Muscles

Biceps Femoris
- multiple insertions
- 3 layers:
  - superficial layer - superficial to LCL
  - middle layer - surrounds LCL
  - deep layer - attaches to head of fibula & Gerdy’s tubercle on tibia
- Function - flex knee & externally rotate tibia

Popliteus
- forms floor of popliteal fossa
- muscle attached to posterior tibia - tendon passes intraarticularly deep to femoral attachment of LCL to attach to femur
- Functions
  - flex & internally rotate tibia when leg is free OR externally rotate femur on tibia when tibia is fixed.
  - dynamic reinforcement of PCL, preventing post. displacement of tibia on femur

Semimembranosus 5 distal attachments:
1. Oblique Popliteal ligament
2. Posterior Oblique Ligament
3. Deep Head - deep to MCL (Pars reflexa)
4. Direct Head - to post medial tibial condyle
5. Anterior Expansion - over anterior leg fascia
6. Popliteal expansion: over the popliteus fascia

Pes Anserinus
1. Sartorius, gracilis & semitendinosus [Say Grace before Tea]
2. Sartorius - very broad insertion, forming a sheath over gracilis & semiT
3. Gracilis - smaller & anterior to semiT
4. Semitendinosus - enveloping muscle belly extending more distally
5. Functions - flexor of knee & internal rotator of tibia

Iliotibial Tract
1. inserts into Gerdy’s tubercle on the tibia & extends to form the lateral patellar retinaculum
2. also attaches to the lateral femoral condyle forming a static lateral restraint
3. Function: since it crosses the centre of rotation of the knee moving from flexion to extension it acts as a flexor in flexion & an extensor in extension
Knee Stabilisers (Anatomical)

I- Extra-articular: see later

II- Intra-articular structures

1. Anterior Cruciate Ligament
   - Intraarticular extrasynovial
   - Anteromedial fibers - tight in flexion - limits anterior translation of tibia on femur
   - Posterolateral fibers - tight in extension - limits anterior translation plus external rotation
   - Bl. supply - middle genicular a. (post) & synovial vv (ant)
   - Mechanoreceptors with a proprioceptive role
   - Acl strength = 50% pcl strength
   - Load to failure = 1700n
   - STRAIN RATE plays a role in the failure location
     1. Midsubstance tears occur at higher rates
     2. Avulsions occur at lower rates

2. Posterior cruciate ligament
   - 2 bundles: posteromedial and anterolateral
   - Function:
     1. Limits hyperextension
     2. Prevents post translation of tibia on femur especially during flexion

3. Meniscofemoral ligaments:
   1. Ligament of HUMPHREY - anterior
   2. Ligament of WRISBERG - posterior

4. Menisci
   - Fibrocartilagenous crescent; triangular in cross-section
   - Lateral meniscus is more circular; medial meniscus more c-shaped
   - Lateral meniscus has twice the excursion of the medial meniscus during knee motion.
   - Anterior horn of LM & post horns of both menisci attach to the intercondylar eminence
   - Anterior horns attached to each other by the intermeniscal ligament
   - Popliteus muscle is attached to lateral meniscus (not the tendon)
   - Semimembranosus is attached to medial meniscus

   Blood supply:
   - From branches of the lateral, middle & medial genicular arteries
   - Vascular synovial tissue from the capsule supplies the peripheral 25% of meniscus

   Constituents:
   1. Type 1 collagen fibers arranged radially & longitudinally (circumferential)
      - Longitudinal fibers - dissipate hoop stresses in the meniscus
      - Radial fibers & longit fibers - allows meniscii to expand under compression
   2. Proteoglycans trapped within collagen fibers to absorb energy

   Functions:
   - Provision of stability
   - Shock absorption
   - Provision of increased congruity
   - Aids lubrication
   - Prevents synovial impingement
   - Limits extremes of flexion & extension
   - Transmits loads across the joint – 50- 100% of load is transmitted through menisci
   - Reduces contact stresses
**Mechanics:**
- The circumferential fibers act in much the same way as metal hoops placed around a pressurized wooden barrel. The tension in the hoops keeps the wooden staves in place.
- The compression of the menisci by the tibia and the femur generates outward forces that push the meniscus out from between the bones.
- The circumferential tension in the menisci counteracts this radial force.
- These hoop forces are transmitted to the tibia through the strong anterior and posterior attachments of the menisci.
- This hoop tension is lost when a single radial cut or tear extends to the capsular margin and that in terms of load-bearing, a single radial cut through the meniscus is equivalent to meniscectomy.

Following total meniscectomy there is a decrease in tibiofemoral contact area and an increase in the contact stresses.

With only one third of the meniscus removed, as with partial meniscectomy, there has been shown to be an estimated 65% increase in articular contact stress.

Total meniscectomy may increase peak loads up to 235%.

In the ACL-deficient knee the menisci, specifically the posterior margins aid in stabilising the knee from anterior translation.

**NERVE SUPPLY**

**Motor**
- Femoral n. - All 4 Quad muscles and the sartorius [Picture]
- Tibial Component of the Sciatic n. - Semitendinosis, Semimembranosis, and the LHB
- Common Peroneal Component of the Sciatic N. - Short head of Biceps
- Obturator n. - Gracilis

**Sensory**
- Saphenous nerve is the largest cutaneous branch of the femoral nerve, and supplies sensation to the skin over the anteromedial aspect of the leg [Picture]
- The main terminal branch, the sartorial nerve, runs distally with the greater saphenous vein
- The infrapatellar branch of the saphenous n. arises proximal to the knee joint, around or through the sartorius, and then crosses underneath the patella to innervate the skin over the proximal anterior tibia
- Free nerve endings are prominent in the peripheral portions of the menisci → transmit pain
- Mechanoreceptors have been identified in the anterior and posterior horns
- ACL & PCL receive nerve fibers branches of the tibial n. → proprioceptive and sensory

**PATELLA**
- Largest sesamoid bone in the body (usually 3 - 5cm in length)
- Patella tendon to patella length usually ratio of 1:1 (+/- 20%)
- 10% of patients have complete supra-patella membranes and 75% will have at least one of the 3 plicae's even if only as a remnant (supra-patella, medial patella or infra-patella)
- Ossification centre appears between 2 and 3 years of age but can be as late as 6 years
- Blood supply is via the anastomosis of the genicular vessels via the anterior-middle 1/3 and is directed mainly upwards, from the inferior pole vessels also enter from the deep surface

**Function:**
1. Quadriceps lever arm → mechanical advantage of the quadriceps
2. Aid articular cartilage nourishment of the femoral condyles
3. Provide some protection of the femoral condyles

Normal function depends on the alignment, stability, articular cartilage and muscle control.
Knee Mechanics

Kinematics

Instant Center of Rotation
- "If one rigid body rotates about another rigid body, its motion at any instant can be described by a point or axis of rotation called the instant center of rotation (ICOR).

Surface Joint Motion
- Motion between the tibia and the femur is both rotational and translational. The femoral condyles both roll back and glide as they articulate with tibial plateaus. As the knee moves from full extension into flexion the ICOR moves posteriorly relative to both the femur and the tibia. This increase the flexion range.

Screw Home Mechanism
- It is the lateral rotation of the medial tibial plateau on femur during stance phase (extension), and internal rotation during swing phase (flexion).
- 3 factors leads to this mechanism:
  1. The more distal alignment of the MFC
  2. The bigger radius of curvature of the MFC
  3. The cruciates crossing in-between; around which this rotation occur
- Its significance it that it tightens both cruciates and locks the knee in the position of maximal stability

Patellofemoral Joint
1. Try function of the extensor mechanism of the knee is deceleration during the swing phase
2. Functions:
   1. Increases the moment arm of the quadriceps
   2. Allows wider distribution of compressive stress between the patellar tendon & the femur
   3. From full extension to full flexion the patella glides caudally 7cm on the femoral condyles.
   4. By 20° of knee flexion the patella first begins to articulate with the trochlear groove. Beyond 90° the patella rotates externally & only the medial facet articulates. At extreme flexion the patella lies in the intercondylar groove.
   5. Initially the patella contact occurs distally and with increased flexion the contact areas shift proximally on the patella
   6. Patellofemoral contact pressure is 0.5 times body weight with walking, and increases to 2.5 to 3.3 times body weight with stair climbing and descending
# Knee Instability

## Definition
- It is the loss of normal relationship among the different parts of the joint throughout the whole arc of motion

## Anatomy:
(knee stabilizers)

### I. Extra-articular

<table>
<thead>
<tr>
<th>Site</th>
<th>Layer</th>
<th>Content</th>
<th>Importance</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Medial</strong> (WARREN &amp; MARSHALL):</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Layer 1</td>
<td>Crural fascia investing sartorius &amp; gastroc Sartorius</td>
<td></td>
</tr>
</tbody>
</table>
|                       | Layer 2 | Superficial MCL  
POL  
Semimembranosus                  | 1ry restraint in 30°  
2ry restraint in 0°        |
|                       | Layer 3 | Deep MCL  
Coronary lig  
True capsule                  | 2ry restraint in 0°        |
| **Lateral**           | Layer 1 | ITB  
Biceps                        |                          |
|                       | Layer 2 | Patello-femoral lig  
Patellar retinacula          | 2ry restraint in 0°        |
|                       | Layer 3 | LCL  
Arcuate Capsule               | 1ry restraint in 30°  
2ry restraint in 0°        |

**PMC** = postero-medial quadruple
(NICHOLAS and MINKOFF  
quadripule complexes)

1. MCL
2. Semi-membranosus & its 5 attachments...
3. SGS
4. OPL / POL / PMC

**PLC** = postero-lateral corner

1. LCL
2. ITB
3. Arcuate
4. Popliteus Tendon
5. Biceps Tendon
6. Popliteo-Fibular Ligament (PFL)

**AMC**

AM capsule  
Medial retinacula  
Patellofemoral & patellotibial lig

**ALC**

AL capsule  
Lateral retinacula  
ITB  
1ry restraint

**Anterior**

ACL
Mid med & lat capsule / ITB / Mensci / collatral
1ry restraint  
2ry restraints

**Posterior**

PCL
1ry restraint

**Internal Rotation**

ACL
POL / PMC  
1ry restraint  
2ry restraints in 45°

**External Rotation**

PFL & MCL  
POL / PMC / LCL / PLC  
1ry restraint  
2ry restraints in 45°

**Arcuate Complex**

1. LCL
2. Arcuate lig (Y shaped condensation)
3. Popliteus tendon
4. Biceps tendon
5. Lateral head gastroc

### II. Internal structures:
- ACL, PCL, Menisci and their ligaments
Knee biomechanics:

- The **MECHANICAL AXIS** of the femur ≠ anatomical axis = 7º
- This produces greater WB stresses on the LFC > MFC
- The mechanical axis generally passes slightly medial to the center of the normal knee
- Because of the disparity between the lengths of the articular surfaces of the femoral condyles and the tibial condyles, **TWO MOTIONS** are produced; ginglymus (hinge) + trochoid (pivot):
  1. Flexion & extension ....................... sagittal plane
  2. Some degree of IR & ER .................. Horizontal plane (5º-25º é the IR > ER)
- The complex flexion-extension = **ROCKING + GLIDING**:
  1. Rolling motion is demonstrable in the first 20º of flexion
  2. Gliding motion predominates after 20º flexion

- The **MENISCI MOVE** ê femoral condyles in flexion and extension & move ê tibia in rotation
- **LFC IS BROADER** in frontal and sagittal planes > medial
- MFC is taller i.e. projects distally to compensate for the 7º valgus
- MFC is prolonged forward > LFC → Screwing and unscrewing occurs around an **AXIS NEAR THE MFC & IS INFLUENCED BY PCL**
- No rotation is possible when the knee is in full extension.
- In full extension; femur IR until the medial articular surfaces are in contact & LFC rotates forward → **SCREWING HOME** movement, locking the knee in the fully extended position.
- **FLEXION AND EXTENSION** are from 0-140º
- Normally there is **SAGITTAL LAXITY** of the tibia over the femur, but not > 3-5 mm
- Also there is a coronal laxity (abduction-adduction) but never > 0-15º (not with full extension)
- When the MCL is ruptured........................ the vertical axis of rotation shifts lat & vice versa.
- Changes in the "ICOR" are responsible for OA associated ê knee instability

**KNEE RESTRAINTS** - **Sectioning Studies**

<table>
<thead>
<tr>
<th>DIRECTION</th>
<th>PRIMARY RESTRAINTS</th>
<th>SECONDARY RESTRAINTS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anterior</td>
<td>ACL</td>
<td>ITB .................................. 25%</td>
</tr>
<tr>
<td></td>
<td></td>
<td>mid medial capsule .... 20%</td>
</tr>
<tr>
<td></td>
<td></td>
<td>mid lateral capsule: ... 20%</td>
</tr>
<tr>
<td></td>
<td></td>
<td>MCL ................................ 15%</td>
</tr>
<tr>
<td></td>
<td></td>
<td>LCL:  ................................ 15%</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Menisci</td>
</tr>
<tr>
<td>Posterior</td>
<td>PCL (large anterior bundle more)</td>
<td>LCL</td>
</tr>
<tr>
<td>Internal rotation</td>
<td>ACL</td>
<td>POL/PMC has secondary effect in 0 - 45º</td>
</tr>
<tr>
<td>External rotation</td>
<td>PFL (Popliteofibular ligament)</td>
<td>POL/PMC</td>
</tr>
<tr>
<td></td>
<td>LCL and PLC ........mainly at 30 º flexion</td>
<td></td>
</tr>
<tr>
<td></td>
<td>MCL  ......................at all degrees</td>
<td></td>
</tr>
<tr>
<td>Valgus</td>
<td>Superficial MCL  ...at all deg (least at full ext)</td>
<td>POL - especially near or at full extension</td>
</tr>
<tr>
<td></td>
<td>PMC  ......................at full extension</td>
<td>ACL</td>
</tr>
<tr>
<td></td>
<td>Deep MCL  ...................little resistance to valgus</td>
<td></td>
</tr>
<tr>
<td>Varus</td>
<td>LCL  ......................all degrees (more at 30º, least at full extension )</td>
<td>PLC (PFL)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>ACL</td>
</tr>
</tbody>
</table>
**Palmer Mechanisms Of Injury**

1. flexion, Abd, IR of the femur on the tibia,
2. flexion, Add, ER of the femur on the tibia,
3. hyperextension,
4. AP displacement.

- **F Abd IR** is the commonest → medial side injury, respectively;
  1. MCL then Medial capsule
  2. ACL
  3. MM = "O’DONAGHUE UNHAPPY TRIAD"

- **F Add ER** → injury of lat ligaments of the knee;
  1. LCL then lateral capsule
  2. ACL
  3. Arcuate complex
  4. Popliteus tendon
  5. ITB
  6. Biceps femoris
  7. Common peroneal nerve,

- **HYPEREXTENSION** mechanism;
  1. ACL
  2. PCL & posterior capsule

- **ANTERO-POSTERIOR DISPLACEMENT**: e.g. dashboard accident:
  1. ACL or
  2. PCL

- **ISOLATED LIGAMENT DISRUPTION**;
  1. Some say all ligaments work in concert especially the ACL, and probably no single ligament can be disrupted
  2. Isolated PCL disruption can result from a direct blow to the front of the tibia with the knee flexed.

- **AVULSIONS** occur either at the femoral, tibial, or mid-substance

**Classification of injury**

1. **PLANES CLASSIFICATION**
   - I. One-plane instability (simple or straight)
     1. One plane medial
     2. One plane lateral
     3. One plane posterior
     4. One plane anterior
   - II. Rotary instability
     1. Anteromedial
     2. Anterolateral
     3. Posterolateral
     4. Posteromedial
   - III. Combined instability
     1. Anterolateral-anteromedial rotary
     2. Anterolateral-posterolateral rotary
     3. Anteromedial-posteromedial rotary

2. **O’DONAGHUE:**
   - 1<sup>st</sup> Degree Sprain: Lig injury with no instability
   - 2<sup>nd</sup> Degree Sprain: partial tear with some laxity
   - 3<sup>rd</sup> Degree Sprain: complete tear è instability

3. **NOYES**
   - Grade
     - Grade 1 < 5mm
     - Grade 2 6-10mm
     - Grade 3 11-15mm
     - Grade 4 16-20mm
### History

<table>
<thead>
<tr>
<th>INJURY</th>
<th>SIGNIFICANCE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Noncontact + Pop</td>
<td>ACL Patellar dislocation</td>
</tr>
<tr>
<td>Contact + pop</td>
<td>Collateral injury Meniscal injury Fracture</td>
</tr>
<tr>
<td>Anterior dashboard</td>
<td>PCL or Patellar #</td>
</tr>
<tr>
<td>Ant + Plantar-flexion</td>
<td>PCL</td>
</tr>
<tr>
<td>Ant + dorsi-flexion</td>
<td>Patellar #</td>
</tr>
<tr>
<td>Hyper-ext + varus + ER</td>
<td>PLC corner injury</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>SYMPTOM</th>
<th>SIGNIFICANCE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pain at sitting &amp; stairs</td>
<td>Patellofemoral pathology</td>
</tr>
<tr>
<td>Locking</td>
<td>Meniscal tear</td>
</tr>
<tr>
<td>Pseudo Locking</td>
<td>Patellofemoral pathology</td>
</tr>
<tr>
<td>Pseudo Giving way</td>
<td>ACL</td>
</tr>
<tr>
<td>Acute swelling</td>
<td>ACL</td>
</tr>
<tr>
<td>Giving way</td>
<td>Ligamentous injury Patellar dislocation</td>
</tr>
<tr>
<td>Pseudo Giving way</td>
<td>Anterior knee pain Reflex inhibition of quad</td>
</tr>
</tbody>
</table>

### Examination:

**LOOK**
- Skin - **SCARS**, redness, swelling ................. Remember to look in popliteal fossa
- Muscle - **WASTING** of quads .......................... compare diameter of thigh if quads wasted
- Varus Valgus **DEFORMITY** (measure intermalleolar distance if valgus)
- **Q ANGLE**
- Bone/joint- **EFFUSION**
- Walking - look for varus **THRUST** = dynamic varus collapse in stance loading (δ laxity)

**FEEL**
- Skin - Temperature, back of hand
- Muscle - Ask patient to contract quads
- Bone/joint- Effusion fluid displacement test, patellar tap test
- Synovial thickening
- Joint line tenderness (with knee bent)

**MOVE**
- Active then passive- Flexion & extension (0-140° normal)
- Feel for crepitus
SPECIAL TESTS

1. **MCL + LCL**
   - Leg under arm, 2 hands, 10º flexion to relax pos capsule (careful not to rotate knee)
   - Valgus stress in flexion ..................... MCL
   - Valgus stress in extension ................... MCL + POL
   - Varus stress (taut in full ext) ............. LCL (normally lax in flexion)

2. **Anterior Cruciate Ligament**
   1- **Anterior Draw** at 90º
   2- **Lachman’s Test** - at 15-30º (put patient’s knee over your knee) - most sensitive
   3- **MacIntosh The Pivot Shift Test** - Knee extended, valgus strain, foot internally rotated, if instability present, tibia is subluxed anteriorly. Now flex knee, clunk at 30º is +ve
   4- **Losee Pivot Shift Test** - knee & hip flexed 45º and the other hand thumb behind the fibula. ER and valgus the tibia + slow extension + push the fibula forward → tibial condyle shifts or subluxes forwards in full extension.
   5- **Modified Pivot Shift Test** - Grasp foot between arm and chest, valgus IR stress, , then flex knee, femoral condyle will ‘appear’ to sublux ant (but tibia is reducing)
   6- **Jerk Test (Hughston)** - patient supine. flex knee 90º & hip 45º. Internally rotate tibia. Let knee slowly extend while applying a valgus stress. High false -ve rate
   7- **Ali Test (Slocum)** - Patient lies on unaffected side, with unstable knee up & flexed 10º. Medial aspect of foot rests on table. Patient maintains ipsilateral pelvis rotated posteriorly 30-50º. Knee pushed into flexion. Easier to do in heavy or tense patients.

