ORTHOPAEDICS AND TRAUMA

LECTURE NOTES

FOR 5th-YEAR MEDICAL STUDENTS

PROFESSOR FREIH ODEH ABU HASSAN

Department of Orthopaedic Surgery

The University of Jordan-Amman

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E-mail: daralfurqan@gmail.com

9 789957 507558

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FREIH ODEH ABU HASSAN,
FRCS (London), FRCS (TR. & ORTH). London
PROFESSOR OF ORTHOPAEDIC AND TRAUMA SURGERY
JORDAN UNIVERSITY HOSPITAL AND MEDICAL SCHOOL
AMMAN – JORDAN

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الموضوع: Orthopaedics and Trauma diseases

- **أعدت دائرة المكتبة الوطنية بيانات الفهرسة والتصنيف الأولية**

PREFACE

For the last 35 years, I noticed the common concern of medical students attending their training in our department regards the book they should read for the orthopaedics syllabus.

Unfortunately, we did find most students reading from written notes contain enormous common facts mistakes.

This led me to write these lecture notes to overcome their concern.

This synopsis aims to provide a theoretical basis in general orthopaedics and trauma for 5^{th}-year medical students.

Each chapter has been written by the author, depending on many sources. It covers the list of topics that have been taught to our medical students yearly at the University of Jordan.

This explains the course syllabus from Introduction to orthopaedics surgery to various topics in orthopaedics.

Each lecture is described in a simple, easy way to enable the student to understand and remember the written knowledge.

I hope these notes fuel you with the best knowledge in orthopaedics and help you deliver a very successful outcome at the end of the year.

Freih Abu Hassan
Amman 2023

5th-year Medical Students Course Syllabus
Special Surgery/ Orthopaedics
The Universities of Jordan

1-INTRODUCTION TO ORTHOPAEDICS

A. Osteology

The skeleton has 206 bones: axial skeleton 80 & appendicular skeleton 126.

1=Components of bone:

- ***Compact bone*:** shaft of the bone (diaphysis).
- ***Cancellous bone****:* end of the bone (epiphysis-metaphysis).
- ***Periosteum*:** covering of bones.
  - Outer fibrous layer → attachment of tendons and ligaments.
  - Inner layer: contains osteoblasts.
- ***Endosteum*:** membrane covers the marrow cavity of long bones
- ***The epiphyseal plate*** separates the epiphysis from the diaphysis and is the center for longitudinal growth in children.

2=Bone Composition

i- ***Bone cells*** *10%:* Osteoblasts, Osteocytes, and Osteoclasts

ii- ***Matrix*** (98% collagen) is a framework for the deposition of mineral salts.

40% **organic matrix (**collagen)

60% **inorganic matrix** (hydroxyapatite), a compound of calcium and phosphate.

iii- ***Mineral salts*** (insoluble calcium & phosphate complex called OH apatite).

3= Types of bone ossification

I-1ry Ossification:

Intramembranous: Woven bone formed directly e.g. clavicle.

*** *Enchondral***: a cartilaginous model of bone is 1st made by chondrocytes → cartilage reabsorbed and replaced by bone, e.g., most bones.

II- Secondary ossification centers usually develop in the periphery of bones.

III- Heterotopic ossification: formation of bone in extra-skeletal location.

5-Types of bones

1- **Long bones**
   The epiphysis (growing end), then the epiphyseal plate, or physis followed by metaphysis and the diaphysis, e.g., humerus.
2- **Flat bones:** Pelvis
3- **Short bones:** tarsus and carpus, they do not have epiphyses.
4- **Accessory ossicles.**
5- **Sesamoid bones.**

4=Anatomy of the immature long bone.

•**Epiphysis** (proximal and distal ends of the bone)
•**Physis** (growth plate): site of longitudinal bone growth
•**Metaphysis**: below the growth plate
•**Diaphysis**: shaft of the bone.
•**Medullary cavity**: contain B marrow

•**Endosteum:**
•**Articular surface**: intraarticular bone ends
•**Periosteum**:
•**Apophyses:** for tendon attachment**,** no rule in the length of the bone. e.g. G & lesser trochanters, olecranon, tibial tubercle & the calcaneum

2-Joints

1- Types of joints

A-Synovial joints: (freely movable)**.**

1- Ball and socket joints: hip, shoulder. **2- Multiaxial**: Shoulder (in all planes).
3- Hinge joint: knee (one plane). **4- Condyloid joints**: radiocarpal (two planes).
5-Pivot or Peg joints: superior radioulnar (one axis only).
6-Plane joints: sacroiliac joint.
7-Sliding joint: Facet joints
8-Saddle-shaped: first carpometacarpal joint.

Components of diarthrotic (synovial) Joints.

A- Joint capsule B-Synovial membrane C-Bursae

B-Cartilage joints: Symphysis Pubis, intervertebral discs.

C-Fibrous joints: the inferior tibiofibular joint is the largest syndesmosis in the body

2- Types of Movements

***Movement*:** a motion, (a change in position).
1-Flexion: bending a body part or decreasing the angle between the parts.
2-Extension: straightening a body part to increase the angle between the parts.
3-Adduction: moving a part of the body towards the midline.
4-Supination: turning the hand, so the palm faces upwards.
5-Pronation: turning the hand, so the palm faces downwards.
6-Inversion: turning the foot so that the sole faces inwards.
7-Eversion: turning the foot so that the sole faces outwards.
8-Rotation: turning the body part on the axis.
9-Circumduction: a combination of movements that results in a cone-like movement (e.g., arm circles).
10-Varus: inward angulation of the distal segment of a bone towards the midline.
11-Valgus: outward angulation of the distal segment of a bone away from the Midline.

2-CLINICAL ANATOMY

Upper limb Anatomy

1-The Skeleton.

2-Anatomy of the shoulder complex.

Shoulder girdle (Pectoral girdle).

Function	Connecting the upper limbs to the axial skeleton
Bones	Scapulae, clavicles, manubrium of the sternum
Joints	Acromioclavicular Joint Sternoclavicular Joint Glenohumeral Joint Scapulothoracic joint Pseudo joint between the humerus and the Coracoacromial arch

The shoulder girdle consists of (5 bones and four joints).

1. Glenoid cavity

- 1/6 of a sphere, with labrum, will be 1/4 sphere.
- Its rim is surrounded by fibrocartilaginous thickening 9mm (the labrum)
  = Deepen the socket
  = Anchor the inferior glenohumeral ligament complex.

2. Humeral head: Normally retroverted an average of 30°

3. Glenohumeral joint (ROM = 120°)

= Ball-and-socket joint, stabilized by both static and dynamic restraints.

- **Static:** -Joint surfaces -Capsulolabral complex -Ligaments.
- **Dynamic:** -Rotator cuff -Scapular stabilizing muscles

= **Glenohumeral ligaments**

Capsular thickenings act as a kind of rein to limit excessive rotation or translation of the humeral head.

1- Superior glenohumeral ligament (SGHL),
2- Coracohumeral ligament,
3- Middle glenohumeral ligament,
4- Inferior glenohumeral ligament complex.

= The rotator Cuff interval,

Composed of the Coracohumeral ligament, Superior glenohumeral lig. (SGHL), and capsular fibers, which all blend along with insertions medial and lateral to the bicipital groove, maintaining the biceps tendon within a groove. It is a triangular anatomic shape, that lies between the supraspinatus & subscapularis tendons.

4-Acromioclavicular joint.

Stabilized by (conoid & trapezoid)

- Acromioclavicular ligaments →resist Antero- post. Translation.
- Coracoclavicular ligaments → prevent inferior translation of the coracoid and acromion from the clavicle.

5-Scapulothoracic joint (ROM = 60°)
Upward and downward rotation of the scapula
The ratio of glenohumeral to scapulothoracic motion during shoulder abduction is approximately 2°:1° (for every 2° ROM of the Glenohumeral Joint, the scapulothoracic joint will move 1°) they move together.

Arm abduction 180° (120° glenohumeral joint, 60° scapular rotation)

- From 0-30°, comes almost entirely from the glenohumeral joint.
- From 0-90° →(60° glenohumeral, 30° scapular rotation)
- From 90-180° →(30° is glenohumeral, 60° scapular motion)

As the arm comes up more and more rotation comes from scapular movement.

The rotator cuff anatomy

The rotator cuff is a sheet of conjoint tendons closely applied over the top of the outer capsule, that mainly acts as a stabilizer of the shoulder.

Rotator cuff, SItS (small t is for teres minor)

Muscle	Action	Pathology
Supraspinatus	Abducts arm initially 1st 15°	Tear, degeneration, impingement ***-Empty full can test***
Infraspinatus	External R	Pitching injury
Teres minor	External R & Adducts arm	
Subscapularis	Internal R & adducts arm	

Movements.

Arm abductors

Degree	Muscle	Nerve	Joint
0°–15°	Supraspinatus	Suprascapular N	Shoulder
15°–90°	Deltoid	Axillary N	Shoulder (main muscle of the shoulder)
> 90°	Trapezius	Accessory N	Scapulothoracic
> 100°	Serratus Anterior	Long Thoracic N	Scapulothoracic

3-Elbow
4-Forearm
5-Wrist
6-Carpal bones

Lower Limb anatomy

A-Femur

- The upper end consists of the head, neck, greater and lesser trochanters.
- The head forms roughly 2/3 of a sphere.
- The shaft of the femur is slightly twisted and curved with convexity forward.
- The neck extends inferior-laterally from the head to meet the shaft of the

femur at an angle of about 125° ***(<120°: Coxa vara >135°: Coxa valgus)***

- First long bone to ossify in the cartilage at 7th week of fetal life.

Femur blood supply (trochanteric anastomosis → retinacular arteries)

1*-Superior gluteal A.* ***2****-Inferior gluteal A.* ***3****-Medial circumflex femoral A.* ***4****-Lateral circumflex femoral artery.*

- Retinacular arteries supply the neck of the femur and the head.
- The artery of ligamentum teres supplies blood to the head of the femur.

Actions of hip muscles

ACTION	MUSCLES
Abductors	Gluteus medius, gluteus minimus
Adductors	Adductor magnus, adductor longus, adductor brevis
Extensors	Gluteus maximus, semitendinosus, semimembranosus
Flexors	Iliopsoas, rectus femoris, tensor fascia lata, pectineus, sartorius
Internal rotation	Gluteus medius, gluteus minimus, tensor fascia latae
External rotation	Iliopsoas, gluteus maximus, piriformis, obturator

A-Proximal femur fractures

1- **Extracapsular fracture**: intertrochanteric, subtrochanteric fracture s (union is good)
2- **Intracapsular fracture**: neck of femur fracture.

1- **Subcapital fracture**: fracture very close to the head of the femur. Common in elderly because the spongy bone of the neck is atrophic and the cortical bone is thinner.

2-Transcervical **fracture:** near the midpoint of the neck.

3- Basal neck **fracture:** very close to the shaft.

Complications → AVN, Non-union in 1+2.

B-The Patella.

Patella is the **largest sesamoid bone** in the body.
Lies within the tendon of the quadriceps femoris.
The stability of the patella is maintained by
1= Superiorly by quadriceps. 2 = Inferiorly by a patellar ligament.
3= Medially by horizontal fibers of vastus medialis.
4= Laterally by prominent lateral condyle of the femur.

C-The Knee Joint

The knee joint is a hinge joint during flexion-extension but in a flexed position modifications enable axial rotation around a central pivot.

Important knee structures.

= 2 menisci = 2 cruciate ligaments = 2 collateral ligaments = 4 bones

I-Knee ligaments.:

1-Collateral Ligaments, on the sides of the knee, controlling the sideways motion of the knee and braces it against unusual movement.

- Medial collateral ligament (MCL) on the inside
- Lateral collateral ligament (LCL) on the outside

2-Cruciate Ligaments, located inside your knee joint, control the back-and-forth motion of the knee.

Running diagonally in the middle of the knee.

- The anterior cruciate ligament (ACL) prevents the shinbone (tibia) from sliding out in front of the thighbone (femur) and providing rotational stability.
- The posterior cruciate ligament (PCL) mirrors the ACL but is attached to the back of the knee, crossing the ACL in an X shape.

II- Knee Menisci

Semilunar cartilages, act as shock absorbers and stabilize the knee**.** They work with the cartilage to reduce stress between the tibia and the femur. Peripheral thirds of the menisci are vascular and can be repaired; the inner two-thirds are nourished by synovial fluid.

	Medial Meniscus	**Lateral Meniscus**
Shape	C-shape	Circular
Size	Large & less mobile	Small & more mobile
Attachment	Adheres to Med collateral ligament	Adheres to Popliteus tendon
Injury	Commonly injured	Less injured
Meniscal cyst	No meniscal cyst	Associated with meniscus cyst and Discoid meniscus

The lateral meniscus is associated with meniscal cysts and discoid menisci and is the most common site of tears in acute injuries to ACL.

D-The Ankle Joint.

The ankle is a hinge joint composed of the fibula, tibia, and talus articulations along with several important ligaments.

The anterior portion of the talus is wider than the posterior portion.

The tibial plafond is also wider anteriorly to accommodate the shape of the talus.

The deltoid ligament supports the medial aspect of the ankle.

The distal fibula is attached to the distal tibia via soft-tissue ligamentous constraint is known as **syndesmosis** (fibrous joint).

The syndesmosis is composed of

A-Anterior inferior tibiofibular, **B-**Posterior inferior tibiofibular,
C-Transverse tibiofibular **D-**Interosseous ligaments.

The fibular collateral ligaments

- Anterior talofibular ligament (ATFL),
- Posterior talofibular ligament, and

- Calcaneofibular ligament

E- The Foot: hindfoot, midfoot, and forefoot.

F-The Muscles of the limbs.
Agonists and Antagonists
Muscles work as balanced groups which oppose each other. If the flexor muscles work more strongly than the extensors, as they do in cerebral palsy, a flexion deformity develops. If the femoral neck is broken, the Iliopsoas cannot act on the hip but spins the femur about its long axis to produce the characteristic external rotation deformity.

Compartments: Muscles contained within fascial compartments.
Locations of compartments in the limbs.
Two in the arm: anterior and posterior.
Three in the forearm: dorsal, superficial volar, deep volar
Four in the leg: anterior, lateral, superficial posterior, and deep posterior
Three in the thigh: anterior, medial, lateral
Ten in the hand plus each finger
Nine in the foot

Forearm.
1- *Ventral compartment*. The ventral compartment includes the median and ulnar nerves and the radial and ulnar arteries.
2- *Dorsal compartment*. includes the posterior interosseous nerve

Lower limb.
1- *Anterior tibial compartment*. (deep peroneal N).
2- *Superficial posterior compartment*. (Gastrocnemius and soleus only).
3- *Deep posterior compartment*. Posterior tibial VAN & the peroneal A.
4- *Lateral (peroneal) compartment*. (Superficial peroneal nerve).

3-PRINCIPLES OF FRACTURES

1- Definition
Fracture (#): break in the structural continuity of the bone.
Dislocation: complete loss of contact between the joint surfaces.
Subluxation: partial loss of contact between the joint surfaces.

Causes of Musculoskeletal Injuries
* Blunt tissue trauma
* Disruption of tendons and ligaments
* Fractures of bony structures

2-Classifications of fractures

A-According to etiology

I- Traumatic: e.g., low/high energy force
II- Pathological: e.g., metabolic, malignancy (Mets.), infection
III- Stress: exposure of normal bone to abnormal stress, e.g., runners, military personnel, ballet dancers.
IV- Insufficiency: exposure of abnormal bone to normal stress, e.g., osteomalacia.
V- Congenital e.g. congenital pseudoarthrosis.
VI- Collagen abnormality, e.g., osteogenesis imperfect.

B-According to location

1-epiphyseal 2- metaphyseal 3-diaphyseal 4- growth plate.

C-According to soft tissue status, open (compound), closed (simple)

D-According to the mechanism of injury (type)
= **Direct trauma** →transverse, a 'butterfly' fragment or Comminuted #.
= **Indirect trauma**: a bone breaks at a distance away from where the force is applied.

Types of injury (force)
(A) Twisting → spiral #
(B) Compression →short oblique #
(C) Bending →triangular 'butterfly' #.
(D)Tension → transverse #, avulsion #
(E) Crushing such as a vertebra or the calcaneus
(F) Repetitive stress →stress #

3-Clinical Features of fractures:
= History of trauma.
= Symptoms & signs.
1. Pain & tenderness 2. Swelling 3. Deformity
4. Crepitus 5. Loss of function 6. Abnormal movement.

Joint injury

Joint Dislocation: complete loss of contact between 2 joint surfaces
Joint Subluxation: partial loss of contact between 2 joint surfaces

Fracture treatment: Reduction →Immobilization →Rehabilitation.

4-Common types of fractures.

A-General types of fractures.

Closed # (simple)	The skin is not communicating with the fracture site.
Open # (compound)	The skin communicates with the fracture site.
Transverse #	The fracture line is at a right angle to the bone's axis. (length of the # equals bone width), direct force, high energy
Linear (fissure) #	The fracture line of the bone is parallel to the bone's long axis.
Oblique #	The fracture line has a curved or oblique pattern. (length of the # equals 1.3-1.5 bone width)
Spiral #	Complex, multi-planar fracture line (length of the # equals > twice bone width). Rotational force, low energy.
Comminuted #	The bone breaks into > two pieces.
Avulsion #	Fragment of bone is separated from the main mass, by the attached tendon or ligament.
Compression or Wedge shape #	Fracture due to vertical load on the bone, e.g., spine, causing a crush or wedging injury.
Pathologic #	Fracture due to the abnormal bone
Stress #	Fracture due to high load on a normal bone.
Segmental #	Two fracture lines with a bone fragment between the proximal and distal portions of the bone
# dislocation	Loss of joint contact associated with bony fracture
Complicated #	Fractures associated with Vascular, nerve injury ..etc.
Greenstick #	Fracture in children, in which one side of the bone is broken, and the other only bent.
Buckle # (Torus)	One side of the bone **buckles** upon itself without disrupting the other side of the bone seen in children.

B-Forearm fractures

Colles' #	Extraarticular # distal radius within 2.5 cm of the wrist (dinner-fork #). Anterior & ulnar angulation. Dorsal & radial displacement of the distal fragment. Impaction of fragments.
Smith's #	Transverse fracture of the distal radius with volar displacement and/or angulation of the distal fracture fragment (reversed Colles' #)
Barton's #	Oblique intra-articular fracture of the distal radius with resultant volar or dorsal displacement of the distal fragment. The radiocarpal joint is intact.
Galeazzi # dislocation	Fracture distal radial shaft, inferior radioulnar joint dislocation.

Monteggia # dislocation	Proximal 1/3 ulna fracture + anterior dislocation of the head of the radius.

C-Special Lower limb fractures.

Tillaux #	Avulsion fracture of the tibia at the inferior tibiofibular ligament
Triplane #	Fracture at the epiphyseal plate of the tibia in early adolescence with the involvement of epiphysis and metaphysis of the tibia.
March #	Stress fracture 2nd metatarsal neck
Jones #	Transverse fracture base of the 5^{th} metatarsal.
Lisfranc's # dislocation	The most common midfoot fracture is at the tarsometatarsal joint.

5-Imaging of fracture.

A-Plain Radiograph (X-ray):

Rule of two

2 Views AP and lateral. 2 joints (in limb fractures)
2 Times (pre-reduction and post-reduction) 2 occasions e.g. scaphoid #
2 Sides e.g. elbow # in children.

Radiographic signs of a fracture include a radiolucent line and cortical disruption of the bone.

B-Special imaging

1- Computed Tomography Scan (CT Scan),
2- Magnetic Resonance Imaging (MRI)
3- Radioisotope bone scanning

6-Deformity (displacement) of the fracture: movement of the distal segment of the fracture relative to the proximal part**.**

I-**Angulation**: Amount of bend at a fracture site described in degrees, with respect to the apex of the angle or concerning the direction of the distal fragment. (varus or valgus, anterior or posterior).

II-**Rotation**: only properly appreciated on an X-ray showing the joint above and the joint below the fracture.

III-**Translation**

The sideways motion of the distal fracture fragment (anteriorly or posteriorly, laterally or medially), usually described as a percentage of movement of distal segment when compared to the diameter of the bone.

IV-**Shortening**: the amount of a fracture collapse expressed in centimeters (bayonet apposition), due to pulling of muscles.

7-Description of the fracture (Personality of the fracture)

Systemic description of radiograph for diagnosis of fractures

Always start with history and physical examination; they will give a lot of help in further diagnosis of the case.

1-Check the **information found on the X-ray**

• Name and date of birth of the patient • Side of extremity • Date of X-ray

2-Integrity of the soft tissue (Closed fracture, Vs. Compound fracture).

3-Look at the **2 views**, AP &Lat.

***Check from outside to inside (air → soft tissue →metal → bone).

1- **Where is the fracture**?
= Diaphysis, (proximal-middle-distal). = Metaphysis = Epiphysis
= Physis = Intra articular.

2- **The shape of the fracture/pattern**
Transverse, Oblique, Spiral, Comminuted, Butterfly, Segmental, Intra-articular …etc.

3- Pieces: two or more (comminuted)

4- **Look for deformity**.

A- Displacement

- Non-displaced: fracture fragments are in anatomic alignment.
- Displaced: fracture fragments are not in anatomic alignment.
- Varus: apex away from the midline.
- Valgus: apex toward the midline.

Causes of fracture displacement

• Muscle forces. • Gravity • Obliquity of the fracture line.
• Improper handling of the fracture.

B- Translated:
C- Distracted: a gap separates fracture fragments.
D- Angulated: direction of fracture apex. e.g., Varus /valgus.
E- Rotated: fracture fragment rotated a long axis of the bone.
F- Shortening

5- Identify whether the bone is **normal or pathological**.

6- **Check joint involvement**: look for the extension of the fracture line into the joint, joint swelling, and evidence of dislocation.

7- Check **soft tissue swelling**: the extent of the soft tissue swelling indicates the severity of the injury.

Stress fracture

Fracture due to exposure of normal bone to small repetitive stresses (bending and compression).

With repeated stress, osteoclastic resorption exceeds osteoblastic formation, and a zone of relative weakness develops – ultimately leading to a breach in the cortex.

Common in athletes in training, dancers, and military personnel.

Pathological fracture

Fracture through a diseased bone with minimal trauma.

Causes of pathological # in Children

1) Bone cyst 2) Non-ossifying fibroma

4) Chronic Osteomyelitis 6) Osteogenesis imperfecta.

Causes of pathological # in Adults

- Metastatic tumor : (Breast, Prostate (sclerotic), Lung, Thyroid (lytic, expansile), Renal (lytic, expansile)
- Myeloma
- Metabolic: Osteoporosis is the commonest cause of pathological fractures in the elderly.

Mangement of fractures

1-Management of closed fracture

Life >Limb > Wound > Fracture

- Life → ATLS
- Limb →decompress in compartment syndrome
- Wound →open (give antibiotics, tetanus, irrigation)
- Fracture → Reduction →immobilization → rehabilitation

Protection, though.

- Prevent further soft tissue damage
- Pain relief
- Decrease the incidence of shock and fat embolism
- Patient transport and radiological imaging.

A- ATLS Protocol

Airway with Cervical spine protection
Breathing and ventilation
Circulation with hemorrhage control
Disability: Neurologic status
Exposure of the patient

B- Assess for neurovascular compromise and compartments.

6 P's:

Pain, Pallor, Pulselessness, Paresthesia, Paralysis, and Poikilothermia

C- Pain management (e.g. Non- Opioid analgesics, Opioids)

D- Dealing with the fracture.

Traditional Principles

1- **Reduction:** manipulation to improve the position of the fragments.
2- **Immobilization:** splint to hold them together until they unite.
3- **Rehabilitation:** joint movement and function.

1-Reduction

Should be done as soon as possible because swelling of the soft tissues during the first 12 hours makes reduction increasingly difficult.

I- Closed reduction: under anesthesia or analgesia**.**

(a) Traction in the line of the bone, ligament taxis (ligament pull).
(b) Dis-impaction.
(c) Pressing fragment into reduced position.

2-Immobilization (No metal – Metal)
To maintain the fracture in a reduced position.
Stability is achieved by one of the following techniques:

1- Intrinsic stability, some fractures require no immobilization
2- External splint.
3- Internal fixation.
4- External fixation

Methods of external immobilization
• Traction by gravity –transverse humeral #, e.g., a U-slab of plaster
• Casting, Functional bracing, special splints.

II- Open Reduction Internal Fixation (ORIF)

Advantages of ORIF
= Allows accurate reduction and maintenance of position
= Allows early mobility of the patient and joints, thus avoiding 'fracture disease.' (Stiffness and edema).
= May encourage union, but only if sufficiently strong.
= Diminishes hospital time.

Disadvantages of ORIF
1-Infection
2-Operative complications due to poor technique, or poor equipment.
3-Union may be disrupted. 4-Implant failure.
5-Refracture: due to the early removal of metals implants 18- 24 m safer.
6-Further surgery may be needed to remove the device.

Types of orthopaedic implants for ORIF

1- *Screws* – inter-fragmentary compression, e.g., the malleoli.
2- *Plate and screws* – most suitable in the forearm or around the metaphysis
3- *Flexible intramedullary nails* – for long bones in children.
4- *Interlocking nail and screws* – ideal for the femur and tibia;
5- *Dynamic compression screw and plate* – ideal for the proximal and distal ends of the femur;
6- *Simple K-wires* – for fractures around the elbow and wrist
7- *Tension-band wiring* – for olecranon or fractures of the patella.
8- *Hemiarthroplasty* for fracture neck femur in elderly.

III- External fixation

= Monoplanar fixator.
= Multiplanar (Circular) fixator.

3-Rehabilitation

Guidelines of soft-tissue care in trauma
= Elevate and exercise; never dangle, never force.

= Soft tissue swelling minimized by improving venous drainage, elevation, and firm support.
= Stiffness minimized by exercise.
Active movement helps to pump away edema fluid, stimulates circulation, prevents soft tissue adhesion, and promotes fracture healing**.**

Open Fractures (Compound)

Fracture hematoma communicates with an epithelialized surface, e.g., skin, rectum, or vagina; maxillofacial fractures with the buccal cavity.
Surgery within 6-8 hours of injury is needed to prevent infection.

Gustilo Classification.

Type	Description	Surgery	Antibiotics
Type I	<1cm and clean		Cefazolin for three days
Type II	>1cm AND no extensive soft tissue damage, avulsions, or flaps		Cefazolin + Gentamicin for three days
Type IIIA	Extensive soft tissue damage, avulsions, or flaps but adequate soft tissue coverage of bone or High-energy trauma cause regardless of the size of the wound.	Closure by an orthopaedic surgeon	Cefazolin + Gentamicin for three days + Penicillin in soil contamination
Type IIIB	Extensive soft tissue loss with periosteal stripping and exposure of bone. Massive contamination common	Needs to be flapped by a plastic surgeon	
Type IIIC	Arterial injury requiring repair.	Needs vascular surgeon	

Tetanus prophylaxis:

- Toxoid 0.5 ml for those previously immunized.
- If not immunized → Add Immunoglobulin Intra muscularly given with two different syringes in <u>two different locations.</u>

Indications for immediate surgery
1-Gross contamination,
2-Compartment syndrome, or
3-Vascular injury

Complications of fractures.

1-Acute

1- Shock (Hypovolemic or Neurogenic)
2- ARDS
3- Fat embolism, DVT, Thromboembolism.
4- Crush syndrome.
5- Fracture Blisters.
6- Neurovascular injuries

2-Chronic

A-General: Post - traumatic psychological disturbances.

B-Local

1- Delayed, non-union, or mal-union.
2- Growth disturbance, LLD in Children.
3- Late wound sepsis with skin breakdown.
4- Failure of fixation e.g. breakage or cutting out of plates or nails
5- Joint stiffness and contracture.
6- Regional pain syndrome
7- Avascular necrosis
8- Volkmann Ischemic Contracture
9- Myositis ossificans
10- Post-traumatic arthritis in intra-articular fractures, mal-union
11- Problems of immobilization. thrombosis, pneumonia, bed sores, UTI, osteoporosis, muscle wasting, renal calculi.

1-Shock (Hypovolemia)

= Femoral shaft fractures-blood loss could range from 500- 2000 ml
= Pelvic fractures-blood loss could range from 1000-2500 ml.
It could be much more in multiple fractures. = More loss in compound #

2-Deep Vein Thrombosis

Homan's sign: when forced ankle dorsiflexion produces calf pain.

