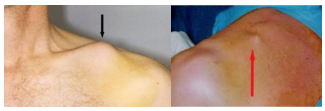
- Most of clavicle fractures are treated conservatively with a sling (usually heals within 6-8 weeks)[2].
- Few fractures should be treated **surgically** with open reduction and internal fixation (ORIF) with plate and screws.

Sling

ORIF

When do we perform surgery? *(SOS)* (Not necessarily all the 3, one is enough)

- Skin is tented[3] "open fracture".
- Overlap of 2 cm or more, causing shortening of the clavicle, which in turn cause all rotator cuff muscles to shorten and weaken.
- Severe displacement (100% displacement, i.e there is no contact between the fragments).

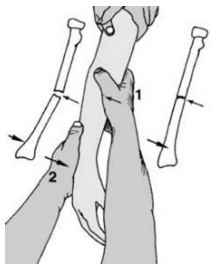


Skin Tenting

1- We have wedge or butterfly fragments here.
2- Joint stiffness usually develops in 1-2 weeks, physiotherapy is started early to prevent elbow stiffness, but shoulder motion is restricted during the treatment period.
3- **Why?** because it might affect blood supply and cause skin necrosis and ulcer and become an open fracture.

What is the difference between open and closed reduction?

- ➔ Closed reduction: is a procedure to reduce a broken bone (put it back in its normal position) without cutting the skin open, see the picture.
- ➔ Open reduction: is reducing the bone surgically, if the fractured bone is exposed and you can see the bone fragments by your eyes and manipulate it by your hands and you reduce the fragments.

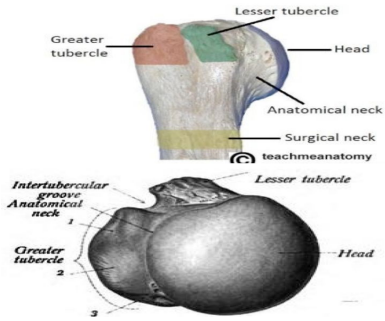


2- Proximal Humerus Fracture

Anatomy

Proximal humerus has 4 anatomic parts:

1. Head (articular surface), the anatomical neck separates the head from the tuberosities.
2. Greater tuberosity ***GT*** → Muscles attached *(SIT)*:
   - ➢ Supraspinatus (initiate abduction 0-15°)
   - ➢ Infraspinatus (external rotation and aid in abduction)
   - ➢ Teres minor (external rotation and aid in abduction)
3. Lesser tuberosity ***LT*** → Muscle attached:
   - ➢ Subscapularis (internal rotation).
4. Shaft.



- Anatomical neck v.s. Surgical neck: The surgical neck is located between the tuberosities and the shaft. **Why is it called surgical neck?** Because this is the location of many fractures that require surgery.

- Bicipital groove runs between GT & LT Attaches Biceps Brachii and Latissimus Dorsi.

- The short head of biceps (with coracobrachialis) goes more medially and attaches to the coracoid process.

Description

Causes of proximal fractures:

- Younger patients: violent (high energy) trauma such as RTA.
- Older patients: minor (low energy) trauma such as falling (osteoporotic or postmenopausal women).
- **Most fractures are minimally displaced** and can be treated with a sling.

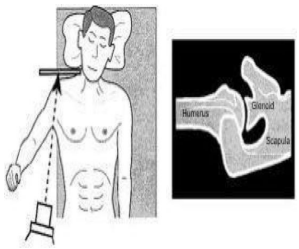
Physical exam:

1. **Expose** the shoulder very well.
2. **Look** for fracture signs (ecchymosis, erythema, swelling, tenderness, inability to move).
3. **Check** the skin, to know if it is an open or closed fracture (although open fractures are unlikely).
   - Examine the **axilla**, most of times they forget to examine it because patient has a lot of pain or they forget.
4. **Peripheral N/V** exam, to make sure there is no neurovascular injury.
5. **Axillary nerve**: the most commonly affected nerve, it innervates the lateral skin patch (area shown in the pic).
   - Patient will lose sensation over the deltoid and will have difficulty in abducting his shoulder.
6. **Examine** cervical spine: (the axillary nerve originates from C5 and C6)
   - You always have to examine the joint above and the joint below of any injury. In case of this fracture, the above is the cervical spine and below is elbow joint.

Imaging

X-Ray (3 views)**:** to make sure proximal humerus is not displaced

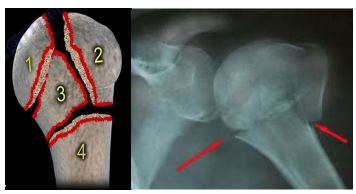
- AP and lateral views: 2 perpendicular (Orthogonal) views, to have 3D image of the fracture.
- Axillary view (special X-ray): the patient is laying down, and the beam will go through the axilla showing the whole joint. It can show you if there is a fracture-dislocation (it has different management).



CT scan: for displaced/dislocated fractures (to categorize the fracture and count the fragments), and intra-articular fractures and to plan surgery if needed.

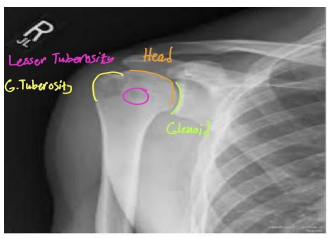
Neer's Classification:

- As discussed in the previous slide, proximal humerus is divided into 4 pieces: head (articular surface), LT, GT and a shaft (figure 1).
- Fracture is defined by the fragments displaced, this classification describes the number of fragments in the fracture with a maximum of 4 pieces, but it only considers the fragment as a separate piece if it was displaced by >1cm and/or angulated by >45°.
- So there are 4 types:
  - **One-part fracture (non-displaced humerus fracture):** No fragment satisfies the displacement criteria (figure 3), although there are many fracture lines, if the fragments are undisplaced, it's regarded as a one-part fracture.
  - **Two-part fracture:** If 1 segment (can be the head, LT, GT or the shaft) is displaced from the main bone by >1cm (figure 4).
  - **Three-part fracture:** If 2 fragments are displaced from the main bone (figures 2&5).
  - **Four-part fracture:** all 4 major parts (both tuberosities, articular surface, and shaft) are separated from each other (figure 6).

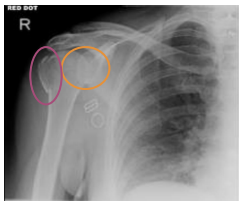


1 2

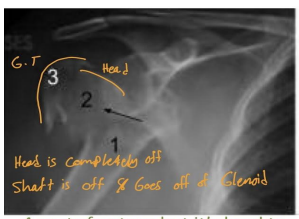
Fun Fact from Apley:
It has always been difficult to apply Neer's classification when based on plain X-rays, and not surprisingly there is a relatively high level of both inter- and intra-observer disagreement. Neer himself later noted that, when this classification was developed, the criteria for displacement (distance >1 cm, angulation >45 degrees) were set arbitrarily. The classification was not intended to dictate treatment, but simply to help clarify the pathoanatomy of the different fracture patterns.



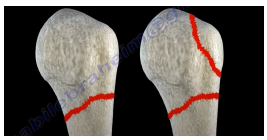
Normal



Three-part fracture
Displaced head: head is tilted back and faces up more than medially.
Fractured & minimally displaced G.T



4 parts fracture but it's hard to tell due to overlap.



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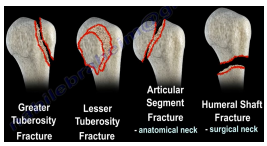

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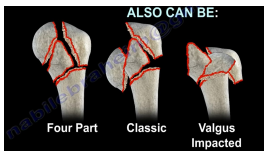
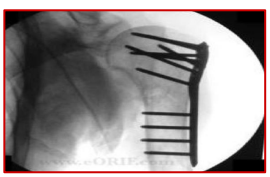


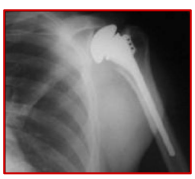
6

Treatment

- If fracture is not displaced or minimally displaced:
  - Treatment with sling and NWB (non-weight bearing) of UP (upper extremity) for 6-8 weeks.
    Why? based on the healing process of the fracture.
  - Early ROM exercises (pedaling exercise) after 2-4 weeks to prevent stiffness.
  - Normal function can be resumed after 3-4 months. (After 6-8 weeks we remove the sling, but the patient might still complain of pain and stiffness up to 3-4 months)
- If the fracture is displaced or intra-articular:
  - Surgery (ORIF with screws and plate) is indicated.
  - Shoulder **Hemiarthroplasty**[1] is indicated in some cases as in severely comminuted fractures.



ORIF with plate and screws



Hemiarthroplasty

1- In hemiarthroplasty, the ball (head of humerus) is changes, while in total arthroplasty, both the ball and socket are changed.

3- Humerus Shaft Fracture

Shaft fractures of the humerus, femur and tibia are classified according to which third was fractured.

General Description

It can be classified based on location of fracture: 1-Proximal. 2-Middle (most common location). 3-Distal.

Clinical Features

Symptoms: Pain, swelling, weakness ± shortening, motion/crepitus at fracture site, bruising.

Physical exam:

- Check the Skin to rule out open fractures.
- Compartment syndrome: Passively stretch distally to check for severe pain.
- Neurovascular (N/V): watch for **radial nerve palsy**. **How to examine the radial nerve?**
  - **Motor:** Extension of the metacarpophalangeal joints "posterior interosseous nerve" (extension of the wrist can be misleading because sometimes supplied by another branch).
  - **Sensory:** over the dorsum of the first webspace.
  - Check if the patient can raise their thumb and wrist "wrist drop & paralysis of the MCP extensors".

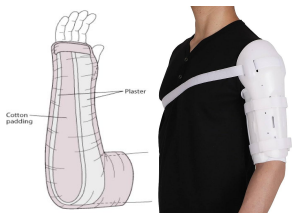
Imaging

X-Ray[1]:

- Left pic: AP and lateral of the right humerus showing long spiral shaft fracture at the junction of the middle and distal thirds of the humerus with lateral displacement, has good alignment with no angulation.

Treatment

Almost all humerus shaft fracture can be treated non-surgically by:



- Closed reduction at the ER under conscious sedation or analgesics.
- Functional brace for 4-6 weeks + NWB. (**Why called functional?** because they can still move their elbow and, to a lesser extent, their shoulders).
- Early ROM of elbow and shoulder to avoid stiffness, but arm abduction is postponed until the fracture has united.

What is the difference between brace and cast? the brace is removable, plastic with velcro tape, clamshell. There is no significant difference compared to the cast but it's easier to the patient.

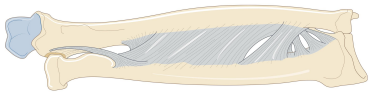
Surgery is indicated[2] (ORIF with plate and screws) for specific conditions like:

- Segmental fracture, (because it is considered unstable).
- Open fracture.
- Obese patient, **why?** Because of body built which will push the humerus and displace it, and also, they have a lot of fat which push the arm into varus, you can't control the fracture with a brace.
- Bilateral fracture, **why?** Patient can't function with 2 casts (inhumane).
- Floating elbow (Fractures of forearm and humerus) difficult to control, so basically like segmental, the elbow being the free segment.
- Multiple trauma: If he will undergoing for surgery, do it surgically because the patient already in OR.

1- Holstein-Lewis fracture is a spiral fracture of the distal one-third of the humeral shaft commonly associated with neuropraxia of the radial nerve (22% incidence).
2- Displacement is not indication.

4- Both Bones Forearm Fracture

Anatomy



- Forearm is complex with two mobile parallel bones; we consider the forearm as a ring or one joint (quadrilateral joint), usually if you break a ring at one side it breaks in another side so if the patient has a fracture in one bone, you should check the other bone or check the joint for disruption, it's rare to see an isolated ulnar or radial injury.

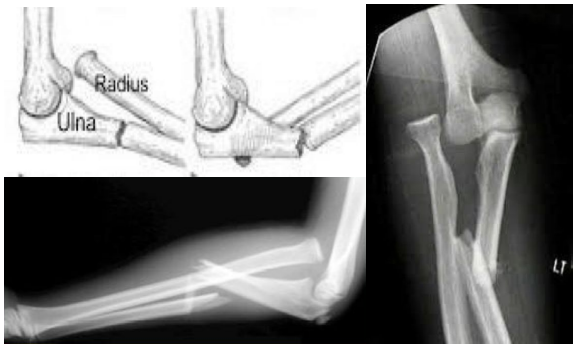
- Radius and ulna articulate proximally and distally, by the proximal and distal radioulnar joint (RUJ) to allow forearm rotation (supination and pronation).

General Description

Fractures are often from fall or direct blow. It's very unlikely to fracture only one bone without disrupting of their articulation.

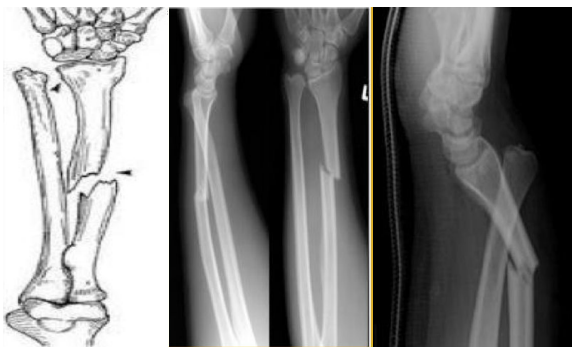
Types:

1. **Both bones fracture:** Both the radius and ulna are broken.
2. **Monteggia fracture:** Proximal or middle third ulnar shaft fracture with dislocation of radius (head) proximally (at elbow).
   - Mechanism: direct blow on the posterior aspect of the forearm, hyper-pronation or fall on a hyperextended elbow.
   - Clinical features: decreased rotation of forearm ± palpable lump at the radial head (dislocation).
3. **Galeazzi fracture:** Distal or middle third shaft radius fracture with disruption of DRUJ.
   - Mechanism: hand FOOSH (Fall on An Outer Stretched Hand) with axial loading of pronated forearm or wrist trauma.



Monteggia Fracture

Radial head can be dislocated to lateral or posterior side



Galeazzi Fracture

Radial fracture with ulnar dislocation dorsally

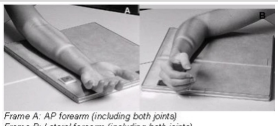
Both Bone Fracture

Clinical Findings

- Symptoms and signs of fracture: Deformity, pain, swelling loss of function in hand and forearm.
- Check the skin: To make sure it is not an open fracture.
- Check the compartments of forearm.
- Check ulnar, median and radial nerve, it bifurcates at the level of wrist into (**PIN**, **AIN**) "Posterior(radial) & anterior(median) interosseous nerves", so examine it separately.
- Check vascularity of the hands: color, temperature, capillary refill and pulse.

Imaging

- **2 Orthogonal views** perpendicular on each other usually AP + lateral with joint above and joint below.
- CT scan if fracture extends into joint "intra-articular to asses the displacement".



Frame A: AP forearm (including both joints)
Frame B: Lateral forearm (including both joints)
Image courtesy of Dr. Naveed Ahmad

Treatment

- **Both bone fracture:** We treat these fractures as a joint
  - Reduce and splint at ER/clinic (this is a temporary solution for pain control).
  - Definitive treatment: Almost always with surgery ORIF with plate and screws even if it didn't displace because we worry that it will later if we left it.
- **Monteggia fracture:**
  - ORIF of the fractured ulna and close reduction of radial head, if closed failed go for open "The key to successful treatment is to restore the length of the fractured ulna".
- **Galeazzi fracture:** Opposite to monteggia
  - ORIF of fractured radius with plate & screws and closed reduction of DRUJ.


Galeazzi managed with ORIF of radius

5- Distal Radius Fracture

General Description

- It is the Most common fracture of upper extremity.
- Most frequently are seen in older women due to osteoporosis.
- Young adults fractures are most commonly secondary to high energy trauma (FOOSH).
- Types:

1. **Extra-articular fractures**:
   A. Colles' fracture: **dorsal** angulation, shortening and radial "displacement" deviation.
   B. Smith's fracture: shortening and **volar** (anterior) angulation (reverse colles').
2. **Intra-articular fractures**:
   A. Barton's fracture: Partial articular (i.e. half of the articular surface is fractured and lost contact with the shaft while the other half is intact and in contact with shaft (fig 1), if no part of the articular surface is in contact with shaft then this is not barton's), could be volar or dorsal (Fig 2), needs lateral view.
   B. Others: Due to a fall on extended pronated wrist (Chauffeur fracture aka Hutchinson or Backfire fracture, which is radial styloid fracture).

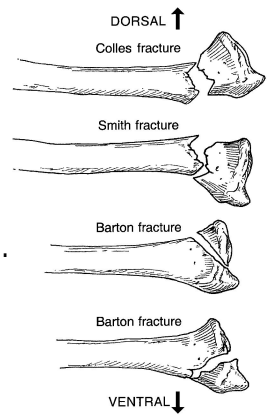


Fig 1

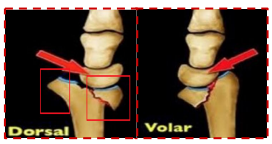
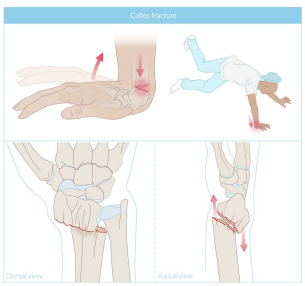
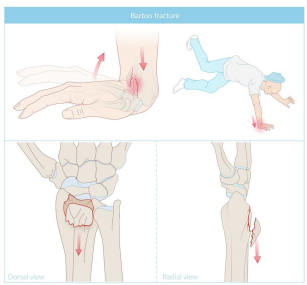
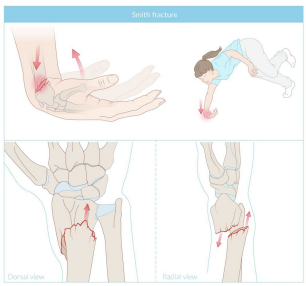
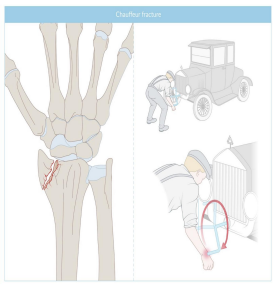
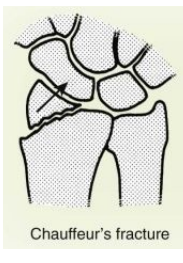


Fig 2

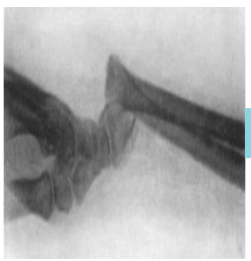





Chauffeur's fracture


Clinical Features

Colles' Fracture	Mnemonic: Colle's restaurant Smith's farm	Smith's Fracture
Can present clinically with a "Dinner Fork" deformity		Can present clinically with a "Shovel" deformity

Imaging

- X-ray: PA not AP → it's hard to ask the patient to supinate his arm.
- In Barton's fracture you have to see it in lateral x-ray because on AP/PA there will be overlap.
- **CT Scan:** if fracture extends into joint (intra-articular).



Smith's fracture

Colles' fracture

Barton's fracture

Treatment

Extra-articular fractures (Colles' and Smith's): you have to know the specific steps, it always comes in OSCE

1. Start with analgesia or preferably, conscious sedation and N/V exam.
2. Closed reduction (traction and countertraction), check N/V again, then apply a splint or below elbow cast (to avoid elbow stiffness, and don't include metacarpophalangeal joint also to avoid finger stiffness), then send the patient to X-ray to check if reduction is acceptable.
    a. If reduction is acceptable:
        - Send the patient home with acute compartment syndrome checklist (tell them if they have pain not responding to analgesia "called pain out of proportion" to immediately come to ER to be checked for compartment syndrome).
        - Patient should be checked once a week for 2 weeks for secondary displacement. If X-ray was good at the 2nd week, continue the cast for 4 more weeks (immobilization for 6-8 weeks in total), then bring them back for out-of-cast X-ray to make sure it is healed.
        - ROM exercises after cast removal.
    b. If closed reduction is unacceptable (failed):
        - Try again if you think you may get it.
        - If very unstable or you think you won't get it, then surgery (ORIF with plate and screws).

ORIF with the plate and screws

Intra-articular fracture (Barton's and Chauffeur's):

- The treatment is the same as extra-articular unless there is an indication for surgery which is:
    - Displacement (also called step or gap)[1] of intra-articular fracture ≥2 mm[2].
    - If Surgery Indication is present → ORIF with plate and screws.

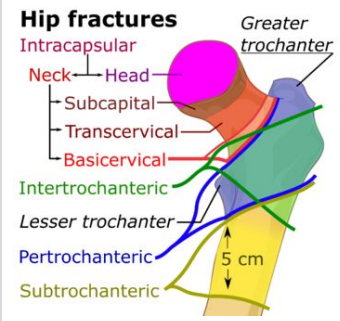
1- The 2 fragments at the articular surface are not at the same level, if it heals like that it will lead to arthritis.
2- If it was less than 2mm (no step or gap), then let them stay in the cast and check them every week for 2 weeks, if stable then we continue to a total of 6 weeks, but most of barton's fractures are unstable "have a step or gap".

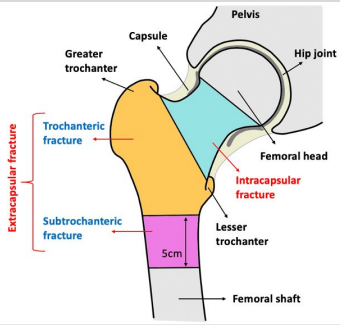
Lower Extremities

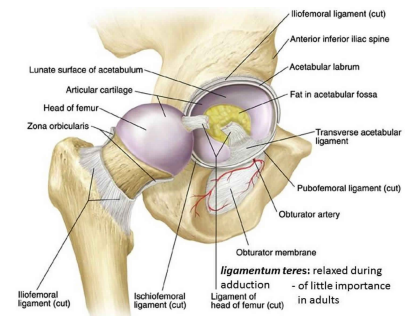
1- Hip Fractures in Elderly (>60 years)

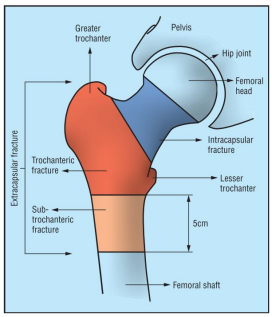
- The physiological age is what matters not the exact age, because you can have a 60 y\o male but he is completely fit and healthy with no issues (in the exam stick to 60 as a cutoff).

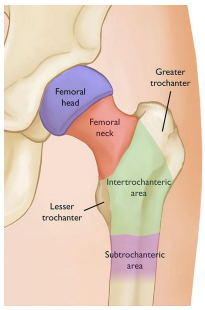
Anatomy









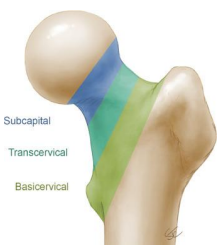


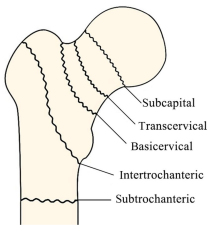
General Description

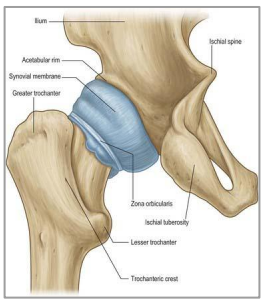
- Hip fractures are defined as fractures that occur between the articular margin of the femoral head to 5 cm below the lesser trochanter.
- The usual story of this fracture: A geriatric patient falls down in the bathroom and it is usually managed by surgery.
- It is the most common fracture of lower limb.
- **It is associated with osteoporosis** (Like colles in old women).
- Most common mechanism is **a fall from standing height**.
- In young people it is usually caused by high energy trauma and is considered a separate entity of fractures that affects elderly.
- **BEWARE** → you have to ask **what caused the fall?** it might be a manifestation of a much more serious medical condition that you first have to deal with or **rule out during clinical evaluation** before managing the fracture such as an **MI or a stroke.**
- Hip fracture is a life changing event, it's not about the fracture itself, but it's about the systemic failure; "The patient starts to be senile". Most people will walk but they will not be the same. Mortality: 20% of these people will die 1 year after the fracture but not because of the fracture, but due to the complications that follow, it just tells you how it's linked to systemic failure.

Fractures can be classified into: intra/extra-capsular (important for prognosis and treatment) **or displaced and non-displaced.**

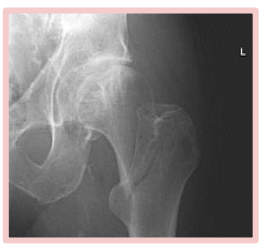
- **Intracapsular** (also called femoral neck fracture) **:**
  - Subcapital: Directly below the femoral head.
  - Transcervical: Mid portion of femoral neck.
  - ❖ Avascular necrosis (AVN) risk is higher with **intracapsular fracture**.
- **Extracapsular :**
  - Basicervical: Base of femoral neck, the end of the capsule.
  - Intertrochanteric.
  - Subtrochanteric: not a hip fracture, rather, a femoral shaft fracture.







Hip capsule



Intertrochanteric fracture

Common associated injuries "at time of fall"**:**

1. **Distal radius fracture.**
2. **Proximal humerus fracture.**
3. **Subdural hematoma.**

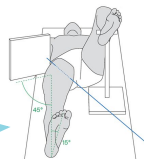
Clinical

- Full detailed history of mechanism of injury.
- Rule out syncope, chest pain, weakness, MI, stroke, etc.
- A detailed systematic review.
- Look for Hip fracture deformity: Abduction, external rotation and shortening "typical but not always".
- **Assess distal N/V status:** The most commonly injured nerve is **the sciatic nerve**.
- **Avoid ROM if fracture is expected**, if patent can not do active, don't try passive ROM.

Imaging

X-Ray:

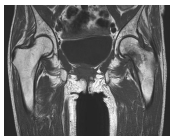
- 3 views are needed:
  1. AP pelvis.
  2. AP hip.
  3. Lateral hip. (Cross-table view)



The X-ray shows either subcapital or transcervical intracapsular fracture

AP of pelvis

Lateral of hip



Occult femoral neck fracture

AP of hip

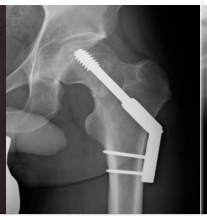
MRI is sensitive for occult (stress) fractures, they don't appear on an X-ray, they happen in people who stress their hip like military or trumps.

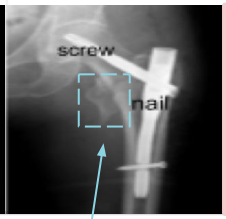
Treatment (includes elderly and young)

IMPORTANT

IMPORTANT

Intracapsular			Extracapsular (age is irrelevant)	
Non-displaced So no need for reduction	**Displaced**		**Stable**	**Unstable**
Internal fixation in situ with 3 percutaneous cannulated screws (PCS)	Elderly	Young	Closed reduction and internal fixation with Dynamic hip screws (DHS)	Closed reduction with internal fixation using a cephalomedullary nail (CMN)
	Hemiarthroplasty	ORIF With cannulated screws		





- **Signs of fracture instability:** Need only 1 to be considered unstable
  1- Four-parts fracture. **2-** Extension to subtrochanteric region. **3-** Large lesser-trochanteric fragment.
- In the ER, no close reduction is needed, **why?** it will displace again anyway. a study showed that there is no difference if you put a traction or not, not cost effective, and no benefit for the patient.
- No traction is needed.
- Patient needs surgery ideally within 48 hours, even less, as studies show it decreases the rate of mortality by more than 40% due to post surgery complications (e.g. atrophy, DVTs, infection, etc)
- The goal is to ambulate patient as soon as possible.
- For all hip fractures, be sure that **DVT prophylaxis is started**.
- Be sure the patient will be evaluated for osteoporosis after discharge, to avoid more fractures.

Why do we do hemiarthroplasty in elderly when they have displaced intracapsular hip fractures, while in young we do ORIF with cannulated screws?

- Patients who undergo hemiarthroplasty can start walking at the same day, ORIF mandates NWB the limb for at least 6 weeks. When elderly are told not to bear weight, they will just lay down in bed all day and be prone to all the complications mentioned below, In the other hand, younger people will stay perform their daily life using crutches.

Complications

1. Nonunion (most common complication in young people):
   - ➔ 2% in IT (intertrochanteric) fractures very rare.
   - ➔ 5% in non-displaced neck fracture.
   - ➔ 30% in displaced neck fracture and among young people.
2. AVN in femoral neck fracture:
   - ➔ 10% in non-displaced.
   - ➔ 30% in displaced.
3. Death:
   - ➔ Early 4%. i.e within the first few weeks of a hip fracture.
   - ➔ At 1 year post-fracture, 20-40% of the patients will be dead, because of some complication like pneumonia, delayed surgery, delayed amputation, bed sores, immobility, and post surgery complication.
4. VTE (venous thromboembolism).

2-Femoral Neck Fractures (young patients)

General

- It is a completely different entity from similar fractures in elderly (>60 years).
- Commonly caused by high energy mechanism (e.g. High-impact trauma such as motor vehicle accidents), start with ATLS protocol.
- 2.5% associated femoral shaft fracture, (needs long femur X-ray), in young people because fractures are caused by high energy.
- Complications:
  - Nonunion: 30% (most common)
  - AVN: 25-30%

Clinical Features (EXTRA)

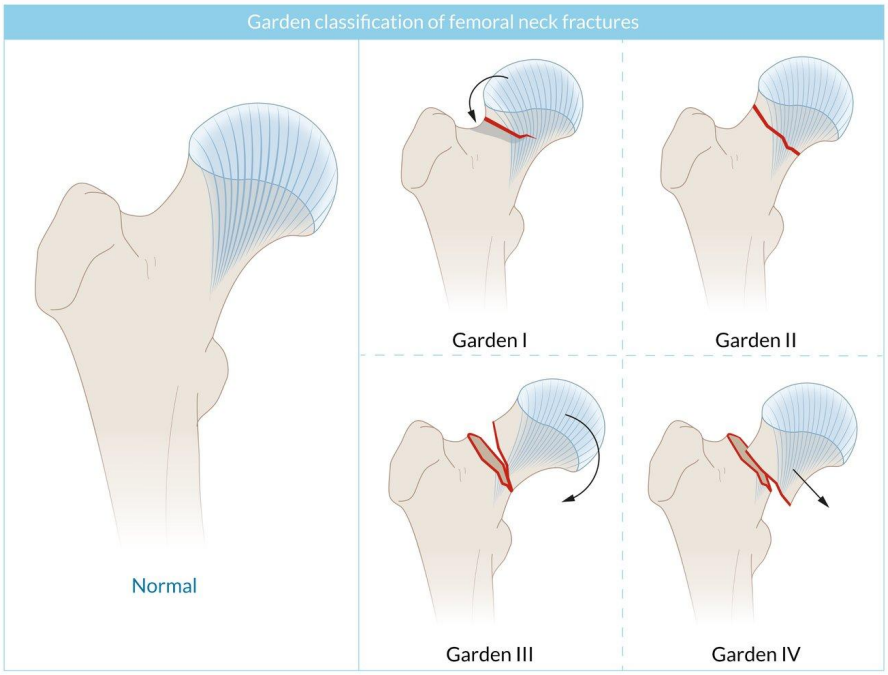
- Groin pain
- Shortened and externally rotated leg
- Minimal bruising

Diagnostic (EXTRA)

- X-ray (AP and lateral view of the pelvis with internal rotation of the affected limb).
- Full femur X-ray of the affected limb should be done to avoid unseen fractures in shaft.
- MRI or bone scan if clinical suspicion is high despite absent findings on x-ray.

Classification (EXTRA)

Garden Classification	
Garden I	Nondisplaced, incomplete, impaction fracture
Garden II	Complete, but nondisplaced fracture
Garden III	Complete and partially displaced fracture
Garden IV	Complete, fully displaced fracture



Treatment (EXTRA)

- Conservative management:
  - Indication: stable, nondisplaced fractures, especially abduction fractures , mostly in debilitated patients.
  - Methods:
    - Temporary bed rest or use of crutches followed by mobilization with physical therapy.
    - Venous thromboembolism prophylaxis.
- Surgical therapy (usually within 72 hours is indicated for unstable fractures, typically adduction fractures, and fragment dislocation:
  - For children and young adults:
    - Attempt preservation of the femoral head.
    - Early (within 6 hours) ORIF.
  - For older adults: total hip replacement (THR) or hip hemiarthroplasty.

3- Femoral Shaft Fractures

If you have femoral shaft fracture look for hip injuries as well

Etiology

More common	Less common
• High energy mechanisms. - Car accidents, fall from a height, gunshot wound. • Young patients "adolescents" (male, < 30 years). • Start with ATLS protocol.	• Low energy mechanism (torsional forces). • Old patients. • Spiral type fracture (twisting).

Clinical Associations

IMPORTANT

MSK injuries	Non-MSK Injuries

1. Ipsilateral femoral neck fracture in 10% (missed in 30-50%, so always check the femoral neck and if you highly suspect it do a CT scan).
2. Knee ligaments injuries in 50%.
3. Meniscal tear in 30%.
4. Floating knee injury: Less common
  - Ipsilateral tibial and femoral shaft fractures.
5. Vascular/nerve injuries: Rare.
6. Contralateral femoral shaft fracture, bilateral:
  - Worse prognosis among above.
	1. Fat embolism, from medullary canal usually to the lung causing shortness of breath "most common associated injury".
	2. ARDS (Acute respiratory distress syndrome).
	3. Head injuries.
	4. Abdominal injuries.

Clinical Examination

- Start with ATLS.
- Look for fracture signs and symptoms.
- Check skin integrity, to rule out open fracture.
- Do N/V exam.
- Do compartment assessment.
- Check for knee swelling or ecchymosis, Indicates ligament Injury.

Investigations

- AP and lateral views of femur:
  - Joint above (spine, looking for bamboo spine) and joint below.
- 15° Internal rotation AP view of ipsilateral hip:
  - To get a good profile of the femoral neck.
- Lateral view ipsilateral view.
- Hip CT if femoral neck fracture is suspected.
- Knee AP and lateral views.

Management

- ATLS: ABC resuscitation, if surgery will be delayed, we can use:
  - Skeletal traction (proximal tibial pin)[1] for pain control and preventing motion which reduces fat embolism risk.
- Early surgical fixation: Is the key
  - ➔ Proven to reduce pulmonary complications: PE, fat embolism, ARDS.
  - ➔ Must be within 24 hours (ideally < 6 hours) "as soon as feasible".
  - ➔ If patient is unstable → External fixation "temporary".
  - ➔ If patient is **stable** → Closed reduction & IM nailing.

Complications

- Malunion:
  A. **Most common**.
  B. More common with proximal fracture (**subtrochanteric fracture**) because of muscles attachments.
  C. Rotational, angulation or shortening (or combination of those).
- Nonunion: Rare.
- Infection: In case of open fracture the skin condition and contamination will change the fixation method from IM to external fixation as temporary and this will worsen the outcome of femur fracture.
- VTE: Always give prophylaxis.

4- Tibial Shaft Fractures

If you have tibial shaft fracture look for ankle injuries as well

General Description

- It is a subcutaneous bone (high suspicion for skin injury), not a lot of muscles are covering it.
- Most common large long bone fracture, and has higher incidence of **open bone fractures** than femur.
- It can be secondary to low or high energy mechanism.
- It carries the highest risk of compartment syndrome, because it is small space and less distal muscles.
- 20% of tibial fractures can be associated with ankle intra-articular fracture.

Classified based on:

A. Location and morphology:
   - ➔ Proximal third.
   - ➔ Middle third.
   - ➔ Distal third.

B. Displaced v.s. Non-displaced.

Clinical Examination

- Check skin integrity to rule out open fracture.
- Assess compartments of leg: Needs serial exam.
- Serial N/V exam.

1- Pins are put proximal and distal to fracture, the pins go through the skin and bone and then out from the other side, the patient would be at the end of the bed with a robe and weight attached to the pins. Benefits: Immobilization of the bone and decrease the risk for fat embolism.

Investigations

X-ray:

- **AP and lateral tib/fib**.
- AP and lateral knee.
- AP and lateral ankle.

CT scan if fracture extends into joints above or below.

- Left pic: AP X-ray of the right lower leg showing comminuted fractures of both tibia and fibula in the middle third + ankle fracture.
- Right pic: AP and lateral X-ray of right leg showing nondisplaced fracture of the tibial shaft in the middle third.

Management

Non-surgical Treatment

Indications:

- No displacement: < 10° angulation on AP/lateral X-rays.
- <1cm shortening.
- Not comminuted.

Procedure: Closed reduction and cast immobilization:

- **Definitive treatment: Above knee full cast, it must be bi-valved** to minimize the risk of compartment syndrome.
  - A bivalve (Anteroposterior or mediolaterally) for 8 weeks, why? If there's any swelling the bivalve gives an area for expansion and this will prevent compartment syndrome.
- In all fractures, if patient is discharged home with cast always provide them with Compartment Syndrome checklist, (pain out of proportion: not responding to analgesia or requiring more analgesia).
- NWB for 8 weeks with cast immobilization.

Surgical Management (most common modality)

Indications: These are also considered "contraindications of non-surgical management".

- Displacement (> 10° angulation).
- Open fracture.
- Compartment syndrome.
- Floating knee.

Procedure:

- **In the ER:** Temporary reduction + Above knee backslab and U-shape slab (3-sided slab) until surgery.
- Most commonly **IM** nail fixation.

IM nail of the tibia with proximal and distal interlocking screws

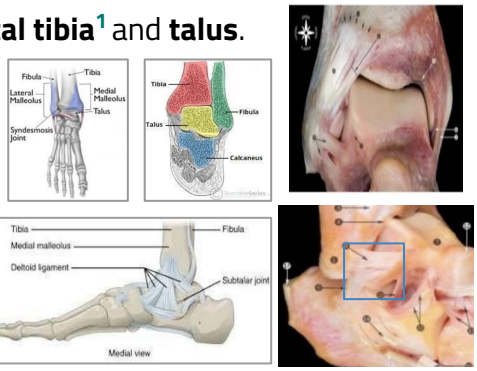
Complications

- **Non-union:** Most common complication, while in femoral shaft it's malunion.
- Delayed union.
- Infection: Open fracture.
- DVT/PE.

5- Ankle Fractures

Anatomy

- Medial (distal tibial extension) and lateral (distal tibial extension) malleoli, **distal tibia**[1] and **talus**.
- Highly **congruent joint** (stable).
- Fibula is held to distal tibia by syndesmotic ligament.
- Medial malleolus is held to talus by deltoid ligament.
- Lateral malleolus is held to talus by LCL (lateral collateral ligament)
- Lateral ligament complex of the ankle is composed of three ligaments:
  - Anterior & Posterior talofibular ligament (ATFL-PTFL).
  - Calcaneofibular ligament (CFL).



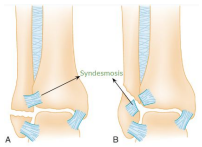
General Description

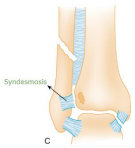
IMPORTANT

- Most of them are low energy "torsional" injuries that cause malleoli fracture.

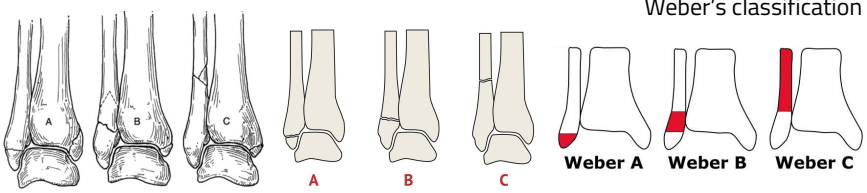
Classifications:

- Stable (non-surgically) v.s. Unstable fracture (surgically):
  - **Causes of instability →** 1- **Lateral displacement of talus**. 2- **Bimalleolar fracture**.
- Medial (usually surgically), lateral (depend on the stability) or bimalleolar fracture (directly unstable and requires surgical fixation).
- For lateral malleolus (and bimalleolar) fractures, we can subdivide them using **Weber's classification**[2]:
  A. Below the level of syndesmosis.
  B. At the level of syndesmosis.
  C. Above the level of syndesmosis.





Weber's classification



Clinical Findings

- Look for fracture signs and symptoms.
- Assess medial joint ecchymosis or tenderness to assess medial malleolus and deltoid ligament integrity.
- Assess N/V status (before and after reduction).
- A valgus deformity is present in medial malleolus fractures.

Imaging

X-ray:

- AP.
- Lateral.
- Mortise view: AP +15° internal rotation (special view).
- Long leg X-rays: If only medial malleolus is broken.

CT scan if fracture extends to articular distal tibia surface.

Unstable: Weber B + very wide medial clear space, so needs surgical fixation with plate & screws

1- The distal articular surface of the tibia is called 'Plafond', so the ankle joint is the articulation between the 'Plafond' and talus.
2- In X-ray we can't see the ligaments, so how to know where is the syndesmosis level? the syndesmosis is present in the area where the distal tibia and fibula overlap on x-ray, look at the AP picture in the imaging part.

Treatment

Medial malleolar fracture:

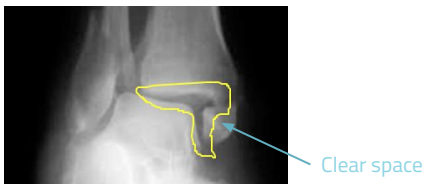
- If non displaced we splint or apply a below knee cast and treat conservatively.

Lateral malleolar fracture "intact medial malleolus":

1. **Weber A:** Stable, No surgery
   - Splint (cast) + NWB for 6-8 weeks.
   - Early ROM.
2. **Weber B:** Plate & screws
   - **We check the clear space on the medial side, if it's wider than 4 mm the fracture is considered to be laterally displaced** making the fracture unstable:
     - ORIF, if the tibia and fibula are displaced we will put a syndesmotic screw between them, it is called **syndesmotic screw** because it acts as a syndesmotic ligament (hold the bones together until syndesmosis heal).
   - If stable "< 4 mm": Cast + NWB for 6 weeks.
   - If not: Call orthopedic for stress film X-rays to see if it's open or not.
3. **Weber C:** Unstable, needs surgical fixation (plate & screws).

Stable: Weber B

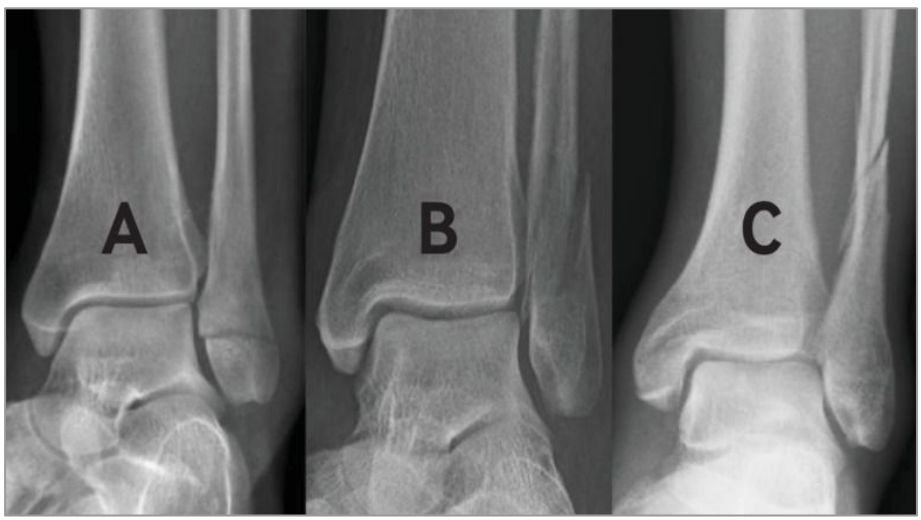
Unstable: > 4 mm lateral translation

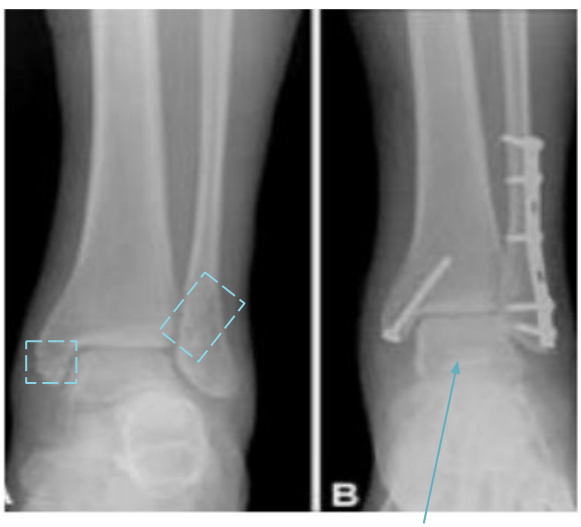


If both malleoli are broken "Bimalleolar fractures": Directly unstable & need surgical fixation

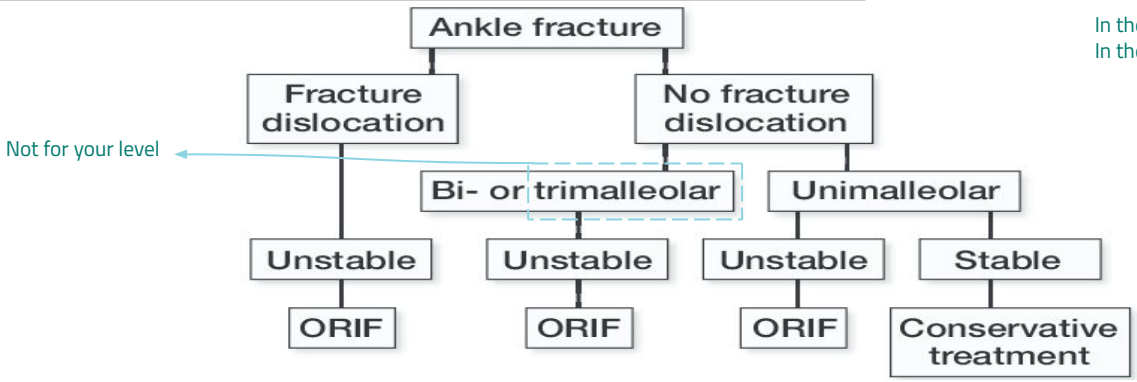
- ORIF with plate & screws (ORIF both bones -/+ syndesmotic screw).

Bimalleolar fracture





In the lateral malleolus: Plate & screws
In the medial malleolus: Screws only



EXTRA

Clavicle Fracture

- incidence: proximal (5%), middle (80%), or distal (15%) third of clavicle
- common in children (unites rapidly without complications)

Mechanism
- fall on shoulder (87%), direct trauma to clavicle (7%), FOOSH (6%)

Clinical Features
- pain and tenting of skin
- arm is clasped to chest to splint shoulder and prevent movement

Investigations
- evaluate NVS of entire upper limb
- x-ray: AP, 45° cephalic tilt (superior/inferior displacement), 45° caudal tilt (AP displacement)
- CT: useful for medial physeal fractures and sternoclavicular injury

Treatment
- medial and middle-third clavicle fractures
  - for nondisplaced fractures, simple sling for 1-2 wk prn
  - early ROM and strengthening once pain subsides
  - if fracture is shortened >2 cm, consider ORIF
- distal-third clavicle fractures
  - undisplaced (with ligaments intact): sling for 1-2 wk
  - displaced (CC ligament injury): ORIF

Specific Complications (see *General Fracture Complications*, OR7)
- cosmetic bump (most common complication)
- shoulder stiffness, weakness with repetitive activity
- pneumothorax, brachial plexus injuries, and subclavian vessel (all very rare)

Open Reduction and Internal Fixation vs. Nonsurgical Treatment in Displaced Midshaft Clavicle Fractures: A Meta-Analysis
J Orthop Trauma 2018;32(7):e276-e283
Purpose: Compare outcomes from ORIF and non-operative treatments in displaced mid-shaft clavicular fractures.
Methods: Meta-analysis with 9 RCTs reporting nonunion, functional outcomes, and subsequent surgeries in patients older than 16 yr.
Results: 9 randomized clinical trials with 1027 total patients were included. ORIF was associated with significantly lower nonunion rate of 1.7% compared to 14.5% for the non-operative treatment groups (RR 0.15, 95% CI, 0.08-0.31). Functional outcomes, rated by either DASH or Constant scores, were significantly better in ORIF up to 6 mo. When excluding elective plate removal, the rate of subsequent surgeries was significantly lower in the ORIF cohort (4.7% vs. 14%, RR 0.36, 95% CI 0.24-0.56).
Conclusions: ORIF is associated with significant reductions in nonunions and earlier functional outcomes in displaced midshaft clavicular fractures.

Associated Injuries with Clavicle Fractures
- Up to 9% of clavicle fractures are associated with other fractures (most commonly rib fractures)
- Majority of brachial plexus injuries are associated with proximal third fractures

EXTRA

Proximal Humeral Fracture

Mechanism
- young: high energy trauma (MVC)
- elderly: FOOSH from standing height in osteoporotic individuals

Clinical Features
- proximal humeral tenderness, deformity with severe fracture, swelling, painful ROM, bruising extends down arm and chest
- physical exam usually reveals diminished forward elevation, with or without disuse atrophy of deltoid and periscapular musculature

Investigations
- test axillary nerve function (deltoid contraction and skin over deltoid)
- x-rays: AP, trans-scapular, and axillary views of the shoulder are essential
- CT scan: to evaluate for tuberosity or articular involvement and fracture displacement, and if the diagnosis of non-union is unclear

Classification
- Neer classification is based on 4 fracture locations or 'parts'
- displaced: displacement >1 cm and/or angulation >45°
- the Neer system regards the number of displaced fractures, not the fracture line, in determining classification
- ± dislocated/subluxed: humeral head dislocated/subluxed from glenoid

Treatment
- assess for and treat osteoporosis if needed
- non-operative
  - nondisplaced and minimally displaced (85% of patients): broad arm sling immobilization, begin ROM within 14 d to prevent stiffness
  - most displaced fractures in low-demand elderly patients
- operative
  - ORIF (anatomic neck fractures, displaced, associated irreducible glenohumeral joint dislocation) or IM nail (surgical neck)
  - hemiarthroplasty or reverse TSA may be necessary, especially in elderly
  - minimally invasive percutaneous pinning and intramedullary nail fixation are indicated in rare instances

Specific Complications (see *General Fracture Complications*, OR7)
- AVN, nerve palsy (45%; typically axillary nerve), malunion, post-traumatic arthritis, persistent pain and weakness, frozen shoulder

Neer Classification
Based on 4 parts of humerus
- Greater tuberosity
- Lesser tuberosity
- Humeral head
- Shaft

One-part fracture: any of the 4 parts with none displaced
Two-part fracture: any of the 4 parts with 1 displaced
Three-part fracture: displaced fracture of surgical neck + displaced greater tuberosity or lesser tuberosity
Four-part fracture: displaced fracture of surgical neck + both tuberosities

EXTRA

Humeral Shaft Fracture

Mechanism
- high energy: direct blows/MVC (especially young)
- low energy: FOOSH, twisting injuries, metastases (in elderly)

Clinical Features
- pain, swelling, weakness ± shortening, motion/crepitus at fracture site
- must test radial nerve function before and after treatment: look for drop wrist, sensory impairment in dorsum of hand

Investigations
- x-ray: AP and lateral views of the humerus, including the shoulder and elbow joints

Treatment
- in general, humeral shaft fractures are treated non-operatively
- non-operative
  - ± reduction; can accept deformity due to compensatory ROM of shoulder
  - hanging cast (weight of arm in cast provides traction across fracture site) with collar and cuff sling immobilization until swelling subsides, then Sarmiento functional brace, followed by ROM
- operative
  - indications: see *NO CAST* sidebar, OR6, pathological fracture, "floating elbow" (simultaneous unstable humeral and forearm fractures)
  - ORIF: plating (most common), IM rod insertion, external fixation (rare)

Specific Complications (see *General Fracture Complications*, OR7)
- failure of functional bracing (seen in up to 30% of patients)
- radial nerve palsy: expect spontaneous recovery in 3-4 mo, otherwise send for EMG
- non-union: most frequently seen in middle 1/3
- decreased ROM
- compartment syndrome

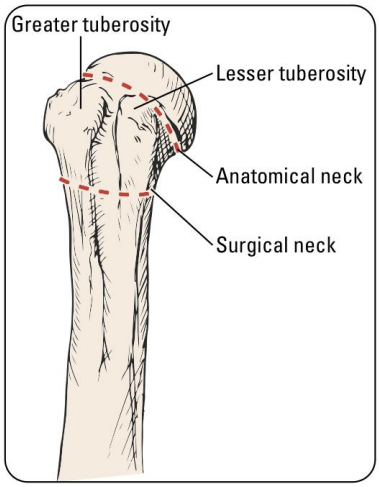


Figure 15. Fractures of the proximal humerus

Acceptable Humeral Shaft Deformities for Non-Operative Treatment
- <20° anterior angulation
- <30° varus angulation
- <3 cm of shortening

Risk of radial nerve and brachial artery injury

EXTRA

Radius and Ulna Shaft Fractures

Mechanism
- high-energy direct or indirect (MVA, fall from height, sports) trauma
- fractures usually accompanied by displacement due to high force

Clinical Features
- deformity, pain, swelling
- loss of function in hand and forearm

Investigations
- x-ray: AP and lateral of forearm ± oblique of elbow and wrist
- CT if fracture is close to joint

Treatment
- goal is anatomic reduction since imperfect alignment significantly limits forearm pronation and supination
- ORIF with plates and screws; closed reduction with immobilization usually yields poor results for displaced forearm fractures (except in children)

Specific Complications (see *General Fracture Complications*, OR7)
- compartment syndrome
- soft tissue contracture resulting in limited forearm rotation – surgical release of tissue may be warranted

Monteggia Fracture

- fracture of the proximal ulna with radial head dislocation and proximal radioulnar joint injury
- more common and better prognosis in the paediatric age group when compared to adults

Mechanism
- direct blow to the posterior aspect of the forearm
- hyperpronation
- fall on the hyperextended elbow

Clinical Features
- pain, swelling, decreased rotation of forearm ± palpable lump at the radial head
- ulna angled apex anterior and radial head dislocated anteriorly (rarely the reverse deformity occurs)

Investigations
- x-ray: AP and lateral views of the elbow, wrist, and forearm

Treatment
- adults: ORIF of ulna with indirect reduction of radiocapitellar joint in 90% of patients (open reduction of radiocapitellar joint if unsuccessful)
- splint and early postoperative ROM if elbow completely stable, otherwise immobilization in plaster with elbow flexed for 2-3 wk
- paediatrics: attempt closed reduction and immobilization in plaster with elbow flexed for Bado Type I-III, surgery for Type IV

Specific Complications (see *General Fracture Complications*, OR7)
- PIN injury: most common nerve injury; observe for 3 mo as most resolve spontaneously
- radial head instability/redislocation
- radioulnar synostosis

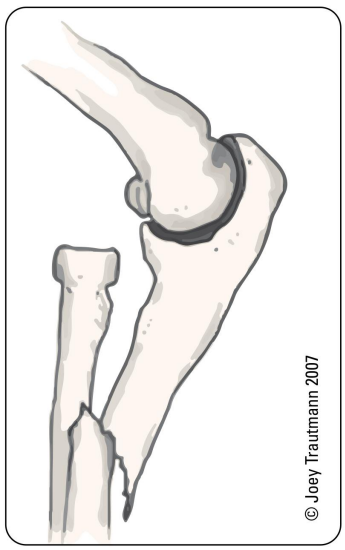


Figure 19. Monteggia fracture

In all isolated ulna fractures, assess proximal radius to rule out a Monteggia fracture

Bado Type Classification of Monteggia Fractures
Based on the direction of displacement of the dislocated radial head, generally the same direction as the apex of the ulnar fracture
Type I: anterior dislocation of radial head and proximal/middle third ulnar fracture (60%)
Type II: posterior dislocation of radial head and proximal/middle third ulnar fracture (15%)
Type III: lateral dislocation of radial head and metaphyseal ulnar fracture (20%)
Type IV – combined: proximal fracture of the ulna and radius, dislocation of the radial head in any direction (<5%)

EXTRA

Galeazzi Fracture

- fracture of the distal radial shaft with disruption of the DRUJ
- most commonly in the distal 1/3 of radius near junction of metaphysis/diaphysis

Mechanism

- FOOSH with axial loading of pronated forearm or direct wrist trauma
- forceful axial loading of radial shaft (e.g. direct trauma to distal 1/3 of radius)

Clinical Features

- pain, swelling, deformity, and point tenderness at fracture site

Investigations

- x-ray: AP, and lateral views of the elbow, wrist, and forearm
  - shortening of distal radius >5 mm relative to the distal ulna
  - widening of the DRUJ space on AP
  - dislocation of radius with respect to ulna on true lateral

Treatment

- all cases are operative ("fracture of necessity")
  - ORIF of radius; afterwards, assess DRUJ stability by balloting distal ulna relative to distal radius
  - if DRUJ is stable and reduced, splint for 10-14 d with early ROM encouraged
  - if DRUJ is unstable, ORIF or percutaneous pinning with long arm cast in supination x 2-3 wk

For all isolated radius fractures assess DRUJ to rule out a Galeazzi fracture

Monteggia vs. Galeazzi Fractures
Remember the mnemonic "**MUGGER**":
Monteggia
Ulnar fracture
Galeazz
Radial fracture

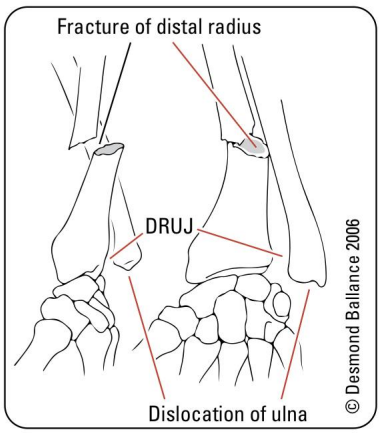


Figure 21. Galeazzi fracture

EXTRA

Colles' Fracture

- extra-articular transverse distal radius fracture (~2 cm proximal to the radiocarpal joint) with dorsal displacement ± ulnar styloid fracture
- most common fracture in those >40 yr, especially in women and those with osteoporotic bone

Mechanism
- FOOSH

Clinical Features
- "dinner fork" deformity
- swelling, ecchymosis, tenderness

Investigations
- x-ray: AP and lateral ± oblique views of wrist

Treatment
- goal is to restore radial height (13 mm), radial inclination (22°), volar tilt (11°), as well as DRUJ stability and useful forearm rotation
- non-operative
  - closed reduction (think opposite of the deformity)
  - hematoma block (sterile prep and drape, local anesthetic injection directly into fracture site) or conscious sedation
  - closed reduction: traction with extension (exaggerate injury); traction with ulnar deviation, pronation, flexion (of distal fragment – not at wrist)
  - dorsal slab/below elbow cast for 5-6 wk
  - obtain post-reduction films immediately; repeat reduction if necessary
  - x-ray at 1 wk, 3 wk, and at cessation of immobilization to ensure reduction is maintained
- operative
  - indication: failed closed reduction, or loss of reduction
  - percutaneous pinning, external fixation, or ORIF

Indications for Direct Surgical Management of Colles' Fracture
- Displaced intra-articular fracture
- Comminuted
- Severe osteoporosis
- Dorsal angulation >5° or volar tilt >20°
- >5 mm radial shortening

Features of Inadequate Closed Reduction that Require ORIF
- Radial shortening >3 mm or
- Dorsal tilt >10° or
- Intra-articular displacement/step-off >2 mm

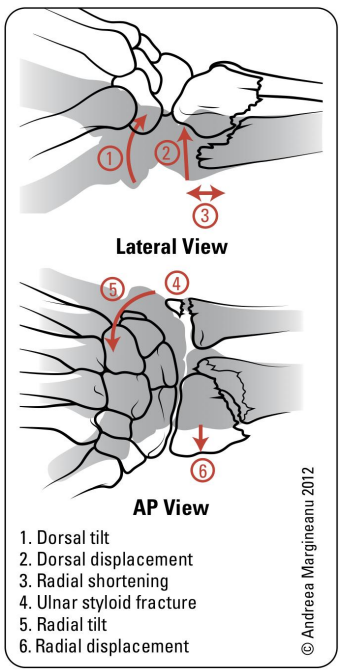


Figure 22. Colles' fracture and associated bony deformity

Smith's Fracture

- volar displacement of the distal radius (i.e. reverse Colles' fracture)

Mechanism
- fall onto the back of the flexed hand

Investigations
- x-ray: AP and lateral ± oblique views of wrist

Treatment
- usually unstable and needs ORIF
- if patient is poor operative candidate, may attempt non-operative treatment
  - closed reduction with hematoma block (reduction opposite of Colles')
  - long-arm cast in supination x 6 wk

Complications of Wrist Fractures

- most common complications are poor grip strength, stiffness, and radial shortening
- distal radius fractures in individuals <40 yr of age are frequently high energy/comminuted and are more likely to require ORIF
- 80% have normal function in 6-12 mo

Table 13. Early and Late Complications of Wrist Fractures

Early	Late
Difficult reduction ± loss of reduction	Malunion, radial shortening
Compartment syndrome	Painful wrist secondary to ulnar prominence
Extensor pollicis longus tendon rupture	Frozen shoulder ("shoulder-hand syndrome")
Acute carpal tunnel syndrome	Post-traumatic arthritis
Finger swelling with venous block	Carpal tunnel syndrome
Complications of a tight cast/splint	CRPS/RSD

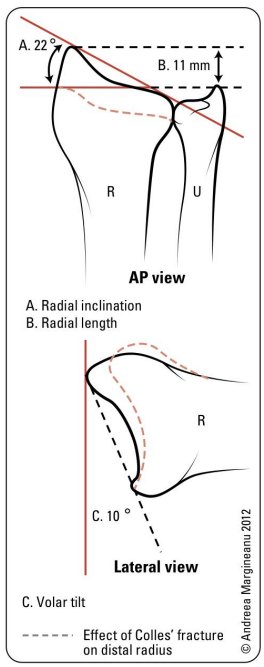


Figure 23. Normal wrist angles+ wrist angles in Colles' fracture
Note the relative shortening of the radius relative to the ulna on AP view in Colles' fracture

Hip Fracture

General Features

- acute onset of hip pain after a fall
- unable to weight-bear
- shortened and externally-rotated leg
- painful ROM

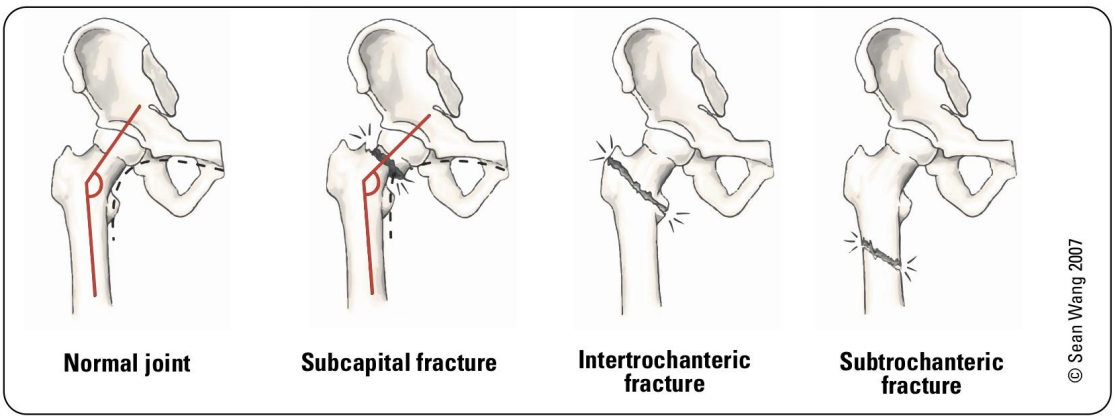


Figure 35. Subcapital, intertrochanteric, and subtrochanteric hip fractures

Table 20. Overview of Hip Fractures

Fracture Type	Definition	Mechanism	Investigations	Treatment	Complications
Femoral Neck (Subcapital)	Intracapsular	Young: MVC, fall from height Elderly: fall from standing, rotational force	X-Ray: AP hip, AP pelvis, cross table lateral hip	See Table 21, OR32	DVT, non-union, AVN, dislocation
Intertrochanteric Stable: intact posteromedial cortex Unstable: non-intact posteromedial cortex	Extracapsular fracture between the greater and lesser trochanters and transitional bone between the neck and shaft	Same as femoral neck fracture Direct or indirect force transmitted to the intertrochanteric area	X-Ray: AP hip, AP pelvis, cross table lateral hip	Closed reduction under fluoroscopy then dynamic hip screw or IM nail	DVT, varus displacement of proximal fragment, malrotation, non-union, failure of fixation device
Subtrochanteric	Fracture begins at or below the lesser trochanter and involves the proximal femoral shaft	Young: high energy trauma Elderly: osteopenic bone + fall, pathological fracture	X-Ray: AP pelvis, AP/ lateral hip and femur	Closed/open reduction under fluoroscopy, then IM nail	Malalignment, non-union, wound infection

Table 21. Garden Classification of Femoral Neck Fractures

Type	Displacement	Extent	Alignment	Trabeculae	Treatment
I	**None**	"Incomplete"	Valgus or neutral	Disrupted	Internal fixation to prevent displacement (valgus impacted fracture)
II	**None**	Complete	Neutral	Aligned	Internal fixation to prevent displacement
III	**Partial**	Complete	Varus	Disrupted	Young: ORIF Elderly: hemi-/total hip arthroplasty
IV	**Complete**	Complete	Varus	Disrupted	Young: ORIF Elderly: hemi-/total hip arthroplasty

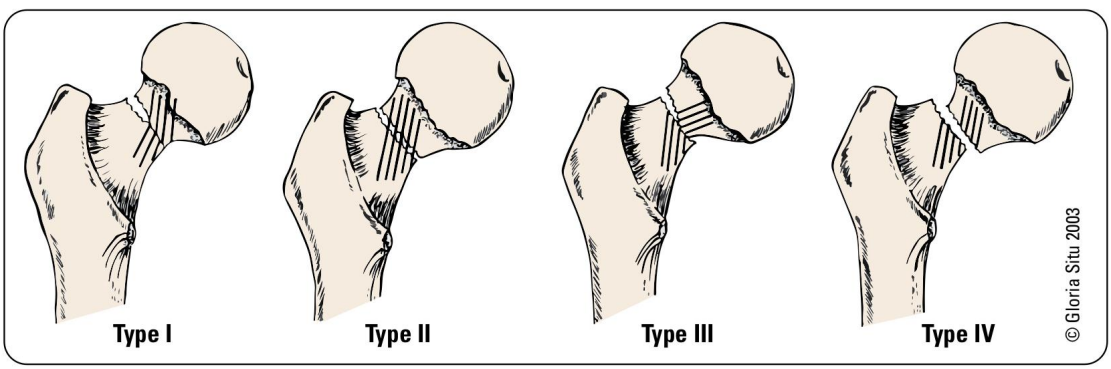


Figure 36. Garden classification of femoral neck fractures

X-Ray Features of Subcapital Hip Fractures

- Disruption of Shenton's line (a radiographic line drawn along the upper margin of the obturator foramen, extending along the inferomedial side of the femoral neck)
- Altered neck-shaft angle (normal is 120-130°)

DVT Prophylaxis in Hip Fractures

LMWH (i.e. enoxaparin 40 mg SC BID), fondaparinux, low dose heparin on admission, do not give <12 h before surgery

AVN of Femoral Head

- Distal to proximal blood supply along femoral neck to head (medial and lateral femoral circumflex arteries)
- Susceptible to AVN if blood supply disrupted
- Etiology: femoral neck fracture, chronic systemic steroid use, SCFE, Legg-Calvé-Perthes, SLE, RA

Comparative Effectiveness of Pain Management Interventions for Hip Fracture: A Systematic Review

Ann Intern Med 2011;155(4):234-245

Study: Randomized controlled trials (RCTs); nonrandomized controlled trials (non-RCTs); and cohort studies of pain management techniques in older adults after acute hip fracture.

Conclusions: Nerve blockade seems to be effective in reducing acute pain after hip fracture. Low-level evidence suggests that preoperative traction does not reduce acute pain. Evidence was insufficient on the benefits and harms of many other interventions.

EXTRA

Femoral Diaphysis Fracture

Mechanism
- high energy trauma (MVC, fall from height, gunshot wound)
  - pathologic as a result of malignancy, osteoporosis, bisphosphonate use
- in children, can result from low energy trauma (spiral fracture)
  - always consider the possibility of non-accidental trauma

Clinical Features
- shortened, externally rotated leg (if fracture displaced)
- inability to weight-bear
- often open injury, always a Gustilo III (see Table 6, OR10)
- Winquist and Hansen classification

Investigations
- x-ray: AP pelvis, AP, and lateral views of the hip, femur, knee

It is important to rule out ipsilateral femoral neck fracture, as they occur in 2-6% of femoral diaphysis fractures and are reportedly missed in 19-31% of cases

Treatment
- non-operative (paediatric, uncommon in adults)
  - possible indication: non-displaced femoral shaft fractures in patients with significant comorbidities who are non-ambulatory
  - most femoral shaft fractures require fixation as this is a life-threatening injury
- operative
  - ORIF with anterograde IM nail (most common) or retrograde IM nail or with plate and screw fixation
  - external fixation may be used initially (e.g. unstable patients or polytrauma patients)
  - early mobilization and strengthening

Complications
- blood loss
- infection
- fat embolism leading to ARDS
- VTE
- malrotation, leg length discrepancy
- malunion/nonunion

Associated Injuries
- extensive soft tissue damage
- ipsilateral hip dislocation/fracture (2-6%)
- nerve injury

EXTRA

Tibial Shaft Fracture

- most common long bone fracture and open fracture

Mechanism
- low energy pattern: torsional injury
- high energy: including MVC, falls, sporting injuries

Clinical Features
- pain, inability to weight bear, deformity
- open vs. closed
- neurovascular compromise

Investigations
- x-ray: full length AP and lateral views
  - AP, lateral, and oblique views of ipsilateral knee and ankle

Treatment
- non-operative
  - indication: closed and minimally displaced or adequate closed reduction
  - long leg cast x 6-8 wk, convert to functional (patellar tendon bearing) brace after
- operative
  - indication: displaced or open
  - if displaced and closed: ORIF with IM nail, plate and screws, or external fixator
  - if open: antibiotics, I&D, external fixation or IM nail, and vascularized coverage of soft tissue defects

Specific Complications (see *General Fracture Complications*, OR7)
- significant incidence of compartment syndrome and neurovascular injury
- knee pain (>50% anterior knee pain with infrapatellar IM nailing)
- malunion, nonunion
- lack of soft tissue coverage secondary to open fracture may require further surgery for muscle flap coverage and can lead to poor outcome

Figure 45. Tibial shaft fracture treated with IM nail and screws

Tibial shaft fractures have high incidence of compartment syndrome and are often associated with soft tissue injuries

EXTRA

Ankle Fracture

Mechanism
- pattern of fracture depends on the position of the ankle when trauma occurs
- classification systems
  - Danis-Weber: based on location of main fibular fracture line relative to the syndesmosis
  - Lauge-Hansen: based on foot position and direction of applied stress/force

Treatment
- non-operative
  - indication: non-displaced, Danis-Weber Type A, and some isolated Danis-Weber Type B
  - NWB in below knee cast, or weight bearing as tolerated in walking boot
- operative
  - indications
    - fracture-dislocation: restore vascularity, minimize articular injury, reduce pain and skin pressure
    - most Danis-Weber Type B, and all Type C
    - any talar displacement
    - displaced isolated medial or lateral malleolar fracture
    - trimalleolar (medial, posterior, lateral) fractures
    - displaced and large posterior malleolar fractures
    - persistent medial clear space widening despite attempt at closed reduction and immobilization
    - open fracture/open joint injury
  - ORIF with plates and screws

Complications
- risk of poor wound healing and deep infections (up to 20%) in patients with DM, particularly if concomitant peripheral neuropathy
- postoperative stiffness
- malunion, nonunion
- post-traumatic arthritis

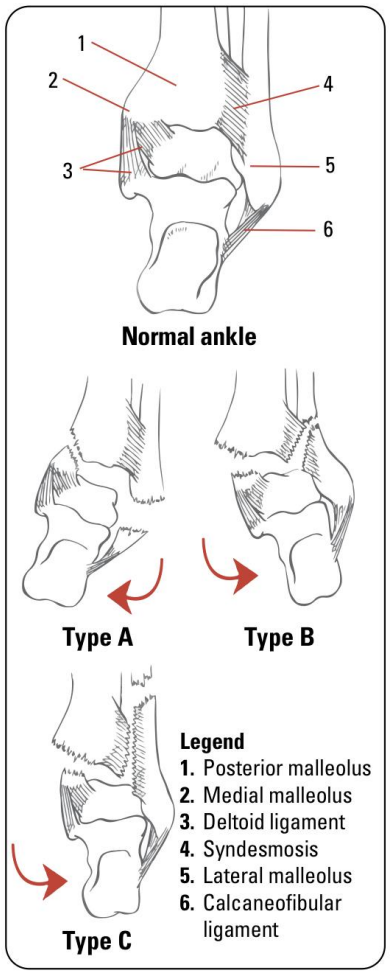


Figure 46. Ring principle of the ankle and Danis-Weber classification

Danis-Weber Classification
- Based on level of fibular fracture relative to syndesmosis
- Type A (infra-syndesmotic)
- Pure inversion injury, tibiofibular syndesmosis remains intact
- Avulsion of lateral malleolus below plafond or torn calcaneofibular ligament
- ± shear fracture of medial malleolus
- Type B (trans-syndesmotic)
- External rotation and eversion (most common)
- ± avulsion of medial malleolus or rupture of deltoid ligament
- Spiral fracture of lateral malleolus starting at plafond
- Type C (supra-syndesmotic)
- Pure external rotation
- Avulsion of medial malleolus or torn deltoid ligament
- ± posterior malleolus avulsion with posterior tibio-fibular ligament
- Fibular fracture is above plafond
- Frequently tears syndesmosis

Extra

Click here
for
Notes from 439 based on (AMBOSS, PubMed & some google researches) for some explanations!

Quiz

Q1: A 22-year-old male had ORIF with plate & screws after a fracture in his humerus, what neurological deficit could happen in his situation?

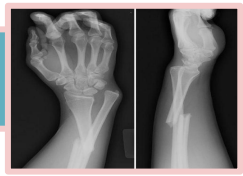
A. Loss of wrist extension

B. Loss of thumb opposition

C. Loss of sensation over the medial 2 fingers

D. Loss of sensation over the medial aspect of the arm

Q2: Which type of fracture is shown on the image?



A. Galeazzi

B. Barton

C. Monteggia

D. Chauffeur

Q3: Which of the following is the best treatment for an elderly with an intracapsular fracture?

A. IM nail

B. Hemiarthroplasty

C. DHS

D. Percutaneous in situ fixation

Q4: A 17-year-old football player had an direct injury to his shoulder, X-ray showed dislocated shoulder anteriorly. What concomitant sign can you see clinically?

A. Deltoid atrophy

B. Claw hand

C. Wrist drop

D. Thenar atrophy

Q5: Which of the following is the best management option for a clavicular fracture with skin tenting?

A. Immobilization with arm sling

B. Immobilization with figure of 8

C. Closed reduction and K-wires

D. ORIF with plate & screws

1:A 2:A 3:B 4:A 5:D

Quiz

SAQs

441 & 439:

1. Name 4 types of distal radius fracture?
   1- Colles' fracture
   2- Smith's fracture
   3- Barton's fracture
   4- Chauffeur's fracture

2. Name 4 indications of operative treatment of humerus fracture?
   1- Segmental fracture
   2- Open fracture
   3- Obese patient
   4- Bilateral fracture

3. What is the treatment for a displaced intracapsular femoral neck fracture in a 75 old lady?
   - Hemiarthroplasty

4. Which nerve might be affected in a proximal humeral fracture?
   - Axillary nerve

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4

Orthopedic 441 Team

MED441 KING SAUD UNIVERSITY

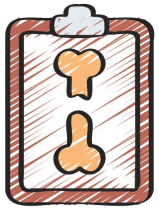
Editing File

Common Pediatric Fractures

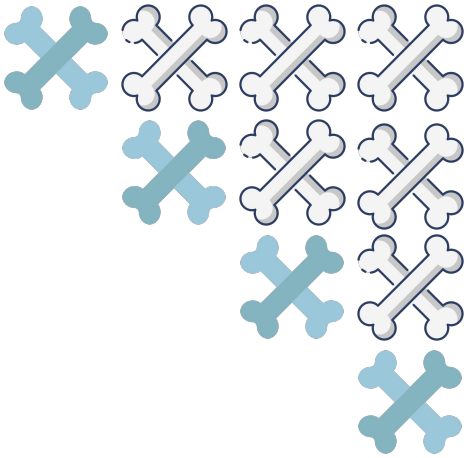
Dr. Khalid Bakarman

Color Index:

- Main Text
- Important
- 441 Notes
- Old Notes
- Extra



Objectives



- Difference between Pediatric & adult
- Physis fracture → Salter-Harris classification
- Indications of operative treatment
- Methods of treatment of Pediatric fracture & trauma
- Common Pediatric fractures:
  - U. L → clavicle, humeral supracondylar, distal radius
  - L. L → femur shaft

Resources

Introduction:

- Different from adult fractures
- Fractures account for ~15% of all injuries in children.
- Boys > girls
- Types of fractures vary in various age groups (infants, children and adolescents).
- Rate increases with age till adolescent.
- Pediatric fractures have **great remodeling potentials. Growth plates** and periosteum are important in remodeling

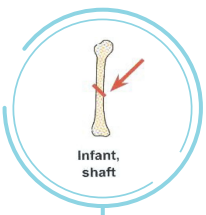
A good number of cases can be treated conservatively, operative fixations aid in avoiding complications

.

- Adults are skeletally mature; pediatrics are skeletally immature. In Saudi Arabia, the pediatric age range is 14 "18 in the west" and below.

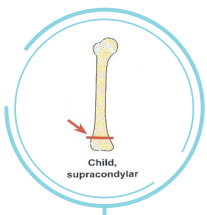
Why are children's fractures different?

Age-related fracture pattern[1]



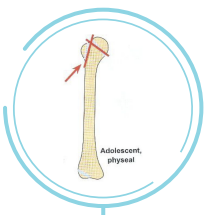
Infants:

diaphyseal fractures



Children:

Metaphysical fractures[2]



Adolescent:

Epiphyseal Injuries

Examples of Remodeling in children:

This is more evident in younger children. Don't expect this in a child aged 13-14 years old. We only realign the bone then apply casting. Fractures near to a joint that moves everywhere (multiaxial ball-and-socket synovial joint like the shoulder) and near to epiphysis → Better for remodeling.

1- Age in pedia MCQs is very important, in 2 Qs they had the same case but different ages which will make the answer for each Q totally different. In real life situation though age related fracture patterns is indicative on the incidence and not accuracy eg: (14 y/o child, most likely epiphyseal fracture but can happen in metaphysis or diaphysis)

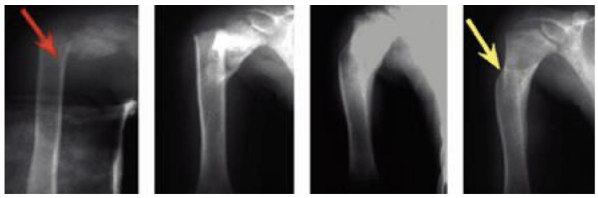
2- Starts with walking age

This radiograph shows diaphyseal humeral fracture
- Note the tremendous amount of callus at day
- 6 months → almost normal

Femur fracture: Big callus forms remodeling 3 segments fracture. You can see the Plaster of Paris (POP) cast in the first picture

100% translation of femur bone fracture Union happened at 15 weeks.

Fracture in the humerus surgical neck with remodeling



Children have different physiology and anatomy:

1-

Growth Plate:
- Provides perfect remodeling power.
- Injury of growth plate causes deformity. It's a two edged sword, can aid in healing the deformity by itself or can cause the deformity depends on the mechanism of the fracture.

May cause angular deformity or "Leg Length Inequality" (L.L.I)
- A fracture might lead to overgrowth.

The growth plate gives the length in long bones, while the periosteum increase the width of a bone. In the picture the lateral side growth is arrested while the medial side continues to grow "genu valgus deformity"
- If there is an injury to the medial growth plate while the lateral side is normal and the bone continues to grow, we will have Varus deformity.
- If the injury is in the lateral growth plate we will end up having valgus deformity.
- If there is destruction of both sides the whole bone will shorten
- (leg length discrepancy).

Procurvatum/recurvatum: This refers to the movement of a single bone; where a procurvatum deformity describes forward bending of the bone and recurvatum deformity is the backward bending of the bone.

Genu valgus deformity

2

Bone:

Increased collagen/bone ratio makes the bone:
- Less brittle
- Becomes deformed

The bone itself has more collagen than actual bone → do not fracture easily and it can bend sometimes, this is what we call plastic deformation.

(Bowing deformity):

Picture shows the cortex and periosteum are intact, it's not a fracture it's called plastic deformation this is not the normal bowing of ulna. The younger the patient the more you will see this.

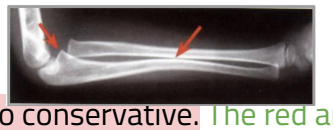
3

Cartilage:

- Difficult X-ray evaluation. Radio translucent.
- Size of articular fragment often underestimated.

We always operate if it involves the lateral condyle because the size of fracture is underestimated. Children and adolescents have more cartilage that's why in an X-ray we see spaces.

Collateral condyle must be treated surgically



- **(red arrow)**: Left elbow joint. You might think that without displacement (less than 2mm) so we can go conservative. The red arrow is pointing to the ossification center (this is not the radial head). This child is so young that the ossific nucleus of radius did not appear yet. This is the ossific nucleus of the capitulum and it should be higher up. - Right picture (yellow arrow): This is an elbow of an older child, the whole epicondyle is fractured and rotated, we call it fracture of necessity 2 (always going intra-articular) we don't go with conservative it should be anatomical reduction to prevent the risk of premature osteoarthritis.

4

Periosteum: (which is an actual layer)

Metabolically active

- more callus, rapid union, increased remodeling

Thickness and strength

- Intact periosteal hinge affects fracture pattern. May aid reduction if it's intact

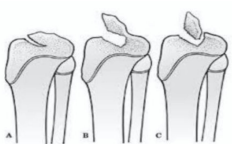
In adults it becomes thinner, but it's very strong in pediatrics, where we need a scalpel to cut it. We can't restore it, but we can bring back the two ends close to each other. If the bone breaks, we don't feel the pain in the cortex + medulla, we only feel the pain from the nerve endings which are in the periosteum. So, fracture movement is what causes pain that's why the first line of analgesia for any fracture is immobilization.

5

Ligaments:

Functionally stronger than bone, unlike in adults. Therefore, Higher proportion of injuries that produce sprains in adults result in fractures in children. You'll never hear a child with ACL tear.

ACL is attached to the tibial eminence; tibial spine fracture is not normally seen in adults. It causes avulsion of the tibial spine. X-ray shows abnormal knee (tibial plateau should be smooth), we use a headless screw to stabilize it (arthrotomy or arthroscopy). ACL is intact, the injury happened in the epiphysis.



The only exception not to do surgery for intra articular fracture is if it's <2mm, will heal with its articular cartilage

6

Physiology:

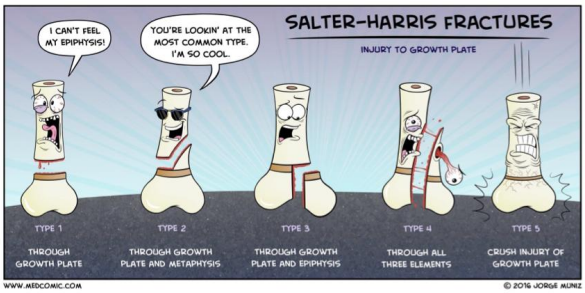
Better blood supply. AKA periosteum. So, the remodeling potential is 6-high. Rare incidence of delayed union and non-union (mainly the problem with children is malunion not non-union. Non-union mainly a problem found in adults)

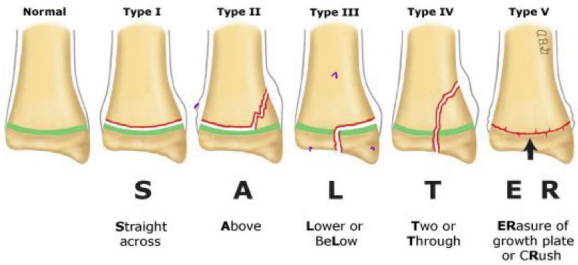
Physis Fracture:

The growth plate, or physis, is the translucent, cartilaginous disc separating the epiphysis from the metaphysis and is responsible for longitudinal growth of long bones. As we said before the growth plate is a two edged sword it helps in remodelling and union but if it gets injured itself there will be a deformity.

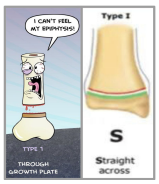
- Account for ~25% of all children's fractures.
- More in boys.
- More in upper limb more in the dominant hand.
- **Most** heal well rapidly with good remodeling, but growth may be affected.

★ Salter-Harris Classification





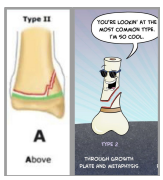
Type I



Through the growth plate.

It doesn't appear on the x-ray, so usually request an x-ray to the other side. In type 1, you go with history and examination mainly (A Child fell down and he is crying and holding his knee, he can't bear weight (femur) sometimes in X-ray you see a fracture that is slightly translucent, but don't forget your history and examination in which you can reach a diagnosis up to 70 % just by proper history. A transverse fracture through the hypertrophic or calcified zone of the plate. Even if the fracture is quite alarming displaced, the growing zone of the physis is usually not injured and growth disturbance is uncommon. Usually apply a cast and it'll be sufficient.

Type II



II

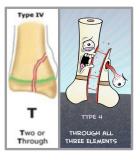
Growth plate with metaphysis.

This is is similar to Type 1, but towards the edge the fracture deviates away from the physis and splits off a triangular piece of metaphyseal bone. Growth is usually not affected. This is the commonest type. Usually apply a cast and it'll be sufficient.

Type III

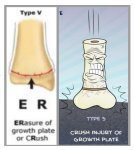
Growth plate with epiphysis.
"Intra-articular", needs anatomic reduction, usually it will go to surgery. This fracture runs partly along the physis and then veers off through all layers of the physis and the epiphysis into the joint. Inevitably the reproductive zone of the physis is damaged and this may result in growth disturbance. one of the complications is premature osteoarthritis.

Type IV



Growth plate with metaphysis and epiphysis.
needs anatomic reduction, usually it will go to surgery. As with Type 3, this fracture splits the epiphysis, but it continues through the physis into the metaphysis. The fracture is particularly liable to displacement and a consequent misfit between the separated parts of the physis, resulting in asymmetrical growth. (corrected with K-wires and screws).

Type V:

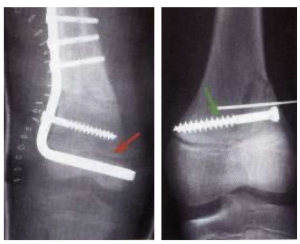


A longitudinal compression injury of the physis, doesn't cause angle deformity, it only causes leg-length discrepancy. Sometimes you have to do X-ray for both limbs to compare. There is no visible fracture, but the growth plate is crushed, and this may result in growth arrest.
The worst Type.

Complications of Physeal Injuries:

Physeal bridging[1] (AKA Physeal bar) a < 1% (from 438), Cause as affecting growth (varus, valgus, or even L.L.I)
Keep in mind:

- Small bridges (<10%) may lyse spontaneously
- Central bridges more likely to lyse
- **Peripheral bridges more likely to cause deformity**



Be Careful:

1. Avoid injury to physis during fixation
2. Monitor growth over a long period (18-24 m). Varus or valgus will not appear immediately, they will take time to appear. Because in the period of 18-24 months, the body is still healing.
3. Image suspected physeal bar (CT/MRI) Not CT only MRI.

-When suspecting physeal bar do **MRI**/CT because you are looking for soft tissue (growth plate) **(MCQ)**

-A scenario where someone fell but got treatment and healed properly, he came after a few years suffering from a deformity or valgus and you check the most recent X-Ray, what's the next step to see the bar? **MRI**

Type 5 salter-harris fractures in the distal tibia sometimes can cause an incomplete crushing of the epiphyseal plate, which in turn will cause the continuation of the bone growth only on one side which lead to valgus or varus with time. The ankle in the picture is showing varus. We check in integrity of the epiphyseal plate using MRI.

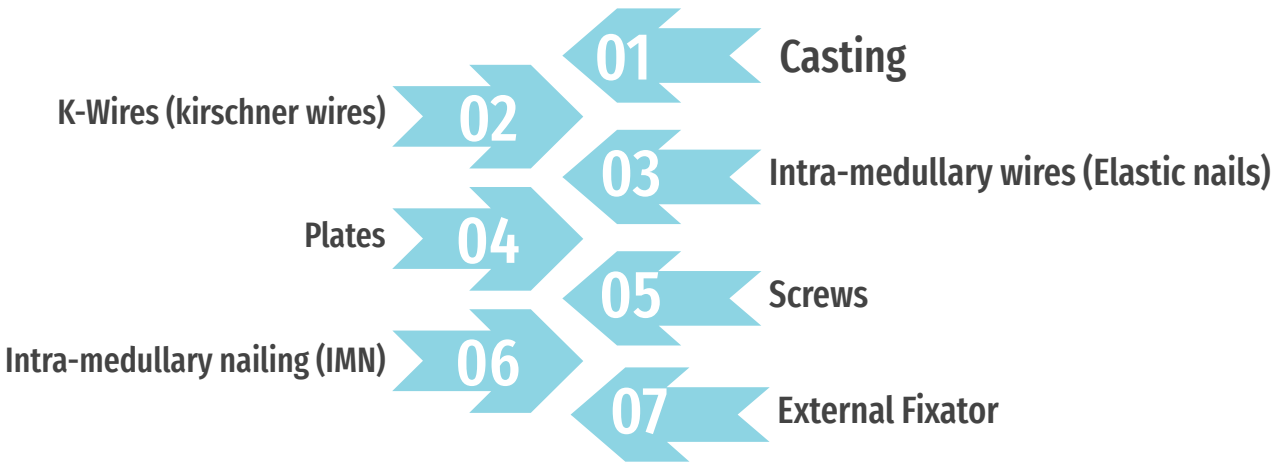
1- Bony bridge crossing the growth plate that results in growth disturbance and/or deformity.

