Anterior Cruciate Ligament (ACL) Injuries

• YELLOW

• Intro:

- Stabilises the knee joint along w/ the PCL (forms a cross "X")
- Prevent excessive forward or backward motion of the tibia relative to the femur during FX & EXT
- **Nerve supply:** middle geniculate artery; **Innervation:** posterior articular n. (branch of tibial n.)
- **Origin:** anteromedial aspect of tibial plateau; **Insertion:** Medial aspect of the lateral femoral condyle
- Has 2 bundles (anteromedial & posterolateral)
-

• Aetiology (risk factors):

- Most commonly injured ligament in the knee (almost ½ of all knee injuries)
- F>M (esp. F athletes 4.5:1)
- Possible factors contributing to increased F risk include: weaker hamstrings, preferential recruitment of quads during deceleration, & weaker core stability

Biomechanics & landing factors:

- F landing mechanics mat ↑ injury risk, w/ ↑ valgus angulation & knee EXT
- ↓ hip & knee FX & ↓ fatigue resistance also contribute to ↑ stress on the ACL

Other risk factors:

- Anatomical: high BMI, smaller femoral notch, impingement on the notch, smaller ACL, hypermobility, joint laxity, & previous ACL injury

Hormonal & genetic factors:

- Preovulatory phase, may affect coordination & predispose females to ACL injury
- Females on OCP were noted less affected

Associated injuries w/ ACL ruptures:

- Both intra & extra-articular injuries can accompany acute ACL ruptures
- Meniscal tears are common, w/ lateral meniscus injury more prevalent in acute cases, & medial meniscus more involved in chronic cases
- Other ligaments (PCL, LCL, & PLC) could also be injured in conjunction w/ ACL

Chronic ACL deficiency effects:

- Detrimental effects on the knee
- Development of chondral injuries & complex, unrepairable meniscal tears is observed (e.g bucket handle medial meniscus tears)

Anterior Cruciate Ligament (ACL) Injuries (cont)

• **Pathophysiology:**	- Common in non-contact sports, esp. non-contact pivoting injuries - Tibia translation anteriorly during slight knee FX & valgus - Direct hits to the lateral knee can also cause ACL injuries - Injury occurs during activity/sports participation that involves sudden changes in the direction of movement, abrupt stopping or slowing down while running, or jumping & abnormal landing
• **Clinical presentation:**	- Hx of injury mechanisms - Pt would complain of hearing/feeling a sudden **"pop"** w/ associated deep knee px - About 70% would experience immediate swelling due to haemarthrosis - Other Ssx: knee "giving way", difficulty ambulating, reduced knee ROM
• **Physical examination:**	- Pt demonstrates quadriceps avoidance gait (no active knee EXT) - Varus knee malalignment should be noted as it increases risk of ACL re-rupture - Palpation: swollen knee, & potential joint line tenderness w/ an associated meniscal injury - Move: knee may be **locked** due to associated meniscal injury (other meniscal & ligamentous structures to be assessed) - Lachman test, Anterior drawer test, Pivot shift test
• **Diagnosis:**	- **MRI** is the 1° modality to diagnose ACL pathology - Knee arthroscopy to differentiate complete from partial tears & chronic tears (gold standard test) - Radiography to rule out fractures & other osseous injuries

Anterior Cruciate Ligament (ACL) Injuries (cont)

• **Complications:**	- Surgical: tunnel malpositioning, posterior wall blowout, graft failure due to various other issues - Post op: infection & septic arthritis - Stiffness & arthrofibrosis - Infrapatellar contracture syndrome - Patella tendon rupture - CRPS - Patella fracture - Tunnel osteolysis - OA in the long term - Saphenous n. irritation - Cyclops lesion
• **Management:**	**Non-operative management:** - **Indication:** when there's reduced ACL laxity on low-demand pts or athletes involved in no cutting or pivoting activities or partial ACL tears - RICE - Non-WB (crutches or wheelchair) - NSAIDs - Phase 1: acute symptomatic treatment - Phase 2: 12 weeks of supervised physiotherapy starting w/ regaining full ROM & progression to quad, hamstring, hip ABD & core strengthening **Operative management:** - **Indication:** complete ACL rupture in younger or older active, high-demand pts, & partial ACL rupture w. functional instability - Two options: ACL reconstruction or repair
• **Ddx:**	- ACL tear - Epiphyseal fracture of femur/tibia - MCL injury - Meniscal tear - Osteochondral fracture - Patellar dislocation - Posterior cruciate ligament injury - Tibial spine fracture