Treatment

Prophylactic methods

= Early ambulation, = Foot elevation, = Compression stocking,
= Pneumatic pumps, = Exercises, etc.

Anticoagulant therapy

3-Vascular injury

Humeral supracondylar # (Brachial A),
Femoral supracondylar # (Femoral A)
Knee dislocation (Popliteal A)

4-Nerve injury in fractures or dislocations

Shoulder dislocation	Axillary N
Humeral shaft fracture	Radial N
Humeral supracondylar fracture	Median N (AIN)

Elbow medial condyle fracture	Ulnar N
Monteggia # –dislocation	Posterior interosseous N
Hip dislocation	Sciatic N
Knee dislocation	Common Peroneal N

7-Compartment syndrome. (Important)

Increased pressure in an enclosed osteo-fascial space that inhibits capillary perfusion necessary for tissue viability.

This is commonest after tibia fractures swelling in the tightly bound compartments cause venous engorgement in the compartment, further raising pressure and subsequently causing muscle necrosis.

Clinical signs ("6 Ps")

*Pain with passive muscle motion *Paresthesia *Pallor; in late stages,
*Pulselessness *Paralysis *Poikilothermia

Swelling, pain with a passive range of motion of the joints distally is the most sensitive early sign of elevated compartment pressure.

Treatment

= Remove casts and circumferential dressings down to the skin
= Splint or immobilize fractures
= Compartment syndrome is an emergent indication for surgery and requires immediate attention if suspected; delayed treatment results in ischemic contracture or limb loss.
= Perform fasciotomies for patients with compartment

N.B: Compartment syndrome can be masked by regional anesthesia & analgesia.

CASTING

Principle of Splint / Cast

To stabilize the joint above and the joint below the site of injury wherever possible.

Types of Cast

1-Plaster of Paris (Gypsum)

Originally found in Paris, it was extensively mined from the Paris district.
It is the white crystalline mineral of hemi hydrated Calcium sulfate (chemical formula **CaSO4. 2H2O**).
On adding water, it solidifies by an exothermic reaction into hydrated calcium sulfate.

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Plaster of Paris disadvantages.

= Heavy and warm.
= Not waterproof.

Strength: 80% strength in 1 hour, 100% in 24 hours

2-Fiberglass cast

Lighter, more moisture resistant, durable, and require less maintenance.

Cotton and other synthetic materials are used to line the inside of the cast to make it soft and to provide padding around bony areas.

Complications of the cast

1-Compartment syndrome., because tight cast restricts swelling.
Thus Plaster needs to check within 24 hours- check the limb, plaster, patient, and split plaster down the skin if too tight.
2-Impaired distal neurovascular.
3-Loose cast →re-displacement of the fracture.
4-Dermatitis
5- Stiff joints, muscle wasting.
6-Plaster Sores.

How do you know if something is wrong with Cast?

= Severe pain that is not controlled with medication prescribed.
= Increasing swelling
= Numbness or tingling in the extremity (hand or foot).
= Inability to move your fingers or toes beyond the cast.
= Circulation problems in your hand or foot.
= Loosening, splitting, or breaking of the cast.
= Unusual odors, sensations, or wounds beneath the cast.

4-UPPER LIMB FRACTURES

1- Clavicle Fracture

The clavicle lies over the brachial plexus, the Subclavian artery and vein, and the apex of the lung →any of these structures may be injured, and careful neurovascular examination and chest auscultation are important.
Fractures of the middle third in 80% of cases**.**

Treatment:
Arm sling or Collar and cuff for 4 weeks
Very rarely surgery is needed
Complications: Malunion

2- Dislocation of the Shoulder Joint (Important)

The shoulder joint is very shallow and relies heavily on the rotator cuff muscles and the cartilage lip around the glenoid for its stability.
It is therefore relatively easy to dislocate.

Risk factors for shoulder dislocation.

* Gender: 70% males
* Age: 80% (15-30y), 20% (female 60-80y), poor collagen crosslink → weak capsule and ligaments.
  Never occur <14y because the growth plate is weaker→ fracture before dislocation.
* Mechanism: 95% anterior dislocation due to traumatic injury, a fall (60%).
* Anatomy and function: shallow joint sockets, weak shoulder muscles, and loose capsule, ligaments laxity increase the risk of shoulder dislocation, although these factors have not been proven in studies.
* Previous shoulder dislocation: The strongest risk factor for shoulder dislocation is the previous dislocation.

Mechanism
Abducted and externally rotated arm (throwing position.)
Occurs in handball sports and quarrels.

Types of dislocation

1- **Anterior**: 97%, sub coracoid
2- **Posterior**: <3% due to epileptic seizures and electric shocks;
   Due to contraction of shoulder internal rotators, (Pectoralis major, Subscapularis and Latissimus dorsi)
3- **Inferior** – very rare <1%.
4- **Multidirectional instability (**Habitual) painless due to lax ligaments.

The patient presents with pain, restricted movement, and loss of the normal shoulder contour.

A-Anterior shoulder dislocation

Clinical Features of anterior dislocation

• Pain, tenderness, deformity (flat deltoid)
• Arm held in slight abduction, external rotation; internal rotation is blocked
• "Squared off" shoulder.

The best way to test the axillary nerve is to assess sensation over the deltoid 'regimental badge' area.

Look for Concomitant Injuries,

1- **Bony:** Bankart lesion, Hill-Sachs Lesion, Glenoid Fracture, Greater tuberosity fracture.

- **Bankart lesion**

  Injury of the anterior (inferior) glenoid labrum of the shoulder due to anterior shoulder dislocation. When this happens, a pocket at the front of the glenoid forms that allow the humeral head to dislocate into it.

- **Hill-Sachs Lesion**

  A cortical depression in the posterolateral head of the humerus. It results from forceful impaction of the humeral head against the anteroinferior glenoid rim when the shoulder is dislocated anteriorly.

2- **Soft Tissue:** Subscapularis tear, Rotator cuff tear (older pts.)
3- **Nerve:** Axillary nerve neuropraxia.

X-ray: AP, Scapular Y, Axillary views

Essential before any manipulation, as fractures may be present, either of the humeral neck, the glenoid, or the avulsion of the greater tuberosity.

Management: Urgent closed reduction

Post reduction

= X-ray confirms reduction or fracture = Pain control.
= Immobilization in a sling for 7-10 days then begin progressive ROM.
= Rehabilitation of shoulder muscles.

Complications

A- According to age

1-Younger age (20s)

= Bankart lesion, (glenoid rim chip #) → recurrent dislocation
= Recurrence rate depends on the age of 1st dislocation:
<20 yrs. = 65-95%; 20-40 yrs. = 60-70%;

2-Middle age (30s) → Fracture greater tuberosity

3-Older age → Rotator cuff tear

4- All age groups → Hill–Sachs lesion, humeral head depression by the glenoid.

B- Others → Axillary nerve injury +AVN

B-Posterior shoulder dislocation (<3%).

Commonly missed injury due to poor physical exams and radiographs.

Mechanism: Adducted, Internally rotated arm (Seizure, Electrocution)
Blow to anterior shoulder

Clinical Features

• The arm is held in adduction and internal rotation; external rotation is blocked

Investigation

X-rays: AP, trans-scapular, axillary views, CT Scan

• ***AP view:***

= Partial vacancy of the glenoid fossa (vacant glenoid sign)

= >6 mm space between the anterior glenoid rim and humeral head

= Humeral head resembles a light bulb due to internal rotation (light bulb sign).

Treatment

• Closed reduction: may need open reduction

• Sling: 3 weeks, followed by shoulder rehabilitation.

3- Proximal Humerus Fracture

Most common fracture of the humerus

Mechanism

= **Young**: high energy trauma (MVA)

= **Older**: fall on outstretched hand in osteoporotic elderly.

Clinical Features

- Pain, swelling, tenderness, painful ROM
- Arm is held close to the chest by the contralateral hand.
- Test axillary nerve function (deltoid function and skin over deltoid)

Investigations

- **X-rays**: AP, trans-scapular, and axillary are essential.
- **CT scan:** to evaluate articular involvement, fracture displacement, impression fractures, and glenoid rim fractures.

Classification (Neer's classification)

Four fracture fragments: head, greater' tuberosity, lesser tuberosity; shaft.

• ***Non displaced***: displacement <1cm and or angulation <45°

• ***Displaced***: displacement >1 cm and/or angulation >45°

• ***Dislocated / subluxated***: humeral head dislocated / subluxated from the glenoid.

Specific Complications

• AVN, axillary nerve palsy, mal-union, post-traumatic arthritis.

Shoulder stiffness and avascular necrosis of the humeral head

4- Humeral shaft fracture (Important)

Direct trauma is the most common, especially MVA.

Indirect trauma such as a fall on an outstretched hand.

Type of fractures

*Transverse (direct trauma). *Oblique (bending force).
*Spiral (twisting injury). *Pathological (in elderly).

Clinical features: Pain, swelling, and deformity of the upper arm
A careful neurovascular exam is required (radial nerve injury is most common).

Radiographic evaluation: AP and lateral views of the humerus

Complications

1-Radial nerve injury 2- Delayed union / non- union, especially transverse #.

Holstein-Lewis Fractures

Distal 1/3 fracture, may entrap or lacerate radial nerve (22%) as the fracture passes through the intermuscular septum. They need ORIF.

5- Supracondylar Fractures of the Humerus

Types: Extension 95% (distal fragment is displaced posteriorly)
Flexion 5% (distal fragment is displaced anteriorly).

Clinical Features

• Pain, swelling, point tenderness.
• Neurovascular Injury- assess median and radial nerve, radial artery

Investigations: X-rays: AP, lateral of the elbow; assess for fat pad sign

Specific Complications

• Brachial A. injury, Ant. interosseous N, compartment syndrome (leads to Volkmann's ischemic contracture), Cubitus Varus (distal fragment tilted into Varus)

6- Elbow Dislocation.

- Posterior dislocations are most common at 90%
- CR is needed followed by placement in a posterior splint for 1 week only
- *Elbow dislocation with radial head and coronoid process fractures are known as the "**Terrible Triad**," due to associated instability.*

7- Olecranon Fractures.

Olecranon fractures are most often caused by:

- Falling directly on the elbow.
- A direct blow to the elbow from something hard, or a dashboard or car door during a vehicle collision.

Non-displaced fractures → Long-arm splint or cast with the elbow flexed from 45-90° for 3 wks. Then physiotherapy to prevent stiffness**.**
Displaced fractures → Fixation.
Complications
1-Prominent implants that require removal after healing has occurred.
2-Elbow stiffness and loss of fixation.

8- Injuries of the forearm.

The forearm acts as a ring: a fracture that significantly shortens the radius or ulna will cause disrupt the proximal radio-ulnar joint or distal radio-ulnar joint.

The ulna acts as an axis around which the laterally bowed radius rotates during supination and pronation.

The interosseous membrane occupies the space between the radius and ulna; it provides a significant contribution to forearm stability.

Clinical assessment

- AP and lateral radiographs of the forearm

Check for compartment syndrome if the pain is out of proportion.

The radial head aligned with the capitellum on all views to rule-out subluxation or dislocation.

A- Isolated Ulnar Shaft Fractures (Nightstick Fractures)

Displaced # (>10°of angulation or > 50% displacement of the shaft) →ORIF.

B- Monteggia's # Dislocation,

Fracture of the proximal ulna with a radial head dislocation.

Treated with ORIF with plates and screws.

Postoperatively: Posterior splint for 7-10 days, followed by ROM exercises.

Specific Complications

• Compartment syndrome • Posterior interosseous N (PIN) injury.

C- Galeazzi # dislocation

Fracture of the distal third of the radius with *dislocation* of the distal radioulnar joint. Needs ORIF for the ulna.

D- Both-Bone Forearm Fractures

Treatment.

ORIF with compression plating using 3.5-mm dynamic plates.

The goals: restore the normal ulnar and radial length, rotational alignment, and radial bow.

Complications

1-Union problems
2-Infection,
3-Compartment syndrome,
4-Synostosis, and loss of ROM.

9-Injuries to the wrist (Important)

Normal anatomic relationships

The radial inclination of 23°, 11 mm of radial length, and 11-12° of palmar tilt.

Distal Radius Fractures,

*Colle's Fracture (important)

Transverse distal radius fracture (about 2.5 cm proximal to the radiocarpal Joint) with dorsal displacement ± ulnar styloid fracture.
Most common fracture of the upper extremity.
Common in younger and older patients.

Mechanism: fall on to an outstretched dorsiflexed hand.
Clinical Evaluation
Patients typically present with a swollen, ecchymosis, tender wrist.
Dinner fork deformity of the wrist
Particular attention is given to median nerve function as carpal tunnel syndrome is a relatively common complication (13-23%) due to traction injury, fracture fragment trauma, hematoma, or increased compartment pressure.

Radiographic Evaluation.: PA, lateral, and oblique views of the wrist**.**
Treatment Goals
(Restore radial height, radial inclination volar tilt, and articular congruity**)**
• All distal radius fractures should undergo closed reduction.
• Dorsal slab/below elbow cast for 4 wks.

Complications 31 %

1- Persistent neuropathies of the median nerve.
2- Radiocarpal or radio-ulnar arthrosis
3- Mal union (very common).
4- Shoulder, hand syndrome.Complex regional pain syndrome (CRPS)
5- Tendon ruptures (EPL), after 6 weeks

*Isolated Radial Styloid Fracture. (Chauffeur's fracture)

*Smith's Fracture,
Volar displacement of the distal radius (i.e., reverse Colle's fracture)
Mechanism: Fall onto the back of the flexed hand
Treatment: ORIF

*Barton's Fracture,
Intra-articular dorsal or volar (more frequent) rim fracture of the distal radius.
Mechanism: Extreme dorsiflexion of the pronated wrist.
Treatment: ORIF.

10- Hand Injuries.

1-Fracture of the Scaphoid (important)

The scaphoid is the carpal bone most commonly fractured.
Physical examination
1-Scaphoid lift test (pain with a dorsal-volar shifting of the scaphoid)
2-Watson test (painful dorsal scaphoid displacement as the wrist is moved from the ulnar to a radial deviation with compression of the tuberosity).

Radiographic evaluation
"Scaphoid view," in addition to the standard wrist series.
Initial radiographs are non-diagnostic in up to 25% of cases.

2-Open Fractures, Fight Bite

Any laceration overlying a joint in hand, particularly the metacarpal-phalangeal (MCP) joint must be suspected as being caused by a human tooth. Should be assumed to have been contaminated with oral flora and treated aggressively with broad-spectrum antibiotics, including anaerobic coverage. (Staph. Strep. Eikenella Corrodens)
Ist drug: Augmentin
Animal bites require antibiotic treatment that covers Pasteurella and Eikenella.

3-Metacarpal Fractures

A= Metacarpal Neck Fracture
"Boxer's fracture" of the fifth metacarpal, usually caused by the fist striking a stationary object.

Treatment: 30-40° for the fourth and fifth metacarpals accepted.
Unstable fractures require surgical intervention with percutaneous pinning or open reduction internal fixation.

5-BONE HEALING

Modes of bone healing.

1- *Primary bone healing* (strain is < 2%)
   Intramembranous healing occurs via Haversian remodeling occurs with absolute stability constructs
2- *Secondary bone healing* (strain is between 2%-10%)
   - Involves responses in the periosteum and external soft tissues.
   - Endochondral healing.
   - Occurs with non-rigid fixation, such as fracture braces, external fixation, bridge plating, intramedullary nailing, etc.
3- *The combination of the above two processes* depends on the stability throughout the construct.

1ry bone healing (there is no callus).

Osteoblastic new bone formation occurs directly between the fragments.
Gaps between the fracture surfaces invaded by new capillaries and osteoprogenitor cells grow in from the edges, and new bone is laid down on the exposed surface (gap healing).

Five stages of secondary bone healing.

(a) Hematoma: there is tissue damage and bleeding at the fracture site; the bone ends die back for a few millimeters.

(b) Inflammation: inflammatory cells appear in the hematoma. Within 8 hours of the fracture cytokines and various growth factors.

(c) Callus: the cell population changes to osteoblasts and osteoclasts; dead bone is mopped up, and woven bone appears in the fracture callus by mineralization. (3-6 weeks)

(d) Consolidation: woven bone replaced by lamellar bone, and the fracture is solidly united. (6-12 weeks)

(e) Remodeling: the newly formed bone is remodeled to resemble the normal structure. (1-2 years).

This is the 'natural' form in the absence of rigid fixation.
According to mechanical stress exposure (Wolff's law).
Fracture healing is complete when the marrow space is repopulated.

Union (Union is incomplete repair): fracture site is still a little tender,
X-Rays show the fracture line still clearly visible,
It is not safe to subject the unprotected bone to stress.

Consolidation (complete repair):

- The fracture site is not tender
- X-rays show no fracture line with a well-defined callus around it.
- Further protection is unnecessary.

A spiral fracture in the upper limb takes 6–8 weeks to consolidate
The lower limb needs twice as long.
Children's fractures, of course, join more quickly.

Causes of non-union

(1) Distraction and separation of the fragments, e.g. interposition of soft tissues between the fragments.
(2) Excessive movement at the fracture line.
(3) Severe injury that renders the local tissues nonviable or nearly so
(4) Poor local blood supply
(5) Infection.

Types of Non-union.

A-Hypertrophic non- union: excess poor callus around the fracture gap, the result of insufficient stability.

B- Oligotrophic non-union: produced by inadequate reduction with fracture fragment displacement

C-Atrophic non-union: usually arise from an impaired repair process.

Factors that dictate the type of healing

Fracture stability (mechanical stability) governs the mechanical strain

- When the strain is below 2%, primary bone healing will occur
- When the strain is between 2% and 10%, secondary bone healing will occur.

1. Inflammation → repair
   (soft callus followed by hard callus) →ending in remodeling)
2. Blood supply (bone blood flow): the most important factor

Type of Fracture Healing with Treatment Technique	
Cast treatment	Secondary: Enchondral ossification
External fixation	Secondary: Enchondral ossification
IM Nailing	Secondary: Enchondral ossification
Compression plate	Primary: Haversian remodeling

Factors that affect bone healing.

A- Internal factors

1- Blood supply (most important)
2- Head injury may increase osteogenic response
3- Mechanical factors

- Mechanical stability/strain
- Location of injury
- Degree of bone loss
- Pattern (segmental or fractures with butterfly fragments)

B -Patient factors

1- Vitamin D and calcium
2- Protein malnourishment decreases fracture callus strength
3- Diabetes mellitus
4- Nicotine
5- Medications affecting healing

- Bisphosphonates → osteoporotic fractures with long-term usage and longer healing times for surgically treated wrist fractures
- Systemic corticosteroids →fracture nonunions
- NSAIDs →prolonged healing time because of COX enzyme inhibition.
- Quinolones →toxic to chondrocytes and diminishes fracture repair

6- Pathologic fracture

Perkin's Formula for fracture healing.

	Children	***Adults***
UL	***3weeks***	***Six weeks***
LL	***Six weeks***	***12 weeks***
Fracture consolidation	***X 2***	***X2***
Remodeling (open medullary canal)	***X2***	***X2***
Smoker		***X2***

N.B: In spiral fractures divide by 2.

6- SHOULDER DISORDERS

Causes of painful shoulder

1-Referred pain: Cervical spondylosis, mediastinal pathology, and MI
2-Joint disorders: glenohumeral arthritis, & ACJ -OA
3-Bone lesions: infection and tumors.
4-Rotator cuff disorders: tendinitis, impingement synd.**,** rupture of rotator cuff & frozen shoulder.
5-Instability: dislocation and subluxation.
6-Nerve injury: suprascapular nerve entrapment**.**

1-Rotator Cuff Disease (RCD)

The rotator cuff stabilizes the humeral head within the glenoid fossa.
RCD is the most common cause of shoulder pain.
Pain can be the result of:

- **Tendinitis**: irritated or damaged tendon.
- **Bursitis**: inflamed swollen bursa with fluid causing pain.
- **Impingement**: narrow space between the acromion and rotator cuffs. The acromion can rub against (or "impinge" on) the tendon and the bursa, causing irritation and pain.
- **Rotator cuff tears**

Etiology
1-Compression between the head of the humerus and the acromion
2- Narrow subacromial space
= Glenohumeral muscle weakness → abnormal motion of the humeral head.
= Scapular muscle weakness→ abnormal motion of acromion.
= Acromial abnormalities (congenital or osteophyte).

Predisposing factors
= Heavy Work =Direct Load, Bearing =Repetitive Arm Movements
= Working with hands above shoulder height =Adiposity
= Metabolic Disorders: Hypercholesterolemia, Diabetes =Advancing Age
= Certain Sports (Repetitive arm motion)

Pathology of rotator cuff damage
Cyclic pathway:
Tendonitis →edema →thickened tendon →Impingement → tear → edema →impingement.

1. Tendonitis (Acute Inflammation)
Tendonitis → pain, inflammation, and irritation. Chronic tendonitis → more tendons can become involved, or it may progress to tendinosis (degeneration).

2. Impingement Syndrome (compression of the tendon)
The most common site of impingements is within the "supraspinatus outlet." This outlet is a space formed by the acromion process of the scapula, the coracoacromial ligament and the upper rim of the humeral head.

The clinical picture of rotator cuff tendonitis / Impingement

* Pain in the area of the four rotator cuff tendons and Tenderness located in the shoulder-joint with a dull pain, especially with overhead reaching, reaching behind the back, lifting, and sleeping on the affected side.
* Pain awakens from sleep
* Loss of mobility and strength in the affected arm.

Diagnosis

1- Empty can test.

The patient stands up with his shoulders in 90°abduction, 30° horizontal adduction, and incomplete end of the rotation. The Physician fixes his hand on the upper arm of the patient and gives downward pressure while the patient tries to maintain his position. Pain at the shoulder or weakness is suggestive of supraspinatus pathology 75%.

2- Hawkin's test.

The patient is standing up with the shoulders abducted at 90° with the elbow flexed at 90° and the internally rotated arm. The presence of pain with movement is an indicator of a possible pathology (impingement).

3-Positive impingement test (**Neer's test),**

Passive elevation of the internally rotated and outstretched arm with simultaneous stabilization of the scapula; this movement will cause pain.

4-Painful arc

(Tendonitis)→pain-related restriction of movement & strength with the abduction of the arm between **60–120°**

5-Lift-off Test

The examiner assists the patient to get in a position where he/ she touches their lower back with the arm fully extended and internally rotated.
A test is judged positive if the patient is unable to lift the dorsum of his hand off his/her, back reflecting the weakness of the subscapularis.

Treatment of tendonitis / Impingement

The goal of treatment is to reduce pain and restore function.

1- Rest and activity modification, such as avoiding overhead activities.
2- **NSAIDS -2-3 weeks**
3- **Analgesics**
4- **Rehabilitation of the shoulder**

2-Rotator cuff tear

Most tears occur in the supraspinatus tendon,

Types of RC tears.

- *Partial tear* (incomplete tear).
- *Full-thickness tear*, (complete tear).

Causes of RC Tear

1- *Acute tear* (traumatic tear) in young people.

2- *Chronic tear* (degenerative tear) in older people, asymptomatic

- **Repetitive stress.** Tennis and weightlifting
- **Lack of blood supply.** The blood supply in rotator cuff tendons decreases with age.
- **Bone spurs** the underside of the acromion bone.

Clinical Features

- Night pain and difficulty sleeping on the affected side.
- Pain is worse with active motion.
  Weakness and loss of ROM (e.g., trouble with overhead activities)
- Tenderness to palpation over greater tuberosity
- Drop arm test.

Investigations

= **X-ray**: AP view show high riding humerus relative to the glenoid, in a massive tear

= **MRI**: useful for assessing full/partial tears and tendinopathy.

3-Acromioclavicular Joint Arthropathy

30% of MRIs in patients with an asymptomatic ACJ demonstrate ACJ arthritis, the most common asymptomatic osteoarthritic joint.

1-OA of the ACJ is due to repetitive use **2-Aging**

Physical exam

= Pain with palpation, pain during "crossover" testing +ve scarf test (i.e., reaching over the body and grabbing the contralateral shoulder with the ipsilateral hand)

4-Adhesive Capsulitis (Frozen Shoulder)

A disorder characterized by progressive pain and stiffness of the shoulder usually resolving spontaneously after 18 months.

A-**Mechanism**

Primary adhesive capsulitis

- Idiopathic, usually associated with DM.
- May resolve spontaneously in 9-18 months.
- More common in older patients (40–60 y).

Secondary adhesive capsulitis

- Due to prolonged immobilization
- Shoulder-hand syndrome - a type of chronic regional pain syndrome
- Following myocardial infarction. Stroke, shoulder trauma.

B-Pathology

Inflammatory thickening of the capsule.

Starts at the rotator cuff interval between the supraspinatus and subscapularis.

The 1st movement to be lost is external rotation while in stiff shoulder abduction.

C-Stages

- 0-6m → painful stage, a gradual increase in pain & decrease of ROM.
- 6-12m →adhesive stage, severe pain, minimal ROM.
- 12-18m →recovery stage, decreased pain, restoration of ROM.

D-PF: ***(****Female****,*** *DM x5****,*** *MI****,*** *Trauma, surgery****,*** *Hyperthyroidism****,*** *Stroke****)***

E-Clinical Features

• Gradual onset of diffuse shoulder pain with decreased active and passive ROM.
• Pain worse at night.
• Increased stiffness as the pain subsides: continue for 6-12 months after the pain has disappeared.
• Loss of both active and passive ROM; internal rotation is usually first affected.
Diagnosis of frozen shoulder is primarily by clinical examination (restriction of both the active and passive range of motion of the shoulder)

G-Treatment

Full recovery may take up to 3 years.

5-Bicipital Tendonitis.

• Tenderness to direct palpation of the tendon with the arm internally rotated 10°.
• **Speed test**: resisted elevation of the supinated arm with the elbow Extended.
• **Yerguson's test**: resisted supination with the elbow flexed is positive for pain at the bicipital groove.

6-Biceps Tendon Rupture

• Seen in long head ruptures.
• Distal biceps rupture produces elbow flexion weakness.

7-Glenohumeral Joint Instability

The majority of glenohumeral dislocations and subluxations are in the anteroinferior direction.
• Chronic unidirectional or multidirectional instability

• **TUBS**: **T**raumatic, **U**nidirectional, associated with a **B**ankart lesion, and <u>often requires **S**urgery</u>

• **AMBRI**: **A**traumatic, **M**ultidirectional, maybe **B**ilateral, best treated by **R**ehabilitation, **I**nferior capsular shift is the surgery performed if rehabilitation fails.
• **Sulcus sign** is suggestive of multidirectional instability (also inferior laxity): with the arm at the side, distraction force to the humerus causes the shoulder the area just below the acromion to hollow out; discomfort occurs if inferior instability is present.

A-Anterior instability:

The apprehension test is positive when the arm is in an externally rotated and

abducted position; pressure on the humeral head in the anterior direction causes guarding.

B-Posterior instability:
The apprehension test is positive when arm the is in an internally rotated, flexed, and adducted position; a posterior force causes guarding

9-Calcific Tendonitis of the Rotator Cuff

- An acute or chronic condition of the shoulder in which calcium deposits (OH apatite) develop within the rotator cuff tendons esp. supraspinatus.
- Night pain, pain with overhead activity).
- A physical exam reveals tenderness over the greater tuberosity.
- Pain with passive forward flexion **(Neer's sign)**.
- Pain with forwarding flexion to 90 degrees with subsequent internal rotation **(Hawkins sign)**
- Plain radiographs typically show the calcified tendon.

7-SPINE DISORDERS

1-Low Back Pain (LBP). important

Epidemiology:

- 80 % lifetime incidence.
  80 % of patients will recover from acute LBP within three days to 3 weeks, with or without treatment.
- Chronic LBP is defined as symptoms persistent for >6 months.
- The recurrence rate of LBP is 40%.

Risk factors for <u>non-specific back pain and chronicity</u>.

• Smoking	• Stress	• Pain behavior
• Depression	• Job dissatisfaction	• Night shifts
• Obesity	• Education level	• Unemployment

Waddell's signs of nonorganic pain

1-Pain in non-anatomical distribution
2-Pain out of proportion to the stimulus
3-Exaggerated pain behavior

Perform four benign tests to assess Waddell's signs.

• *Skin roll test* – gently roll loose skin of the lower back.
• *Twist test* – gently rotate the patient's torso at the hips.
• *Head compression test* – apply a small load to the top of the head.
• *Flip test* – test straight leg raise when seated and supine.