General management:

Indications for surgery: (MCQ) Golden slide

- Open fractures
- Severe soft-tissue injury to do debridement
- Fractures with vascular injury actual cut of the vessels
- Compartment syndrome "it depends when patient came to hospital"
- Multiple injuries.(like both femur fractures)
- **Displaced intra articular fractures (Salter-Harris III-IV).**
- Failure of conservative means (irreducible or unstable fractures).
- Malunion and delayed union very rare because of rich blood supply "when you see nonunion in MCQs **don't** choose it. We know that a child healing is much faster". One of the causes of nonunion is exposure to smoking.
- **Adolescence** we tend to treat them as adults, because the remodeling potentials are really low
- **Head injury (MCQ)** in RTA. **If asked look for operative ones**. When the patient loses consciousness, the body starts healing. At the beginning catabolic stage caused by the injury → they'll develop a lot of callus" what happens in week 4 can happen in week 2 that's why we should fix it as soon as possible before it malunite. Except if the fracture is nondisplaced and immobilizes properly → we don't need surgery.
- Neurological disorder cerebral palsy, myelomeningocele pts. (they're not normal
- and sometimes their bones don't heal in a normal way)
- Uncooperative patient like pts with ADHD

Methods of Treatment (Fixation) of Pediatric Fractures & Trauma



Method	Indication
Casting	Acute fractures or sprains, or for initial stabilization of reduced, displaced, or unstable fractures before orthopedic intervention. Used to correct deformities especially in pediatrics. **IMP Notes: Still the commonest.**   Casting is the first option of treatment. In infants we don't use cast, instead we use tongue depressor.
K-wires (Kirschner wires) (relative fixation)	• Fractures in epi- /metaphyseal areas. Fractures of small bones (e.g. hand and foot). • Small bony fragments. • For fragment reposition in multifragmentary fractures in addition to stable fixation.  IMP Notes: • Most commonly used for internal fixation (I.F). • Usually used in metaphyseal fractures & supracondylar fractures. • Inserted percutaneously in OP under GA. • You can go through growth plate, but it is done by seniors. • Not good for bone fixation in adults. 
Intramedullary wires (Elastic Nails) (relative fixation)	When you bend a K wire it will bend but elastic wire will recoil. The elastic wire uses the technique of 3-points fixation (2C shaped wires, they come straight, and we bend them to give a push in the center. Usually used in mid-shaft diaphysis **"Only in long bones"**→ stabilizes the fracture (3 points: upper crossing/lower crossing/central push). In the radius and ulna we don't have enough space in the medulla to put 2 wires so we put 1 wire. Used mainly in pediatrics but can be used in adults. 

Plates (absolute fixation)	Especially **<u>in multiple trauma</u>** IMP Notes: Comminuted fracture and we use bridging plate. In comminution we don't search for each single piece to fix but what is important is to keep length to prevent LLD.
Screws (relative fixation)	**IMP Notes:** **We NEVER cross a physis with a screw because it will destroy the growth plate, with a k-wire yes because it's smooth.** **Remember in SCFE (salter harris 1) we put a screw for in situ fixation, but we won't fuse it into the growth plate.**
Intramedullary Nailing [I.M.N] (relative fixation)	Only in **adolescents (>12y) (MCQ)** There are many blood vessels come for head of femur and other for greater trochanter. The area between the greater trochanter and the neck is called piriformis fossa (blood supply of the growth plate of the greater trochanter) if I go there with an IM nail I will interfere with the blood supply "AVN" of the greater trochanter, it will affect the growth plate and it will grow into valgus and if it goes into too much valgus it will dislocate. IF I give you **MCQ** and the options are IM nail, K wire, Elastic nail, traction please look at the age: If it's 14 year old patient choose IM nail / If it's 4 year old patient choose elastic nail.
External fixator	Usually in open fractures. Valid in pts who have skin conditions. "crush injury" Used as a temporary treatment for Fractures. Because they are easily applied, external fixators are often put on when a patient has multiple injuries and is not yet ready for a longer surgery to fix the fracture.
Combination	 

Before:

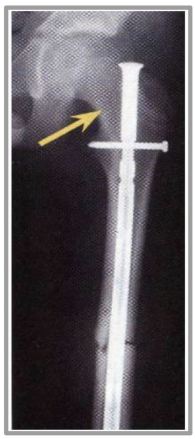
Salter harris 3

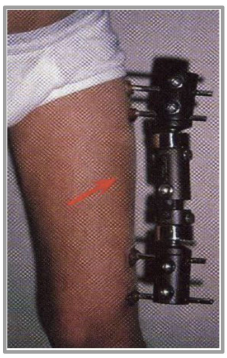
Salter harris 4

After:

In salter harris 3: two screws in the medial malleolus (you can see 2 heads and 2 tips).

We usually treat it with 2 screws in salter harris 4: one for epiphysis and the other for metaphysis.





Side notes

- Usually in pediatrics we remove the screws
- We always remove Plate and screws in pediatrics once it heals bc later on it will grow on top of it through which I will have to cut the cortex for removal
- potential area for pathological fracture. That's why we use wires which can be removed very easily. Plates and screws has limited use.
- In adults: upper limb we don't remove the screws cause there is a lot of nerves and blood vessels.
- In the lower limb we have to remove, cause after it heals, we will have something called shear force (breaks in the sites of the screws)
- Fellow level info: if the pt have some sort of handicap and walks on the upper limb then we have to remove it

Common Pediatric Fractures:

Clavicle Fractures:

Incidence:

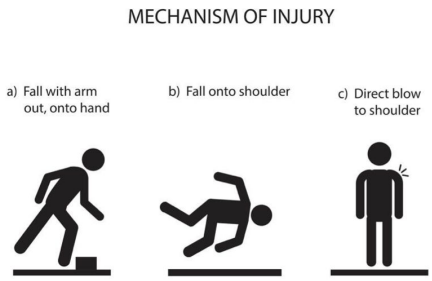
- 80% occur in the shaft
- The periosteal sleeve always remains in the anatomic position.
- Therefore, remodeling is ensured. It's thicker in the pediatric group.

- 8-15% of all pediatric fractures
- 0.5% of normal SVD
- 1.6% of breech deliveries Dr didn't mention it
- 90% of obstetric fractures

Mechanism of injury:

- Indirect (fall onto an outstretched hand)
- Direct
  - Has highest incidence of injury to the underlying:
  - Neurovascular (e.g Subclavian artery)
  - Pulmonary structures (Apex of the lung)

- Birth injury[1] may present with or without Erb's palsy



Going into 4th year like:

1-Shoulder dystocia: Shoulder dystocia is a birth injury (also called birth trauma) that happens when one or both of a baby's shoulders get stuck inside the mother's pelvis during labor and birth. In most cases of shoulder dystocia, babies are born safely, but in other cases it causes fractured clavicle and brachial plexus injury

Clinical evaluation:

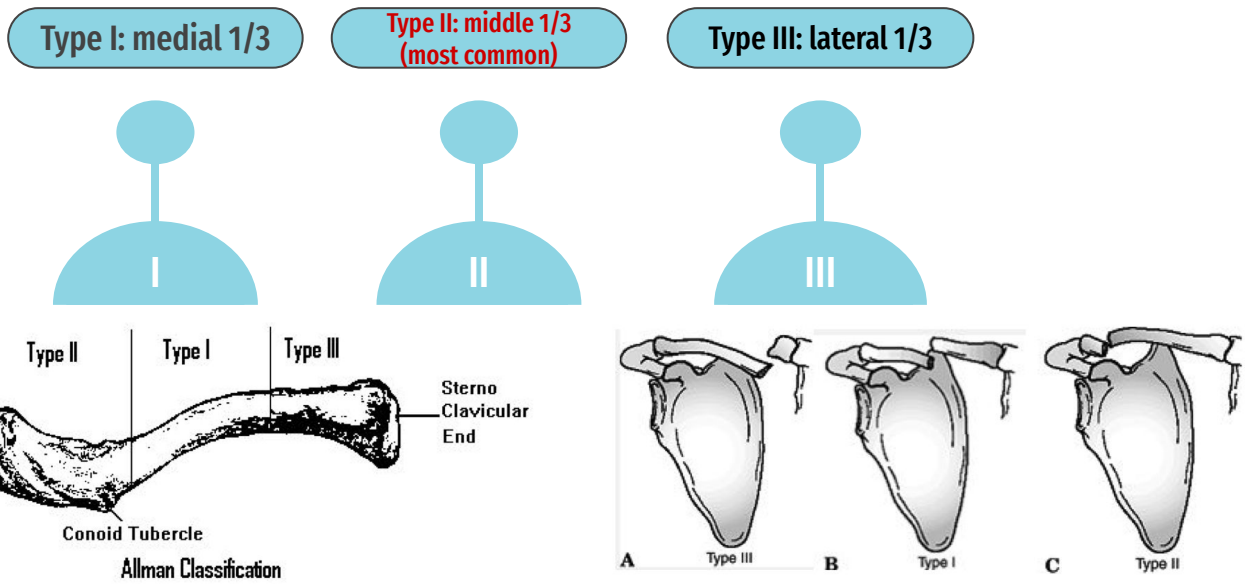
- **Look**: Ecchymosis, swelling, bruises, tenting of the skin.
- Feel:
  - Extreme tenderness at fracture site
  - As a palpable mass along the clavicle (as in displaced fracture) when presented late you can feel the callus.
  - You feel crepitus of the fracture (when lung is compromised) emphysema.

- **Special tests:** Must assess for any.
  - Neurovascular (N.V) injury examine the brachial plexus, subclavian vessels they run behind clavicle
  - Pulmonary injury If the fracture went downward.

Reading X-ray: AP view (438):

- Location: (medial, middle, lateral) ⅓, commonest middle ⅓ fracture site: Junction of middle & lateral ⅓
- Fracture type: transverse, segmental, comminuted, greenstick.
- Displacement in percentage %
- Open or closed: see air on XR it's skin on bone, you have to exclude open fracture.
- If pneumothorax you will see air in the x- ray.
- If subclavian and moved down it will cause vessel injury

Allman Classification[1] :



1- the pictures and context contradict each other, the Dr said most importantly is to know if it's (proximal, middle or distal) moreover the number of the type isn't important but if you want to know the right numbering it's the picture not the context.

Allman Classification[1] :

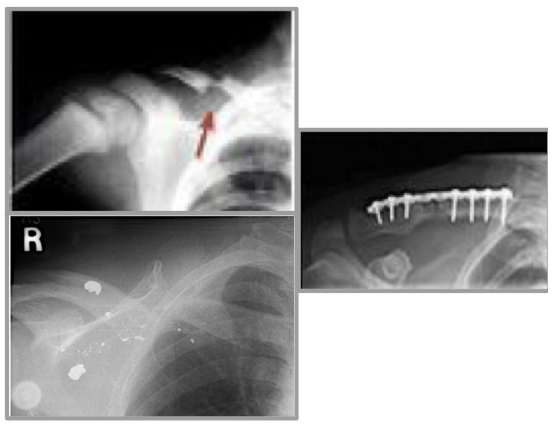
- **Newborn (< 28 days):** Figure-of-eight, untie in 1 week; because the sensory nervous system is not fully developed, Figure of eight we just tell the mother handle, Just leave it.
- **1m – 2y:** Figure-of-eight it just helps in realignment for 2 weeks
- **2 – 12y:** Figure-of-eight or sling for 2-4 weeks.

Figure of eight

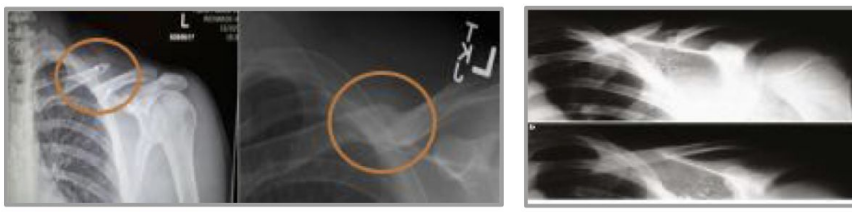
sling

Indication of Operative Treatment:

1. Open fractures (tinting[1] of the skin)
2. Neurovascular, pulmonary. compromise
3. Non-Union
4. Mal-Union



Clavicle Fracture Remodelling:



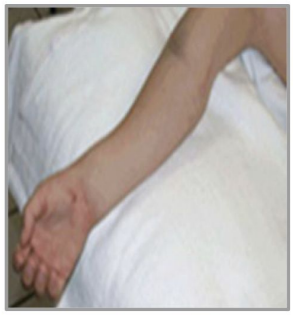
Complications:

From the Fracturerare	Iatrogenic (438)
-Malunion -Nonunion -Secondary from healing 1)Neurovascular compromise 2)Pulmonary injury	-Bad healed scar major indication not to do surgery, keloid in this area is very bad because we are **crossing** the fibers → heals very bad. -Dehiscence -Infection

1- Grayish or blue skin, also called pallor, is a result of a lack of oxygenated blood to the area that is discolored.

Supracondylar Humeral Fractures

Incidence:

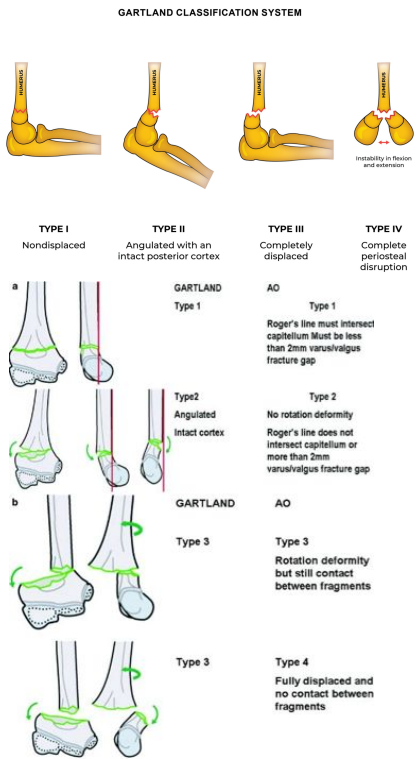


- 55-75% of all elbow fractures
- M:F ratio is 3:2
- Most commonly between 5-8 years, but can occur at any age
- The non-dominant side is most frequently injured (438)[1]

Clinical evaluation:

- Swollen, tender elbow with painful range of motion
- S-shaped angulation causing deformity
- Pucker sign (dimpling of skin anteriorly)[2]
- Neurovascular examination: asses brachial artery,
- median/AIN(most common), and radial nerve.

Mechanism of injury:



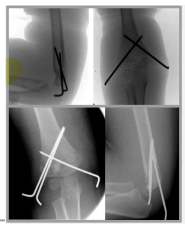
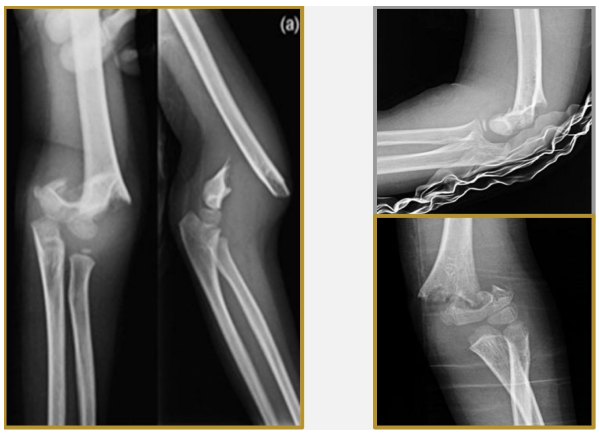
- Indirect >95%
- "Extension type" >95% when falling on stretched hand
- Direct
- Flexion type" <3% when falling on the elbow itself

Gartland's classification(important):

- **Type 1:** minimal or no displacement of the fracture. There is a normal anterior humeral line on the X-ray. Often only abnormality is abnormal fat pads.
- **Type 2:** with posterior displacement, the posterior cortex remains intact and with anterior displacement, the anterior cortex remains intact.
- **Type 3:** completely displaced with complete cortical disruption.2
- **Type 4:** The periosteum is completely torn and is the most unstable type of fracture (can only be diagnosed intraoperatively).

Modified Gartland's classification(important):

Type	Description	Treatment
I	Undisplaced	Casting
IIA	Displaced with angulation, intact posterior cortex	Casting or closed pinning
IIB	Displaced with angulation and rotation, intact posterior cortex	Closed pinning
IIIA	Completely displaced with no contact over posterior cortex	Closed or open pinning
Medial periosteal hinge intact, distal fragment goes posteromedially	Closed or open pinning	Closed or open pinning
IIIB	Completely displaced with no contact over posterior cortex Lateral periosteal hinge intact, distal fragment goes posterolaterally	Closed or open pinning
IV	Lack of periosteal hinge, presence of multidirectional instability	Closed or open pinning

Gartland	Treatment	Radiographs Class
TypeI	• Immobilization in a long arm cast (above elbow) or splint Type 1 • 60 to 90 degrees of flexion • 2 to 3 weeks	
TypeII	• Closed reduction, followed by casting or pinning if unstable or Type 2 severe swelling (with lateral wires to Slight avoid injuring the ulnar nerve)	
Type III	• Attempt closed reduction and pinning (crossed wires) • ORIF for unstable or open fractures, or those with neurovascular injury What is a common nerve injury caused by the crossed wires treatment in the case of a distal humeral fracture? Ulnar nerve injury (MCQ) 	 Extension type(3a). (Pucker sign) Flexion type(3b)

Complications:

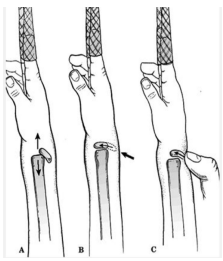
- Neurologic injury (7% to 10%)
  - Most are neuropraxias requiring no treatment (438)
  - Median and anterior interosseous nerves (most common)
  - Ulnar nerve (Iatrogenic)
- Vascular injury (0.5%)
  - Direct injury to the brachial artery or secondary to swelling
- Loss of motion (stiffness)
- Myositis ossificans
- Angular deformity (cubitus varus) caused by injured growth plate, reduction with rotation (malunion)
- Compartment syndrome

cubitus varus

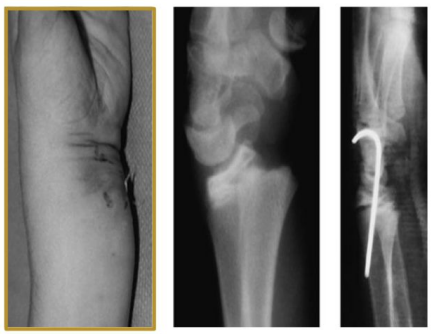
1) not based on scientific evidence
2) caused by bone displacement/angulation against the skin

Distal Radius Physeal Fractures:

Salter-Harris Classification:

Salter-Harris	Type I	Type II	Type III
x-rays			
treatment	• Closed reduction followed by long arm cast with the forearm pronated • 50% apposition with no angular or rotational deformity is treatment acceptable • Growth arrest can occur in 25% with repeated manipulations • Open reduction indication: ○ Irreducible or open fractures 		• Anatomic reduction is necessary • ORIF (open reduction and internal fixation) with smooth pins or screws

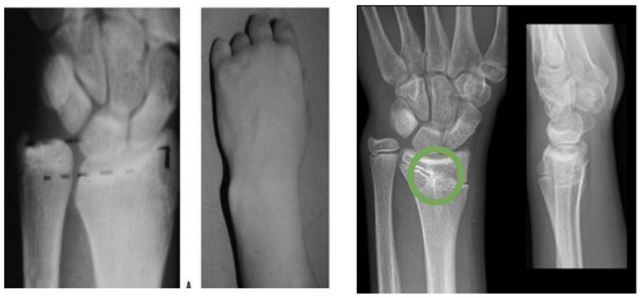

Use k-wires to hold the reduction if closed reduction is unstable



Salter-Harris Types IV and V are rare (in radius) and require ORIF

Complications of SHF:

- Physeal arrest
  - Shortening due to physeal bridge
  - Angular deformity
- Ulnar styloid nonunion
- Carpal tunnel syndrome



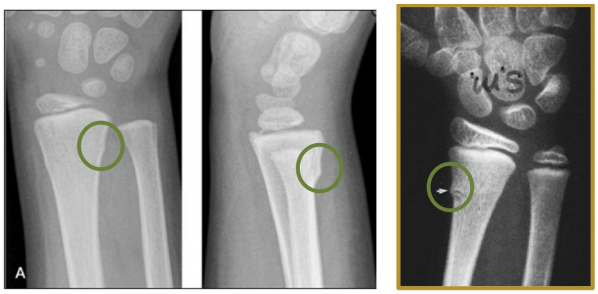
Distal Radius Metaphyseal Fractures:

Classification

- Biomechanical pattern (438)
- Displacement direction (438)
- Ulnar involvement (438)

Torus (only 1 cortex involved)

- Stable but very painful
- Immobilized 2-3 weeks with below elbow cast for pain relief

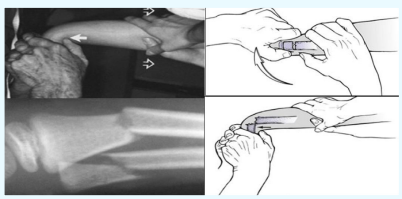


Incomplete/ greenstick fracture

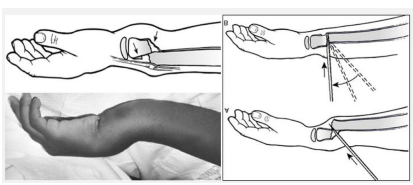
- Greater ability to remodel in the sagittal plane
- Closed reduction and above elbow cast with supinated forearm to relax the brachioradialis

Complete fracture

- Closed reduction
- Well molded long arm cast for **6 weeks**



Indications for percutaneous pinning without open reductions	Indications for ORIF
• loss of reduction • Excessive swelling • Floating elbow • Multiple manipulations	• Irreducible fracture • Open fracture • Compartment syndrome



Complications of DRMF:

- Malunion: residual angulation may result in loss of forearm rotation
- Nonunion: rare (since the metaphysis has good blood supply)
- Refracture with early splint removal and return to activity (before 6 weeks)
- Growth disturbance (overgrowth or undergrowth)
- Neurovascular injuries with extreme positions of immobilization

Femoral Shaft Fractures:

Incidence:

- Boys > girls
- 2 to 4 years of age, mid-adolescence
- In children younger than walking age,
  - 80% are caused by child abuse
- In adolescence, >90% due to RTA

Mechanism of injury:

- Direct trauma
  - RTA, falls, child abuse
- Indirect trauma
  - Rotational injury
- Pathologic fractures
  - Osteogenesis imperfecta, nonossifying fibroma, bone cysts,and tumors

Clinical evaluation:

- Pain, swelling, inability to ambulate, and variable gross deformity
- Careful neurovascular examination is essential
- Careful examination of the overlying soft tissues to rule out the possibility of an open fracture
- Check for knee and hip injuries (joint above and joint below)

AP and lateral views
Must include hip, knee joints

Classification:

Descriptive:

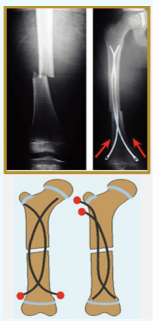
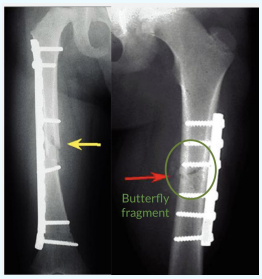
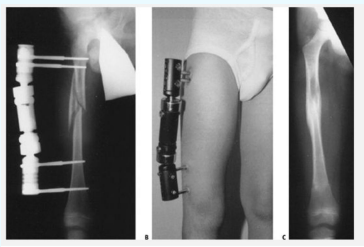
- Open or closed
- Fracture pattern: transverse, spiral, oblique, butterfly fragment
- Comminution
- Displacement

Anatomic:

- Subtrochanteric
- Shaft
- Supracondylar

Femoral Shaft Fractures cont..:

<6mo	• Pavlik harness (best treatment) • Traction and spica casting 
6mo-4yr	• CR and immediate casting (>95%) • Traction followed by spica casting if there is difficulty to maintain length and acceptable alignment 

4yr-12yr	4yr-12yr	4yr-12yr	12yr+
Flexible IMN (Transverse fracture) 	Bridge playing (Comminuted or spiral fracture) 	External Fixation: • Multiple injuries • Open fracture • Comminuted • Unstable patient 	Intramedullary fixation: either flexible or interlocked nails 

Complication:

- Malunion (remodeling will not correct rotational deformities)
- Nonunion: rare
- Muscle weakness
- Leg length discrepancy
  - Secondary to shortening or overgrowth
  - Overgrowth of 1.5 to 2.0 cm is common in 2-10 year of age
- Osteonecrosis with antegrade IMN <16 year (iatrogenic)

Quiz

Q1: Which of the following definitive fixation methods is most suitable for multiple trauma injury?

A. Casts
B. Plates
C. K-wires
D. Screws

Q1: Which of the following physis fractures is most commonly diagnosed late?

A. Salter-Harris type 1
B. Salter-Harris type 2
C. Salter-Harris type 3
D. Salter-Harris type 5

Q1: A 3-year-old patient presented with a painful, swollen left thigh and is unable to bare weight after falling from 3 meters high. Patient is stable, and X-ray shows left femoral shaft fracture. What's the management of choice for this patient?

A. Closed reduction and immediate casting
B. Flexible IMN
C. Bridge plating
D. External fixation

Q1: A 4-year-old boy presented with a closed fracture of the right femur. Reduction and fixation was done and the fracture healed in 8 weeks. What is the most likely complication that can happen to this patient?

A. Knee fraction contracture
B. Leg length discrepancy
C. Femoral condyle avascular necrosis
D. Chronic osteomyelitis

Q5: A 6-year-old boy presented with pain in his left arm after a fall. Physical examination shows a swollen, tender, and angulated elbow with skin dimpling. What is the most commonly injured nerve in this type of fracture?

A. Ulnar nerve
B. Leg length discrepancy
C. Femoral condyle avascular necrosis
D. Chronic osteomyelitis

1:B 2:D 3:A 4:B 5:B

Quiz

SAQ

438:

- List the indications for ORIF in distal radius metaphyseal fractures

439:

- What's the type of fracture, and mention 2 of its complications

Extra:

- Explain the three types of supracondylar fracture based on gartland classification
- Mention three methods of treatment for pediatric fracture & trauma

441:

- Mention 5 differences between pediatric and adult bone features

Q1	Irreducible fracture, Open fracture, Compartment syndrome
Q2	(Salter-Harris type 4). Physeal bridging, joint stiffness..
Q3	**Page 15**
Q4	Casting, K-wires, Screws, Elastic nail
Q5	**In the beginning of the lecture**

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441 Team

Common Foot & Ankle Problems

Dr. Abdulaziz Almaawi

Color Index:

- Main Text
- Important
- Old Notes
- 441 Notes
- Extra

Editing File

Objectives

- To understand the anatomy of Foot and Ankle.
- To get a concise idea on common Foot and Ankle disorders.
- To differentiate from simple disorders and serious ones.
- To learn about initial management and prognosis.

Common foot and ankle problems

1. Flat foot
   very common
   20-25% of population
2. Hallux Valgus
3. Plantar fasciitis
4. Ankle sprains & instability
5. Osteochondral lesion of talus
6. Diabetic foot
7. Charcot foot

Importance of Foot and Ankle

- Subject to most weight bearing (Loading) of the body 1 kg increase in trunk will Increase weight bearing by 8 kg in ankle and 4 kg in knee
- Important proprioception function.
- Their appearance or deformity is easily noticeable while the patient is standing
- Faulty or improper shoe wear can cause symptoms.
- With advancing age; deformity becomes more symptomatic.
- Their sensory role is very important.

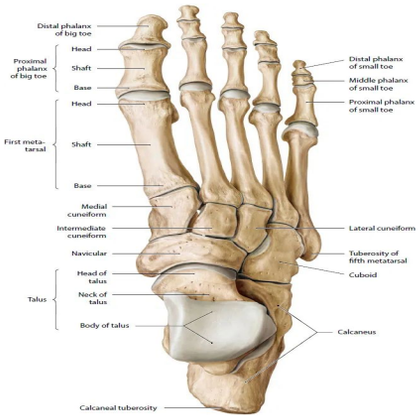
Anatomy of the Foot

- Body of Foot consist of (26 bones) and divided into:

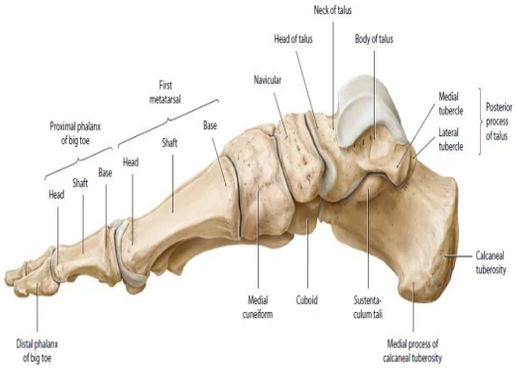
1. Fore Foot: Phalanges + Metatarsals
2. MidFoot : Navicular + Cuboid + 3 cuneiforms.
3. Hind(rear) foot: Talus(articulate with tibia to form ankle joint) + Calcaneus(articulate with talus to form subtalar joint)

Foot Skeleton

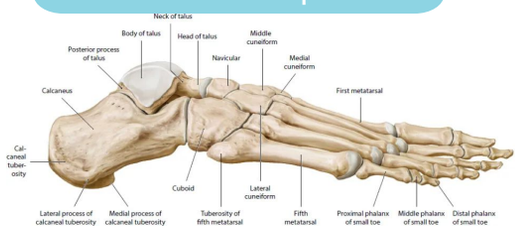
1- superior Aspect



2- Medial Aspect [1]



3- Lateral Aspect [2]



X-ray (standing) [3]

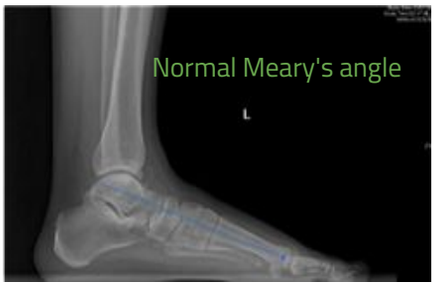
1- Talus and Calcaneus forms subtalar joint. We have more bones on the medial side than we have on the lateral side; that's because medial side gives stability while lateral gives flexibility.
2- Mid Foot forms the arch of the foot.
3- If siting shows no arch; Should be done in standing position since it's the function of the foot to bear weight . Standing to show any deformity

Flat Foot (Pes Planus)

- Reduction of longitudinal arches of the foot.
- Most cases are **developmental**[1]: i.e. arches do not develop normally[2].
- Usually is painless[3]
- Rarely acute flat = loss of the arch in case of an injury of the **posterior tibialis** tendon, unilateral
- Rigid flat (painfull) foot can be the result of tarsal coalition (fibrous or bony cross union between bones of the foot) "abnormal connection between bones in the back of the foot"
- Hind foot> valgus, Mid foot> reduction of longitudinal arch of the foot, Forefoot > abducted foot
- X-ray should be taken in standing position to show deformities

Cavus foot: longitudinal axis of talus is pointing upwards exaggerated arch

Normal X-ray



VS

Flat Foot Lateral weight bearing X-ray

Blue line: along the talus all the way to the metatarsal bone should be continuous with a 0 angle, up to 4 degree is considered normal. If the angle is >4 degree → considered flat foot.

IMPORTANT

Rigid or flexible flat foot

This table is important To differentiate between flexible and rigid flat foot

	**Normal**	**Flexible** most common (can be corrected without surgery)	**Rigid** Rare worse and can come at early age with early complication (needs surgery)
Alignment	Straight or minimal valgus	Extreme valgus	
Standing on tiptoes	The heel moves inward (from valgus to varus) the arch is still present	The heel moves inward (from valgus to varus), The deformity will be corrected the arch will reconstruct in flexible flat foot Hind foot> straight , Mid foot > reconstruction of arch, Fore foot > no abduction	Does not move inward (stay in valgus), Not correctable deformity (still valgus) the arch will remain absent in rigid flat foot
Subtalar motion (inversion/eversion)	Normal ROM	Normal ROM	**Reduced or absent**
Etiology	-	**Ligamentous** laxity (Present in almost 20% of the population)	**Tarsal coalition**[4]: Fibrous or bony union between bones of foot for fibrous we need to do MRI

1-Could be acquired in elderly due to posterior tibialis weakness.
2-Normally, the arch is formed within 4-6 years
3-80:20 rule: 20% of the population has it, 80% of them are asymptomatic,with time its become painful , and might have knee and back pain.
4-For tarsal coalition It has 2 type : 1-talocalcaneal and 2-calcaneonavicular

Too many toes sign

Flat foot:
- Hind foot: increased valgus
- Midfoot: lack of arch
- forefoot : abduction
Flexible tested via Beighton test which tests the ligament laxity

Flexible

Rigid

for example patient has flat foot so they have valgus, If they stand on their tippy toes and the valgus goes to straight and the arch is formed its flexible, if it can't go to normal it rigid

Management

always start with conservative:
1. Weight loss
2. Shoes
3. Orthotics (for arch support)
4. Activity modification
5. Physiotherapy

- Usually NO action is needed. (Asymptomatic)
- If Symptomatic **Always** start with conservative (5stpes)
- **Foot exercises**: Strengthen muscles is important but will <u>not correct deformity</u>.
- **Orthotics/insoles** الدعامات: Protective, <u>correct Malalignment</u>
- **Good shoes:** beneficial on the long run.
- **Surgery**:

1. Rigid[1] flat foot. Removal of coalition for the risk of osteoarthritis
2. Painful flat foot with complication. Like arthritis
3. Acute flat foot. Fix it and the arch will come back

Imp to know difference between flexible and rigid flat foot because the rigid you need to do surgery it's usually show up at 12-14 if you catch it early it will be easy to fix but if you leave it until advanced age the pt will get arthritis

Hallux Valgus

- Means <u>lateral deviation of big toe.</u> it's associated with flat foot
- Usually at the metatarsophalangeal joint.
- Most cases are painless. (If painful, would be due to shoe pressure on large toe or an inflamed bunion due to arthritis.)
- Often is associated with a **bunion**[2] (swelling and protrusion at the medial aspect of big toe[3])
- Severe HV interferes with shoe wear.
- Common at middle age and elderly[4] mainly females[5]
- Possible causes for hallux valgus: 1-Flat foot "if pt is not using the arch the toe will deviate laterally"
  2- Inherited bilateral 3- Shoe wear "heels" 4-Rheumatoid arthritis
- The pain isn't usually caused by the lateral deviation of the big toe, it's rather caused by the medial deviation of the 1st MTP joint.

1-Usually have gastrocnemius muscle tightness
2- Hallux valgus ≠bunion. **bunion** (swelling and protrusion at the medial aspect of big toe). Hallux valgus is the whole deviation of the big toe away from the central line. Sometimes people have a bunion without hallux valgus and we treat it by a bunionectomy; a surgery by which we remove the bunion. A bunion is not a growth instead it's exposure of the metatarsal head.
3-have many reasons most importantly the weight and the shoe.
4-Rheumatoid arthritis
5- Heels

IMPORTANT Hallux Valgus Measurement

Hallux valgus angle: (no. 2)

- Angle between line extending along 1st metatarsal and a line extending along proximal phalanx.
- **Normal**: <15° **Mild HV:** 16-25 °
- **Moderate HV**: 26-35°
- **Severe HV**: > 35°

Normal Foot

Hallux Valgus

1st intermetatarsal angle: (no. 4) most common and very important

- Angle between 1st metatarsal long axis and 2nd metatarsal
- Normal < 10°
- Moderate =12°
- Severe > 15°

Hallux interphalangeus angle: To assess if the deformity within the toe (no.1)

- Angle between long axis of proximal and distal phalanges
- Normal < 8°
- Severe >13°

Management

- If painless (most common) reassure. Never treat for cosmetic purpose
- If painful (interfering with walking), always start with conservative (5-steps)→
- Correct and suitable **shoe wear.** Wider shoes
- Avoidance of **tight shoes.**
- Protection to the bunions. (by cushioning) Using orthotics "like ring cover the toe" or using silicon
- **Surgery** is reserved for symptomatic and disturbing cases. (if conservatives fail to relieve pain for 3-6 months)
  - Surgery is annoying (hardware on skin).
  - Removal of bunion is not cosmetic, patient has to be symptomatic "pain in the joint, pain in the 2nd toe, pain because of shoe wear".
- Following surgery; patient has to continue proper shoe wear.

always start with conservative:

1. Weight loss
2. Shoes
3. Orthotics
4. Activity modification
5. Physiotherapy

Metatarsus went medially
Phalanx went laterally

X-ray Hallux Valgus **pre-op**

X-ray Hallux Valgus **post-op**

There a lot of procedures, but in basic steps:
1- Correct deviated metatarsal.
2- Excise the part of the bunion not all of it.
3- Release Adductor hallucis longus tendon as it is a deforming force.

Plantar Fasciitis

- Common disorder at middle age and elderly. affect about 90% of the population
- Insidious in onset; unilateral or bilateral. Vague pain at heel region. You wake up with sudden pain
- **Localized tenderness** to insertion of plantar fascia into calcaneum.
- Plain lateral X-ray of heel frequently shows calcaneal spur[1] (مسمار القدم), (prominence or ossification at the site of anterior calcaneum at plantar fascia insertion site)[2]
- Commonly associated with flat feet. Also associated with increase weight, walking barefoot on hard surfaces
- No visible heel swelling, no skin changes and no increase in local temperature.
- Inflammatory process is at site of pain; i.e. at **plantar fascia insertion into calcaneum**.
- Heel pain like **stabbing pain** when patient puts foot to the ground first step in the morning; and gets less after some walking.[3]

Some of the possible causes of Plantar Fasciitis:
- Weight (overweight)
- Bad shoes (Too soft and flexible)
- Walking barefoot
- Tightness of the Gastrocnemius
- Flat foot

Imaging

X-ray: Bilateral Calcaneal Spur (Early)	X-ray: Calcaneal Spur (Advanced)
	 The body compensates with formation of calcification

Management

"In many cases, patients will not be compliant with these steps"

Always start with conservative:
1. Weight loss
2. Shoes
3. Orthotics
4. Activity modification
5. Physiotherapy
6. Steroids
7. Surgery

- Most important; **proper shoes & weight reduction**
- NO easy or simple management is available.
- **Mainly conservative.**
- Includes **stretching exercises** to plantar fascia: active and passive.
- Use of **soft heel insoles** (Silicone) may be helpful.
- **Shock wave therapy (SWT)** الموجات الصدمية بالتنظير الخارجي may be effective[4] . Resistant cases only
- Local **steroid** injections are helpful sometimes. Very resistant cases
- Surgery (very rare):last solution Release of gastrocnemius muscle "**NO surgery for plantar fascia itself**"
- Surgery if there's nerve compression as carpal tunnel syndrome "median nerve

(435) If the first 5 steps of conservative didn't relieve the pain;
1. We do **MRI** to confirm diagnoses.
2. Give **steroid injection** if the steroidal injections didn't relieve the pain,
3. Do nerve conduction study to determine other causes. Other causes which although rare but give same symptoms of plantar fasciitis are: **tarsal tunnel syndrome** posterior to the medial malleolus compressing on lateral and medial plantar nerves (branches of posterior tibial nerve) and **nerve entrapment** due to large muscle in athletes.

1-No relation between the spike (spur) you see on x-ray and the pain. some patient have very large spikes with no pain and some have very severe pain with no spike, what we get from that is that it doesn't have to be removed to alleviate the pain.
2- It is an indication of inflammation for many years.
3-When we sleep or set for long periods our feet are in plantar flexion, making the achilles tendon short and when we step on the floor for the first time, we do dorsi flexion which stretches the achilles tendon stretching the plantar fascia with it causing pain.
4- To break down the thick tissue, which we don't know how it happens

Ankle Sprains

- One of the most common injuries.
- Usually occurs during sports activities, but may occur at home or street.
- The injury is **partial or complete ligament rupture.**

Clinical Features

- History of **twisting** injury. Most commonly inversion
- Pain, swelling and bruising at and around ankle. Swelling in look, tenderness in feel usually soft tissue not bone, sometimes can't walk
- No tenderness of lateral malleolus; but tenderness anterior, posterior or inferior to it i.e. over ligaments.
- Dorsiflexion and plantar flexion possible; but inversion and eversion very painful[1]
- Positive anterior drawer test. If both sides positive its a laxity disease if only one side its ligamentous
- X-Rays: **NO fracture**. You have to rule it out.

IMPORTANT

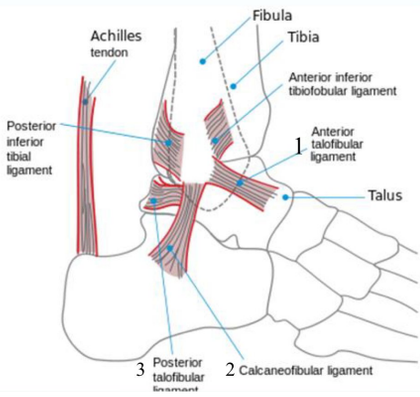
Lateral Collateral Ligament Complex

MRI is choice to determine which ligament is injured

Most commonly injured ligaments are :

1. **Anterior talofibular ligament (ATFL)** الرباط الكاحلي الشظوي الأمامي To test it do it with plantarflexion
2. **Calcaneofibular ligament (CFL)** الرباط العظمي الشظوي to test it do it with dorsiflexion
3. **Posterior talofibular ligament (PTFL)**.
4. **"Anterior drawer test is +",** Compare with normal side.

Most common cause of instability is Lateralization of the talus



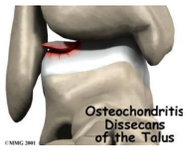
Management[2]

RICE: Rest, Ice, Compression, Elevation

- Apply **Back-slab splints** for few days جبيرة جبس خلفية if not able to weight bear.
- Might use protection with **brace** رباط ضاغط.
- **Early physiotherapy** العلاج الطبيعي and strengthening.(الا لو كان complete tear or unstablitiy)
- Mostly heal without surgery (75% of cases)
- **Surgery**: if physiotherapy fails and there is clear instability.or if there is complete tear
- PRICES: recent view = Protection [3] , relative Rest, Ice, Compression, Elevation and support
- After the Treatment, Do MRI to assess prognosis.
- In summary, We sometimes apply back-slab splint for few days (**10 days**), then start early physiotherapy immediately, but we try to minimize using this option due to complications of cast applying like stiffness and muscle weakness

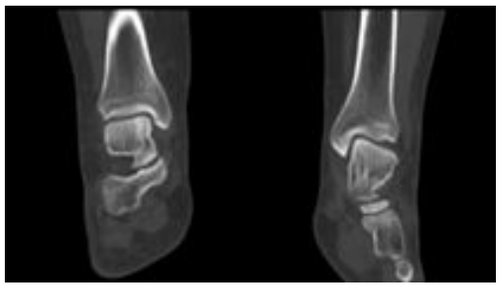
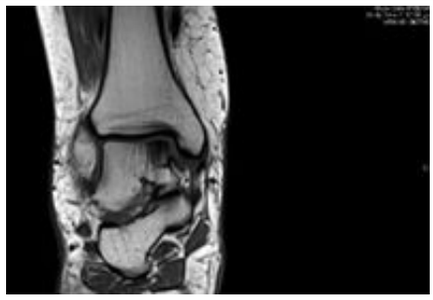
1- always compare, since it might be hyperlaxity
2-Always start with conservative, even if you are treating a professional athlete.
3-Splints are better than back slap casts because you can wear/remove splint anytime and splints allow dorsiflexion and plantar flexion movements

Osteochondral Defects of Talus[1] (OCD)



- Damage at localized areas of Talar articular cartilage.
- Lack of blood supply is often **post traumatic**, but occasionally No cause can be found.
- A local cartilage and varying depth of underneath bone are involved and may separate of main talus inside the ankle joint.
- Usually[2] **postero-medial part** of dome of talus. Bcs commonest sprains happen in inversion, which leads the tibia to impact the talus.
- Localized pain on weight bearing and even at rest may be present. Joint line tenderness especially in plantar- flexion + on & off swelling with walking a lot. المريض يقول رجلي تنتفخ مع المشي وترجع طبيعية " العلامة المهمة

Imaging

Plain AP X-ray: lesion is suspected	CT (bony injury)Coronal view: lesion highly suspected	MRI: lesion is confirmed
		
Bone is white highly sclerotic (marble-like) indicating loss of blood supply, Very fragile and easily broken.	**OCD you need to know** ➢ Most common area is postero-medial part of dome of talus ➢ Best investigation to detect it is MRI	MRI with contrast the bone is white, and the lesion is black, due to loss of blood supply. **Modality of choice**

Management[3]

For size :
1- If less than 1 cm very good prognosis
2- Above 1 cm high risk of Arthritis, bad prognosis

- **Depends on:**

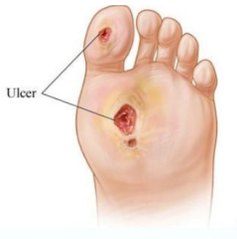
1. Symptoms: pain and recurrent swelling.
2. Size of OCD: large and Loose
3. Loose fragment (urgent surgery within few weeks "injury to cartilage *Calcaneum*")

- **Arthroscopic debridement**[4] تنضير المفصل of the lesion and drilling of its base. Better with minimally invasive + must be in certain site.
- **Fixation with headless screw** of large OCD with large bony part. For big pieces

1. Ankle injury resulting in loss of bone and cartilage in talus.
2. 50% of cases
3. doesn't heal by itself, since it's avascular
4. 90% of cases are managed by Arthroscopic debridement

Diabetic Foot

- **Neuropathy** (nerve damage): Long term diabetes or failure to control diabetes. Sign of poor control
- Numbness, tingling and reduced sensation of the feet.
- Associated **Decreased circulation** (neuropathy, calcification of vessels, CAD).
- May result in delayed healing, Infection, **Gangrene** and **Amputation**.
- How diabetes exactly causes neuropathy? By accumulation of sorbitol (sugar substitute) in nerves, that's happening in UNCONTROLLED DM



Care of Feet in Diabetes

Primary target: **Prevention:**

1. Blood sugar control. Best indicator H1AC should be less than 7
2. Daily self-inspection of feet is mandatory.
3. Member of the family or assistant should do it.
4. Regular inspections by healthcare personnel should be arranged.
5. A visit to a doctor should take place immediately whenever any complication occurs
6. If there's ulcer, orthopedic surgeon, general surgeon, plastic surgeon and vascular surgeon are working as a team to treat it

Surgery in Diabetic Foot

You have to make sure the blood supply is good before do the surgery

- Skilled care of wounds and ulcers in diabetic foot is **required**.
- **Wound debridement** تنضير الجرح, antibiotics and repeated dressing should be done.
- **Amputations**[1] rare may become necessary when there is **Gangrene**.
- Toe amputation or ray amputation, forefoot amputation, below or above knee amputation.

Charcot Foot[2]

- **Significant nerve damage to the foot leads to:**

1. The bones of the foot become weak.
2. Joints inflamed, swollen as if infected and lax.

- Walking on the foot leads to disintegration and collapse of the joints and **deformity:** such as Rocker-bottom deformity قدم روحاء مقوسة .

After long standing diabetes

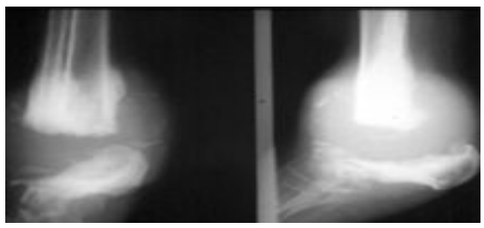
Etiology[3]

- Any disorder which lead to **Neuropathy**.
- There is decreased sensation and decreased ability to feel temperature, pain or trauma
- no pain

1- Our aim is to avoid amputation
2-bone degeneration due to loss of blood supply, in diabetics تتفتت العظام مثل الطبشورة
3-Doctor said that it's a complex mechanism that we are NOT required to know, but in general it's neuropathy at first, then nerves send signals that they require more blood to regain function, so this will cause hyper perfusion of blood to the foot and will increase bone resorption as well, leading to new bone growth.

Charcot Foot Clinical Picture

- **Look**: Foot is red or dusky in color. There's swelling in the area and deformity.
  Often mistaken by osteomyelitis. To differentiate we do **elevation test** we raise the leg up for 20 minutes, If persistent redness and swelling it is infection if redness and swelling subside it is Charcot foot.
- **Feel**: NO Pain or soreness, warmness of foot.
- **Move**: decreased ROM.
- **X-ray** changes are important to detect and interpret;
  - **Early: NO changes.**
  - Later: haziness, osteopenia, irregular joint destruction, subluxation or even dislocation.
- X-Rays: NO fracture. You have to rule it out.

Diabetic Foot	Advanced Case of Charcot
	
Osteolysis of callus and talus ,There's amputation of big toe	Talus and calcaneus almost gone

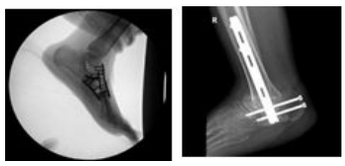
Diagnosis

Staging: development (little osteopenia), then destruction, then absorption then reconstruction.

- Good history[1] and clinical examination.
- Awareness.
- Exclusion of other causes which may give similar picture: like infection or tumor.
- MRI, bone scans can help.
- MRI/ CT to differentiate between Charcot and osteomyelitis

Management

In early stages avoid doing anything other than immobilization if you do the pt may end up with infection and osteomyelitis and amputation

Non-Surgical Management of Charcot Foot	Surgery in Charcot Foot Goal: 90 degree Foot	Amputation in Charcot foot
• **Immobilization** • **Custom Shoes and Bracing** we don't do cast because it might cause ulcers in a diabetic patient with neuropathy • **Activity modification**	Usually we wait for 1.5 or 2 years (because the bone is already متفتت that why not benefit)until reach last stage(by bone scan) then we do reconstruction of ankle or foot depending on area  **[we don't do surgery until blood sugar is controlled]**	• May be indicated as a last option. • Mainly when there is severe instability which cannot be controlled by surgery or orthosis تقويم العظام. • Also when surgery fails to achieve stability. • Presence of refractory infection increase the possibility of amputation.

1-Always ask about diabetes and whether it's controlled or not

439 SUMMARY

Flat foot

-Reduction in longitudinal arches of the foot,Painless.
-Injury of the posterior tibialis tendon causes acute flat foot.
-Hind foot:increase valgus, midfoot:lack of arch, forefoot:abduction.

	Flexible	Rigid
Alignment	Extreme vulgus	Extreme valgus
Standing on tiptoes	Valgus to varus	Not move
Subtalar motion	Normal	Reduce or absent
Etiology	Ligament laxity	Tarsal coalition

-Management:always start conservative,foot exercises,orthotics and good shoes.
-Surgery:Rigid foot, painful flat foot with complications or acute flat foot.

Hallux valgus

-Lateral deviation of big toe,usually at the metatarsal joint, associated with bunion,severe if interferes with shoe wear.
-Hallux valgus angle:between 1st metatarsal and proximal phalanx, normal <15
-1st intertarsal angle:between 1st metatarsal and 2nd metatarsal,normal <10
-Hallux interphalangeus: between proximal and distal phalanges,normal<8
-Management:suitable shoe wear,avoidance tight shoe wear and protection to the bunions.
-Surgery is reserved for symptomatic and distributing cases

Plantar fasciitis

-Localized tenderness to insertion of plantar fascia into calcaneum.
-Xray shows calcaneal spur at plantar fascia insertion site.
-stabbing heel pain when patient puts foot to the ground first step in the morning and gets less after some walking
-Management:mainly conservative, stretching exercises,soft heel insoles, shock wave therapy and local steroid injection.
-No surgery

Ankle sprains

-History of twisting injury, pain swelling and bruising around ankle,dorsiflexion and plantar flexion possible, but inversion and eversion very painful.
-Positive anterior drawer test.
-Xray: rule out fracture
-Most common injured ligaments are :anterior talofibular ligament and calcaneofibular ligament.
-Lateralization of the talus is the most common cause of instability.
-Management:
  Start with RICE
  apply back slab splint
  Might use brace for protection
  Early physiotherapy
-Surgery:if physiotherapy fails and there is clear instability.

Osteo-chondra Defect Of Talus

- damage at localized areas of talar articular cartilage,caused by lack of blood supply is often post traumatic,usually postero-medial part of dome of talus.
-Localized pain on weight bearing and even at rest may be present. Joint line tenderness especially in plantar-flexion + on & off swelling with walking a lot.
-Best investigation is MRI
-Management depends on: symptoms, size and loose fragment
 Arthroscopic debridement
 fixation with headless screw

Diabetic foot

caused by Neuropathy,by accumulation of the sorbitol
Numbness, tingling and reduce sensation of the feet
Prevention:Blood sugar control(H1AC < 7)
Surgery:wound and ulcer in diabetic foot require surgery ,wound debridement

Charcot foot

Significant nerve damage to foot leads to:weak bones,inflamed and swollen,caused by any disorder leads to neuropathy.
To differentiate between infection and charcot we do elevation test.
X Ray:early no changes,late:haziness, rule out fracture.
Management:
We start with non surgical: immobilization, custom shoes and bracing and activity modification, we wait for 1.5 or 2 years until reach last stage,then we do reconstruction of ankle (we don't do surgery until blood sugar is controlled), last choice is amputation.

Quiz

Q1: Which of the following movements is reduced in rigid pes planus?

- A: Plantarflexion/Dorsiflexion
- B: Inversion/Eversion
- C: Internal/External rotation
- D: All of the above

Q2: At which angle is Hallux Valgus considered moderate?

- A: ≤15°
- B: 16-25°
- C: 26-35°
- D: ≥36°

Q3: What's the modality of choice used to diagnose Osteochondral defects of the talus?

- A: U/S
- B: X-ray
- C: CT-scan
- D: MRI

Q4: Tightness in which of the following muscles is associated with Plantar Fasciitis?

- A: Soleus
- B: Gastrocnemius
- C: Posterior tibialis
- D: Flexor hallucis longus

1:B 2:C 3:D 4:B

SAQs

439 & 441 F1 & A:

1. Name Two tests used to differentiate between flexible and rigid flat foot?

   Standing on tiptoe
   Subtalar movement

2. What are the angles used to assess the severity of Hallux Va
   Hallux Valgus angle
   Intermetatarsal angle
   Hallux Interphalangeal angle

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Orthopedic 441 Team

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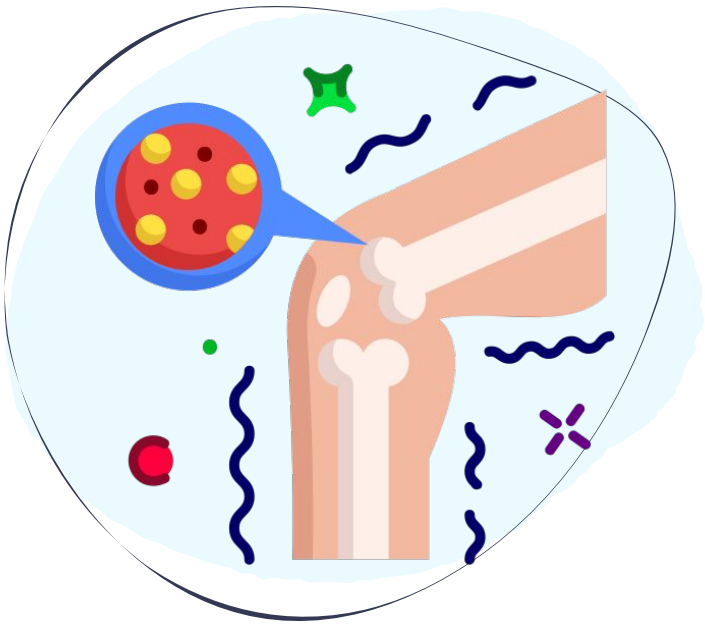
Editing File



Bone & Joint Infections

Dr. Motaz Alaqeel

Color Index:

- Main Text
- Important
- 441 Notes
- Old Notes
- Extra

Objectives

No objectives were provided for this lecture.

Resources

Osteomyelitis
By Osmosis

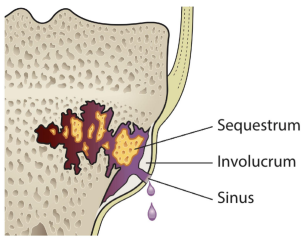
Septic Arthritis
By Osmosis

Osteomyelitis
By Orthobullets

Osteomyelitis

Overview

- Initial treatment → Based on presumed infection type → Clinical findings and symptoms, "you give broad spectrum antibiotics until you can identify the organism from the final culture".
- Definitive treatment → Based on final culture[1].
- Glycocalyx: Polysaccharides biofilm that forms commonly around artificial joints & foreign bodies, that will prevent the antibiotics from reaching, can't be treated without removal of glycocalyx, that's why in order to treat the infection you have to remove the foreign body, otherwise the infection will stay there.
  - Exopolysaccharide coating.
  - Envelops bacteria.
  - Enhances bacterial adherence to biologic implants.
- Terminology:
  - Sequestrum (dead bone): This happens when blood supply is cut off from area of bone due to infection[2].
  - Involucrum: New bone formed at site of infection and trapping a cavity of bone.



Bone Infection

- Osteomyelitis (OM): Infection of bone (the cortex) and bone marrow (inside the bone itself).
- Route of infection:
  - Direct inoculation (organism comes from outside) → Open fractures, cut wounds.
  - Blood-borne organisms (indirect) → Hematogenous.
- Determination of the offending organism is not a clinical diagnosis, but depends on deep culture which it is essential, "the deep culture is taken from deep bone not soft tissue, unless there was pus for example".

Classification

- Acute Hematogenous OM
- Subacute (2-6 weeks)
- Acute OM (Acute <2 weeks)
- Chronic OM (Chronic >6 weeks) Some sources define chronic osteomyelitis as having a duration ≥ 3 months.

1- To determine the organism + its sensitivity to antibiotics, to choose the appropriate antibiotic.
2- In the course of inflammatory response in an infected bone, there is an increased osteoclastic activity resulting in increased bone breakdown, as a result, a segment of bone may become separated and consequently loses its blood supply, therefore bacteria within the sequestrum are isolated from antibiotics carried in the bloodstream, which means the infection can't be properly treated.

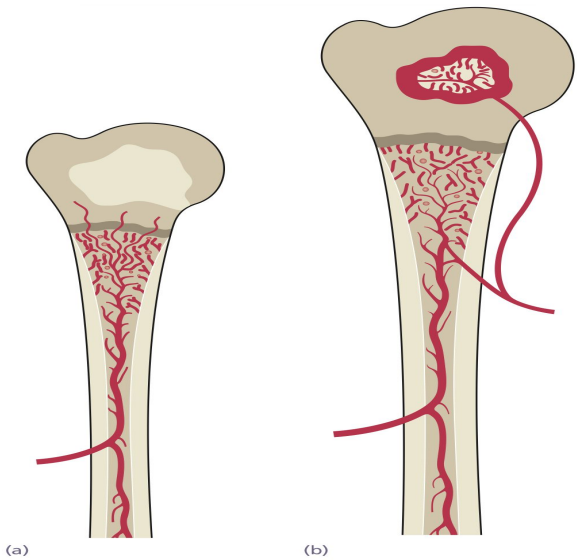
1 Acute Hematogenous Osteomyelitis

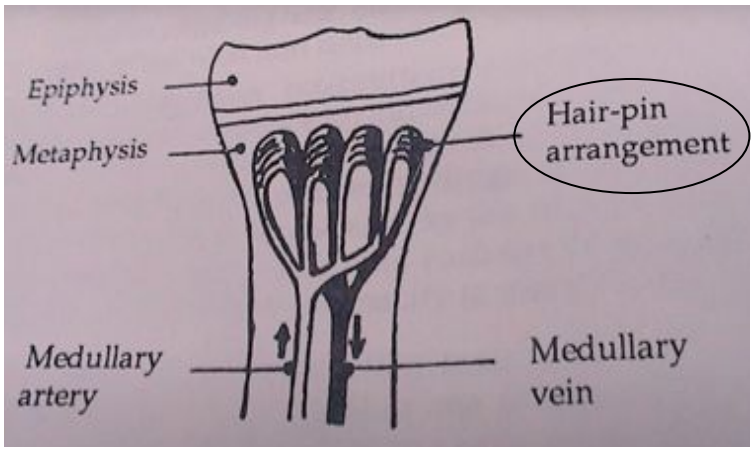
Clinical Features

- Caused by blood-borne organisms.
- More common in children.
  - Boys > girls.
  - Most common in long bone **metaphysis** or **epiphysis**.
  - Lower extremity >> upper extremity.
- Pain, malaise, restlessness.
- Loss of function of the involved extremity, fixed flexion deformity, contractures and stiffness.
- Soft tissue abscess and swelling, especially if the infection spread to soft tissues.

Etiology and Pathogenesis[1]

- Acute haematogenous osteomyelitis is mainly a disease of children. When adults are affected, it is usually because resistance is lowered.
- The bloodstream is invaded, perhaps from a minor skin abrasion, treading on a sharp object, an injection point, a boil, a septic tooth or in the newborn from an infected umbilical cord.
- In adults, the source of infection may be a urethral catheter, an indwelling arterial line or a contaminated needle and syringe.
- In children, the infection usually starts in the vascular metaphysis of a long bone, most often in the proximal tibia or in the distal or proximal ends of the femur.
- Predilection for this site has traditionally been attributed to the peculiar arrangement of the blood vessels in that area: the non-anastomosing terminal branches of the nutrient artery twist back in **hairpin loops** before entering the large network of sinusoidal veins; the relative vascular stasis and consequent lowered oxygen tension are believed to favour bacterial colonization.
- Epiphyseal and metaphyseal blood supply:
  - **In newborn infants (a)** some metaphyseal arterioles from the nutrient artery penetrate the physis and may carry infection directly from the metaphysis to the epiphysis.
  - **In older children (b)** the physis acts as a barrier and the developing epiphysis receives a separate blood supply from the epiphyseal and peri- articular blood vessels.





1- Extra explanation from Apley's & Solomon's System of Orthopedics and Trauma.

Diagnosis

Blood Tests

- ↑ **WBC** count mainly neutrophils.
- ↑ **ESR** not specific (better in total joint arthroplasty infection).
- **Blood cultures** → may be positive in children, must be done especially if patient is febrile.
- **C-reactive protein** monitor response to treatment.
  - **Most sensitive monitor of infection course in children**, respond very fast.
  - Short half-life, gives good indication if the treatment is working.
  - Dissipates "start to reduce" in about 1 week after effective treatment.

Imaging

Radiographic Changes

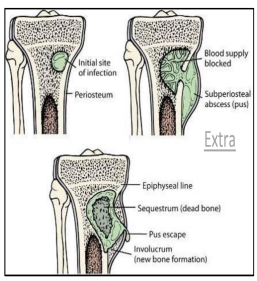
X-ray is the recommended initial imaging modality because it is inexpensive and can rule out differential diagnoses. from amboss

1. **Early:** Soft tissue swelling, no changes visible on X-ray.
2. **10-14 days:** Bone demineralization = Decreases bone density "2-3 weeks to see bone changes on X-ray".
3. **Later** (in chronic stage)**:**
   - **Sequestrum** (indication of OM): Dead bone with surrounding granulation tissue, appears as a dense sclerotic lesion.
   - **Involucrum**: Periosteal new bone formation, considered a periosteal reaction.

- Early X rays may not reveal any findings except soft tissue swelling at site of infection, bony changes take up to 10-14 days to show up, there is decreased blood supply due to it not being managed for a while which in turn causes osteopenia, (osteopenia may appear earlier not diagnostic).
- REMEMBER: The patient can present with symptoms without bony changes in the X ray.

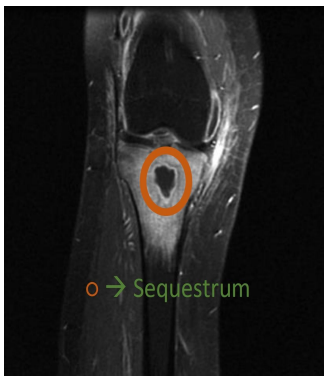
Soft tissue swelling

Sequestrum is the central island, not the border



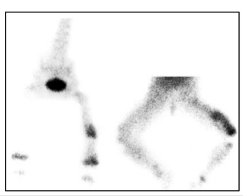
MRI

- MRI bone changes appear before X-ray "best tool for radiological diagnosis as it is sensitive and specific", but difficult in young children as they require general anesthesia".
- Shows changes in bone and bone marrow before plain films.
- Decreased T_1-weighted bone marrow signal intensity.
- **Increased post-gadolinium** (contrast) **fat-suppressed T_1-weighted signal intensity.**
- Increased T_2-weighted signal relative to normal fat.
- **Picture:** There's no activity in this area = dead, it doesn't have any blood supply, it could be a sequestrum or Brodie's abscess.
- MRI can reveal the periosteal reactions.



Nuclear Medicine Studies

- May help when not sure of the course or source of infection.
  - Regular bone scan: It will be positive, if you have anything that increases the activity, like: Infection, tumor, fracture, etc..
  - **Special bone scans for infections include:**
    1- Gallium bone scan **2-** WBCs labeled bone scan (most specific scan for bone infections)
  - Useful in delayed cases, when we want to check if there is more than one focus of infection "if we treated one place but the child is not improving".
  - **Picture:** Increase uptake in femur bone, due to increase activity, so it's possibly infection, tumor or fracture we don't know, the increase uptake in the bladder is because of the urine, it doesn't mean there's an infection.

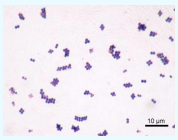


Treatment

1. Identify the organisms
2. Select appropriate antibiotics
3. Deliver antibiotics to the infected site
4. Halt tissue destruction

Empirical Treatment

- Is initiated **before definitive cultures become available**, based on patient's age and other circumstances.
- **NEVER start the antibiotic BEFORE obtain the culture**.

Newborn (up to 4 months of age) 	• The most common organisms:
	  ○ Staphylococcus aureus "most common organism in bone infection".
	  ○ Gram-negative bacilli.
	  ○ Group B streptococcus "after URTI".
	• Newborns: Crying, pain, swelling, stiffness (loss of function)
	  ○ May be afebrile "weak immunity هذه المشكلة إنهم بدون حرارة"
	  ○ 70% positive blood cultures.
	• Primary empirical therapy includes:
	  ○ Oxacillin (penicillin) specific for staph. for group (+).
	  ○ Plus 3rd generation cephalosporin for group (-).

Children (4 years of age or older)	• The most common organisms:
	  ○ Staphylococcus aureus.
	  ○ Group A streptococcus.
	  ○ Coliforms (uncommon).
	• Empirical treatment includes:
	  ○ Oxacillin or cefazolin (1st gen cephalosporins).
	  ○ If suspecting gram-negative organisms → 3rd generation cephalosporin.
	• **Haemophilus influenzae bone infections almost completely eliminated due to vaccination.**

Adults (21 years of age or older)	• **Organisms:**
	  ○ **Most common organism → S. aureus**.
	  ○ Wide variety of other organisms has been isolated.
	• Initial empirical therapy: Oxacillin or cefazolin.
	• Oxacillin is key because it is the most important in all age groups

Sickle Cell Anemia 	• **Salmonella** is a characteristic organism. • The primary treatment is **fluoroquinolones** (only in adults), **can't be given** to children. • Alternative treatment is 3rd generation cephalosporin.

Hemodialysis and IV Drug Abuser	• Common organisms:
	  ○ **Staphylococcus aureus**.
	  ○ S. epidermidis.
	  ○ Pseudomonas aeruginosa.
	• Treatment of choice is penicillinase-resistant synthetic penicillins (PRSPs)[1] + ciprofloxacin.
	• Alternative treatment is vancomycin **with** ciprofloxacin (allergy).

1- PRSPs include: Oxacillin, Cloxacillin, Dicloxacillin, Methicillin, and Nafcillin.

Operative Treatment

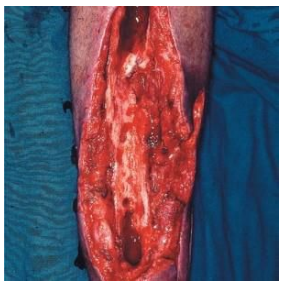
- Started after cultures. Starting treatment beforehand masks the results
- **Indications for operative intervention:**
  - Drainage of an abscess, if we're sure there's pus (as a result of our investigations), antibiotics can't treat the collection of abscess, the efficacy of antibiotics in pus may be limited by various factors like low pH, protein binding and degradation by bacterial enzymes.
  - Debridement of infected (dead) tissues to prevent further destruction, dead tissue can't respond to antibiotics so it's removed.
  - Refractory cases that show no improvement after non-operative treatment, we use nuclear scan for this case, "in case of presence of sequestrum it must be removed (sequestrectomy)".

2 Acute Osteomyelitis

Clinical Features

- Usually comes after open fracture or closed fracture with open reduction and internal fixation (iatrogenic) e.g. somebody have surgery then acute OM.
- Clinical findings similar to acute hematogenous OM "pain, swelling, loss of function, fever, stiffness and tenderness, so patient will be sick and febrile"
- **Most common offending organisms are:**
  - S. aureus.
  - P. aeruginosa.
  - Coliforms.
- **Complications:**

1- Septicemia and distant abscesses. 2- Septic arthritis 3- Chronic osteomyelitis.
4- Growth disturbance in skeletally immature and deformity 5- Pathological fracture



Treatment

- **Empirical therapy: Oxacillin** (for gram+ coverage include MSSA) **+ ciprofloxacin** (for gram- coverage include P. aeruginosa)
- **Treatment:**
  - Radical incision & drainage. Radical means you take everything out, bone, soft tissue and even the skin, "take everything that is dead until you reach the healthy tissue or it will not heal".
  - Removal of orthopedic hardware "device, screws, plates, nail or any implant" if necessary. External fixator, screws, these FB have no communication with blood so they must be removed as they act as a good media for colonization.
  - Rotational or free flaps (tissue transplant → muscles + soft tissue + skin) for open wounds if needed.

3 Chronic Osteomyelitis

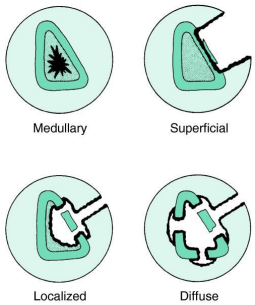
Clinical Features

- **Common in:**
  - Inappropriately treated acute OM.
  - Immunosuppressed, they will present with sinus discharging pus **without fever**.
  - Trauma.
  - Diabetics.
  - IV drug abusers.
- **Most common organisms:**
  - S. aureus.
  - Enterobacteriaceae (mainly with IV drug abuse).
  - P. aeruginosa.
- **Features:**
  - Skin and soft tissues involvement.
  - **Sinus tract** (discharging sinus) → May occasionally develop squamous cell carcinoma.
  - Periods of quiescence followed by acute exacerbations.
  - Presences of **sequestrum** (requires urgent intervention) "source of bacteria" antibiotics cannot reach it.

Anatomical Classification

4 Types:

- Medullary (can erode internal blood vessels and aid in forming sequestra).
- Superficial (only surface "part of cortex and soft tissue around it", because it is superficial we can eradicate it surgically).
- Localized (sinus formation with pus "draining" between outside and medulla).
- Diffuse (**the worst**, whole medullary canal, the sinus has destroyed the whole bone).



Diagnosis

- **Best test to identify the organisms → Operative sampling of deep specimens from multiple foci.**
- Nuclear medicine → Activity of the disease. E.g. Bone scan, gallium scan to tell if the lesion is old, remission, reactive and show us if infection is expanded and destruction more tissue.
- Anything that wasn't useful in acute is useful here: Plain X-rays, CT scans.

Treatment

- Empirical therapy **not indicated** because bone is dead. The main difference between acute and chronic is that in chronic there is no rush to start therapy because the damage has already occurred.
- Do culture and sampling followed by Surgical debridement followed by antimicrobial therapy.
- IV antibiotics must be **based on deep cultures not the superficial culture**.
- **Surgical debridement:**
  - Complete removal of compromised bone and soft tissue.
  - Hardware: **Most important factor**
    - Almost impossible to eliminate infection without removing implant.
    - Organisms grow in a glycocalyx (biofilm) around the foreign body "device" shields them from antibodies and antibiotics, we have to take out the glycocalyx.
  - Bone grafting and soft tissue coverage is often required, you use a flap (containing muscle, soft tissue and skin) rather than skin alone.
  - Amputations are still required in certain cases, diabetes mellitus, if huge area of bone and soft tissue is destructed.

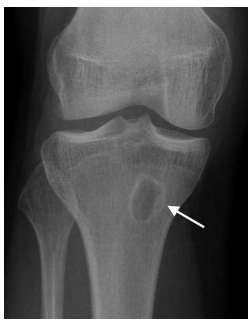
4 Subacute Osteomyelitis

Clinical Features

- **May occur in:**
  - Partially treated acute osteomyelitis.
  - Occasionally in fracture hematoma.
- Painful limp, but not severe like acute.
- **No** systemic and often no local signs or symptoms, the patient is afebrile and doesn't look sick.
- Signs and symptoms on plain radiograph.
- Can happen when the fracture hematoma gets infected.

Diagnosis

- Frequently normal WBC count and negative blood cultures.
- **Usually useful tests:** "any infection you need to rule out tumor"
  - ESR, not very specific could be elevated due to sore throat.
  - **Bone cultures**, only a small percentage have a positive result.
- **Radiographs:**
  - **Brodie's abscess** → Localized radiolucency seen in long bone metaphysis, difficult to differentiate from **Ewing's sarcoma**[1].
  - **Picture:** Cyst surrounded by sclerotic area (means it's well protected, not spreading), localized in the metaphysis and hypodense in the middle. Abscess very well circumscribed. An area of osteopenia surrounded by a thick cortex which is very well localized and won't spread.



Treatment

- Most commonly involves femur and tibia "lower limb".
- It can cross the physis "growth plate" even in older children.
- Metaphyseal Brodie's abscess → Surgical curettage (the only treatment for brodie's abscess).

5 Septic Arthritis

Clinical Features[2]

1- Acute onset.
2- Classical triad of fever, joint pain, and **restricted range of motion**.
3- Arthritis: Usually **monoarticular,** most commonly affected joint "**Knees**", Joints are swollen, red, warm, and painful.
- Those on corticosteroid treatment, may develop a 'silent' joint infection[3].

Diagnostics[2]

1- Arthrocentesis: For all patients with suspected septic arthritis.
- Synovial fluid analysis (SFA) in septic arthritis:
  - Appearance: Often yellow-green and turbid (nonspecific).
  - Cell count: ↑WBC count **(e.g. > 50,000/mm3)**, neutrophil (PMN) dominance of > 90%.
  - Glucose levels: Lower than blood glucose levels.
- Infection of the skin overlying the affected joint is an absolute contraindication to arthrocentesis due to the risk of introducing pathogens into the joint.

2- CBC, CRP, ESR: Leukocytosis and elevated inflammatory markers may be seen (nonspecific).
3- X-ray: Preferred initial imaging modality (prosthetic and native joints)
4- Ultrasonography: The most reliable method for revealing a joint effusion in early cases[3].

1- (Which is a differential diagnosis), you should do more investigations to differentiate between the tumor and abscess, bone scan will show a high uptake for both, you could open the area and send for culture and histopath to differentiate between infection and tumor.
2- Extra explanation from Amboss but important.
3- Extra explanation from Apley's & Solomon's System of Orthopedics and Trauma.

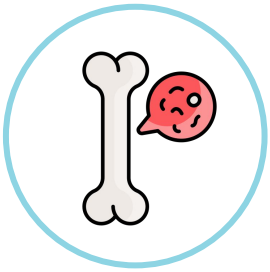
Most Common Site

Infants & children → Hip joint

Adults → Knee joint

Route of Infection

Hematogenous spread (indirect).



Extension of metaphyseal/diaphyseal osteomyelitis in children (direct) e.g. abscess → joint.

Complication of a diagnostic or therapeutic joint procedure "iatrogenic" (e.g. aspiration, steroid injection).

Metaphyseal Osteomyelitis Can Lead to Septic Arthritis in

1. **Proximal femur**
   Most common in this category
   (It may go up to the hip and cause septic arthritis)
2. **Proximal humerus**
   (goes to shoulder joint)
3. **Radial neck**
   (goes to elbow joint)
4. **Distal fibula**
   (goes to ankle joint)

Adults at Risk for Septic Arthritis are Those With

Rheumatoid Arthritis

- Tuberculosis → Most characteristic because of immunosuppression.
- S. aureus most common.

IV Drug Abuse

- Pseudomonas most characteristic.

Treatment

Empirical Therapy

- Prior to the availability of definitive cultures.
- Based on the patient's age and/or special circumstances.

Newborn (up to 3 months of age)	Children (3 months to 14 years of age)

• Most common organisms: Almost same as acute OM in newborn
  ○ S. aureus.
  ○ Group B streptococcus.
• Less common organisms:
  ○ Enterobacteriaceae.
  ○ Neisseria gonorrhoeae.
• 70% with adjacent bony involvement.
• Blood cultures are commonly positive.
• **Initial treatment: PRSP + 3rd generation cephalosporin**.
	• Most common organisms:
	  ○ **S. aureus**.
	  ○ Streptococcus pyogenes.
	  ○ S. pneumoniae.
	  ○ H. influenzae → **Markedly decreased with vaccination**.
	  ○ Gram-negative bacilli.
	• **Initial treatment: PRSP + 3rd generation cephalosporin.**
	• Alternative treatment: Vancomycin + 3rd generation cephalosporin.

Acute Monoarticular (one joint is involved) Septic Arthritis in Adults

- The most common organisms:
  1. S. aureus.
  2. Streptococci.
  3. Gram-negative bacilli e.g. pseudomonas.
- Antibiotic treatment: PRSP + 3rd generation cephalosporin.
- Alternative treatment: PRSP + ciprofloxacin.

Polyarticular Septic Arthritis	Chronic Monoarticular Septic Arthritis (cartilage damage)
Most common organisms: 1. Gonococci. 2. B. burgdorferi. 3. Acute rheumatic fever. 4. Viruses.	Most common organisms: 1. Brucella. Most common 2. Nocardia. 3. Mycobacteria. 4. Fungi.

Surgical Treatment

- **Mainstay of treatment:**
  - Surgical drainage → Open or arthroscopic تنظير المفصل "clean and wash with a scope but if it's difficult you can open"
  - Daily aspiration.
- Tuberculosis infections → Pannus (soft tissue granuloma due to inflammation and proliferation of the synovium leads to formation of pannus) → Similar to that of inflammatory arthritis (rheumatoid arthritis). Pannus "hypertrophied synovium" could cause stiffness & loss of function and has to be removed.
- Late sequelae of septic arthritis → Soft tissue contractures → May require soft tissue procedures (such as a quadricepsplasty "corrective surgical procedure on the quadriceps femoris muscle and tendon to release adhesions and improve mobility"), the quads become short and you can't extend the knees anymore so we have to do surgery to make it more elongate.

6 Infected Total Joint Arthroplasty

Diagnosis

- Most common pathogen:
  1. **S. epidermidis** — Most common with any foreign body
  2. **S. aureus**
  3. **Group B streptococcus**
- Worst type of infection in joints is the artificial joints because it is a foreign body that's why we remove them.
- If joint got infected, we have to take deep aspiration of knee, remove prosthesis, treat, put new prosthesis.
- ESR → Most sensitive but not specific.
- CRP may be helpful.
- **Culture of the hip aspirate → Sensitive and specific**.
- Preoperative skin ulcerations → ↑ **Risk**. If the patient shaves before the operation he will cause small skin openings and ulcerations that will be a good medium for infections, all shaving must be done in the OR to avoid scratches of the skin.
- Most accurate test → **Tissue culture**.
- History of **joint replacement surgery**, pain, fever and joint stiffness (if the patient has normal temperature it is **loose joint arthroplasty** not infected joint arthroplasty).

Prevention

Video explaining Laminar Flow (Air suction mechanism)

- Perioperative intravenous antibiotics is the most effective method for decreasing its incidence.
- Good operative technique.
- Laminar flow → Avoiding obstruction between the air source and the operative wound.
- Special "space suits", everything is sterile even the face area.
- Most patients with TJA **do not need** prophylactic antibiotics for dental procedures, **unless they have an infection/abscess** then antibiotics are given.
- Before TKA revision → Knee aspiration is important to rule out infection. The culture may be negative but it doesn't rule out an infection.

Treatment of TJA

Acute infections within 2-3 weeks of arthroplasty (prosthesis has metal part and plastic part)	• Prosthesis salvage → Stable prosthesis. • Exchange polyethylene (plastic part) components, you open the joint and keep the prosthesis and cover the surface of the tibia and femur with polyethylene (don't remove the prosthesis). • Synovectomy → Beneficial if synovium is infected.

Chronic TJA infections **>3** weeks of arthroplasty	• Implant and cement removal.
	• Staged exchange arthroplasty:
	  ○ Stage 1: After removal of prosthesis, put spacer (cement and antibiotic) and keep it there for 6 weeks, then do new culture.
	  ○ Stage 2: Put the new prosthesis.
	• Glycocalyx:
	  ○ Formed by polymicrobial organisms.
	  ○ Difficult infection control without removing prosthesis and vigorous debridement.
	• Helpful steps:
	  ○ Use of antibiotic-impregnated cement, antibiotics used are usually vancomycin tobramycin and gentamicin.
	  ○ Antibiotic spacers/beads.
	  ○ You could do both antibiotic-impregnated cement and antibiotic spacers/beads.

EXTRA

Osteomyelitis

- bone infection with progressive inflammatory destruction

Etiology
- most commonly caused by *S. aureus*
- mechanism of spread: hematogenous (most common) vs. direct-inoculation vs. contiguous focus
- risk factors: recent trauma/surgery, immunocompromised patients, DM, IV drug use, poor vascular supply, peripheral neuropathy

Clinical Features
- symptoms: pain and fever
- on exam: erythema, tenderness, edema common ± abscess/draining sinus tract; impaired function/WB

Diagnosis
- see Medical Imaging, MI23 and MI28
- workup may include: WBC and differential, ESR, CRP, blood culture, aspirate culture/bone biopsy

Table 7. Treatment of Osteomyelitis

Acute Osteomyelitis	Chronic Osteomyelitis
IV antibiotics 4-6 wk; started empirically and adjusted after obtaining blood and aspirate cultures	Surgical debridement
± surgery (I&D) for abscess or significant involvement	Antibiotics: both local (e.g. antibiotic beads) and systemic (IV)
± hardware removal (if present)	

Acute osteomyelitis is a medical emergency which requires an early diagnosis and appropriate antimicrobial and surgical treatment

Most commonly affected joints in descending order
knee → hip → elbow → ankle → sternoclavicular joint

Plain Film Findings in a Septic Joint
- Early (0-3 d): usually normal; may show soft-tissue swelling or joint space widening from localized edema
- Late (4-6 d): joint space narrowing and destruction of cartilage

Septic Joint

- joint infection with progressive destruction if left untreated

Etiology
- most commonly caused by *S. aureus* in adults
- consider coagulase-negative Staphylococcus in patients with prior joint replacement
- consider *N. gonorrhoeae* in sexually active adults, and newborns
- most common route of infection is hematogenous
- risk factors: young/elderly (age >80 yr), prosthetic joint, recent joint surgery, skin infection/ulcer, IV drug use, recent intra-articular corticosteroid injection, immunocompromised (cancer, DM, alcoholism, RA)

Clinical Features
- inability/refusal to bear weight, localized joint pain, erythema, warmth, swelling, pain on active and passive ROM, ± fever

Investigations
- x-ray (to rule out fracture, tumour, metabolic bone disease), ESR, CRP, WBC, blood cultures
- joint aspirate: cloudy yellow fluid, WBC >50,000 with >90% neutrophils, protein level >4.4 mg/dL, joint glucose level <60% blood glucose level, no crystals, positive Gram stain results
- listen for heart murmur (if concern for infective endocarditis, use Duke Criteria)

Treatment
- IV antibiotics, empiric therapy (based on age and risk factors), adjust following joint aspirate C&S results
- non-operative
  - therapeutic joint aspiration, serially if necessary
- operative
  - arthroscopic or open irrigation and drainage

Serial C-reactive protein (CRP) can be used to monitor response to therapy

Does This Adult Patient Have Septic Arthritis?
JAMA 2007;297(13):1478-1488
Purpose: To review the accuracy and precision of the clinical evaluation for the diagnosis of nongonococcal bacterial arthritis.
Methods: Review of 14 studies including 6242 patients of which 653 had positive synovial culture (gold standard diagnostic tool for septic arthritis).
Results: Age, diabetes mellitus, rheumatoid arthritis, joint surgery, hip or knee prosthesis, skin infection, and human immunodeficiency virus type 1 infection significantly increase the probability of septic arthritis. Joint pain, history of joint swelling, and fever are found in >50% of cases. The presence of increased WBC increases the likelihood ratio (for counts <25000/μL: LR, 0.32; 95% CI, 0.23-0.43; for counts ≥25000/μL: LR, 2.9; 95% CI, 2.5-3.4; for counts ≥100000/μL: LR, 28.0; 95% CI, 12.0-66.0). A polymorphonuclear cell count of ≥90% increases the LR of septic arthritis by 3.4, while a PMN cell count of <90% reduces the LR by 0.34.
Conclusions: Clinical findings may be used to identify patients with a monoarticular arthritis who may have septic arthritis. Laboratory findings from an arthrocentesis are also required and helpful prior to Gram stain and culture.

Quiz

Q1: An 8-year-old girl comes to the emergency department with her mother because of right ankle pain. Her mother states that the pain started two weeks earlier and subsequently became worse. She also reports fever for the last week, and pain around the joint. Physical examination shows the right ankle is erythematous, warm, swollen and tender to palpation over the lateral malleolus. Radiographs of the ankle show soft tissue edema and a large radiolucency in the distal fibula. Which of the following is the most likely organism?

A. Staphylococcus aureus

B. Group A streptococcus

C. Salmonella

D. E.coli

Q2: A 12-year-old girl comes to the emergency department with her father because of right wrist pain. She states that the pain began 17 days ago and has continued to worsen. She reports being ill with a fever and an upper respiratory illness prior to the onset of the wrist pain. Her temperature is 38.5°C (101.3°F), pulse is 92/min, respirations are 22/min, and blood pressure is 132/76 mm Hg. Physical examination shows the right wrist is erythematous, warm, swollen, and tender to palpation over the distal radius. Radiographs of the wrist show soft tissue edema and a large radiolucency in the distal radius. Which of the following is the most definitive treatment for this condition?

A. Excision of the bone lesion

B. IV antibiotics

C. Irrigation and debridement of the bone lesion

D. IV antibiotics with Irrigation and debridement of the bone lesion

1:A 2:D

Quiz

Q3: A 32-year old man comes to the emergency department because of fever, worsening pain, and impaired range of motion in his right knee for 3 days. He states that he often has to clear small nails or glass splinters from the rough pavement of his construction site before kneeling to do work-related tasks. He has never had a prosthetic implant, there is no family history of rheumatoid arthritis or gout, and that he does not have unexplained skin lesions. Examination shows there is an obvious effusion in his right knee, it is visibly red and swollen, and warm and painful to touch. He refuses to bend it. His left knee is unaffected. Which of the following would be the most helpful test in establishing a diagnosis?

A. CBC and cultures

B. Joint fluid analysis and culture

C. Right knee film

D. Polarizing microscopy of synovial fluid for crystals

Q4: A 72-year-old who underwent a total knee replacement 3 weeks ago, presents with increasing knee pain and swelling, with raised inflammatory markers. An aspiration of the joint cultures coagulase-negative staphylococcus. The next most appropriate step in management is?

A. Two-stage revision, with cement spacer plus intravenous antibiotics.

B. Intravenous antibiotics.

C. Open washout/debridement, polyethylene exchange and intravenous antibiotics.

D. Arthroscopic washout/debridement and intravenous antibiotics.

Q5: A 71-year-old patient presents with increasing knee pain 6 weeks after having a total knee replacement. Which of the following would be most reliable in the diagnosis of infection?

A. Erythrocyte sedimentation rate

B. C-reactive protein

C. Microscopy and culture of joint aspirate

D. Triple phase isotope bone scan.

E. MRI

3:B 4:C 5:C

Quiz

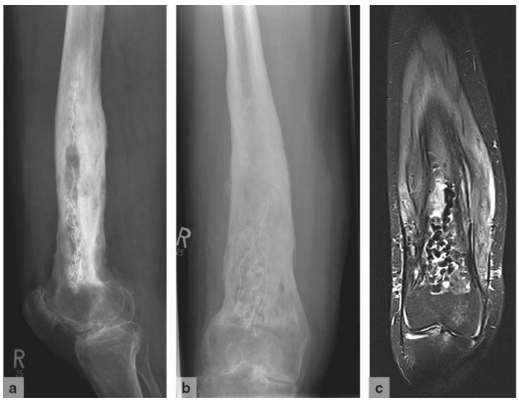
SAQs

441 & 439:

A 45 year old diabetic lady presents to your clinic complaining of a discharging wound in her foot. She reports having this issue for the past 3 years as it started soon after she had developed a calcaneal comminuted fracture.

439

441



1. What is the most likely diagnosis?
   1- Chronic osteomyelitis
   2- Subacute osteomyelitis

2. Name the 3 most possible organisms?
   1- S. Aureus
   2- Enterobacteriaceae
   3- P. Aeruginosa

3. How would you manage this patient (2 investigations & 3 modalities of treatment)?
   1- Nuclear scan
   2- Deep specimen culture
   3- IV antibiotics based on the culture
   4- Complete removal of compromised bone and soft tissue
   5- Hardware removal

Team Leader

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وفّقكم الله

7

Orthopedic 441 Team

MED441 KING SAUD UNIVERSITY

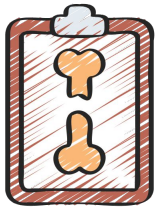
Editing File

Common Pediatric Lower Limb Disorders

Dr. Khalid Bakarman

Color Index:

- Main Text
- Important
- 441 Notes
- Old Notes
- Extra



Objectives

No objectives were found to this lecture.

Resources

Leg Aches

What are leg aches?

- Growing pain at age 2-12 years
- Benign with unknown cause
- **Does not cause** any functional disability or limping
- In 15-30% of normal children
- Females more than males
- Resolves spontaneously

Clinical Features

Leg aches is diagnosed by exclusion through history and screening examination

History

- Dull aching poorly localized pain
- Can be with or without activity
- At long bones of the lower limb (usually bilateral)
- At night (end of the day)
- Responds well to analgesia

Feature	Growing Pain	Serious Problem
History		
Long duration	Often	Usually not
Pain localized	No	Often
Pain bilateral	Often	Unusual
Alters activity	No	Often
Causes limp	No	Sometimes
General health	Good	May be ill
Physical Examination		
Tenderness	No	May show
Guarding	No	May show
Reduced range of motion	No	May show
Laboratory		
CBC	Normal	± Abnormal
ESR	Normal	± Abnormal
CRP	Normal	± Abnormal

Clinical Findings

- Long bone tenderness (non-specific affecting a large area)
  - ➔ leg aches usually don't have tenderness
- Normal joint motion

Differentials

It is **crucial to exclude serious problems** mainly tumors, common tumors that might cause leg aches are:

- Osteoid osteoma [1]
- Osteosarcoma [1]
- Ewing sarcoma
- Leukemia
- Sickle cell anemia
- Subacute osteomyelitis

Management

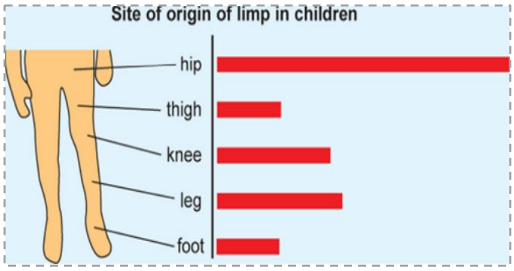
- Reassurance
- Symptomatic: analgesics, massage and bed rest

1- Usually present with night pain, limping, pathological fractures along side with the constitutional symptoms (B symptoms)

Limp

What is limping?