3. **Posterior Cruciate Ligament**
   1- **Posterior Sag** (in extension)
   2- **Danial Drop-Back** in 90º flexion (active & passive)
   3- **GodFrey Chair Test**: the same but on a chair
   4- **Posterior Draw**
   5- **Reverse Pivot Shift Test (Jakob)**: knee 30º flexed [Subluxed] → valgus ER slow extension → reduces anterior

4. **Posteromedial Corner**
   1- **Hughston Postero-Med Drawer**............ ACL, MCL sup & deep. (possible: POL, PMC)
   - knee flexed & IR then post drawer is done; if lax → Postero-med instability

5. **Posterolateral Instability**
   1- **Dial Test** - pt prone & bend knees or perform supine with the knees at 30º (PLC only) and 90º (PLC + PCL) & assistant holding the knees → ER maximally → note the difference between the two sides; > 15º difference = arcuate / PFL tear
   2- **Hughston Posterolateral Drawer**
   - knee flexed & ER then post drawer is done; if lax → Posterolateral instability
   3- **External Rotation Recurvatum Test** - by holding big toes, lifting feet off table & watching to see if tibia falls into ER & Recurvatum

6. **Antero-medial rotary instability**................. MCL, ACL (possible: POL, PMC)
   1- **Slocum Test**: knee flexed & ER then ant drawer is done; if lax → Antero-med instability

7. **Anterolateral rotary instability** ........... ACL (possible: PLC, AC)
   1- **Slocum Test**: knee flexed & IR then anterior drawer is done; if lax → Antero-lat instability
   2- **Mac-Intoch**: see before
   3- **Losee**: see before
   4- **Jerk**: see before

8. **Meniscal, muscle flexibility, others**: see later
### Different Instabilities of the knee

<table>
<thead>
<tr>
<th>Plane</th>
<th>Direction</th>
<th>Test Used</th>
<th>Deficient Structure</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>One plane</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Valgus</td>
<td>Valgus (Full Extension)</td>
<td>MCL sup &amp; deep, ACL (possible: POL, PMC, PCL)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Valgus (20-30° Flexion)</td>
<td>MCL (possible: POL) → O’Donoghue or Hughston</td>
<td></td>
</tr>
<tr>
<td>Varus</td>
<td>Varus (Full Extension)</td>
<td>LCL, PCL, AC, PLC, ACL, ITB</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Varus (20-30° Flexion)</td>
<td>LCL (possible: PLC, AC)</td>
<td></td>
</tr>
<tr>
<td>Anterior</td>
<td>Lachman (Only)</td>
<td>ACL (PLF)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Anterior Drawer (Only)</td>
<td>ACL (AMF)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Lachman &amp; Ant. Drawer</td>
<td>ACL complete (possible: POL)</td>
<td></td>
</tr>
<tr>
<td>Posterior</td>
<td>Posterior Drawer; Daniel; Godfrey</td>
<td>PCL</td>
<td></td>
</tr>
<tr>
<td><strong>Two planes</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>AM</td>
<td>Slocum</td>
<td>MCL, ACL (possible: POL, PMC)</td>
<td></td>
</tr>
<tr>
<td>AL</td>
<td>Slocum; MacIntoch; Losee; Jerk</td>
<td>ACL (possible: PLC, AC)</td>
<td></td>
</tr>
<tr>
<td>PM</td>
<td>Hughston</td>
<td>ACL, MCL sup &amp; deep, (possible: POL, PMC)</td>
<td></td>
</tr>
<tr>
<td>PL</td>
<td>Hughston; Jakob; Dial; ERR</td>
<td>PCL, AC (possible: PLC, LCL)</td>
<td></td>
</tr>
</tbody>
</table>

8). ![Figures A to I](image)

**Figure**: A; Daniel test, B; Godfrey test, C; Slocum AMRI and ALRI test, D; Slocum ALRI test for unrelaxed patients, E; Losee Test, F; Lateral Pivot Shift of MacIntosh, G; Noyes test, H&I; Jacob test.
Meniscal Test

1- Helfet Test: The patient sitting mark the midline of the patella and the tibial tuberosity; normally in 90° flexion, the tibial tuberosity is aligned with the mid-patellar line & in extension, it moves laterally. This indicates the normal screw-home movement. If rotation is blocked, possibly by a meniscal fragment this movement is lost.

2- McMurray: acute knee flexion as possible. The leg is then rotated internally and externally with varus and valgus rotational stresses → Trapping of damaged MM is felt as a clunk
(at 90° = PHMM / at 60° or less = body or AHMM)

3- Anderson Grind Test (if full flexion is difficult) varus and valgus rotational stresses are applied to 45° flexed knee as the knee is extending. The maneuver is repeated with progressive loading → a clunk may be felt

4- Apley Compression Test

5- Steinmann 1st test is done with the patient sitting the same as Apley

6- Steinmann 2nd test is done with the pt supine; ER during flexion & extension → pain moves from posterior to anterior with extension

7- Jump Test (especially for pt with ACL tears): knee flexed to 130°-140°. The proximal tibia is grasped as in anterior drawer and pulled anteriorly with a firm, prolonged force → jump or jerk

8- Bragard’s Extension Test the knee is hyperextended & ER; so the menisci are forced to move forward so the injured meniscus is stretched producing pain over the joint line.

9- Bohler’s Abduction Adduction Test valgus and varus stresses cause pain over the lateral or medial joint line respectively.

10- Dehaven Abduction Gravity Test may help to demonstrate tears of the anterior or middle segments of the lateral meniscus. With the patient on his sound side, the affected knee is flexed and extended from 90°-0°, so the force of gravity apply compression to the lateral joint space

11- Payr’s Test: is the same idea by applying varus stress while the pt is sitting in Buddha position

12- Spring Test is done by grasping the heel with the knee flexed as much as possible, and then extended passively. When a painful “spring block” occur = torn meniscus (fig.2-14; C).

13- Thessaly Test: è the pt. mono-pedal standing on the affected side at 5° & 20° flexion & supported by holding his out-stretched hands; he is asked to rotate his body on his knee internally & externally. Pain, locking, or catching is pathognomonic to meniscal tear (95% accuracy)

14- Merke’s Test: is the same but the patient is standing bipedal unsupported

15- Duck Walk Test: pain is produced at the corresponding joint line
9). **Patellofemoral Joint**

1- **Alignment**
2- **Grass Hopper Sign** of lateral subluxation of patella
3- **Q Angle**: between line joining ASIS to mid-patella & line from mid-patella to tibial tubercle.
4- **‘J’ Sign Tracking** - = maltracking due to lateral subluxation.
5- Grinding/crepitus
6- **Clarke’s [Snatch]** test - patella fixed with hand on its superior border. Patient asked to contract quads. Reproduction of symptoms = +ve (all patients get some discomfort)
7- **Patellar Apprehension Test** (for instability) - Flex knee whilst trying to push patellar laterally, if positive patient will react with apprehension or try to push examiner away.
8- **Plica Grind Test**: flexion 30º then push patella medially → pain if medial plica

10). **Other usually forgotten tests:**

1). Back of the knee
2). Hip
3). Muscle flexibility tests: Ely, Tripod, Ober, Silfverskiold
4). Nerve assessment especially of the common peroneal nerve in PLC injuries

### Radiological

<table>
<thead>
<tr>
<th>X-RAY</th>
<th>ACUTE INJURY</th>
<th>CHRONIC INJURY</th>
</tr>
</thead>
<tbody>
<tr>
<td>AP</td>
<td>Lateral capsular sign = <strong>Segond</strong> #</td>
<td>ACL deficiency: Central osteophytes + Peaking of the tibial spines (TETON sign)</td>
</tr>
<tr>
<td></td>
<td>Growth plate fracture</td>
<td>MCL: <strong>Pellegrini-Stieda</strong> disease</td>
</tr>
<tr>
<td></td>
<td>Avulsed fibular styloid</td>
<td>OCD</td>
</tr>
<tr>
<td></td>
<td>Torn ligaments. [stress film)</td>
<td><strong>Fairbank’s</strong> joint line changes; Squaring of MFC Narrowing of medial joint space Osteosclerosis Marginal and central osteophytes</td>
</tr>
<tr>
<td>Tunnel</td>
<td>In addition to above findings: Loose body Avulsed ACL, or PCL</td>
<td><strong>Discoïd</strong> Lateral meniscus: Squaring of LFC Widening of Lat. joint space Cupping of lat tibial plateau Bipartite patella</td>
</tr>
<tr>
<td>Lateral</td>
<td>Avulsed ACL at tibial spine</td>
<td>Patella alta</td>
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<td></td>
<td>Avulsed patellar tendon</td>
<td><strong>Sinding-Johansson-Larsen</strong> syndrome</td>
</tr>
<tr>
<td></td>
<td>Avulsed quadriceps tendon</td>
<td><strong>Osgood-Schlatter</strong> disease</td>
</tr>
</tbody>
</table>
|         | | Osteophyte [inf. and sup. patellar border]
|         | | Calcified fat pad [Hoffa’s disease]
|         | | Calcified prepatellar bursa |
| Patellofemoral | Avulsed medial capsule | SUBLUXED patella (Merchant 45º & Lauren 20º) |
|         | Fractured lateral osteophyte | Calcification |

2). **MRI**

- Allows assessment of PLC injury as well as ACL & PCL
- Assess which structures of the posterolateral corner are injured and whether the injuries are mid-substance or whether they have been avulsed from the fibula or femur
Non-operative Treatment:
- Good results if:
  - thigh circumference is equal or better than opp. side
  - stable
- Poor results if:
  - thigh atrophy
  - meniscectomy performed
  - instability

Arthroscopic findings:
- "DRIVE THROUGH SIGN" = >1cm of lateral opening and exceptional posterior visualization of the lateral meniscus

Rationale:
- ACL & PCL arthroscopic reconstructions are usually delayed for 6wk then reconstruct
- PLC & PMC are best treated in the acute stage

ACL Instability with degenerative changes
- Ligamentous stabilisation alone often fails
- Usually medial compartment OA
- Rx: combined tibial osteotomy plus extra-articular reconstruction

**ACL**
- Completely intraarticular extrasynovial ligament measures 33mm X 11mm
- ACL max Tensile Strength = 50% PCL strength = 1700n
- Strain Rate plays a role in the location of ligament failure
  - Midsubstance tears occurring at higher rates
  - Bone ligament complex tears occurring at lower rates
- Blood Supply - middle genicular artery (post) & synovial vessels (ant)

Function
1. Anteromedial fibers - tight in flexion - limits anterior translation of tibia on femur
2. Posterolateral fibers - tight in extension - limits anterior translation + ER
3. Mehanoeceptors with a proprioceptive role
4. Important as a secondary restraint to varus and valgus stresses
5. Screw home motion occurs around its axis

Isometry:
1. Isometric placement of ACL refers to the concept that a full range of knee can be achieved without causing long-term ligament deformation
2. Isometry can not exist because, during ROM, there is no one point on femur that maintains a fixed distance from a single point on tibia; elongation always will occur
3. Placement of graft as closely as possible to centers of tibial & femoral attachments of anterior medial band results in least amount of strain (least change in the length of ACL during complete ROM of knee)
Operative Treatment:

1. **ACL Treatment**

   I. Direct Repair
      1. Screw fixation or pull out sutures for avulsion fractures
      2. Not recommended for mid-substance tears due to poor healing potential.

   II. Extra-articular reconstruction
      1. Macintosh Procedure: (historical)
         - Pass a mobilised strip of ITB to the posterolateral corner of the knee through a tunnel deep to the LCL
      2. Losee Modification: Loop through bony tunnel & lat. Gastroc.
      3. Andrews ITB tenodesis

   III. Intra-articular Reconstruction
      1. Clancy BTB reconstruction
      2. Macintosh: distally attached BTB reconstruction
      3. Zaricznyj hamstring reconstruction - semitendinosis + gracilis
      4. Lipscomb distally attached ST graft
      5. Puddu proximally unstripped ST graft
      6. Allograft reconstruction
      7. Synthetic grafts

IV. Intra & extra articular Reconstruction

   1. Zarin ITB & ST in the opposite direction
   2. Losee modification of Zarin ITB & ST in opposite direction
2. **PCL Treatment:**
   - Traditionally, most authors have recommended **Nonoperative Treatment for Isolated PCL**
   - Salvage procedures:
     1. **Müller Olercization** of Patella to prevent posterior sag of tibia
   - Reconstruction procedures:
     2. **O’Donoghue** transosseous suturing
     3. If avulsed **Refixation** of the avulsed fragment
   - Arthroscopic assisted PCL reconstruction:
     4. **Single Bundle** PCL reconstruction
     5. **Double Bundle** PCL reconstruction
     6. **Inlay** PCL reconstruction

3. **Postero-Medial Reconstruction:**
   1. **Repair:**
      1. Anatomical repair from the deepest structures to the most superficial structures
      2. **O’Donoghue** transosseous suturing method
   2. **Reconstruction**
      3. **Hughston** reconstruction of POL through trans-osseous suture
      4. **Slocum** Pes Anserinus transplantation + sartorius advancement to reinforce MCL
4]. **Posterolateral Reconstruction**

1- **Acute Repair:**
   1. Anatomical repair from the deepest to superficial + **Bunnel** LCL suturing
   2. **O'Donoghue** transosseous suturing method

2- **Chronic reconstruction:**
   1. **Hughston** postero-lateral advancement
   2. **Müller** LCL reconstruction using biceps slip
   3. **Clancy** Biceps tenodesis
   4. **Warren** two tailed graft reconstruction (both LCL & popliteo-fibular lig)
   5. **Noyes** allograft reconstruction
   6. **Larsen** semitendinosus for reconstruction

---

**Post-op Care:**
- CPM range of motion immediate post-op
- Closed chain exercises (foot is maintained on the ground or a platform)
- Running & any activity that involves excessive knee rotation (cutting) is discouraged for the first several months.
Complications:

1. Loss of knee extension / Arthrofibrosis
   - more common with early reconstruction
   - incorrect tunnel placement can cause decreased motion and fibrosis
     (diagnose with x-rays & MRI)
2. Tibial tunnel syndrome
   - size of tibial tunnel over 1yr following surgery
3. Graft failures:
   - Improper placement of graft tunnels
   - Impingement of the graft in the femoral notch:
     • due to improper tunnel placement
     • due to inadequate notchplasty
   - Inadequate graft fixation
   - Associated injuries (varus knee, significant osteochondral defects)
     - CRPS (0.5%)
     - Infection (<1%)
4. Patellofemoral pain
5. Patella fractures
6. CRPS (0.5%)
7. Infection (<1%)
**Knee Dislocations**

**Clinical Findings:**
1. **Popliteal artery & vein injury** is common
   1. note that knee dislocations that have spontaneously reduced may look benign but may lead to thrombosis of the popliteal artery
   2. popliteal a. is usually tethered proximal at adductor hiatus & distal by soleus arch
   3. injury to popliteal a. may initially manifest as an intimal tear or intraluminal thrombus
2. **Peroneal nerve injury:**
   1. in 20% to 40% (half of these palsies are permanent)
   2. w/ peroneal nerve injury, be highly suspect for vascular injury; - even if pulse returns following reduction, consider need for arteriogram, since incidence of intimal injury is high w/ concomitant nerve injury
3. **Both cruciates and least one collateral ligament** are usually disrupted

**Classification:**
- **Anterior** (31%)
  - hyperextension of knee (may need > 30 º of hyperextension to produce this injury)
  - often PCL & ACL torn
  - either the MCL or LCL or both will usually be injured
  - alternatively, hyper-extension → ACL & posterior capsule while the PCL is spared
  - Popliteal artery injury
- **Posterior** (25%)
  - disruption of both cruciate ligaments
  - possible extensor mechanism disruption
  - avulsion of or complete disruption of popliteal artery
- **Lateral** (13%) & **Medial** (3%)
- **Rotatory** (4% - usually posterolateral)

**Investigations:**
1. **X-Rays:**
   - Tibial plateau fracture & Proximal fibula fracture & Avulsion of Fibular Head
   - Avulsion fracture of Gerdy's Tubercle
   - Intercondylar spine fracture
2. **Arteriogram** - indications unclear
3. **MRI** - see above

**Management:**
- **Reduction**
  - may be complicated by interposed soft tissue
  - External fixation - it is important that the external fixator pin sites will not interfere with the ACL/PCL tunnel sites (during future ligament reconstruction)
- **Vascular Injuries**
  - the worst error to make is to underestimate the need to promptly treat these injuries
- **Nerve injury:**
  - the location of the nerve injury may be well above the knee joint
- **Assessment of ligament injuries:**
  - EUA
- **Surgical Treatment of Ligament Injuries:**
  - if vascular injury has been previously repair, get clearance from the vascular surgeon to utilize a tourniquet
  - Deep to superficial (as above)
**Patellofemoral Disorders**

**Anatomy**

<table>
<thead>
<tr>
<th>WIBERG’S CLASSIFICATION of patella shape</th>
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<tbody>
<tr>
<td>Type I</td>
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<tr>
<td>Type II</td>
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<tr>
<td>Type III</td>
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<tr>
<td>Type IV</td>
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</tbody>
</table>

**Jagerhut** Rare No articulating medial facet

**Extensor mechanism:**
1. Quad & Quad tendon
2. Medial and lateral retinaculae
3. PF ligaments
4. Patello-tibial lig
5. Patellar tendon
6. Tibial tubercle

**Patellofemoral kinematics**
- Patellofemoral joint reaction force
  - 0.5 times body weight with walking
  - 3.3 times body weight with stairs
- Patella \^ the moment arm of the quadriceps → ^ quad strength by 33-50%
- The femur articulates only with a portion of the patella in each position of flexion

**Clinical**

**History:** Determine if complaint is instability or pain

**Examination** (Also see Torsional Profile Assessment)

- **Standing examination**
  - Varus/ valgus alignment & Q-angle
  - Pelvic obliquity and LLD
  - **Miserable Malalignment $ = femurIR + Tibial ER ± pronated foot ± pes planus**
- **Sitting examination**
  - **Grasshopper Eyes** appearance: high and lateral patellas
  - Lateral patellar tilt
  - Patellar tracking: pain and crepitation, ‘J’ SIGN
  - Position of tibial tubercle relative to trochlear midline ≈ < 20mm lateral to the midline
- **Supine examination**
  - Quadriceps (Q) angle (Normal M 10° F 15)
  - Quadriceps mass (VMO atrophy); hand breadth above patella
  - Hamstring tightness (popliteal angle)
  - Tenderness on quadriceps or patellar tendon insertion, patellar facets, retinaculum
  - Crepitation and patellar compression
  - Apprehension test (20-30°flexion)
  - Examination for medial plica
  - **Clarke’s Snatch Test** (pain on contraction of the quadriceps with the patella fixed)
  - **Patellar Tilt** (evaluates tension of the lateral restraint)
  - **Patellar Glide Test** (knee flexed 20 to 30°)
    - \^: 1 quadrant or less medial glide is indicative of tight lateral
    - \^: subluxable, or dislocatable patella
- **Prone examination**
  - Hip motion - femoral neck anteversion (abnormal if IR exceeds ER > 30°)
  - Quadriceps tightness - Ely Test (especially rectus femoris)
  - Leg-heel alignment (Normal 2-3° of varus)
  - Hindfoot-forefoot alignment: (N: long axis of heel ⊥ to transverse axis of forefoot)
INVESTIGATIONS

RADIOGRAPHY
- AP
- True lateral view
- Skyline views:
  - Sulcus angle of Brattström: 120º-140º
  - Patellar index: \( \frac{\text{PatellarWedth}}{\text{MedialPFW} - \text{LateralPFW}} \approx 17 \)
  - Congruence angle: between the sulcus line and the patellar vector = -16º to + 4º
  - Lateral PF angle: bet. the tangent on femoral condyles & Lat.PFJ line \( \rightarrow N: +ve \)
  - Lateral PF index: PFJ space \( \frac{\text{CentralPFJ space}}{\text{LateralPFJ space}} \approx 1.6 \)
- More accurate assessment of sulcus & congruence angle
- Perform é knee in different flexion degrees ± quad contraction

MAGNETIC RESONANCE IMAGING
- Midpatella transverse images with knee flexed 15º
- Assess other areas of knee: articular cartilage, muscle and supporting retinacular structures
- Dynamic MRI to assess patella tracking

Patellofemoral Trauma

PATELLA FRACTURE
- Patellar Stress Fractures
  - Rare overuse injury reported in endurance runners, volleyball players, and high jumpers
  - Two types: longitudinal and transverse (Iwaya 1985)
  - In cases with delayed diagnosis operative treatment
  - Quadriceps or Patellar tendon rupture

Non-arthritic patellofemoral pain

1. PATELLAR TENDONITIS (JUMPERS KNEE)
   - Secondary to repetitive trauma: running, jumping and kicking sports
   - Tenderness usually on the inferior pole

<table>
<thead>
<tr>
<th>Blazina Classification</th>
<th>Affect Participation</th>
<th>Treatment</th>
</tr>
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<tbody>
<tr>
<td>I</td>
<td>Pain only after activity</td>
<td>N</td>
</tr>
<tr>
<td>II</td>
<td>Pain at the start of activity &amp; fatigue</td>
<td>N</td>
</tr>
<tr>
<td>III</td>
<td>Pain during and after participation</td>
<td>Y</td>
</tr>
<tr>
<td>IV</td>
<td>Complete tendon disruption</td>
<td>Y</td>
</tr>
</tbody>
</table>

2. PLICA SYNDROME
   - Medial patellar (most common), lateral and suprapatellar
   - Inflammation and impingement on medial femoral condyle
   - Treatment
     - NSAID's, quadriceps exercises \( \pm \) Steroid injection \( \pm \) Arthroscopic excision

3. OSTEochondritis DISSEcANS (OCD)
   - Knee is most common site
   - Typically in teenage athletes
   - Location in the patella is rare
   - Medial facet (70%), lateral facet (30%)
4. CHONDROMALACIA
- “softening” of the articular cartilage
- Aetiology
  - 50% idiopathic
  - 15% post traumatic
  - 20% secondary to maltracking - lateral patellar compression syndrome
  - 15% due to unstable (recurrent dislocaters) patellae
- Treatment is dependent on cause

<table>
<thead>
<tr>
<th>Outerbridge Classification</th>
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<tbody>
<tr>
<td>I</td>
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<td>II</td>
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<td>III</td>
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<td>IV</td>
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5. PATELLOFEMORAL MALALIGNMENT
MEDIAL PATELAR SUBLUXATION/DISLOCATION
- Usually iatrogenic, secondary to realignment procedures, but has also been described without previous surgery (Richman 1998)
- When associated with internal tibial torsion
  - If Tibio-Tubercle distance < normal: external derotational tibial osteotomy proximal to tibial tubercle
  - If Tibio-Tubercle distance is normal: external derotational tibial osteotomy distal to tibial tubercle
LATERAL PATELAR SUBLUXATION/DISLOCATION
- 5% are associated with an osteochondral fracture
- Treatment
  - First time instability episode with no malalignment and normal radiographs
  - immobilisation followed by early range of motion and PT
  - If osteochondral loose bodies are present → arthroscopic removal or fixation

<table>
<thead>
<tr>
<th>Fulkerson</th>
<th>Clinically</th>
<th>Q Angle</th>
<th>Lateral Retinaculum</th>
<th>Realignment</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Subluxation alone</td>
<td></td>
<td>Normal</td>
<td>Distal</td>
</tr>
<tr>
<td>II</td>
<td>Subluxation and tilt</td>
<td></td>
<td>Tight</td>
<td>Distal + Proximal</td>
</tr>
<tr>
<td>III</td>
<td>Tilt alone</td>
<td></td>
<td>Normal</td>
<td>Proximal</td>
</tr>
<tr>
<td>IV</td>
<td>No malalignment</td>
<td></td>
<td>Normal</td>
<td>Physiotherapy ± Proximal</td>
</tr>
</tbody>
</table>

- Subdivided:
  A. Absence of articular lesion
  B. Presence of minimal chondromalacia
  C. Presence of osteoarthritis

6. DORSAL DEFECT OF THE PATELLA
- Benign lesion - Non-specific fibrous tissue
- Located along the superolateral aspect of the articular surface of the patella
- Radiolucency with sclerotic margins and intact overlying articular cartilage
- Frequently heals spontaneously by sclerosis

7. BIPARTITE PATELLA
- 15% of people in childhood & 2% in adulthood
- Sometimes a 3rd ossific centre appears (TRIPARTITE)
- 57% are unilateral with a male: female ratio of 9:1

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<thead>
<tr>
<th>Saupe Classification</th>
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<tbody>
<tr>
<td>TYPE I 5% Distal pole</td>
</tr>
<tr>
<td>TYPE II 20% Lateral</td>
</tr>
<tr>
<td>TYPE III 75% Supero-lateral</td>
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</tbody>
</table>
8. Osgood-Schlatter’s
- Traction injury to the apophysis where patella tendon inserts (some inserts on either side)
- 11-15 y male, usually no history of injury and may be bilateral
- Symptoms are aggravated by running, squatting, jumping, stairs etc
- PXR: May show fragmentation of the apophysis
- Spontaneous recovery is usual but takes time
- Where symptoms are persistent and troublesome excision of a separate ossicle

9. Sindig-Johansson-Larsen’s Disease
- Traction apophysitis of the distal pole of the patella → traction tendonitis develops
- Similar pathology to Osgood-Schlatter’s disease and usually resolves spontaneously

10. Torn Meniscus
11. Discoid Meniscus
12. Patellar Bursitis
13. Patella Cysts or Tumours
14. Reflex Sympathetic Dystrophy

Relationship of patellofemoral malalignment to femoral and tibial torsion

Internal Femoral Torsion / Femoral Neck Anteversion
1. Toeing-in if it exists alone and external rotation of hip < 30°
2. Feet straight: if compensatory external tibial torsion, pes planus, or external hip rotation at least 30°

External Tibial Torsion
- Primary or secondary to medial femoral torsion
- Primary deformity responsible for PF malalignment
- Gait with normal foot progression angle (straight ahead) but with kneeing-in medial thrust with stance phase.