Differential Diagnosis for all back pain

1-Mechanical Spinal Condition (97%)
2-Non-mechanical Spinal Condition (1%)
3-Non-spinal /Visceral Disease (2%)

I- Mechanical LBP

1- Lumbar "**Strain**" or "**Sprain**" – **<u>80%</u>**
2- Degenerative changes – 10%
3- Herniated disk – **4%**
4- Osteoporosis compression fractures – 4%
5- Spinal **Stenosis** – 3%
6- **Spondylolisthesis** – 2%

***II- Non-mechanical spinal conditions* (1% of all LBP)**

1-Neoplasia: Multiple myeloma, Metastatic CA, lymphoma (0.7%)
2-Infection: Osteomyelitis, Discitis, Abscess.
3-Inflammatory arthritis: Ankylosing spondylitis, Reiter's syndrome, Scheuermann Disease (osteochondrosis)

***III- Visceral Disease* (2% of all LBP)**

1-Disease of pelvic organs: Prostatitis, Endometriosis, Chronic PID
2-Renal Disease **3-Aortic aneurysm**
4-GI disease: Pancreatitis, Cholecystitis, Penetrating ulcer.

Clinical classification of LBP

1-LBP from radiculopathy or spinal stenosis
2-LBP from serious causes (Malignancy, infection, Cauda equina, Fracture)
3-Non-specific LBP
4-Non-back LBP

Age at onset

Children: congenital, developmental disorders, or infection.
Young adults: disc disease, spondylolisthesis, or acute fractures.
Older adults: spinal stenosis, metastasis, and osteopenic fractures

Systemic symptoms with associated spine pathology.

***Metabolic disease**
***Rheumatologic conditions**: Polyarticular involvement.
***Metastatic disease:** age > 50y, cancer history, rest pain, weight loss**.**
***Infection:** indwelling catheters (hemodialysis)

Referred back pain.

**Viscerogenic* =Peptic ulcer =Cholecystitis =Nephrolithiasis =Pancreatitis
**Pelvic inflammatory disease*
**Vascular*: Abdominal aortic aneurysm
**Distant musculoskeletal areas*: hip arthritis, trochanteric bursitis

Psychogenic pain: Patients with chronic low back disorders.

* Evidence of secondary gain * Workers' compensation or litigation
* Inappropriate physical findings (Waddell) signs, and symptoms.

Chronic back pain: Risk factors for development include:

* Frequent disabling episodes * Availability of workers' compensation
* History of smoking * Age older than 30yr.

Examination of Patients with Disorders of the Spine.

1-Neurological examination

Component	Features
Inspection	Overall alignment in sagittal and coronal planes (sciatic scoliosis)
Gait	Wide-based (myelopathy), forward-leaning (stenosis), antalgic.
Palpation	Localized posterior swelling (trauma), acute gibbous deformity, tenderness
Range of motion	Flexion/extension, lateral bend, full versus limited
Neurologic function	Motor, sensory, reflexes, assessment of long-tract signs long-tract signs**.**
Special tests	Straight-leg raise, Cross SLR, Femoral stretch test, Spurling test, Waddell signs of inorganic pathology

SIJ Dysfunction
*Patrick's Test { FABER test} (Flexion, Abduction, External Rotation)

Red Flag signs.
1-**A**ge > 70 2-**F**evers, chills, recent UTI
3-Recent significant **T**rauma 4-Night **P**ain or pain at rest
5-**C**auda Equina manifestation
- Progressive Motor or sensory deficit
- Saddle anesthesia, bilateral sciatica or leg weakness, difficulty urinating, fecal incontinence.

7-Unexplained **W**eight loss. 8-History of **C**ancer or strong suspicion of cancer
9-History of **O**steoporosis 10-**I**mmunosuppression
11-Chronic oral **S**teroid use
12-**F**ailure to improve after six weeks of conservative therapy.

Investigations in LBP
A- **Laboratory**: to screen for other disease etiologies.
B- **X-Ray:** obtained 4-6 weeks after onset of symptoms.
C- EMG / NCV (Electro diagnostics):
D- Bone scan
E- CT Scan
F- **MRI**

N.B: **36 %** of asymptomatic subjects had "HNP" at L4-L5 & L5-S1 levels.

Treatment of LBP
1- Medications
***Paracetamol**
*** Anti-inflammatory** medications (NSAIDs):
COX1 →GIT problems COX2 → Cardiac problems

2- Physiotherapy and occupational therapy
3- Compresses
- Hot for chronic pain→ relax muscles
- Cold for acute pain → analgesic, decrease swelling

4- Surgery if needed
5- Prevention of LBP
- **Exercise:** Aerobic, back/leg strengthening
- **Education** about proper lifting techniques
- **Weight loss** and smoking cessation

Muscular pain and ligamentous pain (Lumbar Strain)

The most common cause of LBP
Muscles and ligament fibers can be overstretched or injured →Leading to muscular pain and ligamentous pain.

Degenerative back pain (discogenic back pain) 10%

Symptoms of Degenerative Disc Disease
* Back pain greater than leg pain, no radiculopathy.

* Negative tension signs.
May involve buttock or proximal leg pain if associated with facet syndrome.
Radiographs:

- Osteophytes and narrowing of the disk spaces.
- Changes associated with the degenerative spine.

MRI
Reveals decreased signal intensity in the disc space on T2 weighting (dark disc).

Lumbar Disk herniation (4%)

Sciatica: pain radiating to the back of the thigh, below the knee down the foot.
Radicular pain: back pain and unilateral or bilateral leg symptoms.
-The postero-lateral portion of the lumbar disk is the weakest (posterior longitudinal ligament) and as a result the most common location for herniation.
-L4/5, compressing the L5 nerve root, or L5/S1, compressing the S1 nerve root.
-Far-lateral disk herniation at L4-L5 will impinge upon the L4 nerve root.

Pathology of disc herniation.
1-Disc degeneration: Present in 80% of people >50y
2-Disc Protrusion: bulge
3-Disc extrusion
4-Disc sequestration

Central prolapse is often associated with back pain only; however, acute insults may precipitate a Cauda Equina Syndrome.

Features of Cauda Equina Syndrome
1-Bladder and bowel incontinence 2-Perineal numbness
3-Bilateral sciatica 4-Lower limb weakness

Digital rectal examination and evaluation of perianal sensation are important for the immediate diagnosis.
Immediate MRI and surgery (surgical decompression within the first 48 hours were reported to lead to the best outcomes.

Clinical features
Physical examination
Observation (change in posture, gait), sciatic scoliosis.
Palpation of the posterior spine (spasm, localized tenderness).
Measurement of range of motion (decreased flexion).
Neurologic evaluation

- Tension signs such as SLR (L4-L5 or L5-S1).
- Muscle weakness.
- Decrease reflexes, and sensory loss in the affected level
- Femoral nerve stretch test (L2-L3 or L3-L4) are critical findings that suggest HNP.
- A positive contralateral SLR test (pain in the affected buttock/leg when the opposite leg is raised) is the most specific test for HNP.

Diagnostic tests
MRI: The best
False-positive findings are common (occurring in 35% of those younger than 40 years old and 93% of those older than 60 years old).

2-Lumbar Canal Stenosis

Narrowing of the spinal canal, neural foramina, or both, producing nerve root compression, root ischemia, and a variable syndrome of back and leg pain.

Sites of the stenosis:
1-Central stenosis
Absolute stenosis *is defined as* ***< 10 mm*** *of anteroposterior diameter on CT scan.*
2-Lateral recess stenosis
3-Foraminal stenosis
4-Tandem stenosis is the occurrence of both cervical and lumbar stenosis that often presents as both neurogenic claudication and myelopathy.

Causes of stenosis

1- **Congenital** (idiopathic or developmental in achondroplasia)
2- **Acquired stenosis** (most common) is usually:

- Degenerative owing to enlargement of osteoarthritic facets
- Spondylolisthesis
- Post-traumatic
- Iatrogenic (postsurgical)

3- **Secondary** to systemic disease processes
- Paget's disease -Ankylosing spondylitis

Patient history and physical examination
1- Pain and paresthesia with ambulation or prolonged standing are relieved by sitting or with flexion of the spine.
* Walking increases blood flow but due to stenosis venous drainage impaired → edema → more stenosis
* Flexion → increase the size of the canal.
2- Lower extremity pain, (in the buttock and thigh), with numbness.
3- Neurogenic claudication, 50% of patients.

To differentiate from vascular claudication.

- Pain starts proximal (buttock) and extends distally.
- Pain is relieved when sitting, not with standing
- Normal vascular examination (exclude vascular cause)
- Symptoms usually take a few minutes to settle,
- Unlike the situation in a peripheral vascular disease where leg pain from ischemic claudication settles in approximately 30 seconds.

Physical examination
The limited extension is the primary finding →exacerbate pain.

	Neurogenic Claudication	*Vascular Claudication*
Walking	Proximal-distal thigh pain	Distal - Proximal calf pain
Standing	Causes symptoms	Relieves symptoms
Stair climbing	Symptoms develop late (back flexed)	Symptoms develop sooner (straight back)
Stationary bicycle (back flexed)	Relieves symptoms	Causes symptoms
Pulses	Normal	Abnormal

Standing treadmill tests can be a sensitive (>90%), provocative evaluation of neurogenic claudication.

Imaging

Difference between Disc and Stenosis.

	Disc	**Stenosis**
Age	<50y	>50y
Onset	Sudden	Gradual
Posture	Bad in sitting Better in extension	Good in sitting Worse in extension
Weakness	+ve	-ve
Tension sign	+ve	-ve

CT Scan: Osteophyte formation, Axial of axial canal morphology.
MRI (test of choice)

- Hypertrophy of ligamentum flavum
- Foraminal stenosis and nerve root entrapment
- Evaluation for malignancy

3-Spondylolysis and Spondylolisthesis

A-Spondylolysis

Defect in the pars inter articularis (cortical bone between the pedicle and lamina)
* Common causes of low back pain in children and adolescents.
* Usually due to fatigue fracture from repetitive hyperextension stresses.
* Most common in gymnasts.

Imaging

Plain lateral radiographs demonstrate 80% of the lesions.
Another 15% are visible on oblique radiographs, which show a defect in the neck of the "Scottie dog."

CT scan, and more recently **single-photon emission computed tomography** (SPECT) may help identify subtle defects.
Increased uptake on SPECT is more compatible with acute lesions that have the potential to heal.
Prognosis: Unilateral defects never progress to spondylolisthesis.

B-Spondylolisthesis: Forward slippage of one vertebra on another.

Types
Type I: Dysplastic, congenital deficiency of the superior facet of S1.
Type II: Lytic (Isthmic), (defect in the pars inter- articularis)
Type III: Degenerative (L4–L5).
Type IV: Traumatic
Type V: Pathological (e.g., neoplasm).

Severity: five grades according to severity (**Meyerding**);
based on the amount or degree (compared with S1 width).

- Grade I: 0%-25%
- Grade II: 25%-50%
- Grade III: 50%-75%
- Grade IV: greater than 75%
- Grade V: greater than 100% (Spondyloptosis).

• Radiographs show slip on lateral views and "collar" on "Scottie dog" on oblique views with pars inter articularis defects present.

8-PEDIATRIC FOOT DISORDERS

1-Foot terminology

= *Talipes*: (Latin: Tali →talus = ankle, pes →foot) =*Pes*: (Latin: foot)
= *Equinus*: fixed Plantar flexion = *Calcaneus*: fixed dorsiflexion
= *Pesplanus*: flat arch =*Pescavus*: high arch
= *Supination*: 3-plane movement weight rolls onto the outer edges of the feet.
= *Pronation*: 3-plane movement. the amount that the foot rolls inward toward the arch.
= *Inversion*: the soles of the feet face inwards, occurring at the subtalar joint
= *Eversion*: the soles of the feet face outwards, occurring at the subtalar joint
= *Adductus* (Varus): frontal half of the *foot* (forefoot), turn inward.
***Skew foot**: Severe forefoot adductus combined with hindfoot valgus (Tarsometatarsal adductus, talonavicular lateral subluxation, and valgus hindfoot)*

Anatomical divisions of the foot.	Biomechanics of the foot (Tripod)
Hindfoot: talus and calcaneus **Midfoot**: navicular, three cuneiforms, cuboid. **Forefoot**: 5 metatarsals, 14 phalanges	1-Center of the calcaneus 2-Head of the 1st metatarsal 3-Head of the 5th metatarsal

2-Calcaneovalgus foot (Common at birth)

Positional deformity is seen at birth as a result of soft tissue contracture.
Hyper dorsiflexion of the hind foot (improper intrauterine positioning).

Clinically: Dorsal surface of the foot may rest on the anterior tibia. Passively correctable foot to the neutral position.

3-Congenital Talipes Equino Varus (CTEV)

It is the second most frequent pediatric foot condition.

= Classification

1-Postural (Flexible): Postural or positional talipes can be passively fully corrected, or even overcorrected.
2- Rigid (non-flexible): needs correction by serial casting.
3- Syndromic (club foot): stiff foot

Etiology and Associated Anomalies

• **Idiopathic** (most common)
• **Secondary** club foot:
1. Neurological disorders and neural tube defects
2. Arthrogryposis multiplex congenita, Larsen syndrome, Sacral agenesis, tibial deficiency, constriction rings, and amniotic bands.

= Incidence

Male: Female 2:1, 1- 4:1000 live births, Unilateral > bilateral.
10% chance of a subsequent child being affected if positive family history.

= Etiology
Multifactorial inheritance modified by intrauterine and environmental factors

Deformities of Idiopathic Congenital foot deformity
1-Hindfoot Equinus & Varus
2-Midfoot Cavus
3-Forefoot adduction & supination.

Patho-anatomy
A-Bones * Short tibia * Short fibula (most common).
Talus →- Medial and plantar deviation (Equinus) of the neck of the talus
Calcaneus →medial rotation, equinus, Varus
Navicular →medial subluxation on the head of the talus
Cuboid →medial subluxation
Forefoot →adducted and supinated.

B- Joints: ankle & subtalar joint → Equinus
C-Muscles: Atrophy of the leg, especially in a peroneal group, Tib. Post, FDL, FHL contracted a small foot and a small calf.

C- Other soft tissues
Tendon, Tendon sheaths, Joint capsules, Ligaments, and Fascia contractures

Clinical assessment
Examine the whole child to exclude associated abnormalities:
Examine the spine (neurological cause). The affected limb may be shortened, the calf muscle is atrophic, and the foot is short compared to the opposite side.

Radiology. Usually done after correction of the deformity.
1-Dorsiflexion lateral view:
Talocalcaneal angle > 35° is normal; a smaller angle with a flat talar head is seen with clubfoot.
2-Anteroposterior view a Talocalcaneal (Kite) angle of 20- 40° is normal (<20° degrees with clubfoot).
"Parallelism" of the calcaneus and talus is seen in both views

Treatment
1-Started at birth by Ponseti serial casting (4-6 casts) changed weekly, (90% success), the cornerstone for the correction is the head of the talus.
The first deformity to be corrected is the cavus by supination of the foot.
2-Tendo Achilles lengthening (90%) at the end of correction, if not done the foot may end in rocker bottom deformity

The sequence of deformity correction
***C*avus, *A*dductus, *V*arus**, and finally ***E*quinus** (CAVE).
1- Correction of **Cavus** is always the sole aim of the first cast
2- Correction of **Adduction and heel Varus**

3- Correction of **Equinus**
4- Percutaneous Achilles tenotomy is required in 90% of cases (under local anesthetic), followed by a cast for three weeks post-tenotomy.

3- Regular stretching exercises by the mother frequently during the day
4- Special orthosis (Dennis brown boots or Mitchell brace), 23 hr./day for 3-6 months after casting and then at night for 2 to 3 years.
5- 50% need tibialis anterior transfer at the age of 5 years.

4-Flat feet (Very important)

Definition: Reduced longitudinal arch, heel valgus, & forefoot abducted pronated.
Incidence: 20- 25% of flexible flat feet in adults. Very common in children.
Patho-anatomy Generalized ligamentous laxity

Classification

- **Physiologic**: Flexible (Hypermobile): most common.
  = Familial: hyperlaxity of ligaments. = Accessory navicular bone
- **Pathological**: rigid subtalar movement.
  - The tarsal coalition, Idiopathic, juvenile chronic arthritis
  - CVT - Neuromuscular - Tight heel cord 25%
  - Muscular imbalance with tight Achilles tendon and weak tibialis posterior, due to cerebral palsy and spina bifida

Assessment
Usually asymptomatic disorder, but parents are concerned about the shape or worn shoes.
Observing the feet from behind and asking them to stand on tiptoe.
A flexible flat foot will then demonstrate an arch. At the same time, the heel will correct from valgus to neutral or even into Varus (demonstrating normal subtalar function). If compliance is an issue, the great toe manual dorsiflexion and the same features observed
* Neurological examination.
* Check of tightness of Tendoachilles.
* General assessment of signs of hyperlaxity.
- Hyperextension elbows, thumb to the forearm, hands to the floor, knee Hyperextension.

Investigations

1- Asymptomatic: not needed
2- Painful and stiff
- Weight-bearing oblique foot to rule out Calcaneo-navicular coalition
- CT scan for Talocalcaneal coalition.

Management
Flexible flat feet in children → Reassurance as arch grows at 10y.
(no need for any special shoes or arches). Insoles, inserts, or surgical shoes have no beneficial effect on the outcome.

Only if genuine medial foot pain or severe wear of shoes consider soft arch support, remember this does not get rid of the flat foot deformity.
Stretching for symptomatic patients with a tight heel cord

Neuromuscular flat foot.
Orthotics to prevent excessive wear and pressure effects on the foot.

5-Tarsal Coalition

An abnormal connection of two or more bones in the foot.
= Tarsal coalition is an osseous, cartilaginous, or fibrous connection between bones of the hindfoot and midfoot.
= 50% are bilateral, 20% multiple.
Calcaneonavicular coalitions are the most common, followed by talocalcaneal coalitions.

Patho-anatomy
a. The onset of symptoms
i. Age 8- 12 years for calcaneonavicular coalitions
ii. Age 12- 15 years for talocalcaneal coalitions
b. Talocalcaneal Coalitions most commonly in the middle facet

Clinical assessment
i- Pain and limited subtalar motion are the hallmarks of a tarsal coalition.
ii- Pain is typically in the tarsal sinus or the longitudinal arch.
iii-Difficulty with uneven ground and frequent ankle sprains.
iv-Stiff flat feet (no correction on tiptoe test)

Investigations,
Plain radiographs: standing lateral, oblique 45° for calcaneonavicular coalition.
A CT scan for talocalcaneal coalition & clarify whether the child has multiple coalitions in the foot (20%).

6-Congenital Vertical Talus

= Irreducible dorsal dislocation of the talonavicular joint.
= 50% associated with neuromuscular disease (Myelodysplasia, Arthrogryposis, Diastematomyelia, Sacral Agenesis.

Patho-anatomy
Bony
a. The navicular dislocated dorsolateral (irreducible).
b. Vertically oriented talus (hindfoot equinus)
c. Eversion of the calcaneus d. The cuboid is displaced dorsally

Soft tissue
a. Contracture of the dorsolateral muscles. b. Achilles tendon contracture.
c. Tibialis post and peroneal contracture. d. Attenuation of the spring ligament.
Clinically, the foot has a rigid convex plantar surface with a prominent talar head (Rocker's bottom).

Radiographs
Lateral view of the foot in forced plantar flexion→The navicular remains dorsally dislocated in this view.
This differs from the oblique talus, in which the navicular reduces on the plantar flexion radiograph.

7-Pes Cavus (Cavus foot)

A high arched foot deformity where the longitudinal arch fails to flatten with weight bearing.

A- Causes

- Muscular imbalance, variable in each case
- Neuromuscular until proven otherwise, as up to 60-70% % found to be neurological (CMT Syndrome)

1-Congenital 2-Idiopathic: simple cavus 3-Residual CTEV
4-Neuromuscular diseases: usually cavovarus

- Muscular: muscular dystrophies
- Peripheral nerves: HMSN (most common cause), polyneuritis
- Spinal cord: spinal dysraphism, polio, tethered cord, spina bifida
- Central: cerebral palsy, Friedreich's ataxia, Charcot-M-T. disease

5-Trauma: compartment syndrome, crush injuries.

B-Presenting complaints
Foot fatigue, difficulties with shoe wear; metatarsalgia, and lateral foot pain due to reduced area of foot contact with the floor, and pressure areas over the dorsum of PIP joint.

C-Patho-anatomy
a. Fixed forefoot plantar flexion, relative to the hind foot
b. Hindfoot Varus
c. Weak tibialis anterior is relative to the peroneus longus.
d. Intrinsic weakness and contracture→ Clawing toes.
e. Tight plantar fascia.

D-Examination
1. Gait - Look for obvious neurological gait 2. Foot examination
3. Spinal examination 4. Full neurological examination
5. Coleman block test:
Standing with the lateral side of the foot on the block with the big toe hanging, if the varus heel corrects → flexible deformity.

6- Radiological investigation
1. X-rays of the foot: Standing AP and true lateral views.
The first metatarsal and talus long axes should be in line.
The 'Meary' angle is measured between them.
2. X-ray of the spine for spina bifida.
3. MR scan of the spine.

8-Curly toes.

Malrotation flexion deformity of one or more toes (contracture of FDL and FDB).

A common disorder in children.

= Frequently runs in families, often bilateral.

= Noticed when the child walks

=Always asymptomatic (highly symptomatic for the parent!)

Management: Reassurance is the mainstay of management

Surgical tenotomy involves FDL tenotomy at age four years if needed

9- Lesser toes deformities,

1-Hammer - flexion of the PIPJ & extended DIPJ

2-Mallet - flexion at the DIPJ

3- Curly toes -Neutral at MTP, Flexed at PIP and DIP

4-Claw Toes - flexion of the IP joints, hyperextension of the MTPJ (intrinsic minus deformity) often associated with a cavus foot and tight TA.

Associated diseases of claw toes

= Old compartment syndrome = NM disorders = Cavus

= Inflammatory arthropathy

9-CHILDREN FRACTURES

1-Properties of children's bones

1- **Growth plate (GP):**

In infants, GP is stronger than a bone this will lead to

= Increased fractures in the metaphyseal area.

= Provides perfect remodeling power.

= Injury of growth plate causes deformity.

= A fracture near the GP might lead to overgrowth.

Physeal anatomy

- ***The proliferative zone*:** The most metabolically active zone.
- ***The hypertrophic zone***

= The weakest because it lacks both collagen and calcified tissue.

= Most physeal separations occur through this layer.

2- **Bones in children:**

A- Increased collagen/bone ratio → easy fractures

B- Increased cancellous bone →

- Reduces tensile strength
- Reduces the tendency of fracture to propagate →less comminuted #
- Bone fails on both tension and compression ("buckle" fracture)
- Less brittle, therefore may bend but not fracture.

C- Increase Plasticity→ Greenstick fractures

Why do children's bones bend before they break?

*Less osteoid density than the adult.

*More porous than adult bone (Haversian canals occupy a much greater part of the bone.

3- **Cartilage:**

= Increased ratio of cartilage to bone =Difficult X-ray evaluation

= Size of the articular fragment in fractures often under-estimated

4- **Periosteum:**

- Metabolically active →More callus, rapid union, increased remodeling.
- Thick and strong
  i. Intact periosteal hinge affects fracture pattern.
  ii. May aid reduction
  iii. Decrease displacement of the fracture

5- **Age-related fracture pattern:**

a. Infants: diaphyseal fractures
b. Children: metaphyseal fractures
c. Adolescents: epiphyseal injuries

6- **Physiology:** better blood supply → good union

Children have different physiology and anatomy.

- Growth plate. → remodeling of deformity

- Bone.
  = more collagen →easy fractures
  = more cancellous bone →Simple fracture patterns
- Cartilage. (Thick) → not seen on X-ray
- Periosteum. (Thick)→ rapid healing
- Ligaments. (Strong) →Bone fails 1st
- Age-related (Infants) → diaphyseal #
- Physiology (Good blood supply) →good union

The power of remodeling

- Children have a tremendous power of remodeling
- Can accept more angulation and displacement
- Rotational mal-alignment especially diaphyseal does not remodel

Factors affecting remodeling potential.

- Years of remaining growth – the most important factor
- Position in the bone – the nearer to physis the better remodeling (worst) for diaphyseal rotation deformity.
- Plane of motion –
  Greatest in sagittal, the frontal, and least for transverse plane
- Physeal status – if damaged, less potential for a correction
- The growth potential of adjacent physis
  e.g., the upper humerus is better than the lower humerus

Injuries of the proximal humerus and close to the wrist remodel much faster than injuries of the elbow and proximal forearm.
Remodeling is fastest at the knee (distal femur and proximal tibia) than in the proximal femur and distal tibia.
Because of the **relative contributions of the growth plates to the longitudinal growth of the upper and lower limb.**

3-Relationship to joints

Remodeling is *maximal* in proximity to, in the plane of action of, the nearest joint.
The shoulder moves in three planes and remodels in three planes.
e.g., 30° of angulation is acceptable in a child with > 2years of growth remaining.
The elbow is a hinge joint. The cubitus Varus deformity after supracondylar fracture must be prevented because it will not remodel.

Complications of children's fractures

- Mal-union is not usually a problem (except cubitus- varus)
- Non-union is hardly seen (except in the lateral condyle)
- Growth disturbance – epiphyseal damage
- Vascular – Volkmann's ischemia

Functional properties of the child's bones.

= **Growth plate** (physis), increase the longitudinal growth of bone.
= **Periosteum** increase in bone diameter by appositional growth.

= **Good blood supply** Immobilization rarely causes joint stiffness.
= **Thick Cartilage**: Joint surfaces in children are more tolerant of irregularity than those of adults.

Distal radius and ulna is the most common site of fracture in children accounting for nearly 25-30%

The clavicle is the most common fractured bone during birth.

Types of Pediatric fractures

	Radiographic findings	**Treatment**
Torus # (Buckle)	Axial force applied to immature bone cortex buckles on compression (concave) side and fractures. The **t**ension (convex) side **r**emains **s**olid (intact).	Immobilization with a splint or a cast
Greenstick #	Incomplete fracture extending partway through width of bone following bending stress; bone fails on tension side; compression side intact (compare to torus fracture). The bone is bent like a green twig.	***A-Acceptable angulation**** → Cast ***B-Non acceptable angulation*** → CR + Cast
Bowing # (Bent)	No disruption of the cortex or periosteum. Angulation is present. Bones bent to 45 degrees.	
***Acceptable angulation on X-ray** = 0–5 years: Lateral view: 20–25°; AP view: < 10° = 6–10 years: Lateral view: 15–20°; AP view: < 5° = > 10 years: Lateral view: < 10°; AP view: 0°		

Classification of Physeal injuries (Salter-Harris Classification)

Type I: physeal separation
Type II: fracture traverses physis and exits metaphysis, the most common type.
Type III: fracture traverses physis and exits epiphysis
Type IV: fracture passes through epiphysis, physis, metaphysis
Type V: crush injury to physis

Fractures distal forearm

Injury	**Description**	**Treatment**	**Complications**
Distal radius	SH I-V	CRPP types III and IV	Deformity, Loss of reduction, Volkmann IC, Growth arrest, Malunion.
Torus	Tension side intact (failure in compression)	Short cast for 3 weeks	
Greenstick	Tension side with plastic deformation	CR if angle >10°	
Complete	Both cortices disrupted	CR+ long cast	

All injuries that involve physes should be followed at least 12-18 months to confirm that growth has not been disrupted.

II- Pediatric Upper Extremity Fractures & Dislocations

1-Clavicle Fractures

90% of obstetric #s; often associated with brachial plexus palsies.
80% in the middle 1/3 of the clavicle. **Treatment**: arm sling for 2-3 wks.

2-Proximal Humeral Fractures in infants

Separation of the un-ossified proximal humeral epiphysis following difficult delivery of an infant.
Significant displacement and angulation were well tolerated, and the child was treated in an arm sling for three weeks.

3- Proximal Humeral Fractures in children,

= 85% of growth occurs at the proximal physis → good remodeling
= Proximal fragments rotated into the abduction and external rotation by rotator cuff
= Distal fragments adducted and shortened by the pectoralis major & deltoid.
= Accordingly, gravity can be a useful reduction aid.

Treatment

- Non-operative treatment with immobilization
- Children < 12 years old; 100% displacement acceptable
- Age > 12 years, controversial, 50% displacement acceptable.

Complications
Mal-union, Varus deformity, well tolerated due to the wide range of shoulder motion.