- Limping is used to describe an abnormal gait [1] due to pain, weakness or deformity.
- Most commonly caused by hip problems followed by leg problems



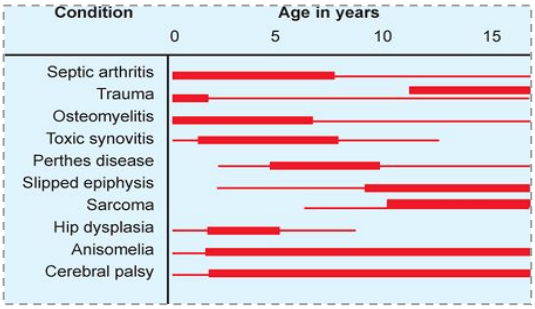
Diagnosis

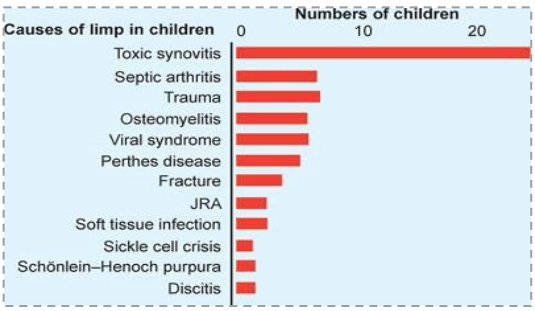
History

- You need to take detailed history specifically the age of onset
- Painful or painless?
  - ➔ Painful is usually unilateral and is caused by trauma, tumors or infections
  - ➔ Painless is usually bilateral and is caused by neuromuscular diseases or congenital

Examination

- You should have a good gait analysis (to determine the site)
  - ➔ Is it above the pelvis? (Back → scoliosis)
  - ➔ Is it below the pelvis? (Hips, knees, ankles and feet)
- Full Neurovascular examination

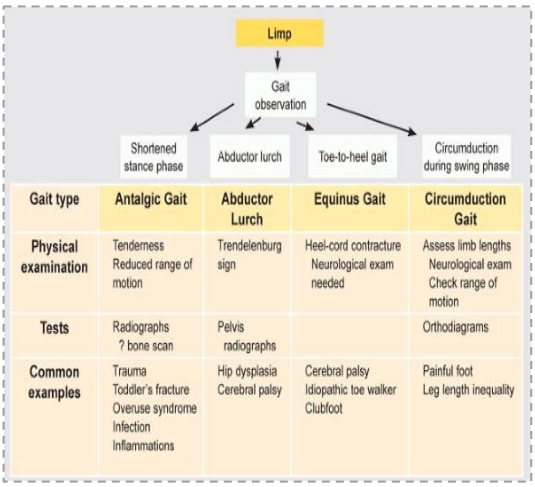




Types of Limps

(Click on the gait)

- We can divide the gait into painful gait (antalgic) and painless gait
- **Antalgic gait:** Abnormal pattern of walking due to pain that results in reduction in the stance phase. **(trauma, tumor, infection)**
- **Abductor Lurch** (AKA trendelenburg gait): Abnormal gait caused by weakness of the hip abductor muscles, leading to contralateral drooping of the pelvis while walking.
- **Equinus Gait:** Seen in children with cerebral palsy, calf spasticity leads to predominant plantar flexion of the ankle joint.
- **Circumduction gait:** Patients with a circumduction gait are unable to achieve adequate clearance for the foot to move through the swing phase on the affected side. To compensate, the patient abducts the thigh and swings the leg in a semi-circle to attain adequate clearance.



Gait type	Antalgic Gait	Abductor Lurch	Equinus Gait	Circumduction Gait
Physical examination	Tenderness Reduced range of motion	Trendelenburg sign	Heel-cord contracture Neurological exam needed	Assess limb lengths Neurological exam Check range of motion
Tests	Radiographs ? bone scan	Pelvis radiographs		Orthodiagrams
Common examples	Trauma Toddler's fracture Overuse syndrome Infection Inflammations	Hip dysplasia Cerebral palsy	Cerebral palsy Idiopathic toe walker Clubfoot	Painful foot Leg length inequality

Management

- Generalization cannot be made.
- Treatment of the cause

1- The normal gait cycle has two phases:
- The stance phase (60% of gait cycle): the phase during which the foot remains in contact with the ground.
- The swing phase (40% of gait cycle): the phase during which the foot is not in contact with the ground.
- If the patient has shorter stance phase >> painful limping. (ddx: trauma, tumor, infection)
- If the patient has normal stance phase but has limping >> painless limping. (ddx: congenital, syndromatic .. etc)

In-toeing and Out-toeing:

Terminologies

There are two words we need to differentiate from each other:

1. Version: is the normal variation of limb rotations
2. Torsion: describes the abnormal limb rotation (internal/ external)
   - ➔ It may be complex if there is compensatory torsion

- When a fetus is developing in the womb, the lower limbs initially point outward, then begin rotating inward around the seventh week. However, this rotation causes the toes to point towards each other. During the rest of fetal development, the legs gradually rotate laterally again. This lateral rotational growth continues slightly during childhood, but by the time of birth, the feet are approximately pointed straight forward. A small amount of rotation in infant legs is considered within the range of normal growth variation and is referred to as **version**. An abnormal amount of rotation is termed **torsion**.

Evaluation

- We usually start with:
  1. History
  2. Screening examination
  3. Rotational profile

History

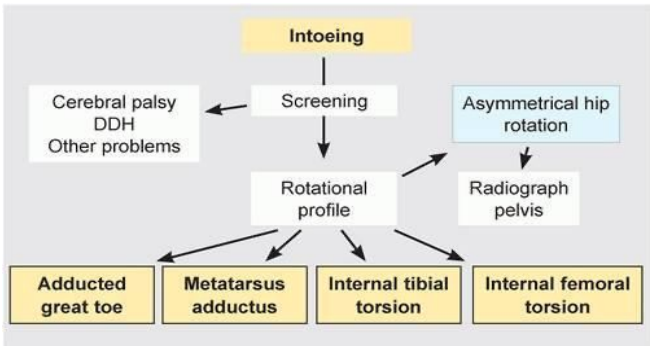
- Onset definitely not seen in 2-3 months old, it occurs after the child walks for few months
- Who noticed it?
- Progression (it is developmental deformity)
- Frequent falling, especially when they run
  - ➔ The main characteristic is that they fall a lot and when they run they fall even more b/c they can't control the rotational profile of their lower limbs.
- Runs with an "Egg-Beater" legs
- Sits in a "W" position
- Family history
- Unilateral vs. bilateral

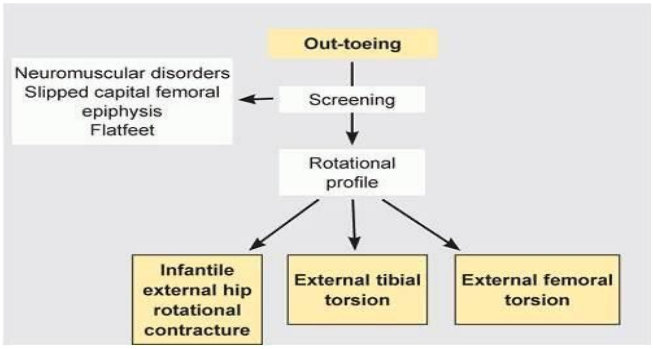
Screening

- We need to screen those patients from head to toe
- These conditions might be associated with neurological disorders such as spina bifida and cerebral palsy.

The figure on the right shows what is known as "W-shaped sitting", a sign to look for when examining such patients.

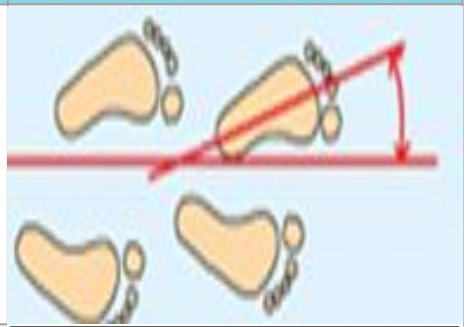
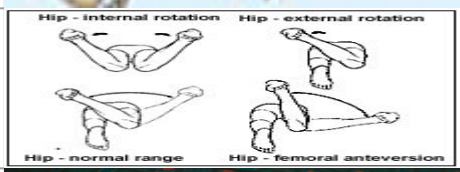
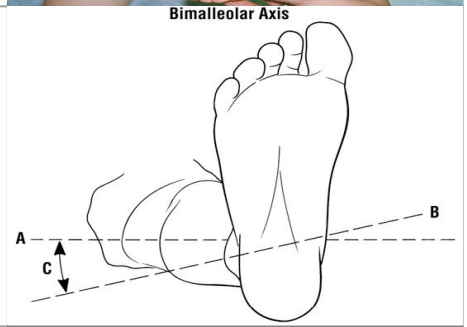
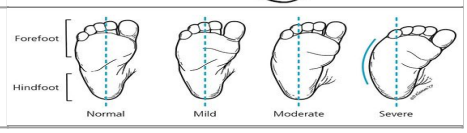
In-toeing and Out-toeing:





Rotational Profile

An assessment known as the rotational profile (also called the torsional profile) which involves taking six different measurements of the angles of the feet, legs, and hips when the child is in various positions and when walking or running. This allows for detection of isolated abnormal angles and facilitates identification of the cause of the rotational problem.

Rotational Profile		
Test	**Description**	**Image**
Foot propagation angle	Done to tell if there is in/out toeing, but it will not tell where it's coming from (source of deformity). Represents the angular difference between the axis of the foot with the direction in which the child is walking. **Normal range:** -10° to +15° → -ve = in-toeing → +ve = out-toeing[1]	
Hip rotation profile	Done for **femoral anteversion**, shows how much internal (N = 40-45°) & external (N = 45-50°) rotation can be done at the hip joint.	
Thigh-foot angle	Tests for **tibial torsion.** **Normal range:** 0 to -10°	
Bimalleolar axis	Tests for **tibial torsion.** - Normally the lateral malleolus tuberosity is posterior to the medial malleolus tuberosity in about 15 degrees, if it's at the same level or anterior then it's tibial torsion. - if the test yield 0 or - degrees then it's tibial torsion	
Heel bisector line	Tests for **forefoot adduction** (metatarsus adductus) Normal along 2nd toe (2nd web space)	

1- Let me be clear, we (the team) are absolutely not sure about the direction of the angle (Is the +ve considered outwards or inwards?), different sources provided different info, we asked the doctor but we got no response, so it's on you, the reader, you're our only hope. We believe in you, ask the doctor, do your own research, find the correct answer, for all of our sakes.

In-toeing and Out-toeing:

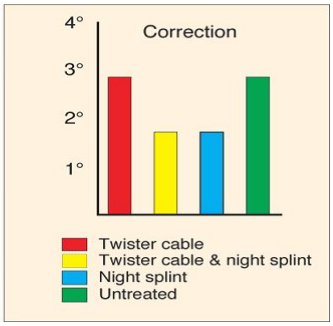
Management

- Establish correct diagnosis
- Parents education
- Allow spontaneous correction
- Control child's walking, sitting or sleeping is extremely difficult and frustrating
- Shoe wedges are ineffective
- Bracing with twister cables limits child's activities
- Night splints have no long-term benefits

Common Causes of Intoeing

Femoral Anteversion (In-toeing)	Description	• Femoral Anteversion is a common congenital condition caused by intrauterine positioning which lead to increased anteversion of the femoral neck relative to the femur with compensatory internal rotation of the femur. 
	Profile	• Increased hip IR with decreased hip ER
	Treatment	• Reassurance and sitting cross legged التربع • Surgery: subtrochanteric osteotomy (over 8y + significant deformity)
Internal Tibial Torsion (In-toeing) 	Description	• Internal Tibial Torsion is a common condition in children less than age 4 which typically presents with internal rotation of the tibia and an in-toeing gait.
	Profile	• Diagnosis is made clinically with a thigh-foot angle > 10 degrees of internal rotation in a patient with an in-toeing gait. (or by the Bimalleolar axis)
	Treatment	• Spontaneous improvement by age 4 • Surgery: supramalleolar osteotomy (over 8y + significant deformity)
Forefoot Adduction (In-toeing)	Description	• Medial deviation of the forefoot with normal hindfoot
	Profile	• Abnormal heel bisector
	Treatment	• Anteversion shoes (if older try physiotherapy)
Adducted Big Toe (In-toeing)	Description	• occurs in children after walking age and presents with varus deformity of the big toe
	Treatment	• Spontaneous improvement

Causes of Intoeing	
Condition	Key findings
Metatarsus Adductus	Medial deviation of the forefoot (abnormal heel bisector), normal hindfoot
Tibial Torsion	Thigh-foot angle > 10 degrees internal
Femoral Anteversion	Internal rotation >70 degrees and < 20 degrees of external rotation



Limb Length discrepancy (LLD):

Definition

An inequality in leg length can be either true or only apparent.

- True LLD: there us an actual difference between the length of either the femur or tibia
  Example: disruption of the growth plate due to trauma
- Apparent LLD[1] : is attributed to something other than the lengths of the femur or tibia
  Example: it has 3 types. Above the pelvis (scoliosis), below the pelvis (knee hyperextension), at the level of pelvis (pelvic muscles imbalance, stiffness)

Etiology

There are many causes of LLD, some are:

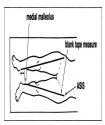
- Congenital as DDH
- Developmental as Blount's (Proximal Tibia Vara)
- Traumatic
- Infection
- Metabolic as Rickets
- Tumors
- Vascular lesions as ischemia or perthes
- Neurogenic as paralysis

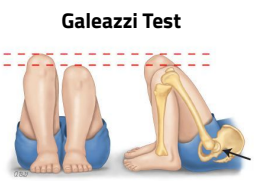
Category	Short	Long
Congenital	Aplasia Hypoplasia Hip dysplasia Clubfoot	Hyperplasia
Neurogenic	Paralysis Disuse	Sympathectomy
Vascular	Ischemia Perthes disease	AV fistular
Infection	Physeal injury	Stimulation
Tumors	Physeal involvement	Vascular lesions
Trauma	Physeal injury Malunion	Fracture stimulation Distraction

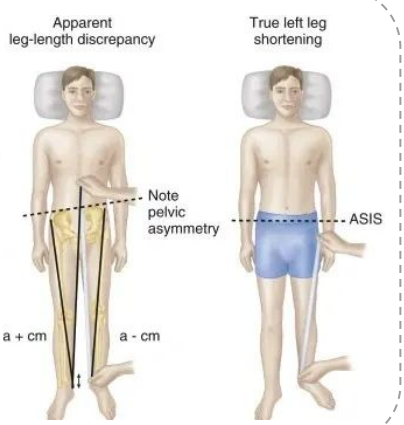
Clinical Picture

- Gait disturbance trendelenburg (if bilateral = waddling gait)
- Equinus deformity
  ➔ Shortening of one side will case an involuntary plantar flexion on the same side (it might become fixed it's persistent)
- Back and leg pain (due to unequal pressure on limbs)
- Secondary scoliosis

Evaluation







- Screening examination (block testing)
- Clinical measures of discrepancy
  ➔ True: from ASIS to medial malleolus (MM)
  ➔ Apparent: from umbilicus to MM
- Galeazzi test
- Imaging methods (centigram/scanogram)
  ➔ A type of x-ray or CT methods of determining LLD. A long film of the 2 limbs from hip to toes is taken, while a ruler is placed in the x-ray to measure the difference between the 2 limbs in length.

Management

Never operate if it is less than 2 cm

For shorter limb

- Shoe raise (< 2 cm)
- Bone lengthening (> 5 cm)
- Ilizarov principle (1 mm /day)

For longer limb

- Epiphysiodesis/ growth plate arrest (2-5 cm)
- Bone shortening (max 2 cm)

1- treat the underlying cause and it resolve spontaneously

Genu Varum & Genu Valgum:

Definition

- Genu varus (bow legs) or genu valgum (knocked knees) are different alignments of the bones at the knee joints.
- These alignments might be either physiological or pathological depending on the age of presentation

Physiological Presentation

★ Genu varum: birth - 2 years
★ Genu valgum: 2 - 5 years
★ Legs should start to become straight by the age of 5 to 7 years

Feature	Physiologic	Pathologic
Frequency	Common	Rare
Family history	Usually negative	May occur in family
Diet	Normal	May be abnormal
Health	Good	Other MS abnormalities
Onset	Second year for bowing Third year knock-knees	Out of normal sequence Often progressive
Effect of growth	Follows normal pattern	Variable
Height	Normal	Less than 5th percentile
Symmetry	Symmetrical	Symmetrical or asym
Severity	Mild to moderate	Often beyond ±2 SD

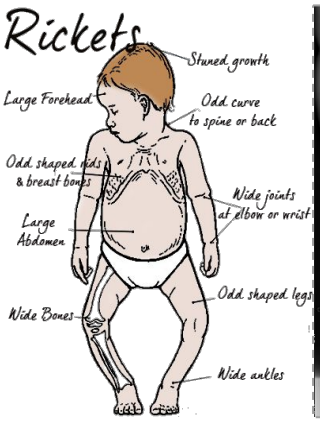
Etiology

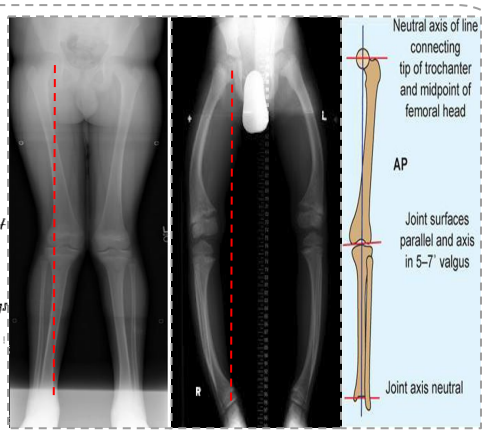
- So, if a child presented with genu varum or valgus different from his physiological alignment at his age, we should investigate him for pathological causes as shown on the table on the right.
- Example: a 3 year old child presented with genu varus, bluish sclera, progressive hearing loss and a history of fractures. X ray showed generalized osteopenia and genu varus.
  Diagnosis: osteogenesis imperfecta

Cause	Genu Valgum	Genu Varum
Congenital	Fibular hemimelia	Tibial hemimelia
Dysplasia	Osteochondrodysplasias	Osteochondro-dysplasias
Developmental	Knock-knee >2 SD	Bowing >2 SD Tibia vara
Trauma	Overgrowth Partial physeal arrest	Partial physeal arrest
Metabolic	Rickets	Rickets
Osteopenic	Osteogenesis imperfecta	
Infection	Growth plate injury	Growth plate injury
Arthritis	Rheumatoid arthritis knee	

Evaluation

- Detailed history
- Examination
  - ➔ Signs of rickets
- Lab
  - ➔ To exclude metabolic causes
- Imaging
  - ➔ Centigram/ scanogram
  - ➔ Rickets: widend growth plates
- **Complications:** early osteoarthritis[1]

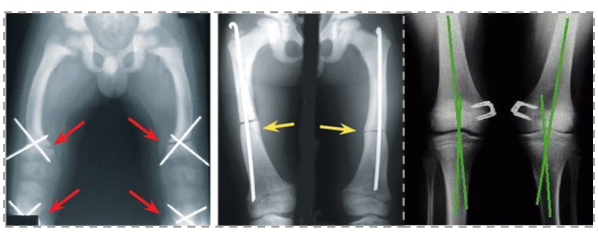




Management

Treat the underlying the cause in pathological and observe in physiological

- Nonoperative
- Epiphysiodesis (temporary vs permanent)
  - ➔ Arrest the growth plate
- Corrective osteotomies (definitive way)



1- If not treated, due to unequal weight distribution

Proximal Tibia Vara (Blount's)

Definition

- Also called blount disease, it is the damage of the proximal medial tibial growth plate (excessive genu varus) due to an **unknown cause**

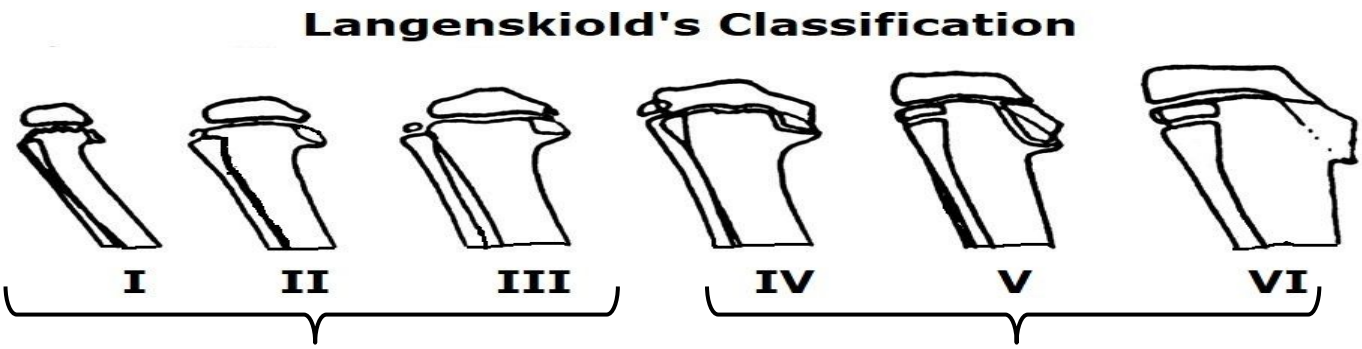
Risk Factors:

- Dark skin
- Overweight

Types:

1. **Infantile**: <3 years usually bilateral and in early walkers
2. **Juvenile**: 3-10 years of age
3. **Adolescent**: >10 years of age, usually unilateral and severe

Classification



Spontaneous improvement

Epiphysis bridges into metaphysis

Evaluation

- In severe or recurrent cases, MRI is mandatory
- On x ray there will be metaphyseal beaking (red arrow)

Metaphyseal-diaphyseal angle (Drennan):

- >16° → Abnormal
- 11-16° → Close observation
- <10° → high chance of resolution

Treatment

Surgical treatment is the definitive choice

Tibial osteotomy

Clubfoot (Talipes Equinovarus)

Normal Foot

- **Stable**: for supporting the body weight in standing
- **Resilient**: for walking and running
- **Mobile**: to accommodate variations of surface
- **Cosmetic**



Etiology

- **Postural**: intrauterine positioning (full correctable)
- **Idiopathic**: CTEV (congenital talipes equinovarus)
- **Secondary**: Spina bifida, myelomeningocele, MSK diseases

Exclusion

Exclude the following to diagnose CTEV

EXCLUDE

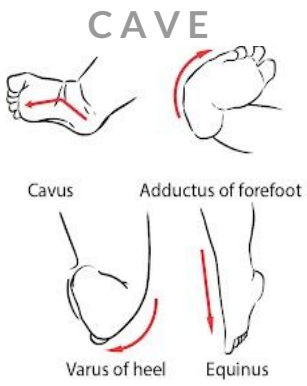
- Neurological lesions such as spina bifida (exclude through an X ray)
- Other abnormalities that explain the deformity such as: Arthrogryposis[1] and myelodysplasia[2]
- Presence of concomitant congenital anomaly such as: proximal femoral focal deficiency[3]
- Syndromatic clubfoot such as Larsen's syndrome[4] and amniotic band syndrome[5]

Deformity (CAVE)

Forefoot
- Adduction

Midfoot
- Cavus

Hindfoot
- Equinus: (Ankle joint)
  - Plantar flexion w/ limited dorsiflexion
- Varus: (Subtalar joint)



Clinical Examination

Check image on top right

- Short achilles tendon
- High and small heels
- No creases behind heel
- Abnormal crease in middle of the foot
- Foot is smaller unilaterally
- Callosities at abnormal pressure points
- Internal torsion of the leg
- Calf muscle wasting
- Deformities don't prevent walking

1- Congenital contracture in two or more areas of the body
2- Are a group of cancers in which immature blood cells in the bone marrow do not mature. EquinoVarus is the most common foot deformity in children with Myelodysplasia
3- A rare, non-hereditary birth defect that affects the pelvis, particularly the hip bone, and the proximal femur. The disorder may affect one side or both, with the hip being deformed and the leg shortened.
4- A disorder of the development of the bones. Include clubfoot and numerous joint dislocations at birth with a distinctive appearance of the face & square-shape finger tips
5- A a rare condition caused by strands of the amniotic sac that separate and entangle digits, limbs, or other parts of the fetus.

Clubfoot (Talipes Equinovarus)

Management

- The **goal of treatment** is to obtain a foot that is plantigrade, functional, painless, and stable over time. A cosmetically pleasing appearance is also an important goal sought by surgeon and family

1 Manipulation and Serial Casting

- Cast must be changed weekly for 6-8 weeks
- Valid up to 12 months of age as soft tissue becomes tighter

1. Ponseti Technique

2. Dennis Brown Splint

- Maintain the correction done by the cast (mandatory)
- Paced 23 hrs/day for 3 months then only during sleep for 3-4 years
- Watch and avoid recurrence till 9 years old
- We must stop if the deformity isn't improving or pressure ulcers were formed

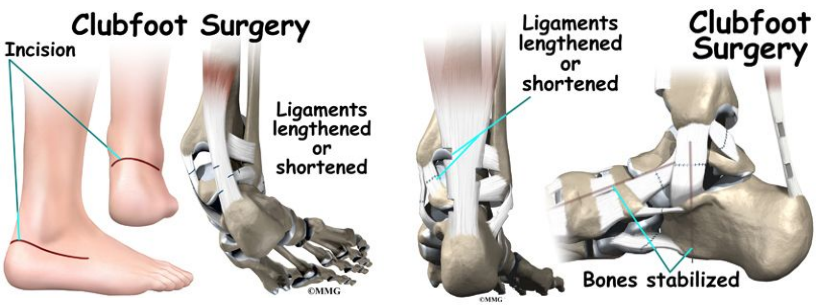
2 Surgery

Indications

- Late presentation (>12 m)
- Complementary to conservative treatment
- Failure of conservative treatment(>9 m)
- Residual deformities after conservative treatment
- Recurrence after conservative treatment

Types

- Soft tissue [1] (9-12 months)
- Bony [2] (3-4 years)
- Salvage [3] (>10 years)



1- lengthening of soft tissue and tendons
2- Wedge osteotomy: wedge removed of calcaneus
3- If severe and rigid arthrodesis

Cerebral Palsy LL Deformities

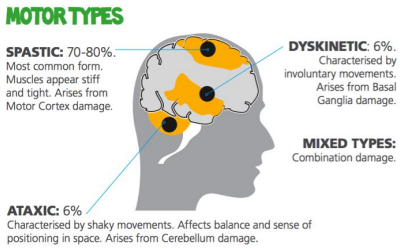
Definition

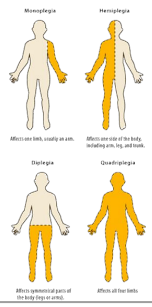
A non-progressive brain insult that occurs during the perinatal period. A deformity might results due to skeletal muscles imbalance that affects joints movement. It might be associated with:

- **Mental retardation**: with variable degrees
- **Hydrocephalus and V.P shunt**
- **Convulsion**

Classification

Physiological Classification	Topographic Classification
• Spastic • Ataxia • Athetosis • Rigidity • Mixed	• Monoplegia (one limb) • Diplegia (two limbs) • Paraplegia (Both lower limbs) • Hemiplegia (One side is affected) • Triplegia (three limbs) • Quadriplegia (All limbs)





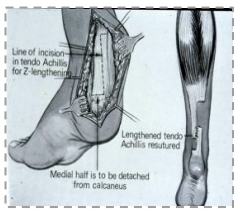
Examination

Hip	• Flexion: fixed flexion - Thomas test • Adduction • Internal rotation	**Ankle**	• Equinus • Varus/ valgus • Achilles tendon shortening
Knee	• Flexion: popliteal angle	**Gait**	• In-toeing • Scissoring tight hip adductors • Crouch

Management

Multidisciplinary approach guided by pediatric neurology

- Physiotherapy for ROM and gait training (most integral part)
- Social/ governmental aid
- Orthotic to maintain correction and aid in gait



Indications of surgery

- Severe contractures preventing physiotherapy
- Perineal hygiene (severe hip adduction)
- Help non-walkers sit comfortably
- To prevent neuropathic ulcers and dislocations

Surgical options

1. **Tendon elongation**
2. **Tendon transfer**
3. **Tenotomy**
4. **Neurectomy**
5. **Bone surgery, osteotomy/ fusion**

Team Leader

Abdulrahman Alroqi

Done by

Mohammed Alwahibi

وفّقكم الله

8

Orthopedic 441 Team

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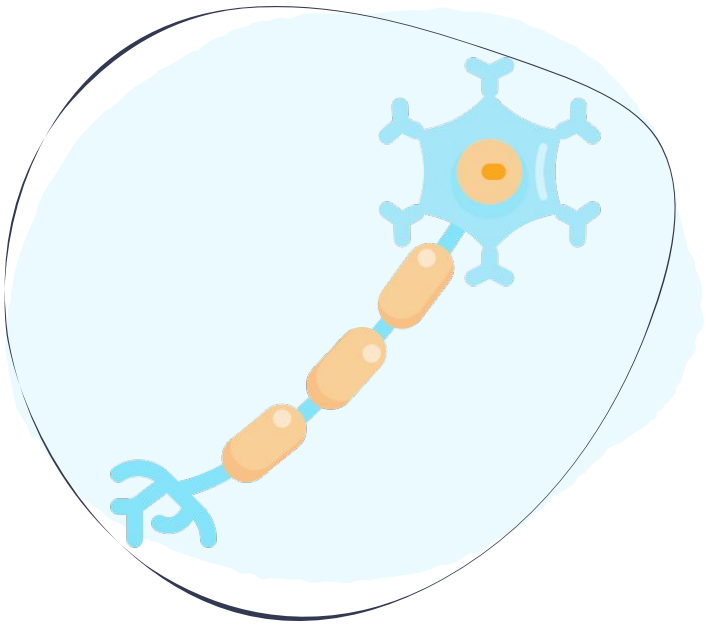
Editing File



Common Peripheral Nerve Problems & Injuries

Dr. Abdulrahman Alaseem

Color Index:

- Main Text
- Important
- 441 Notes
- Old Notes
- Extra

Objectives

No objectives were provided for this lecture.

Resources

Upper Extremity Nerve Injuries

By Dirty Medicine

Nerve Damage and Regeneration

By Armando Hasudungan

Lower Extremity Nerve Injuries

By Dirty Medicine
Our topics starts at (12:50)

Nerve Injury | Nerve Damage

By The Young Orthopod

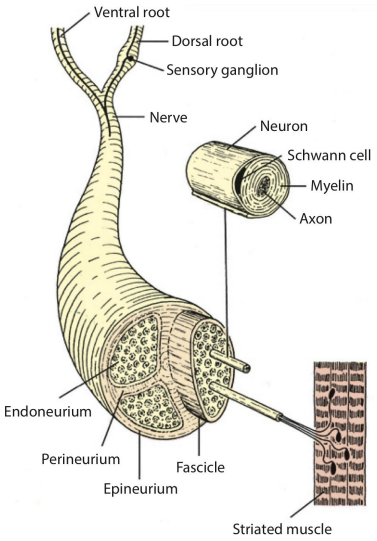
Peripheral Nerve Injury & Repair

By Orthobullets

Peripheral Nerve Injuries

Nerve Structure and Function

- All motor **axons** (main conducting component) and the large sensory axons serving touch, pain and proprioception are coated with **myelin** (lipoprotein derived from the accompanying Schwann cells[1]).
- Neuron is made up by axon covered by myelin and schwann cells.
- Outside the Schwann cell membrane, the axon is covered by a connective tissue stocking, known as the **endoneurium** (between neurons).
- The axons that make up a nerve are separated into bundles (fascicles) by fairly dense membranous tissue, known as the **perineurium** (between fascicles).
- In a transected nerve, these fascicles are seen, their perineurial sheaths are well defined and strong enough to be grasped by fine instruments during nerve repair.
- The groups of fascicles that make up a nerve trunk are enclosed in an even thicker connective tissue coat, known as the **epineurium** (what we see in OR).
- The epineurium varies in thickness according to the area of the nerve and is particularly strong where the nerve is subjected to movement and traction, for example near a joint.
- **Richly supplied by blood vessels** that run longitudinally in the epineurium.



Pathology

- Very commonly to be missed and no body will check it due to trauma, unconscious or comatose patients, all needs ATLS and the nerve injury not a priority in these situations.
- Nerves can be injured by ischemia[2], compression[3], traction[4], laceration[5] or burning[6].
- Damage varies in severity from transient and recoverable loss of function to complete interruption.

1 Transient Ischemia

IMPORTANT

- Caused by: Acute nerve compression.
- For a short period of time (no oxygen), without any damage (myelin is intact), least type of peripheral nerve injury.
- Starts with **numbness** and tingling within 15 minutes.
- Then **loss of pain** sensibility after 30 minutes.
- Followed by muscle **weakness** after 45 minutes.
- Relief of compression is followed by intense **paresthesia** (قرصات) for 5 minutes ('pins and needles' after a limb 'goes to sleep'), feeling is restored within 30 seconds and full muscle power after about 10 minutes.
- Due to transient endoneurial anoxia and **no nerve damage**.
- It could happen with fracture but once you reduce the fracture the compression relief.

1- Schwann cells are responsible for the myelination of the axons in the PNS, while the oligodendrocytes are responsible for that task in the CNS.
2- E.g. Acute limb ischemia (vascular patients), iatrogenic (tourniquet use in long procedures).
3- E.g. Dislocated joint, fracture compress it or carpal tunnel syndrome (very common classic injury).
4- In delivery when pull one hand of baby it might cause traction injury to brachial plexus (Erb's palsy), or when clutching a tree, or iatrogenic (excessive stretch in surgery).
5- Could be iatrogenic in OR, gunshot or stabbing.
6- Regular burn or affect of radiation, e.g. Burn around elbow → Ulnar nerve injury.

Classification of Peripheral Nerve Injury

Seddon's Classification

2 Neurapraxia

- Reversible physiological nerve conduction block (loss of sensation and muscle power) followed by spontaneous recovery after a few days or weeks, up to 6-8 months or a year (not like transient ischemia which is seconds to minutes).
- Due to mechanical **pressure** causing **segmental demyelination**[1] (The axon itself is still **intact**, myelin is the problem)
- Examples: Crutch palsy (brachial plexus), saturday night palsy (alcoholic), tourniquet palsy (iatrogenic)[2], compartment syndrome, carpal tunnel syndrome, distal humerus fracture (holstein-lewis fracture) → Radial nerve injury, posterior hip dislocation (dashboard injury) → Sciatic nerve injury.

3 Axonotmesis (tmesis = cut)

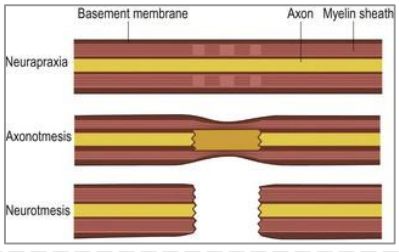
- More severe form of nerve injury.
- Closed fractures and dislocations and open fractures.
- Literally means **axonal interruption**.
- **Loss of conduction but the nerve is in continuity** (so there is chance for recovery), and the neural tubes (nerve sheath) are intact (the axon within is cut)
- **Wallerian degeneration** distal to the lesion.
- Axonal regeneration starts (at the same time) within hours of nerve at a speed of 1–2 mm per day.
- Eventually, they join to end-organs (motor end-plates and sensory receptors), and function is regained again.

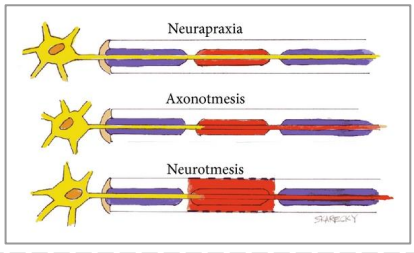
4 Neurotmesis[3]

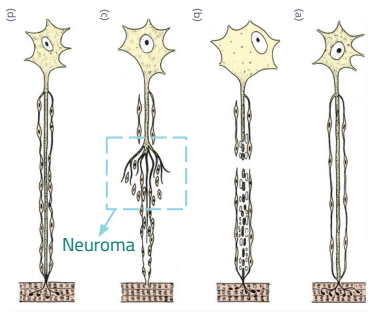
- Division of the nerve trunk as in **open fracture** (high energy trauma).
- There is healing at the injury site but usually the regenerating axons cannot reach the end organ, and the patient will have anomaly.
- If the injury is more severe, whether the nerve is in continuity or not, recovery will not occur (not suspect to heal without surgical intervention "nerve repair").
- Rapid wallerian degeneration.
- The endoneurial tubes are destroyed "whole nerve/fascicle".
- Regenerating axons does not reach the distal segment.
- **Neuroma** formation كرة النار (very painful fibrous tissue around the nerve ending, if you touch the skin → SEVERE PAIN → OR!) at the site of injury (regenerating axons, schwann cells and fibroblasts).
- Function is never normal.

Seddon Classification

Seddon Type	*Myelin intact*	*Endoneurium intact*	*Wallerian Degeneration*	*Reversible*
Neuropraxia	No	Yes	No	Reversible
Axonotmesis	No	Variable	Yes	Variable
Neurotmesis	No	No	Yes	Irreversible







Wallerian degeneration

(a) Normal axon and target organ (striated muscle). **(b)** Following nerve injury the distal part of the axon disintegrates and the myelin sheath breaks up. The nerve cell nucleus becomes eccentric and Nissl bodies are sparse. **(c)** New axonal tendrils grow into the mass of proliferating Schwann cells. One of the tendrils will find its way into the old endoneurial tube and **(d)** the axon will slowly regenerate.

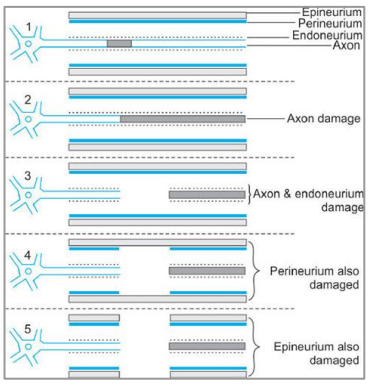
1- Axons are still intact however, dent in myelin sheath/schwann cells (no damage to the nerve).
2- Don't apply a tourniquet for more than 2-2:30 hours, if you need it, remove it each 2 hours and then after 1 hour apply it again.
3- Just know it's the most severe form, involving the whole tissue and there's axonal disruption e.g: knife cut of the forearm leading to drop rest.

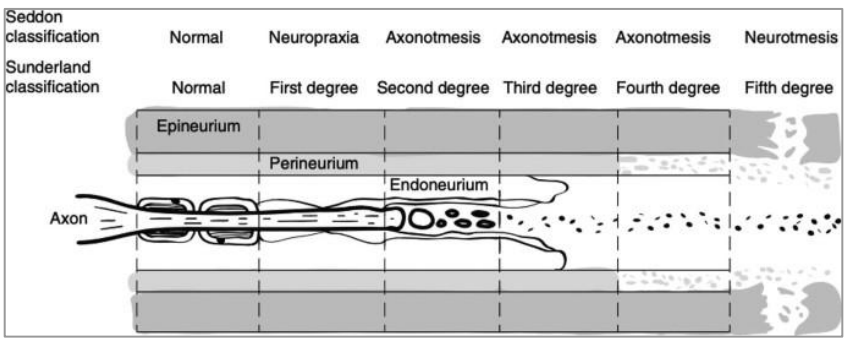
Sunderland's Classification

No MCQs, it's for academic & researches purposes

- More practical classification, not for your level.
- Not important, just try to understand the idea, Seddon's classification is the important one.

First Degree Injury (Neurapraxia)	• Transient ischemia and neurapraxia. • Reversible.
Second Degree Injury (Axonotmesis)	• Axonotmesis. • Axonal degeneration, but because the **endoneurium** is preserved, regeneration can lead to complete, or near complete, recovery without the need for intervention.
Third Degree Injury (Axonotmesis)	• Worse than axonotmesis (but still not neurotmesis). • The endoneurium is disrupted but the **perineurium** is intact. • Chances of the regenerating axons to reach their targets are good. • Fibrosis and crossed connections will limit recovery (when axon heals in wrong direction).
Fourth Degree Injury (Neurotmesis[1])	• Only the **epineurium** is intact. • The nerve trunk is still in continuity, but internal damage is severe. • Recovery is unlikely. • The injured segment should be excised, and the nerve repaired or grafted.
Fifth Degree Injury (Neurotmesis)	• The nerve is divided and will have to be repaired. • Epineurium is injured.





Clinical Features

- Acute nerve injuries are easily missed, especially if associated with fractures or dislocations.
- Ask for numbness, paresthesia or muscle weakness in the related area.
- Examine for signs of abnormal posture (Wrist/foot drop, claw hand, high-stepping gait), weakness and changes in sensibility.

1- This classification is not important to our level as the doctor said, but **in case of clarity**, I found this as an old note, and when I searched for it, all the references said it's equal to axonotmesis **not** neurotmesis (when we compare between the two classifications), so I will leave it, but pay attention to it if you're interested in orthopedics.

Assessment of Nerve Recovery

- Motor recovery is slower (will come last) than sensory recovery.
- **Pain is the first sensation to return.**
- Clinical tests of muscle power and sensitivity to light touch and pin-prick to follow up nerve recovery.
- Low energy injury (high chance of recovery) v.s. High energy injury (like MVA or fall from height, not a good sign for the patient).

Tinel's Sign[1]

- Most accurate and reliable test.
- Positive if patient has peripheral tingling (paresthesia) or dysesthesia upon percussing over the nerve.
- In a neurapraxia, Tinel's sign is **negative** (good indication).
- In axonotmesis, it's **positive** and advance with the regenerating axons.

Two-Point Discrimination

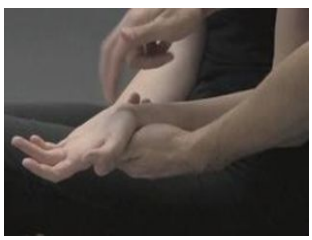
- Measure of innervation density.

Monofilament Assessment

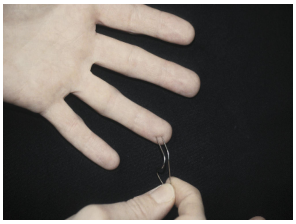
- Small filament used to check if patient has good degree of nerve sensitivity.

IMPORTANT Electromyography (EMG) & Nerve Conduction Studies (NCS) IMPORTANT

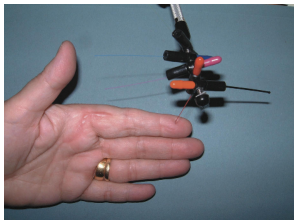
- Denervation potentials by the third week, in the initial stages not helpful to check the nerve so we don't do them before 3 weeks, usually after 6 weeks, then at 3 months, then at 6 months, then at 12 months.
- Distal humeral fracture & nerve injury → Surgery → No improvement → Electronic conductive study after 6 weeks.
- It excludes neurapraxia, and doesn't distinguish between axonotmesis and neurotmesis.
- **MRI** confirms the diagnosis of nerve injury.
- It's helpful to determine if there's a sign of nerve recovery or complete loss of function, because it will dictate the treatment plan.



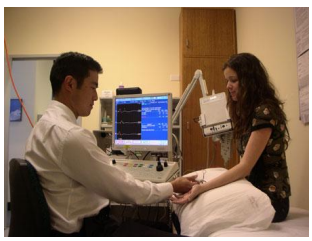
Tinel's sign



Two-point discrimination



Monofilament assessment



EMG/NCS

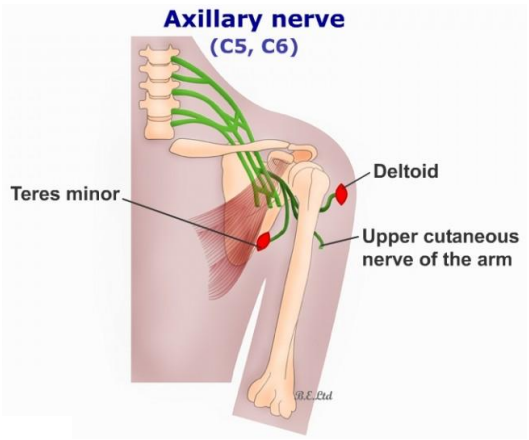
1- Tapping on the nerve pathway.

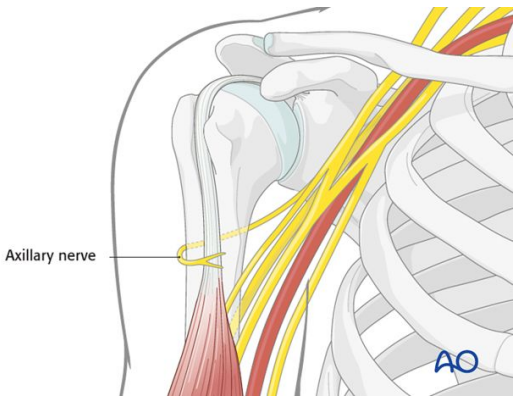
Upper Limb Nerve Injuries

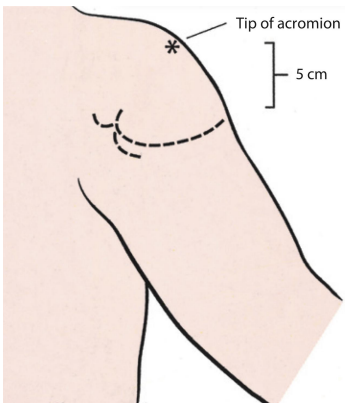
Axillary Nerve

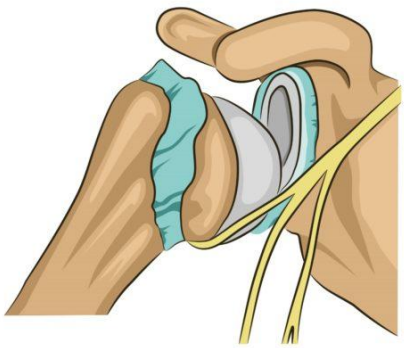
Anatomy	• The axillary nerve (C5, 6) arises from the posterior cord of the brachial plexus.
	• Supplies:
	  ○ Teres minor
	  ○ Deltoid muscle "abduction of shoulder".
	  ○ And a patch of skin over the muscle at proximal lateral arm.
	• Has an anterior branch that curls round the surgical neck of the humerus at 5 cm below the tip of the acromion (it's important to avoid injury during deltoid splitting approach).

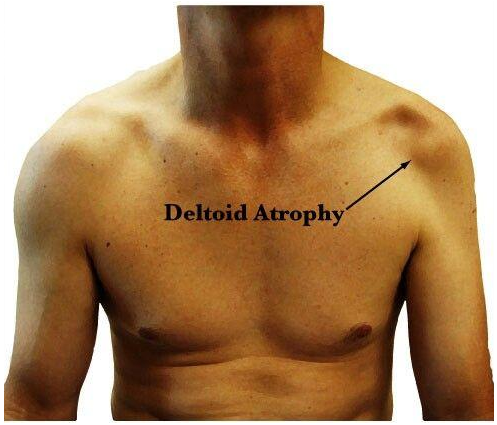
Injuries	• Shoulder dislocation or fractures of the humeral neck. • Lateral deltoid-splitting incisions.
Clinical Features	• Shoulder weakness, and the deltoid is rapidly wasted[1]. • Numbness over the deltoid. • High recovery rate, 80% of cases recover spontaneously. • Compare shoulder contour of both sides.









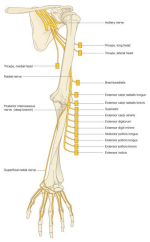


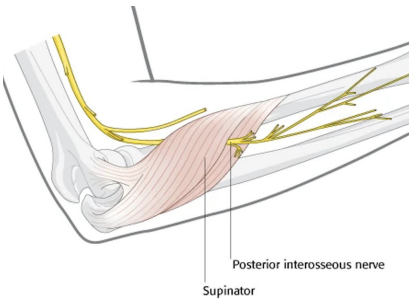
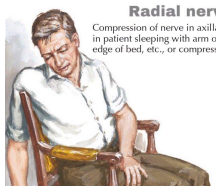
1- Deltoid wasting occurs **only in chronic injuries**.

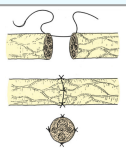
Radial Nerve

IMPORTANT

Most commonly injured nerve



	Low Lesions	High Lesions	Very High Lesion
Level	Elbow or upper forearm.	Humerus (around spiral groove).	Axilla or shoulder.
Injuries	• **Fractures** or **dislocations** at the elbow. • Iatrogenic lesions of the posterior interosseous nerve where it winds through the supinator muscle.	• Fractures of the humerus or after prolonged tourniquet pressure.	• Trauma or operations around the shoulder. • More often, chronic compression in the axilla (Saturday night palsy) or (crutch palsy).
Clinical Features	• Cannot extend the metacarpophalangeal joint (**MCP** joints) of the hand with weakness of thumb extension. • **Wrist extension is preserved** (very imp) because the branch to the extensor carpi radialis longus arises proximal to the elbow.	• Wrist drop. • Inability to extend the Metacarpophalangeal (MCP) joints or elevate the thumb. • Sensory loss at the dorsal first web space. • Reduce the fracture and apply a U-slab and wrist splint and consider nerve exploration in 3 months if no signs of nerve recovery (X-ray pic).	• In addition to weakness of the wrist and hand, the **triceps** is paralysed (loss of elbow extension) and the triceps **reflex** is absent. • For the triceps, test elbow extension AGAINST gravity.
Pictures		   Holstein-Lewis fracture	  Saturday night palsy



Nerve repair

Treatment

IMPORTANT IMPORTANT

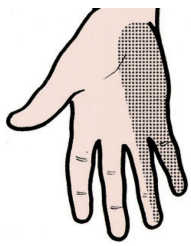
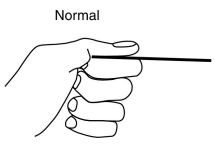
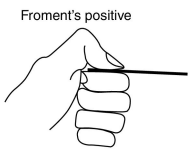
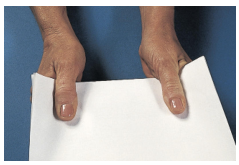
Wrist dynamic splint

Open Injuries	Closed Injuries	If No Recovery
Nerve exploration and - Primary repair[1] or - Grafting[2] as soon as possible E.g. Traumatic amputation, stab wound, gunshot wound and iatrogenic.	Usually neurapraxia, eventually recovers so 1- Observe 2- Splinting (occupational therapy)[3] 3- Physiotherapy (passive exercises to prevent contractures), then send the patient for EMG/NCS.	**Tendon transfer**[4] If not available → Arthodesis (which is joints fusion with a plate or K-wire "old method").

1- Approximate edges and suturing nerve ending, in clean wound and complete transection like in open fractures.
2- In big burn area or big crush injury "big segment", graft is taken from sural nerve "because it has small sensory area in lateral foot without any motor function".
3- Keep the limb at functional position, otherwise he'll have muscle stiffness and deformity so we splint and keep the fingers extended, if can't splinting → Casting.
4- Current literature states that for injury in this nerve specifically it's better to wait, don't do anything, nerve repair or graft, regardless of the injury wait for 6 months and most of the patients will recover.

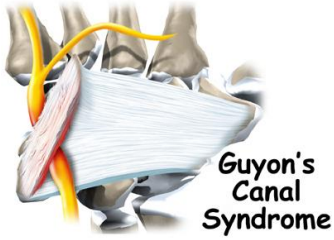
Ulnar Nerve

Signs + symptoms

	Low Lesions	High Lesions
Level	Near the wrist, e.g. Cuts on shattered glass or injury at level of forearm due to compression.	Near the elbow, e.g. Elbow fractures or dislocations.
Clinical Features	• Numbness of the ulnar one and a half fingers. • **Claw hand deformity**, with hyperextension of the MCP joints and flexion of distal IP joints of the ring and little fingers due to weakness of the intrinsic muscles. • Weak finger abduction (because of loss of intrinsic muscles) and **thumb adduction**, makes **pinch** difficult. • **Hypothenar** and interosseous wasting. • Ask the patient to hold a paper by the side, he won't be able to do it using the thumb, and will compensate by using flexor pollicis longus (which is supplied by median nerve) for loss of thumb adductor pollicis (positive **Froment's sign**).	• **Hand:** Not markedly deformed because the ulnar half of FDP (Flexor Digitorum Profundus) is paralysed and the fingers are therefore less 'clawed' **(high ulnar paradox)**. • **Explanation:** In low lesions there is claw hand because the flexor digitorum profundus is working and causes flexion supplied by median nerve BUT if it is high both muscles are affected so there isn't much clawing. • **Ulnar neuritis** (cubital tunnel syndrome)**:** Compression or entrapment of the nerve in the medial epicondylar (cubital) tunnel. • Severe valgus deformity of the elbow or prolonged pressure on the elbows in anaesthetized or bed-ridden patients.
Pictures	     	

Guyon's Canal Syndrome

- Also known as (ulnar tunnel syndrome) or (handlebar palsy).
- Entrapment of the ulnar nerve as it passes through Guyon's canal at the ulnar border of the wrist "compression neuropathy of ulnar nerve in the guyon's canal".
- Caused by ganglion (most commonly) or ulnar artery aneurysm (rare).
- MRI will diagnose the ganglion (which is the most common cause).



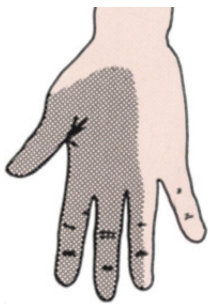
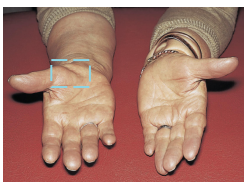
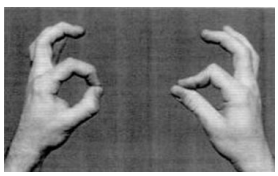
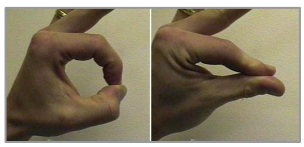
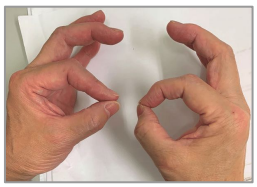
1- Why is the morphology of claw hand like that? Because from your anatomy it is the opposite of the muscles that were paralyzed which are the two medial lumbricals, these muscles flex the fingers at the metacarpophalangeal joints, and extend them at the interphalangeal joints.

Median Nerve

Signs + symptoms

	Low Lesions (most commonly injured)	High Lesions
Level	Near the wrist.	High up in the forearm.
Injury	Cuts in front of the wrist or by carpal, lunate dislocations.	Forearm fractures or elbow dislocation.

Clinical Features	• Unable to **abduct the thumb**.
	• Loss of thumb opposition.
	• Sensation is lost over the radial three and a half digits.
	• Thenar eminence is wasted and trophic changes (chronic injury).
	• Carpal tunnel syndrome (distal within wrist).
		• Same as low lesions, but in addition, all these muscles are paralysed:
		  ○ The long flexors to the thumb, index and middle fingers.
		  ○ The radial wrist flexors.
		  ○ **The forearm pronator muscles**.
		• Typically, the hand is held with the ulnar fingers flexed and the index straight (the 'pointing index sign').
		• Positive "OK" sign.
		• Thumb and index flexors are deficient, there is a characteristic pinch defect with the distal joints in full extension.
		• Positive Pucker sign.

Pictures	  Thenar eminence wasting	   

Isolated Anterior Interosseous Nerve Lesions

- Anterior interosseous nerve syndrome.
- Extremely rare.
- Similar to those of a high lesion but **without any sensory loss** (this is a purely motor nerve).
- The usual cause is brachial neuritis (Parsonage–Turner syndrome) which is associated with shoulder girdle pain after immunization or a viral illness

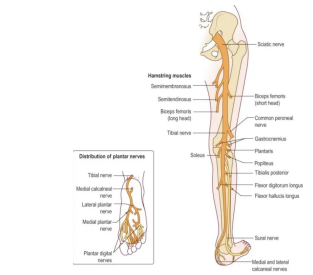
1- What is the most sensitive median nerve clinical test? Durkan test (also called carpal compression test), examiner press with thumb over carpal tunnel for 30 seconds, an onset of pain or paresthesia in median nerve distribution within 30 seconds is positive result.

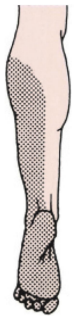
Lower Limb Nerve Injuries

Not important, focus on upper extremity only.

Sciatic Nerve

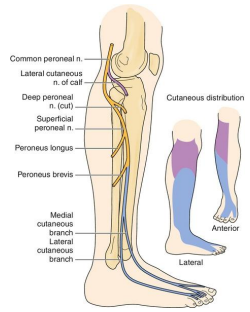
Injury	• Division (cut) of the main sciatic nerve is rare. • Traction lesions (traumatic hip dislocations and with pelvic fractures). • Iatrogenic lesions (posterior approach in total hip replacement).
Clinical Features	• In a **complete lesion** (rare) at the hip**:** The hamstrings and all muscles below the knee are paralysed and the ankle jerk is **absent**. • Very debilitating injury affects patient's quality of life. • Sensation is lost below the knee, except on the medial side of the leg (supplied by saphenous branch of femoral). • **Foot-drop** "weakness in ankle dorsiflexion" and a **high-stepping gait**. • Sometimes **only the deep part of the nerve** is affected (common peroneal nerve lesion), which can happen after hip replacement (more common).

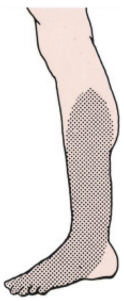
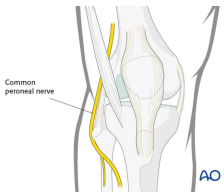



Peroneal Nerve

Level	• Common peroneal nerve or one of its branches. • Often damaged at the level of the fibular neck.
Injury	• Severe traction when the knee is forced into varus (e.g. in lateral ligament injuries). • Fractures around the knee. • During operative correction of gross valgus deformities. • Pressure from a splint or a plaster cast. • From lying with the leg externally rotated (e.g. **ICU patients**).
Clinical Features	• **Foot-drop:** Can neither dorsiflex (deep branch) nor evert the foot (superficial branch). • High-stepping gait. • Sensation is lost over the front and outer half of the leg and the dorsum of the foot.



Tibial Nerve

Injury	• Rarely injured except in open wounds.
Clinical Features	• Unable to **plantar-flex** the ankle or the toes. • Sensation is absent over the sole of the foot and part of the calf. • Because both the long flexors and the intrinsic muscles are involved, there is not much clawing.

Peripheral Nerves

• see Neurology, N38

Classification

Table 20. Seddon's Classification of Peripheral Nerve Injury

Nerve Injury	Description	Recovery
Neurapraxia (class I)	Axon structurally intact but fails to function	Within h to mo (average 6-8 wk)
Axonotmesis (class II)	Axon and myelin sheath disrupted but endoneurium and supporting structures intact → Wallerian degeneration of axon segment distal to injury	Spontaneous axonal recovery at 1 mm/d, max at 1-2 yr
Neurotmesis (class III)	Nerve completely transected	Need surgical repair for possibility of recovery

Etiology

- ischemia
- nerve entrapment nerve compressed by nearby anatomic structures, often secondary to localized, repetitive mechanical trauma with additional vascular injury to nerve
- direct trauma (e.g. transection)
- iatrogenic

Investigations

- clinical exam: muscle bulk and tone, power, sensation, reflexes, localization via Tinel's sign (paresthesias elicited by tapping along the course of a nerve)
- electrophysiological studies: EMG/nerve conduction study (assess nerve integrity and monitoring recovery after 2-3 wk post-injury)
- labs: blood work (e.g. CBC, TSH, vitamin B_{12}), CSF
- imaging: C-spine, chest/bone x-rays, myelogram, CT, magnetic resonance neurography, angiogram if vascular damage is suspected

Treatment

- early neurosurgical consultation if injury is suspected

Table 21. Treatment by Injury Type

Injury	Treatment
Entrapment	Conservative: Prevent repeated stress/injury, physiotherapy, NSAIDs, local anesthesia/steroid injection Surgical: Nerve decompression ± transposition for progressive deficits, muscle weakness/atrophy, failure of medical management
Stretch/Contusion	Follow-up clinically for recovery; exploration if no recovery in 3 mo
Axonotmesis	If no evidence of recovery, resect damaged segment Prompt physical therapy and rehabilitation to increase muscle function, maintain joint ROM, maximize return of useful function Recovery usually incomplete
Neurotmesis	Surgical repair of nerve sheath unless known to be intact (suture nerve sheaths directly if ends approximate or nerve graft (usually sural nerve)) Clean laceration: early exploration and repair Contamination or associated injuries: tag initially with nonabsorbable suture, reapproach within 10 d

Complications

- loss of function (temporarily or permanently)
- neuropathic pain: with neuroma formation
- complex regional pain syndrome: with sympathetic nervous system involvement

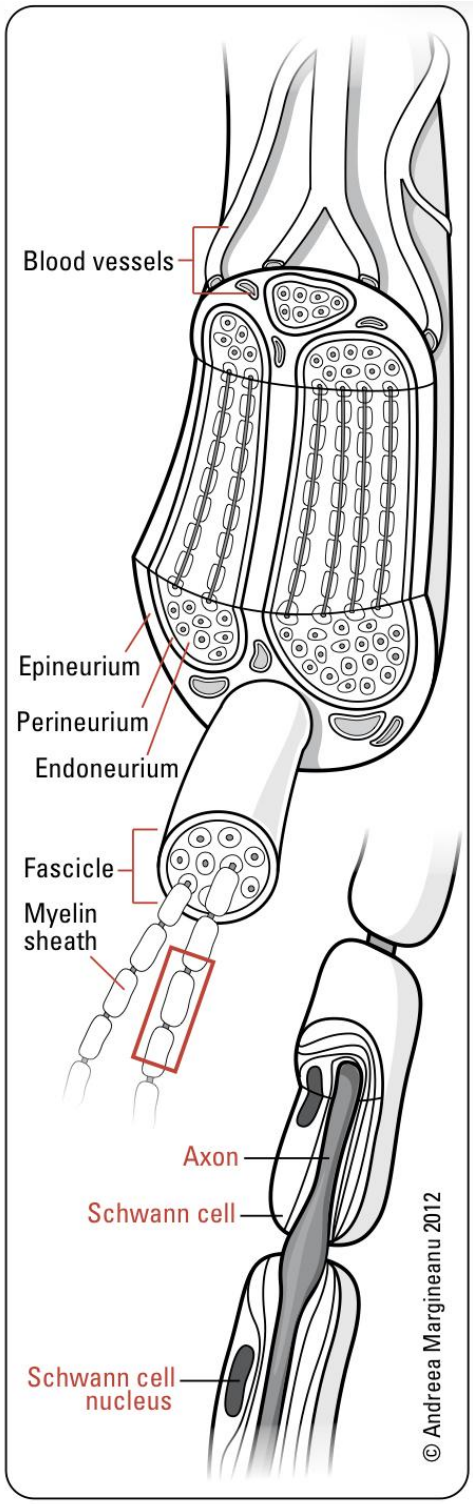


Figure 27. Peripheral nerve structure

Quiz

Q1: After close fracture reduction the pulse was normal but the patient couldn't feel and was unable to dorsiflex, what would you do next?

- A: Go to the OR and do nerve exploration
- B: Nothing it will return by itself
- C: Tell the patient that he has it before the reduction
- D: Tell the patient that he will has a permanent damage

Q2: 11 years old boy sustained left supracondylar humeral trauma following a fall, which of the following statements is correct if the median nerve or its branches are injured as a result of this injury?

- A: Positive OK sign
- B: Clawing of the hand
- C: Loss of thumb adduction
- D: Loss of sensation of little finger

Q3: Loss of forearm pronation is a motor effect of?

- A: Median nerve injury in elbow
- B: Median nerve injury at the wrist
- C: Axillary nerve injury
- D: Ulnar nerve injury

Q4: As a result of injury of the ulnar nerve at the wrist?

- A: Marked atrophy of the thenar eminence
- B: Wasting of hypothenar eminence
- C: Loss of sensation over the thumb
- D: Unable to abduct the thumb.

Q5: A 32-year-old man comes to the physician because of episodic tingling and numbness in his right hand for the past 3 months, his symptoms are worse in the evening, there is no history of trauma, he is employed as a carpenter, physical examination shows decreased pinch strength in the right hand, sensations are decreased over the little finger and both the dorsal and palmar surfaces of the medial aspect of the right hand, which of the following is the most likely site of nerve compression?

- A: Cubital tunnel
- B: Carpal tunnel
- C: Radial groove
- D: Surgical neck of humerus

1:B 2:A 3:A 4:B 5:A

Quiz

SAQs

441 & 439 & 438:

1. Name 3 common classes/types of peripheral nerve injury?
   1- Neurapraxia
   2- Axonotmesis
   3- Neurotmesis

2. Name 3 treatment options for closed non-iatrogenic peripheral nerve injury?
   1- Observation
   2- Splinting (occupational therapy)
   3- Physiotherapy (passive exercises)

Team Leader

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Abdulrahman Alroqi

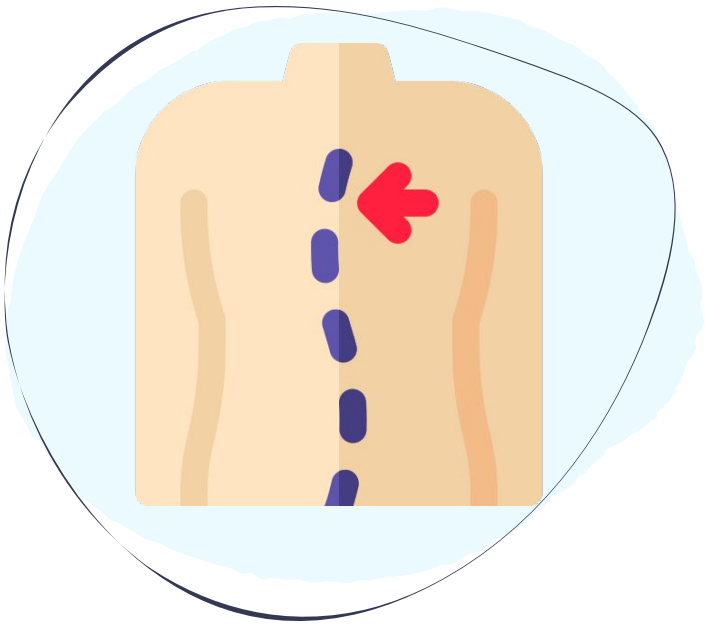
وفّقكم الله

9

Orthopedic 441 Team

MED441 KING SAUD UNIVERSITY

Editing File

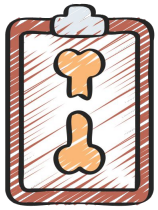


Common Spine Disorders

Dr. Abdulmajeed Alzakri

Color Index:

- Main Text
- Important
- 441 Notes
- Old Notes
- Extra



Objectives

The ability to demonstrate knowledge of the characteristics of the major conditions:

- Degenerative neck or back pain.
- Spinal cord or root entrapment (for example, herniated lumbar disc).
- Osteoporotic vertebral fracture.
- Spinal deformity (scoliosis, spondylolisthesis).
- Destructive (infectious and tumor related) back pain (for example, tuberculosis, metastasis, certain cancers).

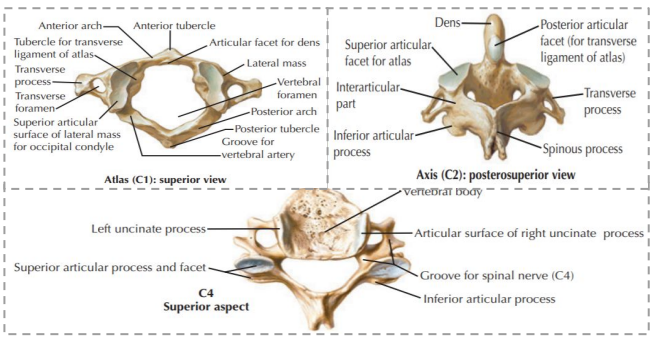
Resources

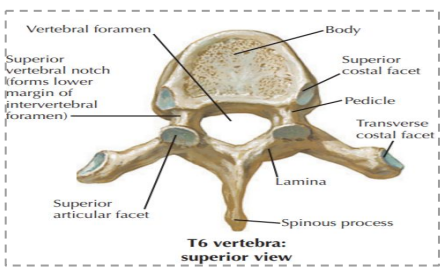
Anatomy of the Vertebrae

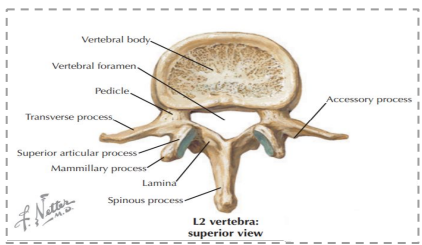
Vertebral Structure

- **Body or Centrum**: Weight-bearing part of the vertebra that lies anteriorly.
- **Vertebral arch:** formed from fusion of: 2 Pedicles and 2 Laminae.
- **The vertebral arch carries 7 process:**
  - 2 Transverse
  - One spinous
  - 2 Superior and 2 inferior articular
- **Vertebral foramen**: between the body and arch, through which the spinal cord passes.
- **Intervertebral disc**: Pads of flexible fibrocartilage that separate the vertebrae.

Vertebrae	Description
Cervical Vertebrae	• **Atlanto-occipital joint** → allow you to nod "Yes". • **Atlantoaxial joint** → allow you to nod "No". • The vertebral vessels passes through the transverse foramen and the vertebral arteries take part in the formation of the circle of Willis in the brain. • Only the vertebral vein passes through C7. • Nerve roots exit above the vertebra (C4 nerve exits between C3 and C4).
Thoracic Vertebrae	• Most thoracic vertebrae are typical, have bodies, vertebral arches and seven processes for muscular and articular connection. • It articulates with the ribs which acts as a splint to stabilize the thoracic spine. • Rarely injured only in high energy trauma or osteoporotic fractures. • **ROM:** Mainly rotation, very limited extension and flexion. Why? due to the way the thoracic articular facets are oriented (most rigid part of the spine).
Lumbar Vertebrae	• The **most common region for fractures and disc herniation**. • Most of the lumbar disc herniation happen posterolateral. • **ROM**: Flexion and Extension. (again, due to facet orientation that goes in the sagittal plane). • Defect in the pars interarticularis (connection between pedicle & lamina) → Spondylolysis. • Nerve roots exit below the vertebra (L4 nerve exits between L4 and L5).







Lower Back Pain Red Flags

- Age more than 50 (of new onset lower back pain)
- History of malignancy tumour weakening the vertebrae → cord compression or fracture
- History of trauma traumatic Spine Fracture → vertebral angulation, pain, or neural sx
- History of infection weakening bone → disc/vertebral destruction or cord compression
- Urinary retention/fecal incontinence "Cauda equina" (perianal numbness, decreased rectal tone)
- Acute motor deficit: Any acute loss of motor power = emergency surgery rush to the OR as soon as possible 6-12 hours to save it. = patient present with developed disc herniation and foot drop.
- Constitutional symptoms: Loss of appetite, loss of weight, fever, night sweat , pain at night.
- IV drug abuse.

Remember that spine fracture can occur without trauma patient can have severe osteoporosis coughs or sneezes (or any minor load) → fracture!

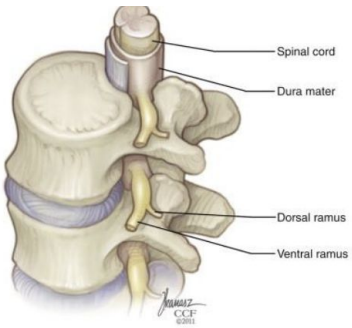
Degenerative Spinal Disorders

- **Degeneration**: deterioration of a tissue or an organ in which its function is diminished or its structure is impaired.
- **Other terms: Spondylosis**[1], degenerative disc disease, facet osteoarthrosis.

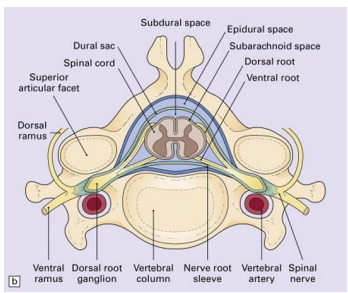
Etiology

Multifactorial in origin

- Genetic predisposition
- Age related
- Environmental factors: **smoking**, obesity, previous injury, fracture or subluxation, deformity, operating heavy machinery such as a tractor.



Anatomy



Anterior elements:

- Vertebral body
- **Inter-vertebral disc**
  - (Degeneration occurs at the disc)

Posterior elements:

- Pedicles, Laminae, Spinous process, Transverse process, **Facet joints** (2 in each level).
  - Osteoarthrosis occurs at the facet joints

Neurologic elements:

- Spinal cord.
- Nerve roots.
- Cauda equina.

1- **Spondylosis**: an umbrella term used to describe pain from degenerative conditions of the spine.

Pathology

Anteriorly
Intervertebral Disc (C2-S1)

The first component of the 3 joints complex:

- Motion segment.
- **It is primarily loaded in Flexion.**
- Composed of "annulus fibrosus" and "nucleus pulposus".
- **Degeneration of the nucleus:**
  - Loss of both: cellular material and hydration → **Pain**
- Can be asymptomatic, Water content drops disc became hard and black on the X-ray.

Disc degeneration will also cause:

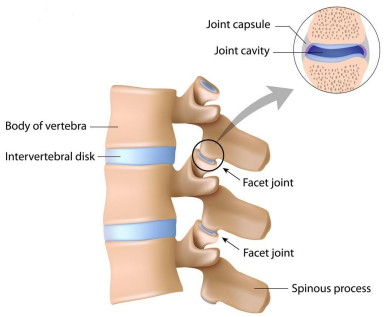
- Bulging of the disc → "Spinal" stenosis.
- Loss of disc height → "Foraminal" stenosis.
- Herniation of the nucleus → "Radiculopathy" (e.g. sciatica in the lumbar spine).

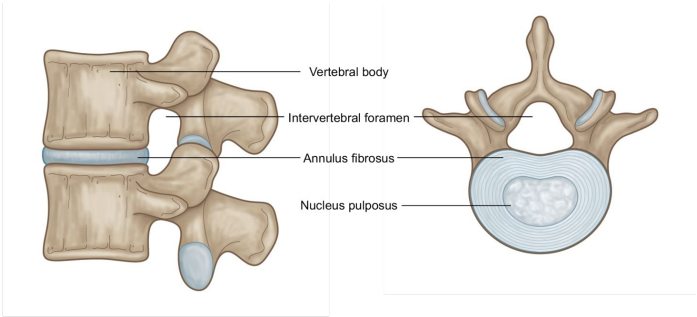
Posteriorly
Facet Joints (Zygapophysial Joints)

- They are synovial joints and we have 2 in each motion segment.
- Are primarily loaded in **extension**.
- Pattern of degeneration similar to other synovial joints:
  - ➔ Loss of hyaline cartilage.
  - ➔ Formation of osteophytes.
  - ➔ Laxity in the joint capsule[1].

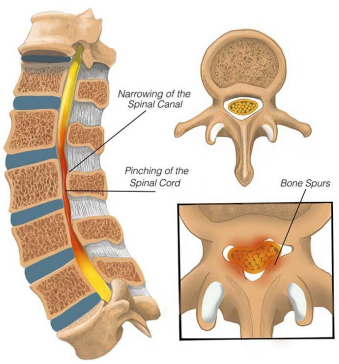
Facet joints degeneration will cause:

1. **Hypertrophy, osteophyte formation**
   - ➔ Leading to spinal stenosis or foraminal stenosis.
2. **Laxity in the joint capsule:**
   - ➔ Leading to **instability** (degenerative spondylolisthesis[2])

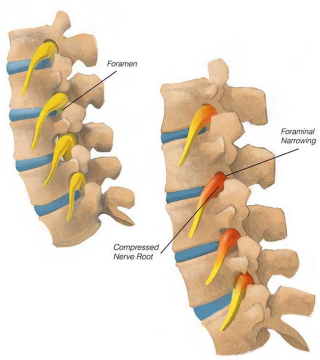




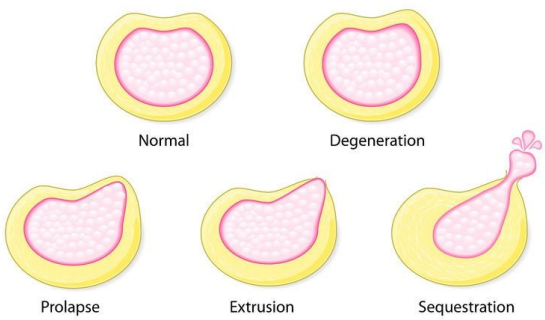
SPINAL STENOSIS



FORAMINAL STENOSIS



DISC HERNIATION



1- It's like any synovial joints in the body the degeneration process leads to loss of joint height, osteophytes formation, redundancy in capsule, inflammatory changes, excessive fluid and eventually instability which cause spondylolisthesis usually anterior posterior slippage "the vertebral above slips anteriorly"

2- Especially at the lumbar vertebrae. Another term is slippage of one vertebra or subluxation of one vertebra or over the other usually in sagittal plane front to back

Clinical Presentation

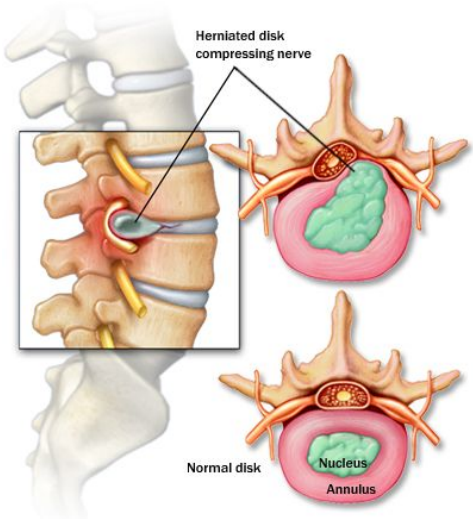
Clinical Presentation of Spinal Disorders Falls Into Two Main Categories

Mechanical Pain

Due to joint degeneration or instability.

- **"Axial pain"** in the neck or back (dull, deep, aching pain).
- Activity related **not** present at rest.
- **Associated with movement:**
  1. Sitting, bending forward **(flexion)**:
     - From the disc **"Discogenic pain".**
     - Pain when praying.
  2. Standing, bending backward **(extension)**:
     - From the facet joints **"Facet syndrome".**
     - Patient presents with the inability to stand or walk for a long time.

❖ Both can occur simultaneously.



Neurological Symptoms

Due to neurologic impingement of the spinal cord, cauda equina or nerve roots.

Spinal Cord:

1) Myelopathy: (compression)

- Loss of motor power and balance.
- Loss of dexterity[1].
  - Objects slipping from hands.
- **UMN deficit:**
  - **Rigidity, hyperreflexia, positive Babinski.**
- Slowly progressive (stepwise) deterioration[2].

2) Spinal Cord Injury:

- Spinal stenosis associated with a higher risk of spinal cord injury.

Cauda Equina and Nerve Roots:

1) Radiculopathy (e.g sciatica)

- **LMN deficit**[3].
- Commonest is sciatica[4] (lumbar).
  - Cervical root impingement causes similar complaints in the upper limb.

2) Neurogenic Claudication:

- Pain in both legs caused by walking.
- Pain relieved by sitting.
- Differentiate from vascular claudication.

Table – Differentiating neurogenic and vascular claudication IMPORTANT

Factors	Neurogenic	Vascular
Evaluation after walking	Increased weakness	Unchanged
Palliative factors	Bending over, sitting	Stopping
Provocative factors	Walking downhill Increased lordosis	Walking uphill Increased metabolic demand
Pulses	Present	Absent
"Shopping cart" sign	Present	Absent
van Gelderen bicycle test	No leg pain	Leg pain

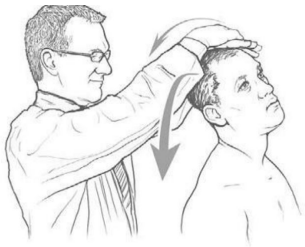
Going downhill is more difficult since you'll need to extend your back for balance, thus worsening the pain, Bending over relaxes the ligaments and relieves the spinal stenosis. While extending the spine does the opposite.

1- Loss of balance, fine movement such as writing, inability to button his shirt, coffee cup will slip from his hands.
2- Unlike spinal cord injury, myelopathies happen over months causing stepwise pain, numbness and paresthesia.
3- Hyporeflexia, hypotonia, dermatomal pain, motor defect, hypoesthesia, ..., etc.
4- Sciatica refers to the symptoms of pain, numbness, tingling, burning sensation or weakness that originate in the lower back, and radiate through the buttock, and continue down the back of the thigh, leg and foot. Sciatica occurs when there is compression, inflammation or injury to the sciatic nerve or to its (spinal nerve) roots L4-S2.

Cervical Spine

- Degenerative changes typically occur in **C3-C7**.

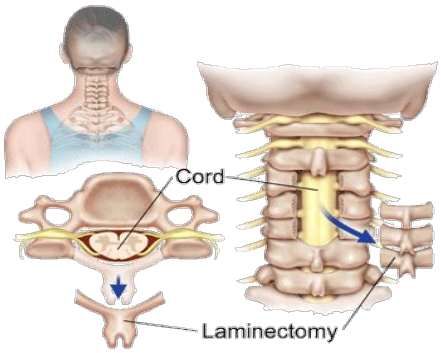
Degenerative Cervical Spine

History		• Presents with axial pain, myelopathy and radiculopathy
Physical Exam		• Stiffness → **Loss of ROM** **Neurological exam:** • **Weakness.** • **Loss of sensation.** • **Hyper-reflexia/tonia (UMN)** • **Special tests: Spurling's sign**[1] 

Management	**Conservative**	• **First line of treatment** for axial neck pain and mild neurologic symptoms (e.g. mild radiculopathy without any motor deficit)
		• **Physiotherapy:**
		  ○ Focus on ROM and muscle strengthening
		• **Non-steroidal anti-inflammatory medications (NSAID)** [2]
		  ○ E.g. Diclofenac, ibuprofen, or naproxen
		• **Neuropathic medication: for radiculopathy pain**
		  ○ E.g. Gabapentin or pregabalin. We don't commonly use it due to It's side effects.

	Surgical	**Indications:** • Spinal stenosis causing myelopathy (urgent surgery) • Disc herniation causing severe radiculopathy and weakness [3] • Failure of conservative treatment of axial neck pain or mild radiculopathy **Procedures:** • Anterior discectomy and fusion • Posterior laminectomy

Cervical compression



Laminectomy

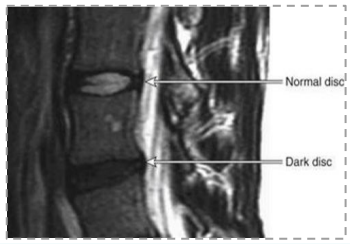
Anterior discectomy and fusion

1- The examiner turns the patient's head to the affected side while extending and applying downward pressure to the top of the patient's head. A positive sign is when the pain arising in the neck radiates in the direction of the corresponding dermatome ipsi-laterally.
2- Pay attention while prescribing such medications to patients with peptic ulcer, nephropathy, HTN.
3- It will present as **acute** limb weakness.

Lumbar Spine

- Degenerative changes typically occur in **L3-S1**

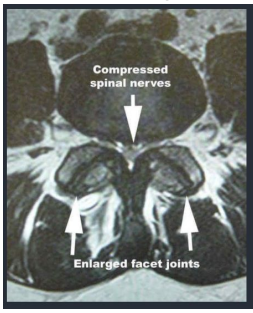
Lumbar Spondylosis

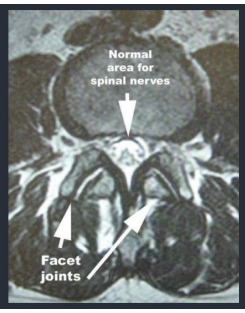


Degenerative Lumbar Spine

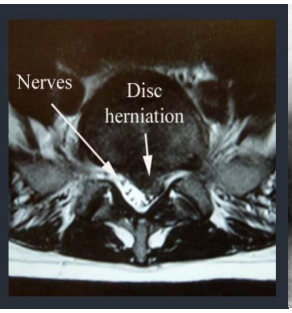
History		• Presents with axial pain, sciatica and neurogenic claudication.
Physical Exam		• Stiffness → **Loss of ROM** **Neurological exam:** • Weakness. • Loss of sensation. • Hypo-reflexia/tonia (LMN). • Special tests: Straight Leg Raise Test (SLRT).
Management	**Axial Lower Back Pain (Spondylosis)**	**Conservative: (first-line)** • Physiotherapy: ○ Core muscle strengthening, posture training. • NSAIDs. **Surgical:** indicated for: • Instability/deformity e.g. high-grade spondylolisthesis. • Failure of conservative treatment.
	Spinal Stenosis	**Conservative: (first-line)** (Especially in elderly or people who can't undergo surgery) • Activity modification. • Analgesics, epidural corticosteroid injection. **Surgical:** indicated for: • Acute motor weakness e.g. Foot drop. • Failure of conservative treatment (minimum 6 months). • **Procedure:** Spinal decompression (laminectomy) **commonest**
	Disc Herniation	**Conservative:** (**first-line** for mild sciatica with no motor loss) • Short (2-3 days) rest • NSAIDs, epidural corticosteroid injection. • Physiotherapy (95% resolves with no surgery in 3 months). **Surgical:** indicated for: • Cauda Equina or motor deficit. • Failure of conservative treatment (minimum 2 months). **Procedure:** Discectomy (only the herniated part).

Spinal Stenosis



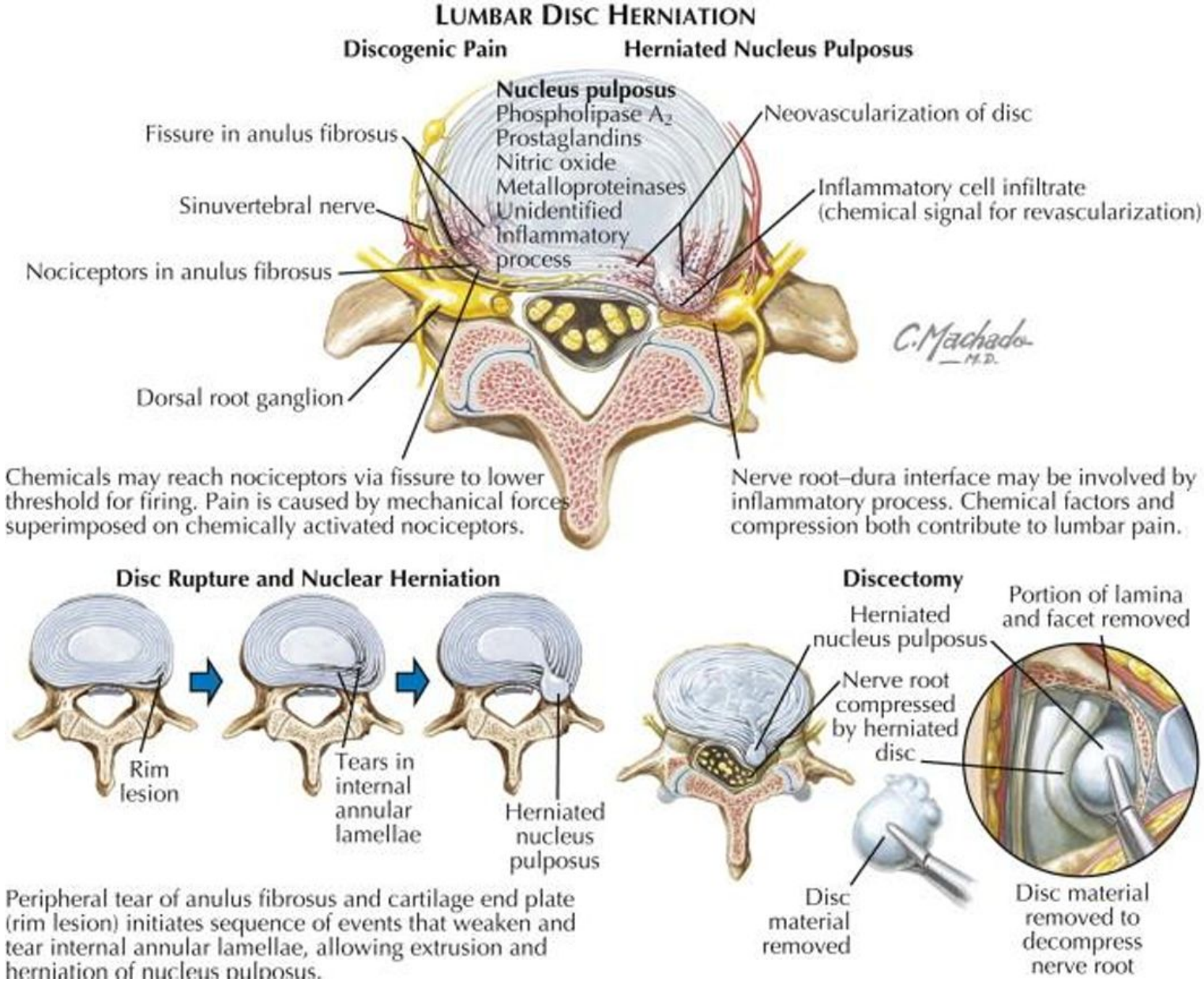


Disc Herniation



Lumbar Spondylosis

Discectomy

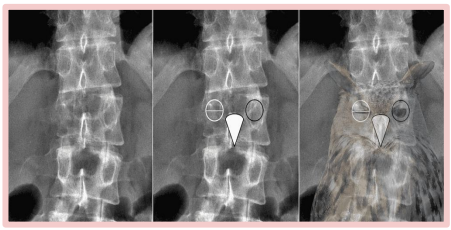

LUMBAR DISC HERNIATION
Discogenic Pain
Herniated Nucleus Pulposus
Nucleus pulposus
Phospholipase A2
Prostaglandins
Nitric oxide
Metalloproteinases
Unidentified
Inflammatory
process
Fissure in anulus fibrosus
Sinuvertebral nerve
Nociceptors in anulus fibrosus
Dorsal root ganglion
Neovascularization of disc
Inflammatory cell infiltrate
(chemical signal for revascularization)
C. Machado M.D.
Chemicals may reach nociceptors via fissure to lower threshold for firing. Pain is caused by mechanical forces superimposed on chemically activated nociceptors.
Nerve root–dura interface may be involved by inflammatory process. Chemical factors and compression both contribute to lumbar pain.