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Lateral Collateral (LCL) & PLC Injuries

• **YELLOW**

Lateral Collateral (LCL) & PLC Injuries (cont)

• **Intro:**	- 1° resistor of varus stress - Provides posterolateral stability (preventing medial translation of the tibia) - LCL & PLT resist external tibial rotation in 0-30° of knee FLX - Minor role in tibial translation (stabilises anterior & posterior tibial translation when cruciate ligaments are torn) - **Origin:** lateral epicondyle of the femur, **Insertion:** fibular head - **Blood supply:** branches of popliteal artery, **Innervation:** common fibular n. - **Surrounding structures:** popliteus tendon (PLT) & iliotibial band (ITB)
• **Aetiology (risk factors):**	- 40% of PLC (posterolateral corner) & LCL injuries result from contact sports - Other causes include trauma, motor vehicle accidents, & falls - F>M - High-contact sports - Sports involving high-velocity pivoting & jumping - Tennis & gymnastics are most specific fro isolated LCL injuries - Prior knee, ankle, or hip injury increases the risk
• **Pathophysiology:**	- LCL injuries rarely occur in isolation - High-energy blow to the antero-medial knee - Involves hyperEXT & extreme varus force - Non-contact hyperEXT & varus stressors can also cause LCL injuries
• **Clinical presentation:**	- Acute event consistent w/ a medial blow to the knee while fully EXT, or extreme non-contact varus bending - Complain of sudden onset lateral knee px, swelling, & ecchymosis after the injury - May report thrust gait, inc. foot kicking in mid-stance - May complain of paresthesias over the lateral lower extremity, & weakness &/or a foot drop - Gain complete Hx inc. bleeding/clot disorders, previous surgeries, occupation, gait, ambulation-assisted devices, living situation (stairs at home)

Lateral Collateral (LCL) & PLC Injuries (cont)

• **Physical examination:**	- Limited ROM - Lateral knee TTP - Ecchymosis, swelling, & warmth - Gait: classical *varus thrust* finding - **Special tests:** Varus stress test, EXT ROT recurvatum test, Posterolateral drawer test, Reverse pivot shift test, Dial test
• **Diagnosis:**	- MRI is the gold standard - US useful for rapid diagnosis **Classification of injury:** - **Grade 1:** Mild sprain - diagnosed w/ lateral knee tenderness, no instability mechanical Ssx - **Grade 2:** Partial tear - diagnosed w/ more severe localised lateral & posterolateral knee px, as well as swelling - **Grade 3:** Complete tear - px & swelling vary in pts, usually associated w/ PLC & other related injuries, & mechanical Ssx
• **Complications:**	- Undiagnosed LCL & PLC injuries have several long-term complications - Continued knee instability & chronic px - 35% of PLC injuries may have an associated peroneal n. palsy (probs due to its proximity to the LCL) - Pts may develop long-term foot drop, as well as lower extremity weakness & decreased sensation - Post op: hardware irritation & stiffness
• **Management:**	- Acutely, all grades treated w/ RICE & NSAIDs - Grade 1 & 2: non-operative, non-WB for 1 week for better px control; next 3-6 weeks, the pt should be in a hinged-knee brace while performing functional rehab - Grade 3: surgical reconstruction (best results), post op rehab and functional exercises
• **Ddx:**	- ACL/PCL tears - Lateral meniscus tears - Popliteal injury - Bone contusion - ITB syndrome

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Posterior Cruciate Ligament (PCL) Injuries