4-Elbow fractures: 5% to 10% of all fractures in children

Ossification centers around the elbow joint.
(C-R-I-T-O-E) === 1-3-5-7-9-11

A-Supracondylar Fractures of the Humerus (important)

Age: male, 4-8 years. As a result of hyperextension injury to the elbow.
Clinical picture: pain, swelling, deformity.

Types

1-Extension type,

Type I: Undisplaced supracondylar fractures.
Radiographs: positive fat pad sign (elbow hemarthrosis)
Treatment: Long arm back slab cast for 3-4 weeks.

N.B: Fat pad sign does not equal fracture.
Anterior fat pad sign →70% chance of fracture.
Posterior fat pad sign → 90% chance of fracture.
The anterior fat pad can be seen in a normal elbow.
The posterior fat pad sign is never visible in a normal elbow.

Type II: Posterior angulation of the distal fragment+ intact periosteal hinge.

Radiographs: The anterior humeral line no longer bisects the capitulum on the lateral radiograph.
Treatment: Closed reduction if stable → long arm back slab cast 3-4wk.
If unstable → percutaneous pinning & long arm back slab cast 3-4wk.

Type III: Completely displaced, may injure neurovascular structures.
If ischemia is present initially, prompt reduction, if pulses are not restored with reduction, then an angiogram and vascular consultation.
When the loss of pulses occurs following attempts at reduction, vascular entrapment should be suspected and prompt surgical exploration of the brachial A.
Closed reduction, percutaneous pinning, and long arm back slab cast for 4 wks., if irreducible open reduction and K- wires fixation.

2-Flexion type: very rare, needs open reduction and pin fixation.

Complications

Early

1-Brachial A. injury: rare
2-Compartment syndrome
3-Ant. I N neuropraxia.

Late

A-Elbow stiffness
B-Mal union (very common) Cubitus Varus / Valgus
N.B: Nerve injury resolve with observation over 3-6 months.

B- Subluxation of the Radial Head (Nursemaid's Elbow)

A common elbow injury in children < 5 years. Caused by a sudden pull on the arm in immature annular ligament slips over the head of radius. Injured arm held in extended /slightly flexed and pronated position.
The child suddenly stops using the arm, holding it in a flexed & pronated position.
Radiographs: Normal.
Treatment:
The reduction is achieved by firmly supinating the forearm and flexing the elbow while pressing down on the radial head. Often a "click" is felt when reduction is achieved.

C- Lateral condyle fractures

2nd most common elbow fracture after supracondylar fracture.
The most common are Salter-Harris IV fracture patterns.

Physical exam
Lateral side swelling and tenderness, lateral ecchymosis implies a tear in the aponeurosis of the brachioradialis and signals an unstable fracture.
Commonly missed fracture.

Radiograph
AP, lateral, and oblique views of the elbow. The internal oblique view most accurately shows fracture displacement because the fracture is posterolateral.

Complications
1-Stiffness: most common complication, by 24 wks., 90% returns normal ROM.
2-Nonunion.
Higher rate of nonunion in non- surgical management due to pulling of the wrist extensors and intra-articular fracture and poor metaphyseal circulation to distal fragment. ***Treatment:*** ORIF with a screw, may require a bone graft.

3- Cubitus Valgus ± tardy ulnar nerve palsy 10%.
Due to lateral physeal arrest or more commonly a non-union of the lateral condyle. Slow, progressive ulnar nerve palsy caused by a stretch.

D-Radial neck fractures

Worse outcomes were seen in patients >10 years of age.

Clinical Picture
Pain is exacerbated by motion, especially supination and pronation.
must have a high suspicion of forearm compartment syndrome

E-Monteggia fracture

Proximal 1/3 ulnar fracture with associated radial head dislocation (Ant., Post. or Lat.) or plastic deformation of the ulna without obvious fracture.

Complications
Posterior interosseous nerve neurapraxia (10% of acute injuries)

5- Forearm Fractures, 40% of all pediatric fractures.
Location: 14% distal physis**,** 60% distal metaphysis**.** 20% mid-shaft.
4% proximal third, Associated elbow injury in 13%.

A- Distal Radius Fractures
Total account for approximately 40% of all pediatric long bone fractures

B- Distal radius physis
- Contributes 75% growth of the radius
- Contributes 40% of the entire upper extremity
- Growth at a rate of ~ 5.25mm per year
- Metaphyseal fracture is most common (60%), followed by physeal #.

III-Pediatric Lower Extremity Fractures & Dislocations.

1-Femoral Shaft Fractures: they often result from significant trauma.
2-Distal femur fracture,
Most commonly Salter-Harris II fractures
30-50% physeal arrest that often leads to growth disturbance and deformity.
Complications
Limb length discrepancy or angular deformity (most common)
Popliteal artery injury and compartment syndrome.

3-Toddler's fracture
An occult undisplaced tibial spiral fracture may cause the toddler to refuse to weight bear. These fractures heal rapidly in a long-leg cast.

4-Tillaux fracture
A fracture involving the anterolateral distal tibial epiphysis.
Mechanism: closure of medial physis earlier than the lateral attachment of anterior inferior tibiofibular (AITF) ligament.
Open reduction and screw fixation.

5-Triplane fractures
Fracture distal tibia identified on the lateral radiograph as SH –IV (two parts SH II and three parts SH III injuries
Anatomic reduction and fixation are critical.

IV-Child Abuse (Battered baby syndrome) (NAI)

A-Suspicion raised NAI
= < 3 years old = Multiple healing bruises, skin marks, burns.
= Unreasonable histories = Delay in seeking treatment = Posterior ribs
= Corner #: Bucket handle # (traction & rotation) at junction of meta. and physis)
B- Nonorthopaedic injuries: Skin, head, burns, and blunt abdominal**.**
B- Fracture Type & location.
= Humerus, tibia, and femur, in that order.
= Spiral humerus # and distal humeral physeal separations are highly suggestive.
= Transverse femur shaft fracture <1 y of age (60-70% NAI)
= Spiral >common than transverse
= Diaphyseal fractures are four times more common in abuse cases than metaphyseal fractures.
Treatment
1- Skeletal survey in children <5y and bone scan if older.
-Early involvement of social workers and pediatricians
2- Treat according to the fracture pattern.
Prognosis
If the abuse is missed, there is a greater than 33% chance of further abuse and a 5% to 10% chance of death in affected children.

10-HAND INFECTIONS

I- Paronychia

Nail fold infection, seen in children due to biting or sucking fingers.

A- *Acute paronychia* → red, swollen, and tender nail fold.
Treatment: In early cellulitis → oral antibiotics. , in an abscess → surgery

B- *Chronic paronychia* caused by Fungal infections due to chronically wet hands (dishwashers) →mildly red, swollen, and mild tenderness, but with no abscess.
Treatment: special medication and stop constant exposure to moisture.
Surgery sometimes is needed to remove infected tissue.

II- Felon

A painful, throbbing infection of the pulp of the fingertip.
This closed space is separated into many small compartments; each of them fills with infection and pus. If not treated early, destruction of the soft tissues and even bone can occur.
*Treatment***:** Anti-staph antibiotics, drainage in abscess formation.

III- Herpetic Whitlow

Viral infection of the hand, usually terminal phalanx of the fingers, is caused by a herpes virus. This is more commonly seen in healthcare workers whose hands exposed to the saliva of patients carrying herpes (HSV1-2). The condition, characterized by small, swollen, painful blood-tinged blisters, and sometimes numbness, is typically treated conservatively and usually resolves in several weeks without any after-effects.

IV-Bites

Mostly during fist injury (fight bites) → Eikenella Corrodens, Staph & Strep (GA) infection of the skin, tendon, joint, and bone.
Treatment: All bites assumed as infected, and the patient should be taken to operating theater directly after the X-ray.

V-Deep Space Infections

The hand is divided into many separate "compartments" or "deep spaces." One or more of these can become infected even from a small puncture wound. An abscess can form in the muscle area at the base of the thumb (thenar space), the palm (deep palmar space), or the web spaces between the fingers. These infections can spread to other areas, even to the wrist and forearm, and need to be drained by surgery.

VI- Flexor Tenosynovitis: Inflammation of a tendon and its sheath.
*Infectious tenosynovitis***:** Patients with infectious FT can present at any time following a penetrating injury, with complaints of pain, redness, and fever.

Kanavel's signs in *Infectious tenosynovitis*
1) Held in flexion posture
2) Pain with passive finger extension 3) Pain on active flexion
4) Fusiform swelling 5) Tenderness along the tendon sheath.

UPPER LIMB DISORDERS

I-HAND

1-Ganglion Cyst

= Fluid-filled swelling overlying joint or tendon sheath, most commonly at the dorsal side of the wrist. (Scapho lunate lig.) arises from the herniation of dense connective tissue.
= Painless Swelling.
Treatment: • Aspiration and steroid injection alleviates ~80% of ganglion cysts, but there is a high recurrence rate
• Recurrent Symptomatic cysts → excision, taking the entire stalk.

2-De-Quervain's Tenosynovitis

Noninflammatory thickening of abductor pollicis longus and extensor pollicis brevis tendons, characterized by pain or tenderness at radial styloid.
Risk in new mothers, golfers, racquet sport players. Dx. **Finkelstein test**.
= Pain at the radial side of the wrist with activities in which the thumb is abducted or the wrist is ulnarly deviated
= Palpation: pain at the site of the retinaculum at the radial styloid.

3-Mallet Finger

= Loss of full, active extension of the DIP joint, resulting in unopposed flexor digitorum longus action to pull the distal phalanx into flexion.
= Loss of extension can be due to avulsion of the tendon with or without a fragment of bone, rupture, or laceration of the tendon inserts on the distal phalanx.
= Usually traumatic (except for rheumatoid arthritis)
= Radiographs to determine if an intra-articular fracture is present.

5- Boutonniere Deformity.

Central slip disruption of the middle phalanx (laceration, closed rupture, synovitis of PIP joint) →Subluxated lateral bands and unopposed flexor digitorum profundus →Proximal interphalangeal (PIP) joint flexion and distal interphalangeal (DIP) hyperextension.

6-Swan-Neck Deformity

• Deformity characterized by hyperextension of proximal interphalangeal (PIP) joint, flexion of distal interphalangeal (DIP) joint, and metacarpophalangeal (MCP) joint flexion, • Due to attenuation or rupture of the extensor tendon insertion into the distal phalanx.

Causes

- Untreated mallet deformity,
- Volar plate laxity,
- Ligamentous laxity.

• The most common mechanism in rheumatoid arthritis is intrinsic tightness with associated MCP synovitis

II-ELBOW

I-Tennis elbow

Overuse injury involving eccentric overload at the origin of the common extensor tendon leads to tendinosis and inflammation at the origin of ECRB → Pain and tenderness over the lateral epicondyle of the elbow.
A common complaint among tennis players – but even more common in non-players who perform similar activities involving forceful, repetitive wrist extension.

Mechanism: Repetitive wrist extension and forearm pronation.
Pathoanatomy: microtear of the origin of ECRB.
Associated conditions: radial tunnel syndrome is present in 5%.

Symptoms
Pain over the lateral epicondyle comes gradually, after unaccustomed activity involving forceful gripping and wrist extension.

Physical exam,
Point tenderness at ECRB insertion into lateral epicondyle.

Management
90% will resolve spontaneously within 6–12 months.

1- Rest + rehabilitation
2- NSAIDs
3- Ice packs and massages also relieve pain and control swelling. Using ice therapy after exercise is an effective way to prevent discomfort.
4- Corticosteroid injections have good results initially but high recurrence rates after 6 wks.

Persistent case → release of origin of extensor tendons.
Surgery is successful in about 85 % of cases.

II-Medial Epicondylitis (Golfer Elbow)

Definition: tendinopathy at the origin of the flexor muscles.
Pain and tenderness along the medial elbow worsen on resisted forearm pronation or wrist flexion.
Ulnar nerve compression syndrome is present in a high percentage of patients

Treatment

- Physical therapy and NSAIDs are successful in >90% of patients with tendinopathy, but the epicondylitis may not resolve for 1y.
- Injections of steroids for tendinopathy (high rate of recurrence).
- Surgical debridement may be helpful in recalcitrant cases, but not be released.

III-Olecranon bursitis.

The olecranon bursa sometimes becomes enlarged as a result of continual pressure or friction; this used to be called the 'student's elbow.'

Causes
= Trauma = Gout →calcification on X-ray. = Rheumatoid arthritis → nodules

11-INJURIES OF THE SPINE

1-Anatomy

Termination of the spinal cord→ L1 vertebral body (L1-L2 disc).
The conus medullaris gives off the motor and sensory nerve rootlets, also known as the "cauda equina" or horse's tail.
No disc between C1-2
In the cervical spine, the C1 root exits above the C1 vertebral body; the C2 root exits below the C1 vertebral body.
In the lumbar spine, each root exits under the pedicle with the same number. e.g. L4 nerve root exits under the L4 pedicle.

The junction between the thoracic and lumbar spines is the most common site of the fracture because it is the transition zone between the relatively stiff thoracic spine and the much more flexible lumbar spine.
As a result, due to its transition zone status, the thoracolumbar junction (T11-L1) is more susceptible to injury, >60% of all thoracic and lumbar fractures.

2-Mechanism of Injury

You should take a proper history of the mechanism of injury

1- High energy mechanisms such as motor vehicle accidents
2- Falls from a height.
3- Pathological #. e.g., osteoporosis or skeletal metastases.

3-Assessment of suspected spinal fractures

- ATLS principles.
- Immobilize the patient on a flatbed, protecting the cervical spine with triple mobilization precautions (3persons head-trunk-pelvis) during the examination of the spine.
- Ask about weakness, numbness in both UL and LL.
- Examination
  = Inspect the spine for bruises, hematoma, or deformity
  = Palpate for tenderness
  = Perform and document a full neurological examination including a rectal exam.

4-Imaging: Obtain AP and lateral views.
* **X-rays of the spine** at the site of tenderness if positive do X-ray for the entire spine (10% risk of different level fractures).

Look for
= The size and shape of the vertebra. = Alignment of the vertebrae.
= Displaced fracture. = Interpedicular distance.

* **CT is indicated.**
1-If patients with multiple injuries. 2-X-rays are inadequate.

Stability of spinal fractures (Stability can be assessed on the lateral X-ray)
I-The anterior column: anterior 1/2 of the vertebral body, along with the anterior

ligaments. → stable

II-The middle column: posterior 1/2 of the vertebral body and the ligaments → unstable

III-The posterior column: everything behind the vertebral body, including the pedicles, facet joints, laminae, and spinous processes. →unstable.

Denis classification system

This system divides the spine into three columns and classifies injuries into minor and major categories based on radiographic and CT imaging.

1-Minor injuries

Fractures of the transverse & spinous processes, lamina, and pars interarticularis.

2-Major injuries

Compression # , burst #, flexion distraction injuries, and # dislocation.

1-Wedge compression fracture

A simple fall onto the bottom (flexion injury) in elderly osteoporotic Women.

Only the anterior column is involved.

Treatment: Conservative if the wedging <50%

- Analgesia and gentle mobilization.
- A corset-style brace may be used to help reduce pain.

Surgery indicated if

- >50% anterior wedging of the vertebral body.
- If the fracture fails to heal (pathological fractures)
- If there is severe kyphosis.

Vertebroplasty, whereby cement is injected into the vertebral body using a long needle inserted percutaneously under X-ray guidance.

2-Burst fracture

Occur as a result of extreme <u>axial loading</u> as falling from a height to the <u>anterior and middle column</u>, leading to the divergent spread of the pedicles and retropulsion of bone into the spinal canal.

N.B: Any structure passed by the gravity line may be involved in axial loading injury,e.g., skull base→C7 →D12→SIJ→femur head → med. knee condyles →calcaneus

There is a risk of neurological injury, either due to instability or by fragments of bone that are pushed into the canal (known as 'retropulsion').

A CT scan is mandatory to evaluate this type of injury.

Surgery indicated in:

1-Neurological symptoms 2-Significant deformity, 3-To stabilize the spine and decompress the cord.

3-Chance fracture (Seat Belt #)

Flexion-distraction injury is the classic "seat belt injury," with failure of the middle and posterior columns and preservation or compressive failure of the anterior column, depending on the location of the axis of rotation.

Abdominal visceral injuries are commonly associated with flexion-distraction injuries in the thoracolumbar spine, in 50% of these patients.

All three columns are involved → Surgical stabilization is necessary.

4-Transverse process fractures

Large muscles, including the trapezius and psoas, originate from the transverse processes. Avulsion fractures are fairly common when these muscles contract strongly in a side-impact collision. Although the fractures are stable and heal without intervention, look carefully for other spinal or pelvic fractures that may coexist.

5-Fracture-dislocations

= Failure of all three columns following compression, tension, rotation, or shear forces.
= They are associated with the greatest incidence of neurologic deficit (paralysis) and are unstable.
= They need surgery for the prevention of chronic back pain
= No cure for paralysis at all even Stem cells are fake.

12-LOWER LIMB FRACTURES

1- Hip Dislocation

= Caused by a high-energy trauma such as an MVA.
= Posterior hip dislocations (85%-90%) are more common
= Dashboard injury (posteriorly directed force against a flexed knee), if the hip is abducted initially → fracture-dislocation of the hip
15% complicated by Sciatic nerve injury.
= Check for other fractures especially femur head, acetabular rim femur neck or shaft fractures, and tibial fractures.

Clinical Evaluation
The classic appearance is shortened extremity with the hip flexed, internally rotated and adducted

Treatment
The hip should be reduced emergently within 6 hours due to the risk of osteonecrosis from associated vascular disruption.
If closed reduction is unsuccessful, open reduction is performed as soon as possible.
After reduction, the patient was placed in skin traction or a Denham pin inserted into the proximal tibia for 3 to 6 weeks of traction.

Complications Posterior hip dislocation
1-Sciatic nerve damage (15%)
2- Osteoarthritis (up to 40%)
2-Associated fractures (acetabulum, femur head, or neck of femur)
3-Avascular necrosis (up to 5%)

2- Hip Fractures

Types of hip fractures. = Intracapsular = Extracapsular.

Risk Factors for Hip Fracture.

Non modifiable
= Age > 65 years
= Family history of hip #
= Female sex
= Low socioeconomic status
= Prior hip #

Modifiable
*Chronic medications
- Levothyroxine (decreases bone density)
- Loop diuretics (impair calcium absorption in kidneys)
- Proton pump inhibitors (reduce calcium absorption)
- Selective serotonin reuptake inhibitors/sedatives (increased risk of falls caused by sedation, postural hypotension)

*Decreased bone mineral density (osteoporosis)
*Falls *Reduced level of activity *Vitamin D def.

Principles of treatment
Early surgery (within 24-48 hr.) is prudent. This allows earlier mobilization and rehabilitation, which speeds functional recovery and decreases the risk of pneumonia, skin breakdown, deep venous thrombosis, and urinary tract infections. Earlier surgery is associated with reduced pain and shorter length of stay in the

hospital.
Patients with comorbidities have an increased risk of mortality; therefore, surgery may need to be delayed until 48 -72 hrs. after the # to stabilize these conditions.

Fractures of the Femoral Head: It is never an isolated injury, usually associated with hip dislocation.

Complications: Osteonecrosis, Nonunion & Post-traumatic arthritis.

The Femoral Neck Fractures. (important)
Fractures of the femoral neck occur most often in elderly patients with the osteopenic bone after a fall directly on the greater trochanter, or fall from twisting injury or catching the toes in the carpet.
In young patients due to high-energy trauma

Clinical picture
Severe pain externally rotated abducted and shortened limb.
Inability to move the hip.

Radiographic evaluation,
AP pelvis radiograph, AP, and lateral radiographs of the hip; If no fractures are detected in an elderly patient with persistent hip pain, one should consider MRI or bone scan to look for a nondisplaced or incomplete fracture.

Fractures of the femoral neck may be classified according to the location (sub-capital, trans-cervical, & basi-cervical), or based on the stability of the # pattern.

The Pauwel's classification.
Describes increasing instability of the increasing fracture angle from the horizontal: type I (30°), type II (50°), and type III (70°).

The Garden classification
Type I (incomplete fracture/ valgus impacted),
Type II (complete fracture, nondisplaced),
Type III (complete fracture, with partial displacement, the trabecular, and bone pattern of the femoral head does not line up with the acetabulum),
Type IV (completely displaced fracture, the trabecular bone pattern of the head does line up with the acetabulum).

Treatment
Type I: internal fixation with multiple cannulated screws.
Type II: (non-displaced) fractures treated with internal fixation.
Type III and type IV (displaced)

- For patients less than 60 years old, with good bone quality, open-reductio, internal fixation is the usual choice.
- For patients more than 60 years old, with osteopenic bone and comminuted fractures, hemiarthroplasty is the treatment of choice.

If the patient has evidence of preexisting acetabular arthritis → THA

Post-operative
= Prophylaxis against thromboembolism
=Early mobilization

Complications
1-Bed sores
2-DVT
3-AVN 30% in displaced fractures, 10% in un-displaced #.

Prognosis of femoral neck fracture

Types	Displacement	Description	Notes
Type 1	Nondisplaced	Incomplete, nondisplaced, including valgus-impacted fractures	**~7% AVN**. Typically treated with percutaneous fixation
Type 2		Complete, nondisplaced	
Type 3	Displaced	Complete, incompletely displaced	~ 37% AVN. Typically treated with ORIF or Arthroplasty
Type 4		Complete, completely displaced	

3- Trochanteric Fractures (important)

A-Fracture of the Lesser Trochanter
Most commonly in adolescent patients 2ry to forceful iliopsoas contracture.
In the elderly patient, think of metastatic disease.

B-Fracture of the Greater Trochanter
The typical mechanism is a direct blow due to a fall in an elderly patient.
Treatment is typically non-operative.
In a young, active patient with a widely displaced greater trochanter→ surgery

C-Intertrochanteric Fractures.
Intertrochanteric fractures describe fractures that occur in the region between the greater and lesser trochanters of the proximal femur.
These #s are extracapsular, in cancellous bone with abundant blood supply.
The typical presentation occurs in an elderly individual after a fall.

X-rays: (AP pelvis, AP, and lateral of the injured hip).

1= Unstable IT fracture
1- "Reverse obliquity" intertrochanteric fractures (oblique fracture line extending from the medial cortex proximally to the lateral cortex distally)
2- Significant posteromedial comminution
3- Fractures with sub-trochanteric extension.

Treatment
(Gamma nail) with the goal being early ambulation with full wt. bearing status.

2= Stable IT fracture →Dynamic hip screw (large screw and side plate)
Unstable fracture →Gamma (Intramedullary hip screws)

D-Sub-trochanteric Fracture,
Location: 5 cm distal to the lesser trochanter.
Mechanism of injury
= Low energy fall in elderly = High energy trauma in young.
Radiographic evaluation includes
AP pelvis, AP, and Lateral views of the hip and femur down to the knee.

Surgical treatment
Cephalo-medullary nail (Gamma nail), typically healed by 3-4 months.

Complications
1-Delayed union and nonunion 2-Hardware failure can occur.
3-Mortality and morbidity

Mortality and morbidity of hip fractures within 1 year
20% will die
30% permanent disability
40% unable to walk

4- Femoral shaft fractures,

Typically occur in young men after high-energy trauma, such as motor vehicle accidents, blood loss, may reach 1200 ml.
Usually displaced due to a muscle pull, this makes reduction difficult.
The proximal segment abducted by gluteus medius and minimus, flexed by iliopsoas
Distal segments are adducted by adductors and the limb becomes short.
Specific attention is paid to the ipsilateral hip and knee joints. Knee ligament injuries are common and easily missed.
Diaphyseal fracture in the elderly should raise the suspicion of pathological fracture.

Radiographic evaluation
AP and lateral views of the femur as well as the ipsilateral hip and knee.
AP pelvis should also be obtained.
Ipsilateral femoral neck and intertrochanteric up 10% of patients with femur #.
Ideally, surgical stabilization should occur within 24 hours of injury.
1- Intramedullary (IM) nailing: The most frequently used surgical treatment for femoral shaft fractures.
IM nailing performed in an antegrade or retrograde fashion.

Complications
i-Blood loss → shock. ii-DVT, PE. iii-Vascular injury. iv-Knee Joint stiffness.

5- Injuries around the knee Joint

1-Fracture of the Patella.

The patella is the largest sesamoid bone in the body. The patella has seven articular facets; the lateral facet is the largest (accounting for 50% of the articular surface).

Radiographic examination

Anteroposterior, lateral, and sunrise views of the knee.
Bipartite patella (5% of the population) may be confused with a fracture.

Bipartite patella

Usually occurs in the superolateral portion of the patella.
It is bilateral in 50% of patients; thus, contralateral knee X-rays may help Dx.

Types of patellar fractures

- Un-displaced fracture
- Displaced (step-off >2mm and fracture gap >3mm)

The most common patella fracture type is stellate, where a direct compressive force results in a comminuted pattern. Similarly, common are transverse patella fractures, typically sustained during hyperflexion of the knee (tensile force) with eccentric contraction of the quadriceps.

Clinically: Acute knee swelling and focal patellar tenderness.

2-Dislocation of the Patella

PF: Increased soft tissue laxity, Valgus knee.
Mechanism of injury: Sudden severe contraction of the quadriceps muscle → pulling the patella outwards →injury to medial patellofemoral ligament.

3- Fractures of the Proximal Tibia.

I. Fractures of the Tibial Plateau

Isolated lateral tibial plateau fractures are the most common

Mechanism of Injury: Axial loading coupled with varus or valgus force.
Associated injuries include meniscal tears as well as injuries to the collateral and cruciate ligaments.

Clinically: severe pain, swelling due to large hemarthrosis, limitation of ROM.

Radiographic Examination

AP and lateral X-rays of the knee are part of the standard evaluation.
CT scan is best for assessing the articular surface and is often used for preoperative planning.

Associated ligamentous injury

Segond sign (lateral capsular avulsion off of the lateral tibial plateau, indicating ACL disruption).

MRI is considered if the ligamentous injury is suspected.

6- Fractures of the shaft tibia and fibula

Most common long bone fractures as it is subcutaneous bone.

Clinical Evaluation

Severe pain, swelling, deformity, and limitation of movement.
The examiner should have a high suspicion of compartment syndrome in the acute setting.
Pain out of proportion, pain with passive stretch, tense compartments, numbness, tingling and cool toes are all signs of compartment syndrome.
Fasciotomy is mandatory by medial and lateral incisions

Radiographic Evaluation

AP and lateral x-rays of the tibia and fibula including the joint above and the joint below

Treatment.

Un-displaced or minimally displaced → long above knee cast
Fractures with significant displacement or comminution → ORIF**.**
Locked Intramedullary nailing (best)

- External fixation,
- Plates, and screws.

Intramedullary nailing is by far the most popular technique as it preserves the periosteal blood supply, optimizing conditions for fracture healing.

7- Injury to the ankle

The fibular collateral ligaments:

- Anterior talofibular ligament (ATFL),
- Posterior talofibular ligament, and
- Calcaneofibular ligament

1-Ankle Ligamentous Injuries (Sprain)

Mechanism of injury

Sudden twist (inversion or eversion with or without external rotation.
= **Lateral (90%)**: anterior talofibular ligaments [ATFL] are most often injured.
= **Medial**: deltoid ligament can be injured as well as syndesmosis

Clinically

History of twisting injury**,** pain, swelling, ecchymosis**,** severe tenderness anterior to the lateral malleolus, and limited ROM**.**

Diagnosis of ankle ligament instability:

= ***Anterior drawer test****:* with the ankle at 30° of plantar flexion and slight internal rotation tests ATFL; inversion stress on the heel with the ankle in dorsiflexion tests CFL
= ***Syndesmotic injury***: assess by palpation, presence of swelling >2 cm above the ankle joint, the presence of tenderness elicited by squeezing the fibula and tibia

together, pain above the ankle with external rotation of the ankle.
• MRI is beneficial for the evaluation of the severity and type of injury.

N.B: Children with talonavicular and calcaneonavicular coalition often have a history of ***recurrent ankle sprains***

Treatment

- **PRICE**: Soft ankle brace, ice, and elevation
- Continue bracing from 1-2 weeks, depending on the severity of the injury
- Acetaminophen or NSAIDs for analgesia
- Air cast brace for 8-12 weeks followed by proper rehabilitation, especially proprioception training.

2-Ankle fractures (important)

Radiographic Evaluation

AP, Lateral, and mortise views (15-20° internal rotation) of the ankle.
If widening > 4 mm talar shift, this indicates the significant syndesmotic injury is likely.