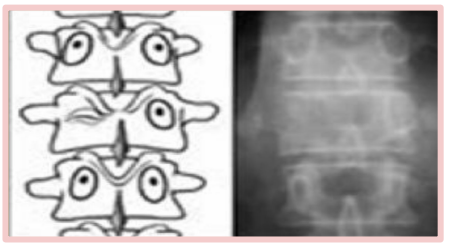
Disc Rupture and Nuclear Herniation
Rim lesion
Tears in internal annular lamellae
Herniated nucleus pulposus
Peripheral tear of anulus fibrosus and cartilage end plate (rim lesion) initiates sequence of events that weaken and tear internal annular lamellae, allowing extrusion and herniation of nucleus pulposus.

Discectomy
Herniated nucleus pulposus
Nerve root compressed by herniated disc
Portion of lamina and facet removed
Disc material removed
Disc material removed to decompress nerve root

Osteoporotic Vertebral Fractures

Description





- Pathologic fractures (low-energy fractures)
- Anterior column (±middle column) only compromised (Wedge/Burst Fracture)
- Often missed (common injury in postmenopausal women)
- Repetitive fractures result in kyphotic deformity (hunchback)
- Treat the underlying cause.

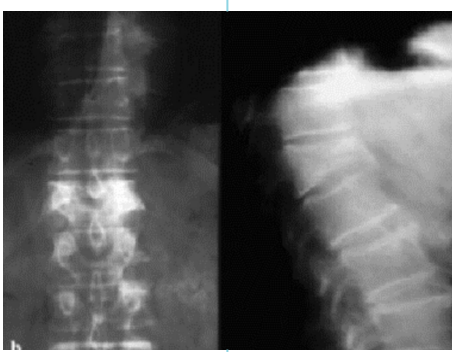
-(e.g. osteoporosis). It increases the mortality rate by increasing the rate of DVT and pneumonia

-Pathological fractures spinal X-ray shows "winking owl sign" (absent pedicles)

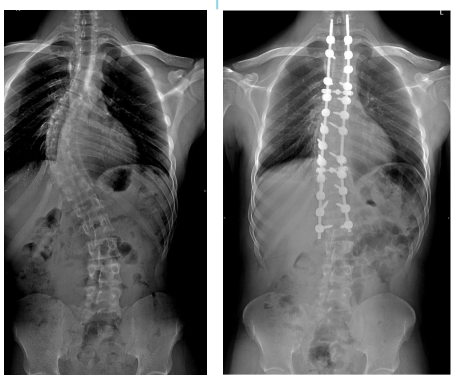
Spinal Deformities

Spinal Deformities

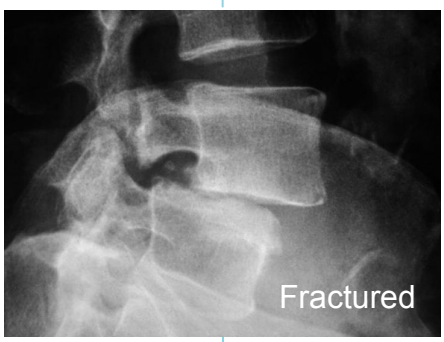
Kyphosis
Sagittal plane



Scoliosis
Coronal plane



Spondylolisthesis [1]
Translation



Give the patient anti-osteoporotic medications

1- Only high grade spondylolisthesis causes visible deformity.

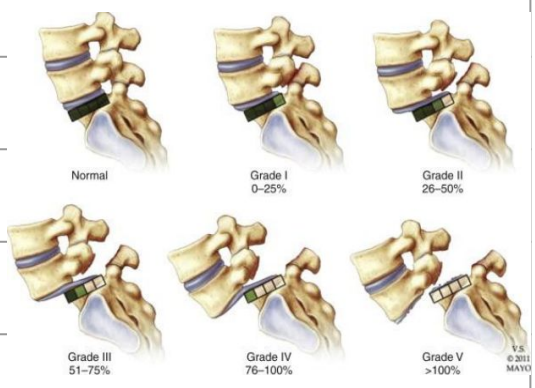
Scoliosis

Types		• **Congenital**: Associated with vertebral anomalies.
		  - E.g. hemivertebra (they have half vertebra).
		• **Acquired**: Secondary to other pathology.
		  - E.g. tumors, infection.
		• **Idiopathic**: Most common is adolescent type.
		• **Syndromic:** Secondary to other syndromes.
		  - E.g. Ehler danlos, marfan, muscular dystrophy.

Adolescent Idiopathic Scoliosis	**Description**	• **Three dimensional** deformity of the spine ★ **Vertebral rotation is the hallmark** • Painless deformity (usually noticed by parents/others)
	Examination	• Neurologically normal. • Positive Adams test[1]. 
	Management	• Depends on age & degree of deformity[2]. Young girl presents with severe idiopathic scoliosis with back pain, next step? MRI

Spondylolisthesis

Description		• Translation (displacement/subluxation) of one vertebra over the another (defect in the pars interarticularis) ○ Most people are asymptomatic
Types		• Degenerative spondylolisthesis (most common) • Isthmic spondylolisthesis ○ Caused by interarticularis defect (spondylolysis)
Grades	**Grade I**	• 25% displacement
	Grade II	• 50% displacement
	Grade III	• 75% displacement
	Grade IV	• Full displacement
	Grade V	• No contact (Spondyloptosis)
Management		• Conservative treatment first. • Surgery is indicated for grade ≥ 3 or failed treatment.

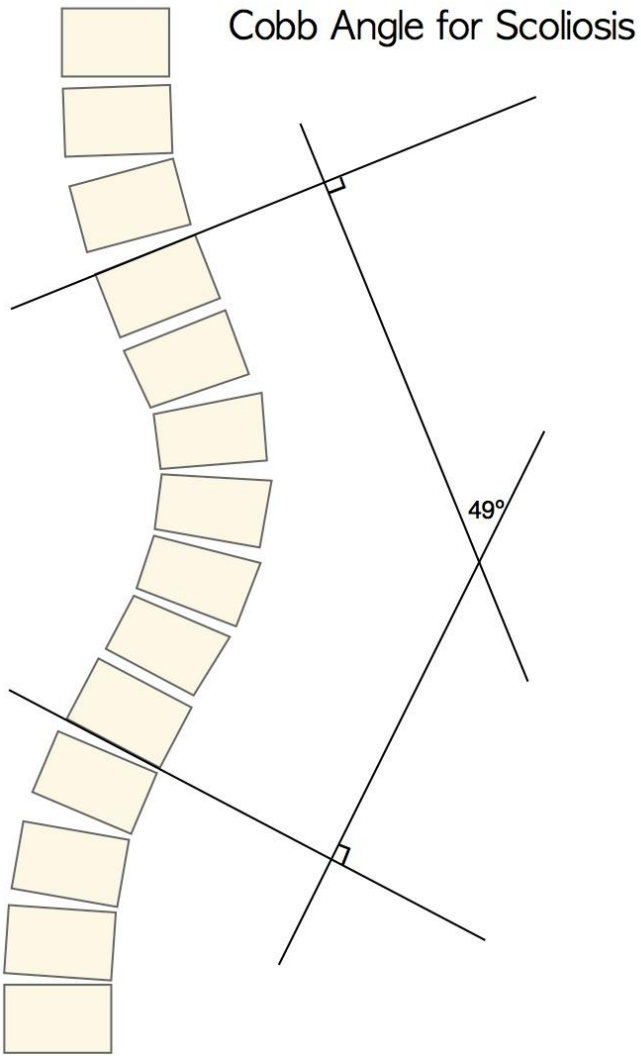


1-In "Adam forward bend" test, The patient bends forward, as if they are diving. If the patient has scoliosis, their back often has a prominent line where the spine is, and one side is higher than the other. Patient's back is completely straight if they do not have scoliosis.
2- Next slide.

Scoliosis Management

IMPORTANT

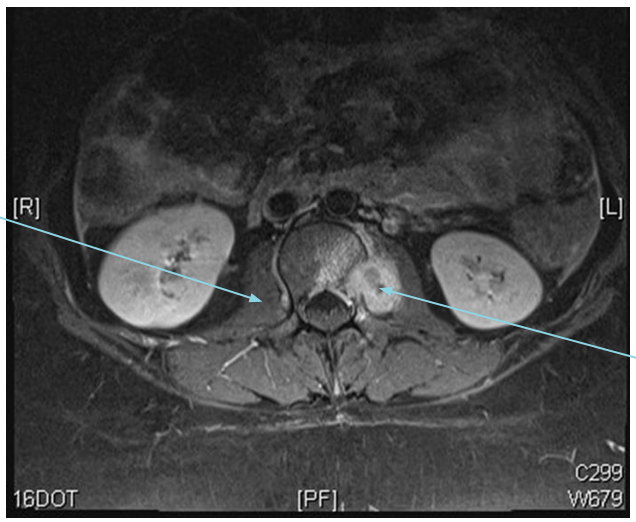
Cobb's Angle	Treatment
< 10	Normal
10-20	Physical therapy
20-45	Brace
> 45	Surgery



Destructive Spinal Lesions

- Present with **pain at rest** or **pain at night**.
  - ➔ The lesion then weakens the bone causing more pain (axial pain with movement) which might lead to fractures with minor trauma and loss of function
- Associated with **constitutional symptoms**.
- Vertebral body and pedicle are the most common sites of pathology
  - ➔ Because it's highly vascular.
- Most common causes are: Tumors and infections.
- If the patient presents without back pain or red flags, we order X-ray to check for stability.

Tumors	Infections
• **Primary Spinal tumors (rare)** ➔ Benign (e.g. osteoid osteoma). ➔ **Malignant** (e.g. chordoma). • Management depends on pathology. • **Spinal metastasis (very common)** because the spine is very close to major organs affected by tumors. ➔ Biopsy required if primary unknown (if you suspect lesion → biopsy). ➔ Spinal cord presentation is according to the site of compression.	• Most common are **TB** and **Brucellosis**. • History of contact with **TB** patient or raw milk ingestion is indicative. • Potentially treatable diseases once diagnosis is established and antimicrobials administered. • Most indicative feature is fever.



Spinal Tuberculosis with psoas abscess: abscess acts just like a tumor and can present like femoral hernia

◆ Compresses adjacent structures and spine

Quiz

Q1: A 35 year old patient's X-ray showed some degeneration at the level of C5 with no red flags which of the following is an indication for surgery?

- A: Mild radiculopathy
- B: Failure of conservative treatment
- C: Myelopathy
- D: All of the above

Q2: Nawaf a 40 year old patient came with repetitive pain after walking a certain distance which of the following is more indicative of neurogenic claudication?

- A: Pain is alleviated by rest
- B: Pain gets worse by going down stairs
- C: Pulses are weak in the lower limbs
- D: Nawaf starts with an N and Neurogenic also starts with an N

Q3: In spondylolisthesis above which grade is it an indication for surgery?

- A: Grade II
- B: Grade III
- C: Grade IV
- D: Grade V

Q4: A 14 years old female brought by her parents. They are concerned about the abnormality seen in her back. On examination, the only positive thing was a unilateral hump on the right side of her back when she bends forward. What is the most likely diagnosis?

- A: Degenerative disc disease
- B: Spinal TB
- C: Spondylolisthesis
- D: Scoliosis

1:B&C 2:B 3:B 4:D

Quiz

SAQs

441 & 439:

1. Mention the posterior elements of the vertebra?
   - Pedicles.
   - Laminae.
   - Spinous process.
   - Transverse process.
   - Facet joints (2 in each level).

2. Mention three features of the neurological claudication?
   1- The pain increases after sitting.
   2- The pulse is present.
   2- Shopping cart sign.

3. What lumbar spondylolisthesis means?
   - Translation of one lumbar vertebra over the other.

438:

A 30 year old female patient presents to your clinic complaining of unilateral pain, burning and numbness in her left buttocks, radiating down the back of her thigh and leg. Which was proven to be sciatica, she does not have any red flags.

1. Name 4 red flags for lower back pain:
   1- History of infection.
   2- History of trauma.
   3- Age more than 50 years
   4- Urinary retention/Fecal incontinence

2. How will you manage this patient?
   1- Physiotherapy.
   2- NSAIDs.
   3- Surgery if necessary.

3. What are the indications for discectomy?
   1- Cauda equina.
   2- Motor deficits.
   3- Failure of conservative management.

Team Leader

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وفّقكم الله

This work was originally done by team 438 & 439

10

Orthopedic 441 Team

MED441 KING SAUD UNIVERSITY

Editing File

Open Fracture, Fracture with NV Compromise & Pelvic Fracture

Dr. Abdulaziz Aljurayyan

Color Index:

- Main Text
- Important
- 441 Notes
- Old Notes
- Extra

Objectives

- To be able to identify and diagnose patients with an open fracture, a fracture with nerve or vascular injury and poly-trauma patients with pelvic injuries.
- To be knowledgeable about the pathophysiology and morbidity associated with these injuries.
- To be able to apply the principles of management of these injuries at the site of accident and In the emergency room.

Resources

Open Fractures - Everything you need to know

By nabil ebraheim

Open Fractures

By Orthobullets

Open fractures

- Open fractures are defined as: "fractures that at some point communicated with the environment". Very serious. Can lead to septic shock, death.
- An open joint is managed similarly.

This patient will be treated as open fracture even if you are not 100% sure

Etiology

- Open fracture usually requires high-energy injuries. (not always)[1]
- Sometimes it can be easily missed, look at the image on the right
- Commonly occurs in bones with minimal soft tissue coverage. e.g. anterior tibia, distal radius and ankle
- The deeper the bone (ex: femur) the higher the energy required.

Pathology

Traumatic injury to the soft tissue and bone

- Inoculation of organisms.
- Injury to vessels and microvasculature.[2]
- Ischemia and lack of immune response.
- This will lead to some necrotic tissue.
- Raised compartment pressure.[3]

The patient will be at a higher risk to develop an infection.[4]

Infections in the Presence of Fractures:

- Difficult to eradicate.
- Requires multiple surgeries.
- Significant costs.
- Need prolonged use of antibiotics
- Lead to significant morbidity.

1- E.g. a 90-year-old thin osteoporotic lady with very delicate skin who just got tripped she might easily break her bone and since her skin is not elastic the result will be an open fracture
2- Less blood delivered to the tissue > decreased O2 transfusion and nutrient > ischemia and lack of immune response > good media for the bacteria to grow > infection.
3- This might lead to compartment syndrome as a result of the swelling. In compartment syndrome the skin is not affected because it has its own blood supply.
4- Infection is the main risk of open fractures, it happens as a result from a combination of: A- communication with the external environment. B- decreased blood supply.

Open fractures

- An open fracture is usually a "red flag" warning of significant trauma so detailed assessment of the patient is necessary.
- An open fracture is associated with significant morbidity so must act quickly.
- A delay in management is proven to increase the likelihood of complications like infection.

So give urgent priority while triaging, provide initial management (antibiotics) and consult urgently.

Diagnosis

Abx and tetanus is the most important thing to prevent infection

- Sometimes it's obvious. Other times, settle; be observant
- A wound close to a fracture is an open fracture until proven otherwise= (the wound is very small, usually oozing blood and sometimes fat droplets from the intramedullary canal, needs high suspicion from the doctor)
- Any wound/small opening in close proximity to a fracture, even if it is a small laceration, is potentially an open fracture until proven otherwise. Whenever a fracture is diagnosed, go back and check the skin.
- Small wound continuously oozing blood, even after applying pressure with a gauze it keeps bleeding, especially if you see fat droplets within the blood like oil within the blood, it is diagnostic, but rarely seen, is an open fracture. Where did the fat come from? from the bone marrow. The most indicative thing of open fracture is wound near fracture site with oozing blood.
- Not always close to the fracture.
- Don't probe any small opening!! Because you might turn a closed fracture into an open fracture (probe: slender surgical instrument for exploring the depth or direction of a wound).
- If in doubt, use good light, if there is a break in the dermis or fat is seen, call it an open fracture.
- Better to overcall than miss it.

Open Fractures Algorithm:

1	Assess and stabilize the patient, ATLS principles (saving life before saving limb)
2	Assess the condition of the soft tissue and bone to help grade the open fracture
3	Manage the wound locally
4	Stabilize the fracture
5	IV antibiotics (Most important)
6	Tetanus status
7	Analgesics

Open fractures Algorithm

1-Assessment

- Whenever you have a polytrauma cause → apply ATLS principles.[1]
- If you have an isolated injury, take a brief history about the following:

➔ Mechanism and circumstances of injury?
➔ Time since injury?[2]
➔ PMH, PSH, allergies, drugs and smoking status (AMPLE)[3]
➔ Tetanus vaccination status

- After stabilizing the patient and taking history, we need to examine the affected region for:
- **Soft Tissue**:

➔ Degree of contamination (clean cut or bulldozer in a farm)
➔ Size of wound
➔ Necrotic and devitalized tissue
➔ Coverage loss break in skin
➔ Compartment syndrome

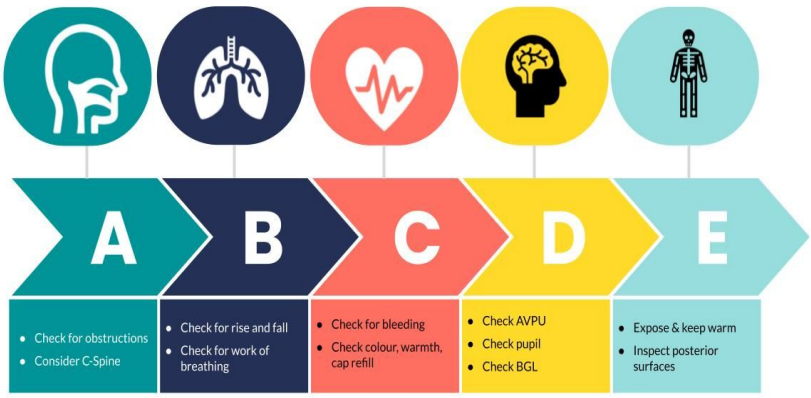
- **Bone**:

➔ Comminution (simple vs. fragmented) multiple small fragments (Indicating a high energy trauma)
➔ Stripping of bone periosteum [4] Provides blood supply
➔ Assess away from injury to joint above and below
➔ X-rays to joint above and below

- **Neurovascular**: **Before and after!!**

➔ Assess on arrival, post reduction and after splinting later

- Assess the condition of the soft tissue and bone to help grade the open fracture.



1- ATLS principles include primary survey which are the ABCDE (airway → breathing → circulation → disability → exposure)
2-The management differs between an open fracture since 1 hour and an open fracture since 12 hours.
3- AMPLE is a quick history : A: allergy / M: medication / P: past med/surgical /L: last meal(for anesthesia) / E: event surrounding the injury
4- Can indicates high energy trauma. Blood supply is affected which increases the incidence of infection.

Open fractures Algorithm

Real case of open reduction and internal fixation of both bones forearm fractures

2-Grading Open Fractures

After debridement in the OR is the best time to grade fractures

This classification will guide the management and makes the communication easier among doctors

- We classify open fractures using "Gustilo Classification". (indicates the severity)

Gustilo Classification			
Grade		**Description**	**Image**
Grade 1		• Less or equal to 1 cm • Less than 6 hours since injury. • Clean, non-segmental nor severely comminuted fracture	
Grade 2		• **> 1 cm** (usually less than 10 cm) • No extensive soft tissue injury or contamination • Non-segmental nor severely comminuted fracture • No bone stripping with adequate soft tissue coverage	
Grade 3	**3A**	• **Any size** (usually more than 10 cm) • Late presentation more than 6 hours • Extensive soft tissue contamination but not requiring soft tissue coverage • Segmental or severely comminuted	
	3B	• Any open fracture that **requires soft tissue coverage** procedure [1] • There is a pulse	
Anything beyond 6hrs , or segmentation/comminutioin= automatically 3C	**3C**	• Any open fracture that **requires vascular repair**. • Patient comes with pulseless limb	

You need the help of the plastic surgeon

It is more than 1 cm and it requires soft tissue coverage. So, it is either grade 3B or 3C. How to know? if there is a pulse then it is 3B, if there is No pulse then it is 3C (You take the higher grade)

Differ depending on the scenario:

- If the injury occurred **<6h** ago, it's **2**
- If the injury occurred **>6h** ago, it's **3A**
- If there is a **vascular injury**, it's **3C**

1-What are the difference between skin graft and flap ? A graft is a piece of healthy skin that is removed from one part of the body and used to cover a wound elsewhere and it's does not have its own blood supply. At first, the graft survives because nutrients pass (diffuse) from the wound site into the graft. Soon, blood vessels grow into the graft. The blood vessels supply the graft with nutrients. A flap is a section of healthy skin that is partially detached and moved to cover a nearby wound. The flap has its own blood supply and usually heal faster than grafts. In the flab you have to take skin, subcutaneous tissue and muscle and it's used in severe soft tissue damage.

Open fractures Algorithm

3- Management

g(+)= 1st generation cephalosporin
g(-)= aminoglycosides

There are 4 phases in management

Local Management

- Take a picture for documentation
- If dirty, irrigate with normal saline (warm) to remove gross contamination. Do not use water even if it was sterile.[1]
- If bone sticking out, try to reduce gently then immobilize and re-check neurovascular status.
- Cover with sterile wet gauze. Wet because a dry gauze will stick to the wound and cause more injury when removing it.
- If bleeding apply direct pressure on wound. **Never use a tourniquet**.[2]
- No culture swabs in ER. you might contaminate the swab and you may induce an infection.

Antibiotics Management

- Most important in managing open fracture grade 3A.
- First generation cephalosporin for gram **positives** (Ex: Cefazolin or clindamycin if allergic) in all open fractures. (all grades)
- Aminoglycoside to cover gram **negatives** (Ex: Gentamicin) sometimes required in grade 2 if you suspect gram negative, but generally give in all grade 3 cases.
- Add penicillin or ampicillin or clindamycin (for **anaerobes**) for clostridium in grade 3 open fractures and all **farm and soaked wounds**.
- Metronidazole can also be used for grade 3.

Extra

Antibiotics based on Gustilo Classification

	Grade I and II	Grade IIIA, IIIB and IIIC	Special considerations
Antibiotics	• 1st generation cephalosporin (e.g. cefazolin)	• 1st generation cephalosporin for gram positive coverage • Aminoglycoside (such as gentamicin) for gram negative coverage	• Penicillin should be added if concern for anaerobic organism (farm injury) • Flouroquinolones (e.g. ciprofloxacin) should be used for fresh water wounds or salt water wounds (can be used if allergic to cephalosporins or clindamycin • Doxycycline and 3rd or 4th-generation cephalosporin (e.g. ceftazidime) can be used for salt water wounds

1- Because osmolality of the water is more so the tissue will loss fluid due to the concentration gradient. Just irrigate, if there is a big piece you can remove it by sterile equipment.
2-it will compromise the blood supply. We apply direct pressure for 5-10 minutes, if bleeding stops it's a venous which is more common to be injured, if doesn't stop, this is arterial injury.

Open fractures Algorithm

3- Management con.

Common MCQ in the exam

Tetanus Management

Clean Wounds			Other Wounds		
Completed vaccination		Not Completed or Unknown	Completed vaccination		Not Completed or Unknown
Last Booster <10 years	Last Booster >10 years	Td 0.5 ml IM	Last Booster <5 years	Last Booster > 5 years	TIG 250 U + Td 0.5 ml IM
nothing	Td 0.5 ml IM		nothing	Td 0.5 ml IM	

Clean wound criteria: (Any wound that doesn't meet all 5 criteria should be dealt with as other wounds)

- <6 hours from injury.
- Not a farm injury.
- No significant devitalized tissue.
- Non immersed wound.
- Non contaminated wound.

Td: tetanus toxoid (vaccine) **TIG**: Immunoglobulin

Surgical Management and Plan

As soon as patient is stable and ready, alert the OR, and get consent from the patient for surgery

- Irrigation, debridement of necrotic tissue (source of infection) and fracture stabilization.
- The sooner the surgery the less risk of further morbidity.

In the OR:

- Extend wound if necessary. (small wound → extend wound until you can reach the bone)
- Thorough irrigation.
- Debride all necrotic tissue.
- Remove bone fragments without soft tissue attachment
- (dead bone) except articular fragments (except the part of bone that have cartilage over it). Cartilage is precious
- Usually requires second look or more every 48-72 hours.
- Generally, do not close open wounds on first look.

This is hematoma blister

Fracture management:

- Generally, avoid internal fixation (plate and screw) because the area is already contaminated.
- Generally external fixator is used.
- Femur and tibia fractures can usually be treated immediately with intramedullary nail except severe injuries and contamination in the intramedullary canal.
- Observe for compartment syndrome post-operatively. Can still happen even in open fractures

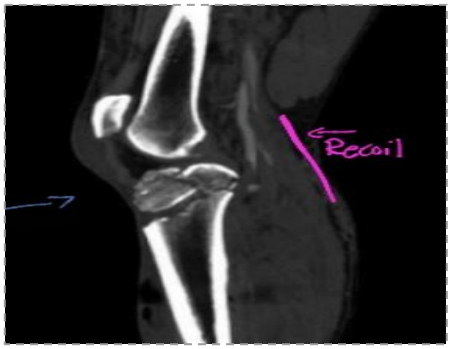
If all principles were applied

1. In grade 1 → 2 % complication rate
2. In grade 2 → 10 % complication rate
3. In grade 3 → **≤50 %** complication rate

Fractures with vascular injury

- Vascular injury is more dangerous than nerve injury because it is usually life-threatening
- Don't miss it. Most vascular injuries are intimal injury, Intimal tear won't cause bleeding
- Always perform an accurate assessment at presentation, post manipulation and reduction, postsurgical fixation, **serially until condition stabilizes.**
- Serial examination helpful in deciding line of treatment and avoiding confusion because sometimes the arterial injury is delayed so you have to check frequently.
- High correlation between vascular injury and nerve injury (proximity)
  - e.g. if a patient injured the brachial artery suspect a median nerve injury.

Mechanism



- Penetrating trauma
- High-energy blunt trauma
- Significant fracture displacement.
- Keep in mind tissue recoil at presentation.[1]
- Direct laceration (vascular) usually from blunt injury or a gunshot
- Traction and shearing. (vascular). Clotting if only injury in intima

Assessment of Vascular Injury

- **Always check for:**
  → Pulse, color, capillary refill, temperature, compartment pressure
- **Keep high index of suspicion in case of the following:**
  1. High energy trauma.
  2. Associated nerve injuries.
  3. Fractures/ Dislocations around the knee (injury to popliteal artery)[2]

IMPORTANT

Hard and Soft Signs of Vascular Injury Associated With Extremity Trauma

Hard signs
Pulselessness
Pallor
Paresthesia
Pain
Paralysis
Rapidly expanding hematoma
Massive bleeding
Palpable or audible bruit
Soft signs
History of bleeding in transit
Proximity-related injury
Neurologic finding from a nerve adjacent to a named artery
Hematoma over a named artery

- Hard signs (ALARMING SIGNS)
  → Realignment of limb → If persistent—> Vascular intervention
  → Realignment of limb → If Improved—> Close observation (with serial examination every 2 hrs)

Why do we perform a realignment?

Realignment results in unkinking of vessel, lowering compartment pressure, relaxing arterial spasm

- **Always measure ABI** (<0.9 → vascular pathology) normal 0.9-1.4
  - Rarely gives false negatives (except profunda femoris injury)
  - Always used in high risk fractures (e.g. around the knee? PA)
  - If positive > Urgent vascular intervention

$$ABI = \frac{\text{Ankle Systolic Pressure}}{\text{Brachial Systolic Pressure}}$$

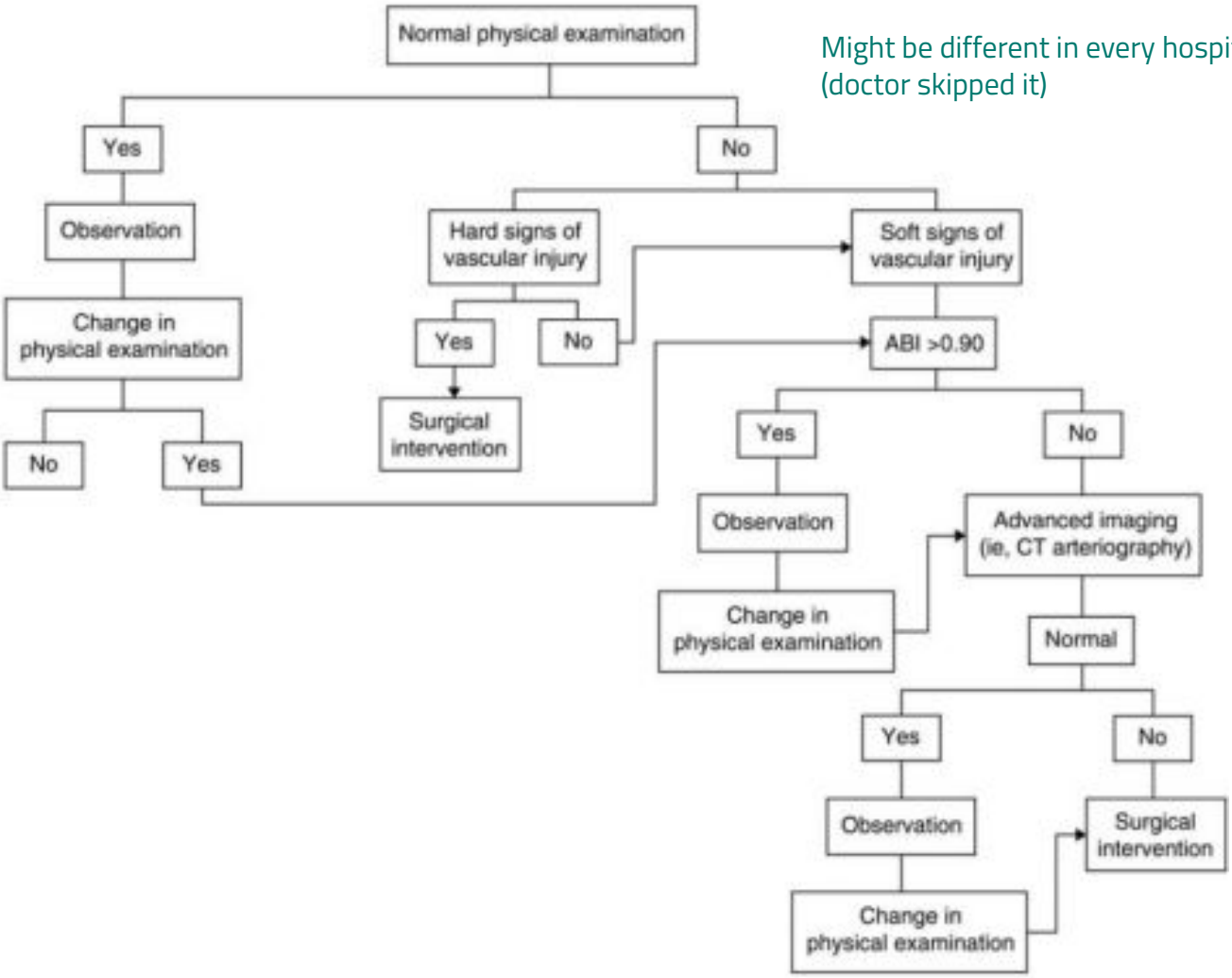
- Angiography and CT angiography are gold standard
  - Comes with risk, so it needs a Vascular surgeon to arrange with interventional radiologist

1- The injury might get displaced then the soft tissue might recoil it back to place; however, the injury still happened (be careful)
2- The popliteal artery is tethered by both ends proximally and distally (so it's very tense)

Fractures with vascular injury

Management of Vascular Injury

- Once a vascular injury is confirmed, coordination between the following must be done:
  - Orthopedic Surgeon 1st - vascular Surgeon 2nd - General Surgeon
- Because the orthopedic intervention is aggressive, due to that it may rupture the newly treated vessels if we start with the vascular team
- To emergently re-establish perfusion, protect, repair and stabilize the bones.
- We should stabilize the bone by an external fixator FIRST in 5 minutes then let the vascular surgeon repair the vessel
- Warm ischemia time dictates treatment.
  - Warm means the limb is still attached to the body,
  - Cold ischemia is when the amputated part was in an ice bag.
  - In cold ischemia it can wait longer because the metabolism is decreased in the amputated part.
- Most times, a quick external fixator is applied, followed by vascular repair.
- Avoid prolonging warm ischemia time.
  - Warm ischemia time is the amount of time from accident to when you intervene/ the time from injury till the limb is really ischemic and not warm anymore.
- Prolonged warm ischemia > 6 hours
  - You have to do prophylactic fasciotomy due to reperfusion injury which might cause CS
- Grade 3C open fractures have the worst outcome.
- Amputation may be necessary in severe cases.
- if you do reperfusion after 6hrs, you must perform prophylactic fasciotomy to prevent compartment syndrome



Might be different in every hospital (doctor skipped it)

Fractures with Nerve Injury

- Usually less critical unless if it's in the neck it might affect respiration
- Cause of medico-legal concern.
- Accurate assessment and documentation at presentation, post reduction, post-surgery is essential.
- Remember to examine for motor and sensation prior to sedation.
- The most common type of nerve injury in bone fracture is **Neuropraxia.**

Management of Nerve Injury

1

Closed fractures with nerve injuries usually doesn't require surgery: why?
- Usually good outcome >80%
- Usually managed conservatively in the early stages
- Recovery may take more than 6 months. most of the time it will recover spontaneously

2

Intact Nerve Before Reduction, absent after reduction:
- Controversial management.
- Usually observe (because most of the time it's neuropraxia)

3

Fracture requiring surgery with nerve injury: what dictates the need for surgery is the fracture not the nerve injury
- Limited exploration.
- Do not perform another incision especially for fixing the nerve itself, we do surgery for fixation of bone only if the nerve is in our way we fix it if not we don't do it..

4

Open Fracture With Nerve Injury:(gunshot +laceration)
- Explore, tag nerve ends for later repair.
- We do suture like a tube between the two ends of the nerve ends)

Follow up for Nerve Injury

- Follow up is usually clinical
- We need to perform an electrodiagnostic assessment starting at 6 weeks (then every 6 weeks)
- If there's no improvement:
  - ➔ Nerve exploration: neurolysis, repair or grafting
  - ➔ Tendon transfers to preserve function: use a tendon supplied by a different nerve (last option)

Common Sites of Nerve Injury

IMPORTANT

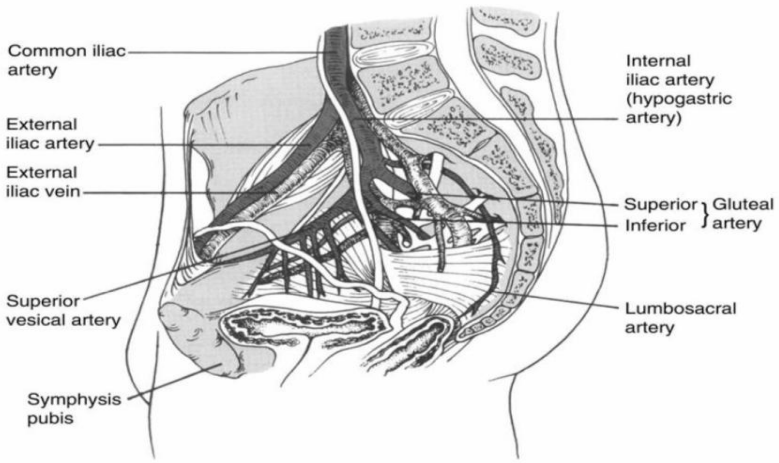
Injury	Nerve
Shoulder dislocation	Axillary
Humeral shaft fracture	Radial
Humeral supracondylar	Radial or median
Elbow medial condyle	Ulnar
Elbow dislocation	Ulnar
Montegia fracture	Posterior interosseous
Hip dislocation	Sciatic
Knee dislocation	Peroneal

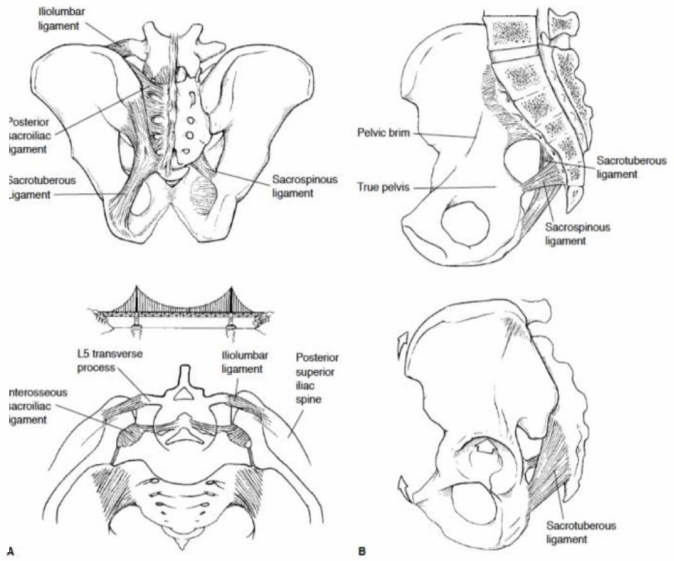
Shoulder fracture/ dislocation	Axillary nerve
Distal humeral shaft fracture	Radial nerve
Elbow fracture/ dislocation	Median, radial and ulnar nerves
Hip fracture/ dislocation	Sciatic nerve
Knee fracture/ dislocation	Peroneal nerve

Pelvic Trauma

Very serious may cause life-threatening bleeding

Pelvic anatomy





What makes the pelvis stable? 3 vertebropelvic ligaments.

1. Iliolumbar ligament: from tip of transverse process of L5 to posterior aspect of inner lip of iliac crest
2. Sacrospinous ligament: from ischial spine to lateral margins of the sacrum
3. Sacrotuberous ligament: from sacrum to tuberosity of the ischium.

The sacroiliac joint is an important joint, composed of:

- Anterior Sacroiliac ligament.
- Posterior sacroiliac ligament (the strongest and most important)
- Accessory ligaments (sacrotuberous and sacrospinous ligament)

Veins bleeding may cause tamponade affect, tamponade effect does not happen with arterial bleeding due to high pressure

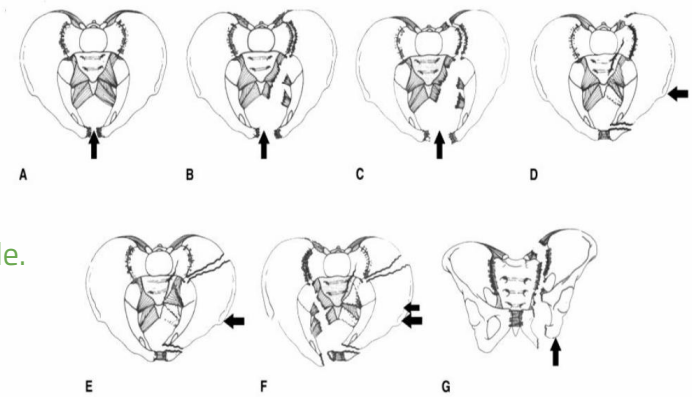
Most common source of bleeding in pelvis? Veins in bone ends

These ligaments, amongst others, provide critical, structural support and connection of various tissues in and around the pelvis.

Pathology

How the fracture causes instability to the patient's pelvis?
We have three mechanisms:
1. Anterior force causing open book. (A, B, C).
2. Lateral compression, e.g. car accident (figure D, E, F).
3. Vertical shears pelvic fracture (figure G). extremely unstable.

Survival is best in no. 1 and worst in no. 3

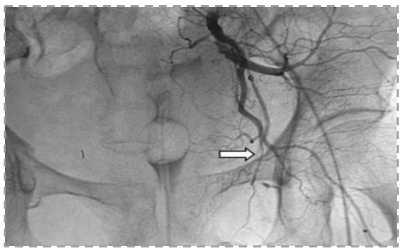


- Figure A: Anterior force causing a little opening in the sacro-illiac joint.
- Figure B: Anterior force causing a little opening in the sacro-illiac joint and instability.
- Figure C: more force caused open book pelvis. In this kind of injury, the left and right halves of the pelvis are separated at front and rear, the front opening more than the rear, i.e. like opening a book.
- Figure D, E, F: lateral compression.
- Figure G: vertical shears pelvic fracture. (falling on one's leg)

Pelvic Trauma in the polytrauma patient

- Pelvic fractures, instability may cause life threatening bleeding. [1]
- Diagnosing pelvic instability early and stabilizing it can save lives [2]

Diagnosis



- History: High vs. Low energy trauma.
- Mechanism of injury: Anterior vs. Lateral vs. Axial force
- Pelvic skin contusion, bruising.
- Short extremity (in vertical shears)
- Careful neurologic assessment.
- Primary survey (pelvic assessment falls under circulation)
  - ➔ Assess stability by gentle compression on the ASIS
  - ➔ Traction on the leg and assess pelvic instability, if unstable/painful:
    - Apply sheet around hips and close the pelvis gently.
    - This results in decreased intra-pelvic volume (tamponade the bleeding) works if venous
    - Traction on the leg to stabilize vertical instability
    - This minimizes ongoing vasculature injury and bleeding
    - If resuscitation doesn't work, that's an arterial bleed.
    - Do an ex-lap or go to angio suite.

RIGHT WRONG

- Rectal exam, check for the following:
  - ➔ Bone fragments (be careful because it's usually sharp)
  - ➔ High riding prostate
  - ➔ Bleeding
  - ➔ Blood at the meatus, labia, or scrotum
- Vaginal exam (if a fractured bone penetrate a hollow viscus (bowel, vagina) ⟶ OPEN fracture

Management

- Stabilize pelvis with binder.
  - To control the volume, you may need an external fixator (leave it until the patient becomes stable).
  - Pay attention, putting the binder for long time can cause skin necrosis (always follow up).
- If vertically unstable apply traction.
- IV resuscitation.
- Look for other injuries.
- Check response (make sure they are responding to fluid).
- If partial response, may require angiography for embolization of bleeders (arterial bleeding).
- May require external fixator and/or pelvic clamp.
- Early diagnosis > Aggressive resuscitation > Coordinated team effort = Save lives

1- The bleeding will be from the venous plexus of internal iliac vein and it will be retroperitoneal that's why it cannot be seen in US (FAST). So a patient comes after a high energy trauma we give him blood his blood pressure will increase then after a while it will go down, so what to do? Give him fluid again the patient will respond then the pressure will go down again this will tell you it is a matter of volume problem not cardiac or something else. So where is the fluid we are giving is going? You have to suspect an internal bleeding.
2- The idea of closing down and stabilizing the pelvis, is to try to close down the volume and allow less blood to accumulate till you have a tamponade effect, which can happen with venous bleeding or bleeding from the bone ends, arterial bleeding is less likely to stop. and fortunately, arterial bleeding is rare (10%).

Open Fractures

- fractured bone and hematoma in communication with the external or contaminated environment

33% of patients with open fractures have multiple injuries

Emergency Measures

- ABCs, primary survey, and resuscitate as needed
- remove obvious foreign material
- irrigate with normal saline if grossly contaminated
- cover wound with sterile dressings
- immediate IV antibiotics
- tetanus toxoid or immunoglobulin as needed (see Plastic Surgery, PL27)
- NPO and prepare for OR (blood work, consent, ECG, CXR)
  - operative irrigation and debridement within 6-8 h to decrease risk of infection
  - ORIF
  - traumatic wound may be left open to drain with vacuum-assisted closure if necessary
  - re-examine with repeat irrigation and debridement in 48 h if necessary

Antibiotics for Preventing Infection in Open Limb Fractures
Cochrane DB Syst Rev 2004;1:CD003764
Purpose: To review the evidence regarding the effectiveness of antibiotics in the initial treatment of open fractures of the limbs.
Methods: Randomized or quasi-randomized controlled trials comparing antibiotic treatment with placebo or no treatment in preventing acute wound infection were identified and reviewed. Data were extracted and pooled for analysis.
Results: Eight studies (n=1106) were reviewed. The use of antibiotics had a protective effect against early infection compared with no antibiotics or placebo (RRR 0.43, 95% CI: 0.29-0.65; ARR 0.07, 95% CI: 0.03-0.10).
Conclusions: Antibiotics reduce the incidence of early infections in open fractures of the limbs.

Table 6. Gustilo Classification of Open Fractures

Gustilo Grade	Length of Open Wound	Description	Prophylactic Antibiotic Regimen
I	<1 cm	Minimal contamination and soft tissue injury Simple or minimally comminuted fracture	First generation cephalosporin (cefazolin) 2 g IV q8 h for 2 d If allergy use clindamycin 900 mg IV q8 h If MRSA positive use vancomycin 15 mg/kg IV q12 h
II	1-10 cm	Moderate contamination Moderate soft tissue injury	As per Grade I
III*	>10 cm	IIIA: Extensive soft tissue injury with adequate ability of soft tissue to cover wound IIIB: Extensive soft tissue injury with periosteal stripping and bone exposure; inadequate soft tissue to cover wound IIIC: Vascular injury/compromise	First generation cephalosporin (cefazolin) for 2 d plus Gram-negative coverage (gentamicin or ceftriaxone) for at least 3 d For soil or fecal contamination, metronidazole is added for anaerobic coverage ± penicillin G If MRSA positive use vancomycin 15 mg/kg IV q12 h

*Any high energy, comminuted fracture, shot gun, farmyard/soil/water contamination, exposure to oral flora, or fracture >8 h old is immediately classified as Grade III

Orthobullets

Gustilo Classification					
	I	II	IIIA	IIIB	IIIC
Images					
Energy	Low	Moderate	High	High	High
Wound size	≤ 1 cm	1-10 cm	usually >10 cm	usually >10 cm	usually >10 cm
Soft tissue damage	Minimal	Moderate	Extensive	Extensive	Extensive
Contamination	Clean	Moderate	Extensive	Extensive	Extensive
Fracture Comminution	Minimal	Moderate	Severe	Severe	Severe
Periosteal Stripping	No	No	Yes	Yes	Yes
Skin Coverage	Local coverage	Local coverage	Local coverage	**Free tissue flap or rotational flap coverage**	Typically requires flap coverage
Neurovascular Injury	Normal	Normal	Normal	Normal	**Exposed fracture with arterial damage that requires repair**

EXTRA

Pelvis

Pelvic Fracture

Mechanism
- young: high energy trauma, either direct or by force transmitted longitudinally through the femur
- elderly: fall from standing height, low energy trauma
- lateral compression, vertical shear, or anteroposterior compression fractures

Clinical Features
- pain, inability to bear weight
- local swelling, tenderness
- abnormal lower extremity positioning: external rotation of one or both extremities, limb-length discrepancy
- pelvic instability

Investigations
- x-ray: AP pelvis, inlet and outlet views, Judet views (visualizes obturator and iliac oblique when acetabular fracture suspected)
  - 6 cardinal radiographic landmarks of the acetabulum: ilioischial line, iliopectineal line, teardrop, weight bearing roof, posterior rim, anterior rim
- CT scan useful for evaluating posterior pelvic injury and acetabular fracture
- assess genitourinary injury (rectal exam, vaginal exam, hematuria, blood at urethral meatus)
  - if involved, the fracture is considered an open fracture

Classification

Table 19. Tile Classification of Pelvic Fractures

Type	Stability	Description
A	Rotationally stable Vertically stable	A1: fracture not involving pelvic ring (i.e. avulsion or iliac wing fracture) A2: minimally displaced fracture of pelvic ring (e.g. ramus fracture) A3: transverse sacral or coccygeal fracture
B	Rotationally unstable Vertically stable	B1: open book (external rotation) B2: lateral compression – ipsilateral B2-1: with anterior ring rotation/displacement through ipsilateral rami B2-2: with anterior ring rotation/displacement through non-ipsilateral rami (bucket-handle) B3: bilateral
C	Rotationally unstable Vertically unstable	C1: unilateral C1-1: iliac fracture, C1-2: sacroiliac fracture-dislocation C1-3: sacral fracture C2: bilateral with 1 side type B and 1 side type C C3: bilateral both sides type C

Treatment
- ABCDEs
- emergency management
  - IV fluids/blood
  - pelvic binder/sheet
  - external fixation vs. emergent angiography/embolization
  - ± laparotomy (if FAST/DPL positive)
- non-operative treatment: protected weight bearing
  - indication: stable fracture (e.g. elderly patient with fracture sustained in fall from standing)
- operative treatment: ORIF
- indications
  - unstable pelvic ring injury
  - symphysis diastasis >2.5 cm
  - open fracture

Specific Complications (see *General Fracture Complications*, OR7)
- hemorrhage (**life-threatening**)
- injury to rectum or urogenital structures
- obstetrical difficulties, sexual and voiding dysfunctions
- persistent SI joint pain
- post-traumatic arthritis of the hip with acetabular fractures
- high-risk of DVT/PE

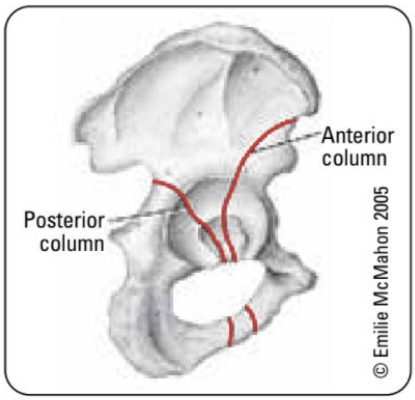


Figure 32. Pelvic columns

Possible Radiological Findings
- Pubic rami fractures: superior/inferior
- Pubic symphysis diastasis: common in AP compression (N=5 mm)
- Sacral fractures: common in lateral compression
- SI joint diastasis: common in AP compression (N=1-4 mm)
- Disrupted anterior column (iliopectineal line) or posterior column (ilioischial line)
- "Teardrop" displacement: acetabular fracture
- Iliac, ischial avulsion fractures
- Displacement of the major fragment: superior (VS), open book (APC), bucket handle (LC)

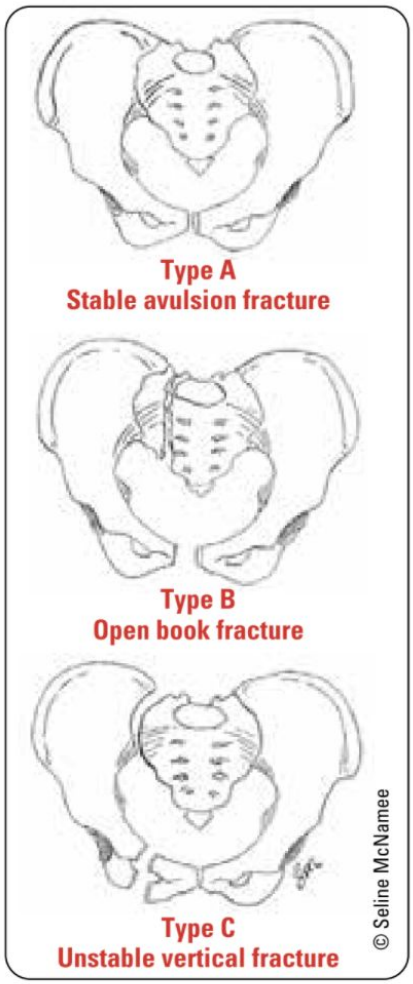
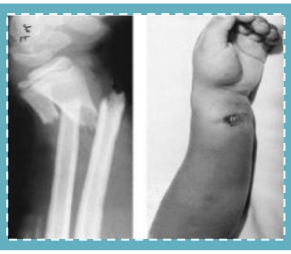


Figure 33. Tile classification of pelvic fractures

Quiz

Q1: A 9 -year-old boy sustained left forearm trauma after a fall. He presented to the emergency room with pain tenderness and inability to move his hand. Clinical and radiological pictures are shown on the figures. What is the diagnosis?



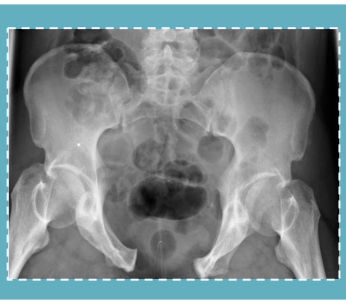
A. Superficial wound laceration with spiral fracture of both radius and ulna

B. Open fracture with Salter Harris type 1 distal radius and ulna

C. Open fracture with transverse fractures of radius and ulna

D. A simple both forearm fracture of radius and ulna

Q2: A 20 year-old male was involved in a motorcycle accident. On arrival to ER, patient was hypotensive and didn't respond well to fluid resuscitation. Examination revealed soft abdomen, clear chest, very unstable pelvis and no external bleeding. The AP pelvis X-ray is shown in the figure. Which of the following is the most appropriate next step?



A. U/S pelvis

B. CT pelvis

C. MRI pelvis

D. Angiogram pelvis

Q3: According to Gustilo Classification of open fractures, a pulseless patient is graded as:

A. Grade 2

B. Grade 3C

C. Grade 3B

D. Grade 3A

Q4: A twenty-five year old male patient sustained an isolated injury shown below following a car accident. He underwent emergency debridement and intramedullary fixation of the fracture.Which of the following post-operative orders would be most appropriate to avoid further morbidity in the early post-op period?

A. Close observation of thigh tightness and increasing pain

B. Weekly X-rays to observe for malunion

C. Daily blood work to observe for infection

D. Observation of distal pulses to identify early compartment syndrome

1:C 2:D 3:B 4:A

Quiz

SAQs

441 & 439:

1. List in details the classification of Open Fracture:
   - Just draw the table and write the information (slide 7)

438:

A 45 years old patient presented to the ER with an open fracture. Picture of the site of injury is shown below.

1. Mention 4 criteria to classify this fracture:
   1- Size
   2- Degree of contamination
   3- Bone fragmentation
   4- Time

2. Mention 2 long term complications of this condition:
   1- Bone infection
   2- Poor bone healing

Team Leader

Abdulrahman Alroqi

Done by

Faisal Alroba

Organized by

Abdulrahman Alroqi

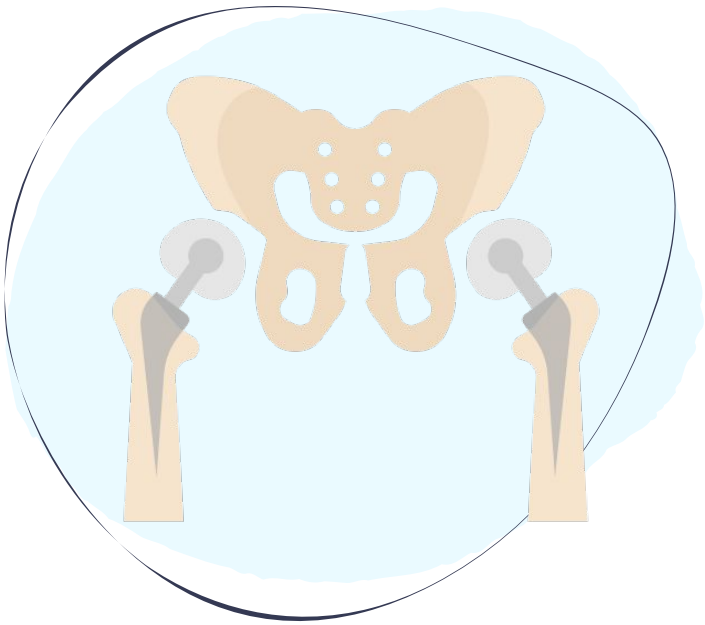
وفّقكم الله

11

Orthopedic 441 Team

MED441 KING SAUD UNIVERSITY

Editing File

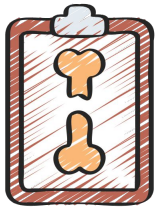


Common Pediatric Hip Disorders

Prof. Abdulmonem Alsiddiky

Color Index:

- Main Text
- Important
- 441 Notes
- Old Notes
- Extra



Objectives

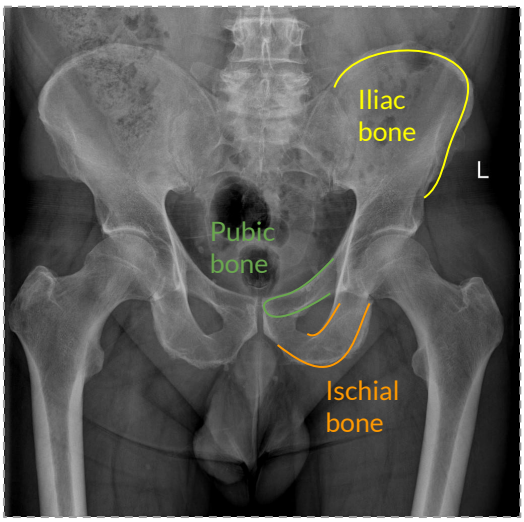
No objectives were given for this lecture.

Resources

Overview:

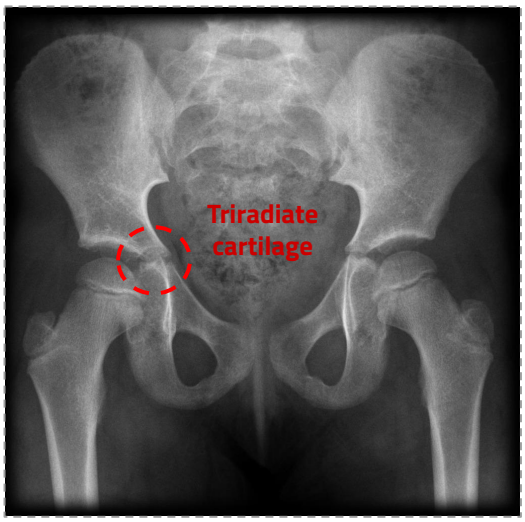
- In pediatrics the meeting area between the **ischium, pubic bone and iliac bone is called triradiate cartilage**. It is a transverse opening near to the top of the acetabulum.
- The femoral head is not present at the very early days (it is only cartilage), after a while the femoral head will start to be formed and the cartilage starts ossification and it will be called ossification center (ossification nucleus).
- Pediatrics have a growth plate, the greater trochanter is not yet formed.

Adult



A Child's Head and neck of femur are separated by the growth plate while in adults there's no separation.

Child



Common Pediatric Hip Disorders

DDH (Developmental Dysplasia of the Hip)
The abnormality is between the head of the femur and the acetabulum.

SCFE (Slipped Capital Femoral Epiphysis)
The problem is between the head and the neck of the femur, growth plate between them becomes weak.

Legg-Calve-Perthes Disease (LCP)
The problem is at the femoral head. (Issue is decreased blood supply to the head of femur).

❖ What is the difference between DDH and CDH?

DDH (Developmental Dysplasia of the Hip):
This is the new name of the disease, why did they change it? Because **it could happen with hip dislocation, subluxation, dislocatable, and Acetabular dysplasia.** It also could happen because of mechanical problems not only developmental problems (not only congenital).

CDH (Congenital Dislocation of the Hip):
This name means this disease will happen **only during in utero development**, and is **limited only to hip dislocation**. This term is no longer used because the disease can also be acquired later in life and can present with multiple presentations other than pure hip dislocation.

Developmental Dysplasia of the Hip:

- The main problem is between the **head** of the femur and **acetabulum**.

Patterns of Developmental Dysplasia of the Hip (DDH)	
Completely Dislocated	• **Completely separated;** out of acetabulum. • There's a special test for dislocated hip & another one for dislocatable hip it's **very important** to differentiate between them.
Subluxated	• Partially separated.
Dislocatable	• Means unstable. Normally, when I want to push out the femoral head it won't dislocate; however in a patient with dislocatable DDH, it will dislocate and return back easily.
Acetabular Dysplasia (Acetabulum doesn't fit the head of femur)	• Normally the head of the femur is inside the acetabulum and both of them are surrounding each other so they will have their shape (the hemispherical shape). • If the femoral head is dislocated the acetabulum will not find anything to surround, so it will become shallow. Or sometimes the acetabulum is formed as shallow shape with no femoral head dislocation, but because the acetabulum is shallow the head can't go in. • The acetabulum takes its shape when the femoral head is attached to it, but if the femoral head is not attached and not inside the acetabulum, it will become flat rather than hemispheric. Evenmore sometimes the baby is born with a flat acetabulum but the femoral head is normal with no pathology but cannot attach to the acetabulum. • What is the X-Ray finding? ○ The angle between the acetabulum and horizontal line is increased.

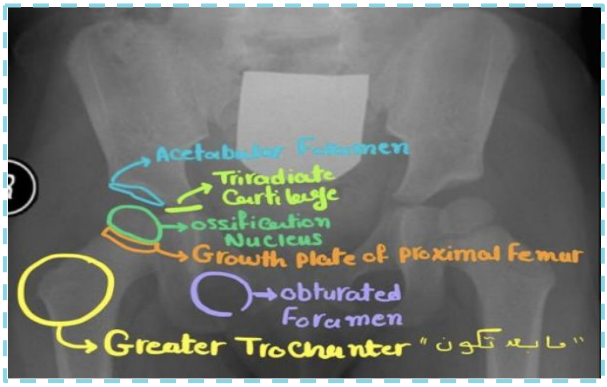
Causes:

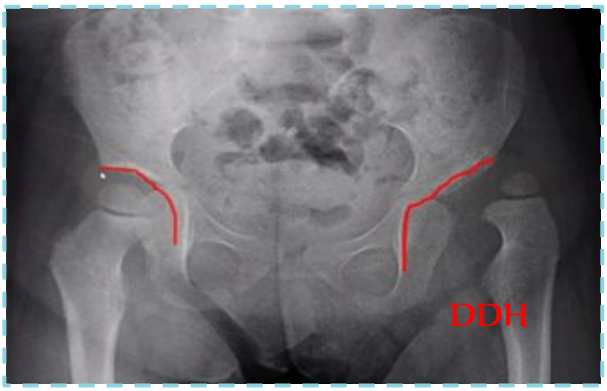
The exact cause is **unknown**, But may be due to:

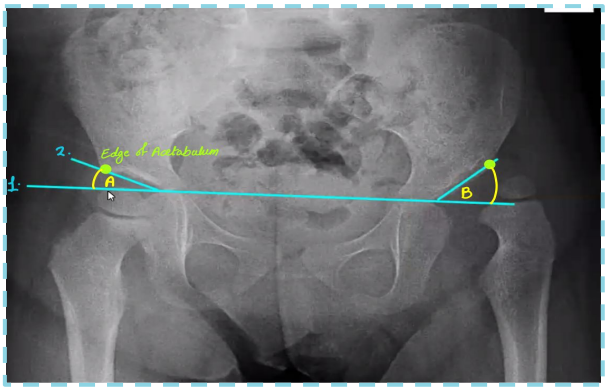
Causes	**Hormonal**		• Oxytocin and Relaxin
	Familial		• Familial ligament laxity diseases
	Genetic		• **Females are affected 4-6 times males.** • **Twins** (40%)
	Mechanical [1]	Prenatal	• Breech presentation[2], oligohydramnios[3], primigravida[4], twins, torticollis[5], metatarsus adductus and C-section
		Postnatal	• Swaddling (المهاد) and strapping (both causes adduction)

1- Anything that causes adduction of the hip might be a predisposing factor of DDH.
2- Breech presentation is when the fetus is lying longitudinally and its buttocks, foot or feet are presenting instead of its head.
3- Oligohydramnios refers to amniotic fluid volume that is less than expected for gestational age which might pressure the baby.
4- Primigravida is a woman who is pregnant for the first time.
5- Torticollis, also known as wryneck, is a twisting of the neck that causes the head to rotate and tilt at an odd angle.

Radiological features:







On Right side:

1. Head is inside the hip joint.
2. Acetabulum is covering the head of femur

Left side shows:

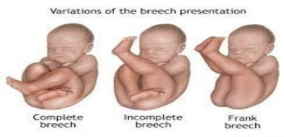
1. Head of femur is smaller
2. Lateralization (going out); of femur head
3. Acetabulum is not covering completely; (Shallow or Acetabular Dysplasia)

1. Draw straight line between the two Triradiate Cartilages. (horizontal line)
2. Draw line from the Edge of Acetabulum to the Triradiate Cartilage. Notice that the angle B is wider than A indicating DDH

Infants at Risk

1. Positive family history: 10X
2. A baby girl: 4-6 X
3. Breech presentation: 5-10 X
4. Torticollis: CDH in 10-20% of cases
5. Foot deformities: Calcaneo-valgus and metatarsus adductus
6. Knee deformities: hyperextension and dislocation.
7. Twins.

-breech birth: is when a baby is born bottom first instead of head first.



-Torticollis: defined by an abnormal, asymmetrical head or neck position

- When risk factors are present? the infant should be reviewed clinically and radiologically. If there are no risk factors one visit with negative findings should be enough to rule out DDH. If there are risk factors, **there must be at least two visits with negative findings plus radiological confirmation to rule out DDH.**
- DDH is increased by adduction and decreased by abduction, so anything that increases the stress on the head causing adduction might be predisposing factors.
- OSCE: History Taking of DDH patient. (the most important thing is to ask about the risk factors that are mentioned above)!

Clinical Examination:

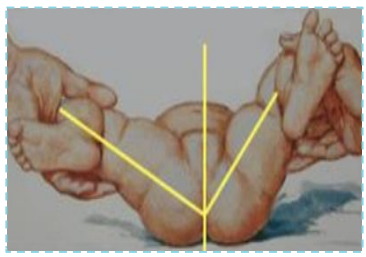
Look

These features are always found until treated:

- External Rotation
- Lateralized Contour
- Asymmetric skin folds (anterior and posterior)
- Shortening
  - Of dislocated limb, which moves up leading to formation of skin folds in the affected limb.

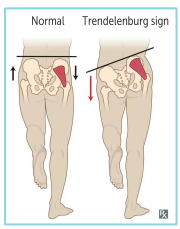
Move

- **Limited hip abduction:** Because there is a dislocation of the hip, that is why there is restricted abduction.
- It is present from birth, the mother will notice it while she's changing the diaper.
- Can be done in any stage and age.



Special Tests

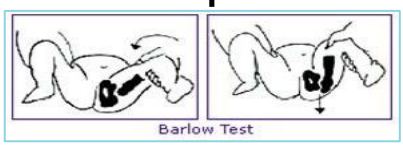
- Galeazzi (Older than 8 months)
- **Ortolani Test** (The most sensitive in Reducible DDH)
  - ➔ This Examination is performed for pediatrics <6 months.
  - ➔ Flex the hip and pull it then abduct it gently, if you pop it back into place or hear a click it is a positive test which means the baby is for sure has DDH.
  - ➔ Helpful in dislocated hip.
- Barlow test
  - ➔ This test is helpful in dislocatable hip, done only for baby <6 months.
  - ➔ You will do adduction and move the femur out a little bit, if you feel the femur moved out that means unstable hip (dislocatable hip)
- Trendelenburg sign (used for late presentations >2 years when the child starts to walk)
  - ➔ Becomes waddling gait if bilateral
- Painless limping



Barlow test

(Used for dislocatable pattern of DDH)

The test will dislocate the hip

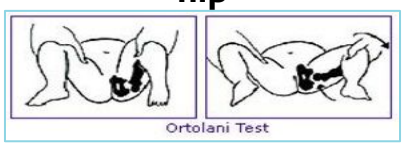


Barlow & Ortolani signs for hip dislocation

Ortolani Test

(Used for dislocated pattern of DDH)

The test will reduce the hip



Both these tests are the most important screening tests

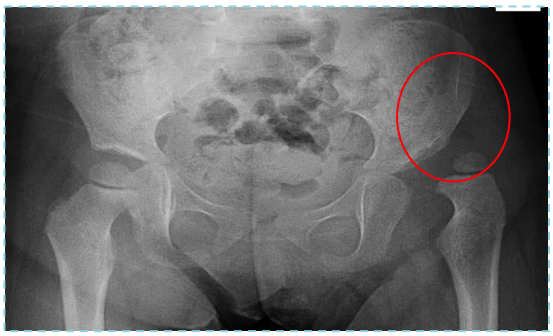
Important Notes ❗

★ **If you have an 8 months old patient with DDH what is the best test to use?**
- The answer is **limited abduction,** key? 8 months.Ortolani and Barlow is for babies less than 6 months & Trendelenburg is for babies after 2y.

★ **If you have a baby older than 6 months, what will you find in the examination which suggest DDH?**
- Limited abduction, shortening, increase skin fold, limping when the baby starts to walk.

Investigations

- **0-3 months:** U/S (bc the head of the femur is not yet formed(it is as cartilage), so X-ray is useless)
- **> 3 months:** X-ray pelvis AP + abduction + inferolateral position.
- **After 6 months:** reliable (the best option for baby 6 months or older is x-ray).

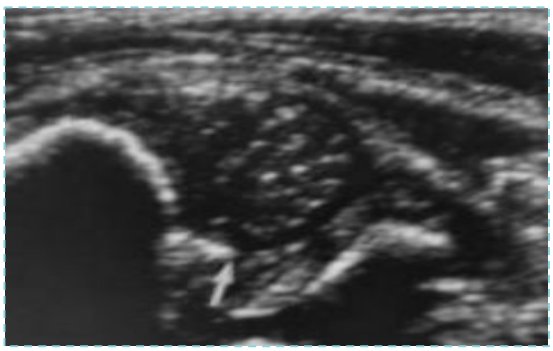


DDH on X-ray

(Very Important!)

In the EXAM:

★ If the baby is **less than 6 months old do US, 6 month or older do X-ray**".

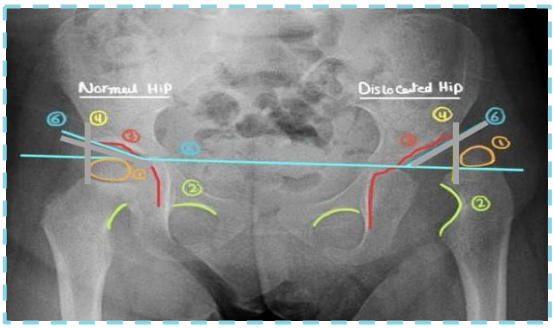


First you need to know some radiological terms found in pediatric hip:

1. Shows the ossification center (nucleus) of the femoral head\
2. Shows "Shenton's line" which runs across the upper border of the obturator foramen to the neck of the femur
3. Acetabulum
4. Shows two perpendicular "Perkin's" lines between the edge of acetabulum and the horizontal line (making an angle). **<u>Normally</u> the femoral head should be medial to the perpendicular line.**

6 Shows a horizontal line "Hilgenreiner's Line" between the two triradiate cartilages.

- **<u>Normally</u> the femoral head should be below horizontal line.**



The image shows left sided DDH

❖ <u>What are the signs of DDH on an X ray?</u> Important!

- Head of the femur (ossification nucleus \ center) is **small.**
- Disrupted **Shenton's line.** In DDH the obturator foramen in seperated from the neck of femur.
- Acetabulum is opened and we call it "**Shallow Acetabulum**". The problem happened in the relationship b\w the head of femur and the acetabulum **that's why it's DDH.**
- Head of the femur (ossification nucleus \ center) is **lateral** to the perpendicular line.
- Head of the femur (ossification nucleus \ center) is **above** the horizontal line.

<u>Note</u>: From base to the tip of the acetabulum: the normal angle is between **18-22 degree**, in **DDH** it's **30, 40 degree and more.**

Treatment

(very important in MCQs)

Treatment of DDH

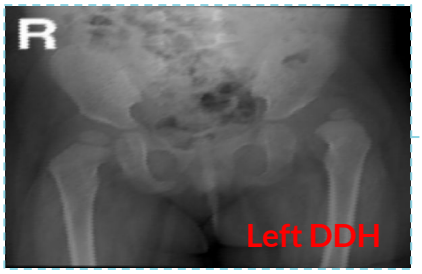
- Our aim in the treatment is to:
  1. **REDUCE**: obtain concentric reduction
  2. **STABILIZE**: maintain concentric reduction
  3. **SAFELY**: in a non-traumatic fashion[1]
  4. **WAY**: refer to pediatric orthopedics

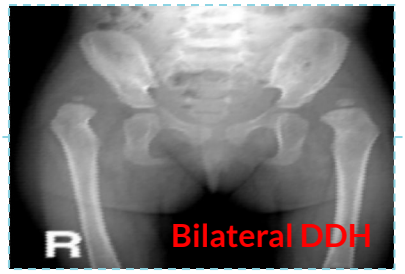
! Important points

- ➔ Method depends on age.
- ➔ The earlier started the easier and better the results.
- ➔ Should be detected EARLY.
- ➔ Could be surgical or non-surgical if you detected early the surgical management is less likely

<table>
<tr><td>Birth – 6 months</td><td colspan="3">• Reduce + maintain with Pavlik harness or hip spica (H.S) in the OR.
• In the clinic and pt is awake, you do ortolani then if it works stabilize the hip.
• First 6 weeks with Pavlik harness then abduction splint for 3 months then we follow up the patient.</td></tr>
<tr><td>6-12 months</td><td colspan="3">GA (general anesthesia) + Closed reduction + maintain with hip spica
• If it fails, we remove fibrous tissues and do an open reduction
• Why we give GA? because we have to do arthrogram (check presence of fibers)
• If the arthrogram showed presence of fibers we might do an open reduction

We do not use pavlik harness because the family can release it and we need to repeat the process and give the child GA again</td></tr>
<tr><td>12 - 18 months</td><td rowspan="4">GA
+
Open reduction</td><td rowspan="4">maintain with hip spica</td><td>Open reduction due to fibers presence</td><td>-</td></tr>
<tr><td>18 – 24 months</td><td rowspan="2">Acetabuloplasty</td><td>-</td></tr>
<tr><td>2 - 8 years</td><td rowspan="2">femoral shortening[2]</td></tr>
<tr><td>Above 8 years</td><td>Acetabuloplasty (advanced)</td></tr>
<tr><td colspan="4">Some hospitals and countries don't treat DDH after 8 years bc there will be erosions and abnormal acetabulum so even if you correct the femur the rate of success is very low. The patient will have to wait to do total hip replacement</td></tr>
</table>



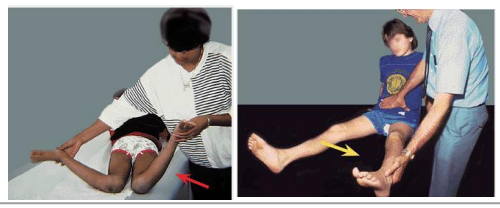
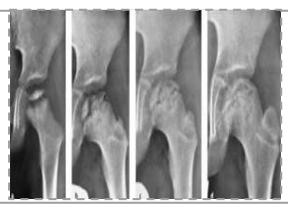


1- So if you try to reduce and it was tight don't try very hard or you will cause AVN
2- At this age the patient start walking, and with walking the femur will move up more and more, so you need to put it back to its place and shorten it

Complications: Important !

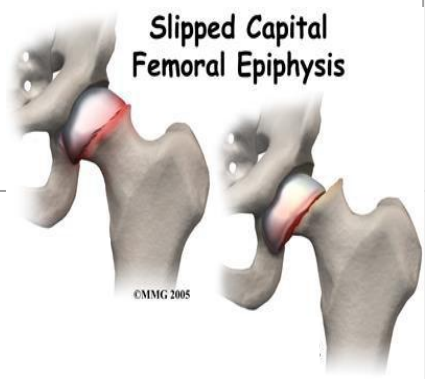
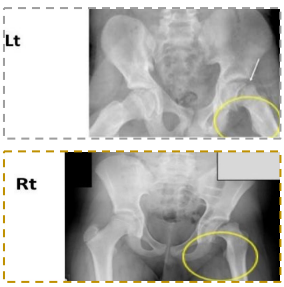
1. Severe pain
2. Early arthritis
3. Leg Length Discrepancy
4. Pelvic Inequality
5. Early lumbar spine degeneration

Legg-Calvé Perthes sease

Legg-Calvé Perthes Disease	
Where?	At the level of head of the femur.
Why?	↓ Vascularity of head of the femur (avascular necrosis)
Causes	Unknown (it's ischemia due to unknown cause that will resupply the tissue however it will be too late)
Risk factors	• 4-8 years • Males • Obesity
Severity	Depends on the amount of femoral head involvement (determinant of prognosis)
History	• Hip pain • Knee pain
Clinical Examination	• Inability for weight bearing • Decrease internal rotation (IR) • Decrease abduction. • Usually painful ROM 
Investigations	**X-ray early X-ray might not show anything** • Pelvis (decreased and irregularly shaped femoral head) • Knee (normal) **MRI** • Used if X-ray is not showing anything (early case) 
Treatment Controversial	**Refer to orthopedic as an urgent case** • Control pain until the body heals the lesion by itself [1] • Maintain ROM to maintain the circular shape of the femoral head • Hip containment inside the acetabulum • If outside we need to do surgery
Late Complications	• Early arthritis • Leg Length Discrepancy (LLD) • Pelvic inequality • Early Lumbar spine degeneration 

1- The blood supply will decrease → some cells will die → the body try to create new blood vessels → the blood supply will return → the dead cells will be reabsorbed → new cell will be formed → the femoral head will go back to its normal shape by remodeling. This process will take up to 4 years. (We need to control it by keeping the hip maintained & reduced + good ROM)

Slipped Capital Femoral Epiphysis:

Slipped Capital Femoral Epiphysis	
Where?	At the level of the growth plate (Between head and neck of femur)
Causes	• Hormonal • Metabolic
Risk factors	• 8-12 years • Males • Obesity • Black • If other side is affected 
History	• Hip pain • Referred knee pain • Minor trauma or no trauma • Painful limping
Clinical Examination	• Inability for weight bearing • Hip in ER (external rotation) • Decrease internal rotation (IR). • Decrease abduction. • Usually painful ROM.
Investigations	**X ray** [1] • Pelvis ➔ **Early**: Normal or increased growth plate (preslip phase) ➔ **Late**: positive slippage • Knee (normal) [2]  **MRI** • Used if X ray is normal or doubtful • If the Hx and PE suggest SCFE and X-ray is normal we do MRI (early cases)
Treatment	Refer to orthopedic as an **emergency** case • In situ pinning – to prevent further damage to the vascularity - Might affect growth slightly (but very crucial) • Protected weight bearing for 3-4 weeks then full weight bearing • No sport for 6 months
Late Complications	• Femoral Acetabular Impingement (FAI). • Early arthritis • Leg Length Discrepancy (LLD). • Pelvic inequality. • Chondrolysis. • Early Lumbar spine degeneration 

1- On the Lt side, there is widening of growth plate (pre-slipped stage) → needs MRI. The Rt one is worse (there is slippage) which increases the risk of early arthritis

2 If the pt comes complaining of knee pain (referred) and I did an X-ray and it was normal, what is the next step? pelvic X-ray

Quiz

Q1: which of the following is a proven risk factor for DDH ?

A. Oligohydramnios
B. Negative family history
C. 3rd child
D. Baby male

Q2: What is the most sensitive test in a 4-months-old child with suspicion of reducible DDH ?

A. Limited abduction
B. Ortolani
C. Barlow
D. Galeazzi

Q3: An 8-months-old child brought by his parents because of painless limping that was noticed recently. Which one of the following test will be positive?

A. Galeazzi
B. Ortolani
C. Barlow
D. Thompson

Q4: A 16-months-old baby has DDH, which of the following is a suitable management plan?

A. Pavlik harness
B. ORIF
C. Open reduction w/acetabuloplasty
D. Open reduction w/o acetabuloplasty

Q5: An 8-years-old child came to the ER with painful limping, limited abduction and painful ROM. An X-ray was done (shown in image). Which of the following is the most likely diagnosis?

A. Perthes disease
B. DDH
C. SCFE
D. Septic arthritis

1:A 2:B 3:A 4:B 5:C

Team Leader

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Done by

Abdulmohsen Albesher

Organized by

Abdullah Alomran

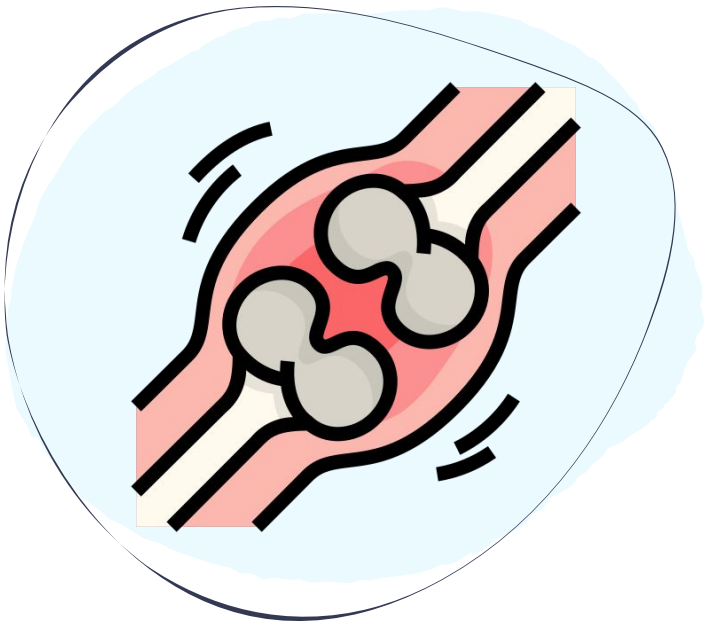
وفّقكم الله

12

Orthopedic
441 Team

MED441
KING SAUD UNIVERSITY

Editing File



Inflammatory & Degenerative Joint Disorders

Dr. Ahmad Bin Nasser

Color Index:

- Main Text
- Important
- 441 Notes
- Old Notes
- Extra

Objectives

No objectives were provided for this lecture.

Resources

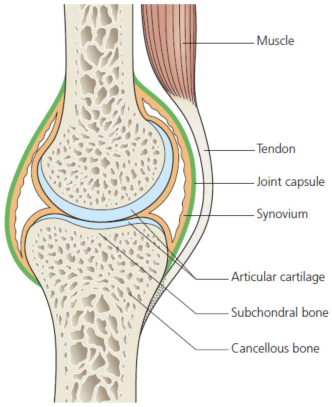
Osteoarthritis
By Osmosis

Osteoarthritis
By Orthobullets

Degenerative Joint Disease

Introduction

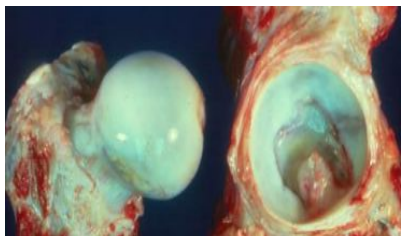
- Synovial joints are the most common type of joint in the body, these joints are termed diarthroses, meaning they are freely mobile.
- The joint is made up of different structures that act together to move, lubricate, nourish and stabilize the joint.



Components of Synovial Joints

1- Articular Cartilage

Cartilage is solid not liquid

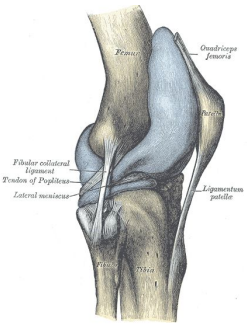


- Hyaline cartilage. The hyaline cartilage is not replaceable, if damaged it's replaced by fibrocartilage.
- Viscoelastic material with variable load-bearing properties.
- Decreases joint friction.
- Avascular and aneural (no nerve ending or vessels).
- Chondrocytes have little capacity for cell division in vivo (even stem cells).
- Direct damage to the articular surface is poorly repaired, or repaired only with fibrocartilage.
- Fibrocartilage has inferior biomechanical properties than hyaline.
- If the collagen network is disrupted, the matrix becomes waterlogged and soft, followed by loss of proteoglycans, cellular damage and splitting ('fibrillation') of the articular cartilage, damaged chondrocytes begin to release matrix-degrading enzymes[1].

Cartilage Composition	
Water (60-80% net weight)	• Pumped in and out of cartilage depending on load. • Contributes to lubrication and nutrition.
Collagen (10-20% net weight)	• Secreted by chondrocytes. • Mostly type-II collagen (90%). • Confers tensile strength to cartilage.
Proteoglycans (10-15% net weight)	• Secreted by chondrocytes. • Composed of GAG (aggrecan, chondroitin and keratin sulfate). • Negatively charged proteins hold water within the matrix. • Provides compressive strength.
Chondrocytes (5% net weight)	• The only cell type in cartilage.

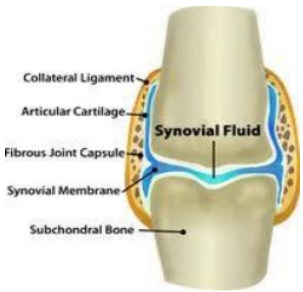
1- Mainly the loss of cartilage is progressive but sometimes the loss happens very quickly like in septic arthritis when the bacteria and the enzymes released by bacteria and phagocytosis cause direct damage to the joint.

2- Capsule and Ligaments



- Fibrous structure with tough condensations on its surface (ligaments).
- Together with the overlying muscles, help to provide stability.
- Ligaments provide stability to the joint when it's torn we lose a major stabilizing factor.

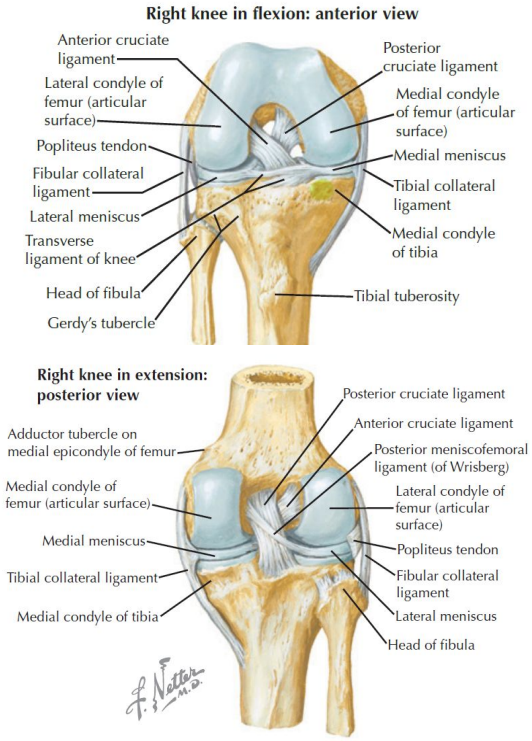
3- Synovium

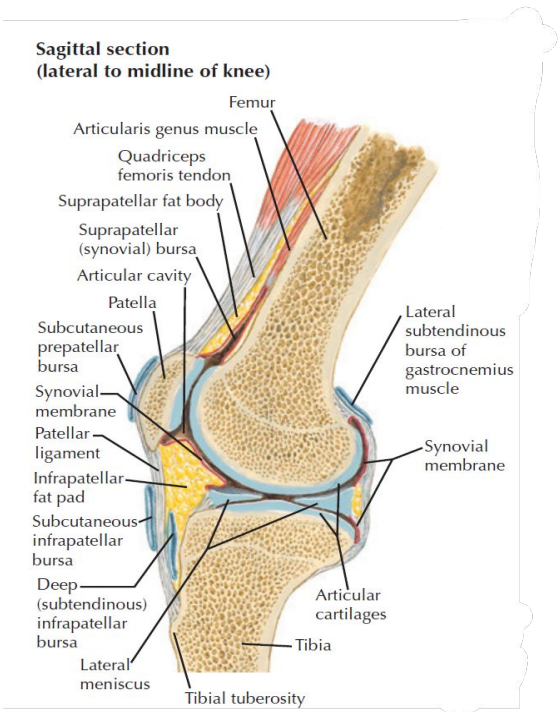


- Thin membrane.
- Richly supplied with blood vessels, lymphatic and nerves.
- Target tissue in joint infections and autoimmune disorders such as rheumatoid arthritis.
- Provides a nonadherent covering for the articular surfaces.
- Produces synovial fluid.

4- Synovial Fluid

- Synovial fluid nourishes the avascular articular cartilage.
- Plays an important role in reducing friction during movement.
- Has slight adhesive properties which assist in maintaining joint stability.
- Synovial fluid functions: Reduce friction, supply nutrition and lubrication.
- The volume remains fairly constant, regardless of movement.
- When a joint is injured fluid increases (joint effusion).
- Increases in case of inflammation (OA), lymphatic obstruction.
- It NEVER decreases.
- Effusion: Increase fluid inside the capsule (Joint effusion).
- Swelling: Increase fluid in tissues outside of the joint.





Osteoarthritis

Primary Idiopathic Osteoarthritis

Chronic disorder, characterized by:

- Progressive softening and disintegration of articular cartilage.
- New growth of cartilage and bone at the joint margins (osteophytes[1]).
  - ➔ Which leads to the loss of the congruency of the articular surface.
- Subchondral bone sclerosis and cyst formation.
- Mild synovitis and capsular fibrosis.
  - ➔ Synovium becomes inflamed and produces more fluid as a protective mechanism.
- Asymmetrically distributed, often localized to only one part of a joint.
  - ➔ The place where the highest pressure is located at, on the hip it will affect the top part more, if it's fully damaged search for another reason.
- Often associated with abnormal loading.
  - ➔ Usually causes of OA is more mechanical (weight and loading), whatever part is loaded more will have early osteoarthritis like, in obese patients patello-femoral part will be affected.

- Unaccompanied by systemic illness.
- Not primarily an inflammatory disorder although there are sometimes local signs of inflammation.
- Not purely degenerative, it's a dynamic phenomenon, it shows features of both destruction and repair.
- Osteoarthritis patients have these 5: 1- Cardiac diseases, 2- DM2 & obesity, 3- Depression, 4- Muscle weakness, 5- Peptic ulcer & Kidney diseases "with treatment".

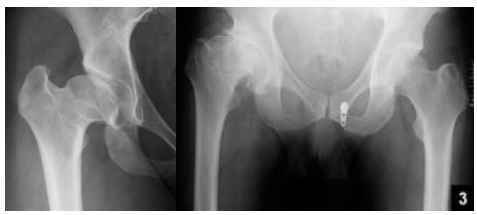
Etiology

1. Increased mechanical stress in some part of the articular surface (abnormal loading).
2. **Disparity between the mechanical stress** to which the articular cartilage is exposed and the ability of the cartilage to withstand that stress.
3. Varus deformity of the knee (that's why the medial side is usually only affected).
4. More of a process than a disease.
5. Increase in frequency with age.
6. Obesity (hips and knees take 3-4x body weight with each step).
7. Family history.

Secondary Osteoarthritis

- Main difference is total symmetric joint involvement (not only medial), universal cartilage loss not due to mechanical stress.

Etiology

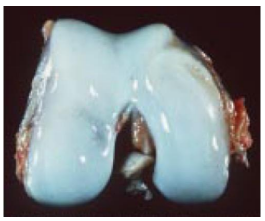


1. Metabolic: Crystalline deposition disease (gout[2], CPPD[3]), Paget's disease.
2. Inflammatory: Rheumatoid arthritis, SLE, Reiter's syndrome.
3. Neuropathic: DM, tabes dorsalis.
4. Hematologic: Sickle cell disease, hemophilia.
5. Endocrine: DM, acromegaly.
6. Trauma: Malunion, osteochondral, sport injury, dislocation, Meniscal tear (Post meniscectomy → OA).
7. Congenital/developmental: Hip dysplasia "DDH", multiple epiphyseal dysplasia.
8. Infection: (Septic arthritis).
9. Necrosis: Perthe's disease, osteonecrosis, steroids.

1- To distribute the pressure.
2- Monosodium crystals with translucent aspiration.
3- Mostly.