Treatment

Conservative
1. Activity modification, rest, NSAID’s and rehabilitation are successful in 90%
2. Supervised exercise program should be continued for at least 6 months
   - Average pain reduction of 78%
   - Taping changes the timing of VMO and VL activity in subjects with patellofemoral pain during step-up and step-down tasks
   - When the patellofemoral joint was taped, the VMO was activated earlier.
**SURGICAL**

**A. PROXIMAL REALIGNMENT PROCEDURES:**

- **LATERAL RELEASE (OPEN OR ARTHROSCOPIC):**
  - Indication: for patellar tilt < 8 ° or lateral retinacular tightness
  - Do only when it is tight
  - Adequate release should allow inversion of patella to 70-90 °
  - The superficial and deep layers of the retinaculum must be divided.
  - Best results in patients with pain. Worse in patients with instability and severe DJD.
  - 85% good results in pts é pain and PXR evidence of tilt and Outerbridge I
  - 92% good & excellent results in patients with minimal articular degeneration and tilt

- **LATERAL RELEASE AND MEDIALL PLICATION (PROXIMAL REALIGNMENT):**
  - Attempt to decrease the valgus quadriceps moment on the patella
  - Indication: retinacular tightness and medial laxity
  - for recurrent lateral subluxation /dislocation in skeletally immature patients
  - 82% good/excellent results in patients with instability (Scuderi 1988)
  - Poor results in patients with significant chondromalacia

- **GALILEAIZI procedure (semitendonosis tenodesis):**
  - Reconstruction with semitendonosis tendon
  - Tensioned at 30° of flexion

**B. COMBINED PROXIMAL AND DISTAL REALIGNMENT PROCEDURES**

- **ELSMLLE-TRILLAT:** Lateral release with medialisation of tibial tubercle
  - Best candidates have recurrent instability, evidence of increased Q angle and minimal, if any articular degeneration

- **FULKERSOON:** Lateral release with antero-medialisation of the tibial tubercle
  - Biomechanical studies have shown that the patellofemoral joint reaction force decreases about 50 % after a 2-cm anteriorisation of the tibial tubercle
  - Due to the obliquity of the osteotomy, no bone graft is necessary.
  - Best for pts é lateral patellar tilt and with Outerbridge grade III-IV chondrosis
  - See Later for details of distal osteotomies

**C. PATELLOFEMORAL ARTHROPLASTY**

- Hemiarthroplasty (PATELLAR RESURFACING) or total patellofemoral arthroplasty
- Indicated for OA of the patella and trochlea
- Great care must be taken to ensure that any malalignment or maltracking is identified and corrected otherwise the procedure will fail

**D. FEMORAL OSTEOTOMY (ALBEE)**

- For trochlear dysplasia
- Elevation of the lateral facet of the femoral trochlea with osteotomy and bone graft

**E. PATELLECTOMY**

- Last resort surgery which may not eliminate pain
- Complicated by loss of quadriceps strength 30 to 50%
B]. DISTAL REALIGNMENT PROCEDURES:

1. **HAUSER (ABANDONED)**
   - Tibial tubercle osteotomy with the tubercle moved medial, distal and posterior
   - Complicated by development of patellofemoral DJD in 70% of patients because of increased PFJ joint reaction forces (Hampson et al, 1975)

2. **MAQUET**
   - Elevation (anteriorisation) of tibial tubercle
   - Salvage operation in patellofemoral DJD without malalignment
   - Risk of anterior skin necrosis when anteriorisation > 2 cm

3. **GOLDTHWAITE-ROUX**
   - The patella tendon is split & the lateral half is passed under the medial half & attached to the periostium on the tibia in a medial position.

4. **HEMI-PATELLAR TENDON TRANSFER ± lateral release/medial reefing**

5. **ELMSLIE-TRILLAT**
   - Medialization of tibial tubercle + lateral release + medial capsular reefing
   - Considered when there is minimal or absent articular injury in patients with lateral instability caused by malalignment
   - Good/excellent results in 81% (Brown, 1984)
   - Best results when postoperative congruence angle < 15° (Shelbourne et al, 1994)

6. **FULKERSON**
   - Anteromedial tibial tubercle osteotomy
   - Malalignment and lateral facet chondrosis
   - Slope of cut dependent on amount of subluxation and DJD
   - Not as successful with medial facet chondromalacia
   - 90% good/excellent results (Fulkerson et al, 1990)
Patello-Femoral Instability

**Patello-Femoral Joint Mechanics**
1. Patella increases moment (lever) arm of extensor muscles
2. Slides 7 cm in trochlear groove
3. Patello-femoral contact minimal until 20° flexion
4. Contact area moves from proximal -> distal in trochlea & distal to proximal on patella
5. >90° flexion quads tendon is in contact with the trochlea
6. 10° flexion -> lever arm increased 10%
7. 45° -> 30 % then decreases
8. Patello-femoral joint reaction force determined by quads force and amount of knee flexion
9. Normal walking -> joint compressive forces = half bodyweight
10. Up stairs -> 2.5 - 3.3 times body wt
11. Deep knee bends -> 7-8 x body wt

**Presentation**
PFJ instability may present as:
- Anterior Knee Pain
- Patello-femoral subluxation
- Patello-femoral Dislocation

**Anterior Knee Pain**
Other causes of anterior knee pain include:
1. Patello-femoral overload (Chondromalacia)
2. Plica Syndrome
3. ‘Jump’ knee (enthesitis of patella tendon origin)
4. Sinding-Johansson-Larsen
5. Torn Meniscus
6. Discoid Meniscus
7. Osteochondritis Dissecans
8. Patella Bursitis
9. Bipartite Patella
10. Patella cysts or tumours

**Risk Factors / Causes**
A. Bony (Static Stability)
1. Shallow femoral trochlea
2. Hypoplastic LFC
3. Patella Shape (Shultz et al.)
4. Patella Alta

B. Malalignment
- External tibial torsion
- Incr. femoral anteversion
- Incr. genu valgum
- Incr. Q angle (unreliable)

C. Soft Tissue (Dynamic stability)
- Ligamentous laxity
- contribution of distal oblique portion of vastus medialis muscle is critical
- (tight hamstring & gastrocnemius w/ pronated feet)

**Clinical**
- Mechanism of injury, acuity, previous treatment, and status of the opposite knee.
Patellofemoral Disorders

**Anatomy**

### WIBERG’S CLASSIFICATION of patella shape

<table>
<thead>
<tr>
<th>Type</th>
<th>Percentage</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Type I</td>
<td>10%</td>
<td>Concave facets, symmetrical and equal</td>
</tr>
<tr>
<td>Type II</td>
<td>65%</td>
<td>Medial is smaller. Lateral is concave</td>
</tr>
<tr>
<td>Type III</td>
<td>25%</td>
<td>Marked lateral predominance</td>
</tr>
<tr>
<td>Type IV</td>
<td>&lt;1%</td>
<td>Tiny medial facet</td>
</tr>
<tr>
<td>Jagerhut</td>
<td>Rare</td>
<td>No articulating medial facet</td>
</tr>
</tbody>
</table>

**Extensor mechanism:**

1. Quad & Quad tendon
2. Medial and lateral retinaculae
3. PF ligaments
4. Patello-tibial lig
5. Patellar tendon
6. Tibial tubercle

**Patellofemoral kinematics**

- Patellofemoral joint reaction force
  - 0.5 times body weight with walking
  - 3.3 times body weight with stairs
- Patella & the moment arm of the quadriceps → quad strength by 33-50%
- The femur articulates only with a portion of the patella in each position of flexion

**CLINICAL**

**HISTORY:** Determine if complaint is instability or pain

**EXAMINATION** (Also see Torsional Profile Assessment)

- **Standing examination**
  - Varus/valgus alignment & Q-angle
  - Pelvic obliquity and LLD
  - Miserable Malalignment $ = \text{femur IR} + \text{Tibial ER} \pm \text{pronated foot} \pm \text{pes planus}$
- **Sitting examination**
  - Grasshopper Eyes appearance: high and lateral patellas
  - Lateral patellar tilt
  - Patellar tracking: pain and crepitation, ‘J’ SIGN
  - Position of tibial tubercle with respect to trochlear midline ≈ < 20mm lateral to the midline
- **Supine examination**
  - Quadriceps (Q) angle (Normal M 10˚ F 15˚)
  - Quadriceps mass (VMO atrophy); hand breadth above patella
  - Hamstring tightness (popliteal angle)
  - Tenderness on quadriceps or patellar tendon insertion, patellar facets, retinaculum
  - Crepitation and patellar compression
  - Apprehension test (20-30˚ flexion)
  - Examination for medial plica
  - Clarke’s Snatch Test (pain on contraction of the quadriceps with the patella fixed)
  - Patellar Tilt (evaluates tension of the lateral restraint)
  - Patellar Glide Test (knee flexed 20 to 30˚)
    - $\downarrow$: 1 quadrant or less medial glide is indicative of tight lateral
    - $\uparrow$: subluxable, or dislocatable patella
- **Prone examination**
  - Hip motion - femoral neck anteversion (abnormal if IR exceeds ER > 30˚)
  - Quadriceps tightness - Ely Test (especially rectus femoris)
  - Leg-heel alignment (Normal 2-3˚ of varus)
  - Hindfoot-forefoot alignment: (N: long axis of heel ⊥ to transverse axis of forefoot)
- Q angle
- The integrity of the ACL (because similar derotation, deceleration mechanisms that cause patellar instability can cause ACL injury)

**Radiology**

**LATERAL X-RAY:** (knee at $30^\circ$ = Patella is centered in trochlear groove at $30^\circ$ flexion)

- **BLUMENSAAT’S** Line: The superior patella pole should not be higher than intercondylar roof line
- **INSALL-SALVATI** index- length of patella to length of patella tendon, normal=1. Patella Alta ❌
- **BLACKBURN-PEEL** index- length of patella articular surface to the distance of its inferior margin from the tibial plateau w/ knee in $30^\circ$. flexion. normal=0.8-1.1. More accurate.

**SKYLINE VIEW:**

- Sulcus angle of **BRATTSTRÖM**: $120^\circ$-$140^\circ$
- Congruence angle: between the sulcus line and the patellar vector $= -16^\circ$ to $+4^\circ$
- Patellar index: \[ \frac{\text{Patellar Wedth}}{\text{MedialPFW - LateralPFW}} = 17 \]
- Lateral PF index: PF joint space \[ \frac{\text{CentralPFJspace}}{\text{LateralPFJspace}} \] = 1.6
- Lateral PF angle: bet. the tangent on femoral condyles & Lat.PFJ line $\rightarrow$ N: +ve
- Trochlear signs- Crossing sign, ‘Bump’ sign, Dysplastic condyles, Trochlear depth < 8mm.

<table>
<thead>
<tr>
<th>View</th>
<th>Flexion</th>
<th>Technique &amp; Position</th>
<th>Measurements</th>
<th>Miscellaneous</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hughston</td>
<td>55 Degree</td>
<td>Prone position &amp; beam is cephalad and inferior 45 degrees from vertical</td>
<td>1) Sulcus Angle $118^\circ$</td>
<td>— Patellar dislocation — Osteochondral fracture — Soft tissue calcification (old dislocated patella or fracture) — Patellar subluxation — Patellar tilt — Increased medial joint space — Apex of patella lateral to apex of femoral sulcus — Lateral patella edge lateral to femoral condyle — Hypoplastic lateral femoral condyle (usually proximal) — Patellofemoral osteophytes — Subchondral trabecular orientation (increase or decrease) — Patellar configuration (Wiberg-Baumgart)</td>
</tr>
</tbody>
</table>

| Merchant   | 45 Degree | Supine and the beam directed caudal and inferior 30 degree from vertical | 1) Sulcus Angle $136^\circ$ | — Patellar dislocation — Osteochondral fracture — Soft tissue calcification (old dislocated patella or fracture) — Patellar subluxation — Patellar tilt — Increased medial joint space — Apex of patella lateral to apex of femoral sulcus — Lateral patella edge lateral to femoral condyle — Hypoplastic lateral femoral condyle (usually proximal) — Patellofemoral osteophytes — Subchondral trabecular orientation (increase or decrease) — Patellar configuration (Wiberg-Baumgart) |

| Laurin     | 20 Degree | Sitting position and the beam directed cephalad and superior 160 degrees from vertical | 1) Lateral Patellofemoral angle N | — Patellar dislocation — Osteochondral fracture — Soft tissue calcification (old dislocated patella or fracture) — Patellar subluxation — Patellar tilt — Increased medial joint space — Apex of patella lateral to apex of femoral sulcus — Lateral patella edge lateral to femoral condyle — Hypoplastic lateral femoral condyle (usually proximal) — Patellofemoral osteophytes — Subchondral trabecular orientation (increase or decrease) — Patellar configuration (Wiberg-Baumgart) |
|           |          |                      | 2) Patellofemoral Index $A/B < 1.4$ | — Patellar dislocation — Osteochondral fracture — Soft tissue calcification (old dislocated patella or fracture) — Patellar subluxation — Patellar tilt — Increased medial joint space — Apex of patella lateral to apex of femoral sulcus — Lateral patella edge lateral to femoral condyle — Hypoplastic lateral femoral condyle (usually proximal) — Patellofemoral osteophytes — Subchondral trabecular orientation (increase or decrease) — Patellar configuration (Wiberg-Baumgart) |

**AP & Tunnel views:** for osteochondral fractures.

**CT Scan** helpful in assessing the relationship of the PF joint in terms of tilt or translation, or both.

**Arthroscopy**

- Use a suprapatella portal to watch patella centre in trochlear groove betw. $30^\circ$-$60^\circ$.  

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[Knee Disorders]
Treatment

- Treatment is individualized and is based on the patient's lower extremity alignment, joint motion, ligamentous laxity, muscle strength, and quadriceps competence.
- The goal of treatment is to prevent recurrence.

Proximal Realignment

- to alter the tension of tissues attached to the patella (Lat release & med. reefing)
- Lateral retinacular release
- Direct medial retinacular repair in acute dislocations.
- Quadriceps transfers, particularly the vastus medialis obliquus are used to restore medial vector balance (Medial plication).
- Medial hamstring transfer (Galeazzi technique) may be required to provide a tenodesis effect in troublesome cases.

DISTAL REALIGNMENT = transpose the tibial tubercle

1. **Hauser Procedure: (Historical)**
   - Involves medialization of the tibial tubercle in order to decrease Q angle. Due to the anatomy of the proximal tibia, translating the tibial tubercle medially, will also translate the tubercle posteriorly. Posterior translation of the tibial tubercle will have the effect of increasing patellofemoral contact pressures which leads to pain & OA. Also can produce a low patella (baja)

2. **Elmslie-Trillat Procedure:***
   - Medial tibial tubercle transfer which has no posterior displacement
   - Does not involve anterior / posterior displacement of the tuberosity

3. **Maquet Procedure: (Historical)**
   - Anterior translation of the tibial tubercle which has the effect of decreasing patellofemoral contact forces. Patients with pain due to early patellofemoral arthrosis may expect pain relief following the Maquet Procedure. Disadvantages with this procedure include high incidence of skin necrosis, compartment syndrome and no effect on the Q angle;
   - A combination of tibial tubercle transfer, proximal lateral release, medial capsulorrhaphy and tendon transfer may be required to establish appropriate alignment.

4. **Goldthwaite-Roux Procedure:**
   - The patella tendon is split & the lateral half is passed under the medial half & attached to the periosteum on the tibia in a medial position.

5. **Fulkerson**
   - Anteromedial tibial tubercle osteotomy
   - Malalignment and lateral facet chondrosis
   - Slope of cut dependent on amount of subluxation and DJD
   - Not as successful with medial facet chondromalacia

Contra-indications to Re-alignment:

1. Absence of clear physical examination and radiographic findings
2. Patellofemoral pain which results from "dashboard" car accident injuries; often have significant patellofemoral pain chondromalacia, but do not improve with surgery
3. Patellar instability associated with abnormal ligamentous laxity; eg, Down $ & Ehlers-danlos $
Osteochondritis Dissecans

**Definition**
- **Non Inflammatory** pathologic condition of the cartilage é **Impending Or Actual** separation of a segment of cartilage é underlying subchondral bone
- Tends to heal spontaneous in skeletally immatures

**Etiology:**
1- Repeated **Trauma**
2- May be **AVN**

**Pathogenesis:**
1. Intraosseous edema Æ capillary compression Æ ischemia
2. Subchondral collapse Æ arteriolar compression Æ necrosis
3. Fragmentation Æ NBF

**Pathology:**
**SITE:**
- Bilateral .................................................................20-30%
- Poster-lateral MFC (ant to PCL footprint). ..........70%
- LFC .................................................................20%
- Patella ..............................................................10%

**MACROSCOPICALLY:**
- **Degenerated** cartilage
- Underlying a segment of **Osteonecrosis**
- Lesion **Dissects** Æ early OA ............................25%
- More common Æ Spontaneous healing ............75%
- Healing takes 3-4 mo Æ ↓ pain

**CLASSIFICATIONS**

<table>
<thead>
<tr>
<th>STAGE</th>
<th>PATHOLOGY</th>
<th>CARTILAGE</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>Subchondral #</td>
<td>soft and yellowish</td>
</tr>
<tr>
<td>B</td>
<td>Incomplete separation</td>
<td>cracked cartilage</td>
</tr>
<tr>
<td>C</td>
<td>Complete undetached</td>
<td>Cartilage lesion all over</td>
</tr>
<tr>
<td>D</td>
<td>Complete detached</td>
<td>Crater</td>
</tr>
</tbody>
</table>

**PAPPAS CLASSIFICATION:** Depends on age at presentation:

<table>
<thead>
<tr>
<th>TYPE</th>
<th>PHYSIS</th>
<th>PROGNOSIS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Juvenile</td>
<td>Open</td>
<td>Excellent</td>
</tr>
<tr>
<td>Adolescent</td>
<td>Partial closure</td>
<td>Either way</td>
</tr>
<tr>
<td>Adult</td>
<td>Closed</td>
<td>Poor</td>
</tr>
</tbody>
</table>

**Clinically:**
- Young male 10-20y * SONK
- No history of major trauma, Usually physically active with multiple minor traumata
- **PAIN:**
  - Insidious onset * SONK
  - Dull aching; if sharp = dissection
  - Deep Pain * SONK
  - ↑ é activity
- If fragmentation: locking, swelling, and sharp pain may occur
INVESTIGATIONS

- **XR** TUNNEL VIEWS (60° flexion é the beam // to the plateau)
  - May be the only key view in 25% of cases
  - Shadow of sclerotic fragment of separation and crater
  - Anterior to BLUMENSAAT’s line and adjacent to PCL femoral footprint
  - DD: CAFFEY ISLANDS of ossification defects develop posteriorly on the condyles (posterior to Blumensaat’s line, painless, and tend to coalesce)

- **MRI:**
  - MRI T1, serpiginous ♦ signal lines
  - MRI T2, Double line = ♦ signal margin & ♦ signal inner border
  - MRI T1Rho sequence can delineate the GAG of the cartilage

TREATMENT (based on Pappas & Ghul)

Other New Treatments for irreparable lesions in WB zone:
1- Electromagnetic pulsed cartilage delivery technique: An idea depends on the injection of Anti CD44 antibody-magnetic beads labeled mesenchymal cells into the knee after very localized provocation of the cartilage lesion by a strong magnetic field; this will affect the concentration of the mesenchymal cells into the cartilage lesion
2- Salu Cartilage implantation
3- Arthro-surface implantation

Patellar OCD
- Uncommon & present as ant. knee pain
- Distal ½ of the patella
- 30% bilateral.
- PROGNOSIS: less clear; but subchondral sclerosis = poor prognosis (as femoral OCD)
- TREATMENT principles are similar to those for femoral OCD.
Knee Bone Infarction

**Definition:**
- Ischemic death of bone & BM of a portion of the knee = AVN, that usually heals spontaneously

**Etiology:**
1. Steroids
2. Alcohol
3. SLE
4. Renal transplantation
5. Gaucher disease
6. Sickle disease
7. Caisson disease

**Pathology:**

**SITE:**
- More metaphyseal than epiphyseal extending to the subchondral plate

**MACROSCOPICALLY:**
- Pale BM
- Zry OA & cystic degeneration of the necrotic area

**MICROSCOPICALLY:**
- Bone death
- Marrow necrosis
- Granulation tissue healing
- NBF

**CLASSIFICATION**

<table>
<thead>
<tr>
<th>CLASS</th>
<th>SYMPTOMS</th>
<th>HISTOLOGICAL</th>
<th>RADIOLOGICAL</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stage 0</td>
<td>Asymptomatic</td>
<td>Abnormal</td>
<td>-ve</td>
</tr>
<tr>
<td>Stage I</td>
<td>Preclinical</td>
<td>Abnormal</td>
<td>+ve MRI only</td>
</tr>
<tr>
<td>Stage II</td>
<td>Pain</td>
<td>Precollapse</td>
<td>PXR: osteopenia, MRI: double line</td>
</tr>
<tr>
<td>Stage III</td>
<td>Pain</td>
<td>Collapse</td>
<td>Collapse</td>
</tr>
<tr>
<td>Stage IV</td>
<td>Arthrosis</td>
<td>OA</td>
<td></td>
</tr>
</tbody>
</table>

**Diagnosis:**

**Clinically:**
- Race, age, and gender are according to the cause
- Generalized pain

**PXR:**
- Lucent lesion surrounded by sclerotic shell

**MRI:**
- T1: serpiginous lines of ♦ signal
- T2: Double line sign: outer ♦ signal line + inner border of ♦ signal
- Chinese letters serpiginous lines

**Tc:** cold spot

**DD:**
- OCD ........................................lateral part of the MFC, young, active, and healthy
- SONK ........................................WB portions of the knee, elder female

**Prognosis:**
- Usually heals spontaneously

**Treatment:**
- if not ............................................as OCD
SONK = Ahlbäck disease

First reported by Alback et al. in 1968. They described a radiolucent area in the femoral condyle surrounded by a sclerotic halo and associated with a focally active bone scan.