Danis-Weber classification

Classifies ankle fractures according to the level of the fibula component of an ankle fracture, concerning the "syndesmosis."
Type A: below syndesmosis
Type B: at the level of the syndesmosis
Type C: above the level of the syndesmosis
Type C1, C2: Other fracture variants include **Maisonneuve fracture** (ankle injury with fracture of the fibula proximal third).

In a mortise view of the ankle, if the distance between the medial malleolus and the medial border of the talus is >4 mm, open reduction is indicated to correct the lateral talar shift.

Remember:

= Avulsion #'s are usually transverse = Shear #'s are usually oblique.

Treatment

ORIF
N.B: If the operation is not done within a few hours, severe swelling forms, thus surgery needs to be postponed for a few days.

Postoperative course

Non–weight-bearing in a splint/cast/removable boot for 4-6 weeks until fracture healing is appreciated radiographically.
Ankle ROM exercises should be started early to prevent postoperative stiffness.

4-Pilon Fractures

A pilon fracture (Plafond fracture), is a fracture of the distal part of the tibia, involving its articular surface at the ankle joint. 75% have associated fibula #.
May have Tibial plateau, Calcaneus, Pelvis, and Vertebral fractures.

Mechanism of Injury
Rotational or axial forces, mostly as a result of falls from a height or motor vehicle accidents →results in an axial compression force directed through the talus into the tibial plafond

Clinical Evaluation
Swelling is often rapid and considerable, potentially resulting in skin necrosis and blistering some authors advocate waiting 7-10 days before taking patients to surgery for swelling to subside or until "skin wrinkling" is appreciated to avoid postoperative wound complications.

8- Injuries to the foot

1. The Calcaneus (Important)

Fracture of the Calcaneus
Most intra-articular calcaneus fractures are the result of axial loading where the talus is driven into the calcaneus during a fall from a significant height or motor vehicle accident. 10-20% of calcaneal fractures are associated with a thoracic or lumbar compression fracture, pelvis, or hip fracture.

Types of fractures
Intra-articular fracture (posterior facet) versus extraarticular pattern
The Bohler angle is usually 20-40°.
A decrease in this angle indicates significant depression of the weight-bearing posterior facet. CT scan offers the best diagnosis
Complications
1-Subtalar OA 2-Broad heel 3-Impingement of peroneal tendons

2. Fractures of the Midfoot.

Tarsometatarsal (Lisfranc) Joint
20% of the time this injury goes undiagnosed initially.
Sprains are the commonest injuries.
Causes include MVAs, falls from height, and athletic injuries
Mechanism of injury, Figure-11

- Indirect rotational forces and axial load through the hyper plantar flexed forefoot.
- Hyperflexion/compression/abduction moment exerted on the forefoot and transmitted to the TMT articulation.
- Metatarsals displaced in dorsal/lateral direction.

The clinical picture of fracture dislocation
1- Symptoms: severe pain and inability to bear weight.
2- Physical exam

- Medial plantar bruising
- Swelling throughout midfoot
- Tenderness over the tarsometatarsal joint

The Lisfranc ligament travels from the medial cuneiform to the base of the 2nd and 3rd metatarsal bone providing additional stability.

Diagnosis: Plain radiographs **and** CT scans can provide greater detail.

3-Jones fracture

Definition

It is a fracture of the diaphysis of the 5th metatarsal bone approximately 1.5-2 cm above the tip of the tuberosity at the metaphyseal junction. It is extra-articular,

Mechanism of Injury

It is an avulsion fracture due to the pull of the peroneus brevis muscle.
It is frequently encountered in athletes.

Clinical Features: The patient complains of pain, swelling, and limp.
On examination, tenderness can be elicited over the base of the 5th metatarsal bone.

Complications of Jones fracture

- Delayed union and nonunion, due to the poor blood supply
- Surgery may be required if it is nonunion.

4-March fracture (Stress #)

Fracture due to an overload on normal bone
This is a stress or fatigue fracture of the metatarsals, particularly the II metatarsal bone.

Clinical picture

Pain in the foot after unaccustomed activity, e.g., running. Initially, no radiological findings. MRI or bone scan can spot the site of the fracture. After 2-3 weeks, the fracture was seen as a transverse hairline with a callus around it.
It is more often encountered in military personnel who indulge in frequent and prolonged marching and hence its name.

Treatment: Rest, NSAIDs, splints, elastic crepe bandage application, etc.

13-BONE AND JOINT INFECTION

Children's bone and joint infections

I-Acute Osteomyelitis

Pyogenic Infection of the bone and bone marrow.
(infections in children are primarily hematogenous in origin)
Causative organisms

1- **Age:** 4m onwards, Staph A, followed by Strep Pneumonia & G A Strep.
2- **Pseudomonas A**: following penetrating wounds of the foot through the shoe.
3- **Salmonella**: in Sickle cell disease
4- **Anaerobes**: human bite.
5- **Neonatal infants** in hospital (0-4 months), with indwelling lines, get Staph. or Gram-negative infections, with multiple sites in 40% and are systemically unwell
6- **'Normal' infants** out of hospital G B strep. at a single site.

PF
= Poor living conditions = Primary focus of infection, e.g., a boil, sore throat, etc.
= Preceding injury with sub-periosteal hematoma. = Virulent organism

Pathogenesis
A high load of circulating organisms tends to start the infection in the vascular metaphyseal ends of the long bones, why?

- Highly growing vascular area
- Sluggish circulation in the metaphyseal right-angled capillary loops.
- Poor phagocytosis in metaphyseal blood vessels.

The presence of vascular connections between the metaphysis and the epiphysis makes infants < 18m prone to septic arthritis of the adjacent joint.

Intra articular metaphysis.
*Shoulder joint *Elbow (radial neck), *Hip Joint *Ankle (distal fibula)

The start of infection gradually causes an inflammatory response → release inflammatory mediators' → vasodilatation → exudate formation
Because of the confined space → increase intraosseous pressure tension, and decrease perfusion pressure → ischemia → tissue necrosis occurs readily and an abscess may form within the bone.
When under pressure, exudate or abscess can extend through Volkmann canals into the medullary cavity, epiphysis, or subperiosteal region stripping it and eventually penetrating to a point on the surface.
Large areas of bone may become necrotic, making penetration by antibiotics difficult and forming 'sequestra 'or hidden areas of dead and infected bone.
If the center of the shaft becomes infected, the nutrient artery may thrombose, leading to the sequestration of the whole shaft.

Common sites of osteomyelitis: around the knee joint

Clinical features of acute osteomyelitis.

- History of septic focus e.g., URTI
- 50% have a history of trauma
- Fever — acute onset with malaise
- Pain — localized throbbing in metaphysis classically,
- Swelling — often associated with joint stiffness
- Tenderness — localized, loss of limb function
- Edema and pus (late sign), pus may break through the periosteum, causing a fluctuant mass
- Swelling of nearby joints is usually due to a 'sympathetic ' effusion.

If the metaphysis is intracapsular, the joint itself may become infected

= In neonates:

*Loss of movement of the limb. *Irritable, refuse to feed
*Failure to thrive. *Cries when the limb is moved.

Radiographic changes include the following:

= Soft tissue swelling (early)
= Bone demineralization and periosteal reaction (10 days after infection).

Differential diagnosis – Osteomyelitis

1- Cellulitis 2- Neoplasm (in leukemia 30% have bone pain) 3- Sarcoma
4-Trauma (but not normally with raised ESR) 5- E. granuloma 6- Bone infarction

Diagnosis

CBC: leukocytosis with a shift to the left
ESR: 90% raised**, CRP** raised**, Blood culture** positive in 50%
C-reactive protein is the most sensitive monitor of the course of infection.
Nuclear medicine studies may be helpful in equivocal cases.
MRI: 100% sensitivity

Management

I.V Broad-spectrum antibiotics

Antibiotics usually control septicemia and fever quite quickly.

Newborn (up to 4 months of age)

Flucloxacillin (anti-staph.) plus a third-generation cephalosporin (gram-negative).
Alternative therapy: Vancomycin (MRSA) plus a 3rd generation cephalosporin (Gram-negative).

Children 4 months of age or older

Augmentin or Flucloxacillin (anti-staph.)
Alternative therapy: Vancomycin (MRSA) plus a 3rd generation Cephalosporin (Gram-negative).
If antibiotics are given early enough (no abscess), the antibiotics may control the infection and complete healing may then take place.

Indication of surgical drainage
1-Failure of response to antibiotics after 2-3 days
2-Abscess formation
3-Associated septic arthritis

Following drainage, antibiotics can be continued or changed according to the culture results
If the condition then settles, a total of 6 weeks of antibiotic therapy is given.
The ESR and CRP are a guide to recovery during this period.

Complications of Osteomyelitis

Neonates	Children	Adult
*Sepsis *Septic Arthritis → joint destruction *Hip dislocation	*Growth disturbances *Chronic osteomyelitis	*Chronicity → Fracture, Amyloidosis, Sq. CC., DVT, PE.

Prognosis: 5-10% of patients may experience recurrence.

II-Acute Suppurative Arthritis (Septic arthritis)

This is a serious and damaging condition to the joint.
Source of infection
1-Spread of osteomyelitis (hip. elbow- shoulder, and ankle)
2-Hematogenous (infants, especially).
3-Spread occurs through the epiphyseal plate into the epiphysis and then into the joint itself (<18m).
The most common site at which septic arthritis follows acute osteomyelitis is the proximal femur (hip).

(MRSA-CA) is an increasingly common cause of septic arthritis in children.

Classical Triad: Fever, Painful Joint swelling, (no active ROM)

The differential diagnosis
1-Osteomyelitis with a sympathetic effusion, 2-Transient synovitis of the hip
3-Rheumatic fever,
4-Rheumatoid arthritis or Still's disease (usually multiple joints)
Neonates and young children often have to coexist with septic arthritis and osteomyelitis, why?.

1- The bony cortex is thin.
2- The Periosteum is loose.
3- Intraarticular metaphysis
4- Blood vessels that connect the metaphysis and epiphysis serve as a conduit by which bony infection may easily reach the joint space.

Differentiating septic arthritis from transient synovitis of the hip

Kocher's criteria assessed children who underwent joint aspiration for suspected septic arthritis.

Septic arthritis is defined as having a pathogen isolated and/or having > 50,000 WBC/mL in the synovial fluid

(1) Inability to bear weight; (2) Peripheral WBC of greater than 12×10^9/L; (3) Sedimentation rate ≥ 40 mm/hr.; and (4) Fever > 38.5°C.

If all four predictors were present, there was a probability of septic arthritis of 99.6%; the probability was 93% if three predictors were present

Investigations

- ESR, CRP, FBC, blood cultures (especially if pyrexia)

- **Plain radiographs**:

After 24-48 hours loss of soft tissue planes and on day 7-10 periosteal reaction is seen. (Periosteal reaction is usually absent in tuberculosis)

Later, subperiosteal new bone may become visible with generalized periarticular porosis.

- **MRI:** best investigation**.**
- **CT** – occasionally to assess bony destruction.
- **USS** –used to assess hip effusion and aspiration.

Treatment of children

1- **≤ Three months**: Augmentin + 3rd Generation cephalosporin

If the risk of MRSA (i.e., prolonged ICU stay) → give Vancomycin

2- **> 3 months**: Augmentin

If the high risk of MRSA →Vancomycin

Complications of septic arthritis

I-Hip dislocation: prevented by the application of Pavlik Harness or hip Spica for 3 months post. hip septic arthritis.

II-Joint destruction

III-Epiphyseal separation (Tom-Smith disease)

IV-Stiff Joint: prevented by early rehabilitation.

III- Transient synovitis of the hip

Transient synovitis is a common cause of a painful hip in young children.

A respiratory illness often precedes complaints of pain, which is localized to the knee, thigh, or hip.

The short duration of symptoms, absence of diagnostic radiographic signs, and nearly normal laboratory studies suggest a benign process.

Children of any age may be affected, with the average age being six years.

Clinical Findings

A. Symptoms and Signs

Pain in the lower extremity with activity (or even with rest) is the most common

complaint for < 1 week.
Limp and refusal to weight bear are also common.
Normally, the child should be able to relax, and motion should be free and easy without "guarding," this is especially noticeable with rotation or at extremes of flexion or extension of the hip joint.
Low-grade fever may be present, but the child does not appear ill.
B. Laboratory Findings Normal WBC, ESR, CRP, or slight elevation
C. Imaging Studies
Radiographs are essential to rule out other diagnoses.
X-rays are usually normal
A hip ultrasound will show little or no effusion.

Differential Diagnosis
1-Septic arthritis 2-Legg–Perthes disease (avascular necrosis). 3-Trauma

Treatment
Rest and anti-inflammatory agents were initiated.
The child should then be reexamined to make certain that normal hip motion and comfort has been achieved.
AP & lateral X-rays are repeated in 3m to ensure that no Perthes disease
Prognosis
Recurrent symptoms may develop after the resumption of activity but usually resolve with more rest.

IV-Chronic Osteomyelitis (>3 weeks)

Causative organism; Staphylococcus Aureus

Pathology
- **Sequestrum: an** avascular piece of bone surrounded by granulation tissue (it is pathognomic of chronic osteomyelitis).
- **Involucrum:** is dense sclerotic new bone surrounding the sequestrum
  At least 2/3rd the surface of sequestrum should be surrounded by involucrum before carrying out sequestrectomy.
- **Cloaca (sinus).**

Treatment
- Remove the sequestrum (Sequestrectomy)
- Identify the organism and control the infection (most important step)
- Fill the gap (Bone graft /Bone cement)
- Provide good soft tissue coverage.

Difference between septic arthritis and other arthritis

	Appearance	WBC/µl (PMN %)	Glucose levels	C&S	Crystals
Normal	Transparent Clear and viscous	< 200 (< 25%)	as blood	-Ve	None
Non-inflamm. arthritis	Transparent Yellow and viscous	200–2000 (< 25%)	as blood	-Ve	Ca. phosphate crystals 60% of OA cases
Inflamm. arthritis	Translucent-opaque Yellow and watery	> 2,000 (≥ 50%)	< blood	-Ve	Mono Na urate crystals: gout Ca. Pyro phosphate crystals: pseudo gout
Septic arthritis	Opaque Yellow or green with variable viscosity	> 50,000 (≥ 75%) Early: > 10,000 (≥ 75%)	<< blood	+ve	None

V-Subacute osteomyelitis

Subacute osteomyelitis can cross the physis, even in older children.

Brodie's abscess
This is a chronic, localized bone abscess. The lesion is typically single and located near the metaphysis of the bone. Preferred sites are the proximal femur, proximal, and distal tibia.

Clinical features
Subacute cases present with fever, pain, and periosteal elevation.
The white cell count is often normal, but the ESR is raised.

Pathology
Typically a well-defined cavity in cancellous bone containing Seropurulent fluid (occasionally pus). The cavity is lined by granulation tissue containing a mixture of acute and chronic inflammatory cells. Typically no organisms are found but, if one is present, it is usually a Staphylococcus aureus (60%).

Management
Biopsy and surgical debridement followed by intravenous antibiotics.

VIII-Puncture wounds of the foot

The most characteristic organism to cause infection as a result of a nail through the sole of a shoe is **Staph aureus** & **P. aeruginosa.**

Adult Hematogenous Osteomyelitis.

The vertebrae (commonest site), tibia, femur, or humerus, caused by S. aureus (Commonest) followed by Pseudomonas and Enterobacteriaceae.

Sources of infection

1- Trauma, (compound tibia fracture)
2- Open reduction and internal fixation of fractures.
3- Prosthetic devices.
4- Spread from soft-tissue infection.

PF for osteomyelitis in Diabetic patients

1-Vascular compromise →inadequate local tissue response.
2- Minor trauma to the feet with multiple organisms.
3- Foot ulcers allow bacteria to reach the bone.
4-Peripheral neuropathy → no pain→ perforating foot ulcer, cellulitis.

Pathology of chronic osteomyelitis.

Sequestrum*:* a devitalized bone that serves as a nidus for infection.
Involucrum**:** formation of new bone around an area of bony necrosis.

Complications of adult limb osteomyelitis

1- Chronic infection. 2- Pathologic fractures. 3-2ry amyloidosis.
4-Squamous cell carcinoma at the sinus tract cutaneous orifice.

Radiological Dx.

A-Plain radiograph

B-MRI: best test for diagnosing early osteomyelitis and localizing infection.
T2 Sequences will show bone and soft tissue edema.
Penumbra sign:
T1- (dark central abscess with bright internal wall and dark ext. sclerotic rim

C-Nuclear medicine (Technetium Isotope bone scan**)**
When radiographs are normal, and MRI is not an option.

D-Gallium scan: for diabetic foot or if MRI is not an option.

Laboratory analysis

Leukocytosis (WBC): 30% of cases. ESR: 90% Positive. C-Reactive Protein: 97% of cases. The culture of bone is the gold-standard for guiding antibiotic therapy.

N.B: S epidermidis in prosthesis infections adhere to a biofilm that protects the organism from phagocytosis and impedes the delivery of the antibiotic

Septic arthritis in adults

S aureus, Streptococcus, and *Neisseria gonorrhoeae* are common causes.
The affected joint is swollen, red, and painful.
Synovial fluid purulent (WBC > 50,000/mm3).

Prosthetic joint infection or Metal (after ORIF)

Etiology
Incidence

- Primary joint replacement 1-2% TKA vs. 0.3-1.3% THA
- Revision joint replacement 5-6% TKA vs. 3-4% THA

Causative organisms

- Early onset (< 3 months of placement): Staph aureus
- Delayed onset (3–24 months of placement): Staph. Epidermidis
- Late-onset (> 24 months of placement): Staph aureus.

Clinical findings: prolonged minimal swelling, mild pain, and diffuse redness.

Risk factors (important)

Pre-operative	Postoperative	Infl. Arthropathy	Lifestyle factors
***Septic focus** ***Previous surgery** ***Old local infection**	***Immuno Supp. Drugs** ***Corticosteroids** ***DM (HBA1c >7)** ***Chronic renal disease** ***Malnutrition (e.g. albumin <3.5; total leukocytes <800)**	**Rheumatoid arthritis**	***Obesity** ***Smoking** ***Poor oral hygiene**

Prevention of prosthetic joint infection:
1-Perioperative intravenous antibiotics are the most effective.
2-Good operative technique.
3-Laminar flow
4-Exhaust suits ("space suits").

Antibiotics are given before insertions of orthopaedic metal or implants.
A. Prophylactic treatment (1st Generation Cephalosporins)
For insertion of a prosthesis or a metallic hardware
For clean surgical cases, administer 1 hour preoperatively and continue for 24 hours postoperatively.

14-Tumors of the Musculoskeletal System

Classification MSK Tumors.

Tissue origin	Benign	Malignant
Osteoid	Osteoma Osteoid Osteoma Osteoblastoma	1ry Osteosarcoma 2ry osteosarcoma e.g. Paget's disease
Chondroid	Enchondroma Osteochondroma Chondroblastoma Chondromyxoid fibroma	1ry Chondrosarcoma 2ry Chondrosarcoma
Fibrous	* Non-ossifying Fibroma (Fibrous Histiocytoma) * Fibrous Dysplasia * Ossifying Fibroma	Fibrosarcoma Malignant Fibrous Histiocytoma
Small round blue cell	E Granuloma	Ewing Sarcoma / PNET Lymphoma, Myeloma
Giant Cell	GCT, ABC	Malignant Giant Cell Tumor

Diagnosis of bone tumors

1-The morphology of the bone lesion on a plain radiograph is the most important. (Well-defined, ill-defined lesion)

Radiological features that are indicative of the pathological process.

● Bone destruction

● New bone formation ● Periosteal reaction ● Soft tissue swelling

2-The age of the patient 3-CT and MRI are only helpful in selected cases.

Most common ill-defined bone tumors and tumor-like lesions.

Malignant bone tumors	Aggressive benign lesions
1-Ewing's sarcoma 2-Osteosarcoma (most common presentation: sclerotic) 3-Leukemia 4-Metastases and Myeloma.	1-Infection 2-Eosinophilic granuloma 3-locally aggressive GCT.

Types of Periosteal reactions, in malignant bone tumors.

A-Continuous periosteal reaction

1- *Sunburst appearance*: divergent spicules that resemble a sunburst
2- *Solid periosteal reaction*: increased formation of new bone →indicates slow tumor growth
3- *Lamellated periosteal reaction*: may occur as single or multiple (onion skin appearance) layers; indicates rapid tumor growth

4- *Spiculated periosteal reaction*: spicules (new bone formations) that grow along Sharpey's fibers; indicate more aggressive tumor growth compared to the solid and lamellated types.
5- *Hair-on-end appearance*: spicules extend perpendicular to the bone surface

B-Interrupted periosteal reaction

Codman triangle: Develops as a result of the destruction of singular or multiple periosteal lamellae

Radiological findings that indicate benign bone tumor

* Well-defined localized, sclerotic margins.
* No cortical disruption or periosteal reaction.

Benign bone tumors

I. Bone producing

A. Osteoid Osteoma: between 5-30 years of age.

Classic symptom: night pain relieved by aspirin or (NSAIDs).

Causes of pain

1- Nerve fibers within the nidus likely play a role in producing pain.
2- High prostaglandin and cyclooxygenase levels within the lesion.

Most common locations

= The proximal femur, (hip is the most common intra-articular location).

= Spine → painful scoliosis (at the center of the concavity of the curve).

Osteoid Osteoma causes extensive inflammatory symptoms in the adjacent tissues (joint effusions, contractures, limp, and muscle atrophy).

Imaging appearance:

1-Plain Radiograph

a. Round, well-circumscribed intracortical lesion with radiolucent nidus
b. Lesions usually <1 cm in diameter.
c. The extensive periosteal reaction may obscure the nidus.

2-Technetium Tc 99m Isotope bone scans Intense focal increased tracer uptake.

3-Thin-cut CT scan (1mm):

Key to diagnosis because it frequently identifies the small radiolucent nidus.

Treatment/outcome

1- The standard of care is percutaneous radiofrequency ablation (PRFA)
A CT-guided probe inserted into the lesion with the temperature 90°C for 4-6 minutes to produce a 1cm zone of necrosis.

B. Osteoblastoma: Aggressive, benign osteoblastic tumor.

Clinical presentation

a. Slowly progressive dull, aching pain of a long duration that is less severe
b. Night pain is not typical, and aspirin does not classically relieve the symptoms.
c. Neurologic symptoms (osteoblastoma most common in the spine).

Imaging appearance,
a. Radiolucent lesion 2-10 cm in size with occasional intralesional densities
b. Cortically based (two-thirds)
c. Expansile with extension into the surrounding soft tissues and a rim of reactive bone around the lesion.
d. (CT, MRI): to evaluate the extent of the lesion before surgical treatment.

Histology as osteoid osteoma, but with increased giant cells.
Treatment/outcome
In most cases, →curettage and bone grafting.
Occasionally, Enbloc resection is required for lesions in the spine.

II. Cartilage producing

1- Enchondroma

- Small bones in the hands and feet can be painful after a fracture. 50% in hands **(**most common bone tumor in hand**).**
- In the medullary cavity of long bones, noted incidentally (If painful suspect low-grade ch. sarcoma). The incidence of malignancy is <1%.

Imaging appearance,
A well-defined, Lucent, central medullary lesion.
The classic radiographic appearance involves rings and stippled calcifications within the lesion: popcorn appearance or salt and pepper appearance.
The cortices in hand enchondromas may be thinned and expanded.
A periosteal chondroma: a surface lesion that creates a defect in the cortex.
Cortical thickening or destruction suggests chondrosarcoma.

Treatment: Asymptomatic → no treatment, otherwise curettage, and bone graft
Related conditions:
I-Ollier disease: Multiple enchondromas
II-Maffucci syndrome: Multiple enchondromas and soft-tissue hemangiomas.

2- Osteochondroma: Most common benign bone tumor.
Usually around the knee (distal femur, proximal tibia), proximal humerus.
It arises from the perichondral ring and extends into metaphysis with growth.

Clinical presentation
Painful swelling from an inflamed overlying bursa, fracture of the stalk, nerve , or tendon compression. Osteochondromas continue to grow until the patient reaches skeletal maturity. The risk of malignant change in solitary osteochondroma is < 1%. Osteochondromas can be sessile or pedunculated. There is a risk of malignant degeneration in sessile lesions.

Plain radiograph
The medullary cavity of the bone is continuous with the stalk of the lesion.
The cortex of the underlying bone is continuous with the cortex of the stalk.

CT or MRI scans can better evaluate the cartilage cap and are useful when

malignant degeneration is a concern.
The gross appearance of a pedunculated lesion is similar to a cauliflower.

Related condition:
Multiple hereditary exostoses, An autosomal dominant pattern
The risk of malignant transformation is (~5%).
The most common location of secondary chondrosarcoma is the pelvis.
Usually, the malignant tumors are low-grade.

3- Chondroblastoma.

80% of patients are younger <25 years. It is thought to arise from the cartilaginous epiphyseal plate. Patients present with progressive pain.
Most chondroblastomas are found around the knee followed by the proximal humerus, and proximal femur. <1% develop benign pulmonary metastasis.

Imaging appearance
Round small radiolucent epiphysis or apophysis, may extend to the growth plate and metaphysis, usually originating in the center of the epiphysis.
Most are 1- 4 cm in size and have a sclerotic rim.
One- third of chondroblastoma have areas of secondary ABC.

III. Fibrous / Histiocytic producing

1-Nonossifying fibroma (NOF)
Very common skeletal lesions (age 5-15 years) frequently called fibrous cortical defect (FCD) or metaphyseal fibrous defect.
Usually, an incidental finding in 30-40% of children lower extremity (80%).
May present with pathological fracture.

2-Fibrous dysplasia. (FD)
Developmental, characterized by hamartomata's proliferation of fibro-osseous tissue within the bone. 75% in patients <30 years

The radiographic appearance of fibrous dysplasia
1-Central lytic lesions within the medullary canal, usually diaphysis/metaphysis. 2- Well-defined borders, endosteal scalloping with intact cortex & periosteum.
3-Sclerotic rim.
4-May be expansile with cortical thinning, "Ground glass" or "shower- door glass" appearance.
(**Ground glass**: glass with a smooth ground surface that renders it nontransparent while retaining its translucency)
5-A shepherd crook deformity refers to a coxa Varus angulation of the proximal femur, classically seen in femoral involvement by fibrous dysplasia.

Other problems of FD
- Vertebral collapse and kyphoscoliosis,
- Extending long lesions in a long bone.

Histology "Chinese letters" or "alphabet soup" appearance.

V. Cystic Lesions

A. Unicameral bone cyst (UBC).
A benign lytic lesion that seen at the dia /metaphyseal region of the proximal humerus. A common, serous fluid inside bone lesion, occurs in patients younger than 20 years. The cyst is active initially when adjacent to the growth plate. The cyst appears to move into the diaphysis with growth. The most common presentation is a pathologic fracture after minor trauma. The most common locations include the proximal humerus and proximal femur, but UBCs can occur in the ilium and calcaneus.

Imaging appearance,
= Purely lytic lesion located centrally in the medullary canal.
= Unilocular or multilocular. = Cortical thinning.
= Bone expansion does not exceed the width of the physis.
= **"Fallen leaf" sign** is pathognomonic (fallen cortical fragment into the base of an empty lesion).

B. Aneurysmal bone cyst (ABC)
= A destructive, expansile bone lesion filled with multiple blood-filled cavities.
= 75% of patients are <20 years of age.
= Can be associated with an underlying lesion that is identifiable in 30% of cases (most commonly chondroblastoma, giant cell tumor, chondromyxoid fibroma, non-ossifying fibroma, osteoblastoma, and fibrous dysplasia)

Clinical presentation
Pain and swelling are the most common symptoms. Neurologic symptoms are possible with lesions in the spine. The most common locations are the distal femur, proximal tibia, pelvis, and spine (posterior elements).
Imaging appearance
Lytic lesion located in the metaphysis. Can aggressively destroy/expand the cortex and extend into the soft tissues. The lesion can expand to greater than the width of the epiphyseal plate. MRI shows fluid-fluid levels on T2-weighted images (separation of serum and blood products).

VI- Others: Giant cell tumor

- Occur in patients 20- 45 years of age (90%).
- Present with pain and swelling.
- It is located most commonly around the knee, distal radius, and ankle.

Imaging appearance,
= Lytic lesion located in the epiphysis/ metaphysis of long bones.
= Lesions extend to the subchondral surface with no sclerotic rim.
= Can destroy the cortex and extend into the surrounding tissues.
MRI is helpful only to define the extent of soft tissue involvement, as plain radiographs are usually diagnostic.