Prevalence

- Osteoarthritis is the commonest of all joint diseases (it's a disease of the cartilage not the synovial fluid).
- Osteoarthritis is much more common in some joints (hip, knee, spine and the fingers) than in others (the elbow, wrist and ankle).
- Much more in **females**, also more joints are affected in **women** than in men.
- Common in our community especially knees (presents earlier than in West).
- About 90% of those over 40 have asymptomatic degeneration of weight bearing joints.
- Commonest joints are knee, hip, cervical spine & lumbar spine, 1st Carpometacarpal, 1st Metatarsophalangeal and Interphalangeal joints.



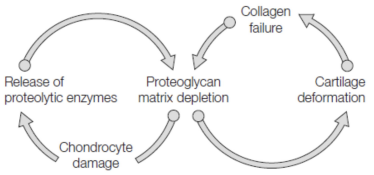
Normal knee cartilage

Pathology

Cardinal Features

1- Progressive Cartilage Destruction (poor cartilage regeneration)

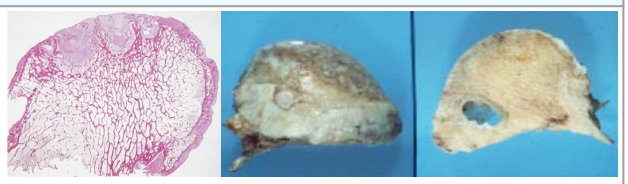
- **Increased** water content which leads to swelling and softening of the cartilage.
- This will lead later on to the **depletion**/loss of proteoglycans.
- Chondrocytes will be damaged and cause synovitis → Release of proteolytic enzymes → Collagen disruption.
- Fibrillation on weight bearing surfaces.
- Destruction of the cartilage → **secondary** inflammation and formation of osteophytes.



2- Subarticular Cyst Formation (doesn't appear in X-rays except in the hip)

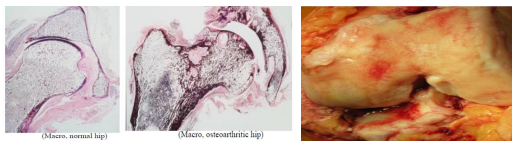
Arises from:

- Local areas of osteonecrosis.
- Forceful pumping of synovial fluid through subchondral bone plate **cracks**.



3- Sclerosis of The Surrounding Bone (seen in all X-rays)

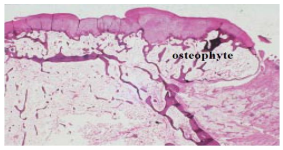
- Bone becomes exposed, may be polished or burnished to ivory-like smoothness (eburnation).



4- Osteophyte Formation (seen in all X-rays)

- Proliferation and remodeling of the adjacent cartilage at the edges.
- This will be followed by endochondral ossification.

➔ The joint try to increase the surface area to distribute the stress so it forms a cartilage at the periphery of the joint which underwent ossification and became a bone.



5- Capsular Fibrosis

- Marked vascularity and venous congestion of the subchondral bone (causes pain).
- The capsule and synovium are often thickened but cellular activity is slight.
- Progressive bone erosion → **BONE COLLAPSE**
- Fragmented osteophyte → **LOOSE BODIES**
- Loss of height and ligamentous laxity → **MALALIGNMENT**

Clinical Features

- Intermittent course, with periods of remission, sometimes lasting for months.
- One or two of the weight-bearing joints (hip or knee) might be affected.
- In our community patients having degenerative knee will have degenerative lumbosacral & cervical spine. So, in a knee surgery check spine and vice versa.
- Pain of OA starts in one of two ways, either suddenly after an event (I did something I'm not used to e.g. Marathon) or progressively (day after day).
- Localized **progressive** pain during or after exertion (e.g. Prolonged walking, at the end of the day) that is relieved with rest.

Symptoms

The most common problem is pain and swelling

1- Pain

- Localized or rarely referred to a distant site (e.g Pain in the knee from hip osteoarthritis).
- Insidious in onset.
- Aggravated by exertion and relieved by rest, so the patient will stop any activities or painful movements to avoid pain → Muscle wasting and stiffness (further worsening).
- In advanced stages there will be night pain or pain at rest.
- The pain is fluctuating (never constant) and not continuous especially during the extremities of motion.

Causes of Pain:

- Bone pressure due to vascular congestion and intraosseous hypertension "**most important**".
- Mild synovial inflammation.
- Capsular fibrosis with pain on stretching the shrunken tissue.
- Muscular fatigue[1].
- Friction.

2- Stiffness

- Initially after periods of inactivity, but later on it will be constant and progressive.
- The worst advice you give the patient is to tell him/her not to use the affected joint, as a result the patient will develop stiffness, weight gain, muscle loss and the symptoms will be worse!

3- Loss of Function

Signs

- Swelling: Intermittent = Effusions, Continuous = Large osteophytes.
- Deformity → Mal-alignment (Primary OA = Varus "genu varus" or "bow legs").
- Tenderness.
- Limited range of movement.
- Crepitus
  - ➔ Joint crepitation is not an indication of OA unless it is accompanied by pain or swelling, or limited ROM.
- Instability
  - ➔ Due to loss of cartilage and bone, asymmetrical capsular contracture and/or muscle weakness.

1- Might be muscle pain, the joint is weak so the muscles contract for a long time, causing pain and fatigue. So the patient avoids fully flexing and extending, at the end they will give up and don't try to move, that's why physiotherapy and exercise is important, it also increases the capacity of the muscle and strengthens it to support the joint, so more movement before feeling pain. Muscle relaxation by message will relief the pain temporarily but won't treat the primary cause.

Imaging[1]

- Used to confirm the diagnosis and rule out other causes (NOT to treat).
- Used to support/confirm the clinical diagnosis (DOES NOT GUIDE MANAGEMENT PLAN).
- Two perpendicular views (AP & lateral), and the other limb to compare.

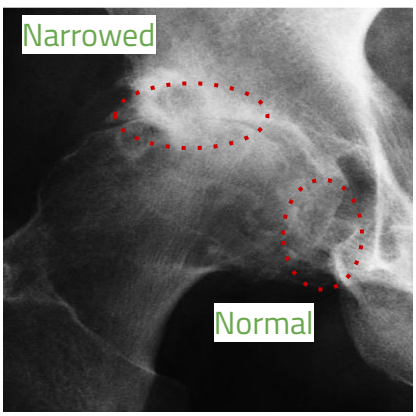
Early

- Asymmetrical loss of cartilage (narrowing of the 'joint space').
- Subchondral bone sclerosis.
- Cysts close to the articular surface.
- Osteophytes at the margin of the joint.

WEIGHT BEARING X RAY

Late

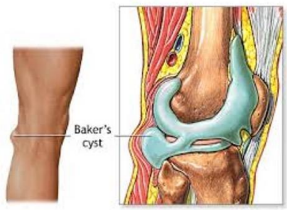
- Malalignment.
- Joint subluxation.
- Bone loss.
- Loose bodies.
- Signs of other disorders.
- Symmetric narrowing in inflammatory OA e.g. Rheumatoid arthritis.

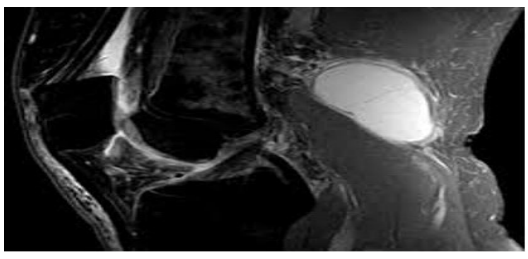


Grade 4 is bone on bone, while grade 1 is only seen on a MRI, X-Ray is normal

Complications

- Capsular herniation: Knee OA; marked effusion and herniation of the posterior capsule (Baker's cyst).
  - ➔ Very common, synovial fluid finds a weak area and goes there.
- Loose bodies
  - ➔ Arthroscopy is done if there are mechanical symptoms (Loose bodies).
  - ➔ Can't move because pebble like structures are preventing it from moving.
- Rotator cuff dysfunction in acromioclavicular joint OA.
- Spinal stenosis.
- Spondylolisthesis.
  - ➔ Severe segmental disability; at L4/L5





1- Imaging is X-Ray and weight bearing **(standing) X-Ray**. Standing X-ray is always better, if you are searching for thinning of cartilage, do weight bearing X-ray to indirectly see the cartilage (you will see the space occupied by the cartilage). MRI is indicated if X-ray is normal.

Management

Depends on: 1- Joint (or joints) involved, 2- Stage of the disorder, 3- Severity of the symptoms, 4- Age of the patient, 5- Functional needs.
2 Factors that you can control (confirmed by literatures): 1- Weight reduction, 2- Muscle strengthening.

Early Treatment

- Maintain movement and muscle strength.
- Protect the joint from 'overload'.
- Relieve pain: Analgesics (1- Ice packs, 2- Paracetamol, 3- NSAIDs).
- Modify daily activities (If obese lose weight, avoid carrying heavy weight, strengthen muscles).

Conservative Management

1- Maintain Movement and Muscle Strength by Physiotherapy

This will help in:

- Pain relief through massage or application of warmth.
- Prevent contractures.
- Muscle strengthening.
- Range of motion.
- It's recommended to do low weight bearing exercises, e.g. Swimming.

2- Load Reduction: Never advice the patient to stop moving or to be immboile

- Weight reduction (if the patient is obese).
- Shock-absorbing shoes.
- Walking stick (carry the stick using the hand on the unaffected side).
- **Unloading brace** (if the knee is in varus, it pushes knee to valgus).
  - ➔ Failed to show any benefits.
- Not bearing heavy objects.

3- Modify Activities and Sitting Habits

- Modify activities and avoid others (pray using a chair for example).
- Change sitting habits (AVOID sitting on the floor 'تربيعه' for example, and prolonged sitting in one position, and sitting in extremes of motion).

4- Medications: Pain relief only as needed

- **Oral**: Paracetamol, NSAIDs (e.g. Ibuprofen), muscle relaxants, narcotics, supplements and herbs.
  - ➔ Supplements has no effect on the disease (placebo effect).
- **Injections**: (Local) **not** recommended in general.
  1- Steroids: It is used for patients who can't use NSAIDs either for kidney disease or peptic ulcer.
  2- Hyaluronic acid injection (oil injection/filler): The goal is to relief pain, it might be an option for patients with early disease or those who can't take medications (not effective).
  3- Plasma: Take blood from patient and separate plasma, limited effect on pain relief (not effective).
  4- Stem cells: No proof of effectiveness.

"Remember injection is not a part of standard treatment of OA"

- If conservative treatment fails and the patient is in pain, we can proceed to surgical management or if the pain started affecting patients quality of life.
- We shouldn't proceed to surgical management based on radiological features alone even if there's severe OA features, the only exception to this is if there's extensive bone resorption along with severe malalignment.

Surgical Management

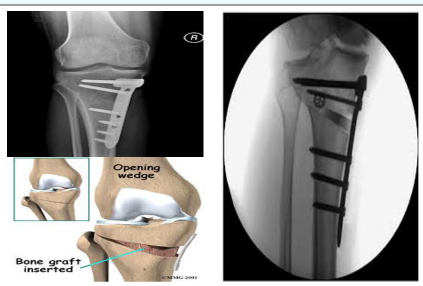
1- Joint Debridement (Arthroscopy): Honeymoon surgery

DON'T DO IT, only limited cases, not common procedure, done under GA, done commonly in private hospital, not indicated and will not correct the disease, used only if there is indication for it (blocking or latching of the joint).

- Removal of loose bodies.
- Removal of meniscal or labral tears.
- For mechanical symptoms.
- Prior to corrective osteotomy to check for areas of osteoarthritis, if generalized we don't proceed.

2- Corrective Osteotomy

- Realign axis and distribute weight.
- We offload the arthritic part, and we load the uninjured part.
- Knee and hip are the common sites.
- Candidate: **Young**, active, mild OA "for early stages".



Minor role in pain relief:

- Vascular decompression of the subchondral bone.
- Redistribution of loading forces towards less damaged parts of the joint.

3- Arthrodesis

- Transfer from painful stiff into painless stiff joint (leads to loss of motion).
- Small joints: Hand (wrist joint), foot and spine (1st CMC).

4- Joint Replacement (Arthroplasty): GOLD STANDARD

- Nowadays the procedure of choice for advanced OA.
- **(indicated if the patient's quality of life didn't improve with the non-surgical options)**

Total joint replacement:

- Knee, hip, shoulder, ankle and elbow.
- **Candidate:** Painful, deformed stiff joint, **old patient**.

Partial joint replacement:

- Knee.
- **Candidate:** Same as for osteotomy (Young, active, mild OA).

Excision arthroplasty:

- Resection arthroplasty.
- Thumb, acromioclavicular joint, hip.

441 Doctor Notes

Osteoarthritis one of the most common topics about musculoskeletal disorders.

It is degeneration of what?? Of the articular (hyaline) cartilage, not bone, not fluid.

One of the most common misunderstandings and complete opposite advices that given to the patient is to avoid walking or remain sedentary!!

Degeneration of cartilage is a progressive disease, once it starts we cannot stop it and we cannot reverse it.

So, the goal of treatment in osteoarthritis is:
1- Delay the progression of the disease.
2- Symptoms management rather than stopping the disease.
3- Enhance the quality of life.

What advice would you give a patient recently diagnosed with osteoarthritis to slow down the progression of disease and improve his symptoms?
1- Maintain movement.
2- Muscle strengthening.
3- lose weight.

Physical inactivity complications:
1- Muscle weakness
2- Obesity
3- Cardiovascular diseases
4- Diabetes
5- Depression

Quiz

Q1: Which of the following non-operative treatments for osteoarthritis has the best evidence to support its use?

A. Combination of supervised and home exercise programs

B. Hyaluronic acid injections

C. Paracetamol

D. Lateral heel wedge

Q2: According to the latest recommendations made for the treatment of osteoarthritis (OA) of the knee, which of the following nonoperative treatment modalities has the weakest supporting evidence for the treatment for knee osteoarthritis?

A. Weight loss

B. Activity modification

C. Intra-articular corticosteroids injection

D. Intra-articular hyaluronic acid injection

Q3: A 61-year-old man with progressive left hip pain comes to the physician for a follow-up examination. One year ago, he was diagnosed with osteoarthritis of the left hip. Since then, he has had an 8-kg (18-lb) weight loss after changing to a vegetarian diet, regular swimming, and physical therapy. The pain worsens when he climbs stairs, which makes it increasingly difficult for him to reach his apartment located on the second floor. Over the last few weeks, he gradually increased the frequency of diclofenac intake but says that even a daily intake does not provide complete pain relief. He asks if there is a treatment that will lead to a long-term improvement of his symptoms. He has no history of major medical illness. His only other medication is pantoprazole. He does not smoke or drink alcohol. He is 179 cm (5 ft 10 in) tall and weighs 80 kg (176 lb); BMI is 25 kg/m2. Physical examination of the left hip shows crepitus, a limited internal rotation, and pain with full flexion and extension. An X-ray of the left hip shows joint space narrowing, osteophytes, and subchondral sclerosis and cysts. Which of the following is the most appropriate next step in management?

A. Refer for arthroscopic hip debridement

B. Perform intra-articular glucocorticoid injections

C. Prescribe walking aids

D. Refer for total hip arthroplasty

1:A 2:D 3:D

Quiz

SAQs

441:

There was a SAQs but the doctor didn't give it to us due to lack of time.

439:

Case…

1. What advice would you give a patient recently diagnosed with osteoarthritis to slow down the progression of disease and improve his symptoms?
   - Maintain movement and muscle strength, lose weight.

2. Please describe the role of radiological investigations in the diagnosis and management of degenerative joint diseases, and mention any relevant technical aspects when requesting these investigations.
   - Imaging is used for confirmation, not for diagnosis. We can use it to differentiate between primary and secondary osteoarthritis. Request images on weight-bearing.

Team Leader

Abdulrahman Alroqi

Done by

Sultan Ahmed

Organized by

Abdulrahman Alroqi

وفّقكم الله

13

Orthopedic 441 Team

MED441 KING SAUD UNIVERSITY

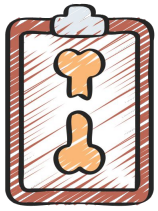
Editing File

Management of Multiple Injured Patients

Dr. Syed Amir Ahmad

Color Index:

- Main Text
- Important
- 441 Notes
- Old Notes
- Extra



Objectives

- Learn to diagnose, start initial management and know when to refer a patient.
- Implement Management as per ATLS protocol.
- Immobilization of cervical spine, in the context of managing the airway.
- Understand the function of spinal board as a transfer tool only.
- Understand Importance of interpersonal communication skills.

Resources

Trauma Overview:

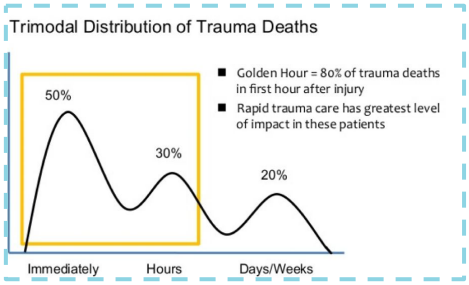
- **Injury:** The result of a harmful event that arises from the release of specific forms of energy.
- **Polytrauma:** Multisystem trauma = injury of two or more systems, or system with deranged vital signs.
- **Scope of the Problem:**
- **UK:** > 18, 000 deaths annually, > 60, 000 hospital admission, > Costing 2.2 billion pounds.
- **USA:** > 120, 000 deaths annually, > 100 billion dollars.

Mechanisms of traumatic injury (Type of Injury):

1. **Penetrating**: Open injury, with direct injury to underlying structures (Liver Laceration)
2. **Blunt** (most common):
   - Closed injury, Indirect injury to underlying structures (Spleen rupture)
   - Transmission of energy into the body (Tearing of the muscles, vessels and bone). (Rupture of solid organ), (Organ injury: ligamentum teres).
- **Blast** (Bombs)
- **Thermal** (burns): heat or electrical
- **Chemical**
- **Others** - crush & barotrauma (e.g. Diving).

Trimodal Death Distribution:

1st peak **Immediate death** (0 to 1 hour)	2nd peak **Early death** (1 to 3 hours)	3rd peak **Late death** (1 to 6 weeks)
• Accounts for **50%** of deaths. • Occurs **within minutes** of injury. • Due to **major neurological or vascular injury** (unsurvivable injuries). • Medical treatment can rarely improve outcome **(die on scene)**	• Accounts for **30%** of deaths. • **Occurs during the golden hour**[1]. • Due to **intracranial haematoma, major thoracic or abdominal injury**. • Primary focus of intervention for the **ATLS** methodology.	• Accounts for **20%** of deaths. • Occurs after **days** or **weeks**. • During staying in **ICU** and due to **complications (sepsis and multiple organ failure)**.



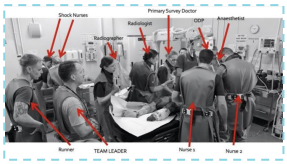
First 1 to 2 hours is the golden hours

Why is it called "the golden hour"? Hence death in this peak is mostly due to hypoxia and loss of blood (hypovolemic shock) which is **potentially preventable**. (80% of deaths happen here.)

1st peak causes immediate death in minutes due to vascular damage like Aortic rupture.
2nd peak happens within 1-3 hrs after the trauma and this is where if ATLS is performed right would save a life.
3rd peak happens in days and is due to infections and sepsis

1- Here's the thing, patients who die in the first peak often die from severe traumas, (like decapitation for example) which means most of the time you cannot save those patients. However, patients who die in the second or third peaks CAN be saved you manage them correctly IN THE GOLDEN HOUR. It's called the golden hour cause it gives you a chance to save the patient.

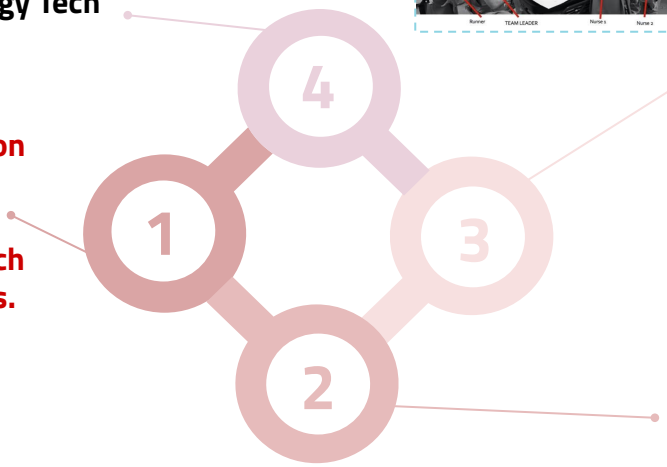
The Trauma Team:



Radiology Tech

The Team Leader[1]**:**

1. Most **experienced**
2. Preferably a **Trauma surgeon or a general Surgeon**
3. Takes all **triage decisions.**
4. **Should be familiar with each member's skills and names.**
5. **Prioritizes procedures**
6. Communicate with consultants and family members



Doctors:

- EM physician
- Anesthetist
- Orthopedic surgeon
- General surgeon.

Nurses[2]: from 3-5 at least

Prehospital Management:

- **The goal of prehospital management:**
  - Gain access to the patient.
  - Smooth transfer.
- **Approaches:** a balance between these two approaches is better.
  1. "Scoop & Run policy" (Take patient to the hospital ASAP, you can give him oxygen mask but we don't do any major intervention here until patient reaches the hospital).
  2. "Stay & Play policy" (Needs expert EMS to do this, full management on scene like intubation etc....).

ATLS - Components Steps: [3]

1. **Primary Survey: Identify what is killing the patient.**
2. **Resuscitation: Treat what is killing the patient**
3. **Secondary Survey: Proceed to identify other injuries**
4. **Definitive Care: Develop a definitive management plan**

*if patient situation during the 2ry survey becomes worse, we return back to the primary survey.

Organisation of Trauma Centers:

1. **Level 1 -** Regional Trauma Centres (All specialities are available and it has a designated research center, polytrauma should be admitted here)
2. **Level 2 -** Community Trauma Centres (Major specialities are available)
3. **Level 3 -** Rural Trauma Centres

Multiple Casualties:

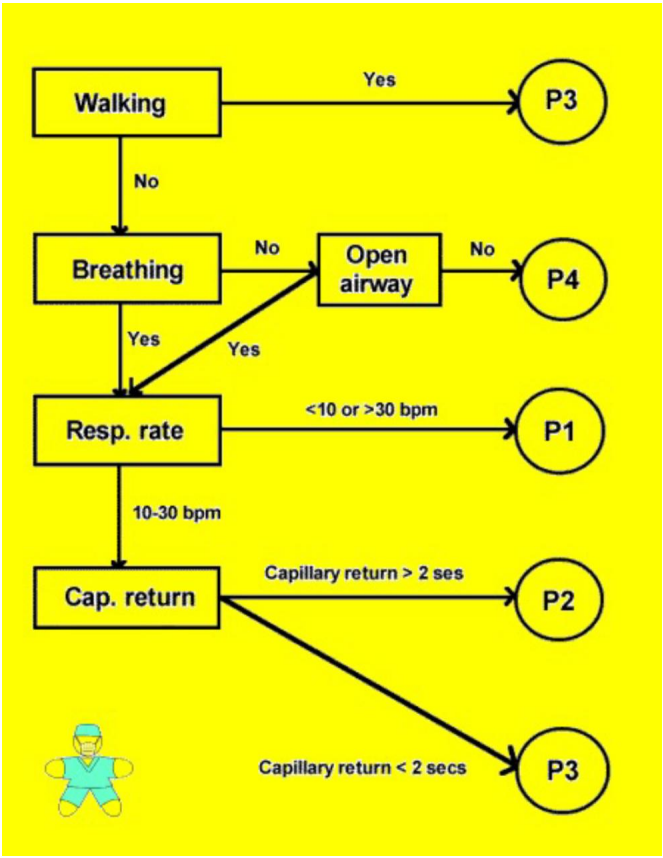
- **Several casualties at the same time** (Multiple injured patients came to the hospital at the same time):

1. **Alert ED services**
2. **Assess the scene - without putting your safety at risk**
3. **Triage 'do the most for the most'**

1- The trauma Surgeon is the team leader in most hospitals, if he wasn't present an EM consultant can be the team leader.
2- 5 Nurses As: 1. Airway 2. Circulation 3. Medication 4. Time Documentation 5. Backup and fetcher (go between)
3- The basic philosophy of ATLS is to save life, followed by saving limb then function. The idea is to prioritize care to the most life threatening injury first, reassess then treat again or move to the next injury. Transfer the patient if the injury requires surgery or any other procedure that you cannot perform.

Triage (Start Triage Algorithm):

- **Ability to Walk**
- **Airway**
- **Respiratory Rate**
- **Pulse rate or Capillary return**



How to Triage?

1. **Can the patient walk?**
- **Yes** → Delayed
- **No** → Check for breathing

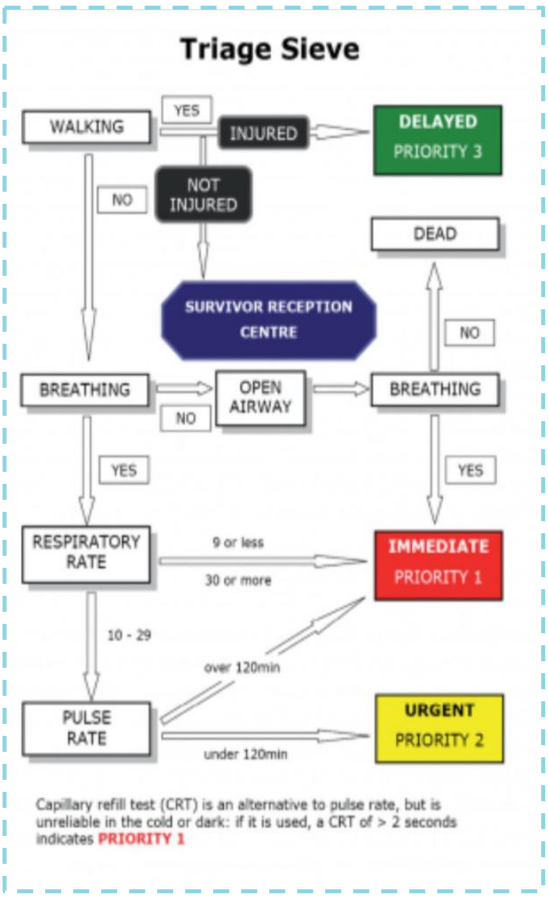
2. **Is the patient Breathing?**
- **No** → Open the airway
  - Are they breathing now?
    - Yes → IMMEDIATE
    - No → DEAD
- **Yes** → Count the rate
  - Less than 10 bpm or **More than** 30 bpm → IMMEDIATE
  - **Between** 10 - 30 bpm → Check Circulation

3. **Check the Circulation?**
- Capillary refill **More than** 2 sec → IMMEDIATE
- Capillary refill **Less than** 2 sec → Urgent

Trimodal Death Distribution:

1. **Triage Sieve (on the field)** - To separate dead & the walking from the injured.
2. **Triage Sort (2nd step)** - To categorize the Casualties according to local protocols:

- Cat 1: **Critical & Cannot wait.**
- Cat 2: Urgent - Can wait for 30 minutes at most
- Cat 3: Less serious injuries
- Cat 4: Expectant - Survival not likely



Triage categories				
Cat	**Definition**	**Colour**	**Treatment**	**Example**
P1	Life-threatening	Red	Immediate	Tension pneumothorax
P2	Urgent	Yellow	Urgent	Fractured femur
P3	Minor	Green	Delayed	Sprained ankle
P4	Dead	White		

1- Make the area safe:

- **Protect yourself, the casualty and other site users**
- Park your car safely, turn lights on, set hazard lights flashing
- Do not across a busy motorway to reach other side
- Assign others to warn oncoming drivers.
- Set up warning triangles or lights 200 metres in each direction
- Switch off ignition of any damaged vehicle
- Is anyone smoking?
- Is there a chemical spill
- Are there live electrical wires
- Could there be a secondary incendiary device (Like if there was 2 bombs in a terrorist attack)

2- Check all Casualties:

- Quick assess (check MOI in accidents)
- Not moving
- Apply life-saving treatment

How to move unconscious casualty?

1. **Do not move** the casualty unless it is absolutely necessary
2. Assume **neck injury** until proved otherwise
1. support head and neck with your hands, so he can breathe freely
2. Apply a collar, if possible
3. There should be only **1 axis** (head, neck, thorax) no moving to sides, no flexion, no extension.
4. Move with help of 3-4 other people 1st person will support the head (he is directing others), the second person will support shoulders and chest, the third person will support hips and abdomen, the last person will support the legs.

Skill Video Demonstrations:

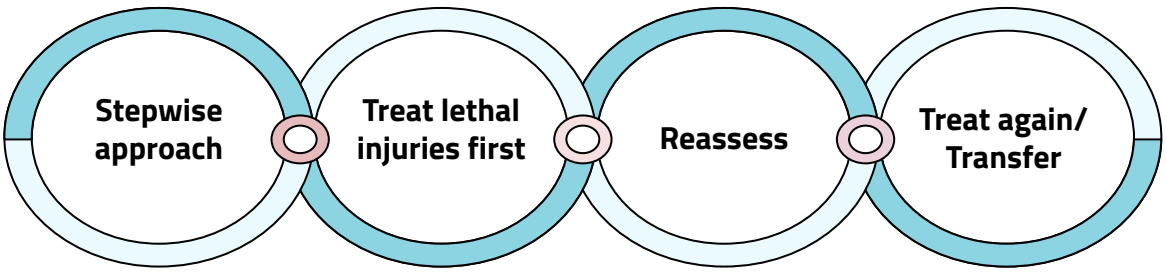
1. Applying Cervical Collar
- Log rolling and Spinal board
- Inline Immobilization
- Spinal Clearance (important)

Trauma Team CALL-OUT Criterion (in hospital):

- **Penetrating injuries**
- **Two or more proximal bone fractures**
- **Flail chest & pulmonary contusion**
- **Evidence of High energy Trauma:**

1. Fall from > 6 ft
2. Changes in velocity of 32 kmph
3. 35 cm displacement of side wall of car
4. Ejection of the patient
5. Roll-over
6. Death of another person in the same car
7. Blast injuries

ATLS Algorithm - Assessment Approach to Trauma Patient in Hospital Settings:



1 Primary survey & resuscitation (ABCDE):

1. **A**irway and securing cervical spine
2. **B**reathing
3. **C**irculation and haemorrhage control
4. **D**ysfunction of the central nervous system
5. **E**xposure.

Adjunct to primary survey (Only imaging permitted during this phase is):

- **X-ray:** AP supine chest, AP plain pelvic, Cross table lateral C- spine X-ray (outdated).
- **Ultrasound:** FAST has replaced peritoneal lavage for detecting intraperitoneal fluid of blood.

2 Secondary survey

(Full physical examination to identify any missed injuries)

3 Definitive treatment

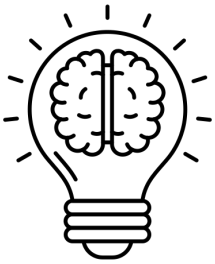
(In most cases surgical management)

4 Tertiary survey

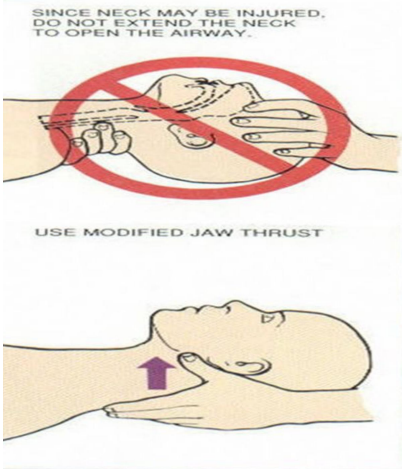
(Another full examination just to be safe)

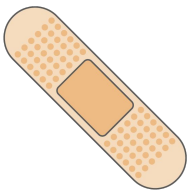
5 Consider Early Transfer

A- Airway



- **Always assume that patient has cervical spine injury**
- **If patient can talk** then he is able to maintain own airway
- If airway compromised initially attempt a jaw thrust and clear airway of foreign bodies, suction, adjuncts to open airways.
- Remember to avoid causing harm eg NP tube, nasopharyngeal airway in base skull fracture
- **Give 100% Oxygen** (face mask, bag valve)
- Assist airway & breathing including ''definitive airways'' (endotracheal tube/cricothyroidotomy)





- A – Airway maintenance & Control of C-Spine (By Applying cervical collar).
- If conscious - Ask the pt's name
- If unconscious - **Look for added sounds** (stridor,cyanosis etc)
- If the pt does not respond to any questions- resuscitate.

Airway maintenance steps:

Chin Lift (If No C Spine Concern) → Jaw thrust → Suction → Oropharyngeal/ Orotracheal Tube → Endotracheal Intubation → Crico-thyroidotomy → Tracheostomy

Manual In-line Stabilization:

Performed by an assistant during airway management to maintain a neutral position and prevent movement of the head and neck, by either:

1. Crouching beside the intubator with hands placed on the patient's mastoid processes (Pic.1)
2. Standing beside the patient in front of the intubator with hands placed on the sides of the patient's head and forearms resting on the patient's chest (Pic.2)

Pic.1

Pic.2

B- Breathing and ventilation

Assessment

- Exposure
- Inspection:
- Palpation
- Movement
- Auscultation

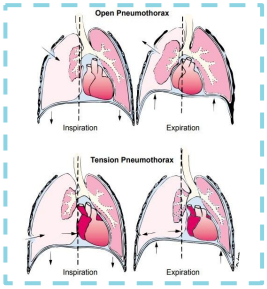
❖ The aim is to hunt out & treat the life threatening thoracic conditions which include :

Life threatening conditions that requires rapid intervention

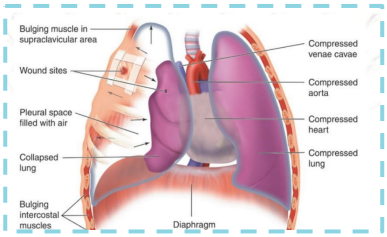
- **Massive Pneumothorax/ Hemothorax**
- **Tension Pneumothorax**
- **Open Pneumothorax**
- **Flail segment**
- **Cardiac Tamponade**

Doing Good 😉
Here's a space for your notes

1- Pneumothorax:



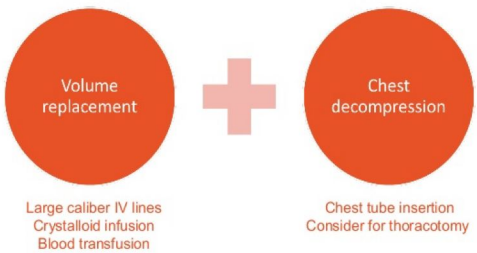
- **Tension Pneumothorax:**
  - Presence of air or gas in the pleural cavity.
  - Can impair oxygenation and/or ventilation.
  - Clinical results are dependent on the degree of collapse in the lung.
  - Pneumothorax is called tension if it was severe enough to shift the mediastinum & compromise hemodynamic stability.
  - Air can enter the intrapleural space through a communication from the chest wall (ie, trauma) or through the lung parenchyma across the visceral pleura.
  - **Clinical Features:**
    - Diminished breath sound (with hyperresonance)
    - Distended neck veins.
    - Hypotension.
    - Tracheal deviation.
    - Respiratory distress.

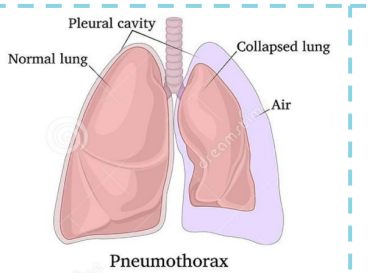


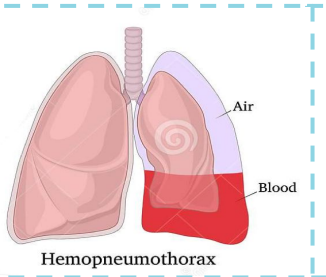
  - **Management:**
    - **Needle decompression:** Immediate needle thoracocentesis in 2nd intercostal space in mid clavicular line, followed by chest tube (definitive) in 5th intercostal space anterior axillary line.
    - Supportive management:
      - analgesia, ventilators support, CXR monitoring, Chest physiotherapy.
- **Open Pneumothorax:**
  - Treated by sealing the wound with occlusive dressing and tube thoracostomy.
- **Suction Pneumothorax:**
  - Sealing of the wound (3 sided occlusive dressing to create one way valve) and Tube thoracostomy

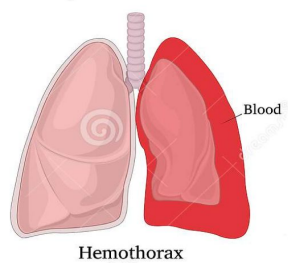
2- Massive Pneumothorax/ Hemothorax:

- **Massive hemothorax is defined by the need for thoracotomy in OR the indications are:**
  - Blood loss > 1,500 mL or 1/3rd of blood volume or
  - Blood loss >200 mL/h (3 mL/kg/h) for 2-4 hours.
- **Recognition:**
  - Hemorrhagic shock:
    - pallor, tachycardia, hypotension, cool peripheries pleural space.
  - Decreased chest movement, dullness and decreased breath sounds ipsilaterally.
  - External evidence of thoracic injury.
  - Persistent blood loss following intercostal cath.
- **Management:**
  - 2 large bore IVs with crystalloid infusion and blood transfusion + Chest decompression with chest tube insertion







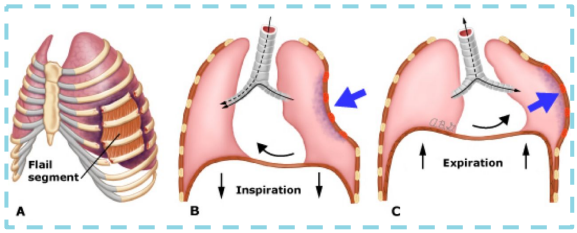


3- Cardiac tamponade:

- Almost always seen with a penetrating wound
- **Clinical Signs and Symptoms:**
  - **Beck's triad :**
    - Hypotension tamponade.
    - Distended neck veins.
    - Muffled heart sounds
- **Management:**
  - Needle pericardiocentesis then thoracotomy & repair as definitive management.

4- Flail segment:

- Fractures of 3 or more consecutive ribs in 2 or more locations.
- Paradoxical movement:
- **Recognition:**
  - Chest pain
  - Respiratory distress
  - Bony crepitus
  - Paradoxical chest wall movement
    - the segment moves inwards on inspiration as the rest of the chest expands and outwards on expiration as the rest of the chest deflates.



- **Management**
  - Requires an endotracheal intubation and mechanical ventilation[1]
  - Supportive care, O2
  - Stabilization of the segment with manual or object pressure
  - Postpositive pressure ventilation

Here's a space for your notes

1. Air is pushed in the patient through +ve pressure

C- Circulation and haemorrhage control

Assessment

- Pulse rate
- Capillary refill
- ECG monitor
- State of neck veins
- LOOK FOR BLEEDING and apply direct pressure
- Place two large calibre intravenous cannulas Give intravenous fluids (crystalloid or colloid)

Assessment of Blood loss

There are 6 keys area to look for evidence of bleeding:

- External or obvious
- Internal or covert
- Chest
- Abdomen
- Pelvis
- Limbs

- **Resuscitation :**
  - Arrest bleeding
  - Obtain vascular access

❖ Tachycardia in a cold patient indicates SHOCK

Types Of Shock Following Injury :

Hypovolemic	Obstructive	Cardiogenic	Neurogenic	Septic
(Most common in trauma) (Resuscitate)	(Mainly because Cardiac Tamponade or tension pneumothorax) (Relieve obstruction)	(Resus + inotropes)	(Resus + vasopressors)	

Consider up to 2 litres of fluids if patient is hypotensive , cardiac arrest (until blood available)

Adults

Children

20 ml/kg of body weight

Response to initial fluid challenge:

- Immediate & sustained return of vital signs
- Transient response with later deterioration
- No improvement

Immediate Responders	Transient Responders	Non Responders
• <20% blood loss • Bleeding ceases spontaneously	• Bleeding within body cavities • Surgical intervention required	• >40% of blood loss • Require immediate surgery • Maybe not enough fluids

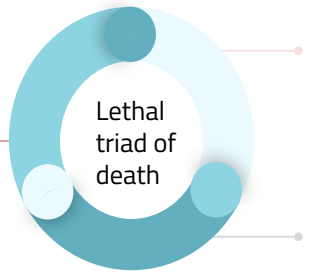
- Continued IV fluids in detrimental

Classification Of Hypovolemic Shock and Physiological Changes

	Class I	Class II	Class III	Class IV
Blood loss (liter)	Up to 0.75	0.75-1.5	1.5-2.0	> 2
% TBV	15%	30%	40%	>40%
Pulse rate	< 100	> 100	>120	>140
Blood pressure	Normal	Normal	Decreased	Decreased
Pulse pressure	Normal or inc	Decreased	Decreased	Decreased
Respiratory rate	14-20	20-30	30-40	>35
Urine output	> 30 ml/hr	20-30	5-15	Negligible
Mental status	Slightly anxious	Mildly anxious	Anxious/confused	Confused/lethargic
Fluid Replacement	Crystalloid	Crystalloid	Crystalloid and blood	Crystalloid and Blood

Estimated blood loss	
Bone	Approximate internal blood loss (L)
• Rib • Humerus • Tibia or fibula or distal femur • Femur • Pelvis	• 2-4 • 1-4 • 2-4 • 2-4 • 2-6

Shock due to primary hemorrhage → Ongoing bleeding 2° resuscitation regimen → Hypothermia



- **Voluminous crystalloid**
  - Dilutes coagulation factors
  - Causes hyperchloremic and lactate acidosis
  - Supplies inadequate O_2 to under-perfused tissue
- **Permissive hypotension**[1]
  - Maintain systolic B.P at 85-95 mm of Hg
  - Turn off the tap and do not infuse too much of fluid and blood products.

Balanced Resuscitation

1. **Fluid resuscitation in Balanced Resuscitation**
   a. Initial fluid replacement with up to 2L crystalloid Permissive hypotension to achieve SBP to 8-9mmHg (radial pulse) until definitive control of bleeding is obtained
2. **Haemostatic Resuscitation**
   a. Early blood versus HBOC transfusion decreases MODS
   b. Packed RBC , FFP and platelets in 1:1 :1 ratio
   c. Cryoprecipitate, Tranexamic acid, Recombinant factor- VIIa
   d. Storage blood of <2 weeks to minimize TRALI, MODS

1- The more pressure the more bleeding so try to control it on the hypotensive border, DON'T use permissive hypotension resuscitation during head injury

D- Disability / Dysfunction

Assessment

- Assess level of consciousness using AVPU method (Check higher Cerebral function)
  - A = alert
  - V = responding to voice
  - P = responding to pain
  - U = unresponsiveness
- Assess pupil size, equality and responsiveness.
- GCS

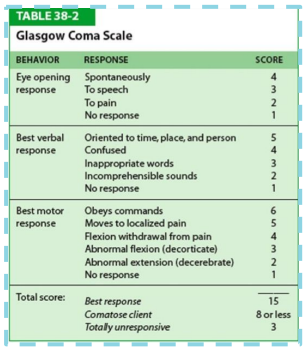


TABLE 38-2
Glasgow Coma Scale

BEHAVIOR	RESPONSE	SCORE
Eye opening response	Spontaneously	4
	To speech	3
	To pain	2
	No response	1
Best verbal response	Oriented to time, place, and person	5
	Confused	4
	Inappropriate words	3
	Incomprehensible sounds	2
	No response	1
Best motor response	Obeys commands	6
	Moves to localized pain	5
	Flexion withdrawal from pain	4
	Abnormal flexion (decorticate)	3
	Abnormal extension (decerebrate)	2
	No response	1
Total score:	*Best response*	15
	Comatose client	8 or less
	Totally unresponsive	3

E- Exposure:

Assessment

- Expose the patient fully while maintaining thermostasis (Avoid hypothermia).
- Hypothermia prevention and treatment strategies :
  - Limit casualties exposure
  - Warm IV fluids and blood products before transfusion
  - Use forced air warming devices before and after surgery
  - Use carbon polymer heating mattress

Secondary Survey (ATLS)

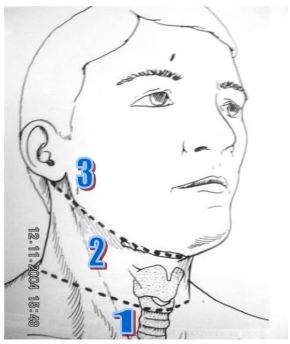
- Comprises of head to toe exam of the stable pt
- Requires
  - Detailed history
  - Thorough examination
  - KEEP MONITORING the vital signs monitoring devices:
    - Pulse oximeter
    - Rectal thermometer
- Detailed radiographic procedures
  - CT , USG , MRI
  - Any multi-trauma/severe trauma patient that is stable we must do Pan CT (Head, Neck, Chest, Abdomen, Pelvic)

HEAD & ENT

- Glasgow coma scale
- Reaction and size of pupils
- Plantar response
- Signs of rhinorrhea, otorrhea (base of the skull)
- Nose Fracture, Septal hematoma

Neck

- Subcut emphysema
- Cervical spine fracture (especially C1, C2, C7)
- Penetrating neck injuries



Thorax

- Search for potentially life threatening injuries
- Pulmonary complication
- Myocardial complication
- Aortic tear
- Diaphragmatic tear
- Esophageal tear
- Tracheobronchial tear
- Early thoracotomy if initial haemorrhage >1500 ml

Abdomen

- Fingers and tubes in every orifice
- Nasogastric and Urinary catheter for diagnosis and treatment
- Rectal examination
- Wound coverage
- Eviscerated bowels packed by warm wet mops

For rigid and distended abdomen :

- Ultrasound
- Four quadrant tap
- Diagnostic peritoneal
- Laparoscopic examination

Any deterioration → Consider rapid surgical exploration

Pelvis

Clinical assessment

↓

X-Ray

↓

Stabilize pelvis with fixator/clamps

↓

If Urethral injury is suspected
- High up prostate in PR
- Blood in meatus
- Perineal haematoma

If not →
- Trial catheter with gentle manipulation
- Fine catheter
- Lots of lubricants in OT

↓

Ascending urethrogram

↓

Suprapubic cystotomy

Spinal Injury

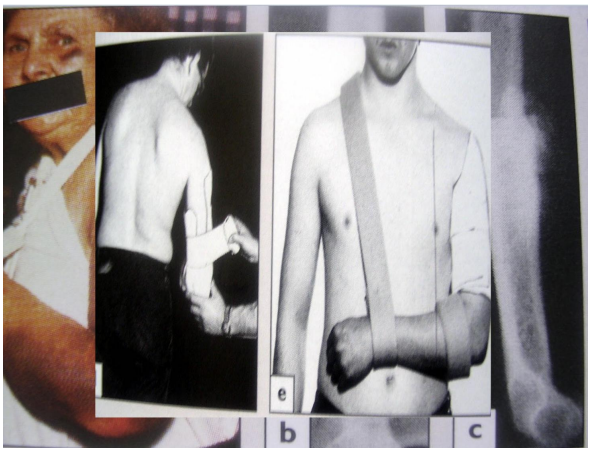
- Thorough sensory and motor examination
- Prevent further damage in unstable fractures
- Log rolling for full neurological examination-5 people required
- Use a long spine board for transportation

Extremities

- Full assessment of limbs for assessment of injury
- Always look for distal pulse & neurostatus
- Carefully look for skin & soft tissue viability
- Look for impending compartment syndrome

F- Fracture management

1. Minor
2. Moderate
   - Open fracture of digits
   - Undisplaced long bones or pelvis fracture
3. Serious
   - Closed long bone fractures
   - Multiple hand/foot fractures
4. Severe
   - Life threatening
   - Open long bone fracture
   - Pelvis fracture with displacement
   - Dislocation of major
   - Multiple amputations of digits
   - Amputation of limbs
   - Multiple closed long bone fractures



The most important thing during this stage is immobilization.

Medication

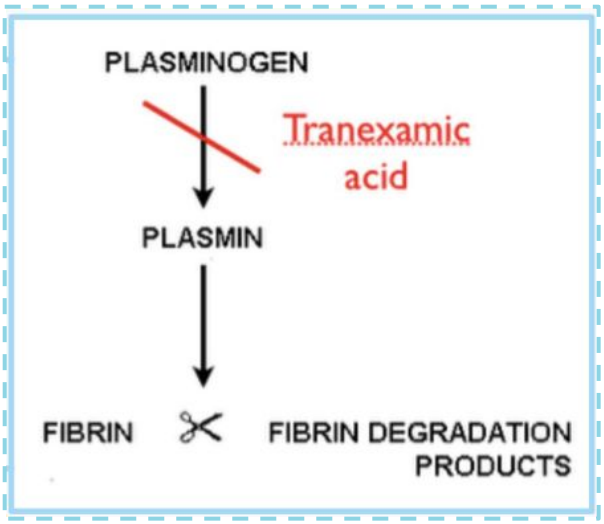
- Tetanus prophylaxis
- Anti D immunoglobulin in possible preg female
- Vasopressors drugs (selective) Shouldn't be given early (first hours) to a trauma patient because they can mask active bleeding by vasoconstriction and increasing BP.
- Antibiotics (selective) Open fractures
- Calcium gluconate (selective)
- Tranexamic acid (TXA)

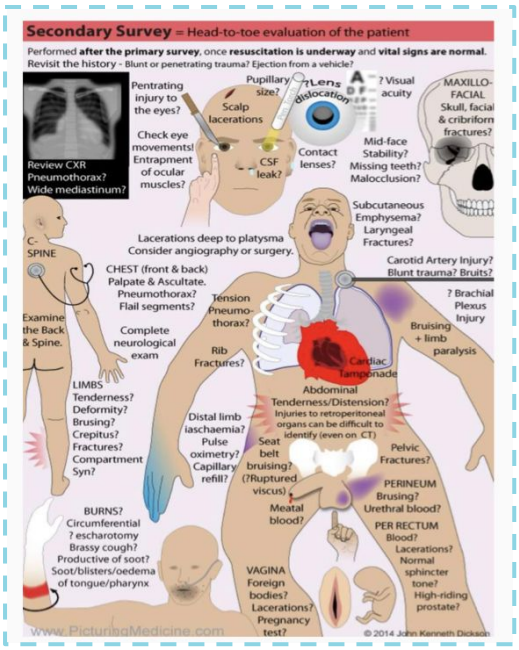
Definitive Care Plan (ATLS):

- Multi-speciality approach (Inter-disciplinary management)
- The most appropriate person in-charge is the General/trauma surgeon.

ATLS - Secondary Survey:

- Carried out after the primary survey and immediate management of potential life threats in a stable patient to identify the presence of other injuries missed in the focused primary survey
- It involves a systematic 'top-to-toe' examination:
  - Head, face, eyes, ears, nose and throat (carefully check the scalp and the oral cavity).
  - Neck, chest, abdomen , pelvis , the back, extremities and all wounds
- AMPLE history:
  - Around the time of the secondary survey clarification of the history is important to ensure that no injuries, or relevant comorbidities, are missed.
- Don't forget the following:
  - Tetanus prophylaxis
  - Anti D immunoglobulin in possible preg female
  - Antibiotics (selective)
  - Tranexamic acid (TXA)





ATLS - Tertiary Survey:

Secondary	Tertiary

• More comprehensive head-to-toe assessment
• Additional historical information
  ○ Significant others
  ○ Past medical records
• Diagnostic studies
	• Assessment of ABCDEs
	• Another head-to-toe assessment
	• Review of lab data and diagnostic studies
	• An injury found within 24 hours is not counted as a "missed" injury

Complications

1- Acute Respiratory Distress Syndrome (ARDS):

- **Signs and Symptoms:** Tachypnoea, Dyspnea, Bilateral infiltrates in CXR
- **Treatment:** Treated with mechanical "Low Tidal" ventilation with PEEP.

2- Fat Embolism:

- Around 72 hours.
- **Signs and Symptoms:** Tachycardia, Tachypnoea, Dyspnea, Chest pain, Petechial hemorrhage.
- **Treatment:** Mechanical ventilation and Fixation of fractures.

3- Disseminated Intravascular Coagulation:

- Follows severe blood loss and sepsis.
- **Signs and Symptoms:** Restlessness, Confusion, Neurological dysfunction, skin infarction, Oliguria, Excessive bleeding, Prolonged PT, PTT, TT,
  Hypofibrinogenemia.
- **Treatment:** Prevention and early correction of shock, warming fluids, giving less Crystalloids.

4- Compartment Syndrome / Crush Syndrome

- When a limb remains compressed for many hours/multiple fractures
- Increased Compartment pressure and further ischaemia (of limb)
- Cardiac arrest due to metabolic changes in blood
- Renal failure
- **Treatment:**

1. Prevention of Renal failure-ensure high urine flow during using IV Crystalloids.
2. Fasciotomy and excision of devitalized muscles
3. Amputation

5- Multisystem Organ Failure (M.S.O.F)

- Progressive and sequential dysfunction of physiological systems
- Hypermetabolic state
- It is invariably preceded by a condition known as **Systemic Inflammatory Response Syndrome (SIRS)**, Characterised by two or more of the following:

1. Temperature >38° C or < 36°C
2. Tachycardia >90 /min
3. Respiratory rate >20/min
4. WBC count >12,000/cmm or <4,000/cmm

- **Treatment:** Key word is PREVENTION

1. Prompt stabilisation of fracture **2.** Treatment of shock **3.** Prevention of hypoxia
4. Excision of all dirty and dead tissue **5.** Early diagnosis and treatment of infection.
6. Nutritional support

Conclusions

- Diagnose, prioritize management as per ATLS PROTOCOL.
- Recognize when to immediately refer a patient that requires urgent specialist management
- Remember to include in-line immobilization of cervical spine while managing the airway.
- Proper priority to orthopedic conditions that affect the patient life/limbs (open book pelvic fracture, bilateral femur fractures, mangled extremity).
- Importance of interpersonal and intrapersonal communication skills.

Quiz

Q1: A patient comes to ER and he unstable, hypovolemic and he responds to resuscitation for short period then declines again, you were consulted if he has pelvic fracture or not, how to R/O the fracture?

A. CT
B. Probe
C. X-ray
D. Pelvis instability (apply gentle pressure on the pelvis)

Q2: Patient came to ER with severe back pain after MVA otherwise normal ATLS was performed on the patient What is the next step

A. Do physical examination
B. X-ray spine
C. CT spine
D. MRI spine

Q3: A 35 y.o patient came to the ER after MVA, ATLS activated, on physical exam there was distended neck veins, diminished breath sounds and hyperresonance on percussion, what is the next step?

A. Apply Cervical collar
B. Consultation
C. Needle decompression
D. Chest tube

Q4: A 59 y.o came to E.R after penetrating injury to his chest, ATLS activated, and he is hypotensive and on auscultation there was muffled heart sounds, what is the next step?

A. Oxygen
B. Needle pericardiocentesis
C. Chest X-ray
D. Intubation

Q5: Alice Brown, a 20-year-old female, presents to the emergency department with a suspected overdose. On examination: She opens her eyes when you say her name. She is making short gasps and other sounds but she is not forming any clear words. She undergoes abnormal flexion in response to a painful stimulus. What is Alice's Glasgow Coma Scale (GCS) score?

A. 8
B. 9
C. 11
D. 5

1:D 2:A 3:C 4:B 5:A

Team Leader

Abdulrahman Alroqi

Done by

Ali Almatri

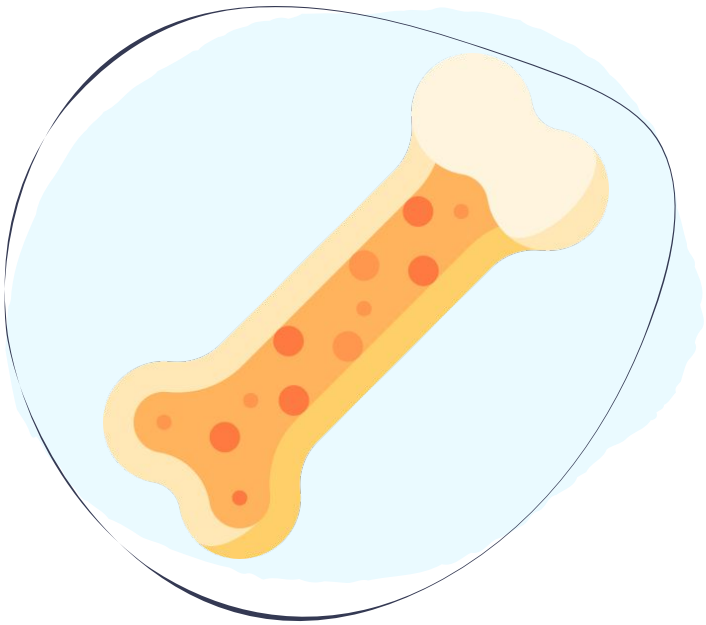
وفّقكم الله

14

Orthopedic 441 Team

MED441 KING SAUD UNIVERSITY

Editing File



Metabolic Bone Disorders

A1 Dr. Hisham Alsanawi

A2 Dr. Abdullah Addar

Color Index:

- Main Text
- Important
- 441 Notes
- Old Notes
- Extra

Objectives

- To be able to specify the symptoms and signs.
- Outline the assessment and appropriate investigation.
- Propose a limited differential diagnosis.
- Outline the principles of management of a patient with:
  - Osteoporosis.
  - Osteomalacia & Rickets.

Although there was 2 different doctors, the questions was from Dr. Hisham's slides, **BUT** this file will include both slides "mixed".

Resources

Rickets & Osteomalacia
By Osmosis

Osteoporosis
By Osmosis

Rickets
By Orthobullets

Osteomalacia
By Orthobullets

Osteoporosis & Osteopenia
By Orthobullets

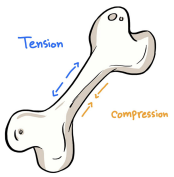
Bones

Outline

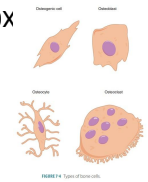
- Basic science "bone metabolism":
  - Metabolic pathways.
- Clinical features & Medications.
- Specific disorders: - Rickets/Osteomalacia. - Renal osteodystrophy. - Hypophosphatasia.
  - Osteogenesis imperfecta. - Osteoporosis.

Introduction

- Orthopedic surgeons have to deal with all types of bone (healthy or diseased), and that's why they have to know about bone metabolism, metabolic bone disorders needs a long term observation that's why it needs a specialized person.
- Bone functions:
  - Protect vital organs & structures.
  - Mechanical support for muscles & tendons[1] and act as lever arm for muscles.
  - Storage and regulation of calcium.
  - Hematopoiesis (bone marrow).
- There is a continuous activity in bone (bone resorption and bone formation as well as remodeling) not a concrete during all stages of life, that means bone is not only for protection and support but its contents play an important part in blood homeostasis, and many factors are involved in this process[2].
- Bone is a living tissue formed by bone matrix which consists of **40% organic** (connective tissue) mainly 'collagen type 1'[3] (responsible for tensile strength[4]) and **60% minerals**, mainly calcium hydroxyapatite, phosphorus, and traces of other minerals like zinc (provides compressive strength), so (collagen → tension | minerals (Ca^{2+}) → compression)[5].
- Bone Mass: Peak at 16-25 years, decreases by 0.3-0.5%/year after skeletal maturity, 2-3% decline/year in untreated post-menopausal women = osteoporosis, Improve/maintain bone mass by resistance medications, bone may become weak in certain conditions.



- All bones aren't straight, normally they're curved.
- Bone cells:
  - Osteoblasts "bone forming cells", its function:
    - Regulate osteoclasts (RANKL + OPG).
    - Produce non-mineralized bone matrix "collagen type 1" → calcium + phosphate = Hydrox
  - Osteoclasts "bone resorbing cells" "bone macrophage".
  - Osteocytes":
    - 90-95% of all cells in bone.
    - Originate from osteoblasts.
    - Function to maintain bone and cellular matrix.



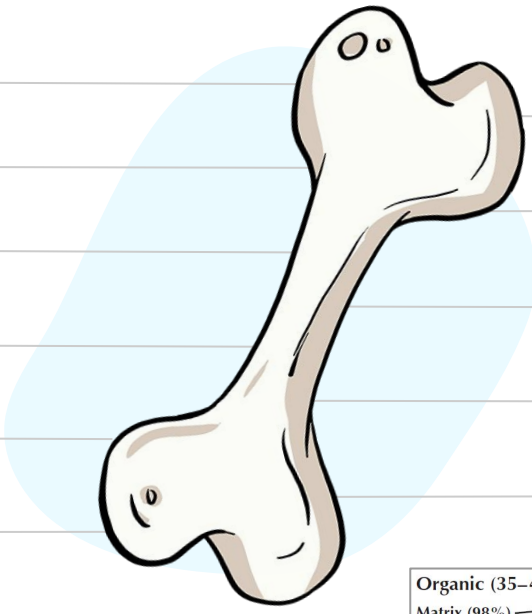
- Constant interplay between bone formation (Osteoblasts) and bone breakdown (Osteoclasts) → Bone remodeling, why remodel? Repair fatigue stresses from repeated micro-trauma.
- Signaling pathways "RANK-RANKL-OPG pathway":
  - Bone formation:
    - Stimulate osteoblasts + inhibit osteoclasts.
  - Bone resorption:
    - Activation of osteoclasts.
  - Osteoblasts produce:
    - RANKL → Bind RANK → Stimulates osteoclasts → Bone resorption.
    - Osteoprotegerin (OPG) → Competes with RANK → Limits osteoclast function → Less resorption.

1- Tendons can't work without bones.
2- While osteoblasts are forming new bones, osteoclasts are removing the dead or aged ones, this process accelerates with aging and when estrogen levels drop (e.g. menopause), the rate of formation decrease and the rate of loss increase (3% of bone mass will be lost yearly), the opposite happens in the childhood where bone formation is higher than resorption, any imbalance in this process will lead to disease.
3- There are other types but the majority of it in the bone are type 1.
4- Tensile strength is the measurement of the force required to pull something.
5- Minerals protect the bone from compressive forces (Push), while collagen protects the bone form tensile forces (Pull).

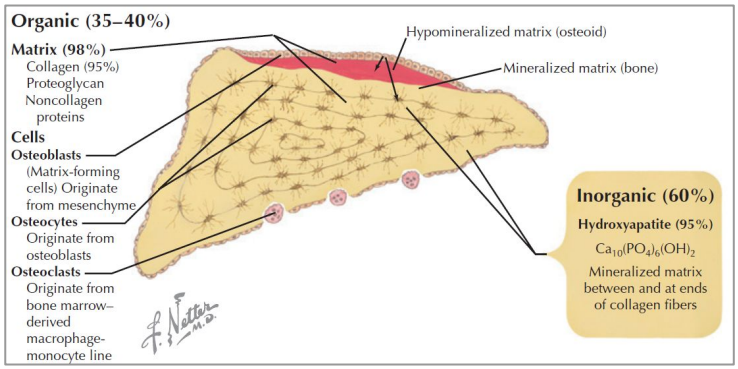
Bone Metabolism Factors

IMPORTANT

- Calcium
- Phosphorus
- Estrogen[1]
- Intestinal absorption[2]
- Renal excretion
- Sun exposure



- Parathyroid gland
- Thyroid gland
- Glucocorticoid hormones "steroids"
- Diet
- Vitamin D



Regulation of Bone Remodeling

Systemic

- PTH:
  - Bone resorption.
- Vitamin D - Calctriol – 1-25 Hydroxyvitamin D:
  - Increases bone mineralization.
- Calcitonin:
  - Limits bone resorption "inhibits osteoclasts".
- Sex hormones (Estrogen/Androgens):
  - Increase BMD "bone mineral density".
- Growth hormone:
  - Positive effect on bone formation.
- Thyroid Hormone:
  - Bone resorption.
- Corticosteroids:
  - Cause bone loss by decreasing bone formation "inhibit collagen synthesis and activate cell death for osteoblasts".

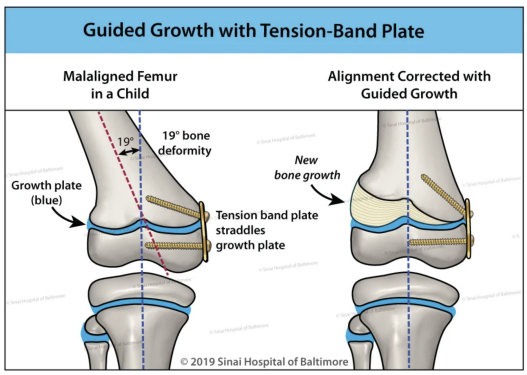
Local

- Pro-resorptive cytokines:
  - IL-1, IL-6, GM-CSF..
  - Stimulate osteoclasts → Bone resorption.
- Anti-resorptive cytokines:
  - IL-4, Interferon-B, Prostaglandin-E2.
  - Inhibit osteoclasts differentiation → Bone formation.
  - NSAIDs inhibit bone healing in rats.
- TGF-B:
  - Inhibits RANKL → Bone formation.
- BMP "bone morphogenetic proteins":
  - Promoted osteoblast differentiation → Bone formation.
  - Given intraoperatively to promote bone formation.

1- Sedentary lifestyle can lead to osteoporosis after menopause.
2- Patients with intestinal problems (such as celiac disease) have poor calcium absorption.

Mechanical Forces

- Wolff's Law:
  - Bone remodels in response to mechanical stress.
  - Osteocytes detect mechanical load → Bone formation/hypertrophy.
  - Clinically: Weight bearing + resistance training → Increase BMD.
- Heuter-Volkmann Law:
  - Mechanical forces influence longitudinal growth.
  - Compression → Inhibits longitudinal growth.
  - Distraction → Accelerates longitudinal growth.
  - Clinically: Guided-Growth.

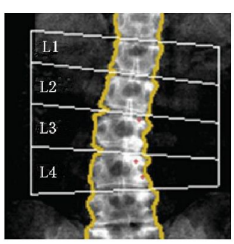


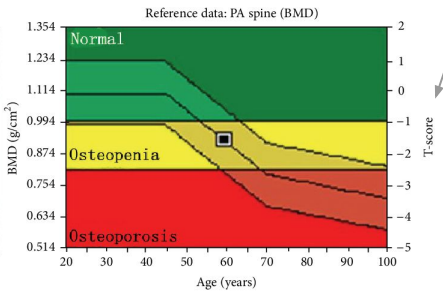
Bone Strength

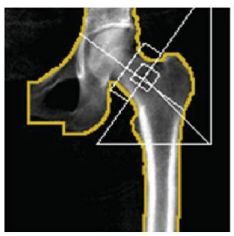
- Affected by **mechanical stress** which means exercise and weight bearing (strengthening the bone) "Wolff's law".
- One of the most important factors which affect bone strength is the exercise, it helps in overcome osteoporitic problems, so any metabolic disease management must include special **workout exercises** with special equipments.
- Gets reduced with menopause and advancing age, and bed ridden patients.
- Reduced bone density on X-rays is called **Osteopenia**.
- Osteopenia is also a term used to describe a degree of reduced bone density, which if advanced becomes **Osteoporosis** (can be diagnosed **only by DEXA**).

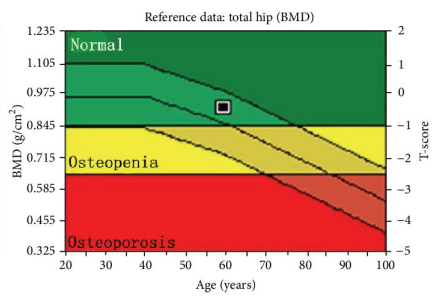
Bone Density

- Bone density is diagnosed at current time by a test done at radiology department called: **DEXA scan** (**D**ual **E**nergy **X**-ray **A**bsorptiometry)[1], which is measured radiation absorption (more absorption → more density), they do it in three areas (vertebrae, wrist "distal of radius", and neck of femur) these bones get affected first and might get fractured easily (we need to protect them).
- However, increased bone density **does not** always mean increased bone strength, as sometimes in **Brittle bone disease** "osteogenesis imperfecta" (which is a dense bone) is not a strong bone but fragile bone which may break easily.
- The measurement of bone density will be expressed as a T score (see image).



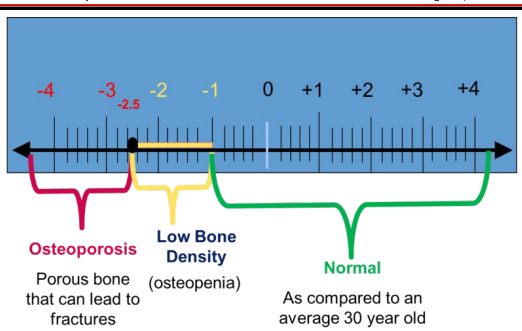




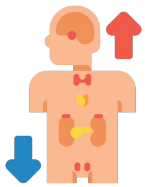


WHO criteria for diagnosing osteoporosis using bone density measurements

Classification	T-score
Normal	-1.0 or greater
Osteopenia	Between -1.0 and -2.5
Osteoporosis [2]	-2.5 or less
Severe Osteoporosis	-2.5 or less with a fragility fracture



1- It's very important to learn how to read the graph, you may be asked to do so.
2- If you suspect osteoporosis in the exam, you should choose the -2.5 or less, NEVER 2.5, FOCUS ON THE MINUS –.



Hormonal Regulations

Plasma levels | **Calcitonin** | **Parathyroid hormone**

Plasma Levels

! There are normal plasma levels in osteoporosis.

- **Calcium**: 2.2-2.6 mmol/l
- **Phosphorus**: 0.9-1.3 mmol/l
  - ➔ Both absorbed by intestine and secreted by kidney in urine.
- **Alkaline phosphatase**: 30-180 units/l (normal in osteoporosis, unless if there's complication like a fracture)
  - ➔ Is elevated in bone increased activity like during growth or in metabolic bone disease or destruction, use it as a clue, it's not specific so can't tell you the diagnosis (indicator of bone metabolism).
- **Vitamin D**: 70-150 nmol/l (promotes Ca^{2+} absorption from kidney and intestines).

Calcitonin

! Not used in treatment due to its side effects.

- Secreted by **C cells** of thyroid gland.
- Its secretion is regulated by serum calcium.
- Its action is to cause inhibition of bone resorption and increasing calcium excretion by this it causes lowering of serum calcium.
  - ➔ Inhibit reabsorption from kidney & intestine and trying to bring it back to the bone, used to be given as supplement but not anymore because of its side effects.

Parathyroid Hormone

IMPORTANT

- Production levels are related to serum calcium levels, PTH secretion is increased when serum calcium is low **($\downarrow Ca^{2+} \rightarrow \uparrow PTH$)**.
- **Action of PTH**:
  1. Increases calcium levels in the blood by increasing its release from bone (osteoclastic activity).
  2. Increases intestinal absorption by activating vitamin D.
  3. Increases reabsorption from the kidney and increases the excretion of phosphorus.
- If the parathyroid hormone is high due to a body demand, that's mean calcium is low we need to increase it, how? From the bone (readily available) so basically you will sacrifice the bone for the seek of heart, brain & vital organs. So, it works as a storage for calcium.

Hyperparathyroidism		
Primary	Secondary[1]	**Tertiary**[2,3] IMPORTANT
Adenoma of the gland.	Low Ca^{2+} (e.g. renal failure).	Prolonged or sustained stimulation = hyperactive nodule or hyperplasia. "when secondary hyperplasia leads to autonomous overactivity"

1- Partial resistance to PTH action leads to parathyroid gland hyperplasia and increased PTH secretion, often in patients with renal failure and osteomalacia (due to low or low normal serum calcium levels).
2- Irreversible clonal outgrowth of parathyroid glands, usually in long-standing inadequately treated chronic renal failure on dialysis.
3- It happened after long standing hyperparathyroidism, you treated the cause like adenoma, but the PTH hormone continues to be secreted.

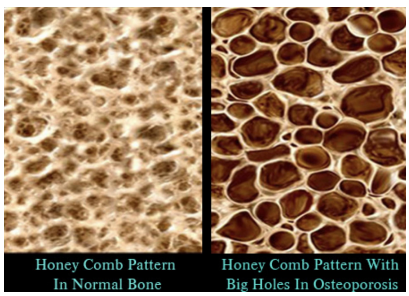
Metabolic Bone Disease

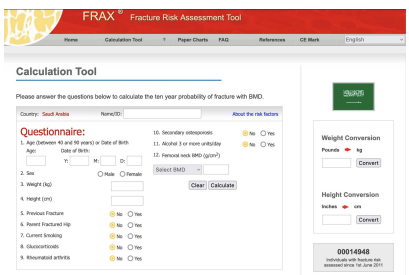
Introduction

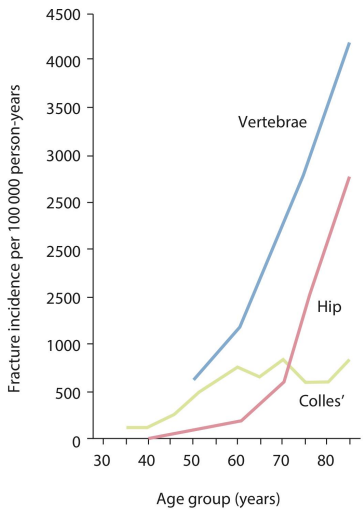
- Pathology in shape, strength, and structure of bone, resulting from altered bone homeostasis, examples:
  - Rickets. - Hypophosphatasia. - Osteogenesis Imperfecta. - Fibrous Dysplasia.
  - Osteomalacia. - Osteoporosis. - Hyper-PTH.
- General clinical features:
  - Electrolyte disturbances. - Fractures (pathological/low-energy).
  - Bone deformity. - Abnormal gait. - Short stature

Osteoporosis + Osteopenia 1

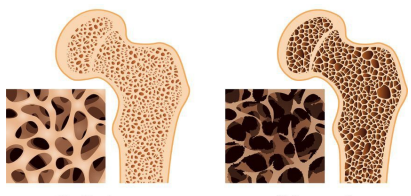
- Decreased bone mass:
  - Decreased total amount of bone/unit volume ⟶ Reduced density (but each unit volume is normal).
- Normal mineralization unlike osteomalacia (the unit volume is reduced).
- Is it important?
  - 200 million people affected yearly, mainly post-menopause and age-related.
  - 1.5 million osteoporotic fractures/year (700k vertebra, 300 hip, 200k wrist).
- **Diagnosis:**
  - Postmenopausal women/men with a T-score of ≤ −2.5 SD bone mineral density (BMD) measured through dual-energy X-ray absorptiometry (DEXA) at minimum of two sites: femoral neck, lumbar spine, or distal radius (in case scanning cannot be done at the hip or spine) is considered to have osteoporosis (this is a diagnostic and not a therapeutic threshold).
  - Presence of low-trauma (fragility) fracture (hip, spine, distal radius, proximal humerus) irrespective of BMD readings.
  - Osteopenia (T-score −1.0 to −2.5) on DEXA scan and high FRAX score based on country-specific threshold.
  - Osteopenia or low bone mass is not a separate disease entity and should be used for epidemiologic purpose only.
- FRAX (Fracture Risk Assessment Tool):
  - For fracture probability, Saudi Arabian FRAX model should be used for citizens, use other country-specific FRAX models for expatriates based on country of birth.
  - FRAX can be calculated without DEXA at baseline:
    - High-risk patients (see below for risk stratification) should be treated.
    - Intermediate risk, BMD should be measured using DEXA and FRAX recalculated with DEXA readings added.
  - Peripheral scanning using ultrasound may be used for screening but not for diagnosing osteoporosis.

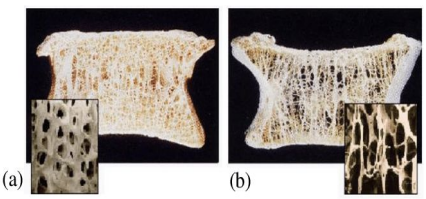




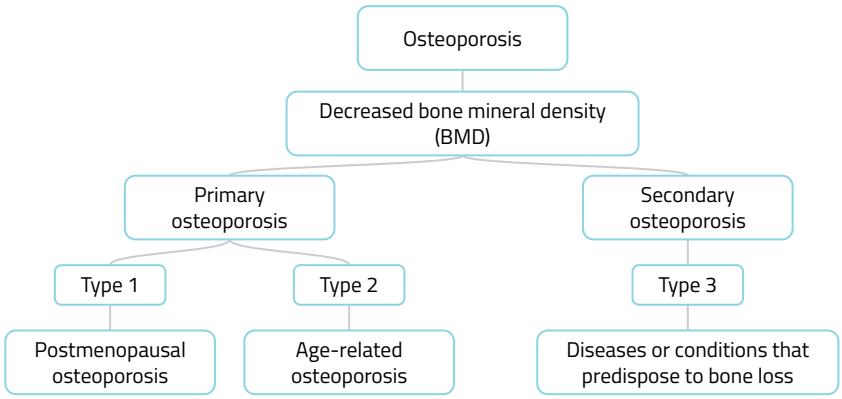


The incidence of fractures of the vertebrae, hip and wrist rises progressively after the menopause.





Osteoporosis Types



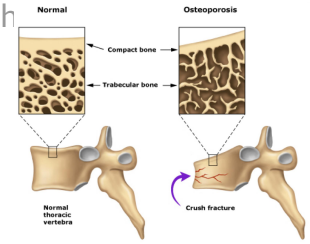
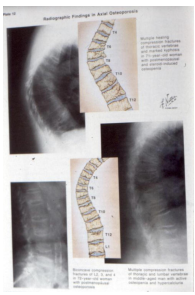
Primary Osteoporosis

Type	Postmenopausal	Senile
Description	• Due to rapid decline in estrogen level. • This results in increased osteoclastic activity. • Normal bone loss usually 0.3% per year (varies between people). • Post-menopausal bone loss 3% per year (10x folds than normal).	• Usually by 7th to 8th decades there is steady loss of at least 0.5% per year. • It is part of physiological manifestation of aging.
Risk Factors	• Race (Caucasian "white ladies"). • Hereditary. • Body build (thin people). • Early menopause. • Smoking, alcohol intake, drug abuse. • Low calcium intake. • Inactivity "chronic lack of exercise".	• Male menopause (decreased testosterone). • Dietary: Less calcium and vitamin D and protein. • Muscle weakness. • **Reduced activity** (exercise is the best way to delay osteoporosis in men).
Clinical Features	• Osteoporosis is a silent disease (asymptomatic until complications happen). • Osteoporosis is serious due to possible complications, mainly fractures. - **Most common** site (vertebra), **2nd** most common (distal radius), **most serious** (hip). • The best way to treat osteoporosis is to prevent it in the first place. • Osteoporosis does not cause pain usually "painless unless it causes fractures". • Osteoporosis causes gradual increase in dorsal kyphosis. • Osteoporosis leads to loss of height due to collapsed vertebrae "shorter back". • **Osteoporosis is not osteoarthritis**, but the two conditions may co-exist.	

Secondary Osteoporosis

It happens most of the time in young patients e.g. 45 years old, causes include:

- **Drug induced: Steroids** (especially in young people), alcohol, smoking, phenytoin, heparin, immunosuppressives.
- Hyperparathyroidism, hyperthyroidism, Cushing's syndrome, gonadal disorders, malabsorption, malnutrition, osteogenesis imperfecta.
- **Chronic diseases:** RA, renal failure, tuberculosis, ankylosing spondylitis, COPD.
- **Malignancy:** Multiple myeloma, leukemia, metastasis.

Kyphosis and Height Loss "Dowager's hump"	• With osteoporosis the anterior part of the vertebra narrows which leads to kypho… …oss of h…    
Osteoporotic Fractures "Fragility Fractures"	• They are pathological fractures. • **Most common** is **vertebral** osteoporotic compression fracture (OVC fractures), they present with **back pain**. • Vertebral micro-fractures occur unnoticed (dull ache). • **Most serious** is **hip** fractures (high mortality rate). • Also common is wrist fractures (**Colles' fracture** "distal radius"). • We must observe the hip & the vertebrae.  
Disuse Osteoporosis	• State of bone loss due to local skeletal unloading or systemic immobilization. • Occurs locally adjacent to immobilised bone or joint. • May be generalised in in bed-ridden patients or ICU patients. • Awareness of and attempts for prevention are helpful (by moving the limb).

General Management

Nutrition Counseling	**1**	Improve calcium intake
	2	Increasing vitamin D intake
Physical Activity Counseling	**3**	Weight-bearing and muscle strengthening exercise
	4	Fall prevention education
Lifestyle Counseling	**5**	Smoke cessation
	6	Limiting excessive alcohol intake
Pharmacotherapy	**7**	Pharmacotherapy
Testing	**8**	Testing bone mineral density: DXA (Dual Energy X-Ray Absorptiometry)
Communication	**9**	Physician referral letter to repot the patient's fragility fracture, risk factors, and recommendations for treatment
	10	Patient education letter to explain bone health risk factors and recommendations for treatment

- Indication: Based on DEXA scan "–2.5 or less"& FRAX score.

Management

Drug Therapy

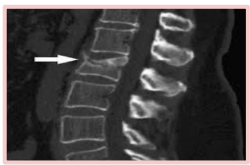
- Estrogen has a definite therapeutic effect and was used extensively as HRT but cannot be recommended now (**not used anymore**) due to serious possible side effects (such as tumors and CV risks).
- Adequate intake of calcium and vitamin D is mandatory (prior to and with treatment).
- Drugs which inhibit osteoclast activities:
  - E.g. Oral **Bisphosphonates as first line of treatment** for most patients like sodium alendronate *(FOSAMAX, BONVIVA)*.
- Drugs that enhance osteoblast activities: Bone stimulating agents like strontium *(PROTELOS)*, teriparatide *(FORTEO)*.
  - The problem in this type of medication is the risk of malignancy.
- Denosumab, zoledronic acid, teriparatide, and romosozumab are other alternative 1st line therapies for specific groups of patients and when bisphosphonates are not feasible, contraindicated or failed.
- Every treatment option has recommended duration for use, after which the patient should be assessed for the need to continue treatment or go through drug holiday, a drug holiday is not feasible with denosumab because of the increased risk of vertebral fractures after stopping treatment, so another agent should be prescribed if denosumab is discontinued.

Exercise

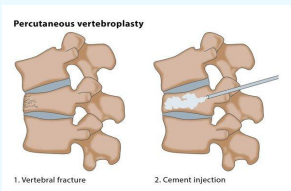
- Resistive exercises.
- Weight bearing exercises e.g. Walking.
- Exercise should be intelligent to avoid injury which may lead to fracture.

Management of Fractures

- Use of load shearing (brace) implants in fracture internal fixation instead of plating.
- Plates = load bearing "all pressure on it" | Screws "IM nail" (preferred)= load sharing "some pressure on it and some on bone".
- Pain relief.
- Prevention of further fractures and instability.
- Vertebroplasty and kyphoplasty.

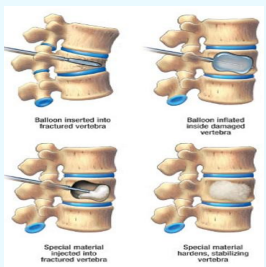


Vertebroplasty



- It's the injection of bone cement into the collapsed vertebra through pedicles.
- The injection is done under X-ray control (image intensifier) by experienced orthopedi or interventional radiologist.
- It results in immediate pain relief.
- It helps to prevent further OVF "osteoporotic vertebral fracture".
- Possible complication is leakage of cement into spinal canal (nerve injury) or venous blood (cement PE), burns around the tissue.

Kyphoplasty



- Used more than vertebroplasty.
- It's the injection of bone cement into the collapsed vertebra **AFTER inflating a balloon** "ballon then cement" in it to correct collapse and make a void (empty space) into which cement is injected.
- It is possible that some correction of kyphosis is achieved.
- It is safer because cement is injected into a safe void (low leakage risk).

Practically, refer to Endocrinology or Fracture Liaison Service.

Prevention of Osteoporosis

- Prevention of osteoporosis should start from **childhood** (especially girls).
- Healthy diet, adequate sunshine, regular **exercise**, avoidance of smoking or alcohol, caution in steroid use.
- At some time in the past there was a recommendation of HRT (Hormone Replacement Therapy) for postmenopausal women and men, **but now this is discontinued**.

Comparison

	Osteomalacia	Osteoporosis
Onset	Any age	Postmenopausal, old age
illness	ill	Not ill (unless complications)
Symptoms	Generalized chronic ache	Asymptomatic until fractures
Muscles	Weak	Normal
X-ray	Looser zones	Nil (late X-ray changes)
ALP	Increased	Normal
PO_4	Decreased	Normal

Comparison of Osteoporosis and Osteomalacia

	Osteoporosis	Osteomalacia
Definition	Unmineralized matrix; Mineralized matrix; Normal	Unmineralized matrix; Mineralized matrix; Bone mass decreased, mineralization normal	Unmineralized matrix; Mineralized matrix; Bone mass variable, mineralization decreased
Age at onset	Generally elderly, postmenopause	Any age
Etiology	Endocrine abnormality, age, idiopathic, inactivity, disuse, alcoholism, calcium deficiency	Vitamin D deficiency, abnormality of vitamin D pathway, hypophosphatemic syndromes, renal tubular acidosis, hypophosphatasia
Symptomatology	Pain referable to fracture site	Generalized bone pain
Signs	Tenderness at fracture site	Tenderness at fracture site and generalized tenderness
Radiographic features	Axial predominance	Often symmetric, pseudofractures, or completed fractures; Appendicular predominance
Laboratory findings Serum Ca^{++}	Normal	Low or normal (high in hypophosphatasia)
Serum P_i	Normal $Ca^{++} \times P_i$ >30	Low or normal $Ca^{++} \times P_i$ >30 if albumin normal (high in renal osteodystrophy)
Alkaline phosphatase	Normal	Elevated, except in hypophosphatasia
Urinary Ca^{++}	High or normal	Normal or low (high in hypophosphatasia)
Bone biopsy	Tetracycline labels normal	Tetracycline labels abnormal

Osteoporosis
Reduced amount of bone

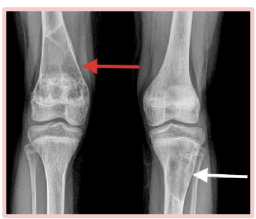
OsteoMalacia
Normal amount of bone, but reduced **M**ineralization of normal osteoid

Hyperparathyroidism 2

Hyperparathyroidism	
• Excessive PTH secretion: Primary, secondary or tertiary. • Leads to increased bone resorption, subperiosteal erosions, osteitis manifested by fibrous replacement of bone. • Significant feature is **hypercalcemia** "renal stones, constipation". • In severe cases: Osteitis fibrosa cystica and formation of brown tumors.	
X-ray Changes	• Generalized decrease in bone density. • Sub-periosteal bone resorption (scalloping of metacarpals and phalanges "hands mainly"). • Brown tumors (empty bone areas with bleeding caused by bone reuptake, not a true tumor). • Chondrocalcinosis "calcification of the cartilage around joints" (wrist, knee, shoulder).
Management	By management of the cause: • Primary hyperparathyroidism due to neoplasm (adenoma or carcinoma) by excision. • Secondary hyperparathyroidism by correcting the cause of hypocalcemia. • Tertiary hyperparathyroidism by excision of hyperactive (autonomous) nodule. • Extreme care should be applied after surgery to avoid **hypocalcemia** due to hungry bones syndrome "severe resistant hypocalcemia". - One of the very important things you must pay attention to, it needs a lot of monitoring, the moment you correct hyperparathyroidism (1st 48 hours is critical), the bone might absorb a lot of calcium leading to severe hypocalcemia, that might lead to cardiac problems (cardiac arrest) or convulsions, if that happens it is very difficult to correct the hypocalcaemia.

Early erosions

Scalloped distal phalanges



Brown tumors

Chondrocalcinosis

TABLE 3

Lab Comparison

Hyperparathyroidism	Calcium	PTH	Vitamin D	Phosphate
Primary	↑	↑→	↑	↓
Secondary	↓→	↑	↓	↑ or ↓
Tertiary	↑	↑↑	↓	↑

Key: ↑Elevated, ↓decreased, →normal.

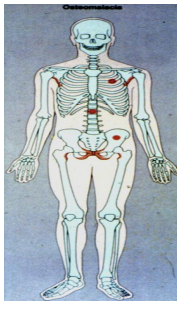
Rickets & Osteomalacia 3

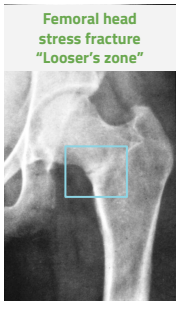
	Rickets	Osteomalacia
	Different expressions of the same disease, which is: Inadequate mineralization.	
Pathology	• Failure of mineralization of cartilage and osteoid tissue. • Decreases longitudinal bone growth and weakness mechanical properties of tubular bone. • Qualitative defect (osteoporosis is a quantitative defect).	
Onset	• Prior to skeletal maturity (open growth plates) in **children**.	• After skeletal maturity (closure of growth plates) in **adults**.
Affected Site	• Areas of endochondral growth (e.g. proximal humerus and proximal femur).	• **All** skeleton is incompletely calcified (slowly progressive).
Biochemistry	• Hypocalcemia (always check Ca^{2+}, PO_4, vitamin D and ALP levels). • Hypocalciuria. • High alkaline phosphatase. Low Ca^{2+}, low PO_4, high ALP	
Causes	• Calcium deficiency (diet, malabsorption). • Hypophosphatemia (to deposit calcium you need phosphate). • **Defect in vitamin D** metabolism: Nutritional "vegan", underexposure to sunlight, intestinal malabsorption "celiac disease", liver & kidney diseases.	

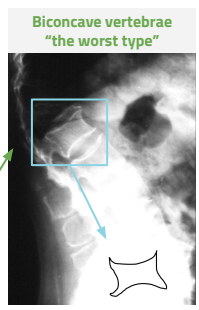
Osteomalacia

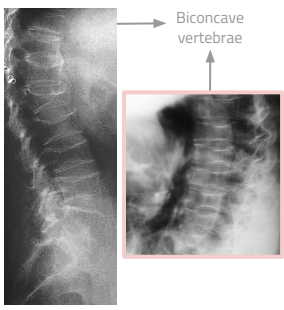
Description	• Metabolic bone disorder in adults.
Clinical Features (The difference here is that the growth is stopped in Adults unlike children so no growth-related symptoms here)	• Bone pain **mainly backache**, muscle weakness, fatigue. • Reduced bone density. • Vertebral changes: **Biconcave vertebrae**, vertebral collapse (shortening), kyphosis, scoliosis. • Stress fractures "late": **Looser's zones** in scapula , ribs, pelvis, proximal femur.
Management	**Medical:**
	• Both "vitamin D + calcium" + lifestyle modification + exercise + sun exposure.
	Surgical:
	• Fracture management. • Correct deformity if needed "the **residual** deformities only (**after treatment**)".