• YELLOW	
• **Intro:**	- 1/4 major ligaments of the knee that function to stabilise the tibia on the femur - **Origin:** anterolateral aspect of the medial femoral condyle in the area of the intercondylar notch, **Insertion:** posterior aspect of the tibial plateau - Prevents posterior translation of the tibia on the femur - Lesser extent, the PCL functions to resist varus, valgus, & EXT ROT forces
• **Aetiology (risk factors):**	- Caused by extreme anterior force applied to the proximal tibia of the FX knee - Dashboard injuries during car accident or falling forward onto a FX knee - M>F (2:1) - Motorcycle accidents (28%) & soccer-related injuries (25%) are the leading causes
• **Pathophysiology:**	- Least common knee injury - Anterolateral portion is more commonly injured due to majority of injuries occurring in knee FX - Resists posterior translation w. the assistance of the posterolateral joint capsule, popliteus, MCL, & posterior oblique ligament
• **Clinical presentation:**	- Pts often present w/ acute onset of posterior knee px, swelling, & instability - Hx includes mechanism of injury, ic. falling onto FX knee or recent vehicle accident
• **Physical examination:**	- Pulses - SMR - Mild to moderate joint effusion - Swelling usually less than in ACL tear - Pt may present w/ antalgic gait & potential difficulty walking up & down stairs - Palpation: potential effusion, joint line for tenderness (suggestive of meniscal tears) - Muscle strength test: should be normal, but there may be weakness w/ knee EXT & FX 2° to guarding - Limited ROM - **Special tests:** Posterior drawer test, Quadriceps active test, Dial test or EXT ROT test, Varus/valgus stress

Posterior Cruciate Ligament (PCL) Injuries (cont)

• **Diagnosis:**	- MRI is the gold standard - Initial imaging w/ plain X-rays **Classification of injury:** - Grade 1 (partial tear) - 1-5mm posterior translation, tibia remains anterior to femoral condyles - Grade 2 (complete isolated) - 6-10mm posterior tibial translation, complete tear of PCL w/o another injury, anterior tibia flush w/ femoral condyles - Grade 3 (complete PCL w/ combined capsular &/or ligamentous injury) - >10mm posterior tibial translation, tibia posterior to femoral condyles which may indicate a concomitant capsuloligamentous injury
• **Complications:**	- Intra & postoperative complications of PCL surgery - Neuromuscular injury (e.g. popliteal artery injury) - Fracture - Residual instability - Osteoarthritic progression - Osteonecrosis - Stiffness - Failure of associated ligament reconstructions or meniscal repairs - Revision of PCL reconstruction

Posterior Cruciate Ligament (PCL) Injuries (cont)

• **Management:**
- **Variables to consider:** Acute or chronic; isolated or combined

Non-operative:
- Acute grade 1 & 2 injuries w/ posterior tibial translation (8-12mm)
- Grade 3 injuries w/ mild Ssx or low-demand activities
- Acute treatment inv. RICE, initial knee bracing, & crutches
- Rehab focuses on knee EXT strengthening
- Est. return-to-play in 2-4 weeks for Grade 1 & 2 injuries
- Grade 3 may inv. knee immobilisation followed by rehab

Operative:
- Acute injuries w/ tibial translation >12mm, associated meniscal tears, dislocation, bony avulsions, & combined injuries
- Chronic injuries w/ posterior tibial translation >8mm, symptomatic cases, instability, & combined injuries
- Arthroscopic procedures
- Reconstruction
- Graft fixations
- High tibial osteotomy

• **Ddx:**
- ACL injury
- LCL injury
- MCL injury
- Meniscus injury
- Talofibular ligament injury

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Baker's Cyst

• GREEN

• **Intro:**
- Also known as popliteal or paramedical cyst
- Fluid-filled sac, typically between semimembranosus & medial head of the gastroc
- Common in adults & associated w/ degenerative knee conditions
- Often linked to degenerative meniscal tears as one of the most common causes
- In children, popliteal cysts more commonly arise as a 1° condition (resulting from herniated post knee joint synovium/capsule)

Baker's Cyst (cont)