**Aetiology**

1. Unknown
2. Vascular theory: Thrombotic veno-occlusive disease → venous hypertension and ischemia
3. **TRAUMA THEORY**: in osteoporotic patients stress fractures may be followed by bone necrosis (most accepted theory)

**Pathology:**

**SITE:**
1. Wt bearing portion of MFC ≠ OCD
2. Wt bearing portion of LFC
3. Tibial plateau

**MACROSCOPICALLY:**
- Crescentic linear subchondral # line
- Over lying hyaline cartilage: discoloration → detach → OA
- Marrow œdema

**MICROSCOPICALLY:**
- Tide mark fibrillation
- Bone necrosis + NBF

**Clinical**
1. Typically female > 60yrs ≠ OCD
2. **SUDDEN** onset of severe pain on the medial side of the knee ≠ OCD
3. pain worse at night
4. **WELL LOCALISED** tenderness over the affected condyle ≠ OCD

**Radiology**

**X-Rays**

<table>
<thead>
<tr>
<th>STAGE</th>
<th>PATHOLOGY</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stage 1</td>
<td>Normal (PXR visible lesion never develops, &amp; symptoms resolve spontaneously)</td>
</tr>
<tr>
<td>Stage 2</td>
<td>Flattening of the MFC</td>
</tr>
<tr>
<td>Stage 3</td>
<td>Radiolucency + sclerotic halo</td>
</tr>
<tr>
<td>Stage 4</td>
<td>Sclerosis enlarges + collapse</td>
</tr>
<tr>
<td>Stage 5</td>
<td>Secondary OA changes</td>
</tr>
</tbody>
</table>

**Bone scans (+ve after 72hr)**
- ♻ uptake is necessary to diagnosis
- The osteonecrotic lesion appears as a focally intense area of uptake over the affected LFC

**MRI**
- Extremely Valuable In Osteonecrosis About The Knee
- T1 - Discrete ♻ Signal In The Femoral Condyle
- T2 - ♻ Signal in the centre + ♻ Signal at the margin
Knee Disorders

Synovial Diseases:
1. PVN synovitis
2. Synovial condromatosis
3. Rheumatoid
4. Hemophilia
5. Plicae
6. Hoffa’s disease (anterior fat pad $): fat pad fibrosis and ca 2ry to trauma

ITB syndrome:
- ITB friction with LFC $\rightarrow$ localized OA especially in hill runners and cyclists
- +ve Noble’s test: Max tenderness at 30º flexion, 3 cm proximal to the joint line
- +ve Ober’s test
- DD:
  1. Lateral meniscal cyst or tear
  2. LCL injury
  3. Chondromalacia of lat patella
  4. Segond #
- Ttt is usually rehabilitation; other wise ellipse ITB excision may be required
**Osteoarthritis**

**DEFINITION**
Non-inflammatory degenerative joint disease ccc by progressive softening & disintegration of articular cartilage é associated new bone formation & capsular fibrosis

1. It is not a simple wear as it is: asymmetrical, localized, & related to abnormal loading
2. It is not purely degenerative (misnomer) as it is accompanied by a healing process in the form of reactive new bone formation

**Epidemiology:** it is the commonest joint diseases and considered a universal disorder

1. > 60y ...............................................................50%
2. Fingers, hip, knee, and spine are more affected than other sites
3. OA hands are more common in females
4. ♀:♂ = 2:1 (more DDH in ♀) unlike China & Africa

**AETIOLOGY**
- Primary - no obvious cause more é aging (effect of aging on cartilage)
- Secondary - occurs following certain predisposing factors:
  - Trauma: .............................................................direct injury, deformity, joint instability
  - Congenital: ....................................................DDH, Perthes, Blount,…etc
  - Infection
  - Metabolic: .....................................................ochronosis, hemochromatosis, gout, Paget
  - Endocrine disorder: ........................................Acromegaly and hyperparathyroidism
  - Occupation: knee (in benders)- UL (vibrating tools) –hands (boxers) –shoulder (pitchers)
  - Obesity: ..........................................................loads across the WB joints

- OA is uncommon in pts é osteoporosis
- OA is due to **FAILURE OF CHONDROCYTES TO REPAIR DAMAGED CARTILAGE**.
- There is a **DISPARITY BET.STRESS APPLIED AND CHONDROCYTE RESPONSE**. = ‘wear vs. repair’

**Mechanical causes of the disparity of the stress and strength:**

**ABNORMAL LOADS ON NORMAL JOINT:**
- articular cartilage can withstand loads of up to 25MPa without damage. Loads exceeding this Æ chondrocyte death & cartilage fissures.
- single impact loads or less intense multiple impact loads can cause articular damage.
- impact loading Æ loss of proteoglycans from matrix Æ ↓ cartilage stiffness, ↓ hydraulic permeability & disrupt collagen meshwork.
- if repair cannot keep up with damage Æ degeneration.

**NORMAL LOAD ON ABNORMAL JOINTS:**
- Instability Æ altered congruence Æ ↑ shear & compression forces on regions of cart.
- Partial loss of joint sensory innervation accelerates joint degeneration
- Forced activity following joint injury or ACL transection Æ joint degeneration, whereas immobilisation after injury prevents degeneration.
PATHOGENESIS

Theory 1
- The initiating event is **Fatigue Of The Collagen Meshwork → Hydration** of the articular cartilage → **Loss Of Proteoglycans** from the matrix into the synovial fluid
- Cartilage **Softens → Chondrocytes Die** → release of proteolytic enz → further damage
- Cartilage deformation → stresses on collagen network → damage
- Cartilage can’t withstand stresses which are concentrated on the subchondral bone → subchondral degeneration, cyst, vascularity, sclerosis
- Repair occurs at the joint margin in the form of growth & endochondral ossification → hypertrophic osteophytes (unlike the atrophic diseases e.g. RA)
- **Evidence of** collagenolytic activity; but collagen loss may be mechanical causes.

Theory 2:
- The initial lesions are **Subchondral Microfractures** following repetitive loading.
- Healing of these microfractures → undue subchondral bone & violation of Tide mark
- A stress gradient develops → articular cartilage is lost in areas of maximum stress
- Underlying bone becomes hardened and eburnated
- Proliferative changes also occur at the joint margins with formation of osteophytes.

Molecular Pathology:
1- **↑ WATER** content (unlike ↓ water content in ageing) – ↓ weakening of type 2 collagen
2- **Proteoglycans** (↑ size, ↑ chondroitin 6-sulphate, ↑ keratan sulphate, ↑ hyaluronic)
3- **↑ Chondroitin / Keratin Ratio** (unlike ageing).
4- **↑ Proteoglycan-degrading Enzymes** (collagenase & stromelysin & plasmin)
5- **↑ collagen, and proteoglycans synthesis and loss** → net result ↓ level
6- Increased levels of:
   - Matrix **METALLOPROTEINASES** (MMPs) (collagenase, gelatinase, and stromelysin).
   - **Cathepsin B & D** (proteases found in synovium, chondrocytes, and PNL)
   - **IL-1** - enhances enzyme synthesis & has a catabolic effect.
   - **IL-6**
   - **TNFα**
   - **TGFβ**
   - GAGs and polysulfuric acid

![Diagram of molecular pathways involving chondrocytes, proteoglycans, and enzymes like collagenase, plasmin, and IL-1.](image-url)
PATHOLOGY

- **Cartilage Damage**: (in pressure areas)
  - softening $\rightarrow$ fibrillation (chondromalacia) $\rightarrow$ cracks $\rightarrow$ ulceration
  - eburnation (loss of cartilage & sclerotic `polished' bone)
  - tufts of fibrocartilage on the bone surface

- **Osteophytes**: (in non-pressure areas) due to:
  - vascularization of subchondral bone
  - develop in the path of least resistance
  - capsule traction & synovial metaplasia
  - proliferation of cartilage adjacent to WB area & endochondral ossification

- Subchondral **Sclerosis**, congestion, and ↑ IOP
- Subchondral **Cysts**:
  - In the areas of max damage
  - containing thick gelatinous material
  - microfractures that degenerate & accumulate synovial fluid

- **Capsular Thickening** and fibrosis $\rightarrow$ deformity F AD IR (antero inferior capsule)
- Mild **Synovitis** usually (sometimes thick and villous)
- **Fragmentation** of osteochondral surfaces
- **Loose Bodies**: usually $\delta$ synovial metaplasia and extrusion and not separation of an osteophyte

**Microscopically**:

- Early:
  - Superficial **Splits** and irregularities
  - Deep **Metachromasia** (depletion of matrix proteoglycans)
  - $\delta$ chondrocytes clusters
  - Subchondral **Osteoblastic** activity, marked vascularity
  - Margin **Osteophytes** $\delta$ cartilage hyperplasia and ossification
- Late:
  - Extensive **CLEFTS**
  - **Cysts** $\epsilon$ amorphous material (squeezed synofluid, disintegrated trabeculae, osteonecrosis)
  - Bone **Necrosis** and denuding

**Types**

1. Hypertrophic 75%
2. Atrophic 20%
3. Progressive 5%

**Variants**:

1. Polyarticular (generalized): commonest, 50y♀, hands painful stiffness, also trapMC OA
2. Monoarticular & Pauciarticular: is the classic form, WB OA or 2ry to a cause (DDH)
3. Endemic OA: either environmental factor or genetic dysplasia
4. Unusual site OA: Milwaukee shoulder
5. Kashin-Beck disease: generalized OA in hands, elbows, knees + short stature
6. Mseleni joint disease: Polyarticular esp hips $\rightarrow$ crippling deformities $\delta$ MED & protrusio
7. Rapid destructive OA: elder $\delta$ under strong NSAIDs, usually hip $\delta$ Ca2+ crystal deposition
8. Charcot’s disease: the most severe form of OA
Knee Disorders

CP
- Old age, +ve family history, + PF
- WB joints (hands in females)
- Insidious onset & Intermittent course é attacks may lasts for few months

1. **PAIN:**
   1. The main symptom: ð ms fatigue, capsular stretch, congestion, ♦ IOP
   2. Site: anterior (sometimes posterior or lateral)
   3. ♦ é exertion, WB, Cold
   4. ♦ é rest (relief ♦ by time)
   5. Progress to be even at rest

2. **Stiffness** & loss of function: after periods of inactivity → constant

3. ♦ ROM ± crepitus

4. **Ms Wasting**

5. **Flexion & Varus Deformity** ð ms spasm → capsular contracture

6. **Tenderness**

7. **Swelling:** intermittent (effusion) or constant (cap/synovial thickening, or osteophytes)

8. **Heberden’s Nodes:** thickening of the DIP joints (*BOUCHARD’S* nodes of RA)

**Radiography**

- **PXR**
  1. Asymmetrical narrowing of joint space
  2. Subchondral sclerosis
  3. Subchondral cysts
  4. Osteophytes at the margins
  5. Late bone destruction and joint deformity and instability

- **Tc-HDP Scan**
  - ♦ uptake in bone phase (¥ vascularity & new bone formation)

- **MRI (T1rho):**
  - new MRI sequence that delineates glycosaminoglycans distribution, so it can assess early degenerative changes in symptomatic osteoarthritic subjects

**COMPLICATIONS**

1. Loose bodies
2. Backer’s cyst: capsular herniation
3. Spinal deformity 2ry to pelvic deformity → lordosis & side bend
4. Ankylosis

**TREATMENT:** Vary according to the site, stage, age, severity, and symptoms

- **Early:** keep moving, prevent overload, relieve pain.
  1. **Exercise** of supporting muscles around joints to avoid wasting and keep ROM
  2. **Protection** of affected joints from overloading: ♦ wt, walking stick
  3. Pain relief by analgesics or **NSAIDS.**
  4. Modify activity: avoid climbing stairs, squatting,... etc
  5. **HYALURONIC** acid injections
  6. **GLUCOSAMINE &chondroitin**

- **Late Surgical treatment** - is indicated for patients é persistent symptoms:
  - Arthroscopic debridement, wash, removal of LBs, and removal of osteophytes
  - Realignment osteotomies may be done in younger patients to redistribute WB load, decompress the congested metaphysis relief pain dramatically
  - Arthrodesis if young and stiffness is accepted
  - Total joint arthroplasties for older patients (over 60) in advanced cases
- **New & Experimental Treatments:**
  1. Soft tissue grafts - periosteal / perichondral
  2. ACI
  3. Mosaicplasty
  4. Artificial matrix - carbon fibre, collagen, polylactic acid
  5. Fresh osteochondral allografts
  6. Doxycycline → cartilage collagenase activity
  7. Transforming growth factor beta (TGF beta) can repair partial thickness lesions
  8. Metalloproteinase inhibitors → matrix destroying enzymes & block cytokines
  9. Gene therapy - Genes that have either anti-arthritic or synthetic properties can be delivered into the joint via non-replicating viral vectors; e.g. IL1

The potential long-term treatment or cure of OA really lies in the development of agents that reverse the balance between degradative and synthetic processes of the chondrocyte.

---

**Forestier’s Disease**

**DISH (Diffuse Idiopathic Skeletal Hyperostosis)**
- a type of OA with significant amount of osteophyte formation
- more common in elderly men
- associated with diabetes & gout
- prevalence of DISH may be as high as 28%
- spine shows calcification of the anterior longitudinal ligament & peripheral disc margins (disc space height is preserved)
- Marginal osteophytes may be seen in all peripheral joints
- Clinically have spinal stiffness & heel pain (with spurs)
- Differs from Ankylosing Spondylitis by absence of inflammatory markers & HLA-B27
- increased risk of heterotopic ossification following THR
Rheumatoid Arthritis

- Affects 3% of women & 1% of men
- Hand > Knee > hip > cervical spine

**Diagnosis:**
- **ARA Criteria** (American Rheumatism Association):
  1. **Morning stiffness**
     Lasting at least 1 hour before maximal improvement.
  2. **Arthritis of 3 or more joint areas**
     At least 3 joint areas simultaneously have had soft tissue swelling or fluid (not bony overgrowth alone) observed by a physician; the 14 possible joint areas are right or left proximal interphalangeal (PIP) joints, metacarpophalangeal (MCP) joints, wrist, elbow, knee, ankle, and metatarsophalangeal (MTP) joints.
  3. **Arthritis of hand joints**
     At least 1 area swollen (as defined above) in a wrist, MCP or PIP joint.
  4. **Symmetric arthritis**
  5. **Rheumatoid nodules**
     Over bony prominences, or extensor surfaces, or in juxta-articular regions
  6. **Rheumatoid factor +ve**
  7. **Radiographic changes**
     - At least 4 of 7 criteria.
     - Criteria 1 through 4 (at least 6 weeks).
  8. **Clinical Staging:**
     - 7 ccc ......................... Classic
     - 5 ccc ......................... Definite
     - 3 ccc ......................... Probable
     - 2 ccc .......................... Possible

**Aetiology:**
- Genetic susceptibility: RA is common in first degree relatives of RA patients and twins
- Immunological process: HLA-DR4 & DW4 encoded on chromosome 6; and is found on the surface of APC (antigen presenting cells); & when interact é the antigen (some times the antigen with the HLA form the activating complex) → autoimmune response
- When APC and T-cells interact → cell proliferation + cyokines secretion → ⊕ phagocytes & B-cells
- Rheumatoid factor: Anti-IgG auto antibodies which is detected in the serum of the patient
Pathology:

Stage 1: Synovitis
- vascular congestion & effusion
- synoviocyte proliferation $\rightarrow$ VILLOUS formation
- infiltration of subsynovial layers by PMNs, lymphocytes & plasma cells

Stage 2: Destruction
- a PANNUS of granulation tissue creeps over the articular surface eroding cartilage & bone
- cartilage destruction occur partly δ proteolytic enzymes & vascular tissue
- bone destruction occur partly by proteolytic enz, & osteoclastic activity
- direct invasion occurs at the margins of the joint
- similar changes occur in tendon sheaths $\rightarrow$ rupture

Stage 3: Deformity:
- a. articular destruction
- b. capsular stretching
- c. tendon ruptures
- acute inflammation subsided

Extra-articular Manifestations:
- Nodules - in 20% of RA - skin, synovium, tendons, sclera, viscera
- Lymphadenopathy & Splenomegaly ...... FELTY'S Syndrome (pancytopenia)
- $\uparrow$ salivary & lacrimal gland secretion .... SJORGEN Syndrome
- Pulmonary (pleurisy & rh nodules) ........ CAPLAN ®
- Vasculitis
- Myopathy & neuropathy, or direct compression from synovitis
- Visceral: pericarditis, nodules

Clinically:
- 40 female may be $\pm$ positive family history
- Early:
  - Painful swollen HAND joints $\pm$ morning stiffness
  - PAINFUL LIMPING
  - $\uparrow$ ROM $\pm$ crepitus
  - loss of WEIGHT, weakness
  - O/E: symmetrical swelling, tenderness, crepitation, synovial hypertrophy
- Late:
  - Valgus Deformity
  - Instability, tendon rupture
  - Path #: from the disease and drugs
  - BOUCHARD'S nodules, Swan neck, Boutonniere, Z-thumb, fingers ulnar deviation, wrist radio-volar deviation, valgus knee, valgus feet, clawed toes, atlanto-axial subluxation
- Types of presentations:
  - Palendromic: starts intermittent episodes of multi joint affection $\rightarrow$ evolve to classic
  - Systemic: severe form $\pm$ visceral affection
  - Monoarticular: usually knee. Also, may present as tenosynovitis or CTS
  - Myalgic: as fibromyalgia rheumatica but with +ve RF
Laboratory Findings:
- ↑. ESR, CRP
- RF +ve in 80%, ANA 30%
- ACCP (anti Cyclic Cetrolinated Peptide): 97% early +ve in RA even in seronegative RA
- anemia: δ abnormal erythropoiesis, and chronic blood loss from analgesic gastritis
- WBC: Normal or ↑ (if ↓ suspect Felty)
- ↓ Complement

Synovial biopsy & fluid:
- Biopsy: is non specific to RA
- Fluid: ↓ ptn, C, glucose / poor clot / ↑ RA cells & PNL

PXR: LARSEN - DALE RADIOLOGIC INDEX
1- Stage I: juxta-articular osteopenia
2- Stage II: Narrow joint space (usually bilateral, symmetrical, concentric ± protrosio)
3- Stage III: Bone erosion of the head near the neck + bone cysts
4- Stage IV: Deformity (don’t forget the cervical PXR)
- Usually no sclerosis nor osteophytes (except if 2ry OA)
- Other complications: AVN, fracture neck

DDx:
1- Seronegative: SLE, Still’s
2- AS: spondarthropathy
3- Reiter’s: Conjunctivits, urethritis, Arthritis
4- Gout & CPPD: crystals
5- OA: DIP affection, osteophytes
6- Polymyalgia rheumatica: pelvic, and pectoral weakness, and aching, +ve steroid test
7- Sarcoidosis: Erythema nodosum, Hilar LN, +ve Kveim test