Looser's zones in the pubic rami and left femoral neck









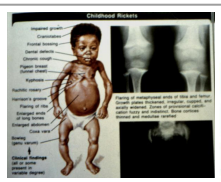
Rickets

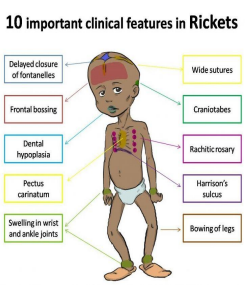
Diagnosis

- Based on: Clinical features, radiographs (X-ray), labs, +/- genetics.

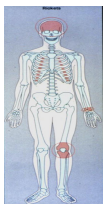
Clinical Features

(The growth plate got affected, many symptoms due to that)



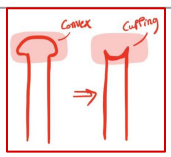


- Depends on age of onset.
- Infants:
  - Generalized muscle weakness.
  - Lethargy, irritability.
  - Hypotonia.
  - Delayed closure of skull fontanelles.
  - **Craniotabes**: Softening of skull bones.
- Child is restless, babies cry without obvious reason.
- **Failure to thrive**.
- Limb deformities, mostly "lower limbs":
  - Bowing: Genu varum "early".
  - Genu valgum "late".
- Bone pain, coxa vara, difficulty walking.
- Joint thickening "hypertrophy" of ankles, and especially around **wrists** and **knees**.
  - Wrist is the most important X-ray to confirm the diagnosis.
- Ligamentous laxity.
- Enlargement of costal cartilage: **Rachitic rosary**.
- Pigeon chest deformity "pectus carinatum", harrison's sulcus.
- In severe cases with **very low calcium**: Tetany or convulsions.



UL: Away from elbow (distal radius & proximal humerus "deltoid hide it").
LL: Around the knee.

X-ray Changes



Growth plate widening and thickening

Metaphyseal cupping

Long bone deformities

- Growth plate (physeal) widening & thickening.
- **Metaphyseal cupping**.
- Long bone deformities.
- Fraying of metaphysis (Indistinct borders).
- Decreased bone density.
- Looser's zones, pseudofracture on the compression side of bone.
- Rachitic rosary: Prominence of rib heads at the osteochondral junction.
- Genu varum/valgum.
- Codfish vertebrae.
- Cat back: Dorsal kyphosis.

Classical nutritional rickets, showing the well-marked physes, the flared metaphyses and the bowing deformities of the lower limb bones

Management

Medical:

- Adequate vitamin D replacement.
- Sun exposure.
- Calcitriol (1-25 vitamin D).
- Phosphate replacement.
- Calcium.
- Burosomab.
- Check if there's no systemic illness "treat the cause".

Very Imp MCQ:
7 y/o with genu valgum of angle 20° has difficulty standing and tripping "X-Ray pic" what will you do to him?
- 1st: Treat him with medication until you normalize every thing, and then see if there are deformity left then correct it "vit D then corrective osteotomy if needed".

Surgical:

- Case by case to **correct residual deformities** (post rachitic) by corrective osteotomy.
  - Don't perform surgery immediately because the bone is still growing, after 18-24 months of treatment.
- Treat fractures.

Child with abdominal symptoms + rickets features → Refer to pediatrician

Rickets Types

Vitamin D Resistant Rickets	• **Most common heritable form** (XLD, AR, AD). • **Inability of renal tubules to absorb phosphate**. • Also known as: **Familial hypophosphatemic rickets**.
Nutritional	• Dietary. • Celiac disease or hepatic disease • **Rickets of prematurity**: - Premature infants in NICU. - TPN, hepatobiliary disease, diuretic therapy, etc.. • **Drug-induced rickets** "Anti-epileptics in children": - **Child + Seizure disorder on medication + frequent fractures**.
Vitamin D Dependent	• Type 1: Inability to hydroxylate. • Type 2: Receptor insensitivity.
Renal Osteodystrophy	• Definition: - Biochemical + skeletal manifestation of CKD or ESRD. • Pathophysiology: - Damaged kidney → Inability to excrete PO_4 → Hyperphosphatemia. • Orthopedic issues: - SCFE "slipped capital femoral epiphysis, discussed in pediatric hip lecture". - AVN "avascular necrosis". - Bowing of long bones.
Hypophosphatasia	• Inherited disorder resulting in rickets-like features. • Main pathology is decreased or lack of alkaline phosphatase (ALP).

Table 42-1 Biochemical Abnormalities in Rickets

	Biochemical Abnormality					
Type of Rickets	**Calcium**	**Phosphate**	**Alkaline Phosphatase**	**PTH**	**25-(OH) vitamin D**	**1,25-$(OH)_2$ vitamin D**
Nutritional	NI	NI↓	↑	↑	↓↓	↓
Vitamin D–resistant (XLH, RTA, Fanconi, oncogenic)	NI	↓	↑	NI	NI	NI
Vitamin D–dependent type I (inability to hydroxylate)	↓	↓	↑	↑	↑↑	↓↓
Vitamin D–dependent type II (receptor insensitivity)	↓	↓	↑	↑	NI↑↑	↑↑↑↑
Renal osteodystrophy	NI↓	↑	↑	↑↑	NI	↓↓

Osteogenesis Imperfecta (Brittle Bone Disease)

Definition

- Genetic connective tissue disorder affecting the formation of type 1 collagen.
- COL1A1, COL1A2.

Pathology

- Type 1 collagen is reduced in quantity.
- Some have a quality issue with mutant collagen.
- Weaker bones.

Types

Sillence classification:

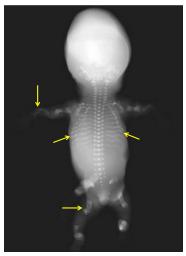
- Type 1: Mild form.
- Type 2: Lethal form.
- Type 3: Severe deforming form.
- Type 4, 5, others: Variable features.

Clinical Features

Orthopedic:

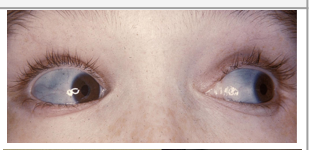
- **Multiple fractures + increased bone fragility**:
  - **Olecranon apophyseal avulsion fractures** in children are pathognomonic for OI.
  - Might be 1st sign of disease, especially milder forms.
- Bowing.
- Short stature.
- Kyphoscoliosis.
- Basilar invagination.
- Ligamentous laxity.
- Radial head dislocation.
- Coxa vara.

Full body X-ray of a neonate demonstrates multiple rib fractures, right femur fracture and right forearm fractures (yellow arrows).

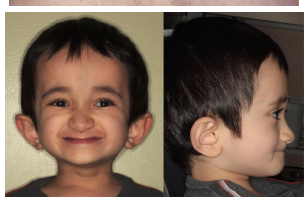


Olecranon fracture

Non-Orthopedic:



- Blue sclera.
- Dentinogenesis imperfecta.
- Hearing loss.
- Bleeding tendency.
- Triangular facies.
- Low set ears.
- Hyper-metabolism (risk of malignant hyperthermia).
- Cardiovascular: Mitral prolapse, aortic regurgitation.

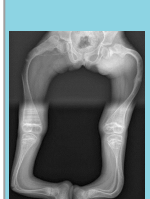


X-ray Changes

- Generalized osteopenia/demineralization of bone
- Frequent fractures:
  - Multiple fractures at different stages of healing.
  - More than 3 rib fractures in a child → Rule out osteogenesis imperfecta.
  - Delayed skull ossification and **Wormian bones**:
    - Wormian bone is extra skull bones occurring within a suture (skull joint).

Management

"Medical + Surgical combined"



Medical:

- Bisphosphonates.

Surgical:

- Indicated in type 3, type 4 and deforming types of osteogenesis imperfecta.
- Long bone telescoping rodding as soon as the child starts pulling to stand.

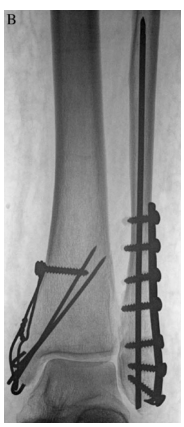
Medications and Bone Metabolism

Table 1. Impact of Medications on Bone Homeostasis

Medication	Mechanism of Action	Effects on Bone
Diphosphonates[19]	Inhibit osteoclast differentiation and function and promote apoptosis	Inhibit resorption and improve BMD
Denosumab[19]	Anti-RANKL antibody	Inhibit resorption and improve BMD
Parathyroid hormone (PTH; teriparatide)[16,29,30]	Promote osteoblast RANKL secretion	Stimulate resorption and may promote fracture healing
NSAIDs[31]	Inhibition of COX-1 and COX-2 enzymes and decrease PGE_2	Impaired bone healing, clinical effect likely duration dependent
Methotrexate (MTX)[19]	Inhibits osteoblast proliferation at high doses	Neutral at low doses used for rheumatologic disease; decreases bone formation at higher doses
Antiepileptics[19]	Effect on the CYP-450 pathway adversely affects the vitamin D-PTH-calcitonin axis	May cause rickets and osteomalacia
Statins[19]	Increase osteoblast BMP-2 release and decrease osteoclast differentiation	Improve BMD
Corticosteroids[32]	Initiate proapoptotic pathways in osteoblasts	Diminish osteogenesis

Surgical Treatment in Metabolic Bone Disease

Principles + Techniques	
Protect the Entire Bone Segment if Possible	• Use intramedullary implants, if possible, nails better than plates.
Promote Weight Bearing ASAP	• Load-sharing implants (nails) better than load bearing (plates).
Augment Fixation (Don't rely on poor biology)	• Use cement. • Use bone substitutes. • Purchase more bone.





Important Notes

Dr. Hisham Alsanawi's Notes:

Bone metabolism:

- **Calcium & Phosphorus** are important factors controlling metabolism.
- **Bone strength** is affected by **mechanical stress** = **Exercise** is important.
- **Osteoporosis** only diagnosed with **DEXA** "-2.5 or less".
- Production of **PTH** is related to serum **calcium** level.
- **Tertiary** hyperparathyroidism: **Prolonged** sustained stimulation.

Rickets & Osteomalacia:

- **X-ray** findings of Rickets: - **Growth plate widening** and thickening - Metaphyseal **cupping** - **Long bones deformity**.
- **First start with vitamin D & calcium replacement, don't do surgery for the deformities at first, after treatment only you can correct the residual deformities by corrective osteotomy.**

Osteoporosis:

- Primary **senile** osteoporosis important risk factor is **reduced activity**.
- The best & first line treatment is bisphosphonates

Important to differentiate between osteomalacia and osteoporosis by the table in the team last slide, but generally **ALP & PO4 are normal in osteoporosis**.

Dr. Abdullah Addar's Notes:

- Patient present with S&S of rickets and have **epilepsy** -> **Drug-induced rickets**.
- **Avulsion of olecranon apophyseal fracture** is pathognomonic for **Osteogenesis imperfecta** in children.
- **Osteogenesis imperfecta** -> **Surgery** (to protect the entire segment) & **medical** (bisphosphonates).
- Patient presented with **hypocalcemia**, **abdominal pain** and S&S of **rickets**, what to do next? - Investigate for **celiac disease**.

Olceranon apophyseal fracture

Long bone deformities

Growth plate widening

Metaphyseal cupping

EXTRA

Metabolic Bone Disease

- see 2010 Clinical Practice Guidelines for the Diagnosis and Management of Osteoporosis for details

Osteoporosis

Definition

- a condition characterized by decreased bone mass and microarchitectural deterioration with a consequent increase in bone fragility and susceptibility to fracture
- BMD is measured at hip and lumbar spine, BMD T-score ≤-2.5 is indicative of osteoporosis
- osteopenia (low bone mass): BMD with T-score between –1.0 and –2.5

ETIOLOGY AND PATHOPHYSIOLOGY

Secondary Osteoporosis

- gastrointestinal diseases
  - gastrectomy
  - malabsorption (e.g. celiac disease, IBD, bariatric surgery)
  - chronic liver disease
  - eating disorder
  - poor nutrition
- bone marrow disorders
  - multiple myeloma
  - lymphoma
  - leukemia
- endocrinopathies
  - Cushing's syndrome
  - hyperparathyroidism
  - hyperthyroidism
  - premature menopause
  - DM
  - hypogonadism
- malignancy
  - secondary to chemotherapy
  - myeloma
- rheumatologic disorders
  - rheumatoid arthritis
  - SLE
  - ankylosing spondylitis
- drugs and chemotherapy
  - corticosteroid therapy
  - anti-epileptic drugs
  - chronic heparin therapy
  - androgen deprivation therapy
  - aromatase inhibitors
- renal disease
- immobilization
- COPD (due to disease, tobacco, and glucocorticoid use)

Clinical Features

- commonly asymptomatic
- height loss due to collapsed vertebrae
- fractures: most commonly in hip, vertebrae, humerus, and wrist (see Figure 22, E49)
  - fragility fractures: fracture with fall from standing height or less (does not include fractures of fingers and toes)
  - Dowager's hump: collapse fracture of vertebral bodies in mid-dorsal region
  - x-ray: vertebral compression fractures (described as wedge fractures, require a minimum of 20% height loss), "codfishing" sign (weakening of subchondral plates and expansion of intervertebral discs)
- pain, especially backache, associated with fractures

Approach to Osteoporosis

1. assess risk factors for osteoporosis on Hx and physical
2. decide if patient requires BMD testing with dual-energy x-ray absorptiometry (DEXA): men and women ≥65 yr (or younger if presence of risk factors, see Table 32, E48)
3. initial investigations
   - all patients with osteoporosis: calcium corrected for albumin, CBC, creatinine, ALP, TSH
   - also consider serum and urine protein electrophoresis if vertebral fractures, celiac workup, and 24 h urinary Ca^{2+} excretion to rule out additional secondary causes
   - 25-OH-vitamin D level should only be measured after 3-4 mo of adequate supplementation and should not be repeated if an optimal level ≥75 nmol/L is achieved
   - lateral thoracic and lumbar x-ray if clinical evidence of vertebral fracture (or in individuals at moderate risk of fracture to help decide if they require medical therapy)
4. assess 10-yr fracture risk by combining BMD result and risk factors
   1). WHO Fracture Risk Assessment Tool (FRAX)
   2. Canadian Association of Radiologists and Osteoporosis Canada Risk Assessment Tool (CAROC)
      - approach to management guided by 10-yr risk stratification into low, medium, and high-risk
5. for all patients being assessed for osteoporosis, encourage appropriate lifestyle changes (see Table 33, E48)

Corticosteroid Therapy is a Common Cause of Secondary Osteoporosis
Individuals receiving ≥7.5 mg of prednisone daily for over 3 mo should be assessed for bone-sparing therapy
Mechanism: increased resorption + decreased formation + increased urinary calcium loss + decreased intestinal calcium absorption + decreased sex steroid production

Calcium plus Vitamin D Supplementation and Risk of Fractures
Osteoporosis Int 2015;27:367-376
Purpose: To review trials of vitamin D and calcium therapy for reducing fracture risk in osteoporosis.
Study: Systematic review searching 2011-2015, inclusive, identified 8 RCTs totaling 30970 participants. RCTs reviewed included healthy adults and ambulatory older adults with medical conditions (excluding cancer). Vitamin D and calcium combination therapy was compared to placebo.
Results: Analysis of RCT data revealed that calcium plus vitamin D supplementation produced a statistically significant reduction in risk of total fractures (0.85; CI:0.73-0.98) and in hip fractures (0.70; CI:0.56-0.87). Subgroup analysis was significant for community dwelling or institutionalized patients.
Conclusions: Systematic analysis suggests that vitamin D and calcium therapy significantly decreases fracture risk. This study did not specifically look at individuals with osteoporosis. However, it still supports that vitamin D and calcium should continue to be used as preventive treatment for individuals at increased risk of fractures.

Vitamin D and Calcium for the Prevention of Fracture: A Systematic Review and Meta-analysis
JAMA Netw Open 2019;2:e1917789
Purpose: To investigate if fracture risk is associated with supplementation with vitamin D alone or vitamin D in combination with calcium.
Study Selection: Observational studies with ≥200 fracture cases and RCTs with ≥500 participants that reported ≥10 incident fractures.
Results: Vitamin D supplementation alone was not associated with a reduced risk of any fracture or hip fracture (RR, 1.14; 95% CI, 0.98-1.32). However, combined supplementation with vitamin D (400-800 IU daily) and calcium (1000-1200 mg daily) was associated with a 6% reduction in fracture risk (RR, 0.94; 95% CI, 0.89-0.99) and a 16% reduction of hip fracture risk (RR, 0.84; 95% CI, 0.72-0.97).
Conclusion: Vitamin D alone was not associated with reduced fracture risk but daily supplementation with a combination of vitamin D and calcium was.

Clinical Signs of Fractures or Osteoporosis
- Height loss >3 cm (Sn 92%)
- Weight <51 kg (Sp 97%)
- Kyphosis (Sp 92%)
- Tooth count <20 (Sp 92%)
- Grip strength
- Armspan-height difference >5 cm (Sp 76%)
- Wall-occiput distance >4 cm (Sp 92%)
- Rib-pelvis distance ≤2 finger breadth (Sn 88%)

Online Clinical Tools
CAROC
www.osteoporosis.ca/multimedia/pdf/CAROC.pdf
FRAX
www.shef.ac.uk/FRAX/tool.aspx

EXTRA

Table 31. Indications for BMD Testing

Older Adults (age ≥50 yr)	Younger Adults (age <50 yr)
All women and men age ≥65 yr Menopausal women, and men 50-64 yr with clinical risk factors for fracture: Fragility fracture after age 40 Prolonged glucocorticoid use Other high-risk medication use (aromatase inhibitors, androgen deprivation therapy) Parental hip fracture Vertebral fracture or osteopenia identified on x-ray Current smoking High alcohol intake Low body weight (<60 kg) or major weight loss (>10% of weight at age 25 yr) Rheumatoid arthritis Other disorders strongly associated with osteoporosis: primary hyperparathyroidism, T1DM, osteogenesis imperfecta, uncontrolled hyperthyroidism, hypogonadism or premature menopause (<45 yr), Cushing's disease, chronic malnutrition or malabsorption, chronic liver disease, COPD, and chronic inflammatory conditions (e.g. inflammatory bowel disease)	Fragility fracture: Prolonged use of glucocorticoids Use of other high-risk medications (aromatase inhibitors, androgen deprivation therapy, anticonvulsants) Hypogonadism or premature menopause Malabsorption syndrome Primary hyperparathyroidism Other disorders strongly associated with rapid bone loss and/or fracture

Prevention - Hip

Alendronate	0.61 RR (0.42-0.90)
Risedronate	0.73 RR (0.58-0.92)
Denosumab	0.56 RR (0.35-0.90)
Teriparatide	0.64 RR (0.25-1.68)
Romosozumab	0.44 RR (0.24-0.79)

Prevention - Nonvertebral

Alendronate	0.84 RR (0.74-0.94)
Risedronate	0.78 RR (0.68-0.89)
Denosumab	0.80 RR (0.67-0.96)
Teriparatide	0.62 RR (0.47-0.80)
Romosozumab	0.67 RR (0.53-0.86)

Prevention - Vertebral

Alendronate	0.57 RR (0.45-0.71)
Risedronate	0.61 RR (0.48-0.78)
Denosumab	0.32 RR (0.22-0.45)
Teriparatide	0.27 RR (0.19-0.38)
Romosozumab	0.33 RR (0.22-0.49)

Table 32. Osteoporosis Risk Stratification

Low-Risk 10 yr fracture risk <10%	Unlikely to benefit from pharmacotherapy; encourage lifestyle changes Reassess risk in 5 yr
Medium-Risk 10 yr fracture risk 10-20%	Discuss patient preference for management and consider additional risk factors Factors that warrant consideration for pharmacotherapy: Additional vertebral fracture(s) identified on vertebral fracture assessment (VFA) or lateral spine x-ray Previous wrist fracture in individuals ≥65 yr or with T-score ≤–2.5 Lumbar spine T-score much lower than femoral neck T-score Rapid bone loss Men receiving androgen-deprivation therapy for prostate cancer Women receiving aromatase-inhibitor therapy for breast cancer Long-term or repeated systemic glucocorticoid use (oral or parenteral) that does not meet the conventional criteria for recent prolonged systemic glucocorticoid use Recurrent falls (defined as falling 2 or more times in the past 12 mo) Other disorders strongly associated with osteoporosis Repeat BMD and reassess risk every 1-3 yr initially
High-Risk 10 yr fracture risk >20%; OR Prior fragility fracture of hip or spine; OR More than one fragility fracture	Start pharmacotherapy (need to consider patient preference)

Factors Necessary for Mineralization

- Quantitatively and qualitatively normal osteoid formation
- Normal concentration of calcium and phosphate in ECF
- Adequate bioactivity of ALP
- Normal pH at site of calcification
- Absence of inhibitors of calcification

Treatment of Osteoporosis

Table 33. Treatment of Osteoporosis in Women and Men

Treatment for Both Men and Women	
Lifestyle	Diet: elemental calcium 1000-1200 mg/d; vitamin D 1000 IU/d Exercise: 3x30 min weight-bearing exercises, balance exercise, and aerobic exercise/wk Cessation of smoking, reduce caffeine intake Stop/avoid osteoporosis-inducing medications
Drug Therapy	
Bisphosphonate: inhibitors of osteoclasts	1st line in prevention of hip, nonvertebral, and vertebral fractures (Grade A): alendronate (PO), risedronate (PO), zoledronic acid (IV)
RANKL Inhibitors	Denosumab: 1st line in prevention of hip, nonvertebral, vertebral fractures (Grade A) *Denosumab should not be abruptly stopped/administration delayed. Increased risk of multiple vertebral fractures due to increased bone turnover on discontinuation. Used as an alternative initial treatment in postmenopausal women with osteoporosis who are at high risk for osteoporitic fractures.
Parathyroid Hormone Analogue	Teriparatide: 18-24 mo duration, followed by long-term anti-resorptive therapy with bisphosphonate or RANKL inhibitor
Sclerostin Inhibitors	Romosozumab: 12 mo duration
Treatment Specific to Post-Menopausal Women	
SERM (selective estrogen-receptor modulator): agonistic effect on bone but antagonistic effect on uterus and breast	Raloxifene: 1st line in prevention of vertebral fractures (Grade A) Advantages: prevents osteoporotic fractures (Grade A to B evidence), improves lipid profile, decreased breast cancer risk Disadvantages: increased risk of DVT/PE, stroke mortality, hot flashes, leg cramps
HRT: combined estrogen + progesterone (see Gynaecology, GY37)	Indicated for vasomotor symptoms of menopause For most women, risks > benefits Combined estrogen/progestin prevents hip, vertebral, total fractures Increased risks of breast cancer, cardiovascular events, and DVT/PE

Effect of High-Dose Vitamin D Supplementation on Volumetric Bone Density and Bone Strength: A Randomized Clinical Trial

JAMA 2019;322:736-45

Purpose: To investigate the effects of vitamin D supplementation on volumetric BMD and strength.

Methods: 311 healthy adults (ages 55-70) without osteoporosis, with baseline concentrations of 25-hydroxyvitamin D of 30-125 nmol/L, were randomized to receive daily doses of 400 IU, 4000 IU, or 10000 IU vitamin D3 for 3 years. For participants with calcium dietary intake <1200 mg/d, supplementation was provided. Primary Outcome: Total volumetric BMD at radius and tibia.

Results: Compared with the 400 IU group, radial volumetric BMD was significantly lower for the 4000 IU group (-3.9 mg HA/cm3; 95% confidence interval (CI), -6.5 to -1.3) and 10000 IU group (-7.5 mg HA/cm3; 95% CI, -10.1 to -5.0) with mean % change of -1.2% (400 IU), -2.4% (4000 IU), and -3.5% (10000 IU). Compared with the 400 IU group, tibial volumetric BMD differences were -1.8 mg HA/cm3 (95% CI, -3.7 to 0.1) (4000 IU) and -4.1 mg HA/cm3 (95% CI, -6.0 to -2.2) (10000 IU), with mean % change values of -0.4% (400 IU), -1.0% (4000 IU), and -1.7% (10000 IU).

Conclusion: In healthy adults, supplementation with daily 4000 IU or 10000 IU vitamin D for 3 years was associated with lower radial BMD compared with 400 IU. 10000 IU was associated with lower tibial BMD. There were no apparent benefits of high-dose vitamin D supplementation for bone health.

EXTRA

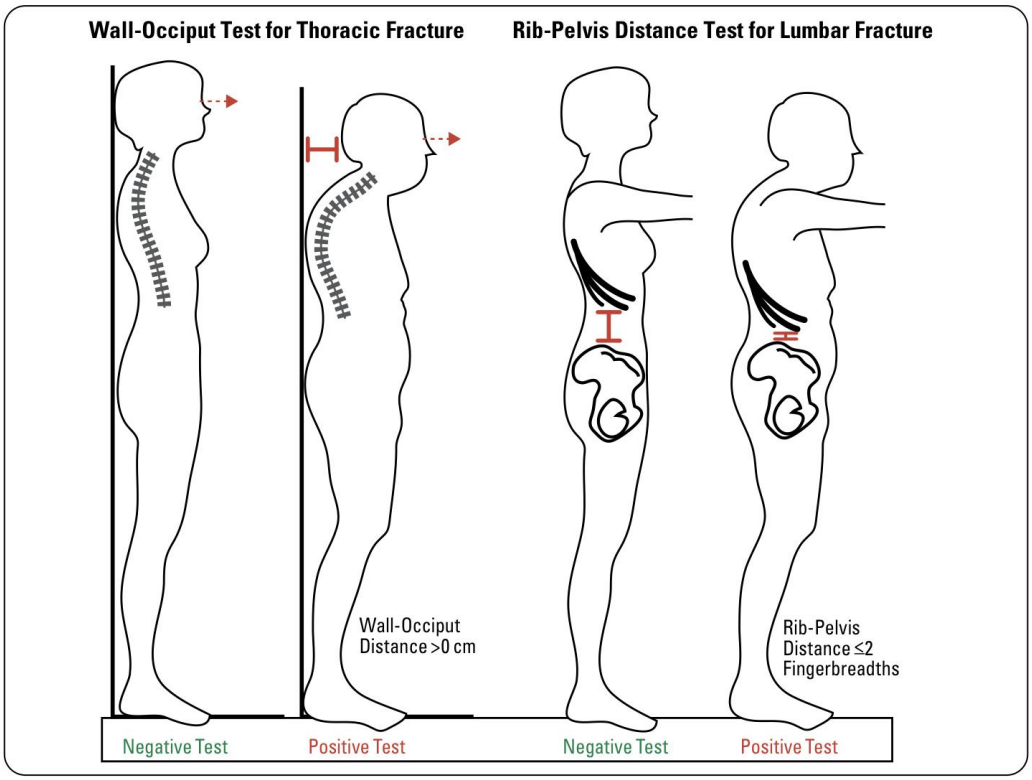


Figure 22. Physical examination test for vertebral fractures

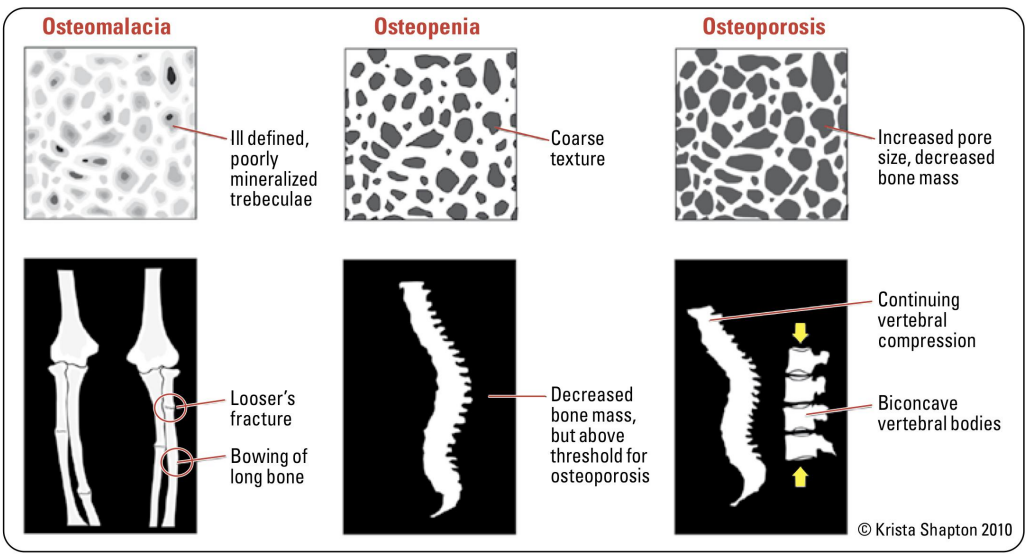


Figure 42. Osteomalacia, osteopenia, and osteoporosis

Disorders Strongly Associated with Osteoporosis Include:
Primary hyperparathyroidism, T1DM, osteogenesis imperfecta, uncontrolled hyperthyroidism, hypogonadism or premature menopause (<45 yr), Cushing's disease, chronic malnutrition or malabsorption, chronic liver disease, COPD, and chronic inflammatory conditions (e.g. IBD)

10 Yr Fracture Risk Assessment
FRAX (WHO Fracture Risk Assessment Tool) and CAROC (Canadian Association of Radiologists and Osteoporosis Canada) have been validated in the Canadian Population
FRAX and CAROC are available online from: https://www.osteoporosis.ca/health-care-professionals/clinical-tools-and-resources/

How Much Calcium Do We Need?

Age	Amount /day
4-8	1000 mg
9-18	1300 mg
19-50	1000 mg
>50	1200 mg

Calcium Content of Common Foods
1 cup milk = 300 mg
¾ cup yogurt = 332 mg
½ can salmon with bones = 240 mg
½ cup cooked broccoli = 33 mg
1 medium orange = 50 mg

Vitamin D Content in Food
- Milk fortified with vitamin D_3 contains 100 IU per 250 mL glass
- Foods such as margarine, eggs, chicken livers, salmon, sardines, herring, mackerel, swordfish, and fish oils (halibut and cod liver oils) all contain small amounts; supplementation is necessary to obtain adequate levels as dietary intake has minimal impact
- Most multivitamins provide 400 IU of vitamin D_3

EXTRA

Osteomalacia and Rickets

Definition

- osteopenia with disordered calcification leading to a higher proportion of osteoid (unmineralized) tissue prior to epiphyseal closure: rickets (in childhood), osteomalacia (in adulthood)

Etiology and Pathophysiology

Vitamin D Deficiency

- deficient uptake or absorption
  - nutritional deficiency
  - malabsorption: post-gastrectomy, small bowel disease (e.g. celiac sprue), pancreatic insufficiency
- defective 25-hydroxylation
  - liver disease
  - anticonvulsant therapy (phenytoin, carbamazepine, phenobarbital)
- loss of vitamin D binding protein
  - nephrotic syndrome
- decreased 1-α-25 hydroxylation
  - hypoparathyroidism
- renal failure

Mineralization Defect

- abnormal matrix
  - osteogenesis imperfecta
- enzyme deficiency
  - hypophosphatasia (inadequate ALP bioactivity)
- presence of calcification inhibitors
  - aluminum, high dose fluoride, anticonvulsants

Calcium Deficiency

- deficient uptake or absorption
  - nutritional deficiency
  - malabsorption
- hypercalciuria (in combination with renal phosphate wasting)

Hypophosphatemia

- gastrointestinal: poor nutritional intake, chronic diarrhea, excessive phosphate binders
- renal phosphate wasting
  - tumour-induced osteomalacia
  - Fanconi syndrome
  - X-linked/autosomal dominant/recessive hypophosphatemic rickets

Matrix Abnormalities

- type IV osteogenesis imperfecta
- fibrogenesis imperfecta ossium
- axial osteomalacia

Table 34. Clinical Features of Rickets and Osteomalacia

Rickets	Osteomalacia
Skeletal pain and deformities, bow-legged	Not as severe
Fracture susceptibility	Diffuse skeletal pain
Weakness and hypotonia	Bone tenderness
Disturbed growth	Fractures
Ricketic rosary (prominent costochondral junctions)	Gait disturbances (waddling)
Harrison's groove (indentation of lower ribs)	Proximal muscle weakness
Hypocalcemia	Hypotonia

Investigations

Table 35. Laboratory Findings in Osteomalacia and Rickets

Disorder	Serum Phosphate	Serum Calcium	Serum ALP	Other Features
Vitamin D Deficiency	Decreased	Decreased to normal	Increased	Decreased calcitriol
Hypophosphatemia	Decreased	Normal	Increased	
Proximal Renal Tubular Acidosis	Decreased	Normal	Normal	Associated with hyperchloremic metabolic acidosis
Conditions Associated with Abnormal Matrix Formation	Normal	Normal	Normal	

- radiologic findings
  - pseudofractures, fissures, narrow radiolucent lines – thought to be healed stress fractures or the result of erosion by arterial pulsation
  - loss of distinctness of vertebral body trabeculae; concavity of the vertebral bodies
  - changes due to secondary hyperparathyroidism: subperiosteal resorption of the phalanges, bone cysts, resorption of the distal ends of long bones
  - others: bowing of tibia, coxa profundus hip deformity
- bone biopsy: usually not necessary but considered the gold standard for diagnosis

Treatment

- definitive treatment depends on the underlying cause
- vitamin D supplementation
- PO_4^{3-} supplements if low serum PO_4^{3-}, Ca^{2+} supplements for isolated calcium deficiency
- bicarbonate if chronic metabolic acidosis

KDIGO 2017 Clinical Practice Guideline for the Evaluation and Management of Chronic Kidney Disease

Kidney Inter Suppl 2017;7(1):1-60

Recommendations for Metabolic Bone Disease (MBD) in Chronic Kidney Disease (CKD)

Screening

- In CKD patients with evidence of CKD-MBD and/or risk factors for osteoporosis, perform BMD testing to assess fracture risk if results will impact treatment decisions
- In patients with CKD-BMD, it is reasonable to perform a bone biopsy if knowledge of the type of renal osteodystrophy will impact treatment decisions

Management

- Treatment of CKD-MBD should be based on serial assessments of PO_4^{3-}, Ca^{2+}, and PTH levels, considered together
- Suggest lowering elevated PO_4^{3-} levels towards the normal range
- Avoid hyperglycemia in adult patients and maintain serum Ca^{2+} in age-appropriate normal range in children

EXTRA

Renal Osteodystrophy

Definition

- changes to mineral metabolism and bone structure secondary to CKD
- represents a mixture of four types of bone disease:
  - osteomalacia: low bone turnover combined with increased unmineralized bone (osteoid)
  - adynamic bone disease: low bone turnover due to excessive suppression of parathyroid gland
  - osteitis fibrosa cystica: increased bone turnover due to secondary hyperparathyroidism
  - mixed uremic osteodystrophy: both high and low bone turnover, characterized by marrow fibrosis and increased osteoids
- metastatic calcification secondary to hyperphosphatemia may occur

Pathophysiology

- metabolic bone disease secondary to chronic renal failure
- combination of hyperphosphatemia (inhibits $1,25(OH)_2$ vitamin D synthesis) and loss of renal mass (reduced 1-α-hydroxylase)

Clinical Features

- soft tissue calcifications, necrotic skin lesions if vessels involved
- osteodystrophy, generalized bone pain, and fractures
- pruritus
- neuromuscular irritability and tetany may occur (with low serum calcium)
- radiologic features of osteitis fibrosa cystica, osteomalacia, osteosclerosis, osteoporosis

Investigations

- serum Ca^{2+} corrected for albumin, PO_4^{3-}, PTH, ALP, ± imaging (x-ray, BMD), ± bone biopsy (gold standard; only done if results inform treatment)

Treatment

- prevention
- maintenance of normal serum Ca^{2+} and PO_4^{3-} by restricting PO_4^{3-} intake to 1 g once daily
- Ca^{2+} supplements; PO_4^{3-} binding agents (calcium carbonate, aluminum hydroxide)
- activated vitamin D (calcitriol) with close monitoring to avoid hypercalcemia and metastatic calcification
- bisphosphates and denosumab are not often used for treatment (can worsen the adynamic components of renal osteodystrophy); bone biopsy may indicate if there are signs of increased bone turnover amenable to bisphosphonates

Quiz

Q1: Which of the following is a sign of rickets on an X ray?

- A: Gunstock deformity
- B: Kyphoscoliosis
- C: Metaphyseal cupping
- D: Bone marrow expansion

Q2: A 45-year-old osteoporotic lady presented for check up. She smokes 10 cigarettes per day and reached menopause at the age of 41, Her mother had a hip fracture at the age of 61. Which of the following is the major risk factor for her to develop osteoporosis?

- A: Smoking
- B: Early menopause
- C: Family history of osteoporosis
- D: Low calcium intake

Q3: An 82-year-old woman presented with back pain. There was no history of trauma, fever or weight loss. Physical examination showed mild thoraco-lumbar kyphosis but no tenderness. Neurologic examination is normal. X ray showed a compression fracture. What is the next step?

- A: Start anti-osteoporotic medication
- B: Admission and bed rest
- C: Open reduction and internal fixation (ORIF)
- D: 6 weeks of halo femoral traction

4: Which of the following is the most common site of clinical and radiological findings in established diagnosis of rickets?

- A: Cranium
- B: Lower limb
- C: Upper limb
- D: Thoracic cage

1:C 2:B 3:A 4:B

Quiz

SAQs

441 & 439 & 438:

1. Name 2 factors mentioned in the lecture that control bone metabolism you think are the most important ones? And explain why for each point.?
   - Page 4

2. Sketch a tibia (bone) demonstrating two rickets radiographic findings labeled in the sketch.
   - Page 14

Done by

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وفّقكم الله

15

Orthopedic
441 Team

MED441
KING SAUD UNIVERSITY

Editing File

Common Foot & Ankle Problems

Dr. Abdulaziz Almaawi

Color Index:

- Main Text
- Important
- 441 Notes
- Old Notes
- Extra

Objectives

- To understand the anatomy of Foot and Ankle.
- To get a concise idea on common Foot and Ankle disorders.
- To differentiate from simple disorders and serious ones.
- To learn about initial management and prognosis.

Resources

Common foot and ankle problems

1. Flat foot
   very common
   20-25% of population
2. Hallux Valgus
3. Plantar fasciitis
4. Ankle sprains & instability
5. Osteochondral lesion of talus
6. Diabetic foot
7. Charcot foot

Importance of Foot and Ankle

- Subject to most weight bearing (Loading) of the body 1 kg increase in trunk will Increase weight bearing by 8 kg in ankle and 4 kg in knee
- Important proprioception function.
- Their appearance or deformity is easily noticeable while the patient is standing
- Faulty or improper shoe wear can cause symptoms.
- With advancing age; deformity becomes more symptomatic.
- Their sensory role is very important.

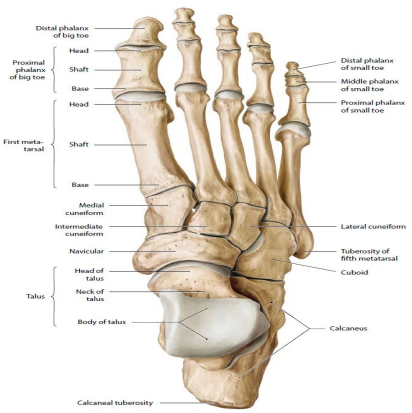
Anatomy of the Foot

- Body of Foot consist of (26 bones) and divided into:

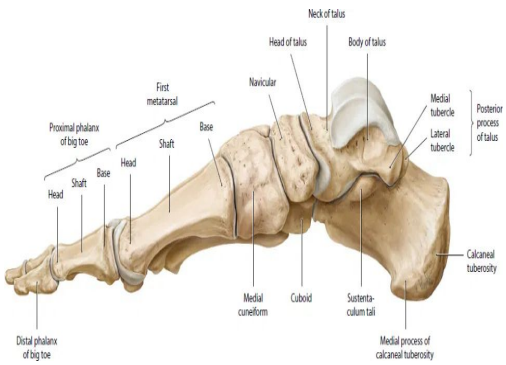
1. Fore Foot: Phalanges + Metatarsals
2. MidFoot : Navicular + Cuboid + 3 cuneiforms.
3. Hind(rear) foot: Talus(articulate with tibia to form ankle joint) + Calcaneus(articulate with talus to form subtalar joint)

Foot Skeleton

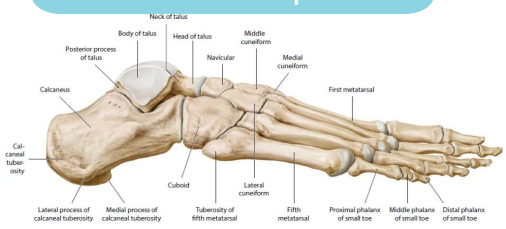
1- superior Aspect



2- Medial Aspect [1]



3- Lateral Aspect [2]

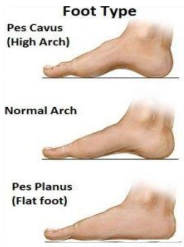


X-ray (standing) [3]

1-Talus and Calcaneus forms subtalar joint. We have more bones on the medial side than we have on the lateral side; that's because medial side gives stability while lateral gives flexibility
2-Mid Foot forms the arch of the foot
3- If siting shows no arch; Should be done in standing position since it's the function of the foot to bear weight

Flat Foot (Pes Planus)

Cavus foot: longitudinal axis of talus is pointing upwards exaggerated arch



- Reduction of longitudinal arches of the foot.
- Most cases are **developmental**[1]: i.e. arches do not develop normally[2].
- Usually is painless[3]
- Rarely acute flat = loss of the arch in case of an injury of the **posterior tibialis** tendon, unilateral
- Rigid flat (painfull) foot can be the result of tarsal coalition (fibrous or bony cross union between bones of the foot) "abnormal connection between bones in the back of the foot"
- Hind foot> valgus, Mid foot> reduction of longitudinal arch of the foot, Forefoot > abducted foot
- X-ray should be taken in standing position to show deformities

Normal X-ray

VS

Flat Foot Lateral weight bearing X-ray

Blue line: along the talus all the way to the metatarsal bone should be continuous with a 0 angle, up to 4 degree is considered normal. If the angle is >4 degree → considered flat foot.

Rigid or flexible flat foot

IMPORTANT

This table is important To differentiate between flexible and rigid flat foot

	**Normal**	**Flexible** most common (can be corrected without surgery)	**Rigid** Rare worse and can come at early age with early complication (needs surgery)
Alignment	Straight or minimal valgus	Extreme valgus	
Standing on tiptoes	The heel moves inward (from valgus to varus) the arch is still present	The heel moves inward (from valgus to varus), The deformity will be corrected the arch will reconstruct in flexible flat foot Hind foot> straight , Mid foot > reconstruction of arch, Fore foot > no abduction	Does not move inward (stay in valgus), Not correctable deformity the arch will remain absent in rigid flat foot
Subtalar motion (inversion/eversion)	Normal ROM	Normal ROM	**Reduced or absent**
Etiology	-	**Ligamentous** laxity (Present in almost 20% of the population)	**Tarsal coalition**[4]: Fibrous or bony union between bones of foot for fibrous we need to do MRI

1-Could be acquired in elderly due to posterior tibialis weakness. 2-Normally, the arch is formed within 4-6 years
3-80:20 rule: 20% of the population has it, 80% of them are asymptomatic,with time its become painful , and might have knee and back pain.
4-For tarsal coalition It has 2 type : 1-talocalcaneal and 2-calcaneonavicular

Flat foot:
- Hind foot: increased valgus
- Midfoot: lack of arch
- forefoot : abduction

Flexible tested via Beighton test which tests the ligament laxity

Flexible

Rigid

for example patient has flat foot so they have valgus, If they stand on their tippy toes and the valgus goes to straight and the arch is formed its flexible, if it can't go to normal it rigid

Management

always start with conservative:
1. Weight loss
2. Shoes
3. Orthotics (for arch support)
4. Activity modification
5. Physiotherapy

- Usually NO action is needed. (Asymptomatic)
- If Symptomatic **Always** start with conservative (5stpes) العلاج التحفُّظي
- **Foot exercises**: Strengthen muscles is important but will not correct deformity.
- **Orthotics/insoles** الدعامات: Protective, correct Malalignment
- **Good shoes:** beneficial on the long run.
- **Surgery**:

1. Rigid[1] flat foot. Removal of coalition for the risk of osteoarthritis
2. Painful flat foot with complication. Like arthritis
3. Acute flat foot. Fix it and the arch will come back

Imp to know difference between flexible and rigid flat foot because the rigid you need to do surgery it's usually show up at 12-14 if you catch it early it will be easy to fix but if you leave it until advanced age the pt will get arthritis

Hallux Valgus

- Means lateral deviation of big toe. it's associated with flat foot
- Usually at the metatarsophalangeal joint.
- Most cases are painless. (If painful, would be due to shoe pressure on large toe or an inflamed bunion due to arthritis.)
- Often is associated with a **bunion**[2] (swelling and protrusion at the medial aspect of big toe[3])
- Severe HV interferes with shoe wear.
- Common at middle age and elderly[4] mainly females[5]
- Possible causes for hallux valgus: 1-Flat foot "if pt is not using the arch the toe will deviate laterally" 2- Inherited bilateral 3- Shoe wear "heels" 4-Rheumatoid arthritis
- The pain isn't usually caused by the lateral deviation of the big toe, it's rather caused by the medial deviation of the 1st MTP joint.

1-Usually have gastrocnemius muscle tightness
2- Hallux valgus ≠bunion. **bunion** (swelling and protrusion at the medial aspect of big toe). Hallux valgus is the whole deviation of the big toe away from the central line. Sometimes people have a bunion without hallux valgus and we treat it by a bunionectomy; a surgery by which we remove the bunion. A bunion is not a growth instead it's exposure of the metatarsal head.
3-have many reasons most importantly the weight and the shoe.
4-Rheumatoid arthritis
5- Heels

Hallux Valgus Measurement

IMPORTANT

Hallux valgus angle: (no. 2)

- Angle between line extending along 1st metatarsal and a line extending along proximal phalanx.
- **Normal**: <15° **Mild HV:** 16-25 ° **Moderate HV**: 26-35°
  Severe HV: > 35°

1st intermetatarsal angle: (no. 4) most common and very important

- Angle between 1st metatarsal long axis and 2nd metatarsal
- Normal < 10°
- Severe > 15°

Hallux interphalangeus angle: To assess if the deformity within the toe (no.1)

- Angle between long axis of proximal and distal phalanges
- Normal < 8° Severe >13°

Normal Foot

Hallux Valgus

Management

- If painless (most common) reassure. Never treat for cosmetic purpose
- If painful (interfering with walking), always start with conservative (5-steps)→
- Correct and suitable **shoe wear.** Wider shoes
- Avoidance of **tight shoes.**
- Protection to the bunions. (by cushioning) Using orthotics "like ring cover the toe" or using silicon
- **Surgery** is reserved for symptomatic and disturbing cases. (if conservatives fail to relieve pain for 3-6 months)
  - Surgery is annoying (hardware on skin).
  - Removal of bunion is not cosmetic, patient has to be symptomatic "pain in the joint, pain in the 2nd toe, pain because of shoe wear".
- Following surgery; patient has to continue proper shoe wear.

always start with conservative:

1. Weight loss
2. Shoes
3. Orthotics
4. Activity modification
5. Physiotherapy

X-ray Hallux Valgus **pre-op**

X-ray Hallux Valgus **post-op**

Metatarsus went medially
Phalanx went laterally

There a lot of procedures, but in basic steps:
1- Correct deviated metatarsal.
2- Excise the part of the bunion not all of it.
3- Release Adductor hallucis longus tendon as it is a deforming force.

Plantar Fasciitis

إلتهاب اللفافة الأخمصية

- Common disorder at middle age and elderly. affect about 90% of the population
- Insidious in onset; unilateral or bilateral. Vague pain at heel region. You wake up with sudden pain
- **Localized tenderness** to insertion of plantar fascia into calcaneum.
- Plain lateral X-ray of heel frequently shows calcaneal spur[1] (مسمار القدم), (prominence or ossification at the site of anterior calcaneum at plantar fascia insertion site)[2]
- Commonly associated with flat feet. Also associated with increase weight, walking barefoot on hard surfaces
- No visible heel swelling, no skin changes and no increase in local temperature.
- Inflammatory process is at site of pain; i.e. at **plantar fascia insertion into calcaneum**.
- Heel pain like **stabbing pain** when patient puts foot to the ground first step in the morning; and gets less after some walking.[3]

Some of the possible causes of Plantar Fasciitis:
- Weight (overweight)
- Bad shoes (Too soft and flexible)
- Walking barefoot
- Tightness of the Gastrocnemius
- Flat foot

Imaging

X-ray: Bilateral Calcaneal Spur (Early)	X-ray: Calcaneal Spur (Advanced)
	 The body compensates with formation of calcification

Management

"In many cases, patients will not be compliant with these steps"

Always start with conservative:
1. Weight loss
2. Shoes
3. Orthotics
4. Activity modification
5. Physiotherapy
6. Steroids
7. Surgery

- Most important; **proper shoes & weight reduction**
- NO easy or simple management is available.
- **Mainly conservative.**
- Includes **stretching exercises** to plantar fascia: active and passive.
- Use of **soft heel insoles** (Silicone) may be helpful.
- **Shock wave therapy (SWT)** الموجات الصدمية بالتنظير الخارجي may be effective[4] . Resistant cases only
- Local **steroid** injections are helpful sometimes. Very resistant cases
- Surgery (very rare):last solution Release of gastrocnemius muscle "**NO surgery for plantar fascia itself**"
- Surgery if there's nerve compression

(435) If the first 5 steps of conservative didn't relieve the pain;
1. We do **MRI** to confirm diagnoses.
2. Give **steroid injection** if the steroidal injections didn't relieve the pain,
3. Do nerve conduction study to determine other causes. Other causes which although rare but give same symptoms of plantar fasciitis are: **tarsal tunnel syndrome** posterior to the medial malleolus compressing on lateral and medial plantar nerves (branches of posterior tibial nerve) and **nerve entrapment** due to large muscle in athletes.

1-No relation between the spike (spur) you see on x-ray and the pain. some patient have very large spikes with no pain and some have very severe pain with no spike, what we get from that is that it doesn't have to be removed to alleviate the pain.
2- It is an indication of inflammation for many years.
3-When we sleep or set for long periods our feet are in plantar flexion, making the achilles tendon short and when we step on the floor for the first time, we do dorsi flexion which stretches the achilles tendon stretching the plantar fascia with it causing pain.
4- To break down the thick tissue, which we don't know how it happens

Ankle Sprains

- One of the most common injuries.
- Usually occurs during sports activities, but may occur at home or street.
- The injury is **partial or complete ligament rupture.**

Clinical Features

★ History of **twisting** injury. Most commonly inversion

★ Pain, swelling and bruising at and around ankle. Swelling in look, tenderness in feel usually soft tissue not bone, sometimes can't walk

★ No tenderness of lateral malleolus; but tenderness anterior, posterior or inferior to it i.e. over ligaments.

★ Dorsiflexion and plantar flexion possible; but inversion and eversion very painful[1]

★ Positive anterior drawer test. If both sides positive its a laxity disease if only one side its ligamentous

★ X-Rays: **NO fracture**. You have to rule it out.

★ You need to know the first two ligaments

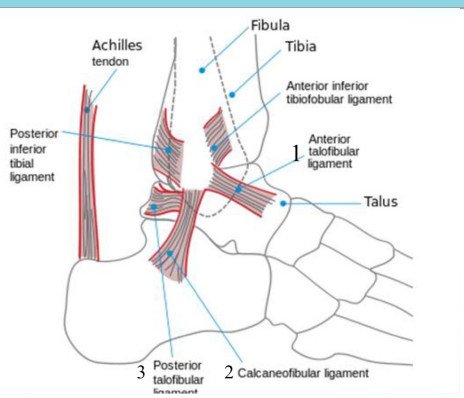
Lateral Collateral Ligament Complex

MRI is choice to determine which ligament is injured

Most commonly injured ligaments are :

1. **Anterior talofibular ligament (ATFL)** الرباط الكاحلي الشظوي الأمامي
   To test it do it with plantarflexion
2. **Calcaneofibular ligament (CFL)** الرباط العظمي الشظوي to test it do it with dorsiflexion
3. **Posterior talofibular ligament (PTFL)**.
4. **"Anterior drawer test is +",** Compare with normal side.

Most common cause of instability is Lateralization of the talus



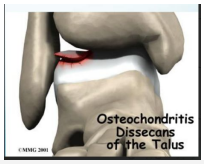
Management[2]

★ **RICE**: Rest, Ice, Compression, Elevation

- Apply **Back-slab splints** for few days جبيرة جبس خلفية if not able to weight bear.
- Might use protection with **brace** رباط ضاغط.
- **Early physiotherapy** العلاج الطبيعي and strengthening.
- Mostly heal without surgery (75% of cases)
- **Surgery**: if physiotherapy fails and there is clear instability.or if there is complete tear
- PRICES: recent view = Protection [3] , relative Rest, Ice, Compression, Elevation and support
- After the Treatment, Do MRI to assess prognosis.
- In summary, We sometimes apply back-slab splint for few days (**10 days**), then start early physiotherapy immediately, but we try to minimize using this option due to complications of cast applying like stiffness and muscle weakness

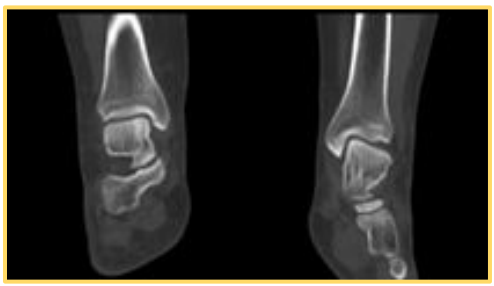
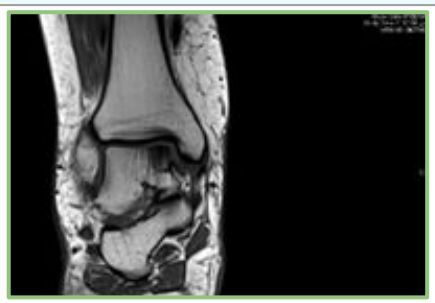
1- always compare, since it might be hyperlaxity
2-Always start with conservative, even if you are treating a professional athlete.
3-Splints are better than back slap casts because you can wear/remove splint anytime and splints allow dorsiflexion and plantar flexion movements

Osteochondral Defects of Talus[1] (ODC)



- Damage at localized areas of Talar articular cartilage.
- Lack of blood supply is often **post traumatic**, but occasionally No cause can be found.
- A local cartilage and varying depth of underneath bone are involved and may separate of main talus inside the ankle joint.
- Usually[2] **postero-medial part** of dome of talus. Bcs commonest sprains happen in inversion, which leads the tibia to impact the talus.
- Localized pain on weight bearing and even at rest may be present. Joint line tenderness especially in plantar- flexion + on & off swelling with walking a lot.

Imaging

Plain AP X-ray: lesion is suspected	**CT Coronal view: lesion highly suspected**	**MRI: lesion is confirmed**
		
Bone is white highly sclerotic (marble-like) indicating loss of blood supply, Very fragile and easily broken.		MRI with contrast the bone is white, and the lesion is black, due to loss of blood supply. **Modality of choice**

Management[3]

For size :
1- If less than 1 cm very good prognosis
2- Above 1 cm high risk of Arthritis, bad prognosis

- **Depends on:**

1. Symptoms: pain and recurrent swelling.
2. Size of OCD: large and Loose
3. Loose fragment (urgent surgery within few weeks "injury to cartilage *Calcaneum*")

- **Arthroscopic debridement**[4] تنضير المفصل of the lesion and drilling of its base. Better with minimally invasive + must be in certain site.
- **Fixation with headless screw** of large OCD with large bony part. For big pieces

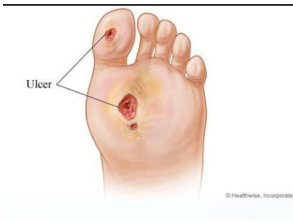
OCD you need to know

- Most common area is postero-medial part of dome of talus
- Best investigation to detect it is MRI

1. Ankle injury resulting in loss of bone and cartilage in talus.
2. 50% of cases
3. doesn't heal by itself, since it's avascular
4. 90% of cases are managed by Arthroscopic debridement

Diabetic Foot

- **Neuropathy** (nerve damage): Long term diabetes or failure to control diabetes.
- Numbness, tingling and reduced sensation of the feet.
- Associated **Decreased circulation** (neuropathy, calcification of vessels, CAD).
- May result in delayed healing, Infection, **Gangrene** and **Amputation**.
- How diabetes exactly causes neuropathy? By accumulation of sorbitol (sugar substitute) in nerves, that's happening in UNCONTROLLED DM



Care of Feet in Diabetes

Primary target: **Prevention:**

1. Blood sugar control. Best indicator H1AC should be less than 7
2. Daily self-inspection of feet is mandatory.
3. Member of the family or assistant should do it.
4. Regular inspections by healthcare personnel should be arranged.
5. A visit to a doctor should take place immediately whenever any complication occurs
6. If there's ulcer, orthopedic surgeon, general surgeon, plastic surgeon and vascular surgeon are working as a team to treat it

Surgery in Diabetic Foot

You have to make sure the blood supply is good before do the surgery

- Skilled care of wounds and ulcers in diabetic foot is **required**.
- **Wound debridement** تنضير الجرح, antibiotics and repeated dressing should be done.
- **Amputations**[1] may become necessary when there is **Gangrene**.
- Toe amputation or ray amputation, forefoot amputation, below or above knee amputation.

Charcot Foot[2]

- **Significant nerve damage to the foot leads to:**

1. The bones of the foot become weak.
2. Joints inflamed, swollen as if infected and lax.

After long standing diabetes

- Walking on the foot leads to disintegration and collapse of the joints and **deformity:** such as Rocker-bottom deformity قدم روحاء مقوسة .

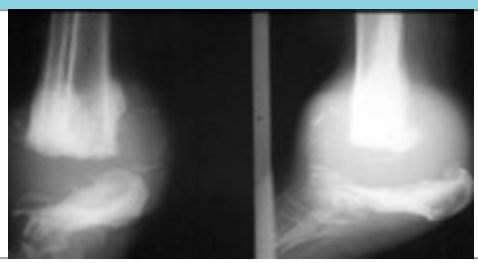
Etiology[3]

- Any disorder which lead to **Neuropathy**.
- There is decreased sensation and decreased ability to feel temperature, pain or trauma
- no pain

1- Our aim is to avoid amputation
2-bone degeneration due to loss of blood supply, in diabetics
3-Doctor said that it's a complex mechanism that we are NOT required to know, but in general it's neuropathy at first, then nerves send signals that they require more blood to regain function, so this will cause hyper perfusion of blood to the foot and will increase bone resorption as well, leading to new bone growth.

Charcot Foot Clinical Picture

- **Look**: Foot is red or dusky in color. There's swelling in the area and deformity.
  Often mistaken by osteomyelitis. To differentiate we do **elevation test** we raise the leg up for 20 minutes, If persistent redness and swelling it is infection if redness and swelling subside it is Charcot foot.
- **Feel**: NO Pain or soreness, warmness of foot.
- **Move**: decreased ROM.
- **X-ray** changes are important to detect and interpret;
  - **Early: NO changes.**
  - Later: haziness, osteopenia, irregular joint destruction, subluxation or even dislocation.
- X-Rays: NO fracture. You have to rule it out.

Diabetic Foot	Advanced Case of Charcot
	
Osteolysis of callus and talus ,There's amputation of big toe	Talus and calcaneus almost gone

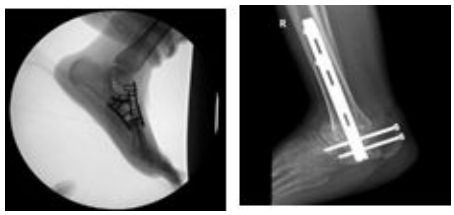
Diagnosis

Staging: development (little osteopenia), then destruction, then absorption then reconstruction.

- Good history[1] and clinical examination.
- Awareness.
- Exclusion of other causes which may give similar picture: like infection or tumor.
- MRI, bone scans can help.
- MRI/ CT to differentiate between Charcot and osteomyelitis

Management

In early stages avoid doing anything other than immobilization if you do the pt may end up with infection and osteomyelitis and amputation

Non-Surgical Management of Charcot Foot	Surgery in Charcot Foot	Amputation in Charcot foot
• **Immobilization** • **Custom Shoes and Bracing** we don't do cast because it might cause ulcers in a diabetic patient with neuropathy • **Activity modification**	Usually we wait for 1.5 or 2 years until reach last stage then we do reconstruction of ankle or foot depending on area **[we don't do surgery until blood sugar is controlled]** 	• May be indicated as a last option. • Mainly when there is severe instability which cannot be controlled by surgery or orthosis تقويم العظام. • Also when surgery fails to achieve stability. • Presence of refractory infection increase the possibility of amputation.

1-Always ask about diabetes and whether it's controlled or not

EXTRA

Diabetic Foot Infections

Etiology
- neuropathy, peripheral vascular disease, and hyperglycemia contribute to foot ulcers that heal poorly, and are predisposed to infection
- organisms in mild infection: *Streptococcus* spp., *S. aureus*
- organisms in moderate/severe infection: polymicrobial with aerobes (*S. aureus, Streptococcus, Enterococcus*, GNB) and anaerobes (*Peptostreptococcus, Bacteroides, Clostridium*)

Clinical Feature
- not all ulcers are infected
- consider infection if: probe to bone (see below), ulcer present >30 d, recurrent ulcers, trauma, PVD, prior amputation, loss of protective sensation, renal disease, or history of walking barefoot
- diagnosis of infected ulcer: ≥2 of the cardinal signs of inflammation (redness, warmth, swelling, pain) OR the presence of pus
- ± crepitus, osteomyelitis, systemic toxicity
- visible bone or probe to bone → osteomyelitis
- infection severity
  - mild = superficial (no bone/joint involvement)
  - moderate = deep (beneath superficial fascia, involving bone/joint) or erythema >2 cm
  - severe = infection in a patient with systemic toxicity (fever, tachypnea, leukocytosis, tachycardia, hypotension)

Investigations
- curettage specimen from ulcer base, aspirate from an abscess or bone biopsy (results from superficial swabs do not represent organisms responsible for deeper infection)
- blood C&S if febrile
- assess for osteomyelitis by x-ray (although not sensitive in early stages) or MRI/bone scan if high clinical suspicion
  - if initial x-ray normal, repeat 2-4 wk after initiating treatment to increase test sensitivity

Treatment
- mild to moderate: cefazolin or cephalexin
- severe: options include- 1. ceftriaxone + metronidazole; 2. piperacillin/tazobactam ± vancomycin; 3. meropenem ± vancomycin
- optimize glycemic control, pressure offloading, wound care, consider revascularization

This is empiric treatment, and specific treatment needs to be adjusted based on culture and response to therapy

Does this Patient with Diabetes have Osteomyelitis of the Lower Extremity?
JAMA 2008;299:806-813
Study: Systematic literature review. 21 studies.
Population: 1027 adult patients with DM being investigated for osteomyelitis.
Intervention: Various aspects of history, physical exam, laboratory tests, and diagnostic imaging studies versus bone biopsy.
Primary Outcome: Diagnostic utility.
Results: No studies examined any part of history taking. Temperature, ulcer characteristics (erythema, swelling, purulence), elevated WBC, skin swabs, and soft tissue cultures were not useful. Nuclear imaging has poor specificity for osteomyelitis (62%-88.5%), and MRIs have greater accuracy in detecting osteomyelitis.

Ankle Ligamentous Injuries

- see Figure 47 for ankle ligaments

Medial Ligament Complex (deltoid ligament)
- eversion injury
- usually avulses medial or posterior malleolus and strains syndesmosis

Lateral Ligament Complex (anterior talofibular, calcaneofibular, posterior talofibular)
- inversion injury, >90% of all ankle sprains
- ATF most commonly and severely injured if ankle is plantarflexed
- swelling and tenderness anterior to lateral malleolus
- ++ ecchymosis
- positive ankle anterior drawer
- may have significant medial talar tilt on inversion stress X-ray

Treatment
- non-operative
  - microscopic tear (Grade I)
    - rest, ice, compression, elevation
  - macroscopic tear (Grade II)
    - strap ankle in dorsiflexion and eversion x 4-6 wk
    - physiotherapy: strengthening and proprioceptive retraining
  - complete tear (Grade III)
    - below knee walking cast x 4-6 wk
    - physiotherapy: strengthening and proprioceptive retraining
    - surgical intervention may be required if chronic symptomatic instability develops

Figure 46. Ring principle of the ankle and Danis-Weber classification

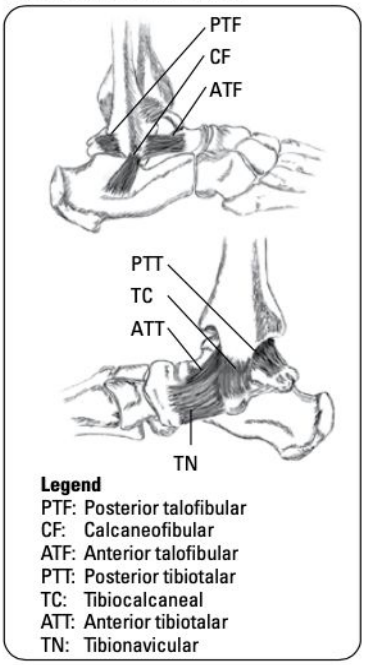


Figure 47. Ankle ligament complexes

With a history of significant trauma from axial loading of lower limb, always consider spinal injuries, femoral neck, tibial plateau, and talar/calcaneal fractures

EXTRA

Plantar Fasciitis (Heel Spur Syndrome)

Definition
- inflammation of plantar aponeurosis at calcaneal origin
- common in athletes (especially runners, dancers)
- also associated with obesity, DM, seronegative and seropositive arthritis

Mechanism
- repetitive strain injury causing microtears and inflammation of plantar fascia

Clinical Features
- insidious onset of heel pain, pain when getting out of bed, and stiffness
- intense pain when walking from rest that subsides as patient continues to walk, worse at end of day with prolonged standing
- swelling, tenderness over sole
- greatest at medial calcaneal tubercle and 1-2 cm distal along plantar fascia
- pain with toe dorsiflexion (stretches fascia)

Investigations
- plain radiographs to rule out fractures
- often see bony exostoses (heel spurs) at insertion of fascia into medial calcaneal tubercle
- spur is secondary to inflammation, not the cause of pain

Treatment
- non-operative
  - pain control and stretching programs are first-line
  - rest, ice, NSAIDs, steroid injection
  - physiotherapy: Achilles tendon and plantar fascia stretching, extracorporeal shockwave therapy
  - orthotics with heel cup – to counteract pronation and disperse heel strike forces
- operative
  - very rarely indicated
  - when performed, includes endoscopic release of fascia

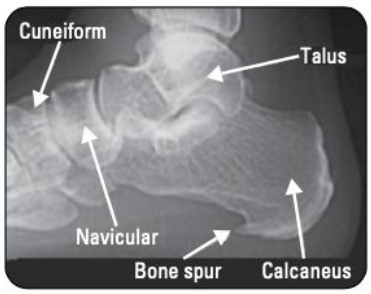


Figure 48. X-ray of bony heel spur

Surgical vs. Nonsurgical Methods for Acute Achilles Tendon Rupture: A Meta-Analysis of Randomized Controlled Trials
J Foot Ankle Surg Am. 2018 Nov - Dec; 57(6): 1191-1199
Purpose: To compare surgical treatment and conservative treatment of acute Achilles tendon rupture.
Methods: A meta-analysis was performed looking at randomized trials comparing surgical with nonsurgical treatment or comparing different surgical treatments of Achilles tendon rupture.
Results: 10 randomized clinical trials with a total of 934 randomized patients were included. Patients in the non-surgical group had a higher re-rupture rate than patients in the surgical group. However, re-rupture rates were equivalent (p = .08) if an early range of motion exercises protocol was performed. Lower incidence of complications (excluding re-rupture) was found in non-surgical patients.
Conclusions: Non-surgical treatment for acute Achilles tendon rupture is preferred if a functional rehabilitation protocol with early range of motion is possible. If not, surgical treatment should be considered because of the lower rate of re-rupture.

Bunions (Hallux Valgus)

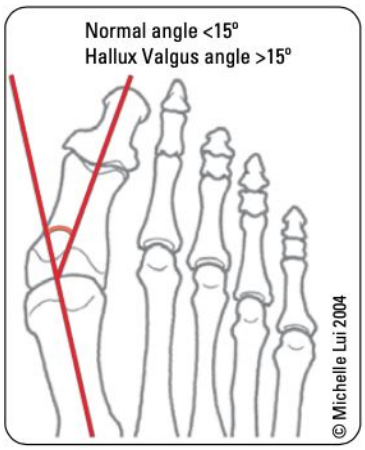
Definition
- bony deformity characterized by medial displacement of first metatarsal and lateral deviation of hallux

Mechanism
- many associated deformities in foot from altered mechanics
- valgus alignment of 1st MTP (hallux valgus), loose medial and tight lateral joint capsule, adductor hallucis becomes a deforming force
- formation of a reactive exostosis and thickening of the skin creates a bunion
- associated with poor-fitting footwear (high heel and narrow toe box)
- can be hereditary (70% have family history)
- 10x more frequent in women

Clinical Features
- painful bursa over medial eminence of 1st MT head
- pronation (rotation inward) of great toe
- numbness over medial aspect of great toe

Investigations
- X-ray: standing AP/lateral/sesamoid view, NWB oblique

Treatment
- indications: painful corn or bunion, overriding 2nd toe
- non-operative (first-line)
  - properly fitted shoes (low heel) and toe spacer
- operative: goal is to restore normal anatomy, not cosmetic reasons alone
  - osteotomy with realignment of 1st MTP joint (Chevron Procedure)
  - arthrodesis



Quiz

Q1: Which of the following movements is reduced in rigid pes planus?

- A: Plantarflexion/Dorsiflexion
- B: Inversion/Eversion
- C: Internal/External rotation
- D: All of the above

Q2: At which angle is Hallux Valgus considered moderate?

- A: ≤15°
- B: 16-25°
- C: 26-35°
- D: ≥36°

Q3: What's the modality of choice used to diagnose Osteochondral defects of the talus?

- A: U/S
- B: X-ray
- C: CT-scan
- D: MRI

Q4: Tightness in which of the following muscles is associated with Plantar Fasciitis?

- A: Soleus
- B: Gastrocnemius
- C: Posterior tibialis
- D: Flexor hallucis longus

SAQs

1. **Name Two tests used to differentiate between flexible and rigid flat foot?**
2. **What are the angles used to assess the severity of Hallux Valgus?**

1. - Standing on tiptoes
   -Subtalar movement
2.
   - Hallux Valgus angle
   - Intermetatarsal angle
   - Hallux Interphalangeal angle

1:B 2:C 3:D 4:B

Team Leader

Abdulrahman Alroqi

Done by

Waleed Alsuhaibani

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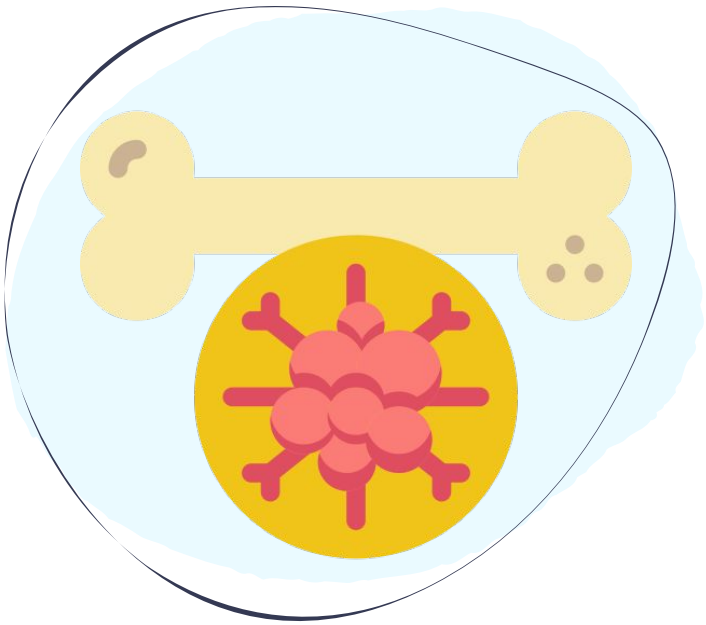
16

Orthopedic 441 Team

MED441 KING SAUD UNIVERSITY

Editing File

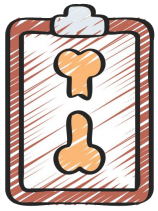


MSK Tumors

Dr. Ibrahim Alshaygy

Color Index:

- Main Text
- Important
- 441 Notes
- Old Notes
- Extra



Objectives

- Discuss presenting history and physical examination features of bone tumors.
- Discuss imaging characteristics of bone tumors.
- Discuss biopsy principles and techniques for bone tumors.

Resources

Classification of bone tumors

- Benign[1]
- Malignant[2]
  - Primary
  - Secondary (Malignant transformation of benign process)
  - Metastatic[3]

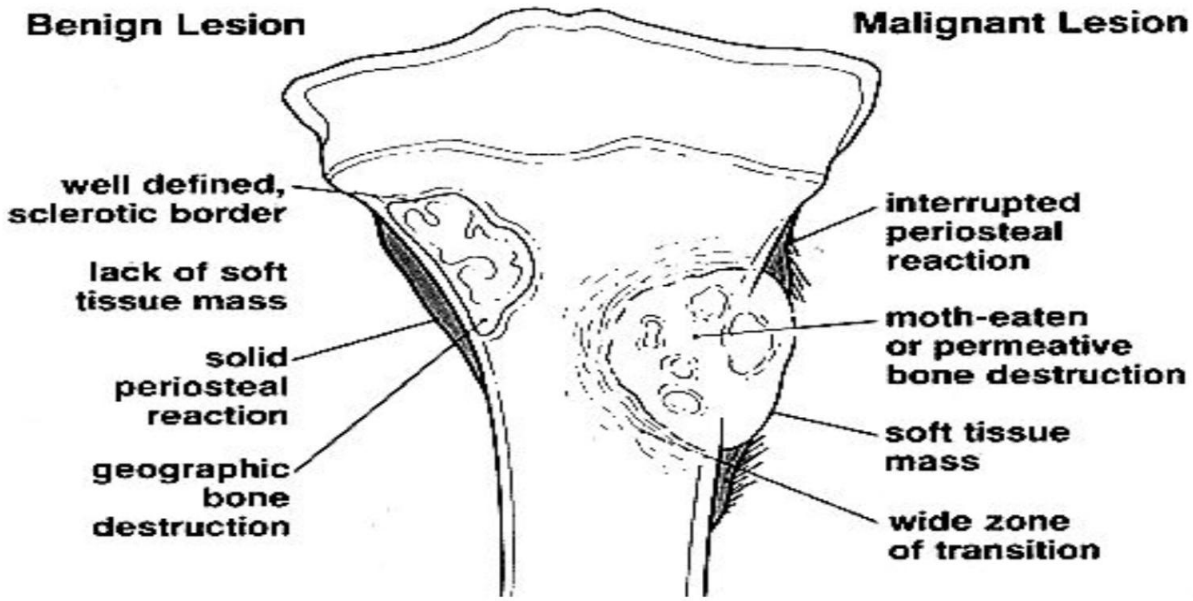


Table 25. Distinguishing Benign from Malignant Bone Lesions on X-Ray

Benign	Malignant
No periosteal reaction or benign appearing reaction (e.g. uniform smooth periosteal thickening as seen in a healing fracture)	Acute periosteal reaction • Codman's triangle • "Onion skin" • "Sunburst"
Sharp, well-demarcated borders, narrow zone of transition (between lesion and normal bone, suggesting slow-growing lesion)	Poorly defined borders, with a wide zone of transition, or infiltrative (suggesting fast-growing lesion)
Well-developed bone formation Intraosseous and even calcification	Varied bone formation Extraosseous and irregular calcification
No soft tissue mass	Soft tissue mass present
No cortical destruction or uniform cortical destruction in some low grade and locally aggressive benign lesions	Aggressive cortical destruction or tumour infiltration without cortical destruction

Adapted from: Buckholtz RW, Heckman JD. Rockwood and Green's Fractures in Adults. Volume 1. Philadelphia: Lippincott Williams & Wilkins, 2001. p558

1-Most bone tumors are benign. Common in pediatrics.
2-Most malignant tumors are metastatic. Primary is very rare.
3-paired organs (such as breasts, lungs, kidneys) and organs with 2 lobes (such as thyroids, prostates) are the most common primary carcinomas which might metastasise to the bone and cause secondary lesions in the bone.
-Embryological origins:

- Ectoderm-nerves, skin
- Endoderm-viscera
- Meso-bone, cartilage, muscle, nerves, blood vessels

Classification of bone tumors

The red color is based on previous teams not 441

Histologic type	Benign	Malignant
Bone marrow Hematopoietic and lymphatic	Giant cell tumor (osteoclastoma), Eosinophilic granuloma, Lymphangioma	Myeloma (plasmacytoma), Malignant giant cell tumor, Histiocytic lymphoma, Ewing's sarcoma, Leukemia
Cartilage-forming Chondrogenic	Enchondroma (chondroma), Periosteal (juxtacortical) chondroma, Enchondromatosis (Oiller's disease), Osteochondroma (osteocartilaginous exostosis, single or multiple) Chondroblastoma, Chondromyxoid fibroma	Chondrosarcoma (central), Conventional, Mesenchymal, Clear cell, Dedifferentiated, Chondrosarcoma (peripheral), Periosteal (juxtacortical)
Bone-forming Osteogenic	Osteoma, Osteoid osteoma, Osteoblastoma	Osteosarcoma (and variants), Juxtacortical osteosarcoma (and variants)
Histiocytic origin	Fibrous histiocytoma	Malignant fibrous histiocytoma
Fibrous and fibrohistiocytic Fibrogenic	Fibrous cortical defect (metaphyseal fibrous defect), Nonossifying fibroma, Benign fibrous histiocytoma, Fibrous dysplasia (mono- and _polyostotic), Periosteal desmoid, Desmoplastic fibroma, Osteofibrous dysplasia (Kempson- Campanacci lesion), Ossifying fibroma (Sissons' lesion)	Fibrosarcoma, Malignant fibrous histiocytoma
Neural Neurogenic	Neurofibroma	Malignant schwannoma
Vascular	Hemangioma, Glomus tumor, Cystic angiomatosis	Hemangioendothelioma, angiosarcoma, Hemangiopericytoma
Fat Lipogenic	Lipoma	Liposarcoma
Notochordal	Neurilemmoma	Chordoma
Unknown origin	Simple bone cyst, aneurysmal bone cyst, Intraosseous ganglion	Adamantinoma

★ The diagnosis of tumors is made by History, physical examination, and investigations.

Key History Questions:

- Onset of pain: traumatic vs. atraumatic (Ask the pts if he/she has this pain prior to the trauma?)
- Progressive pain: indicates metabolic activity in the bone.
- Rest pain (to exclude mechanical pain), Night pain (night pain that fades with NSAIDs is **osteoid osteoma**).
- Relieving factors (NSAIDs)
- Family history.
- History of radiation (radiotherapy), Paget's disease, other cancers, systemic diseases.
- Constitutional symptoms (Weight loss, fever, night sweat, loss of appetite), not the primary presentation, as they present late (metastasis).
- DDx of any tumor is infection.

Clinical Features ★

- Pain
  Not just mechanical, usually even at rest
- Pathologic fractures
  Disease eating the bone —->bone become weak—->easily break
- Mass[1]
- Incidental findings on x-ray

Physical Exam

- Lump/mass physical exam:
  - 2 Ts: Temperature and Tenderness.
  - 3 Ss: Site, Size and Shape.
  - Margins (demarcated or not), consistency (solid or not), pounding pulse (e.g. Pulse proximal to tumor), decreased pulse distal to tumor?
  - Fixed or mobile[2].
- Lymphadenopathy: To check if there is metastasis or not.
- Neurovascular examination.

Investigations

- **Lab**: CBC (infection or anemia), ESR, CRP, Acute phase reactive molecules, hematological investigations
- **Imaging**:
  1. Local: X-ray (for the entire segment, cause some tumors have skip feature), MRI, CT.
  2. Systemic: chest CT, Bone scan.
- Biopsy: biopsy should be done by who will do the definitive surgery, ideally orthopedic oncologist or interventional radiologist who works with the orthopedic oncologist.

1-Progressive, mostly painless..pain if it compressed muscle, nerve ...etc.
2-how do we differentiate?
- Mass is fixed during relaxation and Contraction = fixed to the bone.
- Mass is mobile during relaxation and fixed during Contraction = deep to the superficial fascia (in the muscle).
- Mass is mobile in both contraction and relaxation = above the superficial fascia.

MSK bone tumors

Nine questions of Tumor Staging

1- Where is the lesion?

- Which bone? Which side?
- Which part of the bone? Epiphyseal (such as chondroblastoma), metaphyseal (such as osteosarcoma), diaphyseal (such as Ewing).
- Surface.
- Periarticular.
- Central or eccentric.
- The pic shows a tumor in the epiphyseal-metaphyseal area, eccentric, periarticular with cortical erosion, no periosteal reaction. There is a pathological fracture.

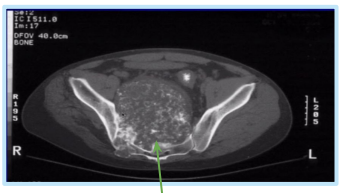
No clear demarcation

2- How big is the lesion

Size of mass is directly correlated to its ability to metastasize (size of army, # of cells)
-track how fast it grows over time
-compression of surrounding structures (localization is a mere important for that though)

osteoid osteoma (Benign)

Neck lesion 1x0.5 cm

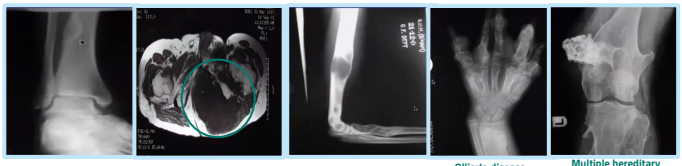


15x8 cm (Huge mass)

3- Is it solitary or multifocal?

The only way to know this is by getting the entire segment of the tumour and sometime we do skeletal survey to upper and lower extremities if we are suspecting it has metastasized to other sides.

Solitary | Multifocal



Note[1] | Ollier's disease (Aggressive) | Multiple hereditary exostosis (Benign)

4- What is the interface between the bone and the lesion?

Don't say it's a malignant say aggressive, cause aggressive either benign or malignant.

Good demarcated sclerotic lesion

عكس هنا ما نعرف وين ينتهي الورم ووين يبدأ

Well defined lytic lesion

Ill defined lesion having Periosteal reaction (codman triangle) = bone try to form periosteal but مايلحق

5- Is there a periosteal reaction? (Thin layer surrounding the bone.)

Body's way of trying to contain it. Not a part of the tumor. "Sclerotic rim"

Sunburst
usually in Ewing Sarcoma

Cloud-like = sign of Bone-forming tumor

Codman triangle
(Commonly with osteosarcoma)

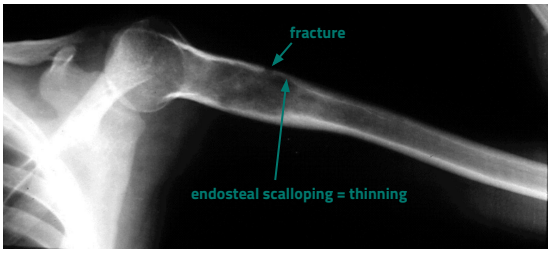
1-Right pic: Here you focused on the big tumor in the humerus so you told me that's a solitary tumor, but there is a pathological fracture in forearm, its thin and not easy to detect but if you look carefully it does not not look normal, so it's not actually solitary. That's why we emphasize to do x-ray for the entire segment.

MSK bone tumors

Nine questions of Tumor Staging

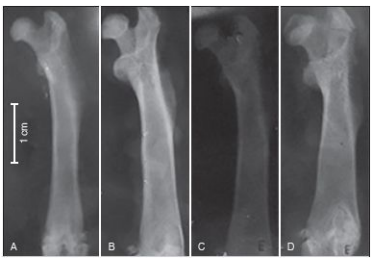
6- Is the cortex eroded (thinning)?

-The more aggressive the tumor is, the more it preserves the cortex.

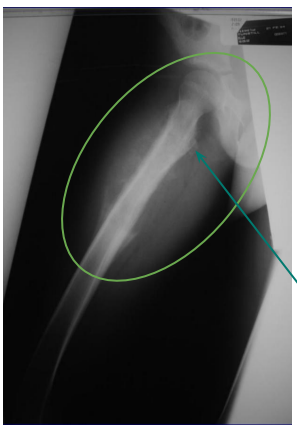


7-Is there bony remodeling?

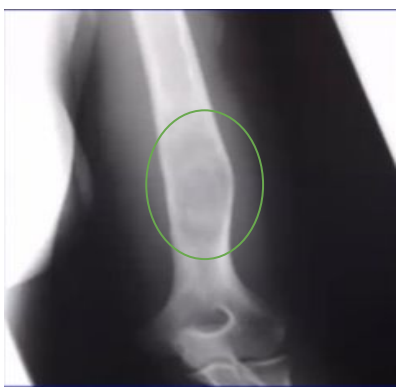
-The normal bone try to contain lesion (and this usually happens in benign lesion).



8- Is there a soft tissue mass?



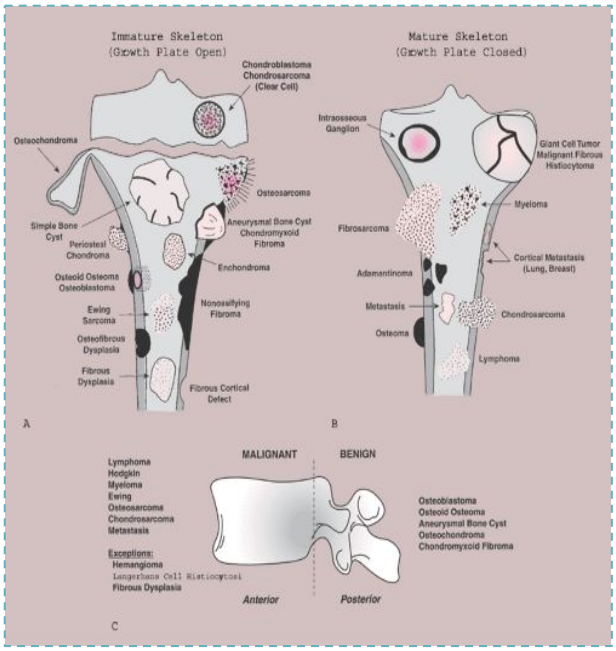
9- Is there any matrix?

	Lytic		Sclerotic
Types of matrix	**Fibrous**	**Cartilaginous**	**Osseous bone**
Picture			
Features	Fibrous: ground glass	Cartilage: popcorn like /arcs and rings appearance كأنه واحد راسم نقاط	Bone forming (Cloud-like)

X-Ray lesion description should include:

Number:

- Is the lesion solitary or are there multiple lesions? [1]



Site:

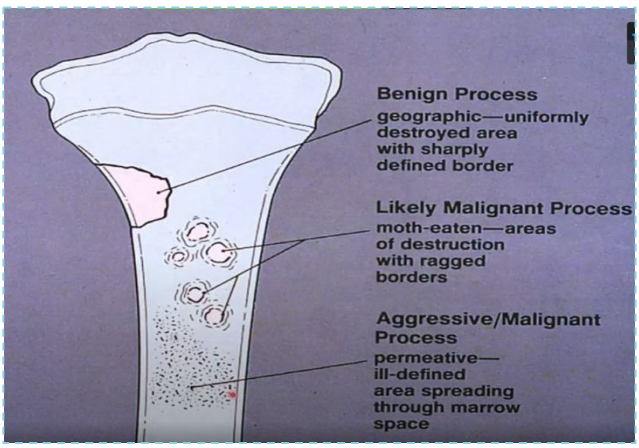
What bone is involved & where is the lesion in the bone?

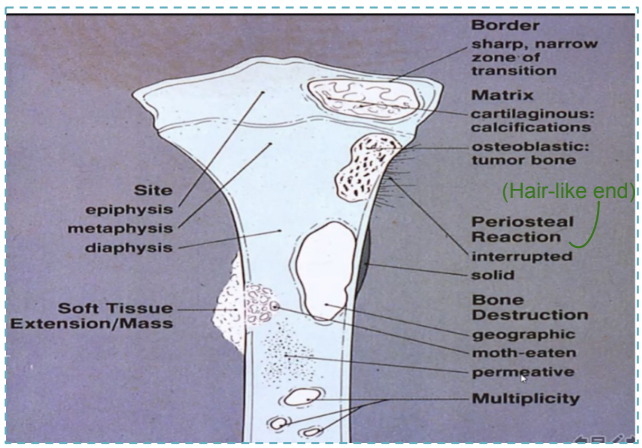
1. Epiphysis.
2. Metaphysis. (most common)
3. Diaphysis.
4. Centric (in the middle of the bone).
5. Eccentric (in the bone border).

Distribution of various lesions in a vertebra:
1- Malignant lesions are seen predominantly in its anterior part (body).
"Most likely" exceptions: hemangioma, Langerhans cells, fibrous dysplasia.
2- Benign lesions predominate in its posterior elements.

Type of bone destruction (morphology):

- Geographic (Organized uniformly destroyed area with sharply defined border): benign.
- Moth eaten كأنها ماكلتها العثة (areas of destruction with ragged/disorganized border): likely malignant.
- Permeative "همجية" (ill-defined areas spreading through bone marrow): aggressive/ malignant.





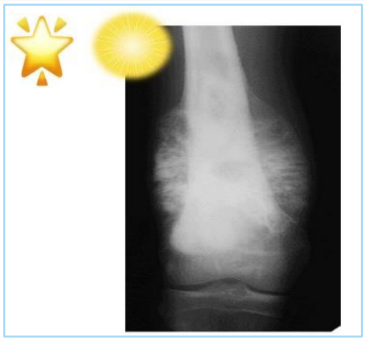
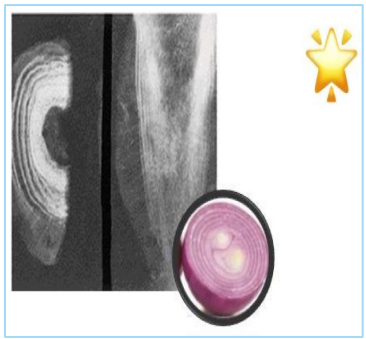
1- DDx for Multiplicity: metastases, myeloma, lymphoma, fibrous dysplasia, enchondromatosis.

Border or zone of transition:

- Well defined & sharp borders (either sharp sclerotic or sharp lytic), narrow zone of transition → benign (grow slowly > encapsulate by bone).
- Ill-defined borders, wide zone of transition → malignant tumors (fast, the bone didn't have the time to encapsulate it).