Malignant bone tumors

Risk Factors for Bone Cancer
Most people with bone cancer do not have any apparent risk factors.

1- Genetic disorders (Retinoblastoma → osteosarcoma facial bones.
2- Multiple osteochondromas →risk of low-grade chondrosarcoma. Multiple enchondromatosis → risk of low-grade chondrosarcoma Tuberous sclerosis → high risk of chordomas during childhood.
3- Paget disease →osteosarcoma develops in about 1%
4- Ionizing radiation → higher risk of developing bone cancer.
5- Radioactive materials such as radium and strontium → bone cancer
6- Bone marrow (stem cell) transplantation →Osteosarcoma

= Primary (arising from abnormal bone or cartilage cells)
= Secondary: on top of a benign lesion.
= Remember stress fracture can mimic osteosarcoma histologically.
The most common primary malignant bone tumor is Osteosarcoma

1- Osteosarcoma

A malignant bone-forming tumor is arising from osteoblasts.
2ry osteosarcoma: Paget's disease of bone. (high malignancy with early Mets)
Primary osteosarcoma: 10-20 years.
Increased incidence in patients with retinoblastoma and Li Fraumeni syndrome

Clinical findings
Pain (progressive, worsens at night and with activity);
Swelling after trauma to the bone.
Limping and decreased range of motion.
Site: around the knee, (90%) arise from the metaphysis; 10% from the diaphysis.
Distal Femur: most common site (40%; about twice as common as proximal tibia)
Proximal Tibia: 2nd most common site
Proximal Humerus: 3rd most common site
Metastasis: lungs, bone.
Skip metastases: metastases within the same bone, or across the joint.

Diagnostics
1-Conventional x-ray
-Mixed sclerotic, lytic & permeative lesions commonest radiographic presentation.
-or Purely osteoblastic, permeative lesion: dense sclerosis and osteoid production.
-or Purely lytic, permeative lesion: little osteoid production and/or minimal calcium deposition in osteoid.
Most conventional osteosarcomas (90-95%) extend through the bone, the soft tissues and form a soft tissue mass outside of the bone.
Sunburst appearance of lytic bone lesions and /or Codman triangle.

2-MRI with contrast (gadolinium)
3-CT Scan:

Laboratory: ↑ alkaline phosphatase, ↑ LDH, ↑ ESR

Prognosis: The five-year survival rate of ~ 70%

2- Ewing sarcoma

Small round blue cell sarcoma, age 5-15y.
A highly malignant bone tumor is arising from neuroectodermal cells.
85% of Ewing sarcoma is associated with a characteristic chromosomal translocation t (11;22) (q24; q12) that results in EWS/FLI-1 chimeric protein.

Clinical features
Localized pain (progressive, worsens at night), hyperthermia, and Swelling after trauma to the bone.
Localization
Primary tumor: often diaphysis of long bones (femur most common), tibia, fibula, and humerus) and bones of the pelvis. **Metastasis**: Lungs, Bone, Bone marrow

Diagnosis
1-Conventional x-ray:

- Permeative or moth-eaten bone destruction.
- Soft tissue mass in 90% of cases.
- Periosteal Reaction in 50% of cases (Onion Skin peel appearance).
- Hair on end (rapid continuous lifting of periosteum).

2-MRI 3-CT chest and abdomen 4-Isotope bone scan
5-Bone marrow aspiration 6-Laboratory findings: ↑ ESR, ↑ LDH, leukocytosis.
Prognosis

- Extremely aggressive, early metastasis.
- The five-year survival rate of ~ 70%.
- Pelvic Ewing sarcoma has a worse prognosis than other areas.
- Patients 10 y or younger have a better response to treatment.

N.B: Patients under five years of age should be carefully evaluated to exclude metastatic neuroblastoma

3- Chondrosarcoma

A malignant tumor arising from mesenchymal cells that produce cartilage.
Types

- 1ry chondrosarcoma 65%: popcorn calcification associated with pain. Cortical scalloping, (poor prognosis), age >50y.
- 2ry chondrosarcoma: e.g., Osteochondroma degeneration of the cartilage cap, younger age (good prognosis)

Clinical features
- Deep, dull pain (worsens at night, insidious progression over the years)
- Local swelling - Pathological fracture in primary chondrosarcoma.
Localization: mainly pelvis, proximal femur, and proximal humerus
Diagnostics: Conventional X-ray or CT.

Osteolysis with a **moth-eaten appearance**

In 1ry chondrosarcoma →calcifications (rings and arcs calcification, popcorn calcification), endosteal scalloping, cortical breach & infiltration of soft tissue.
In long bones: ill-defined margins of the lesion, calcification, the presence of a soft tissue mass, and cortical destruction.
In 2ry chondrosarcoma→ irregular cartilage cap with popcorn calcification.

Treatment
Surgery (definitive resection). resistant to chemotherapy and radiation therapy
Prognosis

- The five-year survival rate of ~ 50–60%.
- Late recurrences are possible → regular follow-ups for ten years.

Bone metastasis

- From Breast, Prostate, and Lung cancer (**80% of cases**), Thyroid, and Kidney.
- Bone metastasis comprises 70% of all malignant bone tumors
- Bones are the 3rd most common site of metastasis, after the lung and the liver.

Localization:
Most commonly spine (~ 40%), pelvis (~ 20%), and proximal femur or humerus.

Radiological Classification:
1-Osteoblastic metastasis: prostatic cancer, small cell lung cancer.
2-Osteolytic metastasis: thyroid ca, renal ca, melanoma, M. Myeloma
3-Mixed metastasis Breast cancer, GIT cancer

Clinical findings
1-Local pain and swelling 2-Pathological fracture
3-Spinal cord compression and/or radicular symptoms

Diagnostics
Laboratory findings (e.g., elevated alkaline phosphatase, hypercalcemia)

Investigation,
For suspected extremity lesions: Conventional X-ray (initial test), followed By lab workup, CT, and MRI if pathological # is identified or suspected for operative planning

PET Scan: useful to detect metastases and for staging, particularly for bone metastasis from breast and lung cancer.

Biopsy.
Preferably taken from a soft tissue mass at the afflicted site.

15-PEDIATRIC SPINE

Kyphosis: abnormal forward curvature of the thoracic spine (hunchback).
Lordosis: abnormal curvature of the lumbar spine, also called (swayback).
The respiratory system develops by the age of 5 years, any spinal deformity before this will affect the pulmonary function

Scoliosis

Definition: 3D abnormal curvature of the spine.
Structural curve: 3D deformity of the spine (curves to the side, rotation of the vertebra, and kyphosis / Lordosis) in the coronal, axial, and sagittal plane.

N.B: If Cobb angle > 10° →scoliosis (error of 3-5°)
Postural curve: sideway bending of the spine.

Types of Scoliosis

A-Nonstructural Scoliosis (postural)
1-LLD 2- Hip contracture 3- Pelvic tilt 4-Discitis 5-Disc herniation

B-Structural Scoliosis
1-Idiopathic
2-Congenital
3-NM (CP. DMD)
4-Genetic: Marfan's S, Down S

C-Pathologic Scoliosis: Painful mild scoliosis: Osteoid O, Osteoblastoma.

Postural scoliosis:
It is a secondary or compensatory deformity to a problem outside the spine.
Diagnosis
1- Positive outside-the-spine problem.
2- Adam bending test → disappearance of deformity.
3- When the patient sits on the couch→ disappearance of the deformity.
Plain radiograph: = Small curve = No rotation of the vertebra

I-Idiopathic scoliosis 75%.

A- According to age	B- Onset
1. Infantile: onset before three years of age 2. Juvenile: onset between 3 and ten years of age 3. Adolescent: onset after ten years of age	= Early onset: <10y = Late onset : >10y

1-Infantile Idiopathic Scoliosis	B-Juvenile Idiopathic Scoliosis (JIS)
= 4% of idiopathic scoliosis = More common in boys = Usually left thoracic curve = Plagiocephaly (skull flattening) = 90% will resolve spontaneously	= 15% of all idiopathic scoliosis = Right main thoracic curve = 18-25% Syringomyelia detected by asymmetric umbilicus reflex = 70% →treatment (50% bracing, 50% surgery).

Imaging OF JIS
= PA and lateral upright images of the whole spine
= MRI: Indicated in children <10 yrs. old with a curve > 20° even in normal neurologic findings to rule out (e.g., Syringomyelia, Dural ectasia, and low-lying conus)

C-Adolescent Idiopathic Scoliosis (AIS)

Affect children > 10 -18y. The most common type of scoliosis
Epidemiology

- 10:1 female-to-male ratio for curves > 30°
- 1:1 male-to-female ratio for small curves
- Right thoracic curve most common
- Left thoracic curves indicate an MRI to rule out cysts or Syrinx.
- Most have a positive family history.
- The primary thoracic curve is usually convex to the right
- The primary lumbar curve is usually convex to the left

Signs of the structural curve.

1- Shoulder height differences.
2- Truncal shift
3- Rib rotational deformity (rib prominence)
4- Waist asymmetry and pelvic tilt

Clinical findings suggestive of intraspinal pathology (tethered cord or a syrinx)
= Asymmetric abdominal reflexes,
= Clonus,
= Muscle weakness or contractures
= Foot deformities.
= Hairy patches, dimples, nevi, hemangioma, cafe-au-lait spots.

VII-Special tests
Adams forward bending test.
To check for the structural curve (examiner stands from behind, if positive → Thoracic rib hump).

16-(Osteoarthritis) (Osteoarthrosis) (OA)

Definition: Progressive degeneration of articular cartilage in synovial joints and formation of new bone in the subchondral region and at joint margins.
OA is a condition caused by degenerative "wear and tear" of the joints.
Most common joint disorder in adults.
The most common cause of disability in orthopaedics

Risk factors for OA
1-Older age, Obese Female *2-Genetic inheritance* *3-Race & ethnicity*
4-Previous joint injury *5-Metabolic syndrome*
6-Local mechanical factors
* Mal-alignment, muscle weakness, internal derangements.
* Excessive joint loading. * Joint overuse. * Joint instability.

Classification
1- Idiopathic OA
2- Secondary OA
** Trauma* **Previous joint disorders* **Infection*
** Metabolic: Gout* ** Inflammatory: Rhd A, AS*

Etiology

Non-modifiable risk factors	**Modifiable risk factors**
*Age (> 55 years) *Familial history *History of joint injury or trauma *Anatomic factors causing asymmetrical joint stress.	* Obesity * Excessive joint *loading or Overuse

Pathophysiology
Joint damage/stress → Cartilage damage →decrease proteoglycan level → cartilage becomes soft, friable and inelastic → fraying and fibrillation → cracks, extending more deeply into the cartilage → degradation → loss of joint space and bony surface → subchondral bone becomes thickened and sclerotic.

Pathoanatomy

1- Articular cartilage
- Increased water content
- A decrease in the amount of proteoglycans
- Loss of collagen organization and orientation

2- Synovium and capsule
Early OA →mild inflammatory changes in the synovium
Late OA → Synovium becomes increasingly thick and vascular

3- Bone
- Sub-chondral bone remodeling →lytic lesion with sclerotic edges
- Bone cysts form in late stages

- Osteophytes form through the pathologic activation of endochondral ossification

Cardinal pathological features of OA

1-Progressive thinning of the articular cartilage → narrow joint
2-Subchondral sclerosis and cyst formation 3-Osteophytes 4-Synovitis
5-Capsular thickening and fibrosis.

Symptoms

1- Pain
   Activity-related or mechanical.
   Exacerbated by use and alleviated by rest.
   Usually insidious in onset; nocturnal in advanced disease.
2- Morning stiffness of brief duration.
3- Reduced range of motion and crepitus**.**

Sources of pain in OA knee

• Inflamed synovium • Microfracture of subchondral bone
• Bursal inflammations • Periosteum stretching by osteophytes
• Venous congestion in intraosseous compartment • Joint distension • Muscle spasm • Affect joint mechanics • Mental depression.

Signs

1-Crepitus**:** audible, palpable grating quality when the knee is flexed /extended**.**
2-Angular deformity (genu varum)
3-Knee swelling in synovitis
4-Tenderness
5-Limited ROM.

N.B: Spine and hip disorders can refer pain to in the knee (evaluate both anatomical sites to isolate the origin of pain)

Associated conditions with OA.

1-Heberden's nodes: nodular thickening on the dorsal sides of the (DIP), ♀ > ♂
2-Bouchard's nodes: nodular thickening on the dorsal sides of the PIP, ♀ > ♂.
3-Hallux rigidus: Arthrosis of the 1st MPJ, characterized by hypertrophy of the sesamoid bones.

In contrast to osteoarthritis, rheumatoid arthritis does not affect the MPJ or DIP joints.

Imaging studies of knee OA

- Diagnose OA based on history and physical exam
- Radiographs are insensitive to early pathologic features
- Plain-film radiography can confirm the clinical suspicion

X-rays (3 views)

1- Weight-bearing AP 2-Lateral 3-Tangential Patellar (Sunrise)

Radiographic findings in OA (remember acronym **LOSS**).

- **L**oss of joint space (thinning of cartilage).
- **O**steophytes (attempts to repair underlying bone).
- **S**ubchondral sclerosis (hardening of bone in response to load).
- **S**ubchondral cysts (resulting from microfractures).

Clinical Criteria.
Sensitivity 95%, specificity 69%
1-Knee pain plus at least 3/6 characteristics
2- >50 y **3**-Morning stiffness < 30 m
4-Crepitus **5**-Bony tenderness **6**-Bony enlargement

N.B: Radiographic signs do not correlate with the patient's perception & clinical findings.

Osteoarthritis Management (AAOS recommendation)

- **NSAIDs (strong evidence)**
- Weight loss (moderate evidence)
- Exercise/physical therapy (strong evidence)
- Total joint arthroplasty

The general approach to OA

1- Diminish joint pain, and enhance functional capacity.
2- Treatment depends on functional impairment and severity of symptoms but not on radiographic findings.
3- Modify treatment according to responses to therapy.
4- Begin with PT, OT, and weight loss.

Aims of knee OA treatment

• **R**elieve pain • **R**estore function
• **R**educe disability if any by **R**ehabilitation.

A-Nonpharmacologic treatments.

1-Patient education 2-Weight loss (if overweight)
3-Aerobic exercise programs 4-Physical therapy.
5-Range-of-motion exercises, Muscle-strengthening exercises.
6-Assistive devices for ambulation, e.g., Cane
7-Occupational therapy 8-Topical Ice and heat therapy

B-Pharmaceutical options If the above efforts did not improve the function
1-Paracetamol Caution in advanced hepatic disease
2-NSAIDS given in moderate-severe pain, **e.g: Cox-2 inhibitors**
It has fewer GI side effects.

3- Opioid analgesics: e.g., Tramadol.
4- Intraarticular Depomedrol Injections, If pain persists despite oral analgesics
5- Hyaluronans if pain persists despite other agents

C-Surgical therapy

- Severe joint pain, resistant to conservative treatment methods
- Limitation of daily living activities
- Deformity, angular deviations, instability

Total Knee Arthroplasty.

- **Indication:** 1-Diffuse arthritis 2-Severe pain 3-Functional impairment
- Pain relief > functional gain.
- Prosthesis 10-yr survival: 90%.

Osteochondritis Dissecans (OCD)

Definition:
A small segment of bone and the cartilage begins to separate from its surrounding region in the joint, due to a lack of blood supply.

Epidemiology

- Juvenile form aged 5-15 years.
- Older adolescents and adults from the disease.
- Occurs in the knee 75%, the elbow 6%, and the ankle 4%.
- In the knee, OCD occurs in the medial femoral condyle 75% of the time,
- In the ankle, OCD occurs in the posteromedial aspect of the talus 56% of the time and in the anterolateral aspect of the talus 44% of the time.

Etiology

- *Multifactorial elements.*
- *Trauma*
  Indirect trauma in case of knee OCD
  In the ankle, traumatic insult is 90%.
  In elbow OCD repetitive microtrauma

Symptoms
Pain and swelling of a joint, often brought on by sports or physical activity, in late cases of OCD may cause joint catching or locking.

A-THE HIP

1-Osteoarthritis of the Hip

Causes
1ry → idiopathic 'wear and tear.'
2ry →
1-Inflammatory arthritis 2-Trauma 3-Osteonecrosis
4-Previous septic arthritis 5-Sickle cell disease.
6-Abnormal hip shape (DDH, old SCFE, and old Perthes')

Clinically

*2ry more common in males over 30 years of age. *Joint stiffness.

*Pain in hip, gluteal & groin areas radiating to the knee (Obturator N).

*Mechanical pain. *Limited walking function.

• History of limited walking distance due to pain, pain going up or downstairs, night pain that interrupts sleep, and pain with sitting, especially with the hip flexed.

Differential diagnosis of hip pain in adult

1-Spine:

- Mechanical back pain → buttock or groin pain.
- Radiculopathy, shooting pains extending below the knee.

2-Knee: knee pathology → thigh pain or limping.

3-Tumor: bony metastases are very common!

4-Stress fracture: NOF in runners, subtrochanteric fractures occur in elderly patients on bisphosphonates due to inhibition of bone remodeling.

5-Greater trochanteric bursitis

Physical examination:

1- Antalgic limping 2-Limitation of ROM (first internal rotation).

3- Painful ROM, +ve Thomas' test. 4-Trendelenburg test positivity.

5- Leg length discrepancy.

• **AP radiographs of the pelvis**

1-Subchondral sclerosis. 2-Narrowing of the joint space.

3-Periarticular cysts. 4-Osteophyte formation. 5-Cystic changes.

Treatment

• NSAIDs plus acetaminophen for pain relief; non–weight-bearing exercise (e.g., swimming); weight loss.

• Corticosteroid injections for patients with moderate joint space narrowing, or to postpone surgery.

• Surgical options: Total hip replacement.

2-Total hip arthroplasty (THA)

Indications: End-stage degenerative joint disease of the hip

Goals of THA

Relieve pain, improve joint mobility, and restore or improve a person's ability to safely perform functional activities like walking, standing, and stair climbing.

Osteonecrosis (AVN)

Infarction of bone and marrow is usually very painful. The most common site is the femoral head (watershed zone) **A** (due to insufficiency of medial circumflex femoral artery).

Common sites of AVN

1- Head of the femur (most common). in fracture neck of femur and

dislocations of the hip (lateral epiphyseal branch of medial circumflex femoral A injury)

2- The body of the talus is fractured through the neck of the talus.
3- Proximal pole of the scaphoid in a fracture through the waist.

Causes

1- Idiopathic (Chandler's disease) – the most common type.
Intravascular thrombosis, extravascular swelling & compression

2- Trauma, e.g., joint dislocation, fracture

3- Secondary nontraumatic causes

a- Alcohol, steroid use. b- Infection c- Gaucher's disease
d- Caisson disease: (N2 accumulates) → block arterioles
e- Hemoglobinopathy and Coagulation disorder: Sickle cell disease
f- Hematological malignancies: Leukemia, lymphoma.
g-Others: SLE, Pregnancy, and irradiation.

Pathology of osteonecrosis

Subarticular bone segmental collapse (Subchondral fracture) of the hip is most clearly seen on the lateral radiograph (Crescent sign).
In the later stages, radiograph shows dense changes in the bone, collapse, and osteoarthritic features.

Clinical presentation

- Limping, thigh pain, or anterior hip pain.
- Initial pain with sit-to-stand, stairs, inclines, and impact loading.
- Limitation of abduction and internal rotation.

Complications of femoral head AVN

-Femoral head collapse -Secondary OA.

Imaging

Plain radiographic changes need months to be seen.

1- Plain radiograph: Pelvis, AP, and frog-lateral of the hips
>50% of osteonecrosis cases have bilateral involvement.

2- MRI (highest sensitivity (99%) and specificity (99%).
In the early stages, avascular necrosis due to trauma is detected by radio-isotope bone scan.
MRI (Investigation of choice) – **Double line sign**

Treatment.

***Nonoperative**

1- Bisphosphonates**:** for pre-collapse AVN
2- In the early stages, protected weight bearing to prevent bone collapse.

****Operative***

1-Hip (femoral head)

A- ***Early:*** **Core decompression with or without bone grafting.**
Before subchondral collapse occurs

B- Late: THR (cementless cup and stem).

17-PERIPHERAL NERVE INJURIES

1- Common nerve lesions

1-Median nerve	2-Ulnar nerve	3-Radial nerve
-Carpal tunnel syndrome -Pronator syndrome -Anterior I N syndrome	-Cubital tunnel syndrome -Ulnar tunnel syndrome	-Radial nerve palsy -Posterior IN syndrome -Radial tunnel syndrome -Cheiralgia paresthetica

2- Peripheral nerve structure.

Composed of bundles (fascicles) of axons conducting

- Efferent (motor) impulses from the AHC of the spinal cord to the muscles.
- Afferent (sensory) impulses from peripheral receptors via cells in the posterior root ganglia to the cord.

*Epineurium covers the whole nerve
*Perineurium covers the fascicle
*Endoneurium covers the axon

Mechanism of injury *Ischemia *Compression *Traction *Laceration

Types of nerve injury

1- **Transient ischemia**
Following acute compression → numbness in 15min. → pain loss after 30 min → muscle weakness after 45 min.
Recovery after 10 min. of the release of compression.
2- **Neuropraxia:** temporary loss of conduction, full recovery days-weeks.
3- **Axonotmesis:** axons disruption → distal regeneration may take 2ys.
4- **Neurotmesis:** complete cut, needs repair, and never returns to normal.

Upper limbs nerve injuries

1-Median nerve (Important)

A- The Sensory Function of the Median Nerve

1-Palmar cutaneous branch – supplies the lateral aspect of the palm (arises in forearm and does not pass through the carpal tunnel)
2-Palmar digital branch – supplies the palmar surface and fingertips of lateral 3½ digits (arises in hand)

B- The Motor Function of the Median Nerve.

Branch	Muscles supplied
Median nerve	- **P**ronator teres - **P**almaris longus - **F**lexor carpi radialis **F**lexor digitorum superficialis
Anterior interosseous N	**F**lexor digitorum prof undus (lateral ½) **F**lexor pollicis longus **P**ronator quadratus

The recurrent branch	**Thenar muscles** - Opponens pollicis - Abductor pollicis brevis - Flexor pollicis brevis
The palmar digital branch	1st and 2nd Lumbricals

A-Carpal tunnel syndrome (CTS). Important

Most common compressive neuropathy in the upper extremity >Idiopathic form

Risk factors

Obesity, Pregnancy, DM, hypothyroidism, Rhd A. Chronic Renal Failure, Advanced age and Vibratory exposure during occupational activity.

Diagnosis

- Paresthesia and pain (often at night) in the volar aspect of radial 3 1/2 digits (thumb, index, long and radial half of the ring).
- **Provocative test**

1- Carpal tunnel compression test (Durkan test). 60 seconds
2- Tinel and Phalen's tests.

- **Sensory testing**

Large sensory fibers (light touch, vibration) are affected before small fibers (pain and temperature).
Semmes-Weinstein monofilament testing is sensitive for diagnosing early CTS. (large fibers).

- Weakness, loss of fine motor control, and abnormal 2-point discrimination are later findings.
- Thenar eminence atrophies but the sensation of the thenar eminence is spared because the palmar cutaneous branch enters the hand external to the carpal tunnel.

Differential diagnoses of CTS.

1-Cervical radiculopathy, 2-Brachial plexopathy, 3-Thoracic outlet syndrome, 4-Pronator syndrome, 5-Peripheral neuropathy.

Treatment

- **Non-operative treatment.**

1- Activity modification, Night splints, and NSAIDs.
2- Single corticosteroid injection yields transient relief in approximately 80% after six weeks, but only 20% are symptom-free by 1year.

- **Operative treatment options**

1- Open, Mini-open
2- Endoscopic release of the transverse carpal ligament.

B-Anterior interosseous nerve syndrome (AIN)

Causes of Compression

Tendinous edge of the deep head of pronator teres. **(**most common)

Clinical picture.
= Involves motor loss of FPL, FDP (index +/− middle finger),& pronator Q.
= No sensory disturbance
= Index FDP and thumb FPL tested by asking the patient to make an "OK" sign (precision pinch),
Benediction hand:
In contrast to the 'ulnar claw,' it is only apparent if the patient is asked to make a fist.

2-Ulnar nerve

Guyon canal syndrome (Ulnar tunnel syndrome)
Compression of ulnar N at the wrist, seen in cyclists due to pressure from handlebars.

The nerve damaged by trauma, deformity from malunion (tardy ulnar palsy, pressure on the medial side of the elbow on the operating table.

Classical examination findings are secondary to motor weakness.

- **Froment's sign**
  Compensatory thumb interphalangeal joint flexion (FPL) due to weak adductor pollicis.
- **Wartenberg sign**: Persistent abduction and extension of small digit during attempted adduction due to weak third palmar interosseous and small finger lumbrical.
  A positive result occurs when the patient is unable to adduct the abducted small finger.
- **Interosseous and web space atrophy.**

What is the ulnar paradox?
The higher the lesion of the ulnar nerve injury, the less prominent the deformity, and vice versa. This is because in higher lesion the FDP is paralyzed →the loss of finger flexion makes the deformity looks less obvious.

Summary of the Ulnar nerve tests.

Card test	Palmar interossei
IGAWA test	Dorsal interossei
Book test / Froment's sign	Adductor pollicis
Wartenberg's sign.	3rd palmar interosseous and small finger lumbrical

3-Radial nerve

1. Proper radial nerve palsy.
Saturday night palsy (weakness triceps, brachioradialis, and ECRL plus muscles innervated by the PIN). Sensory deficits in the distribution of the superficial sensory branch. Exploration if no significant recovery after 3 m.

***2*. Radial tunnel syndrome (PIN compression syndrome)**
Characterized by the lateral elbow and radial forearm pain without motor or sensory dysfunction.
The most common cause is compression by the arcade of Frohse.

3-Traumatic PIN Palsy

* Dislocation head of radius * Fracture neck of the radius

Clinical picture

Fingers drop, radial deviation with active wrist extension (because of the ECRL innervated by proper radial nerve more proximally).

PIN innervates the (ECRB, Supinator, EIP, ECU, extensor digitorum communis (EDC), extensor digiti minimi, APL, EPB, and EPL).

4-Cheiralgia Paresthetica (Wartenberg syndrome)

Compressive neuropathy of the superficial sensory branch of the radial nerve Compressed between the brachioradialis and ECRL with forearm pronation (by a scissor-like action between the tendons).

Symptoms include pain, numbness, and paresthesia over the dorsoradial hand. Surgical decompression is warranted if a 6-month trial of nonoperative treatment fails.

Nerve Specific Sensory autonomous part

•Median Nerve → Pulp of the index

•Ulnar Nerve → Pulp of the small finger

•Radial Nerve → Dorsum of 1st web space.

Lower limb nerve injuries

Sites of lower limb nerve injury.

1-Lumbar nerve roots – prolapsed intervertebral disc.

2-Sciatic nerve:

= Damage to the lateral half of the sciatic nerve from traumatic posterior dislocation of the hip, or THR

= Common Peroneal nerve passes around the fibular neck.

Causes CPN injury

1- # Neck of the fibula
2- Knee dislocation
3- Tight plaster
4- Lateral collateral ligament injury
5- Lying in bed with the leg in external rotation.

Peripheral N injuries due to fractures

Nerve	Trauma	Effect
Axillary nerve	Ant. Shoulder Dislocation # surgical neck	Deltoid palsy
Radial nerve	Humeral shaft # (lower 3rd)	Wrist + finger drops
Ulnar nerve	Medial H condyle #	Claw hand
Sciatic nerve	Posterior hip dislocation	Foot drop
Common Peroneal Nerve	Knee dislocation, The the neck of the fibula #	Foot drop

Post. Interosseous Nerve	Monteggia fracture	Fingers drop
Ant. Interosseous Nerve	Supracondylar H #	OK sign
Median nerve	Supracondylar H #	Pointing index
C5-6 Br. Plexus	Erb's palsy	The waiter's tip def.
C8-T1	Klumpke palsy	Claw hand

Causes of Foot-Drop

General → *Stroke, MS, CP, CMTS, Polio, MD, leprosy*

Local

At the spine (• Spina bifida • Tumors • Disc prolapse, etc.)

- *At the hip* • Posterior dislocation of the hip
- *At the gluteal region* → • Deep intramuscular injections.
- *At the thigh* (• Fracture shaft femur. • Penetrating injury and a gunshot injury.

At the knee (common causes)

• Dislocation of knee. • Fracture lateral condyle of the tibia.
• Tumors. • Tight plaster casts around the knee. • Poor padding during traction.
• *Direct injuries* (gunshot inj., incised and penetrating injuries, etc.)