Management Principles:
- Stop the Synovitis
  - Rest
  - DMAR Drugs (Disease Modifying Anti-Rheumatic) - Pyramid Approach = NSAIDs - antimalarials - sulphasalazine – gold – MTX – D-penicillamine - Azathioprine – Leflunomide + low dose steroids
  - Synovectomy - chemical, irradiation, surgical
- Prevent Deformity
  - Splintage
  - Physiotherapy
  - Tendon repairs & joint stabilisation
- Reconstruct (start ê knee if > 45º flexion deformity)
  - Arthroplasty is the gold standard
  - Osteotomy not done:
    - Doesn’t remove the cartilage ω is a source of inflammation
    - RA is concentric & no healthy cartilage
  - Arthrodesis not to be done (bilateral)
- Rehabilitate & keep moving
  - Occupational therapists - aids, support
  - Physiotherapy
At Onset: NSAID, exercise  
Early: NSAID, Steroids, DMD, local injections, physiotherapy, Rest and splintage  
Erosive: DMD, splintage, operative (synovectomy, tendon repair, joint stabilization)  
Late: Reconstructive arthroplasty

Problems é surgery:
1- Bilateral knee involvement
2- Ipsilateral hip involvement ...........................................start é knee if flexion def >45º start é it
3- UL affection ...............................................................needed for PWB
4- Infections ......................................................................scan for oral, UTI, skin before surgery
5- Bad general condition & systemic disease
6- Cortisone therapy .........................................................need to ♦ the dose preop
7- Atlantoaxial subluxation .................................................difficult intubation
8- Femoral #s .................................................................be gentle & ready
9- Loosening .................................................................Cemented or Hybrid are favored

Drugs Details:

<table>
<thead>
<tr>
<th>Drug</th>
<th>Mechanism</th>
<th>A/E</th>
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</thead>
<tbody>
<tr>
<td>NSAIDs</td>
<td>PG synthesis → ♦ pain and inflammation</td>
<td>Gastric Upset</td>
</tr>
<tr>
<td>Antimalaria</td>
<td>PG &amp; phagocytic activity of PNL</td>
<td>Lucoma</td>
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<tr>
<td>Sulphaslsazine</td>
<td>Anti-inflammatory</td>
<td>Megaloblastic anemia</td>
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<tr>
<td>Gold</td>
<td>Alters the function of macrophages and complement</td>
<td>Thrombocytopenia</td>
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<tr>
<td>Methotrexate</td>
<td>Immune suppression</td>
<td>Liver toxicity</td>
</tr>
<tr>
<td>D-Penicillamine</td>
<td>Dissolve RF complexes in joints to be excreted</td>
<td>Late resp.&amp; nephrotic</td>
</tr>
<tr>
<td>Azathioprine</td>
<td>Immuno suppression</td>
<td>Liver toxicity</td>
</tr>
<tr>
<td>Leflunomide</td>
<td>DiHydro-Orotate Dehydrogenase → ♦ T-cell prolif</td>
<td></td>
</tr>
</tbody>
</table>

Complications:
1]. Fixed Deformities  
2]. Joint Rupture  
3]. Infection  
4]. Spinal cord compression  
5]. PN compression  
6]. Vasculitis  
7]. Amyloidosis, proteinuria  
8]. progressive RF

Poor prognostic signs:
1]. Very high RF  
2]. Peri-articular erosions  
3]. Nodules  
4]. Muscle wasting  
5]. Joint contractures  
6]. Vasculitis

Prognosis:
1]. 10% improve after first attack of synovitis  
2]. 60% have remissions & exacerbations  
3]. 20% have severe joint erosions requiring multiple operations  
4]. 10% become completely disabled
Sero-negative Inflammatory Arthroses
Common Clinical Features (diff. to rheumatoid arthritis)
1. Enthesopathy (inflammation of ligament & tendon insertions)
2. Axial skeleton involvement
3. Asymmetrical joint involvement
4. Heel pain (Achilles tendinitis, calcaneal bursitis, plantar fasciitis)
5. Extraskeletal involvement = skin, mucous membranes, GUT, GIT
6. HLA-B27
7. Radiological:
   1. ankylosis
   2. calcification of adventitia
   3. No osteopaenia (typical of rheumatoid)

- young females
- Arthritis in 75% of SLE patients

Clinical:
- fever, anorexia, weight loss, malaise
- skin rashes (butterfly malar rash)
- Raynaud's phenomenon
- splenomegaly
- nephritis, carditis

Laboratory:
- anaemia, leucopenia
- high ESR
- ANA positive

Treatment:
- Corticosteroids for severe disease

Complications:
- AVN hip (? from steroids)
Ankylosing Spondylitis

Aetiology:
- Affects spine & sacroiliac joints primarily
- Prevalence = 0.2%
- Males > females & Familial involvement
- HLA-B27 marker

Pathology:
- Inflammation & erosive destruction of:
  1. Diarthrodial joints = sacroiliac, vertebral facet, costovertebral
  2. Fibro-osseous junctions - intervertebral discs, sacroiliac ligaments, symphysis pubis
- 3 Stages:
  1. Inflammation - round cell infiltration, granulation tissue, joint erosion
  2. Fibrosis - replacement of granulation tissue with fibrous tissue
  3. Ossification - of fibrous tissue (e.g. syndesmophytes)

Clinical:
1. Spinal stiffness (classical posture)
2. **Wall Test.**
3. Chest expansion < 7cm
4. Hip involvement with FFD
5. Achilles tendon insertion pain
6. Difficult cervical spine fractures with epidural haemorrhage

Extraskeletal:
1. Prostatitis
2. Conjunctivitis & uveitis in 20%
3. Carditis, aortic valve disease
4. Pulmonary fibrosis

Radiology:
- Squaring of vertebral bodies
- Syndesmophytes
- Bamboo spine
- erosive arthritis with progressive ankylosis

Laboratory:
- High ESR
- HLA-B27 in 90%
- RF negative

Management:
- Postural management
- NSAIDs
- Operations to correct deformity & restore mobility
  - Lumbar / cervical spine osteotomies
  - THR

Ankylosing Hyperostosis (Forestier's Disease)
- common disorder in older men
- widespread ossification of tendons & ligaments
- Diff to AS:
  - No erosive arthropathy
  - Normal ESR
Reiter's Syndrome

Hans Reiter, 1916
Triad = Urethritis + Arthritis + Conjunctivitis

**Aetiology:**
- follows dysentery or venereal infection
- Causative organisms:
  - Chlamydia trachomatis
  - shigella, salmonella, campylobacter, Yersinia
  - Lymphogranuloma venereum

**Clinical:**
1. Acute Phase:
   - inflammatory arthritis of: knee or ankle or foot joints
   - pustular dermatitis of the feet (keratoderma blennorrhagicum)
   - balanitis
   - painless buccal ulceration
2. Chronic Phase:
   - spondyloarthropathy
   - sacroiliitis in 60%

**Radiology:**
- erosive arthropathy similar to AS

**Laboratory:**
- HLA-B27 in 80%
- ESR high in acute phase
- organism may be isolated from urethral fluids or faeces

**Treatment:**
- Supportive
- Tetracycline for persistent urethral infection

---

Enteropathic Arthritis

Peripheral arthritis - in 15% of people with Crohn's disease & ulcerative colitis
Sacroiliitis & spondylitis - in 10% of people with Crohn's disease & ulcerative colitis
Must exclude the following first:
1. Septic arthritis of the hip from direct spread from bowel
2. Psoas abscess
3. AVN from steroids
4. Osteopenia from malabsorption & steroids
Psoriatic Arthritis

Prevalence of Psoriasis is 1-2%; only 5% of those will develop psoriatic arthritis.

Aetiology:
- Familial
- HLA-B27 in 60%

Pathology:
- similar to rheumatoid arthritis, but destruction usually severe (arthritis mutilans)
- rheumatoid nodules are not present

Clinical:
- IPJs of fingers & toes affected (Arthritis Mutilans)
- asymmetrical
- 'sausage digits'
- sacroiliac & spine involvement in 30%
- psoriasis of skin & nails usually precedes arthritis - nail pitting
- ocular inflammation in 30%

Radiology:
- 'pencil in cup' deformity
- arthritis mutilans

Laboratory:
- RF negative
- HLA-B27 in 60%

Diagnosis (diff. to RA):
1. RF Negative
2. asymmetrical
3. distal finger joints
4. sacroiliitis & spondylitis
5. No rheumatoid nodules
6. No tendon involvement

Treatment:
- immunosuppressive agents in severe disease
- prevent deformities
- arthrodesis of IPJs to improve function
Tuberculosis is common throughout the world
Usually due to Mycobacterium tuberculosis or Mycobacterium bovis infection

**Pathology:**

1- 1ry lesion:
   - **Site:**
     - Lung usually (sub-pleural **Ghon's Focus** and mediastinal lymphadenopathy)
     - Pharynx  • Gut
   - **Changes:**
     - Local inflammatory focus → Lymphangitis → Lymphadenitis
   - **Seculae:**
     - TB bacilli remain dormant in LN
     - Body is sensitized to toxins (Type IV cell mediated delayed hypersensitivity)

2- 2ry lesion:
   - Due to reactivation, repeat exposure, ♦ immunity (e.g. drugs or HIV infection)
   - Results in more significant symptoms as it spreads to:
     - Lung .................................. military TB, TB bronchopeumonia
     - Meninges: ......................... TB meningitis

3- 3ry lesions (10% affect the musculoskeletal system)
   a. **Tuberculoma** formation:
      - Central **CASEATION** necrosis (coagulation necrosis)
      - Surrounded by **EPITHELIOID** cells, **LANGERHANS** giant cells, **LYMPHOCYTES**
      - They tend to coalesce to form a wide area of caseation necrosis
      - Spread to cartilage → destruction and spread to joint space
   b. **TB Spondylitis:** (mainly thoracic)
      - Starts ant. in the body at multiple level; destroy bone & respect ! disc except late
      - **POTT'S PARAPLEGIA** δ (kyphosis, abscess, ♦ blood supply to the cord)
   c. **TB Arthritis** (hip, knee, ankle, shoulder, then wrist)
      - Synovium is **THICKENED** é Cell rich **EFFUSION**
      - Granulomatous **PANNUS** may form & creaps on the cartilage & bone
      - Cartilage & bone **EROSION** (peripherally at synovial reflection)
      - Juxta articular **OSTEOPENIA** δ hyperaemia
   d. **Appendicular skeleton:**
      - Metaphyseal bone destruction (no sclerosis, no periosteal reaction)
   e. **TB Dactylitis = Spina Ventosa** (middle and distal phalanx)
      - Digit is swollen spindle shape é little pain
      - Starts diaphyseal é bone rarefaction + PNBF + soft tissue swelling
      - PXR: Spina (spindle shaped digit) Ventosa (full of are i.e. rarefied)
   f. **Cold Abscess:**
      - Infected LN may **COALASE** together to form big area of caseation
      - Caseation spread via soft tissue planes
      - May burst to skin to form a sinus
      - May collect at a distant site far from original pathology e.g lumbar TB → groin

4- Healing:
   a. Resolution
   b. Fibrous Ankylosis
   c. Dormant bacilli
**Clinically:**

- **General:**
  - Night sweat, night fever
  - Loss of weight, loss of appetite
- **Local:**
  - **Night Cries:** joint is splinted at day time by spastic ms → spasm is relieved → stretch or compression of the damaged tissue → pain
  - Marked **Ms Wasting**
  - Marked **Synovial Thickening**
  - ↓ ROM & **Stiffness**
  - Joint **Deformity**
  - Spine: pain, abscess kyphosis, neurological manifestation

**PXR:**

- **Juxta Articular Osteopenia** → washed out bone ends
- ↓ **Joint Space** (in children epiphysis → 2ry to hyperaemia)
- Peripheral bone **erosion** and **cystic** subchondral lesions
- **NO periosteal** reaction
- **NO Sclerosis**
- Spine erosion, collapse, paraspinal abscess calcification & kyphosis → crowded related ribs → **Sun Ray Appearance**
- In hip erosions occur in 3 areas (Babcock’s Δ); erosion of superior acetabulum → **Wandering Acetabulum**

**Investigation**

- ↑ ESR
- Leucopenia → relative lymphocytosis
- ↓ Lymphocyte/monocyte ratio may reach 1
- +ve Mantoux test (indicate TB infection recent or old)
- +ve PCR
- Synovial fluid:
  - ↑ ptn, ↓ glucose
  - ↑ viscosity, poor mucin clot
  - Red acid-alcohol fast **intracellular** bacilli → **Zeal Nielsen**...20%
  - Cultivation on **Lovenstein Jensen** media or Dorset egg ..........80%
    - Difficult to culture needs adequate relevant conc. (centrifuged) sample
    - Decontaminate & remove other organism (Petroff method)
    - Keep 35º for 6 wk
  - Organisms also **Fluoresce With Auramine** staining
  - Negative microscopy does not exclude tuberculosis
  - Guinea pig inoculation
- Synovial biopsy:
  - Granulomatous reaction (caseation + Langerhans + epitheliod + lymphocytes)
  - Characteristic evidence of a delayed hypersensitivity reaction
Skin tests
- Delayed hypersensitivity reaction used to diagnose tuberculosis
- The two commonest tests are the Mantoux and Heaf test
  - **Mantoux test:**
    - 0.1 ml of purified protein derivative is injected intradermally
    - +ve if .................................. > 5 mm papule at 72 hours
  - **Heaf test**
    - PPD is inoculated into the skin using a gun to produce multiple punctures
    - +ve if .................................. > 4 papules at puncture sites at 72 hours
- Positive skin test are indicative of active infection or previous BCG vaccination

**DDx**
- Transient synovitis
- Monarticular RA
- Subacute arthritis
- Old septic arthritis

Treatment
- Rest:
  - Splintage and traction to prevent ms spasm and deformity
  - Maintained till pain and inflammation is over
  - Then motion is encouraged; if pain & spasm return, resume splints
- Chemotherapy:
  - RIPES - Rifampicin, Isoniazid, Pyrazinamide, Ethambutol, Spectinomycin
  - Rifampicin + Isoniazid 6-8 mo
  - Ethambutol (or pyrazinamide, spectinomycin) for the initial 8wk
  - Streptomycin is toxic
- When to start surgical ttt:
  - Clinically: ................................................ No fever, no wt loss, no spasm
  - PXR: .............................................. ✶ calcification
  - Lab: .......................................................... ✶ ESR, ✶ lymph/monocyte ratio ≥5
- Operative:
  - Drainage of a TB focus is seldom done nowadays
  - Cold abscess ........................................... calls for urgent drainage
  - Young age .................................................. synovectomy
  - Clearance operation .................................. debride all infected & dead tissue
  - Painful fibrous ankylosis in middle age ...... arthrodesis
  - Painful destroyed joint .................................. spacer then arthroplasty
Charcot's Osteoarthropathy

NEUROPATHIC JOINT

It is a severe form of Degenerative OA caused by loss of proprioception to involved joints

Etiology:

1- DM is the commonest cause ................................. 0.5% will have charcot
2- TABES DORSALIS: .................................................. 10% will have charcot
   • Charcot's is not a syphilitic infection of the joint, it is 2ry to tabes dorsalis
   • Knee & hip are the mostly affected
   • Knee jerk is lost
   • Argyll Robertson Pupil

3- SYRINGOMYELIA: ..................................................... 50% will have Charcot
   • Degenerative condition of the spinal cord (at the shoulder girdle segment)
   • Shoulder and elbow are the mostly affected
   • Lost of reflexes & thermal sensation

4- Other causes:
   • Congenital indifference to pain
   • Meningomyelocele & dysraphism
   • CPPD
   • Multi steroid injections
   • Alcoholic & amyloidosis

Pathogenesis:

• Loss of proprioception \rightarrow repetitive trauma to unprotected joints \rightarrow OA
• Usually extreme movement and harmful strains are prevented by the protective
  contraction of the ms evoked by the Proprioceptive reflex arc
• é loss of joint sensibility this function is lost + no pain \rightarrow trauma & strain are not appreciated
• On the long run this leads to severe OA & disorganization of joint
• Alternatively sympathetic reflexes may lead to hyperaemia & bone resorption

Pathology: exaggerated OA

• Articular cartilage ............................................... Degenerated, destroyed, disorganized
• Metaphysis ................................................................ rarefied & irregular destruction
• Osteophytes & Loose bodies (LB) ........................ is a characteristic feature
• Intra-articular ......................................................... ACL vanish
• Synovium ............................................................... thick, polypoid, chondromatosis, pannus
• Capsule ..................................................................... stretched & lax
• Joint ......................................................................... Disorganized, Grotesque, dislocated

Clinically: Painless joint + lost reflexes

<table>
<thead>
<tr>
<th>TYPE</th>
<th>INCIDENCE</th>
<th>COURSE</th>
<th>TYPE OF JOINT</th>
<th>LB &amp; OSTEOPHYES</th>
</tr>
</thead>
<tbody>
<tr>
<td>1-Hypertrophic</td>
<td>commonest</td>
<td>Chronic</td>
<td>WB</td>
<td>Marked</td>
</tr>
<tr>
<td>2-Atrophic</td>
<td>Rare</td>
<td>Acute</td>
<td>NWB</td>
<td>Not evident</td>
</tr>
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PXR:

• Joint is markedly **DESTROYED, DISORGANIZED, and DISLOCATED**
• Hypertopic NBF

Scans: hot as infection .............................................. but cold indium scan

Treatment:

• Conservative ttt: ♦ wt, orthotics .............................. (no NSAIDs, or steroids)
• Operative ttt: arthrodesis & Ilizarov ..................... (No arthroplasty)
• Aim is to achieve fibrous Ankylosis
• Amputation are rarely indicated .............................. if broken neglected & severely infected
**Haemophilia**

- Combined incidence is 1 in 10 000 male births

**Haemophilia A (Classic haemophilia)**

- Deficiency of factor VIII
- Most common of severe disorders
- XR inheritance
- Only affects males. Carried by females. 30% are new mutations
- Severity varies depending on level of clotting factor VIII:
  - 50% ............................................normal
  - 25-50% .....................................seldom have problems
  - 5-25 % ......................................severe bleeding with operations (mild)
  - 1-5 % .......................................severe bleeding after minor injury (moderate)
  - <1% .........................................spontaneous bleeding (severe)
- 50% of known haemophiliacs are moderate or severe
- 5% of patients develop antibodies to factor VIII
- Very difficult to treat. Infused factors are destroyed

**Christmas disease**

- Deficiency of factor IX
- Less common
- Also XR inheritance
- Similar spectrum of severity

**Von Willebrand’s disease**

- Deficiency of von Willebrand factor
- AD inheritance

**Clinical Presentation**

- Male with positive family history
- May present after circumcision; Otherwise first bruising appears at 3-4 months; Severe bruising at walking age; May be spontaneous haemarthroses; Milder forms may present after dental extraction or operative procedure
- Bleeding In the form of:
  - Bruises
  - Muscle hematomas
  - Haemarthroses
  - May occur sometime after injury
- Defect is in clot formation (2nd phase) rather than haemostasis (1st phase)
- May be no known trauma
- HIV ..........................................................70% of whom received pooled factor VIII

**Investigations**

- Platelet count and bleeding time normal
- APTT prolonged and PT normal ie Intrinsic
- Low factor VIII coagulant activity diagnostic

**Treatment**

1. Blood products
   - Initially fresh frozen plasma used ...........but the volume was too great
   - Then cryoprecipitate used .....................higher level of F VIII; stored at very low temp
   - Then factor VIII concentrates .................Available in convenient dried concentrate form
     - Administration: 1 U/KG elevates VIII by 2% (½ life is 6-12 hrs) till 15-30%
**Haemophilic Arthropathy**

- Recurrent intra-articular bleeding can cause:
  1. Chronic synovitis
  2. Progressive articular destruction

**Pathology:**
- **HAEMOSIDERIN ACCUMULATION** in synovial cells
- **SYNOVITIS** makes synovium more susceptible to further injury
- Intra-articular clots → Organize → **ADHESIONS** → may tear → bleed
- Cartilage gradually eroded ↓ **LIBERATION OF LYSOSOMAL ENZYMES**
- Vascular pannus covers articular surface and further destroys it
- Subchondral bone exposed - Subchondral cysts - Osteoporosis ↓ disuse
- ↑ blood flow to joint → **EPiphyseal Overgrowth** & osteophytes

**Muscle**
- Recurrent bleeds lead to **FIBROSIS AND CONTRACTURE**
- May result in deformity
- May be complicated by:
  1. Compressive nerve palsy
  2. Compartment syndrome
  3. Cysts and pseudotumour
  4. Cyst from massive ms bleed (before it is absorbed draw in more fluid by osmosis)
  5. Erode through skin or viscus and become an abscess

**Pseudotumour** *(From subperiosteal or intraosseous bleed )*
- **SUBPERIOSTEAL** ...........................................shows periosteal stripping + NBF
- **INTRAOSSEOUS** ...........................................ill-defined lesion + extensive osteolysis ± NBF
- May be confused with sarcoma, GCT, ABC
- May destroy bone and lead to pathological fracture

**Stage**

<table>
<thead>
<tr>
<th>Stage</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stage 1</td>
<td>After acute haemarthrosis • Soft tissue swelling</td>
</tr>
<tr>
<td>Stage 2</td>
<td>Subacute haemarthrophy • Epiphyseal osteoporosis; overgrowth</td>
</tr>
<tr>
<td>Stage 3</td>
<td>Chronic ê no narrowing • Squaring off of patella • Widened intercondylar notch of knee • Widened trochlear notch in elbow</td>
</tr>
<tr>
<td>Stage 4</td>
<td>Chronic severe • Marked narrowing of joint space</td>
</tr>
<tr>
<td>Stage 5</td>
<td>Destructive • Joint disintegration</td>
</tr>
</tbody>
</table>

**Clinical**

1. **ACUTE HAEAMARTHROSIS**
   - Most commonly affects - Knee, Elbow, Ankle, Hip and shoulder
   - Joint is Hot red and swollen, Acutely painful, Held in flexion
2. **SUBACUTE HAEAMARTHROSIS**
   - After 2 or more acute bleeds
   - Synovium is thickened and boggy
   - Moderate ♦ ROM
   - Pain not prominent
3. **CHRONIC DISEASE**
   - After subacute form present for 6 m or more
   - Bleeds less frequent and more difficult to detect
   - ♦ ROM due to
     - Fibrous contracture
     - Deformity
     - Muscle wasting
   - Muscles most commonly affected = Iliopsoas, Thigh, Calf, Forearm

**X-rays** ............ Progressive destruction
Treatment

**Acute haemarthrosis**
- Usually treated at home; Usually single dose sufficient to ✶ pain quickly
- Immediate IV dose of factor VIII to achieve level above 30%
- Adequate analgesia (not aspirin or NSAIDs) + Immobilise limb for first 24 hrs
- Place of aspiration or washout controversial
  - Reduces pain and swelling
  - Removes blood ✶ degradation with possible toxic effects
  - No evidence that it decreases risk of arthropathy

**Subacute haemarthropathy**
- Further treatment indicated if:
  i. Failure to respond to above treatment
  ii. Two or three bleeds in short period of time
- Initially:
  - Prednisolone 5 days
  - 2 or 3 doses of factor VIII for level 30%
- If still not settled: 6-8 wk course of Prophylactic factor replacement + Active physiotherapy

**Chronic haemarthropathy**
1- Nonoperative
  - 6 months of small dose of prednisone, prophylactic factor replacement, active physiotherapy
2- Synovectomy
  - When nonoperative treatment fails
  - Not indicated beyond stage 3
  - Open or arthroscopic
  - Reduces number of bleeds
  - Does not slow cartilage degeneration
3- Corrective surgery
  - For stage 4 and 5
  - Presence of antibodies is contraindication to elective surgery
  - Avoid pins that penetrate skin
  - Screen for HIV and hepatitis B preoperatively
  - Check factor levels intraoperatively
  - **Meticulous Haemostasis**
    - Increase factor levels for procedures such as MUA
  - **Osteotomy**
    - Most common deformity is flexion contracture with valgus of knee. Can be corrected with supracondylar osteotomy
  - **Arthrodesis**
    - Not often indicated because multiple joints involved
    - Useful for ankle and subtalar joints
  - **Total Joint Replacement** of hip and knee
    - Higher perioperative complication rate (esp. Haematoma and infection)
    - Only **50% Survival At 10 Yrs**
    - Maintain factor **VIII Levels At 100-130%** for 2 days post-op; 50-60% for 2 wks; 30-50% for 6 wks
  - **Tendon Lengthening** of tendo Achilles for equinus deformity
**Gaucher's Disease**

**Discussion:**
- In 1882, Gaucher described idiopathic hypertrophy of spleen w/o leukemia
- **AUTOSOMAL RECESSIVE** deficiency of lysosomal enzyme **GLUCOCEREBROSIDASE**. It is the most common genetic lysosomal storage disorder & results in accumulation of of glucocerebroside in retinuloendothelia cells
- Commonly seen in children of Jewish descent.