Periosteal reaction:

- ❖ Uninterrupted periosteal reaction usually indicates a benign process (solid clear buttress).
- ❖ Interrupted/large (teeth like) reaction indicates a malignant or aggressive nonmalignant process, and we have 3 types of these:

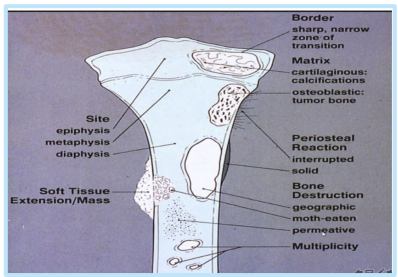
Sunburst pattern[1] in osteosarcoma and in Ewing Sarcoma	**Lamellated or onion-skin type in Ewing Sarcoma**	**Codman triangle (arrow) in Ewing Sarcoma and usually appears in Osteosarcoma**
		

Matrix of the tumor:

- **Sclerotic**: it means bone forming (osseous). So, on X-ray → opaque/white.
- **Lytic**: it means forming tissue other than bone "fluid" (it may be cartilage, fibrou tissue, or cystic), on X-ray → translucent/ black (e.g. popcorn calcification = chondroid).

Soft tissue extension (swelling/ shadow):

- Mostly with malignant tumors (MRI is ideally used).



1-when the periosteum does not have enough time to lay down a new layer and instead the Sharpey's fibres stretch perpendicular to the periosteum.

The spectrum:

Types	characteristics
Benign latent[2] Watch. Follow up in 1 yr.	• Asymptomatic. (if there is pain it may be from trauma, not the tumor) • Well defined. • Narrow zone of transition (geographic). • No soft tissue mass. • No periosteal reaction. • No fracture. • May or may not have matrix. • Examples: enchondroma[1], non-ossifying fibroma
Benign Active[2] Follow up, images. Grows with child.	• **Symptomatic.** (pain not attributed to trauma) • Geographic. • Well-ordered periosteal reaction. • No soft tissue mass. • May or may not have matrix. • e.g: **osteoid osteoma**, UBC, eosinophilic granuloma, fibrous dysplasia, osteochondroma.  Unicameral bone cyst
Benign Aggressive -Destroying bone. -Jump on them! follow up .	• Symptomatic. • Geographic or permeative. • Usually lytic, cortical erosion. • May have soft tissue mass. • Periosteal neocorticalization. (It mean the tumour might go beyond of the cortex, eat the cortex and the bone try to build new cortex) • e.g: **GCT, ABC, osteoblastoma**, chondroblastoma, chondromyxoid fibroma, periosteal chondroma.
Low Grade Malignant	• Usually permeative. • May have matrix. • Cortical erosion. • May have soft tissue mass in continuity with cortical erosion. • Low-grade CSA[1], Adamantinoma (only in ant. tibia), Parosteal OSA, Chordoma (in the sacrum). • Image shows suspicious lytic lesions with popcorn like matrix.  Gas Cortical thinning Chondrosarcoma (CSA)
High Grade Malignant	• Permeative. • Usually has soft tissue mass. • Cortex usually intact. (tumor penetrates the cortex) or destroyed • Malignant periosteal reaction (onion skinning, sunburst, codman's triangle) • May or may not have matrix. • **Osteosarcoma, Ewing's sarcoma**, high grade chondrosarcoma, non-osteogenic spindle cell sarcoma (eg. MFH).

1-Enchondroma vs Chondrosarcoma: both will show popcorn pattern but CSA will have gas and cortical thinning.
2-Benign latent is distinguished from active by the presence of pain.

What's next? Staging:

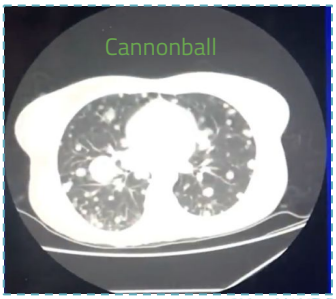
Staging:

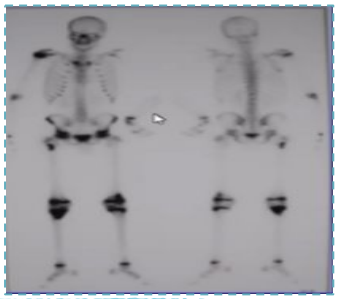
- If lesion is **benign** latent, no further investigation is usually necessary, may follow up after 6m. (Benign active vs benign latent > pain)
- If **benign active or aggressive**, requires further local imaging, perhaps systemic staging.
- If **malignant**, requires further investigation including local and systemic staging.

Local and systemic staging:

- Blood Work – CBC, ESR, CRP, serum calcium, **Alkaline phosphatase , LDH (latter 2 are prognostic in sarcoma)** (Ca, ALP and LDH indicate metabolic activity (bone turnover))
- **Local x-ray =** whole bone (Because some tumours have something called skip lesions (which is mass completely separated from the primary location of the tumour in the same bone)., chest x-ray (To rule out lung* metastasis)
- MRI of local site: must cover entire bone (full length MRI) may find lesions in proximal which is called skip lesions (occasionally CT = if there is soft tissue) (whats the **only** tumor that requires a full body MRI? **Myxoid liposarcoma**)
- CT chest to rule out metastasis.
- Total body bone scan for other lesions.
- For Ewing sarcoma we should order a gallium scan.
- ★ We should take a bone marrow aspiration for lymphoma, ewing sarcoma and MM.

Skip lesions (full length MRI)





Systemic staging

Metastasis?

>40 = metastasis | <40 = primary tumor

- Bloodwork – same bloodwork plus PSA prostate surface antigen, serum immunoelectrophoresis (in case of lymphoma and multiple myeloma for M band).
- CT chest and abdomen and pelvis to look for the source (lungs most commonly affected)
- Mammogram.
- Bone scan* other area are involved.

How to stage tumors?[1]

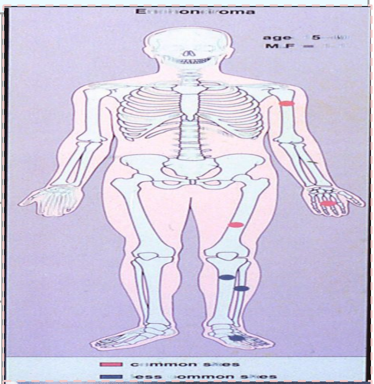
- Benign Latent/Active: Local - X ray +/- CT/MRI +/- TBBS = total body bone scan.
- Benign Aggressive: Local - X Ray/CT/MRI /Systemic - TBBS, CXR.
- Malignant: Systemic - CT Chest (<40) and bone scan , TBBS.
- Special: Gallium scan, CT Abd + Pelvis, Bone marrow biopsy.

1-Because lungs receive large venous supply which drains tumor .
2-PET Scan (how biologically active the tumor is).
-There is only one case in which we do total body MRI which is mexoid liposarcoma. The doctor told us it is coming in the exam but it did not :(

Primary Benign Bone Tumors:

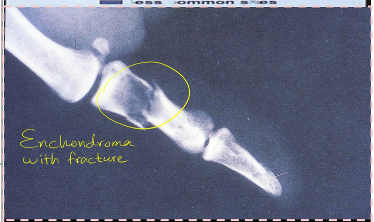
Benign Latent Tumors

"441: ماني سائلكم فيها" *(1) Enchondroma*

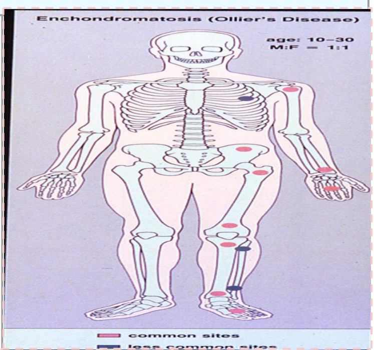


- Middle aged patients (15-50 age group).
- It is composed of translucent hyaline cartilage (chondroid).
- **Mostly found in the fingers.**

Site	Mainly small bone e.g. phalanges in hand & foot…etc.
Presentation	• **Asymptomatic**: Usually found incidentally. • Rings may become tight due to the swelling. • Pathological fracture which may cause pain, affect one side and prolonged healing.
Radiographic features	• **Popcorn matrix is characteristic for cartilaginous content.** • Benign features. • Can be seen on CT.
Treatment	• If symptomatic (pain, bulky, fracture) → Curettage + bone graft ± fixation.

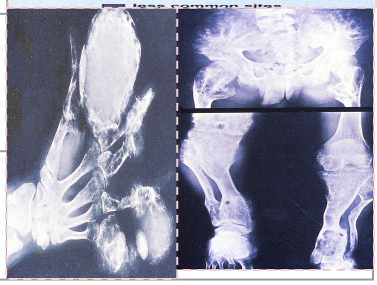


Enchondromatosis (Ollier's Disease)



- Multiple sites of enchondroma in the body most common sites are proximal humerus/tibia, which are premalignant.
- Autosomal recessive
- Benign, **affects both sides**. Usually seen in children. Rare.
- The difference between single enchondroma and multiple enchondromatosis is the high risk of malignant transformation (10-15% transforms into **chondrosarcoma,** which is chemo- and radio- resistant).

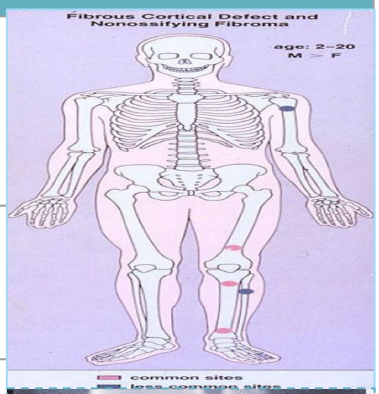
Presentation	• Not painful. • Very disabling deformity → restricts movement.
Treatment	• Only remove painful lesions.



Primary Benign Bone Tumors:

(2) Fibrous Cortical Defect and Non-ossifying Fibroma (NOF)

- A benign incidental finding, it is benign latent (never transfers to malignant).
- Found in children and it's usually an incidental finding.



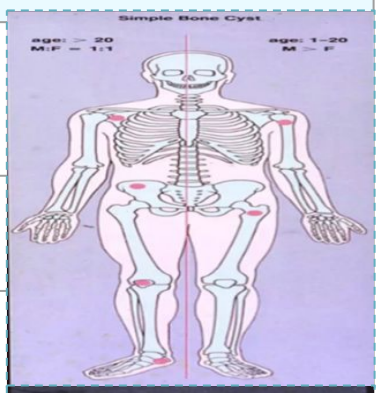
Site	• Around the knee (lower femur and proximal tibia) • Lower tibia • May appear on proximal humerus
Presentation	• **Asymptomatic.**
Radiographic features	• On x-ray, always found on the cortex (eccentric). Contains fibers and not clear material (ground glass appearance). • **FIBROUS COMPONENT!!!** • Metaphyseal Mixed lesion (cystic + sclerotic components) • Lesion that appears to be 'central' is actually adjacent to or within the cortex, cortex intact, hence the alternative name 'fibrous cortical defect'. • Benign features: No periosteal reaction, No Soft tissue swelling. • Well-defined, sclerotic margin. X ray description: (bottom image) - X ray of a child, growth palate is seen. - Cloud shaped, defect in the metaphyseal area, geographic shape and fibrous material. - This x-ray shouldn't be worrisome and could be observed with no Tx.
Treatment	• **Self-limited (Reassurance).** • If large or fractured → curettage + bone graft.

Primary Benign Bone Tumors:

Benign Active Tumors

(1) Simple Cyst or Unicameral Bone Cyst (UBC)

- A common benign tumor, it is benign latent except if it ruptures.
- Usually seen in young patients.
- Less than 20 year olds.

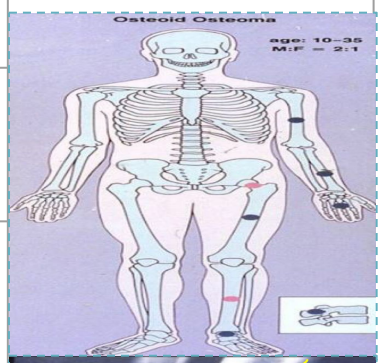


Site	• Pelvis, calcaneus, scapula and around the knee • Proximal part of long bones (e.g. proximal humerus, femur...)
Presentation	• Most commonly as incidental finding (asymptomatic) or pathological fracture.
Radiographic features	• **Metaphysic lytic lesion** (it contains fluid like suntop juice) • Well defined sharp border • No periosteal reaction • Fallen leaf sign indicates intra-cystic fracture (pathognomonic)
Treatment	• Observation unless there's a stress fracture • Often supportive, lesions will regress following skeletal maturity • Curettage and bone grafting may be required in areas at risk of fracture

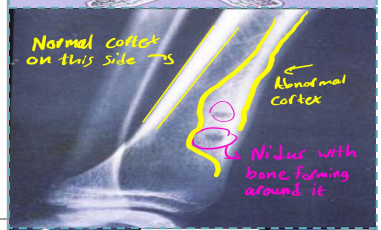
★ Fallen Leaf Sign

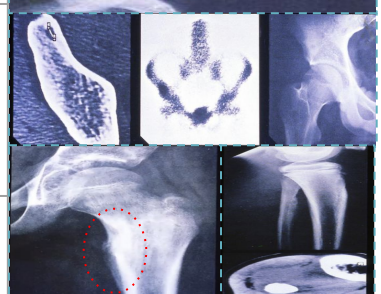
(2) Osteoid Osteoma (*night pain that improves with NSAIDs* احفظوها بيجي عنها سؤال)

- Bone-forming tumor



Site	• Found in the diaphysis and neck of the femur, tibia, humerus and posterior element of the spine • May arise in the cortex of long bones and rarely in the talus
Presentation	• Main presentation with **pain.** • Well localized pain (in the back or groin area) • Worse at night and prevents patient from sleep. ➔ Because of prostaglandin release. • Responds well to NSAIDs, leading to complete resolution • Painful scoliosis if affecting the spine. While idiopathic scoliosis is painless. • Nothing on physical exam.
Radiographic features	• Lytic lesion (central nidus), surrounded by a reactive zone of dense sclerotic new bone formation • Fine cut CT scan → nidus (modality of choice) • Cortical thickening on X ray
Treatment	• NSAIDs challenge (aspirin) • **Radiofrequency ablation**

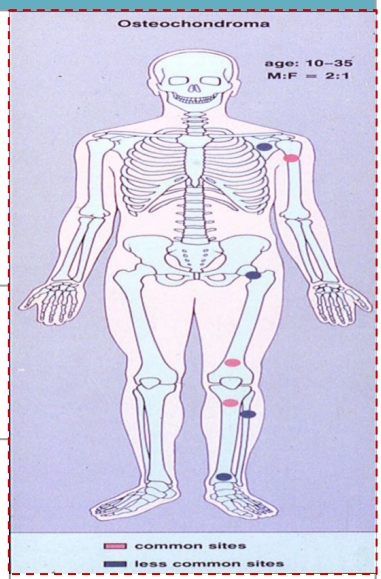




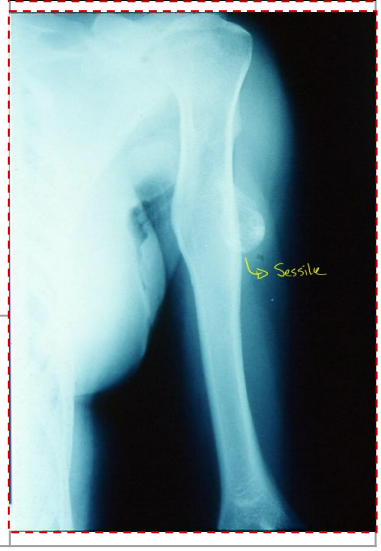
Primary Benign Bone Tumors:

★ (3) Osteochondroma

- Exostosis
- The only surface tumor (outside the bone)
- It is a combination of both bone & cartilage.
- Starts from the growth plate and Stops growing when patient stops to grow usually at 18 yrs
- > 2 cm masses have a risk of malignant transformation



Site	• The commonest are around the knee (**distal femur** & proximal tibia) • Proximal humerus, scapula and neck of femur
Presentation	• Patients usually present for cosmetic purposes or pain • Painless progressive swelling • Painful in children due to growth plate compression and pressure effects • On adjacent nerve or vascular structures (obese patients won't notice it) Complications: 1. Pressure symptoms: - Pseudoaneurysm → artery - Hypoesthesia/ paresthesia → nerve - Limited ROM → tendon - Formation of an overlying bursa due to friction 2. Fractures especially in the pedunculated type
Radiographic features	• Exostosis (fungated): mushroom-like stalk of the bony tumor (connected to the bone) • Benign features. • Metaphyseal lesion. • MRI: cartilaginous cap • **A distinctive feature is the continuity of the medullary canal content with tumor** • According to the shape of the neck, we divide it into: A. **Pedunculated type** (more common): long & thin neck. Directed away from the bone. B. **Sessile type**: has short & thick neck.
Treatment	• We usually resect the lesion even if it's benign • Surgery is indicated for: **(NEVER for cosmetic reasons)** 1. If the lesion is obstructing a nerve, vessel or tendon. 2. If the lesion limits movement. 3. If the lesion is quickly increasing in size

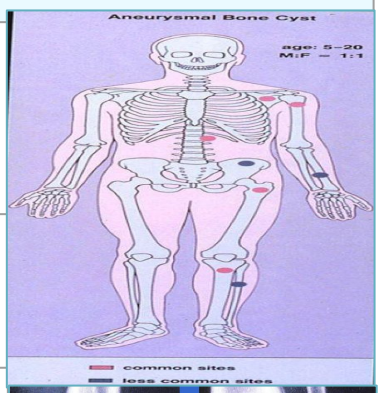


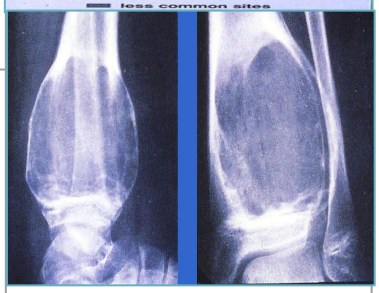
Primary Benign Bone Tumors:

Benign Aggressive Tumors

(1) Aneurysmal Bone Cyst (ABC)

- They can recur and may transfer to osteosarcoma
- Blood-filled cystic spaces, bigger than simple cysts
- Simple cyst is not wider than the growth plate and has fallen leaf sign, aneurysm bone cyst is wider than growth plate





Site	• Around joints: upper humerus, femur, and tibia • Spine (neural arch: lamina and pedicle) • Flat bones: scapula and pelvis
Presentation	• Asymptomatic
Radiographic features	• **Balloon like lesion** no periosteal and soft tissue swelling • Metaphyseal, septated lytic lesion • Well defined and sclerotic border • Expansile with thinned cortex (egg-shell) ⟶ • MRI shows fluid fluid level

(2) Giant Cell Tumor (GCT) very high recurrence rate (18%)

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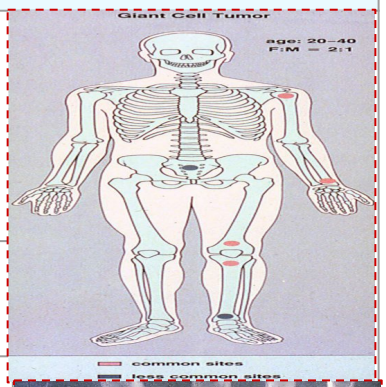
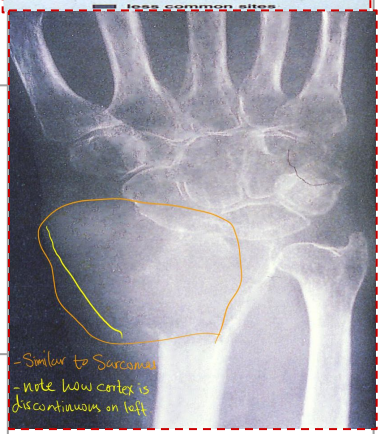
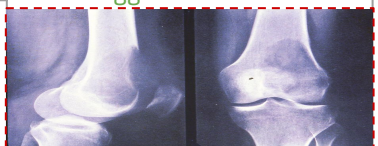
- Benign but very aggressive tumor (most aggressive benign one)
- The only benign that can metastasize (to the lung ⟶ get chest x-ray)
- GCTs can occasionally be seen with Paget's disease of bone and can arise in association with focal dermal hypoplasia (Goltz syndrome).
- In middle age group, In 20-40 years





AP x-ray with a lesion on metaphysis or epiphysis w soft tissue involvement, cortical destruction
Either aggressive b. or mal.



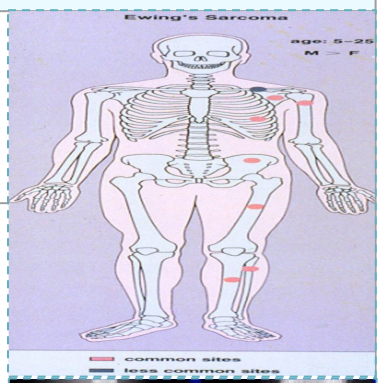
Site	• Proximal humerus, distal femur, proximal tibia. • Most common: distal radial Metaphyseal-juxta articular.
Presentation	• Presents with: pain, swelling & later pathological fracture
Radiographic features	• **Aggressive features:** - Permeative destruction - Ill defined borders - Huge soft tissue component • No new bone formation because this tumor is made up of osteoclasts (little or no periosteal reaction)
Investigation	• Soft tissue extension ⟶ MRI • Joint extension: the articular surface prevents extension • Bone extension ⟶ CT scan • Bone scan ⟶ metastasis • **Biopsy** ⟶ rule out malignant transformation
Treatment	• No place for conservative treatment. • Excision (Curettage) followed by either bone graft or prosthesis depending on the site of the tumor.

Primary Malignant Bone Tumors:

Malignant Tumors

(1) Ewing Sarcoma

- **One** of the most common tumors in **children (3-5y)**
- Most Ewing's sarcomas are misdiagnosed as acute osteomyelitis because of systemic symptoms, elevated ESR, elevated CRP, high WBCs. Even with biopsy some **pus** comes out



Site	• Around the growth plate → femur (most common), tibia, and humerus. • It is the only bone tumor which takes origin from **diaphysis,** Iliac flat bone, and ribs.
Presentation	• Pain • Huge Swelling • Lump with constitutional symptoms
Radiographic features	• Onion-skin periosteal reaction. • Very significant soft tissue component (characteristic of Ewing sarcoma)
Investigation	• MRI and biopsy make definitive diagnosis • N.B. you can't differentiate from osteosarcoma unless in biopsy. But hints are age group, radiological location (diaphysis in Ewing and metaphysis in osteosarcoma)
Treatment	For both Ewing and osteosarcoma: • Neoadjuvant chemotherapy followed by tumor excision and reconstruction. Why do we give neoadjuvant chemotherapy prior to surgical resection? 1. **To shrink the tumor for easier removal** 2. **To kill micrometastasis** 3. **Hints on post surgical chemotherapeutic agent**

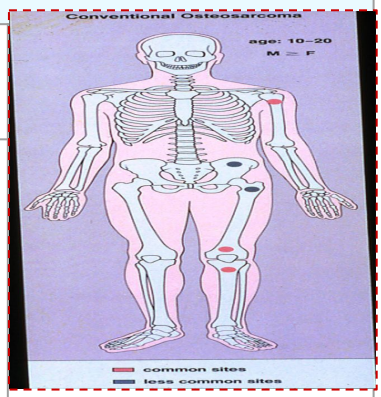
Primary Malignant Bone Tumors:

Malignant Tumors

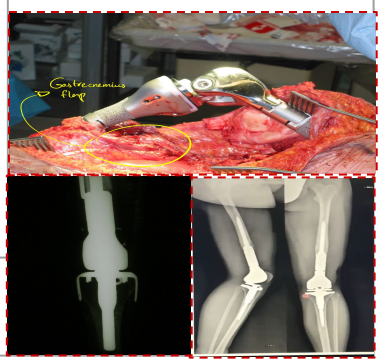
(2) Osteosarcoma

- More common than Ewing's sarcoma. **(age 10-20)**
- **History of radiation and paget's disease.**

Types	1. Primary (conventional, low-grade central, telangiectatic, multicentric (multifocal), juxtacortical; no need to know types of primary sarcoma) 2. Secondary (malignant transformation of benign process): A. Paget's disease → Paget's sarcoma **B. Post radiation sarcoma** 3. Metastatic from breast, lungs or bones.
Site	• Around the growth plate → around the shoulder, knee. • Distal femur, proximal tibia/Humerus
Presentation	• Pain, pathological fracture (typical presentation of malignant tumor) • Swelling (palpable mass in the site of the tumor "metaphysis commonly" but not as huge as Ewing's) • There might be prominent veins overlying the skin over the swelling • Overlying skin is warm due to high vascularity • The patient may look cachectic (in late and advanced cases)
Radiographic features	• Codman triangle • Irregular medullary and cortical destruction of metaphysis • Cortex is intact
Investigation	• CT scan → bone extension, lung metastasis. • MRI → soft tissue extension, neurovascular bundle involvement • Bone scan → metastasis to other bones *characteristic feature*. • Fever, elevated alkaline phosphatase (ALP) and lactate dehydrogenase (LDH) • Definite diagnosis made by **biopsy**
Treatment	• Similar to Ewing's Sarcoma

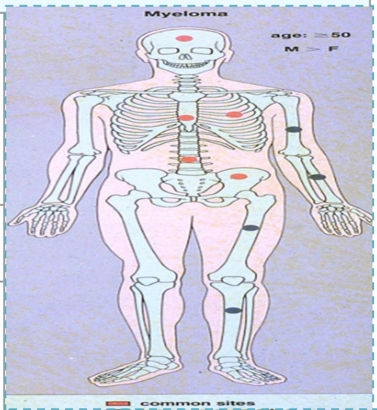


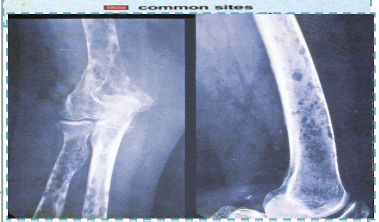
Bone producing, cortex is intact

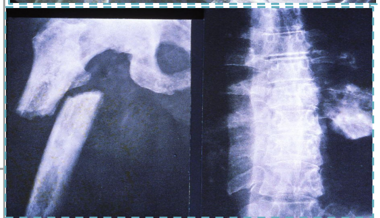


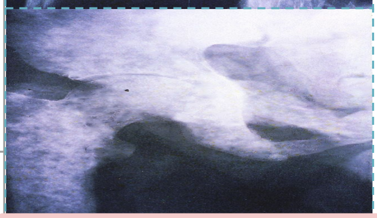
Primary Malignant Bone Tumors:

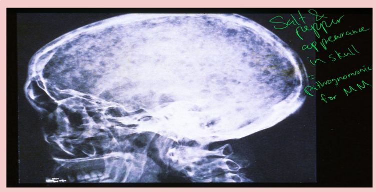
Malignant Tumors	
(3) Multiple Myeloma	
• Systemic disease. • Arise from plasma cells in the bone marrow. • The most common primary malignant tumor in elderly > 50. • Some consider it a bone tumor, and some consider it hematological	
Site	• Central bones (axial skeleton): skull, ribs, pelvic girdle. & spine
Presentation	• Pain, spinal cord compression, Osteoporosis, Pathological fractures. • Medical complications include anaemia, hypercalcaemia, hyperviscosity, • Immunosuppression and renal dysfunction. • CRAB: C = Calcium (elevated), R = Renal failure, A = Anemia, B = Bone lesions (bone pain)
Radiographic features	• Radiological features: Multiple Lytic or sclerotic lesions. "Moth-eaten appearance" • In skull, there will be pepper (lytic) & salt (sclerotic) appearance (pepper-pot). You have to do skull x-ray.
Investigation	• Bence jones proteins test found in 24h urine collection (highly suggestive)(protein electrophoresis) (urine & serum electrophoresis) UPEP and SPEP • Only definitive diagnosis is by bone marrow aspiration
Treatment	• Radiotherapy, Chemotherapy (mainly medical) • Bone marrow transplant: Success rate is 30% and it costs millions • The role of orthopedic surgeon is only fix pathological fractures, do internal fixation.









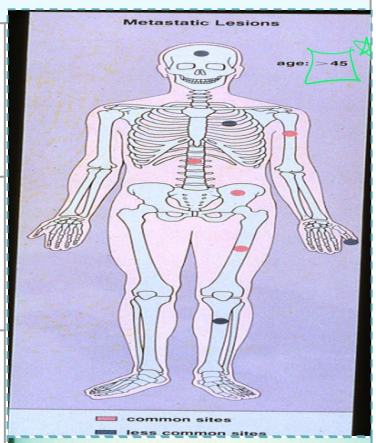


Secondary Metastatic Bone Tumors:

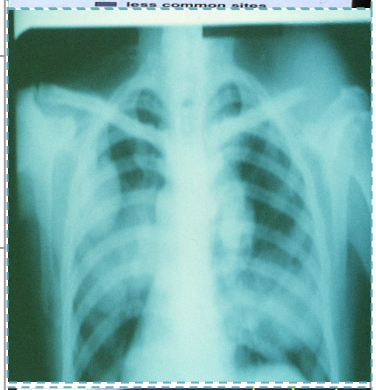
Malignant Tumors

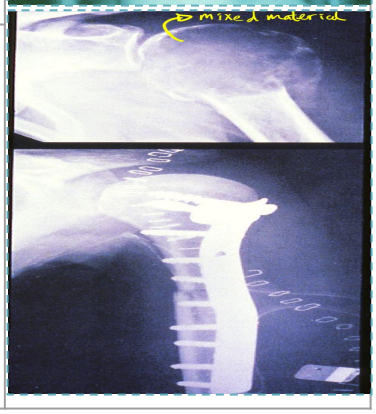
Metastasis

- More common than all primary tumors. Common in adults (>45 y)
- The most common tumors are: thyroid, lung, breast, colorectal, prostate and kidney



Site	• Usually metastasis occurs in the **highly vascular bones** e.g. vertebral body, hip, ribs, pelvis, upper end of femur, and humerus
Presentation	• Patient may present with known primary tumor. • May present with secondary metastasis. So, we must identify the primary sit and treat it
Radiographic features	• X-ray shows lytic lesions (bone eaten away) with thinning of the cortex. • Resembles bone cysts but the **age group** directs the diagnosis
Investigation	• CT scan of the chest. • Take biopsy to know where is the primary. • You can do bone scan to check metastasis in other areas
Treatment	• Palliative chemotherapy (not surgical). • The new trend is to downgrade the tumor then allograft it Surgery indications: ❖ Severe pain ❖ Mechanical instability: pain in the spine with neurological symptoms (spinal cord compression) ❖ Prophylactic (preventive fixation): in weight bearing bones with involvement of >60% of the cortex ❖ Fixate a fracture (curettage, grafts and plates)



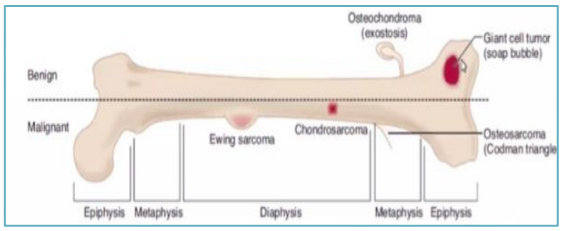


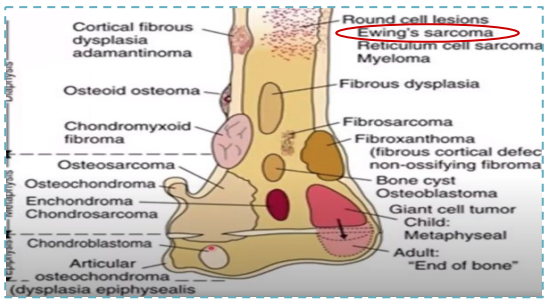
Important locations

Tumors usually have a common site (not necessary)

Common sites of tumor in bones:

- **Chondroblastoma**: epiphysis
- **Giant cell tumo**r: cross metaphysis and epiphysis
- **Osteosarcoma**: cross **metaphysis** and epiphysis
- **Ewing sarcoma**: **diaphysis**
- **Osteoid osteoma**: cortex





What's Next? Biopsy.

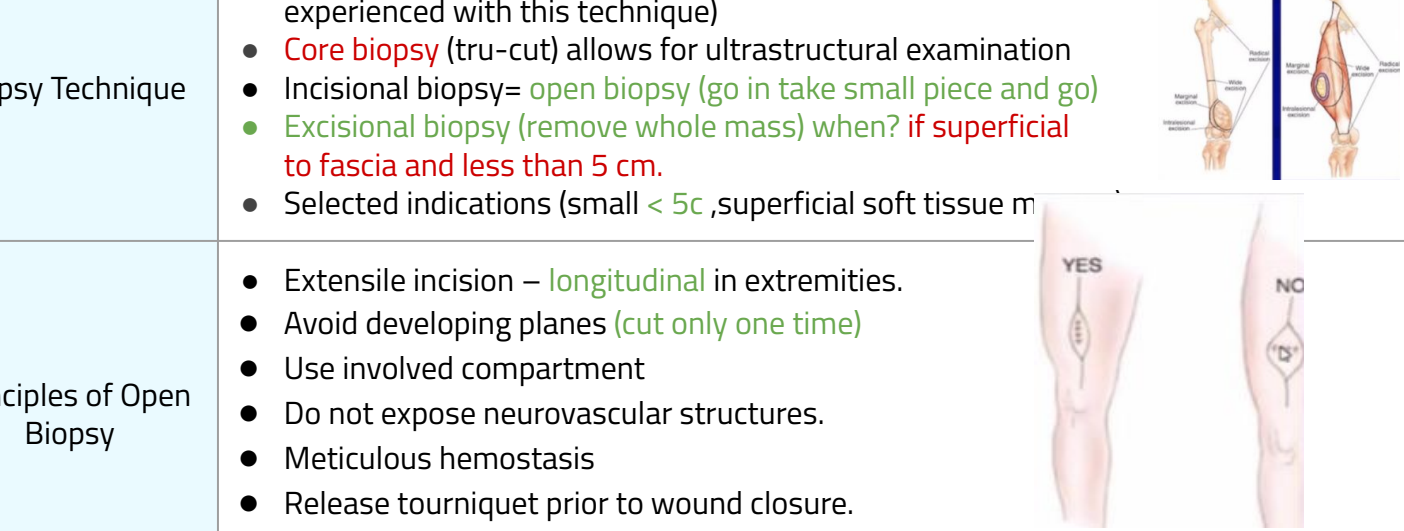
Biopsies

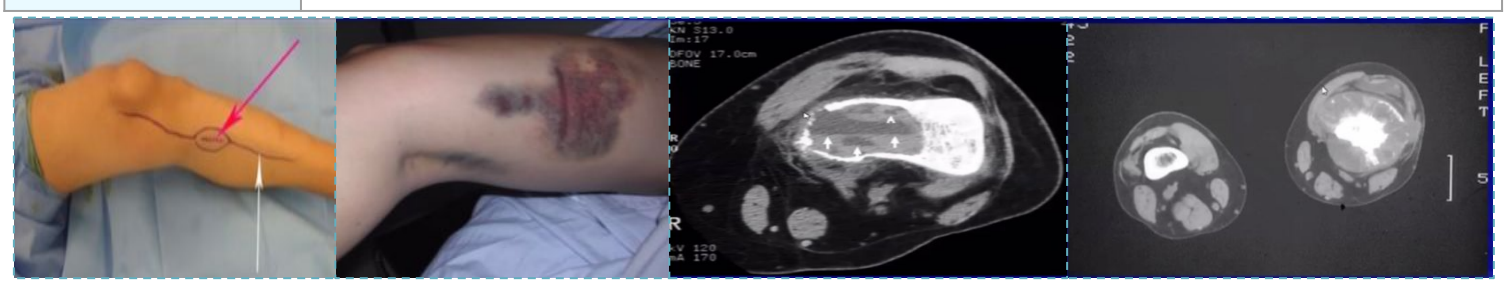
- Is not a substitute for thorough history, physical examination and investigations.
- Serves to confirm diagnosis suspected from above
- If you don't know what it is before the biopsy you won't know what it is after.

Not every lesion needs a biopsy!

- An asymptomatic (latent) or symptomatic bone lesion (active) that appears entirely benign on imaging doesn't need a biopsy
- A soft tissue lesion that appears entirely benign on MRI (lipoma, hemangioma) does not need a biopsy
- When in Doubt, it is safer to do a biopsy

Indications for Biopsy **V. Important**	• Aggressive or malignant appearing bone or soft tissue lesions. • For soft tissue lesions > 5 cm, deep to fascia, bone or neurovascular structures. • Unclear diagnosis in symptomatic patient • Special situation - solitary bone lesion in a patient with a history of carcinoma
Prerequisites for Biopsy	• CBC, platelets, coagulation screen • Cross sectional imaging- depicts local anatomy , solid areas of tumor • Experienced musculoskeletal pathologist is available.
Biopsy Technique	• Fine needle aspirate - gives cytologic specimen (adequate for some pathologists experienced with this technique) • Core biopsy (tru-cut) allows for ultrastructural examination • Incisional biopsy= open biopsy (go in take small piece and go) • Excisional biopsy (remove whole mass) when? if superficial to fascia and less than 5 cm. • Selected indications (small < 5c ,superficial soft tissue m
Principles of Open Biopsy	• Extensile incision – longitudinal in extremities. • Avoid developing planes (cut only one time) • Use involved compartment • Do not expose neurovascular structures. • Meticulous hemostasis • Release tourniquet prior to wound closure. • If using drain, bring out in line with incision.
General Recommendations Not important for you	• For benign aggressive tumours without soft tissue mass, plan biopsy through area of maximal cortical weakening based on CT or MRI. • For malignant tumours or benign aggressive with soft tissue mass, biopsy soft tissue rather than creating hole in bone.





Quiz

Q1: A 13-year old boy comes to the clinic because of a 6-week history of pain in his right shoulder. His pain is aggravated by the recent start of football practice and associated increased activity, but it persists after practice as well. He has no prior history of trauma. He denies fevers, weight loss and night sweats. His temperature is 37.1°C, pulse is 70/min, respirations are 16/min, and blood pressure is 120/82 mm Hg. Physical examination shows a hard immobile mass on the right proximal humerus which is slightly tender. His range of motion in the shoulder is minimally restricted. A radiograph obtained of his right upper extremity shows a sunburst pattern. Which of the following is the most likely diagnosis?

- **A** Ewing Sarcoma
- **B** Osteosarcoma
- **C** Giant cell tumor
- **D** Metastasis

Q2: A 15-year-old comes to his pediatrician's office because of pain in his right humerus for the past 3 months. He also reports a recent low grade fever. Further examination reveals midshaft swelling on his right humerus. The patient denies any recent history of trauma. Genetic analysis shows an 11;22 translocation. An x-ray of his right arm is taken. Which of the following answer choices will most likely show up on imaging for this patient?

- **A** Onion-skinned appearance
- **B** Punched-out lytic lesion
- **C** Necrosis surrounded by sclerotic bone
- **D** Central nidus, hyperactive cortex

Q3: A 10-year-old boy is brought to his pediatrician's office by his parents because of worsening pain over his left lower leg for the past 2 months. He denies any recent trauma to his leg within the past year and has been generally healthy. Physical examination shows a discrete, hard, and exquisitely tender mass over the anterior aspect of the right tibia about 3 inches below the knee. ESR today is 20 mm/hr. An X-ray of this patient's left leg is shown . What is the most likely diagnosis?

- **A** Osteosarcoma
- **B** Ewing Sarcoma
- **C** Giant cell tumor
- **D** Osteoid osteoma

Q4: Q4: A 17 year old with swelling and pain at the right knee for the last 6 months, lost 7 kg, no history of trauma or infection, blood work done and was normal besides high alkaline phosphatase. X-ray was done which showed highly suspicious tumor most likely to be an osteosarcoma. What is the confirmatory diagnosis test?

- **A** Knee MRI
- **B** Knee CT scan
- **C** Bone scan
- **D** Biopsy

1:B 2:C 3:A 4:D 5:C

Quiz

Q5: A 12-year-old boy comes to the clinic with his mother because of severe pain in his right leg for the past month. He says that the pain is mostly located in his thigh and that it was initially mild but now the pain is so bad that he cannot walk on his leg. His mother says that aspirin relieves his pain temporarily. Physical examination shows some localized pain over the area of the proximal femur. There is no palpable joint deformity, redness or swelling. He also walks with a visible limp. Neither increasing nor decreasing activity alleviates the pain, which tends to be worse at night. He has not had any recent trauma to the leg. An MRI of the leg shows a 1 cm lesion within the femur (shown below). Which of the following is the most likely explanation for this patient's symptoms?

- A: Aneurysmal bone cyst
- B: Non-ossifying fibroma
- C: Osteochondr-oma
- D: Osteoid osteoma

Q6: A 23-year-old man comes to the emergency department because of a 10-month history of gradual painful swelling of his wrist. His temperature is 37.9°C (100.2°F), pulse is 82/min, respirations are 16/min, and blood pressure is 120/77 mm Hg. An X-ray is obtained and is shown below. Which of the following is the most likely diagnosis?

- A: Osteoid osteoma
- B: Osteosarcoma
- C: Ewing sarcoma
- D: Giant cell tumor

Q7: Which of the following is the most aggressively benign tumor?

- A: Osteoid osteoma
- B: Endochondr-oma
- C: Giant cell tumor
- D: Simple bone cyst

SAQs

List three bone tumors that require bone marrow aspiration

1- Multiple Myeloma
2- Ewing Sarcoma
3- Lymphoma

What are 3 prerequisites to biopsy a highly suspicious bone tumor?

1- CBC, platelets, coagulation screen
2- Cross sectional imaging to depict local anatomy and solid areas of the tumor
3- Experienced musculoskeletal pathologist available

MCQ	
Q1	A
Q2	A
Q3	B
Q4	D
Q5	D
Q6	D
Q7	C

Team Leader

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Orthopedic 441 Team

MED441 KING SAUD UNIVERSITY

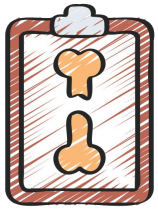
Editing File

Sport & Soft Tissue Injuries

Prof. Abdulaziz Alomar

Color Index:

- Main Text
- Important
- 441 Notes
- Old Notes
- Extra



Objectives

Specify the symptoms, signs and potential immediate complications of common sport and soft tissues injuries involving muscles, tendons, and ligaments for commonly injured joints; like shoulder, knee, and ankle.

Outline the assessment and appropriate investigation and to outline the immediate and long term management of patients with muscles, tendons, ligaments and meniscus.

Demonstrate knowledge of indications for non-operative and operative treatment and to know the most common non-operative and operative measurements used for sport/soft tissue injuries.

Resources

Introduction

Soft Tissue Injuries Include:

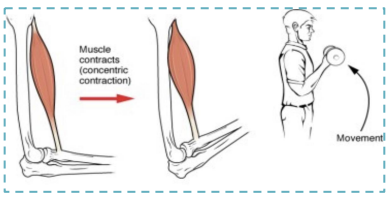
- **Muscle**
  - Muscle Strain
  - Muscle Contusion
  - Muscle Laceration
  - Delayed Onset Soreness
- **Tendon**
  - Overused tendinopathies
  - Tendon Rupture
- **Knee**
  - Ligament
    - AC
    - PC
    - MC
    - LC
  - Dislocation
  - Meniscus
- **Shoulder**
  - Discussed in separate Lecture
- **Ankle**
  - Ankle Sprain

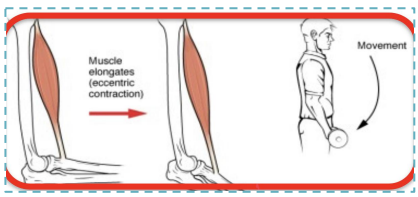
Initial Management

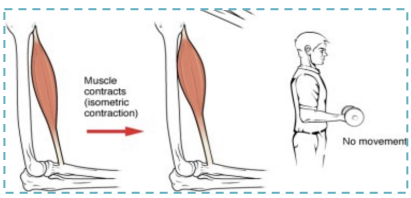
- **In acute phase** (first 2-3 days):
  - **PRICE** protocol, Analgesia, Muscle contusion: immoblize in maximum muscle length position
- **Early mobilization, Physiotherapy, Stope sports until full recovery Duration: based on injury severity**
- **Prevention of recurrent injury protocol.**

Protect	the injury (stop using the injured limb, pad to protect)
Rest	the injury It can help in detecting the real side of injury , controlling the damage prevent further damage
ICE	apply a wrapped ice pack It can help in Pain and swelling relief and prevent further damage. It's golden period is : first 72 hours , after that it's useless
Comfortable	**support -** apply a supportive bandage It can help in swelling relief , controlling the damage and prevent further damage.
Elevation	to reduce swelling

Types of Muscle Contraction







Muscle Injuries

- The muscles most at risk are those in which the origin and the insertion **cross two joints**
- Frequently injured muscles act in an eccentric fashion (i.e., lengthening as they contract).
- Muscle injuries are the most frequent cause of physical incapacity in sports practice
- It has been estimated that 30 to 50% of all sports-related injuries are caused by soft-tissue lesions

3 types of muscle contractions: contraction (shrink) , **elongation "eccentric" MOST INJURED**, isometric

Muscle Strain[1]

Characteristics

- The most **common** muscle injury suffered in sports. due to noncontact injury
  - How does it happen? overuse, or improper use of a muscle result in → muscle overstretched (muscle strain) → could lead to muscle tear.
  - (39 slide) Both complete and incomplete muscle tears can occur by passive stretch of an activated muscle. (Trying to stretch a contracted muscle by force)
- Caused by non-contact (indirect) trauma.
  - Excessive tensile force (eccentric load) subjected onto the muscle leads to the overstraining of the myofibers
- Muscle tears also typically occur at or near to the myotendinous junction.
  - It can be complete or partial tears
- Clinical features:
  - Immediate pain associated with diminished function.
  - Localize tenderness
  - -/+ swelling
  - Pain/weakness with resisted muscle contraction

Muscle strain can happen in such a football player had sudden pain, the difference from cramps is the cramps lasts mere seconds only.

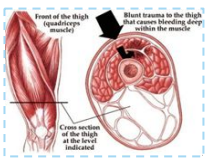
Treatment (439 slides)

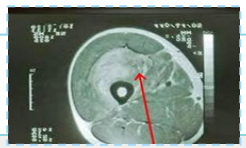
- PRICE, NSAIDs, Physical Therapy

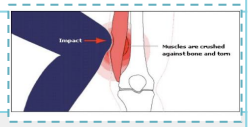
Muscle Contusion

Characteristics

- Caused by a non-penetrating blunt injury (direct blow) to the muscle resulting in hematoma and inflammation.
- Quadriceps and Brachialis muscles are **common involved regions**
- Clinical features:
  - Pain with active and passive motion +/- swelling.
  - Decreased range of motion of joints spanned by the injured muscles.
  - Occasionally a permanent palpable mass.







Treatment (439 slides)

- **Short** period of immobilization
- Followed by **early** mobilization and Physiotherapy
- NSAID

Muscle Laceration (439 slides)

Mechanism

- Caused by a direct injury to the muscle by a sharp object.

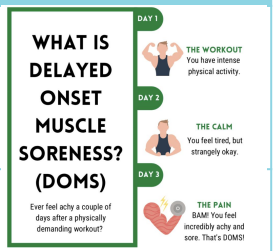
Treatment

- I&D (irrigation & debridement) followed by suture repair of the fascia, if possible.

Delayed Onset Soreness (DOMS) (Muscle fever)

Characteristics

- Structural muscle injury leads to progressive edema formation and resultant increased intramuscular pressure.
- Is primarily associated with eccentric loading type exercise.
- **Clinical features**: muscular pain that occurs **1-3 days after** vigorous exercise[2].



Treatment

- It's self-limited and **will resolve in a few days**
- NSAIDs

1- Strain is used instead of "tear".
2- Like going to the gym for the first time.
*muscle injury treatment: mainly conservative "RICE", analgesia, immobilization (fully lengthened position; quads injured ->in full knee flexion)

Complications of muscle injuries

Scar formation and muscle weakness

- Mainly due to laceration.
  - How the scar formed inside the muscle? The space between ruptured muscle fibers fills with blood which clots and gradually converted into connective tissue, which converted into scar tissue.
  - This leaves the muscle with areas of varying elasticity.
  - In some cases, this scar tissue may need surgical excision. Scars will lead to muscle weakness

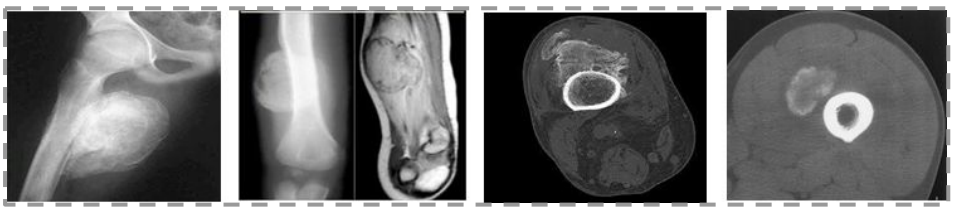
Compartment syndrome

- At the level of the muscle fibers, capillary bleeding and edema can lead to hematoma formation and can cause compartment syndrome in areas in which the volume is limited by the fascial envelope.
- Patients with Bleeding disorders is at high risk

(Example: Hemophilic patient that suffers muscle contusion)

Myositis Ossificans (AKA heterotopic calcification)

- **What is it?** Bone formation (calcification) within muscle secondary to blunt trauma (Muscle contusions).
- Clinical features :
  - Early:
    - Pain, swelling and decreased ROM
    - Erythema, warmth, induration, tenderness
  - Late:
    - Painless swelling with decreased ROM.
- This sometimes mimics osteogenic sarcoma on radiographs and biopsy. Which is why a good history is essential, as many patients forget to mention that they suffered some sort of trauma earlier.
- Increased ESR and serum alkaline phosphatase
- Myositis ossificans becomes apparent approximately 2 to 4 weeks post-injury.
- ~~Does not appear in x-ray until 2-4 weeks later~~
- Management is conservative unless if it is huge then we should excise it Orthobullets: if it remains a problem+do not operate in acute phase, wait at least six months



(39 slides pics)

others

- **Infection:**
  - Pyomyositis (*S. aureus)*
- **Rhabdomyolysis** (skeletal muscle necrosis)
  - Crush injury , myoglobin in circulation , renal impairment
- **Recurrent injury**

Tendon Injuries

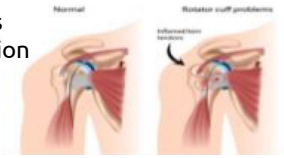
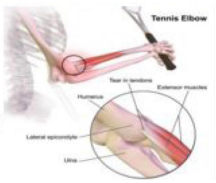
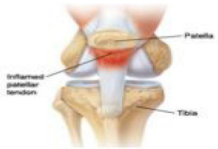
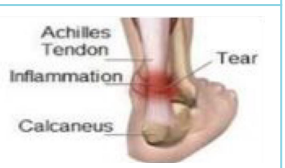
- What are the functions of tendon? To transfer force from muscle to bone to produce joint motion.
- Type of injuries:
  - **Overuse tendinopathies.**
  - **Tendon rupture**. It's a traumatic tear usually .

Overuse Tendinopathies

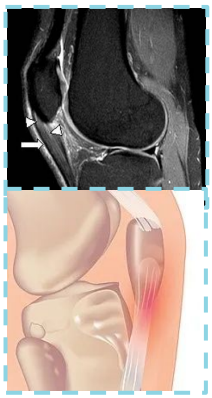
Common in athletics

- Result of repeated **mechanical** loading (overuse).
- **Osteotendinous junction** is the most common site of overuse tendon injury. Why? Tendons are relatively hypovascular proximal to the tendon insertion. This hypo-vascularity may predispose the tendon to **hypoxic tendon degeneration** and has been implicated in the etiology of tendinopathies.
- Tendinopathy NOT tendonitis.

Most Common Diagnoses and Locations of Chronic Tendinopathies

Diagnosis	Symptoms	Location
Rotator cuff Tendinopathy is when a tendon in your shoulder has tiny tears in it or is inflamed and hurts	• Pain and swelling in the front of your shoulder • Pain triggered by raising or lowering your arm • A clicking sound when raising your arm • Stiffness	Supraspinatus tendon insertion 
Lateral epicondylosis (tennis elbow) (because of the **overuse of arm extensor** will lead to tear in tendon)	• Tenderness on the outside of the elbow. • Morning stiffness of the elbow with persistent aching • Soreness of the forearm muscles • Elbow pain is worse when grasping or holding an object	Common wrist extensor tendon origin mainly involved extensor carpi radialis brevis (ECRB) 
Medial epicondylosis (golfer's elbow) **(Baseball elbow)**	• Pain when **flexing the wrist** toward the forearm • Pain that extends from the inside of the elbow through the wrist to the pinky • A weak grip • Pain when shaking hands	Common wrist flexor tendon origin 
Hamstring Tendinopathy	• Pain in or close to the knee joint that radiates up the thigh and possibly into the hip or pelvis & gets with activity , especially repetitive motions . • Swelling in or around the knee or thigh	Hamstring tendon origin 
Quadriceps Tendinopathy	• Swelling around the quad tendon • Sensitivity to touch • Warmth or burning pain in the affected area . • Stiffness in the knee in the early morning	Quadriceps tendon insertion 
Patellar Tendinopathy (jumper's knee)	• **Pain around your patellar tendon .** • Swelling • Pain with jumping , running , walking bending or straightening your leg • Tenderness behind the lower part of your kneecap	Patellar tendon origin 
De Quarvain's disease	• Pain & swelling near the base of your thumb . • Difficulty moving your thumb and wrist when you're doing something that involves grasping or pinching . • A "sticking" sensation in your thumb when moving it .	Sheath/pulley of **abd**uctor pollicis longus 
Achilles Tendinopathy (runners)	• Increasing pain , usually at the back of your leg or heel . • Stiffness in the Tendon • Swelling & tenderness at the back of your ankle • Crepitus when you move your ankle	Sheath, midsubstance, or calcaneal insertion 

Overuse Tendon Injuries



Patellar Tendinopathy (jumper's knee)

Overuse Tendinopathies Treatment

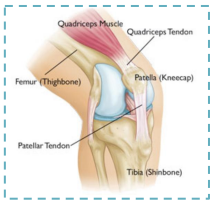
- **Goal: reduce pain and return function**
- **Mainly is Conservative**
  - **Rest**
  - **Physiotherapy** (stretching and eccentric strengthening)
  - **Corticosteroids injections**
    - Injected in the sheath(around the tendon), not the tendon itself to prevent weakness and tendon rupture
  - **Other modalities:**
    - U/S
    - ESWT (extracorporeal shockwave therapy)
    - **iontophoresis**
      - Introduction into the tissues, by means of an electric current, of the ions of a chosen medicament.
    - **phonophoresis**
      - The use of ultrasound to introduce medication into a tissue.
  - Ice (Cryotherapy)
  - Analgesics
  - Orthotics and braces

- **Surgical treatment**:
  - very rarely
  - Failed conservative treatment (at least 3-6 months)
  - Excision of abnormal tendon tissue and performance of longitudinal tenotomies to release areas of scarring and fibrosis.

Tendon Rupture

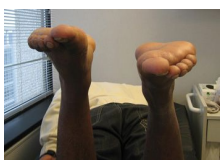
- Knee extensor mechanism: Quadriceps tendon, and Patellar tendon
- Achilles tendon (more common than patellar tendon)
- Partial vs complete

Patellar/Quadriceps tendon rupture

Predisposing factors	**Steroid use**, Chronic disease, Tendinopathy
Age	• Patellar < 40 usually in young • Quadriceps > 40 That's why if you examined randomly people with patellar pain you'll find - >40 Pain above Patella - <40 Pain below Patella 
Location	At the tendon attachment to the patella
Physical Examination	• Tenderness at the site of the injury , hematoma , and a palpable defect (gap) in the tendon • Unable to extend the knee against resistance or to perform a straight -leg raise . can do passive movement only **How to differentiate between them clinically ?** The most significant sign is extension lack. Patient is unable to do active extension and if you can do it passively it can be fully extended.
X-ray	How to differentiate between them in X-ray ? **Best diagnostic is: examination + MRI** • Patella-alta > Patellar tendon rupture (Patella goes up) • Patella-infera(Baja) > Quadriceps tendon rupture (Patella goes down) You don't need MRI for diagnosis, but you may use it to exclude other injuries or to determine how you will reconstruct in the surgical treatment . Sometimes the Patella is above its normal position which indicate patellar tendon rupture patella-alta. While in patella baja it will deviate below its normal position. 
Treatment IMPORTANT	Treatment in tendon repair is usually **surgical (primary repair).**

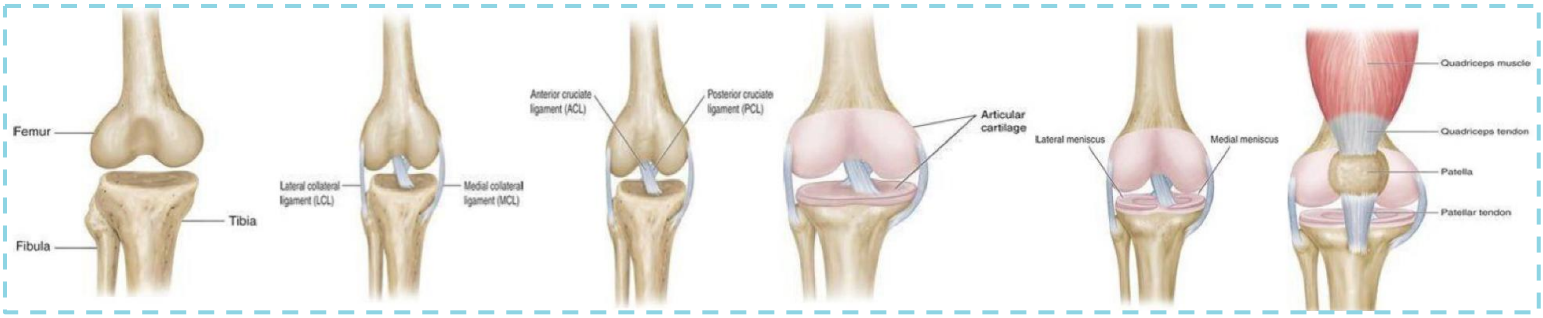
Common scenario: 20 y boy came to ER with inability to rise his right lower limb "knee extension" . What is your DDx? Knee ligament tear , fracture of patella , quadriceps or patellar tendon rupture, femoral nerve injury or psychology

Achilles Tendon Rupture

Occurrence	Most common rupture (75%) during sporting activities.
History	• The patient reports a "pop" or the sensation of being kicked in the heel during the injury. The stored energy will be released suddenly, creating what the patient perceives as a pop. • Weakness and difficulty walking
Physical Examination IMPORTANT	Increased resting dorsiflexion with the knees flexed, a palpable gap, **weak plantar flexion**, and an abnormal Thompson test (lack of plantar flexion when squeezing the calf). **One leg standing test.** 
Diagnosis	Diagnosis is clinical, but MRI or ultrasound can confirm
Treatment	**Usually surgical** (repair)(always) as we said before in tendon rupture the management is surgical only. Conservative treatment is not recommended as it usually leads to chronic weakness.

Knee Injuries

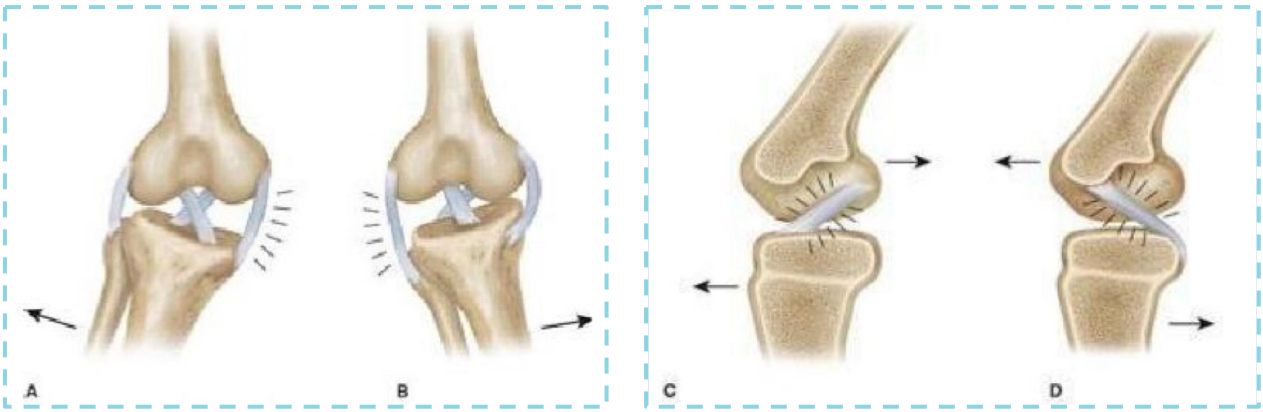
Knee Anatomy



- Joint stability: bone stability + soft tissue
- Dynamic Stabilizer: Tendon/Muscles
- Static Stabilizer: Ligaments ± meniscus

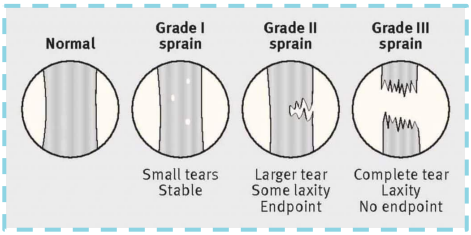
Complex Synergy leading to a Functional and Stable joint.

Functions of The Knee ligaments



- The medial collateral ligament (MCL) prevents valgus deformities.
- The lateral collateral ligament prevents varus deformities.
- The anterior cruciate ligament prevents anterior tibial translation over the femur.
- The posterior cruciate ligament prevents posterior tibial translation over the femur.

Types of Knee Injuries



1	Ligaments injuries (ACL, MCL, LCL, PCL)
2	Knee Dislocation (red flag)
3	Menisci

Knee Injuries

Ligaments injuries

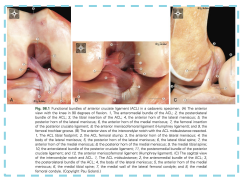
Common Signs & Symptoms:

- Some patients will offer that they felt, or even heard, a "pop" when the ligament was injured. Knee ligaments are very strong structures. They can store a tremendous amount of energy before failing. If the load is big enough to fail the ligament, then the ligament will rupture, and that stored energy is released suddenly, causing the tibia to swing back hitting the femur creating what the patient perceives as a pop.
- Many patients present a long time after injury with symptoms of instability. In these patients, the pain and swelling from the initial injury has been resolved, but, because the ligament did not heal, they are prone to intermittent episodes of instability.
- Ligaments are more vascular than meniscal tissue, and patients with ligament injuries tend to develop effusions within an hour of their injury. In patients with meniscus tears, effusions usually develop much more slowly.

Anterior Cruciate Ligament (ACL) injury إصابة الرباط الصليبي الأمامي

Anatomy and function

Function: Prevents anterior translation of the tibia relative to the femur
Anatomy:
- Extrasynovial but intracapsular
- Origin: Lateral femoral condyle
- Insertion Between the intercondylar eminences of the tibia



Mechanism of Injury

- About 70% of ACL injuries are caused **without contact,** by cutting or pivoting. Sport such as Basketball, football, Soccer. الرياضات التي تتطلب تغيير الاتجاه بشكل سريع
- **Contact** = associated with Medial collateral ligaments (**MCL**) injury.
- Sports-Related (80%)
  - 70% of patient will hear a "POP" sound. Very loud!
  - Female: 2-4x > Male. if both female and male are active, but in Saudi Arabia it is less than in males. **Why it's more common in females abroad? Because:**

1. Outside, women play sports more than men, while here men are 4 times more.
2. Their notch (the place where the ACL & PCL cross -between the 2 condyles) is narrower than the males, so any over activities makes the ACL more prone to injury.
3. The pre period hormones change so they develop laxity.
4. The way they have it: they always jump with valgus so there is stress on ACL.
5. Neuromuscular balance+strengthening (decreases risk of injury); more effective in females

Symptoms

In acute phase the patient will present with pain, swelling, instability but in chronic phase only instability and it's usually not because of ACL injury! but because of associated injuries like meniscus tear or cartilage damage.

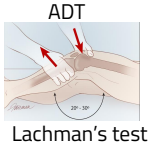
- Instability "giving way episodes" أو مو ثابتة "تخوني" ركبتي Later this is the only symptom left
- **Immediate Swelling (Hemarthrosis** دم في المفصل **)** is noted within 1-2 days of the injury. And a "pop" sound.
- Pain : (in acute stage):▪ Bone contusion ▪ Meniscus tear /MCL injury ▪ Chondral injury ▪ Severe effusion
- Pain if associated with Meniscus tear or cartilage damage. After acute injury we will have pain because of meniscus injury, or bone contusion so the tibia will sublux interiorly. In case of femur it subluxed in the middle.

Dx is done clinically. MRI r/o soft tissue injury, xray r/o fractures

Physical Examination

- The patient needs to be relaxed and comfortable.
- Must be compared with those of the normal knee.
- A moderate to severe effusion is usually present in the acute cases.
- **ROM:** in acute injury the range of motion may limited by Pain, Effusion, Hamstring spasm, ACL stump impingement (قطع الرباط الممزق تتكدس بالمفصل وتعيق حركته) , or Meniscal pathology.
- **Special tests:**
  1. Lachman's test. (at 20-30 degree) (the most sensitive test)
  2. Anterior Drawer test (ADT) (at 90 degree)
  3. Pivot shift test: is pathognomonic for ACL ry (best in Chronic settings).


ADT


Lachman's test


PST

Knee Injuries

Ligaments Injuries con.

Anterior Cruciate Ligament (ACL) injury إصابة الرباط الصليبي الأمامي

Investigations[1]

X-ray (Important)

Segond Fracture (avulsion fracture)

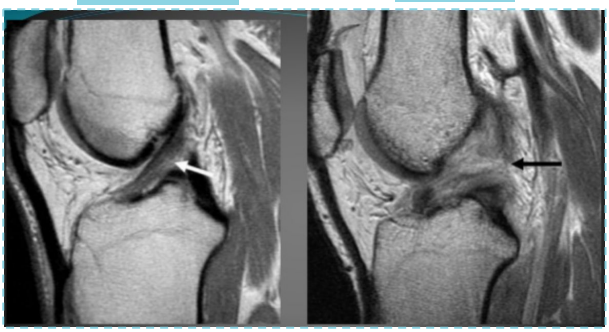
Pathognomonic for ACL injury. There is avulsion of anterolateral capsule attachment & its sign of ACL

Tibial spine avulsion

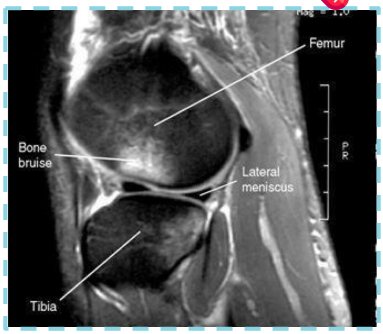
Here we see growth plate still opens = immature skeleton so this is a pediatric pt.. In pediatric the ligament is stronger than adult so there will be avulsion without piece of bone usually.

MRI

Normal ACL **Torn ACL**



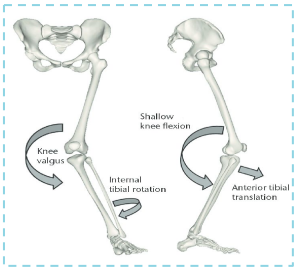
Bone Bruise[2] IMPORTANT

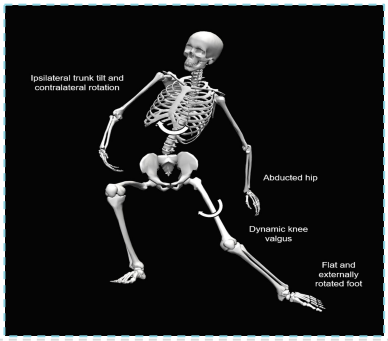


Injuries Associated with ACL disruption

- Injuries of the **ACL rarely occur in isolation**. The effects of other injuries, including:
  1. Other ligament sprains (MCL). Contact injury = MCL
  2. Meniscal tears = pain (40% -30%)
  3. Articular cartilage injuries (chondral injuries)
  4. Bone bruises causes pain and "pop" sound, there is subluxation of tibia over femur especially on the lateral aspect leading to impingement of the posterolateral part of the tibia against the middle part of the lateral femoral condyle.
- Complicate the treatment and eventual outcomes of ACL disruptions.

Mechanism for non-contact ACL injury





1- ACL is mainly diagnosed by H&E, you don't really need any investigations mostly.
2- The POP sound heard in ACL tear is caused by the impact between the tibial and femoral condyles, this contact may lead to bone contusions "Bruises", which can cause pain for some time.

Knee Injuries

Ligaments Injuries con.

Anterior Cruciate Ligament (ACL) injury إصابة الرباط الصليبي الأمامي

Treatment

WHEN ACL IS TORN IT DOES NOT HEAL! why not repair? the difference is in repair we repair the same ligament while in reconstruction we change it to new one, so we do reconstruction because the studied shows that the healing in repair is very poor.

1. **Nonsurgical treatment:**
   - Appropriate for asymptomatic patients with partial injuries to the ACL.
   - Patients who are older or less physically active may elect to modify their activities and proceed with nonsurgical treatment.
   - Nonsurgical treatment involves rehabilitation to strengthen hamstrings and quadriceps, as well as proprioceptive training.
   - Activity modification is also an important part of nonsurgical management, as patients who avoid cutting and pivoting sports are at lower risk for knee instability.
   - ACL sports braces have not been shown to prevent abnormal anterior tibial translation

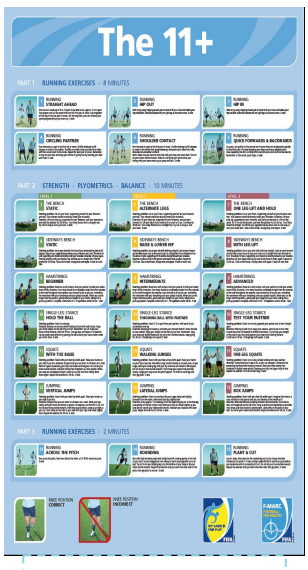
2. **Surgical Treatment indications:**
   - Athletes with ACL injuries rarely return to **cutting and pivoting sports** (e. basketball, football, soccer) without first undergoing surgery. Or walking on uneven ground
   - For individuals who wish to return to such sports, surgery is generally recommended to avoid instability and secondary meniscal and/or articular cartilage damage.
   - Individuals who work in occupations that may involve physical combat, such as police officers, or risk, such as firefighters, should have ACL reconstruction before returning to work.
   - Most patients can function well and perform activities of daily living (ADLs) without instability after a complete ACL injury. However, some have difficulty performing even simple ADLs because of ACL deficiency related instability, and they may require surgery.
   - Young patients.
   - **In summary**: Surgery needed if unstable during activities or someone who use his legs for living even if he didn't reach instability stage.

❖ **Summary of the treatment from the doctor** IMPORTANT

★ **Surgical (ACL reconstruction)** عملية بناء الرباط أو وضع بديل له:
1. **Young, athletic and active patient.**
2. **Middle age not having arthritis:**
   - If there is instability during daily activity.
   - If there is **NO** instability and the patient is active and want to play sports.

★ **Conservative: (exercise, and physiotherapy)**

★ **Isolated ACL injury, Old patient, no symptoms, not active**
1. **Middle age not having arthritis:**
   - If there is **NO** instability and the patient will not play sports.
2. **Old or osteoarthritis patient.**
   - To come to the point, patient who are old or not willing to participate in any kind of sports, or asymptomatic or no instability, treat them conservatively; this is only exception



ACL Prevention

In general surgical intervention for tendon injury is a primary repair and for ligaments reconstruction

Ligaments Injuries con.

Posterior Cruciate Ligament (PCL) injury إصابة الرباط الصليبي الخلفي

PCL Function	• The PCL is the primary restraint to posterior tibial translation in the intact knee

Mechanism of Injury (MOI)	• A direct blow to the proximal aspect of the tibia is the most common cause of PCL injury.
	• Dashboard injury:
	  ○ اللي يركب قدام بالسيارة لما يصير فيه تسارع بعده فرامل تروح ركبته تضرب بدرج السيارة فتدخل داخل خصوصا اللي جالس 90 درجة وينقطع الرباط
	  ○ "Direct blow to flexed knee"
	• **In athletes:** a fall onto the flexed knee with the foot in Plantar flexion, which places a posterior force on the tibia and leads to rupture of the PCL. or hyperflexion

PCL special test	Posterior sag sign   Posterior drawer test
Complications	• PCL insufficiency significantly increased the risk of developing medial femoral condyle and patellar cartilage degeneration over time.
Treatment	1. **Non operative:** They do healing without surgical intervention, it won't affect our performance. Mainly non-operative unless there is combined ligament injury. 2. **Surgical** if combined ligament injury , or symptomatic grade 3 rarely because it's hard to get there, it doesn't cause frank instability, if it does, we do surgery. if the extensive physiotherapy doesn't succeed we do surgery

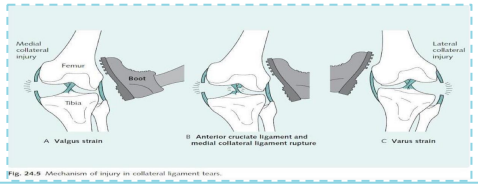
Medial Collateral Ligament (MCL) injury إصابة الرباط الجانبي الأنسي

Anatomy	• The main function of this complex is to resist valgus and external rotation loads. (stress)
Occurrence	• The tibial MCL is the Most coMMonly injured ligament of the knee. 40%
MOI IMPORTANT	• Usually result from **contact** injury like a direct blow to the lateral aspect of the knee Like what happened to THE GOAT in EURO final 
Associated Injuries	• Concomitant ligamentous injuries (95% are **ACL**) • Concurrent **meniscal injuries** have been noted in up to 5% of isolated medial ligamentous injuries

Physical Examination	• Valgus stress test should be performed with the knee at 0° and 30° of flexion:
	  ➜ **Laxity at 30° : isolated MCL**
	  ➜ **Laxity at both 0° and 30°: concurrent injury** to the
	• Posteromedial Capsule and/or cruciate ligament
	• Rule out associated injuries (ACL and Medial Meniscus) 

Investigations	• It Is a clinical diagnosis and most of the time does not need further investigation. • If the injury is severe or suspecting associated injuries (e.g. significant knee effusion) • then the MRI will be modality of choice. • **X Ray:** to rule out fracture (lateral tibial plateau fracture)
Treatment	• **Conservative Rx:** Is the mainstay of treatment for the isolated MCL injuries • Crutches, PRICE, and anti-inflammatory/pain medication • No brace is usually required for partial tear • A knee brace is recommended for complete tear. • **Surgical Rx:** Very rarely if failed conservative Rx + complete tear (Grade 3) + associated with other ligaments (Combined) injury. When I have ACL and MCL I will treat ACL surgical and MCL conservative.

Knee Injuries

Lateral Collateral Ligament (LCL) injury إصابة الرباط الجانبي الخارجي	
LCL Function	• The LCL is the primary restraint to varus stress at 5° and 25° of knee flexion.
Occurrence	• Less commonly injuries than MCL Usually comes with posterolateral corner injury which is way above your level
Mechanism of Injury	• **Varus strain:** Injuries to the lateral ligament of the knee most frequently result from injury motor vehicle accidents and athletic injuries. 
Treatment	• **Isolated injury:** non operative • **Combined injury:** surgical

Knee Dislocation

ACUTE EMERGENCY!

★ Multiligament knee injuries are usually caused by high-energy trauma and are often considered knee dislocations. A lot of cases come late to the ER after spontaneous reduction. At least 3 ligaments are injured: ACL, PCL, and one of The collateral ligaments, why? Because the dislocation is either laterally or medially.

★ Less frequently, low-energy trauma or ultra-low-velocity trauma in obese patients can also result in this injury pattern.

★ A bicruciate (ACL+PCL) injury or a multiligament knee injury involving three or more ligaments should be considered a spontaneously reduced knee dislocation.

★ A knee dislocation should be considered a limb-threatening injury, and careful monitoring of vascular status after the injury is imperative.

★ Popliteal artery (estimated at 32%) or peroneal nerve injury (20% to 40%) also can occur. Look for pulse and perfusion sign: color, temperature, capillary refill time (exam question)

★ Vascular examination is **critical** in an acutely dislocated knee:

- Pulse and ankle-brachial index (ABI) should be carefully assessed. An ABI of less than 0.90, and most certainly less than 0.80, should be considered abnormal.
- If there is any concern about an abnormal vascular examination, there should be a low threshold for ordering an angiogram.
- **MCQ**: pt have ABI less than 0,9 what is the next step? Angiogram
- If pulses are still abnormal or absent following reduction of the dislocation, immediate vascular surgery consultation with intraoperative exploration should be the next step in management.
- A vascular injury in a knee dislocation is a limb-threatening injury and needs to be corrected within 6 to 8 hours the golden period. If not corrected, amputation may be required.

★ Neurologic examination is also critical, as peroneal nerve injury can occur with multifilament injuries, particularly in concomitant lateral/posterolateral corner injuries.

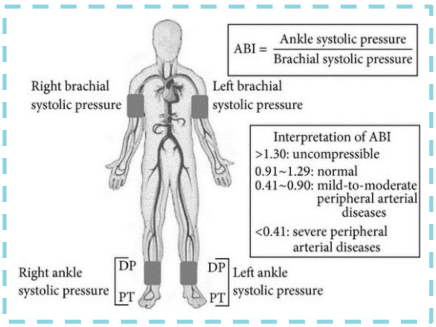
Knee Injuries

Knee Dislocation Management

Need Emergent Reduction

Multiligament knee injury requires v high energy trauma

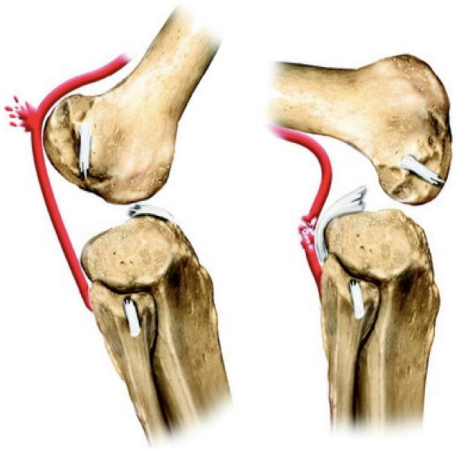
- Emergent closed reduction and splinting or bracing should be performed immediately. Post reduction radiographs should be taken to confirm knee reduction.
- What are the steps of reduction in ER?
  - Analgesia ➜ Reduce joint ➜ Immobilization ➜ Neurovascular assessment before reduction and after ➜ Vascular ABI ➜ X-ray ➜ Call OR



Extra pic but important

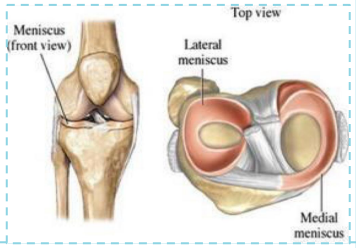
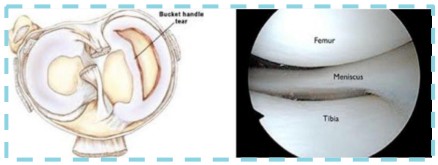
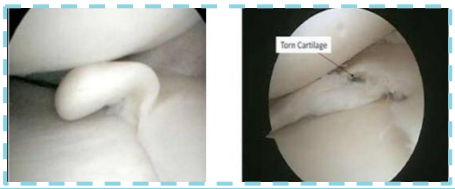
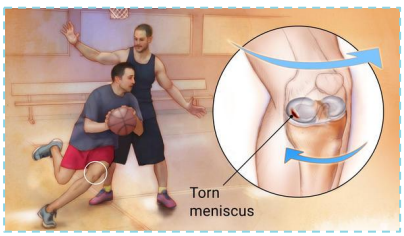
Acute knee Dislocation

1) Activate ATLS if high energy trauma or associated with other injuries
2) Analgesia++
3) Quick clinical/NV assessment
4) 2 view x-rays (if you can get it quickly!)
5) Urgent reduction (should not be delayed!)
6) Check stability and safety zone
7) Re-check neurovascular status after reduction (including ABI)
8) Examine the compartment to R/O CS
9) Immobilize the joint
10) Post reduction 2 view X-rays
11) Consult Orthopaedics



Knee Injuries

Menisci injury

Menisci Injuries تمزق الغضروف الهلالي	
Meniscus Anatomy	• The menisci are crescent-shaped, with a triangular appearance On cross-section. • The lateral meniscus covers 84% of the condyle surface; it is 12 to 13 mm wide and 3 to 5 mm thick. • The medial meniscus is wider in diameter than the lateral meniscus; it covers 64% of the condyle surface and is 10 mm wide and 3 to 5 mm thick. 
Meniscus Function	• The meniscus provides stability, absorbs shock, increases articular congruity, aids in lubrication, prevents synovial impingement, and limits extremes flexion/extension. • The **most important function** of the meniscus is **load-sharing** across the knee joint, which • it accomplishes by increasing contact area and decreasing contact stress[1].
Epidemiology of Meniscus injuries	• Meniscus injuries are among the most common injuries seen in orthopaedic practices. • Arthroscopic partial meniscectomy is one of the most common orthopaedic procedures Bucket-handle tear Normal Mechanical block Torn (rupture) Cartilage  
Incidence	• Meniscal tears are unusual in patients younger than age 10 years. • Most meniscus tears in **adolescents** and young adults occur with a twisting injury or with a change in direction. In young patients, the meniscus is tough and durable, and it is hard for a person under the age of 25 to tear their meniscus without some element of knee trauma. Usually, this is a weight-bearing, twisting injury. (acute kind) • **Middle-aged and older adults** can sustain meniscus tears from squatting or falling. As we age, the meniscus cartilage becomes more fragile (degenerative), and it is possible to tear the meniscus cartilage by simply squatting. (degenerative kind) 
History (symptoms)	• With an acute meniscal tear, an effusion may develop slowly several hours after injury. This differs from an anterior cruciate ligament (ACL) injury, where swelling develops rapidly within the first few hours. • Patients with meniscal injuries localize pain to the joint line or posterior knee and describe • mechanical symptoms of locking or catching. نسأل المريض هل ركبته تخونه؟ • Mechanical symptoms: Locking or catching. • Chronic meniscal tears demonstrate intermittent effusions with mechanical symptoms

1- Which is why meniscectomy is often associated with early Osteoarthritis.

Knee Injuries

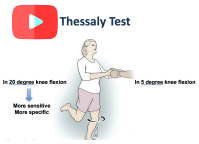
Menisci Injuries تمزق الغضروف الهلالي

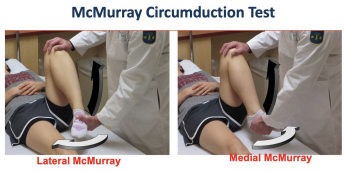
Physical Examination

- Small joint **effusions** and **joint line tenderness** with palpation are **common findings** with meniscus tears, palpation with patient has osteoarthritis isn't useful. Joint line tenderness is a most sensitive sign.
- Manipulative maneuvers, including the **McMurray** and **Apley tests** may produce a palpable audible **click** with localized tenderness, but they are **not specific** for meniscal pathology.
  - ★ Range of motion is typically normal, but longitudinal bucket-handle tears may **block** full **extension** of the knee joint. -> locking. (+Know difference between leg lag and lock?)
  - ★ **Combined testing has improved accuracy**
  - ★ **An effusion combined with joint line tenderness (JLT) is one of the most sensitive and reliable signs of a meniscal tear.**

Doctor skipped this table

Test	Sensitivity	Specificity
Joint line tenderness	71% MM 78% LM	87% MM 90% LM
Apley grind test	41% for both	93% MM 86% LM
McMurray test	48% MM 65% LM	94% MM 86% LM
Thessaly test	89% MM 92% LM	97% MM 96% LM





Imaging

- Standard knee radiographs should be obtained for evaluating for: Bone injuries or abnormalities, Osteoarthritis, x ray we can't see anything regards of meniscus but to see possible arthritis.
- **MRI** remains the noninvasive diagnostic p**rocedure of choice for confirming meniscal pathology**.

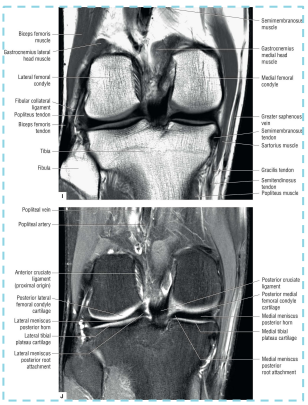
Differential Diagnosis

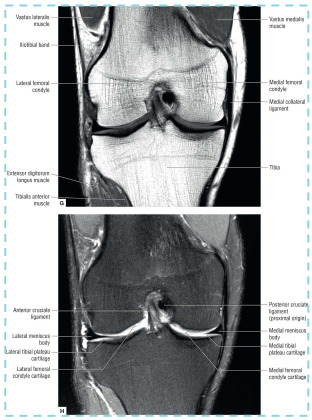
- Differential diagnosis Prior to MRI, several large studies demonstrated accuracy of the clinical diagnosis of meniscus tears to be 70% to 75%.
- **The differential for meniscus tears includes intra-articular and extra-articular diagnoses:**
  - **Intra-articular:** possibilities include: osteochondritis dissecans, medial patella plica, patellofemoral pain syndromes, loose bodies, pigmented villonodular synovitis, inflammatory arthropathies, and osteonecrosis.
  - **Extra-articular:** possibilities include: collateral ligament injuries, slipped capital femoral epiphysis, bone or soft-tissue tumors, osteomyelitis, synovial cyst, pes or medial collateral ligament bursitis, injury, reflex sympathetic dystrophy, lumbar radiculopathy, iliotibial band friction, and stress fracture.

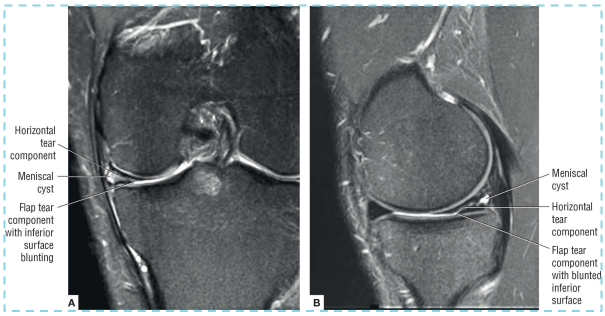
Management

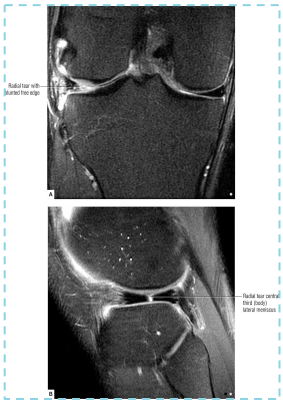
- **Nonsurgical:** if no mechanical symptoms
  - Not all meniscus tears cause symptoms, and many symptomatic tears become asymptomatic.
  - All degenerative meniscus tear.
  - Nonsurgical management include: ice, NSAIDs, or physical therapy for range of motion
  - and general strengthening of the lower extremities. they respond well
- **Surgical indications:** no need for surgery unless it disturbs his life his daily activities not
  - his hobbies or there is pain or mechanical block
  - Failure of conservative treatment
  - Locked knee blocking or displaced bucket handle tear
  - Concomitant ACL surgery. We do meniscectomy with ACL reconstruction.
  - Mechanical symptoms (unstable tears)
  - Young active patient
  - **Type of surgical intervention:**
    - Repair whenever possible, meniscus tear should be repaired and saved. first choice, but if the tear is at an avascular zone we might have to do meniscectomy
    - Meniscectomy (Arthroscopic partial/subtotal/ or total meniscectomy) Only if NOT REPAIRABLE tear – Tears not amenable to repair (complex, degenerative, radial tear patterns) More risk of osteoarthritis progression !

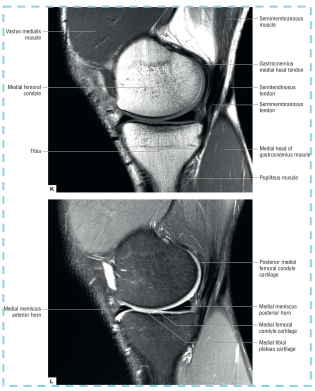
Imaging

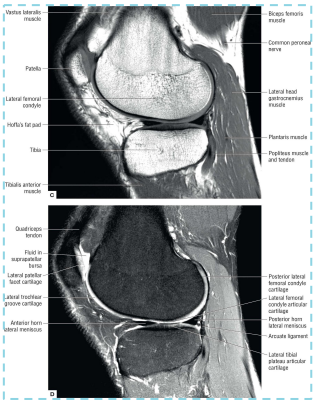


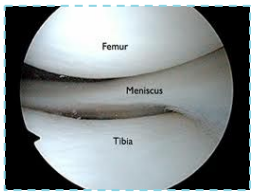


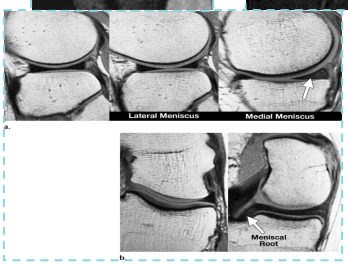


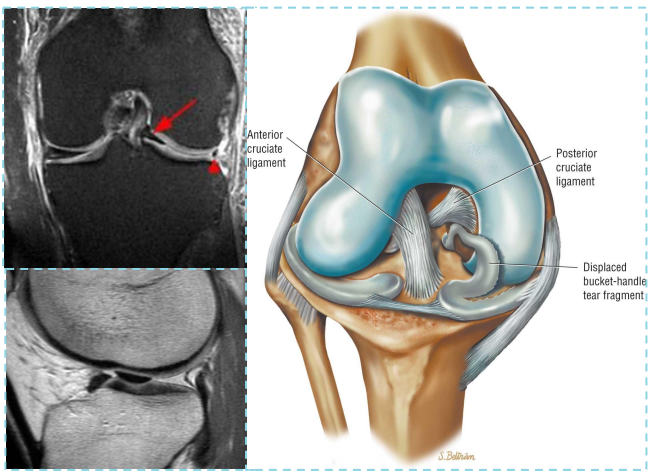








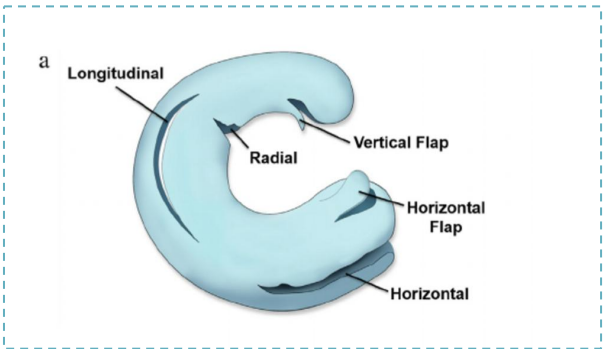


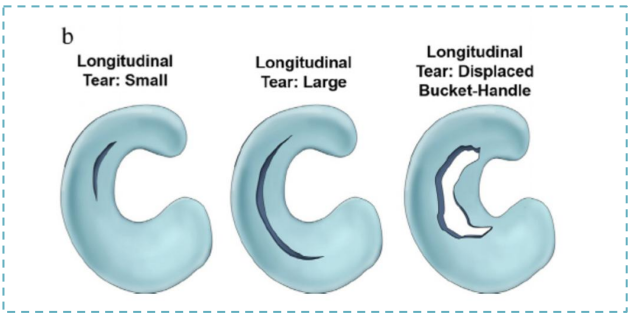


Bucket handle tear

[Mensical radiology guide for dummies]

Meniscal tear types



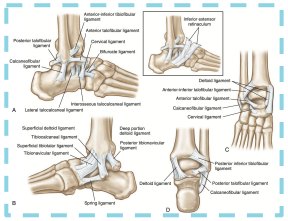


Ankle Sprain

Ankle Sprain التواء الكاحل

Characteristics

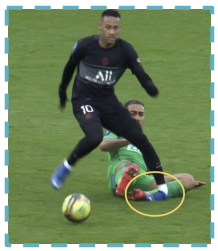
- Ankle sprain is a common sports related injury. more than ACL
- Lateral sprains accounting for 85% of all such injuries.
- The most common reason for missed athletic participation
- We have something called anterior fibular ligament which is more common to be injured than the ACL. Especially in female but they can live with it to the point that the stress start to develop upon the bones so she can't handle it anymore.
- Females more common because of laxity and high heeled shoes.