Bilateral drop feet

1- Peripheral neuropathy 2- Cauda equina syndrome
3- Motor neuron disease 4- Spastic paraparesis
5- Bilateral L5 radiculopathy

Symptoms

1-High steppage gait 2-An exaggerated, swinging hip motion
3-Tingling, numbness & slight pain in the foot 4-Muscle atrophy in the leg

Differential Diagnosis of drop foot

1- Weak dorsiflexion & eversion, decrease sensation dorsum of the foot → CPN palsy
2- Weak dorsiflexion, eversion, and inversion → L5 radiculopathy
3- Weak dorsiflexion, plantarflexion, eversion, and inversion → Sciatic nerve
4- UMNL signs, e.g., Babinski reflex +ve →Central lesion

Investigations

Electromyography (EMG) and nerve conduction studies

18-KNEE DISORDERS AND SPORTS

I-Meniscal injuries and diseases

A. Meniscal Tears

= Caused by axial loading with rotation, but maybe a trivial incident
= Lateral meniscus tears may be associated with ACL tears and major sports injuries.
= The medial meniscus is torn approximately three times more often than the lateral meniscus.
= Patients may complain of pain at the joint line area, locking, clicking, and giving way, and swelling with activity.
= Swelling (effusion) in the joint after 24 hours.
= Joint line tenderness to palpation
• Obtain radiographs to rule out extra-articular causes of knee pain.

Severe knee injury involving complete or partial tears of three major structures of the knee occurs with contact sports or MVA

Unhappy Triad (ACL tear + MM tear + MCL tear).

There is an increased rate of OA in knees after meniscal tears and meniscectomy.

Clinical picture

1-Instant acute swelling. 2-Audible pop or tear in the knee at the time of injury.3-Pain upon knee movement., 4-Bruising appears within a couple of days.

Types of meniscal tears.

Traumatic meniscal tears are common in young patients with sports-related injuries. Degenerative tears usually occur in older patients and can have an insidious onset. Tears in the peripheral third have the highest potential for healing MRI or arthroscopy confirms the Dx.

B. Meniscal cyst. Occur in conjunction with horizontal cleavage tears of the lateral meniscus.

C. Popliteal (Baker's) cysts

A fluid-filled cyst that occurs between the medial head of the gastrocnemius and semimembranosus tendons.

The name of Baker's cyst derives from the **London surgeon M.W. Baker**. Normally a weakening of the joint capsule exists (mostly in combination with meniscus damage).

Symptoms

Mass and pain in the posterior aspect of the knee; the mass tends to enlarge after vigorous exercise and subside during rest. Cysts are frequently associated with intra-articular pathology (i.e., meniscal tears, degenerative arthritis, or rheumatoid arthritis). 82% of popliteal cysts are associated with meniscal tears (two-thirds, medial meniscus tears; one-third, lateral)

MRI is more frequently used because it is also helpful in identifying concurrent intra-articular pathology.

Knee Arthroscopy

1. This is the gold standard for the diagnosis of knee disease.
2. The benefits of arthroscopic surgery include (smaller incisions, less morbidity, improved visualization, and decreased recovery time).

Complications of knee arthroscopy

1. Iatrogenic articular cartilage damage.
2. Instrument breakage, hemarthrosis, infection, and injury to the infrapatellar branches of the saphenous nerve.

2-Ligaments injuries

A. ACL injury

Mechanism of injury (non-contact injury)

- Rapidly changing direction
- Deceleration coupled with cutting, pivoting, or sidestepping moves
- Suddenly stopping
- Slowing down while running
- Direct contact or collision (like a football tackle)

Symptoms of ACL injury

* A "popping" noise * The knee gives out *Loss of ROM
* Discomfort while walking
* Pain with swelling (hemoarthrosis) within 24 hr. after the injury.

50% of ACL injuries are associated with other injuries such as the meniscus, articular cartilage, other ligaments, or bruising of the bone.

ACL+ MCL + Medial meniscus injury → "unhappy triad."

Grades on a severity scale.

Grade 1 Sprains – slightly stretched (the knee joint stable).
Grade 2 Sprains – loose. (a partial tear), rare.
Grade 3 Sprains – complete ligament tear, (knee joint is unstable).

History and physical examination.

Noncontact pivoting injuries are associated with an audible "pop" and an immediate hemarthrosis.

Associated injuries,

Acute lateral meniscal tears are more common.
Medial meniscus tears occur > with chronic ACL deficiency.
The Lachman test is the most sensitive examination for acute ACL injury, followed by the anterior drawer test and the pivot shift test.

Performance on the pivot shift test most closely correlated with outcome after ACL reconstruction. Done under GA

Plain radiographs

A lateral capsule sign or Segond fracture may be present.

(An avulsion fracture of the anterolateral capsule of the knee and posterior fibers of ITB).
MRI is useful in confirming the diagnosis.

Chronic ACL deficiency is associated with a

- Higher incidence of complex meniscal tears and chondral injury.
- Bone bruises (trabecular micro-fracture) occur in > 50% of acute ACL injuries.

Prevention of ACL injury
Athletes should participate in neuromuscular and proprioceptive strengthening, strengthening of knee flexors, and conditioning programs. These should include plyometric exercises and coaching regarding proper positioning while landing.

B. Posterior Cruciate Ligament (PCL) Tear

• **Mechanism**: dashboard injury to the tibia or a fall on a flexed knee
C/O: pain and instability
O/E: positive "sag" (Godfrey) test and a positive posterior drawer sign at 90° (>10 mm, compared with the opposite knee)
MRI is diagnostic for PCL tear.

3- Collateral ligament injury

Medial Collateral Ligament (MCL) Injury

The MCL functions
Stabilize the knee (keeps it from sliding inwards) and allow it to rotate. Caused by direct valgus stress to the knee or non-contact rotational injury.
Three grades of injury, based on the opening of the knee to valgus stress at 30° flexion.
Grade I: 1–4 mm, **Grade II:** 5–9 mm, **Grade III**: 10–15 mm.
Immediate swelling is more likely to be an (ACL) tear or fracture
• Grade III injuries are often less painful than lower grades
• Obtain radiographs to rule out other pathology;
• MRI to confirm the diagnosis.

Symptoms:
Pain on the inner side of the knee, the feeling of instability, hearing a "pop" sound, possible swelling (ACL injury) and bruising.
Patient history, the examination of the knee, and some imaging studies (MRI) are used to make a diagnosis of MCL injury.

Lateral Collateral Ligament (LCL) Injury.

Three grades of injury (I–III), based on the opening of the knee to varus stress at 30° of flexion, Grade III is complete
• Varus laxity at 30° of flexion indicates an isolated injury; varus laxity at 0° indicates a more extensive injury.
• Average laxity at 30°of flexion is 7° of opening

Symptoms:
Swelling, pain on the outer side of the knee, and instability. It is diagnosed using patient history, the examination of the knee and imaging studies, and radiographs to rule out other pathology. MRI can confirm the diagnosis

.

Functions:
1-Fulcrum for the quadriceps 2-Protects the knee joint
3-Enhances lubrication and nutrition of the knee.

4-Diseases of the patella

I-Patellofemoral Pain / Idiopathic chondromalacia patellae (Patellofemoral syndrome) (important)

Idiopathic articular changes of the patella, are more common in females.
Symptoms
Diffuse pain in the peripatellar or retro patellar area of the knee (major symptom)
Aggravated by specific daily activities including

- Climbing or descending stairs
- Prolonged sitting with knee bent (known as theatre pain)
- Squatting or kneeling

Physical exam
1-Quadriceps muscle atrophy 2-Signs of patella mal-tracking.
3-Increased femoral anteversion or external tibial rotation
4-Lateral subluxation of patella or loss of medial patellar mobility
5-Positive patellar apprehension test 6-Palpable crepitus
7-Pain with compression of the patella with knee range of motion or resisted knee extension.

Radiological imaging

- **Plain radiographs of the knee**
  (Sunrise view is essential)→ slight subluxation of the patellae.
- **MRI.** best modality to assess articular cartilage.T2 best sequence to assess the cartilage. (abnormal cartilage is seen as a high signal compared to normal cartilage)**.**

Treatment
• Ice, rest, and NSAIDs for acute pain are indicated, along with quadriceps strengthening and hamstring stretching
• A patellar stabilizing brace may be helpful.

II-Patellar instability
Injuries that occur when the patella, is displaced from its resting place

Types
1-Acute traumatic
2-Chronic recurrent subluxation episodes, > in women, associated with malalignment.

3-Habitual, painless, occurs during each flexion movement, due to tight ITB and vastus lateralis.

Risk factors

1- General factors

A-Ligamentous laxity B-Previous patellar instability event

C-Malalignment syndrome" →increased Q angle.

(Femoral anteversion, Genu valgum, External tibial torsion, Pronated feet.)

2- Anatomical factors

1-Patella Alta 2-Trochlear dysplasia 3-Excessive lateral patellar tilt.

4- Lateral femoral condyle hypoplasia.

5-Dysplastic vastus medialis oblique (VMO) muscle.

Radiographs

Rule out a fracture or loose body, medial patellar facet (most common)

- **AP views**: to evaluate lower extremity alignment and version
- **lateral views**:
  = To assess for trochlear dysplasia
  = Evaluate for patellar height (patella Alta vs. Baja)
- **Sunrise / Merchant views**
  = Lateral patellar tilt.
  = Lateral patellofemoral angle (normal: angle opens laterally)
  = Congruence angle (normal is -6°)

MRI

* Suspected loose bodies. * Osteochondral lesion and/or bone bruising
* Medial patellar facet (most common) * Lateral femoral condyle
* Tear of MPFL (tear usually at medial femoral epicondyle)

III-Jumper's Knee (Patellar Tendinopathy)

• Described as tendonitis or tendinopathy.
• Common in volleyball players; caused by eccentric overloading
• Pain occurs along the patellar tendon and the inferior pole of the patella and is elicited when the knee flexed while actively trying to extend.

Radiographs are negative, but an ultrasound may be helpful.

5-Bursae around the knee

Bursae are small, jelly-like sacs located throughout the body, including around the shoulder, elbow, hip, knee, and heel.
They contain a small amount of fluid and are positioned between bones and soft tissues, acting as cushions to help reduce friction.

Prepatellar bursitis.

Inflammation of the bursa in front of the kneecap (patella). Caused by pressure from constant kneeling, Plumbers, carpet layers, and gardeners are at greater risk for developing the condition.

6-D.Dx of knee pain.

1- Referred Pain	2-Patellofemoral pain	3-Knee joint Disorders	4-Periarticular disorders
From hip	-Patellar instability -Patellar chondromalacia -Osteochondral lesion -OA	-OCD -Loose body -Synovial chondromatosis -Plica syndrome	-Patellar tendinitis -Bursitis -Osgood Schlatter disease

SPORTS MEDICINE

Definition
Sports Injuries: damage to the body tissues, as a result of sport or exercise.
The most common sports injuries are (more strains) and sprains.
Sprains: Sudden stretching to ligaments past their limits→ tears them.
Strains: Tears in the muscle fibers or tendons.

Ligaments and muscle-tendon units like springs (lengthen with stress and return to their normal length, unless it is pulled too far out of their normal range)

Common sites of Sports-related injuries

Site	Pathology	Sport
Bone	Metatarsal stress # Tibial stress syndrome Osgood Schlatter disease	Running Running & Dancing Running
Tendon	Patellar tendinosis (Jumper's knee) Achilles tendinitis Supraspinatus	Volleyball Running Swimming
Joint	SLAP lesion	Throwing athletes
Ligament	Ulnar collateral ligament injury	Baseball
Muscle/fascia	Iliotibial band syndrome	Running
Bursa	Trochanteric bursitis	Race walking
Nerve	Ulnar neuropathy (handlebar palsy)	Cycling

Evaluation of Common Sports Injuries

A- Athletic injuries

1-Acute injuries: caused by sudden trauma (contusions, strains, and sprains)

2-Chronic injuries: e.g., stress fractures.

B- Soft Tissue Injuries

1-Closed

- ***Contusion:*** An injury to soft tissue that results from direct trauma and is usually caused by striking a body part against a hard object.
- ***Hematoma:*** A large area of local hemorrhage.

2-Open: *Laceration,* an injury in which the skin continuity is torn

C- **Joint Injuries.** Fractures, dislocations.

Physical Examination

- Ecchymoses, abrasions, and swelling,
- Range of motion of the joint in question, both actively and passively.

Treatment of Sports Injuries

Macro traumatic Injuries

"PRICE" (Protection, Rest, Ice, Compression, and Elevation).

1- Surgical repair or immobilization with a cast or an orthosis

2-Avoid heat and massage to the affected area → pain, swelling.

3-Avoid NSAIDs in the acute stage to minimize bleeding.

4-Physical rehabilitation to provide normal strength and motion.

Micro traumatic Injuries

1-Avoid activity that causes the athlete's symptoms.

2-Rest to heal and resolve the inflammatory process

3-Non-steroidal anti-inflammatory drugs (NSAIDs).

4-Ice, heat, electrical stimulation, ultrasound, and massage → decrease pain and associated swelling.

5-Physical therapy program

The PRICE principle for treating sports injuries,

1- ***P -Protect*** by a splint, or pad → prevent further injury
2- ***R- Rest*** prevents worsening.
3- ***I -Ice:*** anti-inflammatory." Use ice for 20 min_ every 1-2 hours for the 1st 48 hours after the injury.
   Don't use heat→ swelling and inflammation.
4- ***C- Compression:*** with an elastic bandage.
5- ***E- Elevation:*** reduce pain & swelling.

Mechanism of sports injuries

=Overuse =Direct impact

=Application of greater force > the body part can withstand.

Risk Factors Related to Development of Sports Injury

Extrinsic Factor

1-Training Errors 2- Excessive Load on the Body 3-Environmental Mal conditions

4-Poor Equipment (Worn shoes, Faulty rackets) 5-Ineffective rules (violent play)

Most common Types of sports injuries

1. Ankle sprain 2. Hamstring strain 3. Shin splints

4. Knee injury: ACL tear, meniscus injury, Patello-femoral syndrome.

5. Tennis elbow (Epicondylitis), Golfer's elbow 6. Shoulder injuries

1-Ankle Sprain, most common.

2-Achilles Tendon Rupture,

The Achilles tendon is the <u>largest tendon in the body</u>.

There are three sources for the tendon's blood supply:

(1) Musculotendinous junction. (2) Osseous insertion.
(3) Multiple meso sternal vessels on the tendon's anterior surface.

Mechanism of injury
Secondary to eccentric contraction of inflamed gastrocnemius-soleus complex
• Ruptures usually occur 3–6 cm proximal to the insertion on the calcaneus because the blood supply there is the poorest
•The patient complains of sudden pain and often a "pop" after a push-off movement; History may be negative for pain before rupture.

• **Thompson test** (Simmond's test) (squeezing calf with patient prone and knee flexed to 90 °) is positive (no motion at the ankle) with a complete rupture, and the patient cannot do a heel raise on the affected side.

•The patient can plantarflex at the ankle as flexors of the toes intact.
• Palpable defect and swelling is present a few centimeters above the heel
• 23% of ruptures missed.
• MRI and ultrasonography can help diagnose recurrent or partial ruptures but should not be necessary with complete rupture.

Plantar fasciitis

Inflammation of plantar aponeurosis due to running or prolonged standing is characterized by heel pain (worse with first steps in the morning or after a period of inactivity) and tenderness.

3-Posterolateral Corner Injury (PLC)

- 70% usually combined with PCL injury
- Missed PLC injury is a common cause of ACL reconstruction failure.

Static stabilizers of the lateral knee.
1-Lateral collateral ligament (LCL)
= Most anterior structure inserted on the fibular head
= Primary varus stabilizer of the knee
2-Popliteus tendon (PLT) 3-Popliteofibular ligament
4-lateral capsule thickening
Dynamic structures
1-Biceps femoris inserts on the posterior aspect of the fibula posterior to LCL.
2- Iliotibial band (ITB). 3-Lateral head of the Gastrocnemius.
Symptoms: instability when the knee is in full extension

4- Osgood-Schlatter disease (traction apophysitis). important

Overuse injury caused by repetitive strain and chronic avulsion of the secondary ossification center of the proximal tibial tubercle.
Occurs after a growth spurt in running and jumping athletes.
Presents with progressive anterior knee pain.

Clinical Findings
Symptoms vary from mild aching at the tubercle to severe pain with patellar function and exaggerated bursal tenderness.
Radiographs of the proximal lateral tibia show characteristic fragmentation.

Treatment
Treatment is symptomatic, including analgesics, knee pads to avert direct pressure, quadriceps stretching, avoidance of sports activities, and brief casting or splinting for painful cases. The disorder resolves spontaneously when the physis closes at skeletal maturity. No evidence indicates that physical activity within the limits of pain is harmful to the child with Osgood-Schlatter disease.

5-Medial tibial stress syndrome (shin splints).
Shin pain and diffuse tenderness in runners and military recruits.
Caused by bone resorption that outpaces bone formation in the tibial cortex.

6-Iliotibial band syndrome
Overuse injury of the lateral knee that occurs primarily in runners.
Pain develops 2ry to the friction of ITB against the lateral femoral epicondyle.

7-Patellofemoral syndrome
Overuse injury commonly presents in young, female athletes as anterior knee pain.
Exacerbated by prolonged sitting or weight-bearing on a flexed knee.
Treatment: NSAIDs, thigh muscle strengthening.

Prevention of Sports Injuries

1. Fitness training
2. Proper clinical examination to identify anybody defects that may affect performance and cause injury during sports activity
3. Correcting wrong body postures
4. Conditioning exercises to overcome particular deficiencies
5. Cardiopulmonary conditioning exercises to help increase endurance during Sports.
6. Proper warm-up exercise before starting any sport activity
7. Proper relaxation after sports
8. Wearing protective wear such as shin guards to protect the legs, helmets to protect the head, and gloves to protect the hands (especially goalkeepers)
9. Overuse injuries are common and preventable. Don't come out and hit the ball for an hour after not playing for a while.

19-METABOLIC BONE DISEASES

The fundamental problem in metabolic bone diseases is an imbalance between Osteoblasts and Osteoclasts in normal remodeling.

Types

1-Osteoporosis: quantitive diminution of bone mass (Osteoid).

2-Osteomalacia: qualitative insufficient skeletal mineralization, with a normal amount of osseous connective tissue (Osteoid).

3-Rickets: impaired mineralization of cartilage matrix (chondroid) affecting the physis zone of provisional calcification) in children.

4-Hyperparathyroidism

1-Osteoporosis (Most common)

Trabecular (spongy) and cortical bone lose mass and interconnections despite normal bone mineralization and lab values (serum Ca & PO4).
Due to bone resorption related to estrogen levels and old age.

Diagnosed by DEXA (dual-energy X-ray absorptiometry) at the lumbar spine, total hip, and femoral neck, with a T-score of ≤ −2.5 or by a fragility fracture (e.g., fall from standing height, minimal trauma) at hip or vertebra.
One-time screening is recommended in women ≥ 65 years old.

Risk factors in osteoporosis

1-Nonmodifiable factors	2-Potentially modifiable factors
* History of fracture as an Adult * Positive family history * White race * Advanced age * Female sex * Dementia * Poor health or fragility	* Current cigarette smoking * Low body weight (< 58kg) * Estrogen deficiency (early menopause (age < 45 y) or bilateral ovariectomy and prolonged pre-menopausal amenorrhea (>1y) * Low life long calcium & protein intake * Alcoholism * Inadequate physical activity * Poor health or frailty * Sedentary lifestyle

Types

A-Primary osteoporosis

	Type I (Post. menopausal)	Type II (Senile)
Cause	Loss of estrogen	Poor calcium absorption
Age group	Postmenopausal (50-70 years old)	>75 years old
Bone affected	Almost exclusively trabecular (Cancellous bone)	Both (Trabecular > cortical Bone)
Bones fractured	Distal radius and vertebral	Hip
Effect on calcium	Decreased intestinal absorption and increased urinary excretion of Ca.	Poor calcium absorption

Turnover	High turnover osteoporosis	Low turnover osteoporosis

B-Secondary osteoporosis

1- Chronic medical conditions: Endocrine, GIT, chronic liver disease, CRF.
2- Environmental/lifestyle: smoking, excess alcohol, inactivity.
3- Nutritional: Malabsorption, malnutrition.
4- 2ry to drugs (e.g., steroids, alcohol, anticonvulsants, anticoagulants, thyroxin therapy)

Clinical features of osteoporosis

1-This disease is a "silent thief" that generally does not become clinically apparent until a fracture occurs.
2-Two-thirds of vertebral fractures are painless 3-Loss of height.
4-Fractures (vertebra, hip, and wrist) 5-Positive risk factors.

Assessment Pitfall

Avoid commenting on osteoporosis on a single plain radiograph; the most you can say is that the bones appear osteopenic.

Dual Energy X-ray Absorptiometry (DEXA).

The standard gold method for detecting and assessing osteoporosis.
It is quick and accurate with a low radiation dose.
The technique involves simultaneous measurement of the passage through the body of X-rays with two different energies.
By using two different energy beams, it is possible to minimize the effect of soft tissues, particularly fat, on the result.

Indications of DEXA

- Screening for risk patients
- Diagnose & assess the degree of bone mineral density and follow up after treatment (each year).

T scores result in the number of standard deviations above or below the mean peak bone mass for a race- and sex-matched population.

Z scores result in the number of standard deviations above or below the mean bone mass for age, race, and sex population.
The T score is used for the diagnosis of osteoporosis.

Reading	Meaning	T score	Color
Normal Bone Density	Bone is fine	– 1.0 & above	**Green→Fine**
Osteopenia – low bone density	Bone has been lost & continuing to be lost	between – 1 to -2.5	**Yellow→ Beware**
Osteoporosis	Increase fracture risk	-2.5 and below	**Red→High Alert**

The FRAX® assessment includes questions about:

1-Age 2-Smoking 3-Family history of hip fracture
4-Glucocorticoid use (e.g., Prednisone)
5-Arthritis 6-femoral neck bone mineral density.

Recommendations (for prevention)
1-A balanced diet high in fruits, vegetables, calcium, and vitamins.
2-Calcium. 1,200 mg daily; calcium carbonate is taken with food, but calcium citrate is taken on an empty stomach.
3-Vitamin D. 1,000-1500 IU per day for adults.
4-Avoid alcohol and smoking.
5-Regular weight-bearing exercise every day, walking 30 min /day.
6-Therapeutic medications for osteoporosis
7-Eliminate environmental factors that may contribute to accidents. Falls→ 90% of all osteoporotic fractures.

- ***Measures to Improve home circumstances in old people.***

= Ample lighting, removing obstructions to walking, using nonskid rugs on floors, and placing mats and grab bars in showers)
= Be aware of medication side effects
= Regular checking for eyes.

Management

A- Prevention of osteoporosis

- Raise awareness among patients and health professionals

B- **Pharmacologic Agents**

1-Calcium and Vitamin D (for all patients)
Decreases bone resorption but does not increase bone mass or density
Evidence to suggest modest protective effect – more effective for type II osteoporosis.

2-Bisphosphonates (1st line therapy)
Decrease osteoclastic bone resorption, and increased osteoclast apoptosis

- ***Denosumab (Prolia),*** 60 mg given subcutaneously every 6m for 2-3 years.

Decrease bone loss	Halt bone loss	Bone gain
Bisphosphonates	Ca, Vit D Alendronates (Fosamax) Mild exercise Zoledronic acid Risedronate (Actonel) Denosumab (Prolia)	Fluoride + Ca, Vit.D Extensive exercise

C- Fractures management
Can lead to vertebral compression # → acute back pain, loss of height, and kyphosis.
Also can present with fractures of the femoral neck, and distal radius (Colles #)

2-Vitamin D deficiency

Rickets

Failure of mineralization of the physis → poor calcification of the zone of calcification → weak bone with joint loading→metaphysis becomes broad and cup-shaped.

Causes

Decrease Vit D or impairment of its metabolites (defect in the pathway)

- Malnutrition
- Underexposure to sunlight
- Malabsorption syndrome
- Liver diseases, anti-epileptics → decrease 25-OH**lase**
- Renal failure → decrease 1@OH**lase**
- Hypocalcemia.

Pathogenesis

Decrease vitamin D → decrease serum Ca2+ → increase PTH secretion → decrease serum PO4 3−. Hyperactivity of osteoblasts → increase ALP.

Clinical features of rickets

A-General features

- Retarded bone growth → short stature
- Symptoms of hypocalcemia

Under the age of 18 months→ failure to thrive, restlessness, muscular hypotonia, convulsions or tetany but only minimal bone changes.

B-Localized orthopedic features

1-Genu valgum or varum	2-Anterolateral bowing of the distal tibia
3-Coxa vara	4-Anterolateral bowing of the femur
5-Waddling gait.	6-Kyphosis

Blood investigations: Decrease Calcium, phosphate levels, increase alk. Ph.
Treatment: Vitamin D + Calcium

X-linked hypophosphatemic rickets (VDRR).

* Resistance to treatment with UV radiation or vitamin D.
* Dominant inherited systemic disorder, from mutation of the phosphate-regulating gene homologous to endopeptidases on the X chromosome (*PHEX*).
* Normal Calcium level, high alk.ph, low phosphate levels.
* Massive urinary phosphate loss

Clinical features

- A slower growth rate in the first year of life.
- The next clinical sign is the patient's reluctance to bear weight when beginning to stand or walk.
- Delayed dentition in older children.
- Angular deformity in lower limbs.
- Short stature.

3-Osteomalacia (Adults)

Defective bone mineralization →a large amount of unmineralized osteoid → soft bone. (qualitative defect as opposed to a quantitative defect like osteoporosis).

Causes

1- An inherited defect in the metabolic pathway of Vit. D,
2- Hypocalcemia.
3- Tumors (tumor-induced osteomalacia)
4- Lack of sunlight.
5- Malnutrition.

Clinical features of Osteomalacia

- Generalized bone pain is initially, vague and non-specific but gradually becomes more severe and sometimes localized.
- Proximal muscle weakness.
- Fractures of long bones, ribs, and vertebrae.

Physical exam.

- Waddling gait from hip pain and thigh weakness
- Difficulty rising from the chair and climbing stairs

Radiology of Osteomalacia.

1- Diffuse osteopenia
2- Looser's zones (stress fractures on the concave side of bones)
3- Milkman 'Pseudo fractures' on the concave side of bones (fractures that have united but not mineralized).
4- Protrusio acetabulum.
5- Trefoil pelvis.
6- Biconcave 'codfish' vertebrae → kyphosis

Treatment

- **Large doses of oral vitamin D (10.000 IU/day) + Vit. D**
- **Treat underlying cause.**

20-PEDIATRIC HIP

Normal foot walking is (+10-15°) with external rotation.

Causes of In-toeing

1- Metatarsus adductus (in infants' 0-yr.).
2- Internal tibial torsion (in toddlers' 1-yr.).
3- High femoral anteversion (in children 3-10 years).
4- Residual Talipes equinovarus (clubfoot)

Causes of Out-toeing

A-Calcaneovalgus feet.
B-External rotation hip contracture (in infants 0-1year).
C-External tibial torsion.
D-Femoral retroversion (older children & adolescents).
E-Hypermobile pes planus. Mostly corrected after 1-2 years of walking.

1-Pediatric hip (important).

I. Developmental Dysplasia of the Hip (DDH)

***Normal growth of the acetabulum** depends on

1-Normal epiphyseal growth of the triradiate cartilage and on the three ossification centers located within the pubis (os acetabulum), ilium (acetabular epiphysis), and ischium.
2-The presence of the spherical femoral head within the acetabulum is critical for stimulating the normal development of the acetabulum. The hip is a "ball-and-socket" joint that is held together by ligaments and joint capsule.
3- At birth, the hips are lax, the head of femurs are cartilaginous, the acetabulum has more cartilage than bone and the fibrocartilage labrum widened the acetabulum to accommodate 50% of the head cover.
4-Few weeks after delivery, 90% of the hips become mature and stable.
5-Babies whose legs are swaddled tightly with the hips and knees straight are at a notably higher risk for developing DDH after birth.

***Definitions of neonatal hip disorders**

A. Developmental dysplasia of the hip (DDH).
The spectrum of abnormal growth of the developing hip, (ranging from acetabular dysplasia, hip subluxation, hip dislocation, or hip instability).