• **Aetiology (risk factors):**	- Children aged 4-7 yo - Adults aged 35-70 - Most commonly found in adults w/ Hx of trauma, knee joint diseases (OA, RA, etc), or as incidental findings - They form due to accumulation & extrusion of synovial fluid between the semimebranosus & medial head of the gastroc - Popliteal cysts are located on the medial side of the popliteal fossa just below the crease at the posterior knee - Prevalence increases w/ age, likely due to an increase in knee-burial communication
• **Pathophysiology:**	**Several mechanisms:** **1.** Joint-cyst communication **2.** Sequestration of synovial fluid in popliteal fossa due to a valve-like effect between the joint & cyst (controlled by gastroc-s-emimembranosus m. w/ FX & EXT at the knee) **3.** Negative intraarticular knee pressure during partial FX combined w/ a positive pressure during extension (as a result directing fluid flow towards the cyst from the suprapatellar bursa during FLX) **4.** Gastroc-semimembranosus bursa enlargement resulting from micro-traumas to the bursa w/ muscle contractions **5.** Herniation of the joint capsule into the popliteal fossa
• **Clinical presentation:**	- Sensation of tightness, discomfort, or px behind the knee - Swelling Moree noticeable when standing w/ full knee EXT - Swelling reduces or disappears when the knee is flexed to 45° (Foucher's sign) - Px worsens w/ increased activity & may limit full knee FX & EXT
• **Physical examination:**	- Compression of surrounding vessels - Lower extremity oedema due to venous obstruction - Enlargement into the calf m. (dissecting cyst) can cause swelling, erythema, distal oedema, & +ve Homan's sign - Venous obstruction can mimic Ssx of DVT or thrombophlebitis

Baker's Cyst (cont)

• Diagnosis:	**Diagnosis methods:** - Pt stand & full knee EXT - Mass is most prominent in this position - Mass often softens or disappears when the knee is FX to 45° (Foucher's sign) - Supine exam: knee passively moved from full EXT to at least 90° FX **Imaging:** - Plain radiograph & US - MRI is recommended, esp. if considering surgery
• Complications:	**Complications & Ssx of ruptured Baker's cyst:** - Rapid fluid accumulation may cause cyst rupture - Released fluid into surrounding tissues can lead to inflammation - Ssx similar to thrombophlebitis: sharp pc in the knee & calf; swelling or erythema of the calf; sensation of water running down the calf **Complications of cyst rupture:** - Post tibial n. entrapment: posterior plantar numbness & calf px - Popliteal artery occlusion: lower extremity oedema - Anterior compartment syndrome: foot drop, oedema of anterolateral leg - Posterior compartment syndrome: plantar dysesthesia, weakness of toes, calf swelling, px worsens w/ passive toe extension

Baker's Cyst (cont)

- **Management:**
  - Asymptomatic cases are managed through observation & reassurance alone
  - Essential to treat any underlying joint disorder in pts w/ symptomatic Baker's cysts
  - Helps reduce synovial fluid accumulation & cyst enlargement

  Non-operative:
  - Rest/activity modification
  - NSAIDs
  - Physical therapy & rehab regimes are effective for minimal Ssx & smaller degenerative meniscal tears
  - Aspiration & steroid injection
  - Lower recurrence in younger pts
  - Higher recurrence rates in older pts & those w/ degenerative meniscal tears

  Operative:
  - Arthroscopy
  - Open cyst excision (not recommended in case of underlying degeneration due to recurrence risk)
- **Ddx:**
  - Abscess
  - Arteriovenous fistula
  - DVT
  - Ganglion cyst
  - Hemangioma
  - Lipoma
  - Lymphadenopathy
  - Malignancy (e.g. fibrosarcoma, liposarcoma)
  - Popliteal (Balker's) cyst

link text

Chondrocalcinosis (pseudogout)

- YELLOW

Chondrocalcinosis (pseudogout) (cont)

• Intro:

Overview:
- Calcium pyrophosphate deposition disease (CPPD)
- Crystal deposition arthropathy involving synovial & periarticular tissues
- Asymptomatic to acute or chronic inflammatory arthritis