**Pathology:**
- Bone involvement is common in Gaucher's disease:
  - expansion of metaphyses - failure of remodeling → **'ERLENMEYER FLASK'** distal femur (also found in Niemann-Pick disease and osteopetrosis).
  - erosion of the cortices of bone ('moth eaten')
  - AVN femoral head ('Bone within bone' appearance)
  - Bone crisis similar to Sickle Cell Anaemia
  - Pathologic frx.

**Histology:** lipid-laden Histiocytes.

**Classification:**
- **TYPE I:** (adult form) - chronic noneuronopathic type; - central nervous system is spared & disease is characterized by slowly progressive visceral and osseous involvement; enlarged spleen may cause mechanical problems, including abdominal distention and abdominal pain as well as shortness of breadth, as well as causing pan cytopenia; splenectomy may be performed for thrombocytopenia; bone pain is a common complaint, which often resolves after 1-2 days; bone pain may also be due pathologic frx, osteomyelitis, or DJD;
- **TYPE II:** acute neuropathic form; most patients don't survive more than 9 months;
- **TYPE III:** sub acute neuronopathic; convulsions are frequent;

**Clinical Presentation:**
- Bone Crises (episodes of pain and swelling), sometimes accompanied by fever but w/o x-ray changes, are common, recurrent manifestations of the disease. - often the pain cannot be controlled w/ narcotics; pain may last days or several weeks;
- **AVN OF THE FEMORAL HEAD** - is the most symptomatic lesion; bilateral disease is comon;

**MRI:**
- $T2$ signal is seen in the marrow of long bones, vertebrae, & hips and is related to the areas of marrow infiltrated by Gaucher's lipids.

**Treatment:**
- **PATHOLOGIC FRX:** - are best treated conservatively; frxs are esp common in yound children. Frx proximal end of femur may occur in children & adults - these may be treated non operatively although there may be with some varus deformity
- **BONE CRISIS:** .............................................................. brief bed rest + NSAIDs → crutches as tolerated
- **AVN:** .............................................................. brief bed rest + NSAIDs → crutches as tolerated
  - THR carry $\uparrow$ risk of infection, bleeding, loosening
- **ENZYME REPLACEMENT** therapy is now available.
• **Pellegrini-Stieda:**
  - Deep MCL laceration → hematoma formation
  - Ossification of the hematoma → painful lump deep to the MCL
  - ttt: removal if symptomatic

• **Semimembranosus Bursa**
  - The bursa between the semimembranosus and the medial head gastroc
  - CP:
    - Painless lump behind the knee slightly to the medial side
    - Painful on knee flexion
    - Fluctuant lump and the fluid could not be pushed into the joint → ms that obstruct the communication
    - Sometimes painful
  - ttt: excision via transverse incision
  - Recurrence is common

• **Baker's cyst**
  - Synovial herniation of the posterior capsule
  - Occur → OA, RA, TB
  - CP:
    - Older people
    - Midline at the level of the joint
    - Fluctuant lump
    - Sometimes rupture and become painful → release of the synovial content into the calf (DD: DVT)
  - Arthrography:
    - Reveal the communication to the joint
  - ttt: aspiration + cortisone instillation
  - Excision is not recommended as the cyst usually recur unless the cause is treated
Knee - Osteotomies

- 60% of the load of the body weight passes through the medial compartment of the knee.
- Loads up to 4 times body weight are produced on climbing stairs.
- Osteotomy redistributes the force.
- Valgus osteotomy most commonly performed (varus knee) and is indicated in patients that have unicompartmental disease, are less than 60 - 70 years old, are of optimal weight, have an active occupation or lifestyle where they want to maintain and have a good range of motion.
- NB: ROM is not likely to improve with an osteotomy.

**INDICATIONS**

1. **Age:** physiologic age < 60 yrs in an athlete, laborer, or anyone who needs to kneec down such as for gardening (TKR will generally not allow the patient to kneel)
2. **Weight:** > 80 kg are at increased risk for component failure;
3. **Angular Deformity:**
   1. >15º varus deformity (often patients will have varus laxity)
   2. > 10º valgus deformity
4. **Sagittal Range Of Motion:**
   1. < 15 º flexion contracture
   2. > 90 º flexion
5. **Unicompartmental Involvement**

**Contraindications:**

1. **Tibial Subluxation** > 1 cm
2. Bicompartmental or tricompartmental arthritis
3. OCD lesion of more than 5 mm deep
4. ACL tear
5. RA & inflammatory arthritis
6. Inadequate bone stalk

**CLINICAL**

- Observe patient **Walk** (look for varus thrust & foot progression angle)
- Test **Ligamentous Stability** tests
- **Q Angle**
- **Compensatory Arc Of Motion** - to correct a valgus knee deformity - for a 20º varus osteotomy, 20º of hip abduction is required so pt does not end up in an add deformity
- Examine the **Foot Fixed Varus Deformities** which may worsen med compartment loading
- **LDD** Coventry closing wedge osteotomy might be indicated, where as, if the arthritic side is shorter (than the other leg), then consider opening wedge osteotomy
- **Staheli Rotational Profile** is done to rule out rotational malalignment

**Preoperative Planning**

**Radiology**

- **Standing Long-Leg** film with patellae facing forward (rather than the patient's feet)
  - Mechanical axis = centre of femoral head - medial tibial spine - centre of ankle joint
  - Weight-bearing axis = centre of femoral head - centre of ankle joint
  - Anatomical axis = line along axis of the femur to the intercondylar notch and the line formed by the interspinous region to the centre of the ankle
- **Supine** films
- Perform the **Alignment Test.**
**Technique**

**Medial Compartment Disease**
- High Tibial Osteotomy (HTO) above the tibial tubercle:
  - Lateral closing wedge & fibular shortening
  - Open medial wedge
  - Dome shaped osteotomy
  - Rap osteotomy in severe degrees and Blount’s

- **OVERCORRECTION** of the mechanical axis by 3º is ideal (the mechanical axis pass through the medial 1/3 of the lateral plateau)

- **COMPLICATIONS:**
  1. Undercorrection - most common
  2. Overcorrection
  3. Penetration of the articular surface
  4. AVN of tibial plateau
  5. Anterior compartment syndrome
  6. Peroneal nerve injury
  7. Patella baja
  8. TKR may be more difficult

**Lateral Compartment Disease**
- >12º ......................................femoral (as the LFC usually erodes → accentuating the valgus)
  - Distal femoral medial closing wedge
  - Distal femoral lateral opening wedge

- < 12º .................................HTO (if preop. plan shows no joint line obliquity will result)

**Other Osteotomies Around The Knee:**
- Osteotomies of the patellofemoral malalignment
- Osteotomies of Blount’s
Knee Arthrodesis

Indications:
1. FAILED KNEE REPLACEMENT (most common)
   1. better fusion rate following failed condylar components (80%) than failed hinged prostheses (55%)
2. uncontrollable SEPTIC ARTHRITIS with complete joint destruction
3. young patient with SEVERE ARTICULAR DAMAGE as in joint tumors
4. SEVERE LIGAMENTOUS INSTABILITY that can not be addressed by TKA
5. NEUROPATHIC JOINT disease

Contra-indications:
1. Contralateral knee disease
2. Contralateral leg amputation
3. Ipsilateral ankle or hip disease
4. Ipsilateral severe segmental bone loss

Techniques of Arthrodesis:

1- External Fixation
   o Ilizarov technique
   o minimal soft tissue dissection
   o allows for late adjustment
   o allows arthrodesis in the presence of active infection

2- Intramedullary Nailing Arthrodesis
   o most reliable for achieving fusion
   o 2 stage technique in active infection
   o can insert nail antegrade thro the piriform fossa or thro the knee joint
   o complication rates of 20-50%

3- Plate Fixation
   o 2 twelve hole plates

Complications:
1- Non-union- 20%
2- Malunion
3- Delayed union
Uni Compartmental Knee replacement

**Rational**
- Used to treat unicompartmental knee arthroses where it is better than the osteotomies as it offers less early complications and fast initial success

**Indications**
1. Unicompartmental knee arthroses
2. <60 y
3. degenerative arthritis
4. Range of motion at least 15-90° flexion

**Contraindications**
1. Bicompartmental arthroses
2. Crystalline deposition arthroses
3. ACL & Ligamentous instability
4. Subluxation > 1 cm
5. Large intercondylar erosion [sign of subluxation] convert of TKR
6. Marked coronal malalignment > 10° varus, > 15° valgus

**Technique**
- Midline, subvastus, or lateral approach
- Protect the ACL and anterior horn meniscus
- Keep moist the contralateral cartilage surface
- Distal femoral cut should be minimal
- Anterior cut should allow the anterior flange to be recessed below the level of the normal cart
- Tibial cut parallel to the femoral cut (8-10 cm)
- Soft tissue balance and gap measurement are done as in TKA

**Complications**
- Anterior impingement of the patella
- Pes Anserinus bursitis
- Arthritis of the contra lateral compartment
- Loosening, vascular, thromboembolic...

**Results**
- Controversial, most surgeons stick to the indications only
- Up to 90% success

<table>
<thead>
<tr>
<th>Arthrodesis</th>
<th>Osteotmy</th>
<th>Unicompartmental</th>
<th>TKR</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>Any</td>
<td>&lt;60</td>
<td>&gt;60</td>
</tr>
<tr>
<td>Patient</td>
<td>Active</td>
<td>Heavy active</td>
<td>Light sedentary</td>
</tr>
<tr>
<td>Arthritis</td>
<td>Severe</td>
<td>uni</td>
<td>uni</td>
</tr>
<tr>
<td>Pathology</td>
<td>Infective</td>
<td>degenerative</td>
<td>degenerative</td>
</tr>
<tr>
<td>Range</td>
<td>Any range</td>
<td>15° - 90°</td>
<td>15° - 90°</td>
</tr>
</tbody>
</table>
Primary Total Knee Arthroplasty

Rational
1. Pain relief
2. Restoration of normal limb alignment
3. Restoration of a functional range of motion

Indications
1. The most common indication for TKA is **Primary And Secondary Osteoarthritis**
2. The 2nd is **Rheumatoid Arthritis**
3. **Hemophilic Arthritis** and other types of arthropathy
4. **End-Stage Arthritis** that is nonresponsive to the usual nonoperative treatment
5. **Severe Intractable Pain**
6. Ideally indicated for old ages; although also done in young patient with severe pathology
7. **Failed Alignment Osteotomies**

Contradictions
Absolute
- **Active Infection** in the knee, ipsilateral extremity, or elsewhere in the body

Relative
1. **Inadequate bone stock** due to osteoporosis or other metabolic bone disease
2. **After arthrodesis** or scaring & muscle fibrosis that needs highly constrained knee
3. **Marked Recurvatum** in polio as the prosthesis is unable to address the recurvatum such a deformity that may lead to early loosening and failure
4. **Neuropathic Joint** or preclude motor control of the knee or lead to a Charcot knee
5. **Weak Extensor Mechanism**
6. Generally **Poor Health** or **Systemic Disease**

Classification

Three broad types of arthroplasty are used in uncomplicated primary TKR today: **Cruciate-Retaining, Cruciate-Substituting, And Mobile-Bearing Knees**. For more complex reconstructions in which instability due to ligament failure is present, nonlinked constrained arthroplasties have been used. (Linked constrained devices such as “hinges” are rarely used or even indicated.) All of these arthroplasties are descendants of the successful Total Condylar knee, the gold standard for resurfacing types of total knee replacement. In the best designed of each category, long-term results are excellent in terms of fixation, function, pain scores, and motion.
**PCL-Retaining Knees**

1. The theoretical basis for retention of PCL in TKA suggests that with a posterior tether between the femur and the tibia, **Stress Bypass** the TKA components.

2. **Proprioception** is maintained, and normal motion (femorotibial rollback) is preserved.

3. Preservation of **Quadriceps** power and

4. Preservation physiologic **Rollback** may theoretically result in a more normal gait in walking stairs. The demands of balancing the anatomic function of the PCL dictate that the normal joint line be closely restored, possibly further improving patellofemoral kinematics. In order to facilitate the femoral rollback, tibial polyethylene components were historically kept relatively flat in the anteroposterior (AP) direction, with a physiologic posterior slope. This avoided the “**Kinematic Conflict**” of a dished polyethylene, preventing normal rollback. Although some laxity is necessary to preserve range of motion, more anatomic posterior cruciate–sparing prostheses depend entirely on the soft tissues to resist posterior translation. Later iterations of cruciate-retaining implants have added more congruence to the articular polyethylene to improve stability and contact stresses.

5. Restoration of the proper posterior translation of the femoral component → flexion; this is reproduced by **High-Flexion Designs** in ω there is ↑ in the thickness of the posterior condyle → delay of the tibial impingement during flexion.

---

**Precautions:**

1. **Level of the Joint Line** must be near normal height → or may cause tight PCL.

2. Avoid **Tight PCL**. ω will produce:
   - i. Restricted flexion
   - ii. Excess femur on tibia rollback = **Booking**
   - iii. High posterior polyethylene-bearing load → Articular failure.

3. **Flat Tibial Insert** avoids that conflict.

4. The technical challenge with a PCL-retaining knee design is to realign the limb and restore full motion while keeping the PCL under proper tension.

- Normal physiologic tension is found in only 1/10 knees undergoing a PCL-retaining
- Well-functioning PCL-retaining TKAs may have nonphysiologic AP movement.
- Some cases demonstrated “**Roll Forward**” rather than “roll back”
**PCL-Substituting Knees**

- Sacrifice and substitution for the PCL gives predictable femorotibial kinematics and may facilitate balancing knees with greater angular deformity, especially when secondary changes in the PCL contribute to the deformity.
- The function of the PCL is partially replaced by:
  1. More **CONGRUENT** articular surface
  2. Tibial **SPINE**
  3. Femoral **CAM** mechanism
- Historically, the most popular designs had a higher rate of patellofemoral complications.
- Patellar pathology may be partially due to:
  1. Relative elevation of joint line
  2. Relative patella baja
  3. Impingement of fibrous tissue proximal to patella → **“PATELLAR CLUNK”**
  4. Boxy femoral profile & no recession of trochlear groove → ↑ patellar retinacular tension
- Recently, improved designs replicate patellofemoral anatomy and mechanics.

**Mobile-Bearing Knees**

- Potential design advantages:
  1. Highly congruent femoral and tibial articular surfaces → very low contact stresses
  2. ↓ PE wear
  3. Tibial articular surface “float” by gliding → ↓ kinetic conflict by highly congruent surfaces
- Fluoroscopic information suggests that *in vivo* kinematics may imperfectly replicate design goals
- In USA:
  1. 45%.................................. PCL substituting
  2. 45% ................................. PCL retaining
  3. 10% ................................. Mobile bearing

<table>
<thead>
<tr>
<th>Advantage</th>
<th>PCL retaining</th>
<th>PCL substituting</th>
<th>Mobile bearing</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Preserve anatomy</td>
<td>1. ▲ congruence</td>
<td>1. Ultraconforming</td>
<td></td>
</tr>
<tr>
<td>2. Preserve Roll Back</td>
<td>2. Fixed roll back</td>
<td>2. Floating tibial insert ▲ conflict</td>
<td></td>
</tr>
<tr>
<td>3. ▼ stress; dissipated to soft tissue</td>
<td>3. Less sensitive to JL changes</td>
<td>3. ▼ contact stress</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Disadvantage</th>
<th>PCL retaining</th>
<th>PCL substituting</th>
<th>Mobile bearing</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Less congruent</td>
<td>1. Patello-femoral clunk</td>
<td>1. Diff precise flex-ext gap</td>
<td></td>
</tr>
<tr>
<td>3. Late PCL instability</td>
<td>3. Loss of PCL proprioception</td>
<td>3.</td>
<td></td>
</tr>
</tbody>
</table>
Accurate limb alignment \( \Rightarrow \) stresses. The main goal of instrumentation is to provide accurate alignment.

- The mechanical axis = line that passes from hip center to ankle center & falls on the med side of knee center.
- Consistent reproduction of a mechanical axis landmarks are difficult intraoperatively.
- Anatomic axis landmarks, by contrast, are available, easy to instrument, and approximate mechanical axis.
- Precision is paramount because an asymmetry of only 5\% \( \Rightarrow \) Lift off of the femoral component on tibia, with compressive forces all on one side and tensile forces on the other.
- Subtle malalignment \( \Rightarrow \) by time as soft tissues attenuate under excessive forces.
- Fixation is reliably achieved with both cemented and cementless techniques, but PMMA remains the standard and generally the most widely used technique.

Planning of skin incision:

1. Medial Parapatellar Approach
2. The Subvastus Approach
3. Lateral Parapatellar Approach
4. Modified Lateral Capsular Approach
5. Mid-Vastus approach

**Kinetics** is the study of forces.

**Kinematics** is the study of motion.
**Surgical Procedure**

**Preoperative Evaluation**

7. **TAKE GOOD-Quality Radiographs** on large cassettes in both the AP and lateral planes. AP weight-bearing films demonstrate better any potentially dynamic instability. They also accentuate joint space narrowing. A Merchant view (or other similar radiograph) is useful to evaluate patellofemoral mechanics. A **Tunnel View** may be useful to evaluate for osteonecrosis.

8. Fixed **Angular Deformities** and **Contractures** need to be released at the time of surgery either through soft tissue or bony maneuvers.

9. **Ligamentous Instability**, particularly in the medial-lateral plane, may need to have specially stabilized components or ligamentous augmentation for a successful procedure.

10. **Vascular Disease** in LL, may be there is a contraindication for use of a tourniquet.

11. Assess **Skin** condition; **avoid flaps** and **undermining** of the skin, particularly laterally over the patella to avoid potentially devastating problems. In general, **old incisions** performed. Cross transverse incisions for patellectomy or high tibial osteotomy at 90°. More recent **parapatellar incisions** are problematic. In general, include recent parapatellar incisions in the line of the main incision if practical.

12. In the absence of long bone and hip deformities, **Standard 36-Inch Radiographic** films with the patient standing are adequate for preoperative planning.

13. **Template** for the size of prosthetic components on the radiographs with acetate overlays that are compensated for radiographic magnification. Preoperative templating is only an approximation of the required prosthetic sizing—never use templating to determine the precise size used at surgery.