Classification of Acute Lateral Ankle Sprains

- **Grade I :**
  - **Mild injury to the lateral ligamentous complex.**
  - No frank ligamentous disruption is present.
  - Mild swelling, little or no ecchymosis on the lateral aspect of the ankle, and no or mild restriction of active ROM. Difficulty with full weight bearing is sometimes seen. No laxity on examination.

- **Grade II:**
  - **Moderate injury and partial tear to the lateral**
  - ligamentous complex.
  - Restricted ROM with localized swelling, ecchymosis, hemorrhage, and tenderness of the anterolateral aspect of the ankle.
  - Abnormal laxity may be mild or absent.
  - May be indistinguishable from a grade III injury in the acute setting.



Inversion type ankle injury on a plantarflexed foot

- **Grade III:**
  - **Complete disruption of the lateral ligamentous complex.**
  - Diffuse, swelling,tenderness and ecchymosis on the lateral side of
  - the ankle and heel. ++ instability

Ankle sprains types

- **High ankle sprain**: Syndesmosis injury. 1-10% of all ankle sprains
- **Low ankle sprain**: Lateral ankle sprain. ATFL and CFL injury. >85% of all ankle sprains
- **Medial ankle sprain**: Deltoid ligament injury

CFL: Calcaneofibular Ligament ATFL: Anterior talofibular ligament

Presentation

- **History:** History suggestive of inversion injury
- **Physical examination:** Localized tenderness, swelling, and ecchymosis over the lateral ankle.
- **Special tests:**
  - The anterior drawer test may demonstrate anterior talar subluxation.
  - The talar tilt stress test may demonstrate positive tilt to inversion stress

Treatment

- **Non-surgical management:** Start with Conservative; (functional treatment) consists of 4 (RICE-proper shoes - brace - physiotherapy)
  - Initial treatment consists of RICE. + short period of immobilization (10 days or less)) followed by early physiotherapy.
  - Early weight bearing and use of a protective brace during functional activities facilitates recovery better than non–weight bearing or immobilization.
  - Functional instability may result and should be treated with a course of Physical therapy and proprioceptive training[1].
  - Residual mechanical instability may be managed effectively with bracing or taping.
  - Patients may return to unrestricted activity when cutting, running, and hopping on the affected leg are no longer painful.
  - 90% of acute ankle sprains resolve with PRICE and early functional rehabilitation.
- **Surgical indications :**
  - when an adequate trial of nonsurgical treatment fails to control symptoms for grade III.
  - Ligaments repair/reconstruction

Quiz

Q1: An 18 year old young man presented to the orthopedic OPD with a H\O twisting injury to his left knee 10 days ago. He reported that his knee is (locked) since the injury. O\E: left knee medial side tenderness and ROM from 150 to full flexion. MRI showed a tear of the medial meniscus. Which of the following is the most appropriate treatment ?

- A: Manipulation under anesthesia
- B: Physiotherapy
- C: Steroid injection
- D: Urgent Arthroscopy

Q2: A 35 years old man C/O severe knee pain for 2 hours sustaining a non-contact twist while playing soccer. O/E showed massive effusion in the knee with ecchymosis. What's the most important initial investigation?

- A: MRI
- B: Plain X-ray
- C: US
- D: Nuclear Medicine Studies

Q3: A 30 years old man came to the ER with pain and swelling of the knee after Dashboard injury, what is the most common injury in this case?

- A: ACL injury
- B: MCL injury
- C: PCL injury
- D: Meniscal injury

Q4: A 21 years old came to the ER due to Knee dislocation, there was multiligament injury involving ACL, PCL, MCL, after checking NV examination there was no distal pulse, what is the most likely injured artery and injured nerve respectively in this case?

- A: Popliteal Vein, Peroneal Nerve injuries.
- B: Popliteal Artery, Sciatic Nerve Injuries
- C: Popliteal Artery, Peroneal Nerve Injuries.
- D: Popliteal Artery, Vagus nerve Injuries.

1:D 2:B 3:C 4:C

Team Leader

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18

Orthopedic
441 Team

MED441
KING SAUD UNIVERSITY

Editing File

Compartment Syndrome & Acute Joint Dislocation

Dr. Hamzah Alhamzah

Color Index:

- Main Text
- Important
- 441 Notes
- Old Notes
- Extra

Objectives

Compartment syndrome

- To explain the pathophysiology of CS.
- To Identify patients at risk of developing CS.
- To be able to diagnose and initially manage patients with CS.
- To be able to describe the possible complications of CS.

Acute joint dislocation

- To describe mechanisms of joint stability.
- To be able diagnose patients with a possible acute joint dislocation.
- To be able to describe general principles of managing a patient with a dislocated joint.
- To describe possible complications of joint dislocations in general and in major joints such as the shoulder, hip and knee.

Resources

Acute Compartment Syndrome
By Armando Hasudungan

Hip Dislocation
By nabil ebraheim

Leg Compartment Syndrome
By Orthobullets

Hip Dislocation
By Orthobullets

Shoulder Dislocation
By Rhesus Medicine

Shoulder Dislocation
By nabil ebraheim

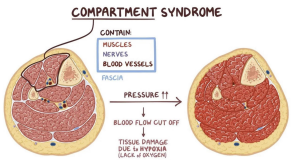
Knee Dislocation
By Orthobullets

Anterior Shoulder Dislocation
By Orthobullets

Posterior Shoulder Dislocation
By Orthobullets

Compartment Syndrome

Definition



Group of muscles and nerves wrapped in a layer of deep fascia

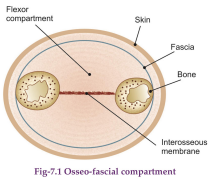
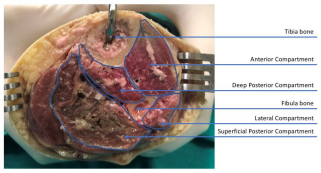


Fig-7.1 Osseo-fascial compartment

- It's a potentially devastating condition in which the pressure within an osteofascial compartment rises to a level that decreases the perfusion gradient across tissue capillary beds, leading to cellular anoxia, muscle ischemia, and death.
- It's an **orthopedic emergency**, in which intercompartmental pressure "tissue pressure within a closed muscle compartment" exceeds perfusion pressure, results in muscle & nerve ischemia which lead to muscle necrosis, and it can develop wherever a compartment is present[1], and it can be either due to increased compartment content or decreased space, can happen with or without fracture (e.g. burn, crush injury, cast, etc..).

Pressure Gradient



- Arteries pressure is greater than > arteriole > capillary bed which is (diffusion/exchange) site > venule > vein[2].

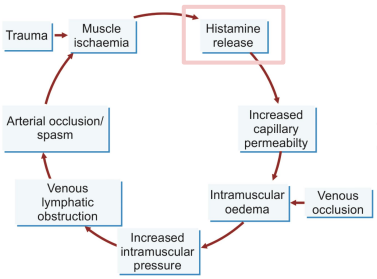
Pathophysiology

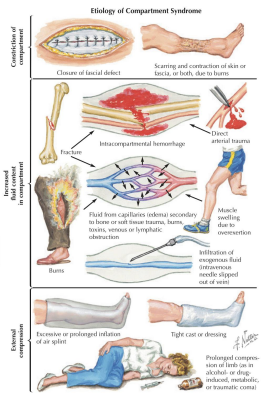
IMPORTANT

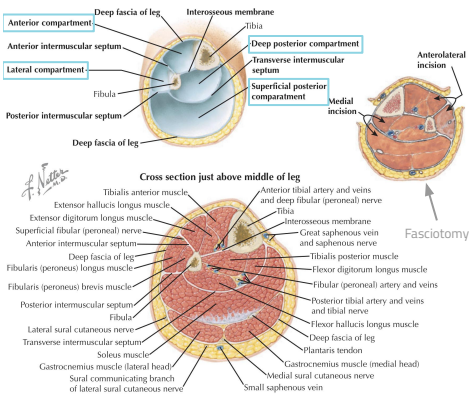
- Risk factor (warfarin, valve replacement) → elevated tissue pressure → absence of diffusion at the capillary bed → cell damage and swelling → further increase in tissue pressure → lack of oxygenation → vicious circle
- **Threshold pressure:** "Absolute measurement"
  - It's CS if the Intercompartmental pressure exceeds 30 mmHg (rigid measurement). Because this pressure is enough to close the capillary bed perfusion pressure.
- **Relative measurement:**
  - Δ P = **DBP - Intercompartment** = <30 (**if less than 30 it is CS**)[3].
  - **Less than 30 mmHg difference** between compartment pressure and preoperative diastolic pressure = it is more relative to perfusion = (clinically relevant)[4], normal CP must be < DBP by 30 mmHg.
- **Examples:**

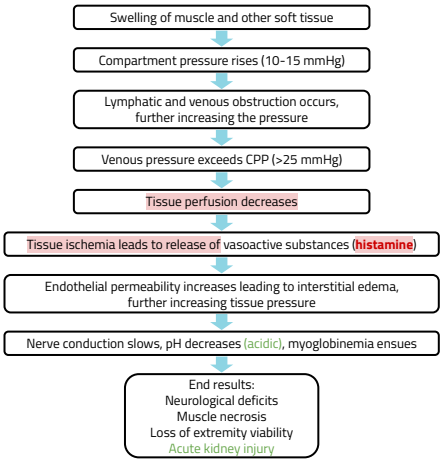
1- Patient's BP was 100/60 and his intracompartmental pressure (IMP) was 25 mmHg, is this CS?
- No, this patient doesn't have CS because (60-25=35) which is not <30 mmHg which means that the compartmental pressure didn't exceed the perfusion pressure (no ischemia).

2- Patient had a diastolic pressure of 50 and Intracompartmental pressure of 25 mmHg, is this CS?
- Yes, because (50-25=25) which is <30 mmHg.









1- It typically occurs following a traumatic event, most commonly a fracture. Also, could happen due to burns or a cast.
2- Very unlikely to have an absent artery pulse because it's unlikely for compartment pressure to be > artery pressure. However, when it reaches the level of perfusion pressure the compartment cascade will begin.
3- First patient 80 years old with BP 90/40 and relative compartment pressure is 20, second patient 20 years old muscular bodybuilder with BP 120/80 and relative compartment pressure is 29. Which one has a high risk of compartment syndrome? **First one** due to low relative compartment pressure and low diastolic pressure.
4- Relative measurement is more important than absolute measurement, because variation between people limits the use of it, since a big muscular guy differs from an old thin lady.

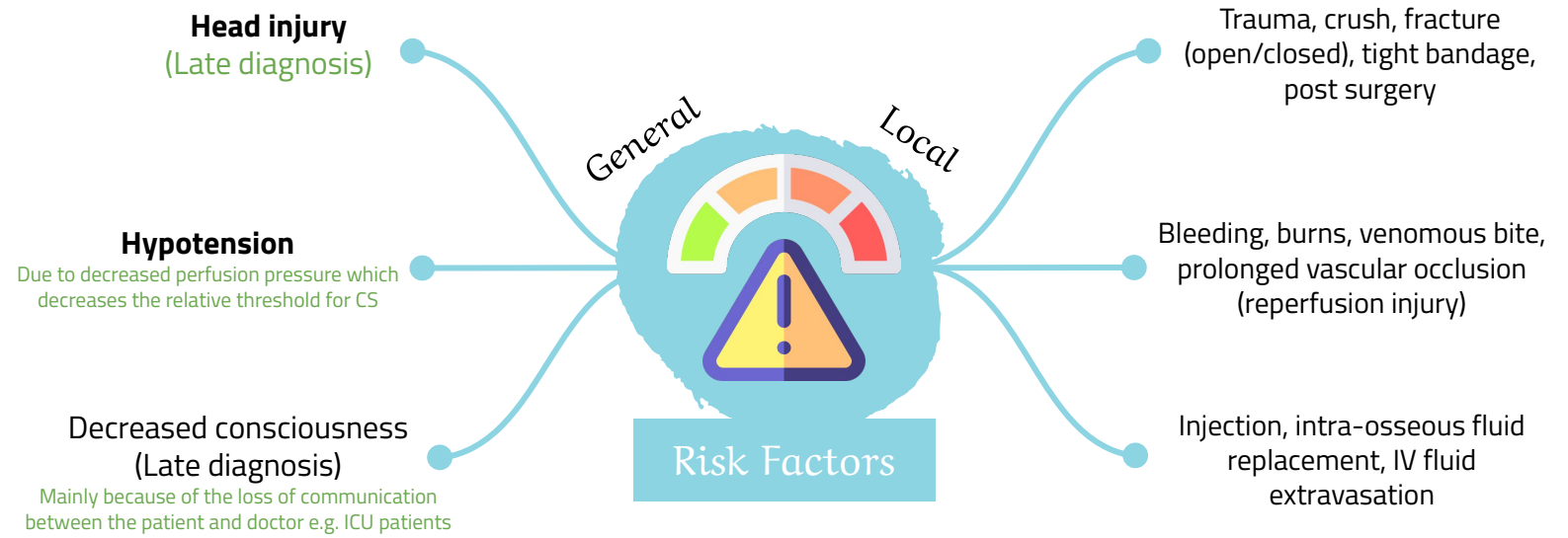
Etiology

Increased Compartmental Volume (internal expanding forces)

- Close soft tissue injury/crush injury. Local hematoma inside
- Close fracture. Internal medullary artery bleeding
- Open fracture[1]. → Higher risk than closed fracture
- Hemorrhage. From medulla
- Vascular injury. Like laceration, popliteal artery damage if waiting for > 6hrs can lead to reperfusion syndrome, which is the damage done by restoration of blood flow to prolonged ischemic tissues, resulting in sudden release of muscle necrosis products into the circulation (e.g. reactive oxygen species, myoglobin, K^+), causing many adverse effects (e.g. AKI, DIC, Arrhythmias, CS, etc..)
- Coagulopathy (anticoagulation therapy).
- Increased capillary permeability after burns (especially circumferential).
- Infusions or high-pressure injections (eg, regional blocks, paint guns). Radiology contrast injection intervention/extravasation
- Reperfusion after prolonged ischemia[2]. (most ischemia → fasciotomy)

Reduced Compartmental Volume (external compressing forces)

- Tight circumferential dressings (artificial) (e.g. can occur with cotton cast padding alone).
- Cast or splint (artificial).
- Prolonged limb compression as in Trendelenburg, lateral decubitus, alcohol or drug abuse[3].



"Young, male, high energy mechanism injury, E.g. Young patient coming with a tibial diaphyseal fracture"

1- It is a common mistake that people make thinking that an open fracture helps in releasing the pressure. In fact, usually the hole is small and isn't enough to overcome the pressure caused by the internal bleeding. The blood also can close the hole by acting like a valve. Moreover, the hole is usually in one compartment and we have more than one (4 in case of the tibia).
2- Damage to the basement membrane can cause leakage of intravascular fluid into the interstitium increasing the intracompartmental volume. The cut off in ischemia is usually 6 hours until the cellular death of the structures (vessels, nerves, ...)
3- It happens more in western countries by getting drunk and not moving for a long time (>24 hrs). This can compress one side of the leg causing a compartment syndrome on the other side.

Orthopedic Conditions

Underlying Condition	% of Cases
Tibial diaphyseal fracture	36
Soft tissue injury	23.2
Distal radius fracture	9.8
Crush syndrome	7.9
Diaphyseal fracture forearm	7.9
Femoral diaphyseal fracture	3.0
Tibial plateau fracture	3.0
Hand fracture(s)	2.5
Tibial pilon fractures	2.5
Foot fracture(s)	1.8

Why tibial fractures are number 1 ?

- Scientifically, it's proven by evidence.
- Logically, the tibia is a large bone placed in a small compartment.

- When you hear in MCQ or OSCE or anywhere "Tibial shaft fracture, young adult, male, pain" immediately think about CS.

Diagnosis

- It's a clinical diagnosis! (No X-ray, US, CT or MRI).
- You need to keep a high index of suspicion to perform immediate surgery and save the patient's limb.

Signs & Symptoms

Anterior compartment of the leg (the most commonly affected compartment)

- Tense swelling.
- Tight (woody compartment):
  - Most reliable clinical sign.
  - Calf muscle would be hard (rigid) and woody (normally soft and fleshy).
- Tender compartment:
  - Movement of anterior or posterior muscles of leg elicits severe pain.

Early

The earliest of the 'classic' features is severe pain (or a 'bursting' sensation) and this may be the only feature seen.

- Very severe **pain**, not responding to analgesics:
  - **Out of proportion** to the injury.
  - Patient cries from the pain.
  - Increase need of analgesics, if the patient respond to analgesics, then it's not CS.
  - This is the most sensitive & 1st symptom.
- Pain increase with passive **stretching** of the muscles in the involved compartment:
  - Dorsiflexion → Posterior compartment.
  - Plantarflexion → Anterior compartment[1].
  - Ischemic muscle is highly sensitive to stretch.
- Pain might be worse with elevation.
- Patient will not initiate motion on their own.
- Presence of risk factor.
- Measurement of the IMP is high.

Late

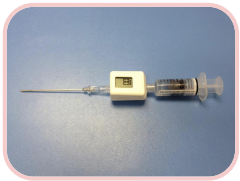
- 4 P's: Paralysis, Paresthesia, Pallor and in severely high pressure Pulselessness.
- Pulselessness is RARE, and it's sign of severity, only severely high compartment pressure causes it because the pressure need to exceed the arterial pressure to occlude it (almost never), don't say it's not CS because there is pulse. The ischemia occurs at the capillary level, so pulses may still be felt and the skin may not be pale!

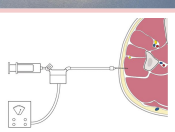
Pediatrics

3 A's: (Increasing Analgesic requirement, Anxiety, Agitation)

IMP (Intramuscular Pressure)

Not used anymore





- We insert a device in the compartment that injects fluid and collect it and provide us with the reading.
- Rarely necessary and might give a false low result, so must be done at the area of the highest expected pressure.
- We can use it as a relative measure if the patient is unconscious and cannot communicate e.g. ICU patients, those who are comatose, have psychiatric problems, or are under the influence of narcotics.
- Issues:
  - Not reliable (if injected in muscle → very high reading).
  - High false positive rate (perform unnecessary procedures).

1- Most common affected compartment.

Management

Initial (Undeveloped CS)

- Maintain normal blood pressure.
- **Remove** any constricting bandage. if there is no improvement within 2 hours of splitting the dressings, fasciotomy should be performed.
- Keep limb **elevated** at heart level.
- Regular close monitoring (observation) (15-30 minute intervals).
- Ice
- Avoid nerve blocks, sedation and strong analgesia to obtain patients feedback. Anything that can block pain response.

Late (Fully Developed CS)

- Remove any constricting bandage. (The first & immediate thing to do)
- Maintain normal blood pressure.
- Keep limb elevated at heart level.
- Diuresis to avoid kidney tubular injury if late. Diuresis isn't for CS it's to protect the kidney from myoglobinuria (That results from rhabdomyolysis "muscle damage").
- Urgent surgical decompression (**Fasciotomy**). The sooner the better, because we're not treating CS we're preventing the sequelae of CS.

Summary

1. Split the cast and dressing down to the skin.
2. Elevate the leg (to the level of the heart).
3. Ice to decrease the swelling.
4. Feel the compartments, if not tight, observe the patient every 2 hours.
5. If compartments are tight, take the patient to OR for emergency fasciotomy.

Fasciotomy

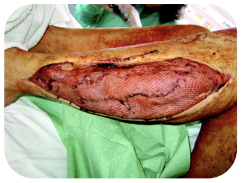
- It is a prophylactic procedure[1] done by releasing the compartment fascia to allow swollen muscles to expand which will decrease IMP and prevent further damage "the deep fascia is the most important to cut".
- This procedure doesn't reverse the damage (so better to have a low threshold), and should be done as soon as possible, and if there's still viable tissue (otherwise infection is likely).

Indications:

- Within 6 hours of total ischemia time (e.g. arterial embolism).
- Significant tissue injury.
- Worsening initial clinical picture.
- Delayed presentation with a picture of developed CS.
- Absolute compartment pressure >30 mmHg or relative pressure (DBP-IMP) <30 mmHg.

Technique[3]:

- Debridement of all necrotic tissue is necessary[2].
- Second and third look surgeries are often required, for reassessment and further debridement.
- Closure of skin is usually done after the swelling subsides.
- Skin grafting is often required.



- Patient has CS for 4 hours → Do fasciotomy.
- Patient has CS for 48 hours → Don't do fasciotomy, because there will be dead muscle and the damage already happened, so the late presentation of CS will not be treated with fasciotomy, we just prevent further complications.

1- It is a prophylactic procedure intended to save what's left of the viable tissue (never wait!). So if someone came with a >24 hours missed CS, there'll be no benefit for the fasciotomy, in general the fasciotomy time is (<24 hours → Do fasciotomy, >24 hours don't do fasciotomy).
2- Leave the skin open for 48 hours for debridement of dead tissue then check before closing the skin incision.
3- **Summary**: Take the patient for faciatomy: we do debridement and removing all dead tissue then we keep the wound open with sterile dressing that says "Do Not Touch", after 48 hours we go in again for a second, third look for dead tissue removing, then we try to close (we try to close only the skin if that didn't work we do a graft).

Complications

IMPORTANT

Renal failure

- Myonecrosis → myoglobinemia → myoglobinuria → AKI (tubular damage)
- We can measure creatinine kinase (CK) to check for rhabdomyolysis, also urine output will be decreased and turned to dark brown (black).

Loss of function

- Flexion contracture
- Paralysis (nerve damage)
- Loss of sensation and chronic pain
- Loss of limb "Amputation"

Leg complications

Anterior compartment:

- Drop foot

Deep posterior compartment:

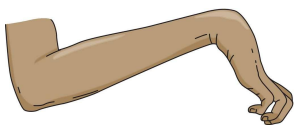
- Clawed toes, Ischemia of the calf muscles
- Loss of sensation in the sole

Forearm complications

Volar "palmar side" **compartment**:

- Volkmann contracture[1]

- Muscles are dead and start to be contracted → fibrosed → holds in a position → not allowed to move. The most commonly affected sites are the forearm and hand, leg and foot. In a severe case affecting the forearm, there will be wasting of the forearm and hand, and clawing of the fingers.



The possible complication upon opening the fascia is such as when the patient already developed rhabdomyolysis, the patient could develop **hyperkalemia** and cardiac arrest eventually, so you have to be in certain with anesthesia, first with opening of compartment there will be gush of lysed cells that contains lactic acid and potassium into the blood and to the body causing something called (**reperfusion injury**), so after cutting, you should tell anesthesia to inject calcium gluconate.

Extra

- **Chronic Compartment Syndrome** "Chronic exertional compartment syndrome (CECS)":
  - Young athletes especially long-distance runners sometimes develop pain along the anterolateral aspect of the calf, brought on by muscular exertion. Swelling of the anterior calf muscles contained within the nonexpansile deep fascia causes ischaemia of the deep peroneal nerve as it traverses the compartment. The condition is diagnosed from the history and can be confirmed by measuring the compartment pressure before and after exercise. Release of the fascia is curative. The same syndrome is very rarely seen in the forearm muscles (known as 'arm pump' in motorcyclists).
- **Interesting information:** The ischemic calcification and even ossification that occur in traumatic compartment syndromes in the lower extremity can often mimic a tumor. The initial injury is usually a crushing type that causes increased compartment pressure from muscle swelling. This pressure eventually leads to ischemic necrosis of the compartment muscle, which several years later becomes calcific or even ossified. Because the muscle appears firm and calcified on radiographic examination, the clinician may not relate the finding to an old injury and may suspect a calcifying sarcoma such as synovial sarcoma. The most common place for this pseudotumor is in one of the muscle compartments of the leg, and it causes stiffness and muscle weakness at the ankle and foot area.

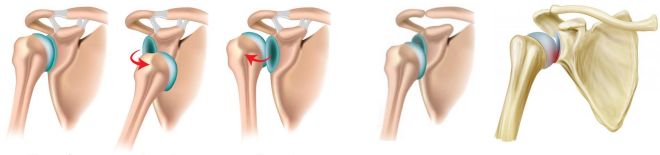
Radiograph (A) and CT scan (B) of an old compartment syndrome of the anterior compartment of the leg of an 81-year-old woman who had a history of fracture treated with internal fixation 60 years prior.

1- Permanent flexion contracture due to shortening of forearm muscles ("claw-like deformity" of the hand, fingers, and wrist) usually caused by ischemia due to compartment syndrome, muscle once infarcted is replaced by inelastic fibrous tissue (Volkmann's ischaemic contracture).

Acute Joint Dislocation

Definition

- A joint dislocation is described by stating the location of the distal segment in relation to proximal.
  - Anterior shoulder dislocation: anterior displacement of the humeral head relative to the glenoid
  - Posterior hip dislocation: posterior displacement of the femoral head relative to the acetabulum
- **Dislocation**: is the total loss of contact between the articular surfaces of the joint.
- **Subluxation**: is the partial loss of contact between the articular surfaces of the joint.
- **Joint stability**:
  - Bony stability:
  - ➜ Shape of the joint (ball and socket vs round on flat)
  - Soft tissue:
  - ➜ Dynamic stabilizer (tendons/muscles)[1]
  - ➜ Static stabilizer (ligaments ± meniscus/labrum)[2]

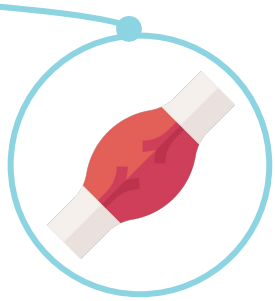


- Complex synergy leading to a functional and stable joint.
- It takes higher energy to dislocate a joint with bony stability (e.g. Hip) than a joint with mainly soft tissue stability (e.g. Shoulder) like weak ligaments.
- Connective tissue disorders may lead to **increased joint instability** due to abnormal soft tissue stabilizers[3].
- Dislocation of a major joint should lead to considering other joints[4].
- When a joint is subjected to sufficient force in certain directions it might sustain a fracture (in stable joint usually like hip joint), a dislocation (with torn ligament) or a fracture dislocation.
- Different joints have different force vectors that may lead to a dislocation.
- A joint might dislocate in different directions; in acute dislocation mainly is in one direction.
- Chronic joint dislocation, more in small joints, chronic doesn't mean recurrent.

Groups at Risk

Major trauma victims especially unconscious patient.
(e.g. Dashboard injury)

Athletes and sport enthusiasts.
(e.g. Anterior shoulder dislocation)



Connective tissue disorder patient;
due to increased joint instability in result of abnormal soft tissue stabilizers and increased soft tissue laxity.

1- The body move the joint by contracting the muscles and these muscle cause compressive force to hold the joint in place, such as in shoulder deltoid muscle.
2- One which is holding the joint in the place regardless of the movement status of joint (mainly ligaments) e.g. acute knee dislocation happens because there is injury in the ligament of knee that stabilize the knee joint, shoulder dislocation rely mainly on soft tissue called labrum which is a kind of cartilage that surrounds the periphery of the glenoid, if torn the join is no further stable.
3- Ehlers-Danlos Syndrome has high tendency to have dislocation at lower threshold of energy.
4-If an energy is sufficient to dislocate a joint you should suspect other injuries including muscle/neurovascular.

Diagnosis

- History of a traumatic event (major trauma or any trauma with the limb in high risk position).
- Very severe pain and inability to use the limb.
- Deformities:
  - Squaring of shoulder in shoulder dislocation (may have axillary nerve injury, deltoid atrophy and loss of sensation)
  - Shortening
  - Malalignment
  - Malrotation
- Should check for other injuries (distracting injury), pain can hide things.
- Should always check the distal neurovascular status, before & after to protect the patient from further damage and yourself from medicolegal issues.
- Should check for compartment syndrome.

loss of the usual rounded contour of the shoulder

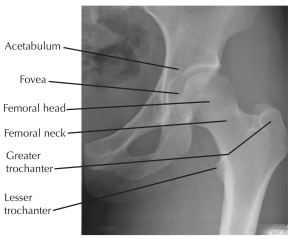
Squaring of shoulder (right shoulder dislocation)

Internal rotation, shortening of the leg & posterior hip dislocation

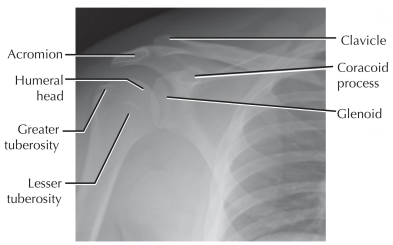
Summary:
1-Severe pain
2-Seen deformity
3-Loss of function

X-ray

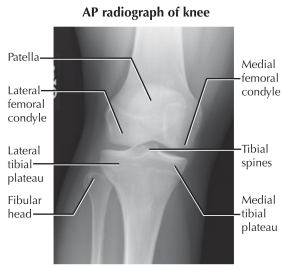
- Should be done urgently without delay if dislocation is suspected, the dislocation must reduced after obtaining X-rays, because maybe there's a fracture and you make it worse.
- Two perpendicular views of the involved joint, it's 2D image, one view is misleading.
- Occasionally, special views are required such as the **axillary view** (to isolate the joint) for shoulder dislocation.
- X-rays to the joint above and below.
- Normal X-rays:



Hip, AP xray



X-ray, AP



Management Principles

Don't forget to take consent from the patient

1. Must rule out other injuries
2. Pain relief (consent for reduction/conscious sedation "no intubation")
3. Urgent reduction (By traction and countertraction until we heard clunk "pop" sound)
4. Check stability and safety zone
5. Check neurovascular status after reduction (even before)
6. X-ray after reduction to confirm the reduction[1,2]
7. Protect the joint (by immobilization and stabilizing for 2-3 weeks)
8. Rehabilitation (physiotherapy)
9. Follow for late complications

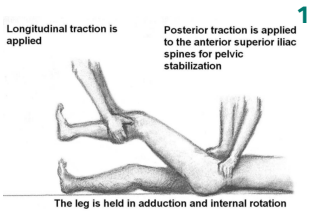
1- If the patient is in hospital setting already, then it's better to do X-ray before reduction for documentation if available.
2- Meanwhile, if patient is in the scene of injury then came to hospital, then it's better to reduce right away because the muscle are softer at that time and it's easier to reduce.

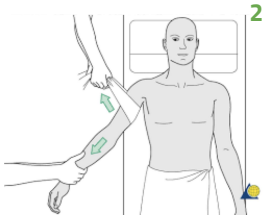
Reduction Technique

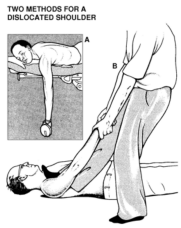
- The best treatment of pain is reduction of joint not medications.
- During reduction, follow the following steps:

1	Monitor vitals.
2	IV analgesia (opioid).
3	IV sedation (to relax the muscles).
4	Gradual traction to distract the joint.
5	Realignment and rotation to reduce the joint based on direction of dislocation.
6	A palpable clunk will be felt.
7	Check ROM "range of motion" and stability of the joint.
8	Once joint is felt to be reduced, check distal NV "neurovascular" status: • If it was intact before but not after → further urgent management is needed. • If it was not present before but intact after → check again later to confirm.
9	Observe patient vitals until medications wear out.
10	Stabilize joint and get X-rays.

- If irreducible or partial reduction only:
  - Urgent closed reduction under general anesthesia and possible open reduction if closed reduction fails.
- Usually due to:
  - Insufficient muscle relaxation.
  - Entrapment of soft tissue.







Summary

1. Start with (closed reduction with sedation) if that didn't work
2. Do (closed reduction under general anesthesia) if that didn't work as well
3. Do (open reduction)

Fracture and dislocation, you must rush the patient to the OR in 6 hours to prevent AVN

Special Consideration

- A fracture dislocation is usually reduced in an open fashion in the operating room.
- Must confirm **concentric reduction** on the X-rays, otherwise an open reduction should be performed.
- Make sure that the neurovascular status is documented before & after.

1- Posterior hip dislocation technique: Stabilize the pelvis with sheet → hip flexion → axial traction → wait for a 'Clunk' (if needed you could do internal rotation) → joint is reduced.
2- Shoulder dislocation technique: Has many techniques one of them is dislodging by using body weight of patient and pull the arm along the joint then use a folded sheet by a helper to pull shoulder.

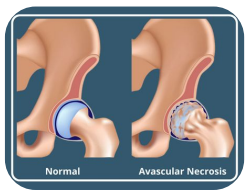
Complications

Early (Acute)

- **Heterotopic ossification**, formation of extraskeletal bone in muscle and soft tissues
- Neurological injury (reversible or irreversible)
- Vascular injury (common in neurological injuries)
- **Compartment syndrome**
- Osteochondral fracture/injury

Late (Chronic)

- Heterotopic ossification
- Stiffness
- Chronic instability, recurrent dislocation (common in shoulder)
- Avascular necrosis, It's a local bone ischemia due to destruction of blood supply (common in femur head)
- Osteoarthritis

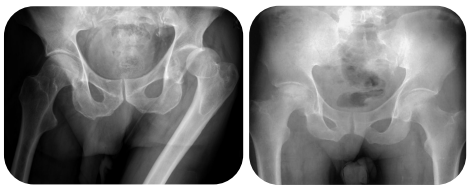


Examples of Joint Dislocation

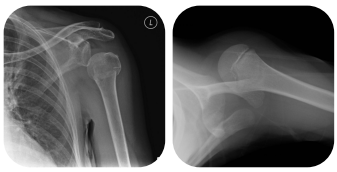
IMPORTANT

Hip Dislocation

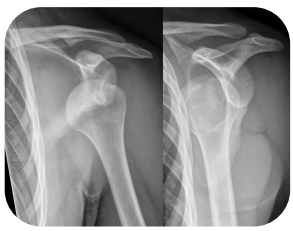
- **Posterior** dislocation is commonest among hip dislocations (high energy mechanism injury).
- Major trauma with hip flexed (dashboard injury in RTAs).
- Sciatic nerve injury is common; Most likely temporarily and take 9 months to resolve. check extension/flexion of big toe
- Loss of ankle dorsiflexion (foot drop), due to injury to peroneal nerve.
- High incidence of late avascular necrosis (10% in the beginning, reaching 80% after 6 hours), you have to follow up for 1 year.
- An orthopedic emergency!. Low recurrence rate.
- Immobilized by knee immobilization (brace), hip immobilization is hard.
- Traction by pulling flexed hip + counter-traction by holding pelvis on table.



Shoulder Dislocation



- Common. Low energy mechanism injury (sports)
- **Anterior** dislocation is more common.
- Patients presents with pain and limited range of motion after shoulder injury.
- May cause chronic instability (damaged labrum). Most common complication is recurrent dislocation.
- Patients with **seizures** (epilepsy) prone to **posterior** dislocation.
- Can result in axillary nerve injury (wasting in deltoid muscle and numbness over its area).
- Young patient with dislocation = Higher rate of recurrence.
- Immobilized by a normal sling.
- Image is showing anterior shoulder dislocation (humerus is anterior).
- Traction by pulling arm + counter-traction by a sheet around arm and pulling it.



Knee Dislocation

- Very serious emergency (could be either anterior or posterior).
- Usually with severe (high energy) trauma.
- Three or more ligaments are teared[1].
- May be associated with **popliteal artery injury** (50%) "limb threatening", peroneal nerve injury (foot drop) or with fracture/compartment syndrome.
- Needs accurate vascular assessment (popliteal artery).
- Most require surgery either early or late or both, prognosis is not great.
- Image is showing anterior knee dislocation (tibia is anterior).
- The pathognomonic sign of a posterolateral knee dislocation is the anteromedial distal thigh transverse "pucker" or "dimple sign".

1- Have medial & lateral collateral ligaments and, anterior & posterior cruciate ligaments, these main ligaments hold the knee + meniscus.

EXTRA

Compartment Syndrome

- increased interstitial pressure in an anatomical compartment (forearm, calf) where muscle and tissue are bounded by fascia and bone (fibro-osseous compartment), with little room for expansion
- interstitial pressure exceeds capillary perfusion pressure, leading to irreversible muscle necrosis (in 4-6 h) and eventually nerve necrosis

Etiology

- intracompartmental
  - fracture (particularly tibial shaft or paediatric supracondylar and forearm fractures)
  - reperfusion injury, crush injury, or ischemia
- extracompartmental: constrictive dressing (circumferential cast), poor position during surgery, circumferential burn

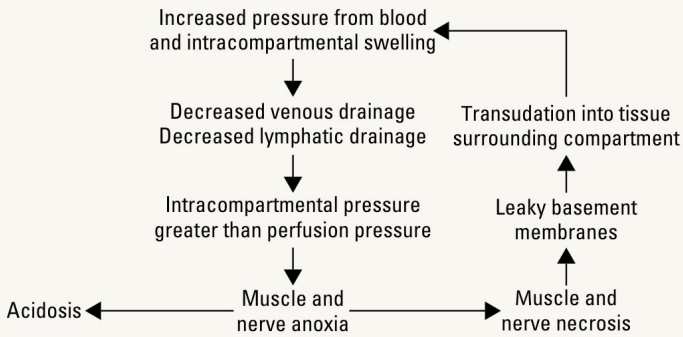


Figure 8. Pathogenesis of compartment syndrome

Most important sign is increased pain with passive stretch. Most important symptom is pain out of proportion to injury

5 Ps of Compartment Syndrome
Pain: out of proportion for injury and not relieved by analgesics
- Increased pain with passive stretch of compartment muscles

Pallor: late finding
Paresthesia
Paralysis: late finding
Pulselessness: late finding

Clinical Features

- pain out of proportion to injury (typically first and most significant symptom)
- pain with active contraction of compartment
- pain with passive stretch (most sensitive sign)
- swollen, tense compartment
- suspicious history

- **5 Ps**: late sign – do not wait for these to develop to make the diagnosis!

Investigations

- compartment syndrome is a clinical diagnosis; investigations usually not necessary
- in children or unconscious patients where clinical exam is unreliable, compartment pressure monitoring with catheter (normal = 0 mmHg; elevated ≥30 mmHg or [dBP – measured pressure] ≤30 mmHg)

Treatment

- non-operative
  - remove constrictive dressings (casts, splints), elevate limb to the level of the heart
- operative
  - urgent fasciotomy
  - 48-72 h postoperative: necrotic tissue debridement + wound closure
  - may require delayed closure and/or skin grafting

Complications

- Volkmann's ischemic contracture: ischemic necrosis of muscle; followed by secondary fibrosis; and finally calcification - especially following supracondylar fracture of humerus
- rhabdomyolysis, renal failure secondary to myoglobinuria

EXTRA

Shoulder Dislocation

- complete loss of continuity between the two articular surfaces of the glenohumeral joint; may be anterior or posterior

Investigations

- anterior disloction x-rays: AP, trans-scapular, and axillary views of the shoulder
- posterior dislocation x-rays: AP, trans-scapular, and axillary views of the shoulder; or CT scan

Prognosis

- recurrence rate depends on age of first dislocation
- <20 yr = 65-95%; 20-40 yr = 60-70%; >40 yr = 2-4%

Specific Complications

- recurrent dislocation (most common complication)
- unreduced dislocation
- shoulder stiffness
- rotator cuff or capsular or labral tear (Bankart/SLAP lesion)
- injury to axillary nerve/artery, brachial plexus

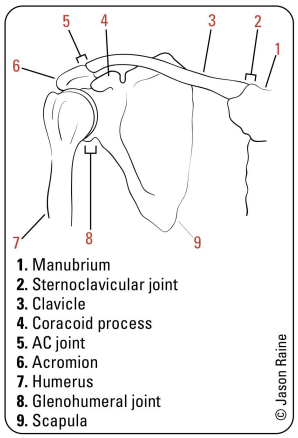


Figure 9. Shoulder joints

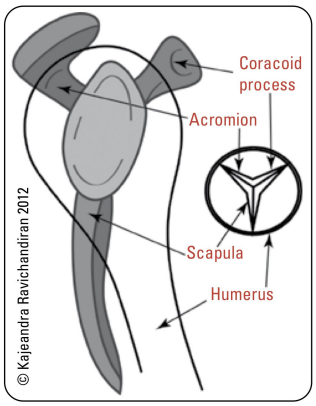


Figure 10. Mercedes-Benz

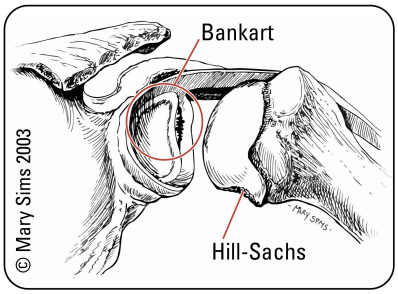


Figure 11. Posterior view of anterior dislocation causing Hill-Sachs and Bankart lesions

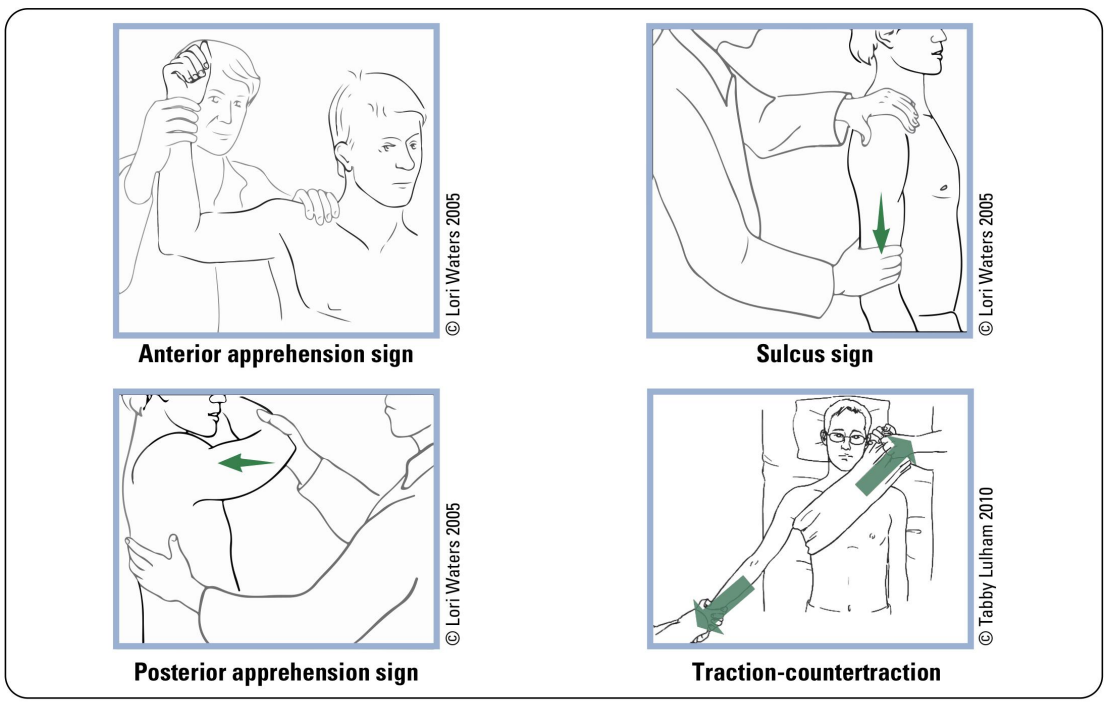


Figure 12. Shoulder maneuvers

Posterior Shoulder Dislocation
Up to 60-80% are missed on initial presentation due to poor physical exam and radiographs

There are 4 Joints in the Shoulder
Glenohumeral, AC, sternoclavicular (SC), scapulothoracic

Shoulder passive ROM: abduction – 180°, adduction – 45°, flexion – 180°, extension – 45°, int. rotation – level of T4, ext. rotation – 40-45°

Factors Causing Shoulder Instability

- Shallow glenoid
- Loose capsule
- Ligamentous laxity
- Frequency of Dislocations
- Anterior shoulder > Posterior shoulder
- Posterior hip > Anterior hip

The glenohumeral joint is the most commonly dislocated joint in the body since stability is sacrificed for motion

Table 8. Anterior and Posterior Shoulder Dislocation

	Anterior Shoulder Dislocation (>90%)	Posterior Shoulder Dislocation (5%)
MECHANISM		
	Abducted externally rotated/hyperextended arm Blow to posterior shoulder Involuntary, usually traumatic; voluntary, atraumatic	Adducted, internally rotated, flexed arm FOOSH 3 Es (epileptic seizure, EtOH, electrocution) Blow to anterior shoulder
CLINICAL FEATURES		
Symptoms	Pain, arm slightly abducted and externally rotated with inability to internally rotate	Pain, arm is held in adduction and internal rotation; external rotation is blocked
Shoulder Exam	**"Squared off" shoulder** **Positive apprehension test**: patient looks apprehensive with gentle shoulder abduction and external rotation to 90° as humeral head is pushed anteriorly and recreates feeling of anterior dislocation **Positive relocation test**: a posteriorly directed force applied during the apprehension test relieves apprehension since anterior subluxation is prevented **Positive sulcus sign**: presence of subacromial indentation with distal traction on humerus indicates inferior shoulder instability	**Anterior shoulder flattening**, prominent coracoid, palpable mass posterior to shoulder **Positive posterior apprehension ("jerk") test**: with patient supine, flex elbow 90° and adduct, internally rotate the arm while applying a posterior force to the shoulder; patient will "jerk" back with the sensation of subluxation Note: the posterior apprehension test is used to test for recurrent posterior instability, NOT for acute injury
Neurovascular Exam Including	**Axillary nerve**: sensory patch over deltoid and deltoid contraction **Musculocutaneous nerve**: sensory patch on lateral forearm and biceps contraction	Full neurovascular exam as per anterior shoulder dislocation
RADIOGRAPHIC FINDINGS		
Axillary View	Humeral head is anterior	Humeral head is posterior
Trans-scapular 'Y' View	Humeral head is anterior to the center of the "Mercedes-Benz"sign	Humeral head is posterior to center of "Mercedes-Benz" sign
AP View	Sub-coracoid lie of the humeral head is most common	Partial vacancy of glenoid fossa (vacant glenoid sign) and >6 mm space between anterior glenoid rim and humeral head (positive rim sign), humeral head may resemble a lightbulb due to internal rotation (lightbulb sign)
Hill-Sachs and Bony Bankart Lesions	± **Hill-Sachs lesion**: compression fracture of posterior humeral head due to forceful impaction of an anteriorly dislocated humeral head against the glenoid rim ± **Bony Bankart lesion**: avulsion of the anterior glenoid labrum (with attached bone fragments) from the glenoid rim	± **Reverse Hill-Sachs lesion** (75% of cases): divot in anterior humeral head ± **Reverse bony Bankart lesion**: avulsion of the posterior glenoid labrum from the bony glenoid rim
TREATMENT		
	Closed reduction with IV sedation and muscle relaxation **Traction-countertraction**: assistant stabilizes torso with a folded sheet wrapped across the chest while the surgeon applies gentle steady traction **Stimson**: while patient lies prone with arm hanging over table edge, hang a 5 lb weight on wrist for 15-20 min **Hippocratic method**: place heel into patient's axilla and apply traction to arm **Cunningham's method**: gentle longitudinal support and traction of the arm at the patient's side, massage/relaxation of deltoid, trapezius, and biceps to allow atraumatic shoulder reduction. Low-risk, low pain; if not successful try above methods Obtain post-reduction x-rays Check post-reduction NVS Sling x 3 wk (avoid abduction and external rotation), followed by shoulder rehabilitation (dynamic stabilizer strengthening)	Closed reduction with sedation and muscle relaxation Inferior traction on a flexed elbow with pressure on the back of the humeral head Obtain post-reduction x-rays Check post-reduction NVS Sling in abduction and external rotation x 3 wk, followed by shoulder rehabilitation (dynamic stabilizer strengthening)

EXTRA

Hip Dislocation

- full trauma survey (see Emergency Medicine, *Patient Assessment/Management*, ER2)
- examine for neurovascular injury prior to open or closed reduction
- high index of suspicion for associated injuries
- reduce hip dislocations within 6 h to decrease risk of AVN of the femoral head
- hip precautions (no extreme hip flexion, adduction, internal or external rotation) for 6 wk post-reduction
- see *Hip Dislocation Post-Total Hip Arthroplasty*, OR32

Up to 50% of patients with hip dislocations suffer fractures elsewhere at the time of injury

ANTERIOR HIP DISLOCATION

- mechanism: posteriorly directed axial loading of the femur with hip widely abducted and externally rotated
- classified into inferior (flexion, abduction, external rotation) and superior (extension and external rotation)
- clinical features: shortened, abducted, externally rotated limb
- treatment
  - closed reduction under conscious sedation/GA
  - post-reduction CT to assess joint congruity

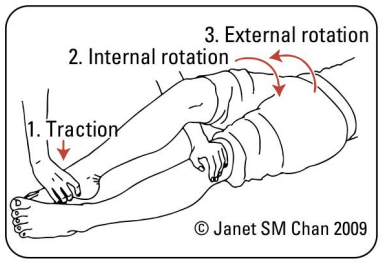


Figure 34. Rochester method

POSTERIOR HIP DISLOCATION

- most frequent type of hip dislocation (90%)
- mechanism: severe axial load to knee with hip flexed and adducted
  - e.g. knee into dashboard in MVC
- clinical features: shortened, adducted, internally rotated limb
- x-ray: affected femoral head will appear smaller than unaffected femoral head
- Thompson and Epstein classification – posterior dislocation:
  - I – with no or minor posterior acetabular wall fracture
  - II – with large posterior acetabular wall fracture
  - III – with comminuted acetabular fracture
  - IV – with acetabular floor fracture
  - V – with fracture of femoral head
- treatment
  - closed reduction under conscious sedation/GA only if no associated femoral neck fracture or ipsilateral displacement
  - ORIF if unstable, intra-articular fragments, or significant displacement
  - post-reduction CT to assess joint congruity and fractures

COMPLICATIONS FOR ALL HIP DISLOCATIONS

- post-traumatic OA
- AVN of femoral head
- associated fractures (e.g. femoral head, neck, or shaft)
- sciatic nerve palsy in 25% (10% permanent)
- HO
- thromboembolism – DVT/PE

EXTRA

Dislocated Knee

Mechanism
- high energy trauma more common (i.e. MVC) or low energy (sport related, obesity)
- by definition, caused by tears of multiple ligaments

Clinical Features
- knee instability
- effusion
- pain
- ischemic limb

Classification
- Kennedy classification (based on direction of tibial displacement) classified by relation of tibia with respect to femur
  - anterior, posterior, lateral, medial, rotary
- Schenck classification (based on pattern of ligamentous injury)

Schenck Classification
Type 1
Single ligament injury (ACL or PCL)
Type 2
Injury to ACL and PCL
Type 3
Injury to ACL, PCL, and either MCL or LCL
Type 4
Injury to ACL, PCL, MCL, LCL
Type 5
Multiligamentous injury with periarticular fracture

Investigations
- x-ray: AP and lateral
  - associated radiographic findings may include extensor mechanism injury, tibial plateau fracture dislocations, proximal fibular fractures, and/or avulsion of fibular head
- assessment of NVS:
  - ABI (abnormal if <0.9)
  - arteriogram or CT angiogram if abnormal vascular exam (such as abnormal pedal pulses)
  - detailed neurologic assessment, paying close attention to the peroneal nerve (foot drop is common)

Treatment
- urgent closed reduction
  - can be complicated by interposed soft tissue (posterolateral variant)
- assessment and management of neurovascular injuries
- emergent operative repair if vascular injury, open fracture or dislocation, irreducible dislocation, or compartment syndrome
- ligament reconstruction to restore knee stability is typically performed in a staged fashion
- comprehensive physiotherapy

Specific Complications
- high incidence of associated injuries (tibia/fibula fracture, extensor mechanism injury)
- popliteal artery injury
- peroneal nerve injury
- capsular tear
- chronic: instability, stiffness, post-traumatic arthritis

Quiz

Q1: A 35-year-old male sustains a closed tibial shaft fracture after falling from 12 feet. Which of the following measurements would be concerning for an evolving compartment syndrome?

A. Preoperative anterior compartment measurement of 25, with preoperative DP of 60

B. Intraoperative anterior compartment measurement of 25, with intraoperative DP of 54

C. Intraoperative anterior compartment measurement of 29, with preoperative DP 58

D. Intraoperative anterior compartment measurement of 28, with intraoperative DP of 72

Q2: During a dual incision fasciotomy of the leg, the soleus is elevated from the tibia to allow access to which of the following compartments?

A. Superficial posterior

B. Deep posterior

C. Lateral

D. Anterior

Q3: An 80-year-old woman presents to the emergency room with right shoulder pain and immobility. She fell down the steps outside her house and landed on her right side two hours prior to presentation. On exam, her right arm is abducted and externally rotated. She has decreased sensation to touch over the lateral aspect of her right shoulder. What is your diagnosis?

A. Anterior shoulder dislocation

B. Posterior shoulder dislocation

C. Impingement syndrome

D. Adhesive capsulitis

1:C 2:B 3:A

Quiz

Q4: 50 year old patient admitted to the ICU and intubated, a day later the nurse calls you and says he has compartment stiffness with an intramuscular pressure of 30 mmhg and a DBP of 70 mmHg, which of the following is the most appropriate management?

A. Fasciotomy
B. Observation
C. Ice
D. Angiogram

Q5: 23 year old male after MVA, came with limited shoulder abduction and loss of sensation over the lateral shoulder, what nerve is affected?

A. Radial
B. Ulnar
C. Axillary
D. Median

Q6: 17 year old male was presented to the ER with blunt trauma to his left leg, upon evaluation it appears that he developed compartment syndrome. According to the picture which compartment is mostly affected?

A. Anterior compartment
B. Lateral compartment
C. Superficial posterior compartment
D. Deep posterior compartment

4:B 5:C 6:A

Quiz

SAQs

441:

20-year-old man is post-surgical fixation of Rt tibia fracture earlier today. He has below knee back slab. He has increasing leg pain that cannot be controlled with pain meds.

1. What's your initial management in the floor?
   - Remove the back slab

2. Mention 3 possible complications if left untreated?
   1- Muscle necrosis
   2- Acute kidney injury
   3- Limb loss

22-year-old man sustained posterior hip dislocation.

3. Please describe reduction technique in Emergency department:
   1- Use analgesic and tell the patient to relax
   2- Stabilize the pelvis with sheet
   2- Do hip flexion
   3- Do axial traction
   4- If needed do internal rotation

439:

A 23-year-old patient presents to the emergency department after a motor vehicle accident. The patient was an unrestrained driver involved in a head-on collision. The patient is heavily intoxicated on what he claims is only alcohol. An initial trauma assessment is performed, and is notable for significant bruising of the right forearm. The patient is in the trauma bay, and complains of severe pain in his right forearm. A physical exam is performed and is notable for pallor, decreased sensation, and cool temperature of the skin of the right forearm. Pain is elicited upon passive movement of the right forearm and digits. A thready radial pulse is palpable. A FAST exam is performed, and is negative for signs of internal bleeding. The patient's temperature is 99.5°F (37.5°C), pulse is 100/min, blood pressure is 110/70 mmHg, respirations are 12/min, and oxygen saturation is 98% on room air. Radiography of the right forearm is ordered. The patient is still heavily intoxicated.

1. What is your diagnosis?
   - Compartment syndrome

2. How would you manage such patient?
   - Page 6

Done by

Abdulrahman Alroqi

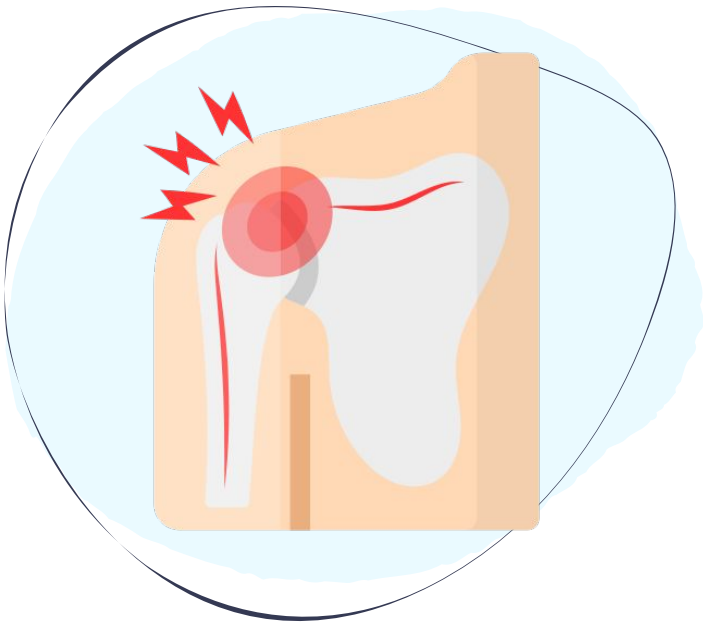
وفّقكم الله

19

Orthopedic 441 Team

MED441 KING SAUD UNIVERSITY

Editing File

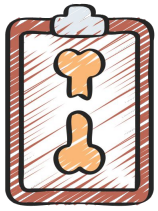


Common Shoulder Problems

Prof. Abdulaziz Alahaideb

Color Index:

- Main Text
- Important
- 441 Notes
- Old Notes
- Extra



Objectives

Specify the symptoms, signs and potential immediate complications of **common shoulder disorders.**

Outline the assessment and appropriate investigation and to outline the immediate and long term management of patients common shoulder disorders.

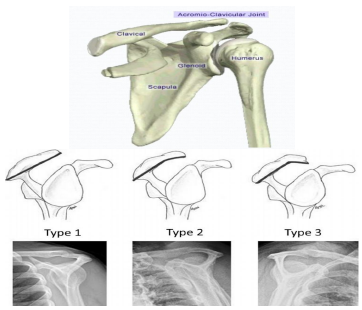
Demonstrate knowledge of indications for non-operative and operative treatment and to know the most common non-operative and operative measurements used for common shoulder disorders.

Resources

Shoulder Anatomy

Bones

- Humerus.
- Scapula (Glenoid, Coracoid, scapular body and acromion).
- The acromion has 3 different variations:
  Type I is Flat, type II is curved and type III is hooked.
  (Type I is the normal variation).
- Clavicle.
- Sternum.



Joints

all of them called the shoulder girdle but when we say shoulder joint we mean GHJ

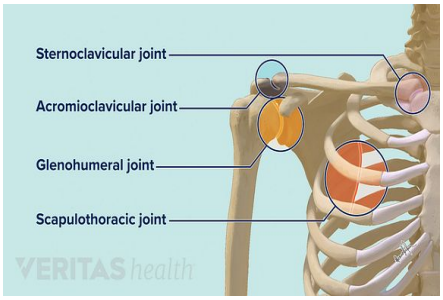
1- Glenohumeral joint (GHJ): The main joint, most common dislocated joint in the body, Why? Because it has the widest range of motion among all the joints and lacks bony stability.
Composed of:

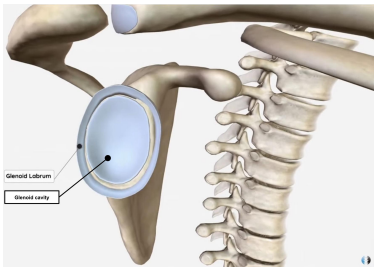
- Fibrous capsule.
- Ligaments.
- Surrounding muscles.
- Glenoid labrum. Anterior and posterior labrum which plays a very critical role in stability

2- Acromioclavicular (AC) joint.

3- Sternoclavicular (SC) joint.

4- Scapulothoracic joint is in the back.

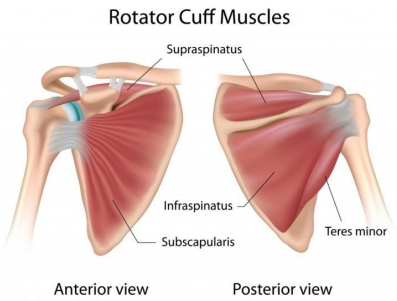


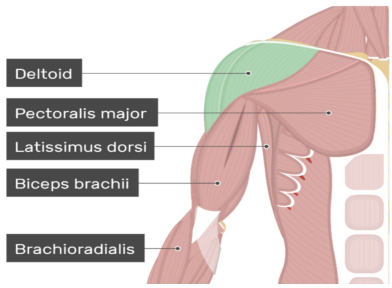


Muscles

- Rotator Cuff Muscles (SITS): depress humeral head against glenoid, it's main function is rotation hence the name
1- Supraspinatus: Initiation of abduction + external rotation.
2- Infraspinatus: External rotation.
3- Teres Minor not very important: External rotation.
4- Subscapularis: Internal rotation.

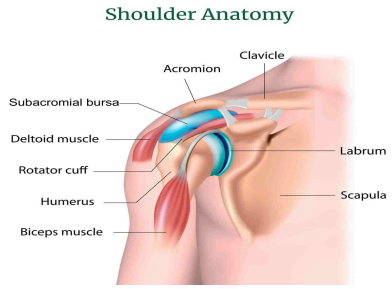
5- Deltoid: largest & strongest muscle of the shoulder. Most imp muscle of the shoulder, innervated by the axillary nerve. It has three attachments: from clavicle, acromion, scapular spine and attaches to the lateral aspect of the proximal humerus. It counteracts the action of the rotator cuff muscles and moves the humeral head upwards.
6- Pectoralis major.
7- Biceps.
- Posterior scapular muscles:
  8- Trapezius.
  9- Rhomboids (major & minor).
  10- Levator scapulae.
  11- Latissimus dorsi.
  12- Serratus anterior.



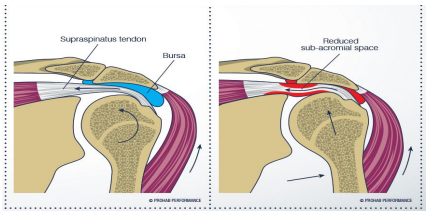


Subacromial bursa

- A bursa is a small fluid-filled sac that decreases the grinding between bones and muscle.
- Located between the acromion and the rotator cuff tendons.
- Protects rotator cuff tendons from grinding against acromion.
- Pathology → irritation → thickening → subacromial space narrowing → further impingement.



Impingement syndrome[1]



Definition

A condition in which the supraspinatus and bursa are pinched as they pass between the head of humerus (greater tuberosity) and the lateral aspect of the acromion usually happens with the proximal migration of humeral head. It is the most common disorder of shoulder, accounting for 44–65% of all complaints of shoulder pain during a physician's office visit.

-Mechanism: most of the time it is a combo between the 2

1- Subacromial contents gets bigger like in subacromial bursitis and supraspinatus tendonitis.

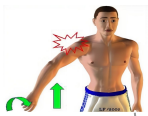
Or

2- The space gets smaller; e.g osteoarthritis (because of spurs/osteophytes), the acromion becomes curved or hypertrophy of greater tuberosity.

Risk Factors

1. Age: over 40y.
2. Overhead activity e.g. lifting, swimming, tennis, combing hair, wearing.
3. Bursitis and supraspinatus tendinitis: Inflammation—> swelling—-> space narrowing.
4. Acromial shape: type II (curved) & III (hooked).
5. AC joint arthritis or osteophytes may result in impingement and mechanical irritation to the rotator cuff tendons.

Symptoms



- **Pain in the acromial area** (may radiate to the neck) when the arm is flexed and internally rotated → Inability to use the **overhead position** (difficulty in taking clothes off, hair brushing, putting things in high places).
- Pain could be due to Subacromial bursitis or rotator cuff tendinitis.
- Worse at night as the subacromial bursa becomes hyperemic after a day of activity.
- Pain when sleeping on the affected side.
- After time, the pain decreases ROM especially abduction and forward flexion.
- Weakness.

DDx

- Rotator cuff tear: leads to proximal migration of the humerus. they have similar presentation.
- Calcific tendinitis: Unknown why it happens.
- Biceps tendinitis.
- Cervical radiculopathy.
- AC joint arthritis.
- Glenohumeral osteoarthritis.
- Glenohumeral instability: dislocation or subluxation, usually present as vague pain.

1-Means انحشار.

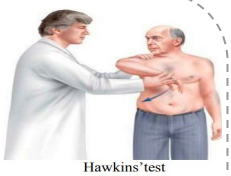
Diagnosis

- History: Pt comes with pain upon doing overhead activities and pain in lateral aspect of arm.
- Physical Examination.
- Imaging.

Physical examination

- ↓ ROM in Internal rotation "IR" & Abduction "ABD".
- Weakness in flexion and external rotation.
- Pain on resisted abduction and external rotation.
- Pain on "Impingement tests".


Neer's test


Hawkins'test

➔ ***Impingement tests:*** negative results doesn't rule out it and positive doesn't specify it

- **Neer's impingement test:** (not specific because it can be positive with other) passive elevation of the internally rotated arm in the sagittal plane (shoulder forward flexion). Test is positive if he felt pain.
- **Hawkins' impingement test:** (more sensitive) with the elbow flexed to 90 degrees, the shoulder passively flexed to 90 degrees and internally rotated. Test is positive if he felt pain.

Radiological Findings

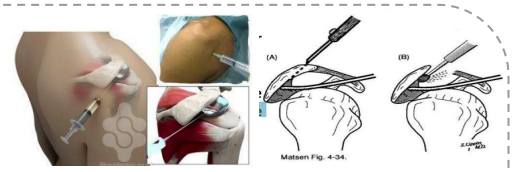
Supraspinatus outlet view

Plain x-ray, mostly normal but you may find:

- Acromial spurs: small osteophytes due to stress.
- AC joint osteophytes.
- Subacromial sclerosis.
- Greater tuberosity cyst is common (clear on MRI).
- Greater tuberosity hypertrophy.
- There is a special view on x-ray called **supraspinatus outlet view,** it gives exactly the shape of acromion but we don't use it very often with the presence of MRI.

MRI (Best test): To confirm dx and rule out rotator cuff tear.

Management



Conservative treatment: Always start with it.

- Avoid painful activities → especially overhead activities.
- NSAIDs: To decrease the inflammation.
- Physiotherapy: if doesn't work we give steroid injections
  1- Stretching and range of motion exercises. 2- Strengthening exercises.
- Subacromial space steroid injection: very potent but should be under aseptic technique to avoid infections. May increase blood sugar for 2-3 days in diabetics. May cause pain for 2-3 days. If injected multiple times it will weaken the tendons which might lead to rotator cuff tears so we maximally inject it twice.

Operative treatment: Indicated if there is no improvement **after 6 months** of conservative treatment

- Goal → remove the impingement and create more subacromial space for rotator cuff.
- Open (Called: Acromioplasty) or arthroscopic technique (Called: subacromial decompression).
- The anterolateral edge of the acromion is removed.
- At the same time we do bursectomy.
- Success rate 70-90%.

Rotator Cuff Pathology

Functions Of Rotator Cuff Muscles

- Keep the humeral head centered on the glenoid regardless of the arm's position in space.
- Generally work to depress the humeral head while powerful deltoid contracts.

Rotator Cuff (RC) Tear

- A large tear in the tendon of one or more muscles in the rotator cuff. Often results from progression of smaller tears and inflammation; may be degenerative or traumatic in nature.

Causes of rotator cuff tear

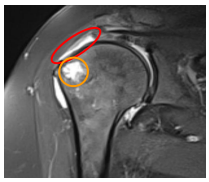
Intrinsic factors	Extrinsic factors	Traumatic
- Vascular : Musculotendinous junction has poor vascularity 15:15 - Degenerative : (wear and tear) Age-related and overuse	1- Repetitive use. 2- Impingement : • Acromial spurs. • AC joint osteophytes. not treated > chronic impingement > cuff tear	-e.g. a simple fall (FOOSH) or trying to catch or lift a heavy object. -If a patient >40 presents with shoulder dislocations DO MRI!!, labrum in elderly is often stronger than rotator cuff muscles. (important in life and exam). -Hx of trauma + not able to move >> high suspicion of rotator cuff injury
Presentaion		
similar to impingement; mild progressive pain, pain when sleeping on the affected side, decrease range of motion. There is no specific incident after which the symptoms started.		Acute or sudden symptoms and can specify an incident, after which, the symptoms started such as falling

Diagnosis

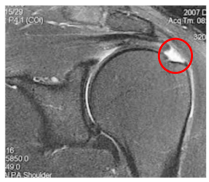
- History (Wide range of symptoms: range from inability to lift the arm to minimal pain) and Physical Exam (Do first impingement tests bc it's more common, then test rotator cuff muscles).
- X-rays to check if there is any impingement signs (acromial spurs....etc). Findings could be normal.
- Ultrasound; Highly operator dependent, Does not provide information regarding concomitant pathologies.
- MRI (Sensitivity of 84% and a specificity of 96%): Best for RC evaluation, **MODALITY of choice**. (CI in claustrophobic, metals in sensitive locations or pacemakers and its relatively CI in pregnancy).

Wide Spectrum

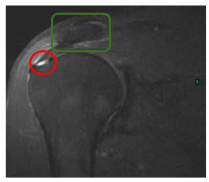
- Partial thickness about 3mm.
- Full thickness:
  - Small about 1cm.
  - Medium 1-3 cm.
  - Large about 4cm.
  - Massive.


Full thickness large with a cyst


Full thickness large


Full thickness small

Tendinitis


Partial[1]

1- Partial thickness RC tears has 3 types based on location: articular side, bursal side, and intratendinous. The one in the pic is articular side.

Treatment: Non-Operative

➢ **Indications:**
- All partial thickness tears.
- Full thickness tear esp small type:
  - Chronic + degenerative.
  - Elderly low demanding + not active.

Degenerative & Young ??? **OPERATIVE**

➢ **Modalities**: same as impingement
- Activity modification.
- NSAIDs.
- Physical Therapy:
  1- Range of motion.
  2- Strengthening of the rotator cuff and periscapular musculature.
- Steroid injections.

Treatment: Operative

- Indications:
- ❖ **All Acute** traumatic tears regardless of the age. It has to be treated urgently, why? with time, the muscle will shrink and retract (it might reach the level of glenoid if left long enough) so you won't be able to pull it back and reattach it, also there will be fatty infiltration (the muscle would have fatty texture making it hard to anchor a suture in it). This happens only in the traumatic type.
- ❖ Failed non-operative treatment within 6 months.
- ❖ Full thickness tear:
  - ➢ Active, young, painful. if young regardless of the cause do surgery.
  - ➢ Old but active.

-Options:
- ❖ Rotator cuff repair (Arthroscopic or open).
- ❖ +/- Subacromial decompression (SAD) (done especially with the degenerative type).

-Complications of surgery:
- ❖ Not improving, stiffness, and re-rupture (esp if we repair a retracted muscle or pt lifted heavy object within recovery period).

Natural History

- **If not treated** → rotator cuff muscles retract progressively further and further → chronic pain and loss of motion and with time becomes irreparable → deltoid will pull the humerus up to the acromion (called proximal migration of humerus) → rotator cuff arthropathy which is arthritis between humeral head and acromion (unusual place) due the tear. Treated by reverse shoulder replacement الكتف الصناعي المقلوب.

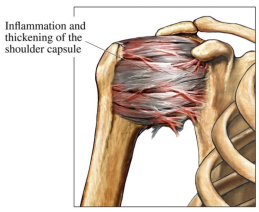
Deltoid pulls humeral head and causes arthritis in an unusual position called rotator cuff arthropathy.

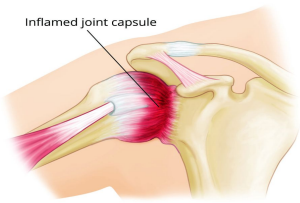
Adhesive Capsulitis

مقطع سواه الدكتور عن الموضوع

Scenario : DM pt with severe shoulder pain and limited range of motion in all directions.

- Inflammation of capsules with adhesion.
- Also called "frozen shoulder" which is not specific term and should not be used.
- Usually self-limiting (typically begins gradually, worsens over time and then resolves but may take 2-4 years to resolve).
- 10 % is bilateral.
- More common in females.
- The cause is unknown.





Risk Factors

1. **DM** (esp. insulin dependent) (Most common RF by far) (أول سؤال يطلع ببالك للمريض : "عندك سكر؟").
2. Hypo and Hyperthyroidism.
3. Following injury or surgery to the shoulder (Called secondary adhesive capsulitis).
4. Hyperlipidemia.

Mechanism (Extra)

- ❖ Primary adhesive capsulitis: Idiopathic, usually associated with DM. Usually resolves spontaneously in 9-18 months.
- ❖ Secondary adhesive capsulitis → Due to prolonged immobilization or previous shoulder surgery, poorer outcomes.
- ❖ Shoulder-hand syndrome: CRPS/RSD14 characterized by arm and shoulder pain, decreased motion, and diffuse swelling → following MI, stroke, shoulder trauma.

Symptoms

- It is characterized by pain and restriction of **all** shoulder movements (ROM) **actively AND passively** "global stiffness" due to fibrosis and contracture of the capsule. (impingement syndrome has restricted IR & ABD).
- Pain so severe, worse at night and often prevents sleeping on affected side.

Stages

1. Pain (**freezing stage**):
   The hardest stage because it's very painful.
   Pain+++/ Hot++, ROM mildly limited, duration: 3-9 Months.
2. Stiffness (**frozen stage**):
   Pain decreases, ROM more restricted, duration: 4-12 Months.
3. Resolution (**thawing stage**):
   Slow improvement in ROM, duration: 12-42 Months.

Investigations

- The diagnosis of adhesive capsulitis is often one of **exclusion;** we have to rule out other pathologies.
- **Mainly clinical diagnosis:** all movements are restricted both actively and passively.
- Most of the time **normal investigations:**
  - X-rays: b/c pt can't use shoulder there will be disuse osteopenia or mainly no findings.
  - MRI: thickening of the joint capsule and diminished filling of the axillary pouch.
  - There are the findings of adhesive capsulitis. However, not always seen.

Treatment

- If untreated, it would resolve spontaneously over 2-4 years.
- Pain relief and anti-inflammatory medications: it breaks the adhesions.
- Aggressive Physiotherapy (most imp).
- Steroid injections.

❖ If not improved:

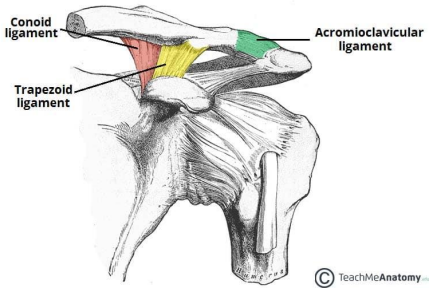
- Manipulation under anesthesia (MUA): you break the adhesions by manipulation, but there's risk of bone fractures.
- Arthroscopic capsular release: Best treatment (if 6 months of physiotherapy and steroid injection failed).

Summary: Physiotherapy for 3m if not improved give steroids injection not improved so go to surgery

Acromioclavicular Pathology

Anatomy

➢ The AC joint is different from joints like the knee or ankle, because it doesn't need to move very much (it has minimal motion). The AC joint only needs to be flexible enough for the shoulder to move freely. The AC joint just shifts a bit as the shoulder moves.
➢ The joint is stabilized by the AC ligament and the 2 coracoclavicular (CC) ligaments: Coracoid ligament and Trapezius ligament.

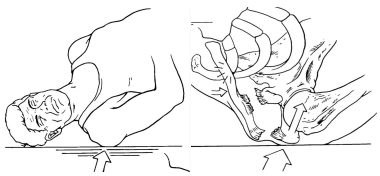
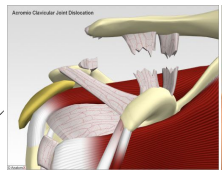


AC Joint Common Conditions

- Traumatic AC joint separation/dislocation.
- Osteoarthritis.
- Osteolysis of distal clavicle.

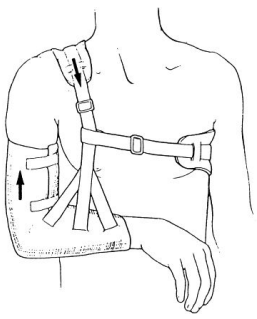
Traumatic AC Joint Separation/Dislocation

Mechanism: Almost always a direct blow or fall onto acromion (so FOOSH can't cause it)
يكثر في الدول اللي تستخدم السيكل
The patient will complain of pain and something protruding (deformity)

Treatment

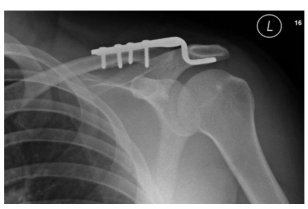
- Partial dislocation: Conservative.



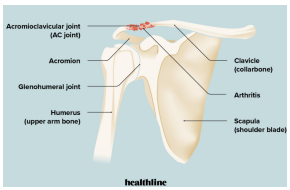

- Complete dislocation: Surgery.
  - Number of different approaches involving AC/CC ligament reconstruction (tightrope) or screw/hook plate insertion.

Tightrope

Hook plate



AC Joint Arthritis



- Arthritis is a condition characterized by loss of cartilage in the joint, which is essentially wear and tear of the smooth cartilage which allows the bones to move smoothly.
- Motions which aggravate arthritis at the AC joint include reaching across the body toward the other arm.

Causes

- Degenerative osteoarthritis; wear and tear in old aged people (the most common).
- Rheumatoid Arthritis.
- Gouty Arthritis.
- Septic Arthritis.
- Atraumatic distal clavicle osteolysis in weight lifters; result of repeated movements that wear away the cartilage.
- Post-traumatic osteolysis of lateral end of clavicle such as like dislocation or a fracture.

Signs and Symptoms

- Progressive pain, Which worsens with movement.
- The patient may suffer a night pain which is a sign of arthritis.
- It is commonly associated with impingement syndrome.

Diagnosis

- Clinical: tenderness over the AC joint.
- X-rays: Since it is rare to have isolated AC joint arthritis, sometimes we do MRI to rule out other diseases, except in the distal clavicle osteolysis will be isolated.

ACJ osteoarthritis with osteophyte

Distal clavicle osteolysis

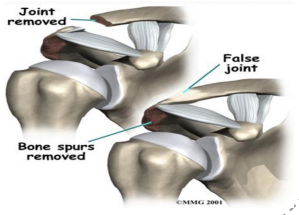
Treatment

Non-Surgical treatment (start)**:**

- Modify activities: rest, avoid weightlifting and push-up.
- Pain medications and NSAID to reduce pain and inflammation.
- Physiotherapy.
- Steroid injection.

Surgical treatment: if conservative fails

- We excise 1cm from the lateral clavicle, can be done open or arthroscopic. Do not exceed 1cm bc you will reach the CC ligaments and cause separation/dislocation.



Distal Clavicle Osteolysis

- This disease only affect **weight lifters**, but it doesn't mean every weight lifter will get it, only a small portion.
- Normally stress causes arthritis, but here the body reacted by causing resorption (osteolysis) as you can see in the pic.
- What is the treatment?
  - Stop weight lifting.
- What if they didn't stop? They will come back due to pain and we would give steroid injection but they will come back again, what do we do? The lateral part of clavicle is being actively resorbed, so we excise this part and even a bit further to the non-active area in order to stop it. This procedure has 50% success rate.

Shoulder dislocation

Acute dislocation is a surgical emergency and demands urgent relocation. (While RC tear is urgent).

- The shoulder joint has the greatest ROM among the body's joints.
- It relies on soft-tissue restraints (including the capsule, ligaments, and musculature) for stability (lacks bony stability).
- Therefore, it is the most frequently dislocated joint followed by elbow.

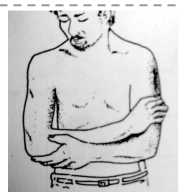
Classification

Atraumatic (AMBRI)	Traumatic (TUBS) — We are going to talk about this type in the lecture
- Multidirectional instability. - Generalized ligamentous laxity, they would live normally but have higher risk of shoulder and knee dislocation. more common in girls. - Bilateral. - Habitual. - Responds well to nonsurgical management (physiotherapy, we rarely operate on them).	- 96%. - Such as FOOSH injury. - Unidirectional. - Further classified by the direction of the humeral head dislocation: 1- Anterior. {commonest >95%} 2- posterior < 4%, caused by 3 Es: A- Electrical shock. B- Epilepsy. C- Ethanol (alcohol). 3-inferior. < 1%

Anterior Shoulder Dislocation

- Actually it is anterior inferior.
- **Mechanism:**
  a. Usually Indirect fall on Abducted and extended shoulder (external rotation).
  b. May be direct when there is a blow on the shoulder from behind humerus pushed anteriorly.
- Maybe associated with: explained in slide 14
  a. **Bankart's lesion.**
  b. **Bony bankart lesion.**
  c. Hill-sachs lesion.

Clinical Picture


- Patient is in severe pain they come screaming.
- Holds the injured limb with other hand close to the trunk.
- The shoulder is abducted and the elbow is kept flexed.
- There is loss of the normal shoulder contour may appear as a step, very clear when pt is exposed.
- Anterior bulge of head of humerus may be visible or palpable If pt thin.
- A gap can be palpated above the dislocated head of the humerus.

Investigations

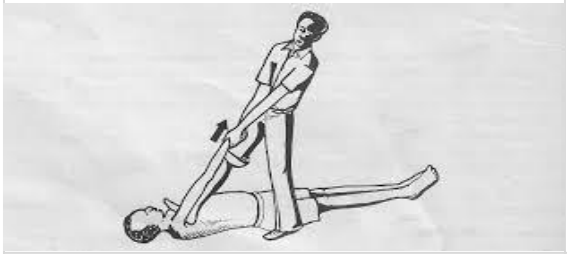
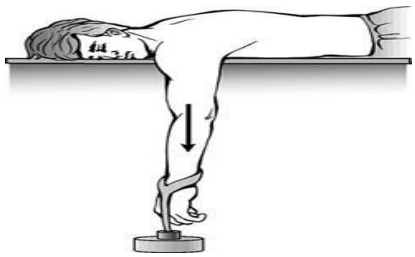
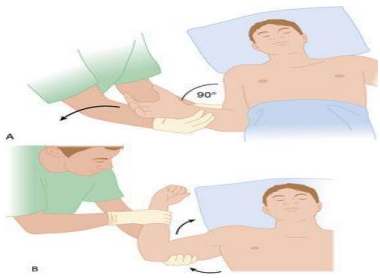
- It is hard to do physical examination when the patient in pain.
- You must obtain AP, Lateral (we have 2 lateral views: trans-scapular (right pic) & more importantly axillary view).

Management of Anterior Shoulder Dislocation

- Is an Emergency.
- It should be reduced in less than 24 hours or there may be Avascular Necrosis of head of humerus.
- If you try in the ER and you cannot do it, take the patient to OR under GA.
- Following reduction, the shoulder should be immobilized strapped (sling) to the trunk for 3-4 weeks and rested in a collar and cuff. (sling then physiotherapy)
- In ER N/V exam → closed reduction (traction and counter traction) → NVE → X-ray to make sure it is in place → immobilization → ortho clinic → MRI (can wait few weeks but if patient >40y/o we have to be urgent about it to rule out RC tear).
- If 1st time then we immobilize with sling for 3-4 weeks then physiotherapy, unless pt is under 20y/o and active we have to do surgery due to high recurrence rate (90%). The older they get the less likely they redislocate.
- If patient dislocated 2nd time we do surgery.

most common way is traction/counter traction

★ Methods of reduction of Anterior Shoulder Dislocation:

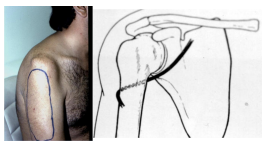
Hippocrates Method	Stimpson's technique	Kocher's technique
A form of anesthesia or pain abolishing is required	Some sedation and analgesia are used but No anesthesia is required	It is the method used in hospitals under general anesthesia and muscle relaxation. This is what we nowadays do with conscious sedation.
- Put your foot in axilla to counteract and pull the elbow. - An old way used now by soldiers in wars. -الطريقة هذي تنفع في الزنقات.	- There is a lot of spasm in muscles after dislocation bc of pain which makes the reduction harder. - This technique need strong analgesia (midazolam..). - Put pt in Prone position and put Axillary pad to prevent brachial plexus strain put traction (3-4 kg or less) leave him (15-20 min) until muscles relaxes, most of the time it reduces by itself.	- Efficient and quick technique. - The dislocation in this case is inferior internal. - We need good muscle relaxation and good analgesia. -How to reduce? Exaggerate the deformity by Applying traction 'pull the arm down' then hold arm and do external rotation then push up and internal rotation. (Need someone to support the axilla)
		

❖ Post-reduction rehabilitation:
- ➢ The goal of rehabilitation is to regain maximum ROM while retaining stability.
- ➢ The affected arm can be immobilized for 3 weeks, and limited physical rehabilitation is recommended.

Complications of Anterior Shoulder Dislocation

Early:

1. Injury to the neurovascular bundle in axilla, the most common is Axillary Nerve Injury:
   - It is a branch from posterior cord of Brachial plexus, It is sensory and motor so, you have to examine both, sometimes only the sensory part is affected or only the motor part is !!!!
   - It hooks close round neck of humerus from posterior to anterior.
   - It pierces the deep surface of deltoid (abduct the shoulder) and supply it and the part of skin over it.
   - Mostly "Neuropraxia" and usually resolves with time.

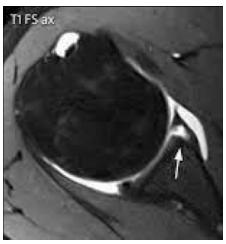
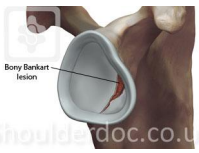
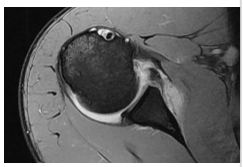


2. Associated Fracture such as humeral neck or LT or GT (the one in the pic).
3. Rotator cuff rupture: If a patient >40 presents with shoulder dislocations DO MRI!!, labrum in elderly is often stronger than rotator cuff muscles. (important in life and exam).

Late:

1. Avascular necrosis of the head of the Humerus (high risk with delayed reduction).
2. Recurrent shoulder dislocations (most imp one), the younger the pt the more common.

Associated lesions

Bankart's lesions	Bony Bankart's lesions	Hill-Sachs lesion
- Detachment/avulsion of the anteroinferior labrum from the glenoid, **seen on MRI**. present in 80-90%. - We reattach it with surgery. - If it was posterior shoulder dislocation we call it reverse bankart.	Avulsion of the anterior rim of the glenoid bone. present in up to 49%	An impaction injury in the posterosuperior humeral head secondary to forceful contact with the glenoid rim during the acute dislocation. Present in 80%
 	  	 

If there is a big bony Bankart and big Hill-Sachs lesion, the patient needs Latarjet procedure (transfer of the coracoid into the glenoid rim)

Important questions read them!

45y lady with Shoulder Pain with overhead activity and limited abduction = rotator cuff and impingement

Q: a 20y old male who was involved in car accident and was unable to abduct- MRI shows tear of supraspinatus? SURGERY

Q: What is the treatment for 50y old have chronic pain for long time and unable to abduct? Conservative

Q: Overhead activity? limited abduction? think about impingement and rotator cuff tear

Extra

-The anatomy in the shoulder is very special. In joints we have two things to consider: Range of motion and Stability, you have to sacrifice one to gain the other. E.g. the ROM in the knee is limited only in one axis: flexion and extension (although it's not very accurate to say one axis), but the shoulder has many axes: abduction, adduction, forward flexion, extension, internal rotation, external rotation and circumduction (basically all kind of ROM).

-Stability is either by static structure (always there, always acting the same way) or dynamic:
Stability by static structures can be due to:

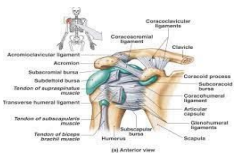
1-Bony structures:

- Humerus: humeral head is big, glenoid is wide (humerus head is much bigger than glenoid) which can give you some stability but not the best, unlike the femoral head which is more like spherical in shape and the acetabulum is covering most of it (Ball in socket) → more stability, It typically takes a major force or trauma to dislocate the hip (It's a big issue to have someone with hip dislocation!)
- Scapula (Glenoid, Acromion, Coracoid, scapular body)
- Clavicle.
- Sternum.

Shoulder injury can be caused by low energy trauma; certain movements can get your shoulder out.

2-Ligaments:
The shoulders' ligaments are not as big and strong as the knees' ligaments, why? The ligaments in the shoulder are just thickening of the capsule.
There are 3 anterior ligaments which prevent anterior dislocations: superior, middle and inferior Glenohumeral ligaments. You have to keep in mind! 95 % of the dislocations are anterior (commonly anterior- inferior), while the posterior is extremely rare and only certain traumas will cause the inferior, posterior or superior dislocations.
The anterior dislocation happens when the shoulder is abducted in 90 degree and externally rotated, So in this position Which one of the three ligaments has more chance to get strained and also **affected in the anterior dislocation? inferior glenohumeral ligaments.**
↪ How? While abducting and externally rotating the shoulder → the inferior ligament will be more stretched, but the ligaments are stronger than any structures although they are only thickening but still they are the strongest → they will not tear but they will detach the labrum from the bone when there is dislocation. Very IMP I WILL ASK YOU ABOUT IT!! (What is called? What will you see in MRI? **BANKART LESION;** There is detachment of the anteroinferior labrum from the underlying glenoid.
Labrum is fibrocartilage that gives the depth for accommodating the head also the ligaments attach to it.

○ Acromion is very important, it's a landmark for almost everything you do around the shoulder, and it's supporting the rotator cuff and acts like a roof over the glenohumeral joint. In case of abduction if there was a proximal migration what will happen? The GT will hit the acromion → supraspinatus impingement.
○ Coracoid is attached to the conjoint tendon (tendons of short head of biceps and coracobrachialis).
○ Long head of biceps is a trouble maker passes through the bicipital groove and attached to superior labrum (doesn't attach to bone which is unusual) with age there will be pulling out and detachment of labrum which is called (superior labrum anterior posterior (SLAP) lesion) or becomes inflamed in old people, very common problem in the West (white ethnicity) but here it is not bad as in the West, what do we do for them? (Shoulder arthroscopy).
In women base of thumb is usually affected by instability then arthritis in the West, here is not that common.



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