1- Acetabular dysplasia: abnormally developed, shallow acetabulum, with an oblique roof and a thickened medial wall.
2- Subluxation – Incomplete contact between the articular surfaces of the femoral head and acetabulum.
3- Dislocation – Complete loss of contact between the articular surface of the femoral head and acetabulum.
4- Instability – Ability to subluxate or dislocate the hip with passive manipulation.

B. **Teratologic (Congenital) dislocation of the hip** (CDH)
Antenatal dislocation in utero and usually stiff irreducible on neonatal examination. (Remember Pavlik harness should not be used in CDH)
Teratologic dislocation is the result of congenital abnormal neuromuscular development. e.g., arthrogryposis multiplex congenita, Larsen Syndrome, and myelomeningocele.

***Epidemiology of DDH**

1-20:1000 neonatal hip instability, 1-2:1000 treated for dysplasia.
2-80% of affected children are female.
3-The left hip is more commonly involved (60%)., as the commonest head presentation is left occipito posterior, during delivery the left hip lies against the mother's sacrum which forces the hip to be in the adducted position, favors instability.
4-20% bilateral. 20% right.
5-60% of newborns with hip instability become stable by age 1w, and 90% become stable by age two months, leaving only 10% of them with residual hip instability.

***Etiology and risk factors in DDH.**

1. Etiology:

I-Anatomical factors.

The shallow acetabulum and capsule laxity often coexist at birth, improving the range of hip movement to aid delivery. The femoral head is >50% uncovered at birth, and this predisposes to subluxation/dislocation.

II- 1ry idiopathic hip dysplasia.

III- Multifactorial.

* *Genetic inheritance* → positive family history
* *Racial*: absent in Africa
* *Mechanical*: Breech, Oligohydramnios, 1st born (tight uterus).

A breech presentation may exert its effects using the strong hamstring forces on the hip that result from a knee extension. The increased tension on the hamstrings pulls the femoral head out of the acetabulum.

* *Maternal hormone*: female child. Maternal hormones & fetal estrogen that is produced by the female infant's uterus → Ligament laxity

2- Associated Risk factors

A- **F**rank breech presentation (30 - 50% risk).
B- **F**emale, **F**irstborn, and **F**amily history is a strong risk factor.
i. One child has DDH, risk of another child is 6%
ii. At least one parent involved: 12% risk
iii. Parent and sibling involved: 36% risk
D-**F**luid abnormality Oligohydramnios)
E- **F**eet Deformity (Metatarsus adductus)
F- **F**etal anomalies **(for CDH)**
G-**F**aulty Habits (Swaddling) esp. with ligament laxity.

Secondary Hip Dysplasia (Pathological)

1-Neurological conditions: Cerebral palsy, Down syndrome.

2-Connective tissue diseases: Ehlers-Danlos syndrome

***Pathology of DDH**

Spectrum: 1-Acetabular dysplasia 2-Subluxation 3-Dislocation.
In dislocation, there will be soft tissue interposition

***Evaluation for DDH**

1. Clinical presentation

The clinical presentation varies with age and type of DDH.
1-In complete dislocation
= Pre-walking: limitation of abduction while changing nappy.
= Post walking: limping, tiptoe gait (in unilateral DDH), LLD or waddling gait (in bilateral DDH).
2-In acetabular dysplasia: Asymptomatic.

2. Clinical examination (in frank dislocation)

A. In the neonatal period, (<6 months), e.g., Ortolani test, (Barlow test is not recommended).
B. In infants older than six months *–walking,*
* Limitation of abduction (>20°), most sensitive test for DDH,
* Apparent limb shortening in unilateral DDH.
* Abnormal deep long groin crease.

C. In toddlers, *(+after walking)*
* Wide perineum in bilateral DDH
* Lumbar lordosis in bilateral DDH
* Trendelenburg's sign and gait.
* Limping in unilateral DDH
* LLD in unilateral DDH

Special Clinical tests in DDH

A- The Ortolani test (reduction test): best tested before 3m of age
N.B: Ortolani test is not for dysplastic or subluxated hips**,** only for completely dislocated hips. (poor screening test)

- Thus, a negative Ortolani test does not mean no DDH; it means only you need further evaluation for risky newborns by other methods.
- The test is positive if the dislocated hip is reducible (Clunk), in cases of teratological dislocation, (CDH) the hips are irreducible.

B-The Barlow test: (Dislocation test), a harmful test
C-The Galeazzi (or Allis) test is positive only in a unilateral dislocation in children above six months of age.
The hips and knees lexed to 90°; the test is positive if one knee (the involved side) is lower than the other.

Diagnostic tests (confirmatory)
A-Ultrasonography (USS).
= At six weeks in patients who are considered high risk (female, family history or

breech presentation) despite a normal exam.
= Done, if available proper ultrasonography service in the first 4- 6 months of life before the appearance of the ossific nucleus.

At the age of 6 weeks,
= Normal α angle is >60°. (Acetabular roof angle).
= Normal β angle is <55°. (Labral cartilage roof angle).
= The amount of femoral head coverage should be >50%.

Neonatal screening for DDH

1- Routine clinical screening (thorough history taking and physical examination) for all newborn infants.
2-Routine ultrasound screening performed for infants with at-risk factors for the condition at the age of 6 weeks, (to give a chance to neonatal unstable hips to be mature and stable (85-90%), otherwise you will over-diagnose and over-treat hips.
3-If no proper ultrasound facility is available, do a plain radiograph for the hips at **the age of 3 months** (make sure the hips are at 40° abduction & 30° internal rotation).

B. Plain radiographs

I. Hilgenreiner line

A line is drawn horizontally through each triradiate cartilage of the pelvis.

II. Perkin line

Perpendicular line to the Hilgenreiner line at the lateral edge of the acetabulum.

III. Shenton line. !!!!!!

N.B: False positive in young children with high femoral anteversion

IV. Acetabular index angle (AIA) (Mirror of DDH)

The angle formed by an oblique line (through the outer edge of the acetabulum and triradiate cartilage) and the Hilgenreiner line.
(a) In the infant <3months, a normal value < 30°.
(b) By the age of 6 months, the acetabular index decreases to 25°.

***Natural History of DDH**

= 90% of neonatal unstable hips will stabilize by 4-6 weeks of age.
= The maximum remodeling of the acetabulum occurs below the age of 18 months.
= Dislocated hips per se do not develop AVN.
= In a dislocated hip the affected leg will become shortened, and this will put pressure on the back, increasing the risk of osteoarthritis to the spine.
The false acetabulum is smaller than a true acetabulum and will develop osteoarthritis between 40-60 years of age if not treated.

***Management of Developmental Dysplasia of the Hip**

The basic principles for treatment are

• Concentric reduction of the dislocation or subluxation.
• Hold the reduction until the hip stabilizes.
• Careful follow-up until skeletal maturity.

Treatment is based on the age of the child, the stability of the hip (unstable versus dislocated hip), and the severity of acetabular dysplasia.

Age	Type	Treatment
0-6 m	Dysplastic	Pavlik Harness
6-18 m	Dislocated	Arthrography 1st. Closed reduction or Open reduction

1-Dysplastic hips in neonates < 6 months of age

In a child with an abnormal α angle on ultrasound or with an unstable hip (subluxated hip on examination), initial treatment usually includes a Pavlik harness. (**Gold standard treatment**).
A-The hips flexed to 90-100° with 45-60° abduction.
B-Excessive hip flexion → risk of femoral nerve palsy.
C-Excessive hip abduction → increased risk of osteonecrosis
The Pavlik harness is a dynamic flexion/abduction orthosis
The child's weight must be <9kg to avoid tearing the harness by the child.

Duration of the Pavlik Harness: 8-12 weeks**,** until the AIA <30°

Success rates for Pavlik harness treatment in this setting have been reported at >90% if applied before 2m of age.
The recurrence rate of hip problems in the future is 10-20%; therefore, follow-up evaluation until maturity is necessary.
N.B: No role for double napkins in the treatment of DDH.

***Complications of DDH**

1-Joint stiffness after open surgery
2-Residual acetabular dysplasia, subluxation, and /or re-dislocation despite adequate treatment. (residual subluxation is ~20%) → Follow up till maturity.
3-Early osteoarthritis in the hip joint(the 30s)
4-Leg length discrepancy→ back pain, functional scoliosis, and knee pain
5-Genu valgum: Unilateral hip dislocations →fixed adduction deformity in the hip → increased medially directed stress on the knee joint.
6- **Trochanteric overgrowth** → abnormal gluteus function
6-Avascular necrosis (AVN) of the femoral epiphysis, is the most devastating. 0-73%. Extreme abduction, especially when combined with extension and internal rotation, results in a higher rate of avascular necrosis.

Prognosis
Children with DDH who receive early treatment are generally good.

Factors associated with poor prognosis in DDH
A- Late initiation of treatment (especially after six months)
B-Need for open reduction.
C-Failure of first-line treatment.

II. Legg-Calvé-Perthes Disease (LCPD).

1. Definition: non-inflammatory idiopathic AVN of the femoral head in a growing child, caused by temporary cessation of the blood flow to the femoral head resulting in venous occlusion and necrosis of the femoral head.

2. Epidemiology

a. The disease more commonly affects boys than girls (5:1).
b. The hips are involved **bilaterally in 10%** to 12% of cases.

3. Patho-anatomy

1. Etiology: The exact cause of LCPD is not known

a. Disruption of the vascularity of the capital femoral epiphysis.
b. Hydrostatic pressure theory.
Reactive synovitis → capsular distension → compression on retinacular vessels
c. Thrombophilia(protein C and S def.)
d. Microtrauma or passive smoking (affects fibrinolysis)

2. Risk factors (Susceptible child)

* Boys (80%). * Poor Social class.
* Short stature with delayed bone age (usually by two years). (90%)
* The child is often thin, and very active. * Smaller than his age group.

4. Pathology.

The capital epiphysis and physis are abnormal histologically, with disorganized cartilaginous areas of hypercellularity and fibrillation.

5. Evaluation

1. Clinical presentation

a. Age 4-9 years.
b. Commonly have a limp and pain in the groin, hip, commonly thigh, or knee regions (referred pain).

2. Physical examination

a. Abnormal gait (antalgic). b. Decreased abduction and internal rotation.
c. Late Limb-length inequality, is mild due to femoral head collapse.

3. Diagnostic tests

a. Plain Radiographs.

Standard AP and frog-leg lateral views of the pelvis are critical in making the initial diagnosis and assessing the subsequent clinical course. (best)

Radiographic Feature (according to the stage)

• Widening of the joint space and minor subluxation
• Sclerosis
• Caffey's sign (Salter), a subchondral # (Crescent sign) on lateral X-ray, (an anterolateral aspect of the femoral capital epiphysis).
• Fragmentation and focal resorption of the epiphysis.
• Loss of epiphyseal height.

• Widening of the femoral neck & head (coxa magna).
• Lateral uncovering of the femoral head.
• Metaphyseal cyst formation.
• Sagging rope sign in adults.

Radiographic stages (**Waldenstrom**)
a-Necrosis. (Ischemia stage).
b-Fragmentation stage (Revascularization)
c-Re ossification stage (healing)
d-Final stage (Remodeling).
N.B: In general, necrotic and fragmentation stages last approximately six months each, the re-ossification stage 18 months, and the remodeling stage 3 years.

Late Stage
= Coxa magna = High-riding trochanter
=Flattened femoral head =Irregular articular surface

Other imaging studies:
Bone scans, MRIs, and arthrography are not routinely necessary.

6. Classification
1. The Herring (lateral pillar) classification (most accurate)
Based on the height of the lateral pillar of the capital epiphysis on the AP view of the pelvis.
a. Group A: Normal height
b. Group B: More than 50% of the lateral pillar height is maintained.
c. Group C: Less than 50% of the lateral pillar height is maintained.

7. Treatment
Conservative (this is the mainstay of management).
NSAIDs, pain killers, physiotherapy.

1. General Principles
a. Most patients (60%) with LCPD will not require surgical treatment. In patients with
i. Herring group A disease in any age group.
ii. Young age (< 6 years) at the onset of disease.
b. Patients with poor prognosis will usually need containment.
i. Herring groups B and C disease.
ii. Children are older than six years at the onset of the disease

Poor prognostic factors in Perthes disease.

1->6 years old **2**-Female: mature earlier with less remodeling potential
3-Obesity
4-Progressive loss of hip motion
5-Advanced stage of disease at diagnosis (B, C)
6-Advanced grade (loss of containment).

8. Complications of Perthes disease.

a. Femoral head deformity.

* Premature physeal arrest patterns * Osteochondritis dissecans,
* Labral injury, and * Late osteoarthritis.

b. The most important *prognostic factor*

1- Shape of the femoral head and its congruency at skeletal maturity
2- patient age at onset of disease.

c. Degenerative changes in the hip joint in the fifth or sixth decade of life.

Differential diagnoses

Unilateral Perthes disease	Bilateral Perthes disease
= Transient synovitis = Infection = Lymphoma, and leukemia. = Juvenile chronic arthritis = Rheumatic fever. = Sickle cell disease.	= Hypothyroidism = Multiple epiphyseal dysplasias = Spondyloepiphyseal dysplasia = Gaucher's disease.

III. Slipped Capital Femoral Epiphysis SCFE

1-Definition

A displacement through the growth plate of the immature hip occurs during the rapid growth period in the hypertrophic zone of the physis

The femoral head remains in the acetabulum; the neck displaces anteriorly and rotates externally.

2-Epidemiology

a. Most common disorder of the hip in adolescents.
b.> Male (12–14 years)
c. Unilateral (80%),
d. Obese hypogonadal male (adiposo genital syndrome), or excessively thin and tall.

3-Etiology and PF: Idiopathic, but in general, weakness of the perichondral ring.

Conditions that weaken the physis

* **Endocrinopathies**

If the patient is <9 years or >16 years and has a retarded bone age or short stature.

* **Systemic diseases** such as chronic renal failure.

*** Mechanical factors** increase the load across the physis → SCFE.

(a) *Overweight children* (b) *Increase in femoral retroversion*.
(c) *Decreased femoral anteversion* and femoral neck-shaft angle.
(d) *Vertically oriented physeal plate*. (e) *Thinning of the perichondral ring*.

4-Pathology

The physis abnormally widened with the irregular organization.
The slip occurs through the hypertrophic zone of the physis.

5-Evaluation

1. Clinical presentation

Alimp and pain in the groin, hip, thigh, or knee region, (Unexplained antalgic limp).

i. Pain in the distal thigh and/or knee region in 30% of cases.

ii. Symptoms are usually present for weeks- months before a diagnosis is made.

2. Physical examination,

a. Abnormal gait (antalgic), waddling gait in bilateral cases, and decreased ROM (flexion and internal rotation). b. Obligatory external rotation, i.e., ER of the hip as the hip is brought into flexion. c. Walking with the externally rotated foot.

3. Diagnostic tests

a. Plain radiographs—Standard AP and frog-leg lateral views of the pelvis.

* Widening and irregularity of physis (appears woolly, earliest sign).
* Decreased epiphysis height (slipped posteriorly).
* **The Klein line**, a line tangential to the superior border of the femoral neck on the AP view intersects the proximal femoral epiphysis in a normal hip.
  SCFE fails to intersect the proximal femoral epiphysis.
  Frog lateral radiographs are more sensitive in detecting an SCFE.

*** Trethowan's sign** is when Klein's line does not intersect the lateral part of the superior femoral epiphysis on an AP radiograph of the pelvis.

Classification of SCFE

1. The Loder classification (based on SCFE stability)

A. Stable (85%) if the patient can weight bear on the involved extremity (with or without crutches).

B. Unstable if the patient is unable to weight bear on the involved extremity.

- Sudden onset of pain, often after a fall or injury
- Inability to walk or bear weight on the affected leg
- Outward turning (external rotation) of the affected leg
- The discrepancy in leg length—the affected leg may appear shorter than the opposite leg

N.B: AVN in unstable hips 50%; in stable hips, 0%).

6-Treatment

The primary goals of management are stabilization of the slip to prevent further progression and promotion of physeal closure.

Surgical

i. In situ screw fixation is the preferred initial treatment of SCFE.

NO manipulation for reduction because it is associated with AVN

8-Complications of SCFE

i. Osteonecrosis (ON):

- 50% risk in unstable slips and 25% in severe slips.
- Screw placement in the posterior and superior femoral neck
- With open reduction.

ii. Chondrolysis: due to pin penetration of the joint and multiple screw fixation. If penetration is recognized during surgery and corrected, the hip will be safe. Diagnosis indicated by virtually nil range of hip movement, hip pain, and a narrowed joint space. Confirm with MRI.

iii. Slip progression: in 1% to 2%

iv. Subtrochanteric #: with entry sites through the lateral cortex and those at or distal to the lesser trochanter.

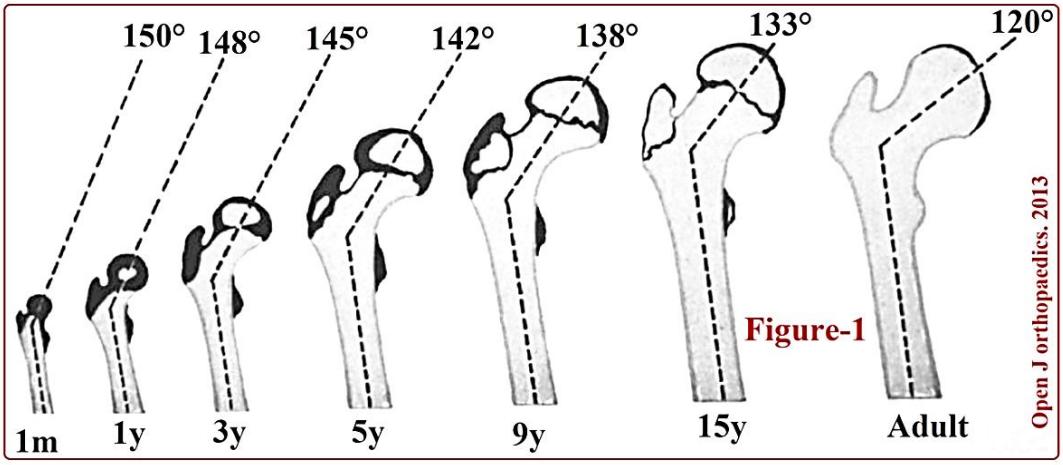
v. Degenerative joint disease: 10% of patients with SCFE develop OA.

vi. Residual leg length inequality and rotational deformity
(severe slips that may require late corrective osteotomy)

IV. Coxa Vara.

1-Definition: abnormally low femoral neck-shaft angle (<120°).

Evolution of the neck-shaft angle in the normal hip. **Figure-1**



Types of Coxa vara in children.

1- **Congenital**
2- **Acquired (secondary)**
Trauma, infection, SCFE, LCPD, Rickets, or Fibrous dysplasia.
3- **Developmental**
Localized developmental bone dysplasia characterized by decreased neck-shaft angle (<110°) owing to a defect in ossification of the inferomedial femoral neck (Fairbank's triangle). (including the inverted Y sign)

21-History and physical examination in Orthopaedics

I- Important Musculoskeletal Symptoms

A-Pain

• Acute onset of pain is often a manifestation of infection, such as septic arthritis or crystal arthropathies (e.g., gout).
• Osteoarthritis and rheumatoid arthritis (RA) can cause chronic pain.

Common sites of referred pain

(1) Arm pain from shoulder pathology (2) Knee pain from the hip pathology
(3) Shoulder blade pain from the neck pathology
(4) Buttock, thigh, and leg pain from the lumbar spine pathology

• Bone pain is boring and penetrating and is often worse at night. e.g., tumor, chronic infection, and Osteoid Osteoma.
• Shooting pain is suggestive of nerve entrapment (e.g., disc protrusion).

B-Stiffness

The inability to move the joints after a period of rest. It may be due to mechanical dysfunction, local inflammation of a joint, or a combination of both.
e.g., rheumatoid arthritis & ankylosing spondylitis.

C-Swelling

Inflammation of the synovial lining, an increase in the volume of synovial fluid, bone swelling, or swelling of structures surrounding the joint.

D-Deformity

= Acute deformity may arise with a fracture or dislocation.
= Chronic deformity is more typical of bone malalignment and may be partial / subluxated or complete/dislocated.

E-Locking

Sudden inability to complete a certain movement secondary to a mechanical block or obstruction, usually caused by a loose body or torn cartilage within the joint (often secondary to trauma).

F-Weakness

Localized or generalized weakness, which suggests a peripheral nerve lesion or systemic cause (neurogenic or myopathic in origin).

G-Sensory disturbance

Ask about the exact distribution of any numbness or paraesthesia and document any exacerbating and relieving factors.

H-Loss of function

Loss of function caused by a combination of muscle weakness, pain, mechanical factors, and damage to the nerve supply.

J-Instability

The sensation of the knee twisting or moving from side to side when doing basic activities. (Due to damage of the knee ligaments, meniscus tears, arthritis, patellar (knee cap) instability, or even nerve damage).

K-Extra-articular features

Several musculoskeletal disorders (e.g., rheumatoid arthritis) cause extraarticular or multisystem features.

II- History

Routine history as usual.

III- Physical examination

Pre-requisites: good history.

Set your mind for inspection, palpation, and movement.

1- Before inspection

* Adequate exposure. * Look from all directions.
* Normal first. * Bilateral comparison.

2- Before palpation

* Ask the patient where is the pain?
* Check every anatomical landmark in detail.

3- Before movement

* Check both active and passive ROM. * Wash hands afterward.

A- General examination (routine)

B- Local examination

I-Walking

The unusual pattern of gait

= **STRAWS** (**S**hort, **T**rendelenburg, **R**igid, **A**ntalgic, **W**eak, **S**pastic)

Look at the trunk, hip, knee, ankle, and foot at the swing and stance phases.

II-Posture

- See how well they undress for examination. e.g., takes off their sleeves.

Standard local Exam

= Look, Feel, Move, Percussion, and Auscultation.

1= **Skin**

2= Muscle

*Bulk *Mass *Tenderness, Spasm / Tone, (**Feel**)

*Fasciculation (**Percussion**)

3= Bone

- Length –apparent, real.
- Deformity - frontal, sagittal, rotational.
- Tenderness (**Feel**)

4= Joint

*Deformity *Effusion *Soft tissue swelling

*Tenderness, effusion, synovial thickening (**Feel**)

*ROM, crepitation, click (**Active movement**)

5= Swelling

- The site, size, and shape
- **S**oreness, **s**tructure, **s**urface, **s**kin, tissue plane (**Feel**)
- Bruit (**Auscultation**)

6= Neurovascular

IV-Examination of Specific joints.

1-Upper Limb

I- SHOULDER

Important points to notice.

- Expose both shoulders.
- Compare sides: swelling, discoloration, deformity, atrophy.

Look: deltoid wasting, rotator cuff in a thin patient.
Feel: clavicle, ACJ, acromion, coracoid, scapula, humeral head, biceps T.
Move: ROM
= Flexion, extension, (with shoulder IR and ER)
= Abduction, adduction, (glenohumeral and scapulothoracic) (with ER)
= ER, IR (with elbow flexion).

If abduction restricted

- Cannot initiate → rotator cuff injury.
- Painful arc →tendonitis/ rotator cuff impingement.
- Restricted movements →fix scapula; if after fixing scapula cannot abduct at all it suggests fixed glenohumeral joint with previous movements entirely scapular (adhesive capsulitis).

Shoulder-specific tests.

A-Shoulder instability tests

1-Apprehension test *2-Load and shift test*
3-Relocation test *4-Sulcus sign*

B-Impingement tests +ve Neer's impingement sign. +ve Hawkins sign.

C-Biceps tendon problem

- Palpation for tenderness
- *Speed's test*
- *Yergason's test*

D-Rotator cuff tests

- ***External rotation against resistance*** → infraspinatus, teres minor
- ***An empty beer can test*** → supraspinatus
- ***Painful arc*** → supraspinatus
- ***Lift-off test*** →subscapularis

E-ACJ tests

* Direct palpation for tenderness**.** ***** Cross-arm flexion test **(Scarf)**

F-Scapular winging: in long thoracic nerve injury.

G-Neurological exam – power, reflexes, and sensations**.**
H-Pulses
J-Examine the neck (Cervical spine).

B- ELBOW

Look: - Carrier angle, Cubitus (valgus, varus).
Feel: - Bony landmarks
Move: - ROM, (Flexion, extension, supination, pronation).
Elbow specific tests

1- **Elbow laxity**
- *Varus stress test*

Valgus stress test.

2- **Tests for Tennis elbow**
3- **Tests for golfer's elbow**

C- WRIST

Feel:
- Bony landmarks

Move: - ROM.
Wrist specific tests

* DRUJ stability. * de-Quervain's disease: **Finkelstein test**.
* CTS tests: Compression test, Phalen's test, Reversed Phalen's test.

D- HAND AND FINGERS

- **Important deformities to notice**

= Trigger finger = Mallet finger = Swan neck deformity
= Boutonniere deformity. = Z-deformity of thumb

- **Hand and finger**

* Pinch grip, thumb to the side of index grip.
* FDS/FDP action. * Cross-finger test.
* Benediction sign * Froment's test.
* O sign (FPL, FDP).

2-Lower Limb

A-HIP

Look:
Feel:
Move:
Measure: lower limb length measurement
I-Apparent leg length discrepancy
II-Real leg length discrepancy
III- Leg length
Hip-specific tests.
1-Thomas's test
2-Trendelenburg's test
3-Trendelenburg's gait (waddling -Duck gait)

B-KNEE

= *Look* (on standing from front-side-back)

= *Feel* (Effusion).

- **The bulk of quadriceps** (ask the patient to press down against your hand)
- **Patellar tap**
- **Fluid displacement (Bulge, Wipe, Milk, or Stroke test)**

= Strokes upwards with the edge of the hand on the medial side of the knee to

- **Ballottement test.**

- The examiner strokes the proximal hand on the thigh toward the knee and taps on the patella with the index finger of his distal hand.

Specific knee tests.

- Menisci (McMurray test, Grinding test).
- Collateral ligaments (Varus / Valgus stress tests) at 30°.
- Cruciate (Lachman test, Ant. Drawer, Post drawer, Pivot shift).

- Lachman test – flex knee 20°, shift articular surfaces forward & backward.
- Flex knees, put feet together, and look for sag sign (PCL tear).
- Drawer test with the examiner sitting on feet.

- Menisci exam.

- Lateral meniscus – Internal rotation, **AD**duct, flex
- Medial meniscus – External rotate, **AB**duct, flex

- Patellar apprehension test for recurrent dislocation/subluxation
  Press the patella laterally with the thumb, and asks the patient to flex – if in pain, apprehensive suggests impending subluxation.

C-ANKLE

1-Look: Deformity

2-Move**:** ROM

Differentiate between the ankle and subtalar joint movement.

- Ankle: - dorsiflexion, plantarflexion
- Subtalar: - inversion, eversion

Ankle specific tests

I-Ligaments:

1- Inferior tibiofibular syndesmosis → Squeeze test

2- Lateral ligaments

= Anterior drawer test for ankle joint (Compare both sides)
Test the anterior talofibular ligament

= Talar tilt test (varus)
*With ankle neutral: test calcaneofibular ligament
*With ankle plantarflexed: test anterior talofibular ligament

3- Medial ligament
Talar tilt test (valgus) (Test integrity of deltoid ligament)

II-Tendons

- Tendo-Achilles examination
* Look and Palpate for a gap. * Test plantarflexion power
* Single leg heel raise.* Simmonds/Thompson's test for rupture.

III- Sole callosities.

3-Spine

THORACIC AND LUMBAR SPINE

On standing
Look

- Scoliosis (postural –improves on flexion - or structural?)
- Kyphosis
- Lumbar lordosis/ loss of it
- A tuft of hair/ lipoma.

Feel.
Move: Flexion/ extension/ lateral flex extend/ rotation
Power: Tiptoes test, Stand on heels

Thoracic spine-specific tests

1-Forward bending test.
2-Neurology of lower limbs
Motor, Myotome, Sensory, Dermatomes, Abdominal reflex.

Lumbar spine-specific tests
A-SLR
B- Lasegue's test
C-Bowstring sign
D-Cross SLR
E- Femoral stretch test.
F-Neurology of the lower limbs (Motor and sensory)
Sacroiliac joint.
1-Pelvic compression. 2-Pelvic stretch. 3-FABER test (Patrick test).

MRC grading for muscle power

0	No muscle contraction
1	A flicker of contraction but no movement
2	Active joint movement with gravity eliminated
3	Active movement against gravity, but no resistance from the examiner
4	A movement against resistance, but reduced power
5	Full and normal power against resistance

The End.