---

**SURGICAL PREPARATION**

- **Position** the patient carefully in the supine position on the operating table.
- Protect the elbows with foam pads and gently flex them approximately 30°.
- Elevate the patient’s buttock ê soft roll (hard ones Þ sciatic nerve palsy)
- Contralateral leg often lies in external rotation; so pad the la
- Position & secure **TWO SEPARATE BUMPS** to the table as leg pc
- When the leg lie on top of both full extension
- When the foot lie on the 1st bump 30° flexion
- When the foot lie on the 2nd bump 100° flexion
- Rotate the table toward the opposite limb ê tendency to E
- Confirm that **Preop AB** have been given before tourniquet inflation; cephalosporin is an appropriate choice.
- **Tourniquet** should have been placed on the thigh as high possible and the skin protected with soft webril padding.
- Finally, take a “**Walk Around**” the patient just before scrub: Check all peripheral nerve sites for appropriate padding.
- **Shave** the skin around the knee immediately before op to compromise of the skin surface. Extended time between shaving and surgery promotes colonization of small nicks with bacteria.
- Reaffirm that the **Correct Limb** is being prepared. Although it is rare, wrong limb side surgery does occur and is disastrous for patient and surgeon. AAOS recommended confirmation of the side to minimize this complication
- Avoid **Excessively Bulky Drapes** as precise alignment in total knee replacement depends on the ability to palpate the bony landmarks
Operative Technique

- Flex the knee to a 30° position and mark the surgical incision. Flexion produces tension on the anterior tissues, which allows a more precise surgical exposure.
- A midline incision is preferred, extending ~ five fingerbreadths above & below the patella.
- Carry the incision through the midpoint of the knee and distally just medial to the tibial tubercle.
- Then carry the incision through the deep fat and the prepatellar bursa. Preserve the epitenon for later closure.
- Perform a medial arthrotomy with a new knife blade. The location of the quadriceps tendon can be very deceiving proximally, as it may be laterally if only the visual landmarks are used, and this may lead to transection of the bulk of the quadriceps. Often, feel the tendon.
- Use a #10 blade perpendicular to the tendon, longitudinally in a gentle curve, which curve around the patella.
- In the inferior region of the arthrotomy, split the fat pad with the medial bulk of fat maintained. This facilitates the lateral exposure, and leave a fair tissues for closure.
- When the incision comes to the tibial tubercle, bring the incision parallel to the shaft of the tibia and carry it distally for a few more millimeters.
- Place a right angle retractor medially and transect the anterior horn of the medial meniscus, with the medial soft tissue from proximal to distal, staying strictly at the subperiosteal level to maintain the vascularity of the medial flap of tissue.
- If severe varus knee, the medial exposure can be extended into a medial release.
- Once the deep portion of MCL has been elevated, place the Z-type retractor between the MCL and bone to gain medial exposure.
- Remove large medial osteophytes at this point as part of the medial release.
- Flex the knee to 90° or 100°. The potential for patella tendon avulsion exists in the tight knee. As the knee is flexed
- Watch the insertion of the patella tendon to avoid any excessive flexion that may avulse the patella tendon. If the patella cannot be easily everted without the risk of patella tendon avulsion, obtain more extensile exposure either by:
  i. Extending the arthrotomy proximally
  ii. Doing more distal soft-tissue release
  iii. Quadriceps release
  iv. Tibial tubercle osteotomy: this is mostly preferred
- This situation most often occurs in:
  i. Very obese patient
  ii. Patient with prior knee surgery, e.g. HTO → patella tendon is scarred and tight
- Once patella tendon avulsion occurs, it is very difficult to treat, so this complication is best avoided.
- Flex the knee 100°, patella is everted; so femoral condyles, and tibial plateau should be at least partially visualized.
- ACL is resected to enhance exposure.
- Using a 1-inch osteotome, remove medial and lateral osteophytes to expose the PCL attachment.
- If the PCL is being intentionally sacrificed, it is best done at this point. The PCL should not be sacrificed in a cavalier fashion, but it should be done precisely by electrocautery subperiosteally off
- Release the lateral periphery of the meniscus with a #15 blade in a precise curvilinear fashion along to its insertion next to the PCL.
- Branches of the lateral geniculate artery are just outside the insertion of the lateral meniscus and should be cauterized at this time.
- Once the lateral meniscus is removed, place a Z-type retractor in the corner overlying the lateral tibial condyle to gain extensile exposure of the flexed knee.
Principles of Balancing:
- At least four directions for spatial relations must be carefully maintained:
  1. **Coronal Angulation**
  2. **Rotation**
  3. **Sagittal Angulation**
  4. **Joint Level**
- In tibia use IM or extramedullary alignment guides, but in femur only use IM guides

1. **Angulation:**
- Femoral medullary hole ≈ 9 mm, just med to notch center & above PCL insertion → best alignment
- Avoid midline drill hole in femur → valgus angulation, as it is not aligned with medullary canal.
- Put your opposite hand on the femoral shaft to provide appropriate directional control
- Once the hole is drilled, aspirate BM to prevent embolization.
- Distal cut is made in a preset angle from mechanical axis = 5°-6° valgus
- Tibial cut is made ⊥ to its long axis

2. **Rotation Of The Femoral Component**
- Patello-femoral mechanics are altered dramatically by femoral rotation.
- Excessively IR of femoral component → patellar subluxation
- Some ER of femur is desirable to achieve equal flexion gap, as tibial cut is done ⊥ to tibial long axis, not in I normal 3° Medial Inclination
- ER of femoral cut is necessary to make flexion gap equal, achieved by:
  1. **Whiteside's Line** and draw the transverse axis ⊥ on it
  2. **Transsepicondylar Axis** gives 3-5° ER to post condylar axis
  3. **Posterior Condylar Axis** in cut is made 3° ER to that line
  4. Parallel to the tibial cut (Gap Technique)
- No one landmark is always reliable. Use all three in combination;
  o Epicondylar Axis .................. is the most reliable, but difficult to palpate
  o Post condylar axis ............ not for valgus knees or RA
  o Transepicondylar ............................. not for lat condylar hypoplasia

**Femoral cut depth and rotation techniques:**

1. **Gap Technique**
- Begins ⊥ to tibial axis 5-10 mm below the articular surface of the more normal side, then check ligament balance.
- Measure the **Whiteside's Line** (AP axis) is the line between the deepest part of the patellar groove anteriorly & the center of the intercondylar notch posteriorly; this gives you an idea about the femoral size
- Posterior femoral cut is made // to tibial cut
- Disadvantages:
  o Midrange laxity (only 0° - 90° are checked)
  o Possible elevation of JL (in flexion contracture + flexion gap cut → undersized femoral)

2. **Measured Resection Technique**
- Replace resected bone from femur & tibia = thickness of implant
- **Joint Line Technique**, as the joint lines are fairly well maintained and the normal anatomy is essentially resurfaced.
- Femur is usually resected first
- It is very important in PCL retaining; as slight elevation of the joint line in PCL-retaining designs can have adverse effects on knee kinematics
- Advantages:
  o Close matching of the AP dimension of femur
  o Preserve femoral rollback without the “booking” phenomenon
  o Avoids post stresses
  o Avoids limited flexion associated with tight PCL
  o Avoids excessive translation with loose PCL
  o Whiteside line ..................... not for big anterior osteophytes
Preparation of Femoral Cut

- The distal level of the femoral implantation is critical to preserve the proper level of JL.
- The flexion and extension gap must be equal.
- The coronal alignment and proper ER are critical.

1). Anterior and Posterior Sizing
- After the distal femoral cut is made and the ER has been determined, make the anterior and posterior femoral cuts. In general, there are two references:
  1). **ANTERIOR REFERENCING** technique, where the anterior cut is made first by referencing from the anterior cortex. This approach ensures no femoral notching.
  2). **CALIPER TECHNIQUE** (Jig system technique, mostly used): measure the distance between post-condyles & ant cortex. If size is between 2 sizes, take the smaller one to avoid overstuffing.
- **OVERSTUFFING** is a phenomenon that occurs when too large femoral component is placed. If the component is placed posterior → tight flexion gap & flexion. If an oversized component is placed too far anterior → extensor mechanism impinge on the femoral flange → tight knee.
- In the so-called "GREYHOUND KNEE," named for its resemblance to canine anatomy, there is large AP diameter & narrow medial-lateral diameter → medio-lateral over hanging femoral component. Proper implant system selection → this phenomena.
- Restoration of the proper posterior translation of the femoral component → flexion; this is reproduced by **High-Flexion Designs** in → there is in the thickness of the posterior condyle → delay of the tibial impingement during flexion.
- Lastly, check the bone cuts for:
  1). Appropriate rotations.
  2). **MATTERHORN SIGN** (after a mountain) = longer lateral condylar ant cut than the medial one.
  3). Posterior cuts should show asymmetry, e more > lateral bone in most cases.

2). Removing the Posterior Osteophytes
- Remove posterior osteophytes after the chamfer and other finishing cuts. The posterior osteophytes do not act as a mechanical block to limit flexion only, but also tent the posterior capsule and limit full extension.
- With the knee held in extreme flexion, retract the femur superiorly with a blunt bone hook placed in the notch. Use a 1-inch sharp, curved osteotome to resect posterior osteophytes.
- Remove adherent capsular attachments by either curettage or rongeur. A soft sulcus should result behind the posterior condyles.
- This approach will maximize the potential extensio.

**Check for pitfalls:**
1). Oversized femoral component
2). Improper ER
3). Notching of the femur
4). Improper axial alignment
5). Flexion & extension gaps: this can be a guide to the amount of tibial cut needed
Tibial Resection

- Tibia is resected ⊥ to its long axis. The anatomic axis is in 3° varus.
- Implant systems initially attempted to replicate this slight varus profile 2° to 3° varus that produced 5-6° varus cut → early component loosening.
- Therefore, attempt a ⊥ cut with the knowledge that a slight varus cut 2-3° should not alter clinical results. We prefer extramedullary technique.

Multidirectional orientation of tibial cut:

1. **ROTATION**
   - This is best determined by sighting down “bird’s-eye view” relying on:
     a. Tubercle line (most reliable): keep the profile centred on the medial third of the tubercle
     b. Posterior condylar axis
     c. Transcondylar line

2. **CORONAL ALIGNMENT**
   - Secure the yoke of the tibia cutting guide at 2/3 of the way down the tibia above the ankle; not at the ankle joint, as the fibula widens it → produce a lateral offset → varus angulationion.
   - Place the proximal jig of the tibia cutting guide right over the center of the tibial tubercle. The bulk of the patellar tendon may bias the cutting jig medially → varus angulation, so a smaller footprint cutting block is better
   - Distally, place the guide // to the chin of the tibia / its tip over TA tendon / pointing to 2nd ray
   - Always, place the distal tip of the guide medially (over the anterior tibialis tendon) → † varus risk

3. **HEIGHT**
   - Variety of height guides are available:
     1. Curved wing-shaped device
     2. Measuring Jigs that measure from the cutting surface to fixed points above the tibial surface
   - In general, it is preferred to take 10 MM OFF THE “NORMAL” SIDE → slight subchondral resection on the “abnormal” side.

4. **POSTERIOR-TIBIAL TILT**
   - AP Caudal downslope of the tibia ≈ 5º (most systems have it built in i.e. use a neutral cut)
   - Avoid putting the tibia in extension → tight flexion gap & † motion

5. **TIBIAL COMPONENT SIZE**
   - The largest size is used to † force/area unit
   - Avoiding overhang of the tibial plateau, particularly posterolaterally.

Resection

- Resect the proximal tibia, protecting the medial collateral ligament on the inside of the knee, the patellar tendon on the outside of the knee, and the posterior vascular structures.
- Then lift off the plateau one piece after releasing it from soft-tissue

Saving the PCL

- Make a slot ant to PCL by a saw, then place a rectangular osteotome into that slot.
- Start to do tibial cut ê that osteotome in place

When to sacrifice PCL

1. Bowstring tension of PCL
2. Incomplete flexion or forced flexion
3. Excess rollback at >90º flexion
4. Anterior lift off ð tight flexion gap
5. Femoral sublux ð tight flexion gap

Check for pitfalls

1. Varus alignment caused by:
   o Patellar tendon bias
   o Medial ankle offset
2. Mal-rotation
3. Over hang tray
4. Elevation of joint line
5. Reversed tilt → tight flexion
Balance of the Knee
- Perform rigorous medial and lateral ligament balancing, and flexion and extension gap balancing using appropriately sized blocks.
- If femoral preparation is completed, trial components are inserted & lig balance checked
- In general, balance the knee with soft-tissue releases rather than by bony cuts.

1. Ligament Balancing
- Lig balancing can be straightforward or complex
- Plan to have more constrained implants available if the deformity is severe e.g. > 15° of varus or valgus, but remember that constrained knee implants have poor longevity
- There are 7 points of lig balancing that must be achieved
- At this point, balancing may require conversion from PCL retaining → PCL–substituting, as PCL may contribute to the deformity esp if severe

Seven points of ligamentous balance
1. Achieve full extension of the knee
2. Achieve full flexion & out excessive rollback of femur
3. Achieve good medio-lateral balance at full extension
4. Achieve good medio-lateral balance at full flexion
5. Track the patella over a full arc of motion & no thumb
6. Good rotational balance in full extension
7. Good varus-valgus must be correct

2. Varus Deformity
- Is the commonest & usually the simplest deformity to correct by cutting the tibial \( \perp \) to the axis.

Steps To Correct Varus Deformity:
1. Remove medial OSTEOPHYES on the femur and tibia
2. Release the MEDIAL CAPSULE with care to MCL
3. Release DEEP MCL subperiosteally
4. Release SUPERFICIAL MCL subperiosteally (é break SHARPEY frs.)
5. Release PES ANSERINA as a secondary constraint medially.
6. Release the POSTERIOR CAPSULE and PCL
7. Release MEDIAL GASTROC
- However, commonly there is still medial tightness in extension, after appropriate cuts.

3. The Valgus Knee
- Technically is more difficult to realign
- The tightest structure is usually the ITB with the knee in full extension, Release this from inside out \( \perp \) to ITB long axis at the joint line.

Steps To Correct Valgus Deformity:
1. Release ITB at the joint line ............... *
2. Release popliteus tendon ................. ♠
3. Femoral attachment of the LCL .......... ♠
4. Release fibular attachment of LCL ......... ♠
5. Release PLC .................................................. ♠
6. PCL Is Resected Routinely
7. Biceps tendon is transected from fibular insertion
8. Consider other implant system
- Combined deformity; valgus flexion are the most challenging
- Valgus + flexion deformity = tight in extension
- Bony lateral release (lateral femoral sliding osteotomy) & fibular head decompression could be done as an alternative to LCL release
2]. Coronal balance

<table>
<thead>
<tr>
<th>VARUS DEFORMITY (medial release)</th>
<th>VALGUS DEFORMITY (lateral release)</th>
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<tbody>
<tr>
<td>1]. Osteophytes</td>
<td>1]. Osteophytes</td>
</tr>
<tr>
<td>2]. MCL deep (coronary) then superficial</td>
<td>2]. Lateral Capsule</td>
</tr>
<tr>
<td>3]. Postero-medial corner + semi-membranosus</td>
<td>3]. ITB If Tight In Extension</td>
</tr>
<tr>
<td>4]. Pes Anserinus</td>
<td>4]. Popliteus If Tight In Flexion</td>
</tr>
<tr>
<td>5]. PCL in rare cases</td>
<td>5]. LCL</td>
</tr>
</tbody>
</table>

Patella (follows femoral & tibial components, & ligament balancing)
- Indications:
  i. Inflammatory arthritis
  ii. Severe OA
  iii. Patellar deformity & insufficiency
  iv. Some do it routinely
- PF articulation is the source of the MAJORITY OF COMPLICATIONS
  i. Boxier femoral components
  ii. Inadequate patellar tracking
- PATELLAR CLUNK results from fibrous quads tendon that catches on the ant aspect of the femoral component. Offending fibrous tissue may be resected arthroscopically.

Patellar maltracking etiology and prevention

<table>
<thead>
<tr>
<th>ISSUE</th>
<th>GUIDE LINE</th>
<th>PROBLEM</th>
<th>SOLUTION</th>
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<tbody>
<tr>
<td>1]. Femoral rotation</td>
<td>Never IR femur beyond neutral axis</td>
<td>Lateral patellar tilt &amp; lat sublux</td>
<td>Slight ER of femoral component</td>
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<tr>
<td>2]. Femoral position</td>
<td>Never medialize femur</td>
<td></td>
<td></td>
</tr>
<tr>
<td>3]. Tibial rotation</td>
<td>Never IR tibia past med side of tibial tubercle (TT)</td>
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<tr>
<td>4]. Leg alignment</td>
<td>Never leave excess valgus</td>
<td></td>
<td></td>
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<tr>
<td>5]. Patella position</td>
<td>Avoid lateralization</td>
<td></td>
<td></td>
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<tr>
<td>6]. JL position</td>
<td>Never raise joint line level</td>
<td>Patella baja</td>
<td></td>
</tr>
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</table>

**Patellar Clunk:**
A. Result from ingestion of fibrous tissue bet patellar dome and the ant femoral condyle
BE: This causes pain during extension as the issue is cranked over the condyle.
- Reflect the fat pad & remove all osteophytes til you see patellar margin all around é out compromising quadriceps tendons.
- Cut the patella flat to give symmetric cut & drill til you see patellar margin all around.
- Most components are pegged.
- Check PF tracking by "No Thumb" or touch-free tech.
- Tracking & lateral retinacular tension is controlled:
  i. Subvastal approach is better for tracking
  ii. Appropriate ER of femoral & tibial components
  iii. Appropriate alignment
  iv. Lateralization of femoral and tibial components → retainacular tension & improve tracking
  v. Never lateralize the patella ⇒ usually lateralize every thing except patella
  vi. If still there is mal tracking ......................... do lateral retinacular release (5%)
  vii. Distal realignment procedures or medial capsulorrhaphy, μθs needed in extreme cases
- Pitfalls:
  i. Under resection .......................................... overstuffed PF joint
  ii. Too much resection .................................. patellar fracture
  iii. Lateralized patella ...................................... maltracking
  iv. Asymmetrical cut .......................................... patellar tilt

Cementation
- Remove the trial components → lavage the bone surfaces é normal saline → Clear the debris
- Drill sclerotic bone small shallow holes, to ⋁ cement interdigitation (avoid many holes in 1 line)
- Mix the cement → pressurize some cement into the plateau 1st → add another 1-mm cement layer on plateau → gives adequate cement mantle in spite of bone irregularity
- Apply the tray and impact it. Vigorous impaction improve pressurization, but may #
- Remove excess cement, particularly posterior to the tibial tray.
- 2nd apply a donut-shaped piece of cement to femur & pressurize it into a uniform thickness
- Place a small amount of cement on the posterior rails of the femoral component
- Impacat the femoral component neither in flexion nor in extension.
- Always remove excess cement before and after every component cementation
- Place a trial insert and extend the knee fully till the cement sets.
- Patellar cementation is done é a cut insulin syringe used to pressurize the cement into holes
- Apply the patellar component onto the three holes & pressurize é the patella clamp.
- After the cement has hardened, verify the seven points of ligament balancing

Wound Closure
- Irrigate all of the soft tissue, and prosthetic and bony surfaces of the knee joint vigorously.
- Now close extensor mechanism watertight, with monofilament resorbable suture.
- Distal seal is difficult but imp (that’s why distal incision incorporating the patellar tendon is VIP)
- Close the deep fat layer, peritenon, and the skin with staples → dress the wound.

Tibial Tubercle Osteotomy
- Indications:
  i. Difficult exposure  iv. Prior surgery
  ii. Severe angular deformity  v. Obese patients
  iii. Bone deficiency  vi. Associated patellar disorders
- Place the oscillating saw // to the tibia and make a 1cm thick, 8cm long cut from medial to lat
- Keep the lateral periosteal hinge intact laterally.
- Make a 2nd cut // to JL and 2 cm distal to it, at a 45° angle to meet the initial osteotomy site.

POSTOPERATIVE CARE
- Begin early motion according to your preferences and the clinical situation
- Early motion can improve recovery and shorten the hospital stay.
- CPM is useful for obtaining early motion (achieve extension early and advance CPM by 10°/d)
- WB allowed on the first postoperative day
- Discharge from the hospital at 90° in an immobilizer until sufficient quadriceps control
PITFALLS AND COMPLICATIONS
Long-lasting complications of TKA are rare, and long-term success is generally the rule.

NONMECHANICAL COMPLICATIONS
The most common nonmechanical complications of TKA are thromboembolism and anemia.

1- THROMBOEMBOLISM
- 50% of pts mainly at or below knee; 10% are proximal to the knee; < 2% pulmonary
- Pulmonary emboli may be fatal esp if large
- Dx:
  - Contrast venography: gold standard, sensitive, specific, nephrotoxic
  - B-mode duplex: not for iliac or deep vv, technician dependant
  - Other: clinical (Homann, Hoffman,...), $^{131}$I labeled fibrinogen scan, plethysmography
  - No test yet proven to be effective as a postop screening test to pick risky pts
- Prophylaxis:
  - For those reasons; most surgeons favor chemical, mechanical, or both
  - Mechanical: all may prevent leg DVT but not the serious higher embolism
    1- elastic calf
    2- thigh hose
    3- sequential intermittent pneumatic compression stockings
    4- Plantar foot compression pumps
    5- Early mobilization
    6- CPM
    7- Inferior vena caval filters; but invasive, expensive, hazardous contrast
- Chemical:
  1- Continuous IV heparin
  2- LMW heparin sc
  3- Oral warfarin
  4- IV dextran
  5- Aspirin
  6- All predispose to bleeding, hematomas, hemorrhathrosis, GI & CNS bleeding
- Treatment:
  - Immediate IV heparin infusion (keep PTT 1.5-2 times the control)
  - Discontinue heparin and replace by warfarin (the same level) till 6wk-6mo
  - Discontinue afterward
  - Continuously monitor the patient for bleeding (40%)

2- NEUROLOGIC INJURY after knee arthroplasty is
- most common in valgus deformity ± flexion deformity of severe rheumatoid arthritis
- Mainly peroneal nerve (1/3 complete, and 2/3 incomplete)
- Due to:
  - Traction upon the nerve during surgery after realignment of the deformity
  - Compression by a tourniquet
  - Compression by a tight bandage or orthosis
- Prognosis:
  - ½ recover and ½ would have partial recovery
- Treatment:
  - Prophylactic decompression is not recommended
  - Remove all the tight bandages
  - Flex the knee
  - Conservative ttt + rehabilitation
  - AFO
  - Surgery after several months of no improvement → decompression
3- **Vascular Injury:**
   - Most common vessels to be injured:
     - Superficial femoral
     - Popliteal
     - Genicular
   - Type of injuries:
     - Direct lacerations
     - Late AV fistulae
     - Aneurysm
     - Thrombosis: esp in predisposed pt é peripheral vascular disease
   - Dx: diminished pulses (esp é lacerations)
   - Treatment:
     - Prophylaxis: consultation of vascular surgery – non use of tourniquet in risky pt
     - TTT:

4- **Fat Embolism Syndrome (FES)**
   - Mainly occur with trauma; but also é joint replacements (3% of TKA)
   - Pathogenesis:
     - Incompletely understood
     - Introduction of BM elements into the systemic circulation
     - Eventually deposited in the pulmonary vv → liberates inflammatory mediators
     - Mediators cause compromised gas exchange and respiratory failure
   - PDF:
     1. Intramedullary drills
     2. Guide rod system
     3. Stemmed prosthesis
     4. Sudden release of the tourniquet
   - Clinically: (usually mistaken as narcotic sequelae, or senile sundowning)
     - Respiratory insufficiency
     - Mental confusion and agitation
     - Petechae (trunk, axilla, conjunctiva)
     - Fever & tachycardia
     - Retinal changes
   - Prophylaxis:
     1. Use of fluted IM guides
     2. Lavage the IM marrow fat
     3. Over drilling of the guide holes
     4. Slow advancement of the IM guides and stems
   - Treatment: (mainly supportive)
     - Mechanical ventilation
     - IV fluids & glucose
     - Heparin, Aspirin, Dextran, Ethanol (proven to have no effect)
     - Steroids may be effective if given early
5- **Wound Problems**:
- More in obese, DM, Poor nutritional status, RA, Multiple scars
- ✪ hematoma formation, clumsy technique, inadequate tight closure of all layers
- No relation to tourniquet deflation time or to CPM usage had been attributed

1- **Prolonged Drainage**:
- Expected for several days. After 7-10 days considered prolonged
- ✪ incomplete closure, poor superficial healing
- the problem is that it promotes bacterial contamination of sc tissues
- ttt: immobilization till closure → explore → irrigation → close in layers

2- **Superficial necrosis**:
- Uncommon
- ttt: do not overlook and do gastroc rotational flap

3- **Wound dehiscence**:
- Rare
- Usually 2ry to trauma, obesity, patellar instability, premature stitch removal
- ttt: as a surgical emergency, copious lavage, Broad AB, closure & 2ry sutures

### Mechanical Complications
- The majority of the mechanical complications of TKR can ✪ careful attention to proper soft-tissue balance in all planes. Patients ✪ more challenging preop deformity or motion restriction should be counseled about possible postoperative complications

#### 1- Aseptic Loosening
- Typically is different from aseptic loosening of the total hip arthroplasty. Polyethylene debris-induced, macrophage-initiated osteolysis is considerably more rare. This problem is thought to be due to the different wear environment of the TKA. Threefold larger wear particles are produced in a shearing, less-conforming environment. These larger particles are less likely to induce a macrophage response and lead to a lower rate of aseptic loosening. Malalignment, especially of the tibial component in varus, can lead to mechanical subsidence of tibial component & loosening.

#### 2- Infected TKA:
- Costly complication that occurs in approximately 1% of all knees

**PDF:**
- **Exogenous:** In the surgical technique
- **Endogenous:** In the patient e.g. DM, poor nutritional status, steroid, immunocompromized

**Bacteriology**
- **Low** virulent organisms:
  - Staph epidermidis (methicillin sensitive, non glycocalyx forming)
  - Staph aureus (methicillin sensitive, non glycocalyx forming)
  - β hemolytic strept
  - Anerobic strept

- **High** virulent organisms:
  - Gram negative organisms E. coli, Pseudomonas, Proteus, & Entero-cocci
  - Staph epidermidis (methicillin resistant, glycocalyx forming)
  - Staph aureus (methicillin resistant, glycocalyx forming)

**Others:** Corynlobacterium, Group B strep, Strep faecalis

**Sources**
- **Intra-operative**:
  - Sampling of theatre air identified the same organisms & same proportions
  - Contamination with the surgeons gloves, gowns, light handles, skin blades and needles mainly affected. 76% of the organisms grown were coagulase-negative staphylococcus.

- **Post-operative**:
  - Drains left for 48 hours allow ingrowth of organisms (Willett et al. JBJS 1988)
  - Haematogenous spread: Most common source is skin(46%), mouth(15%), UTI(13%)
Organism Virulence

1. **ORGANISM RESISTANCE TO AB:**
   - [i]. Altered target: altered DNA gyrase (Quinolones)
   - [ii]. Detoxifying enzymes: β lactamase (penicillins)
   - [iii]. ♦ uptake: membrane transport (erythromycin & tetracyclin)

2. **RACE FOR SURFACE THEORY**
   - When a total joint prosthesis is placed into the human body, the body's cells hurry to get hold on the surface of the prosthesis.
   - Once the body cells colonize the surface of the total joint prosthesis they can protect this non living surface against the bacterial colonization.
   - If any bacteria (usually skin bacteria) are present in the operation wound, they hurry to get hold on the prosthesis surface too, racing the body cell to colonize the surface.
   - If bacteria win, they evolve the capability to adhere to surfaces for their survival, by secretion of a glyco-protein cell wall called **GLYCOCALYX:**
     - i. Very strong adhesive
     - ii. Mask the bacterial antigens
     - iii. Colonize inside this biofilm away from immune system
     - iv. Invite other types of bacteriae to trick the immune system
     - v. When they adhere to the inert implant surface, bacteria are protected by the antiphagocytic effect of biomaterial.
   - All these factors will create a very powerful resistance 100 to 1000 times against AB & immune system.

Classification

1. Stage 1= immediate postoperative:
   - o Staph-aureus /β hemolytic strept
   - o Usually needs irrigation, unless glycocalyx forming organism is identified
2. Stage 2 = 6-24 mo
   - o Methicillin resistant staph epidermidis
   - o Usually needs staged operation
3. Stage 3: after 2y
   - o Hematogenous spread from other septic focus

New Classification

1. Type I | Early < 6wk
2. Type II | Late > 6wk
3. Type III | Hematogenous
4. Type IV | Incidental +ve culture
PREVENTION

Pre-operative:
1. All septic lesions must be identified & treated (feet, dental, urinary, prostate)
2. Admit patient as late as possible & into a clean ward
3. Shave in the anaesthetic room

Intra-operative:
[1]. Surgical Technique:
   1. Careful tissue handling & Haemostasis
   2. Length of procedure
   3. Wound lavage - mechanical effect more important than type of fluid used.
[2]. Antibiotic prophylaxis
   - Systemic antibiotics: methicillin-realted penicillin or cephalosporin
   - Antibiotic Loaded Cement: reduces risk of infection by 11 fold
[3]. The team: keep the operating team as small as possible
[4]. Movement: shed up to 10 000 skin scales/min
[5]. Theatre gowns.
   - Standard Gowns are permeable to organisms when wet “moist bacterial strike through”
   - Weaved Gortex & disposable non-woven ‘paper’ gowns are preferred
   - Body Exhaust Suits (Charnley): maintain –ve pressure, cool surrounding (shedding)
[6]. Footwear: no evidence that outdoor shoes are a source of infection; but overshoes wearing
   can result in the contamination of the hands of the clinicians.
[7]. Headgear.
[8]. Face masks.
[9]. Gloves and hands: scrub in a separate area, wear 2 gloves, change the outer after prepping.
[10]. Sterile drapes
[11]. Drainage of wound has risk of becoming infected than closed wound (only when sure)
[12]. Transporting the patient to operating theatre on a separate clean trolleys in the induction zone
[13]. Theatre equipment & instruments
[14]. Ventilation System: Vertical Laminalr Air Flow is recommended for arthroplasties
[15]. Ultra-violet Light: bacteriocidal; but not commonly used, potential dangers to staff

Post-operative:
   - In the early post-op phase - antibiotic cover for urethral catheterisation (Gentamycin IM)
   - AB cover for all GI & UTI & skin & Dental lesions è antistaphylococcal agent

DIAGNOSIS

   - You must suspect infection all the time & use every tool to reach the final diagnosis

1- Clinically
   - History of pain, swelling, erythema, fever problems post-op

2- Radiology:
   - Need serial radiographs (get original post-op films)
   - Osteolysis around the components
   - Periosteal reaction

3- Blood: WBC, ESR, CRP usually not helpful (usually –ve or irrelevant) except if strongly suggestive

4- Bones scans (incl. white cell scan):
   - usually unable to differentiate septic from aseptic loosening
   - Indium111 may be helpful

5- Aspirates:
   - CELL COUNT .........................>25,000/MM3 (May be helpful 85%)
   - DIFFERENTIAL COUNT...............>75% PNL
   - Gram stain..................................+ve 25%
   - Glucose........................................v
   - Protein.........................................v
   - PCR ............................................Newly introduced to diagnostic tools
   - IL6 serum level ..............................Newly introduced to diagnostic tools

6- Open biopsy:
   - Often identifies organism & sensitivities.
TREATMENT

1- **Antibiotic suppression**
   - Antibiotics alone will not eradicate deep sepsis so it is rarely indicated alone
   - Considered in:
     - Elderly, unfit for surgery.
     - Early infection
     - Single organism
     - Low virulent
     - Gram +ve organism

2- **Debridement alone:**
   - 20% success rate in TKR.
   - Considered in the same conditions but can combat also virulent organisms

3- **Resection arthroplasty:**
   - for the unfit or unwilling to have revision surgery & severe RA
   - not good for knee

4- **Arthrodesis:**
   - Remain a last resort especially for young active patients

5- **Amputation:**
   - for knee - High functional cost - end of line.

6- **Revision Arthroplasty:**
   - 75% success is achievable
   - Pre-requisites:
     - Healthy individuals who can stand long treatment
     - Organism sensitive to AB
     - Adequate bone stalk

<table>
<thead>
<tr>
<th>One Stage</th>
<th>Two Stage</th>
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<td><strong>Advantages:</strong></td>
<td>probably safer &amp; more successful</td>
</tr>
<tr>
<td>1. single operation</td>
<td></td>
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<td>2. quicker return to normal function</td>
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<tr>
<td><strong>Disadvantages:</strong></td>
<td>1. two procedures</td>
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<tr>
<td>1. Prolonged procedure</td>
<td>2. prolonged bed rest betw. stages</td>
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<tr>
<td>2. AB sensitivity must be known pre-op</td>
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<tr>
<td>3. Can’t use cementless prosthesis (lose benefit of antibiotic in cement)</td>
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<tr>
<td><strong>Contraindicated:</strong></td>
<td></td>
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<tr>
<td>1. If AB sensitivities not known pre-op</td>
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<tr>
<td>2. Mixed infection</td>
<td></td>
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<td>3. Gram –ve organism</td>
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<td>4. bone loss requiring allograft</td>
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<td>5. Doubt about debridement adequacy</td>
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<tr>
<td>6. Failure of previous one stage revision</td>
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</tbody>
</table>

Two stage Revision:
- **First stage:**
  - Debridement (Capsule, prosthesis, inf tissues, Cement, & cement restrictor) & lavage
  - Tissues must be sent for microbiology (even if organism is diagnosed pre-op)
  - Insert AB loaded cement beads or a ab cement spacer
- **Between stages:**
  - Continue iv antibiotics
  - Traction if beads used (or ext fix)
- **Second stage:**
  - Timing:
    - Early at 3wk
    - Late: after 6 wk, more effective (the wound would have healed & -ve CRP); but more tissue scarring that’s why many surgeons use AB spacers (AB-PMMA)
  - Continue antibiotics for 3 months.
3- **Periprosthetic Fracture**
- Is the fracture that occurs within 15 cm of the prosthesis or adjacent to prosthesis stem
- 1%, more in femur > in tibia, more in Revision & osteoporosis
- **Etiology:**
  - Traumatic
  - More in poor bone quality
  - Intraop during impaction, during trailing
  - Post op of anterior femoral notching → fatigue fracture
- **Treatment:**
  - Conservative as long as the alignment is preserved
  - Operative ORIF with plate and screws or IMN

4- **Tibial Fracture** is associated with malalignment or component loosening. The fracture may be bypassed with a stemmed prosthesis.

5- **Patellar Fracture:**
- Well known complication that may occur with or without resurfacing of the patella
- **Etiology:**
  - Direct trauma
  - Under resection (marked stresses over the patella)
  - Over resection (weak patella)
  - Patellar pegs create stress risers (some use multiple small rather than one large peg)

6- **Prosthetic Failure**
- In contemporary arthroplasties is limited to delamination or fracture of tibial polyethylene components or failure of metal-backed patellae. In the compromised environment with either mechanical imbalance or secondary to polyethylene failure, a tibial metal backing may rarely crack. This type of failure may often lead to particulate-induced synovitis & mechanical symptoms.

7- **Patellar Instability:**
- **Etiology:**
  - Excessive internal rotation of the tibial or femoral components
  - Over sized or overhang patellar component
  - Lateral placement of the patellar component
  - Avoid thick patellar component → tight patellar tracking
  - More to occur with valgus deformities with tight lat retinaculum ± medial reefing of the VMO
- **Treatment:**
  - Revision arthroplasty
  - If sure about the rotation of the major components → proximal of distal realignment
  - are the most common reason for revision knee arthroplasty. These complications were addressed in the technique section.

8- **AVN Patella**
- May predispose to patellar fracture
- **Etiology:**
  - ð sacrifice of the supero medial genicularte and inferior medial geniculate w in medial parapatellar approach

9- **Patellar Loosening**
- Uncommon event
- More é patellar maltracking
- Require revision of the patellar component
- If no bone stalk; retain unsurfaced patella or do the hated patellectomy
10-**Premature Failure É Metal Backed patella**
- Previously used to improve the stress transfer from the implant of the bone and consequent decrease in deformation and shear rate on PE. More with patellar maltracking
- Failure modes:
  - PE wear
  - PE metal dissociation
  - Peg failure
- Dx:
  - Anterior knee pain and swelling
  - PXR: Ficat views metal to metal articulation / AP metal debris
  - Aspiration: dark + metallic debris
- Treatment:
  - Patellar revision ± total synovectomy
  - Some times tricompartmental TKA revision

11-**Disruption of the extensor mechanism**: (rare but disastrous)
- Ätiology:
  - After difficult exposures and over release of the tendon
  - Tibial tuberosity osteotomy
  - After revision TKA
  - É limited ROM
  - Poor bone stalk
- Prophylaxis:
  1- Thorough lateral gutter debridement
  2- Cut Patellofemoral ligament
  3- Lateral retinacular release
  4- Quad snip
  5- Quad V-Y turn down
  6- Distal tibial osteotomy
- Treatment:
  - Immediately postop:
    - Immediate reattachment ± screw fixation
  - Later postop
    - Autograft or allograft reconstruction of the patellar tendon
- Prognosis:
  - persistent extension lag → abnormal gait and walking stairs
  - Rerupture

12-**Catastrophic Wear**: is the premature failure of prosthetic implants

<table>
<thead>
<tr>
<th>Factor</th>
<th>Problem</th>
<th>Solution</th>
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</thead>
<tbody>
<tr>
<td>1]. PE thickness</td>
<td>PE &lt; 8 mm → past the yielding strength</td>
<td>Thinnest PE portion must be &gt;8mm</td>
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<tr>
<td></td>
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<td>Thicker tibial cut</td>
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<td></td>
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<td>All PE tibia</td>
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<tr>
<td>2]. Articular design</td>
<td>Flat PE Low contact surface area High contact stress load</td>
<td>Congruent articular surface design</td>
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<tr>
<td></td>
<td></td>
<td>High contact surface design</td>
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<tr>
<td></td>
<td></td>
<td>Low contact stress load</td>
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<tr>
<td>3]. Kinematics</td>
<td>Femoral role back</td>
<td>PCL retained design</td>
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<tr>
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<td>Posterior slope to improve flexion</td>
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<tr>
<td>4]. Kinematics</td>
<td>Diskinetic sagittal motion Sliding wear</td>
<td>PCL substituting design</td>
</tr>
<tr>
<td>5]. PE sterilization</td>
<td>PE chain session Subsurface delamination</td>
<td>Vacuum PE γ irradiation</td>
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<td></td>
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<td>Vacuum packing</td>
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<td></td>
<td></td>
<td>Metal-on-metal / ceramic-on-ceramic</td>
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<tr>
<td>6]. PE machining</td>
<td>PE stretching at subsurface zone → delamination</td>
<td>Direct compression molding instead of surface machining</td>
</tr>
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Controversies In TKA

Cementless Vs cemented:
- The brittle nature of the PMMA and its association to osteolysis sight was aroused towards cementless TKA
- Porous coating provides biological interlock
- Disadvantages:
  - Radiolucent lines
  - Tibial subsidence
  - Loose beads
  - More pain and patellar failure and inadequate bone ingrowth
- Some suggested hybrid cementless femur + cemented tibia
- Till now the cemented TKA remain the gold standard procedure

Retaining vs substituting:
- Retaining Rational:
  - Normal knee kinematic
  - Preserve proprioception
  - Minimal constraint
  - Preserve femoral bone stalk
  - Normal gait
- Substituting Rationale:
  - PCL even if present never normal → alter the normal kinematics
  - Difficult balance with PCL sparing
- Almost same clinical outcome and scores are noticed for both

Obesity
- Was considered to be a relative contraindication for TKA
- Associated with high rate of complications, PE wear & hospital stay

Age
- Young age has been a relative contraindication for TKA
- But if the conservative fail to control the condition → make the pt is candidate for surgery
- Surgery start → arthroscopic debridement → realignment osteotomies → arthrodesis
- If all fail → pain persistence + functional limitations → TKA
- Long term results shown 94% results → PE wear being the major failing factor

Hemophilia
- Recurrent hemarthrosis → articular degradation and severe arthritis → Ankylosis
- Pathogenesis of arthritis: free radical formation 2ry to hemoglobin breakdown → synovitis & cartilage damage
- Knee is the most common affect joint
- Arthrodesis is an option; but UCR and osteotomies are contraindicated
- Treatment:
  - Prevent bleeding: Factor VIII (may secrete anti F VIII → make things worse)
  - Preserved joint space.................................Splintage for rest followed by physiotherapy
  - Narrowed joint space.............................TKA
  - Synovitis .....................................................synovectomy
  - Any surgery require F VIII monitoring:
    - > 100% ...............................................till 3rd day postop
    - > 50% ...................................................till 3rd week
    - 30-50% ...................................................till 6th week
- Prognosis there is a high level of adverse effects & HIV transmission is common

Paget’s
- Not uncommon disorder in UK and USA; and usually asymptomatic & discovered accidentally
- Most symptomaticating patients are DJD of the knee
- TKA results are satisfactory
Revision TKA

- **Rationale:**
  - Done when it is expected to achieve a good function
  - Can neutralize minor deficiencies (major bone deficiencies call for arthrodesis)

- **Indications:**
  1. Aseptic loosening
  2. Prosthesis failure
  3. Severe pain

- **Contraindications:**
  1. Active infection
  2. Marked bone deficiency
  3. Severe ligamentous laxity and subluxation
  4. Extensor mechanism insufficiency
  5. Bad general condition

- **Preoperative Planning:**
  - Address cause of failure (pt selection, implant failure, malalignment, instability)
    - Poor pt selection: young, active, heavy wt
    - Failures are common with the old constrained designs
    - Alignment, rotation, and soft tissue balance are crucial

- **Exposure:**
  - Through the previous planes
  - New incisions → hinders the superficial blood supply → sloughing
  - If previous transverse incision → crossed at right angle
  - Usually median parapatellar incision is used
  - Care should be taking during eversion of the patellar tendon
  - Thorough synovectomy, lateral release, and lateral gutter release for better exposure
  - Quad snip or turndown may be needed
  - Remove the components and the cement anchor

- **Implantation:**
  - Coronal orientation is adjusted ....................... 7° valgus
  - Femoral cut is done using IM guide & keep it at minimum (usually augments needed)
  - Rotational orientation is difficult → absent reliable bony land mark; but depend on epicondyles; the medial being more anterior
  - Tibial cut is a straightforward procedure .. ⊥ to the anterior tibial border
  - JL should mimic the normal for better flexion extension balance and PF tracking
  - Normal joint line is determined by ............... Preop AP view (1.5cm above fibular head)

- **Implant size:**
  - Difficult → bone loss; so preoperative planning and templating is mandatory
  - Intraoperative posterior augments are the key for proper sizing → the best balance
  - Tibial size is selected as in primary
  - If upper tibial deformity → custom tibial component → asymm stem orientation is used
  - Usually **Cemented Unconstrained Stemmed PCL Substituting** device is used
    - Unconstrained ........................................ Unstressed across the joints
    - Stemmed ............................................... Stresses distribute away from bone interface
    - PCL ...................................................... Usually scarred and un physiologic
    - Cemented ............................................. Immediate strong fixation

- **Bone deficiency:**
  - Central..................................................... cementation or BG (acc to age, size of loss)
  - Peripheral................................................ bone or metal wedge, custom implant

- **Patella:**
  - Revise only if maltracking + good bone stalk
  - Rule of thumb; at least 10mm is needed as a stalk for revision

- **Results:** up to 75% with semiconstrained & 90% è unconstrained
• Complications:
  o PF malalignment
  o Slough
  o Wound problem
  o Sepsis

• The modern TKA has instrumentation that facilitates reproducible alignment and proper ligament balance, and materials and design that permit functional and durable recovery of motion. When implanted in closely monitored and maintained surgical and rehabilitation environments, it has a remarkable and predictable record of success. Most of the mechanical and some of the nonmechanical complications of knee replacement are preventable by meticulous surgical technique with careful cross checking of anatomic landmarks and soft-tissue tensions with appropriate jigs.