Phenotypes & terminology:
- Various terms used for different phenotypes
- Acute CPP deposition arthritis: *"Pseudogout"*
- Chronic CPP deposition arthritis: informally called *"pseudo-rheumatoid arthritis"*
- Characterised by a waxing & waning clinical course, resembling RA

Radiological findings:
- Term: ***chondrocalcinosis***
- Describes intra-articular fibrocartilage calcification

Commonly affected joints:
- Hips
- Knees
- Shoulders

Underlying factors:
- Often associated w/ underlying joint disease or metabolic abnormalities
- Predisposing factors: OA, trauma, surgery, RA

• Aetiology (risk factors):

- Often pts >65 yo, w/ 30-50% >85 yo
- M>F
- Rare <60 yo
- High prevalence of radiographic **chondrocalcinosis** in the general population

Comorbidities associated w/ CPPD:
- Hyperparathyroidism
- Gout
- OA
- RA
- Hemochromatosis

Other related comorbidities:
- Osteoporosis
- Hypomagnesium
- Chronic kidney disease
- Calcium supplementation

Chondrocalcinosis (pseudogout) (cont)

• **Pathophysiology:**	- Caused by an imbalance between pyrophosphate production & pyrophosphate levels in diseased cartilage - Pyrophosphate deposits in the synovium & adjacent tissues combine w/ calcium to form CPP - Deposition of calcium pyres-hate can activate the immune system, leading to inflammation & soft tissue injury
• **Clinical & physical presentation:**	**Acute cases:** - Typically, self-limiting, & inflammation resolves within days - weeks w/ treatment - Similar to acute rate arthropathy - Joint oedema, erythema, & tenderness - Up to 50% may have a low-grade fever - Most commonly affected joint: knee - Other affected joints: hip, shoulders **Chronic cases:** - May show Ssx of RA inc. morning stiffness, localised oedema, & ↓ ROM - Waxing & waning episodes of non-synchronous, inflammatory arthritis - Affeects multiple non-WB joint: wrist & MCP joint - Causes *"crowned dens syndrome"* (deposition of CPP around C2) - Mostly asymptomatic
• **Diagnosis:**	- Confirm diagnosis through synovial fluid analysis **Imaging:** - For involved joints is recommended - Presence of *chondrocalcinosis* in imaging supports CPPD Dx - Absence of *chondrocalcinosis* doesn't rule out CPPD - US may reveal early signs like cartilage abnormalities - Radiographic imaging may show joint cartilage calcification - MRI is useful - can evaluate crystal deposition in joint cartilage

Chondrocalcinosis (pseudogout) (cont)

- **Complications:**
  - Potential of triggering inflammatory responses
  - Presence of *chondrocalcinosis* has associations w/ degradation of menisci & synovial tissue
  - Pts rarely present w/ palpable nodules (resembling gout) that may lead to further joint degradation
  - Rare spinal involvemen, causing clinical manifestations like spine stiffness & bony ankylosis (resembling AS)
  - Some pts present w/ manifestations similar to DISH w/ PLL calcification leading to spinal cord compression Ssx
- **Management:**
  - 1st step reduce inflammation & addressing underlying metabolic conditions
  - NSAIDs
  - Acute flares inv. 1-2 joints often treated w/ joint aspiration
  - Medication
  - Low-alkaline diet
- **Ddx:**
  - Tenosynovitis w/ carpal or cubital tunnel s. can occur (multiple joints affected)
  - RA
  - AS
  - Erosive OA
  - Gout

link text

Acute Compartment Syndrome

- RED
- **Intro:**
  - Increased pressure in closed osteofascial compartmetn
  - Leads to impaired local circulation
  - Surgical emergency

-Untreated ACS can lead to ischemia & necrosis

Compartments:

- Lg has 4 compartments: anterior, lateral, deep posterior, & superficial posterior
- Anterior compartment is most common ACS
- Contains extensor m., tibialis anterior m., deep peroneal n., & tibial artery

**Open fractures:*

- Skin laceration doesn't relieve compartment pressure
- ACS is still predictable, esp. in open Gustily type 2 & 3 lesions in proximal intra-articular tibia fractures

Acute Compartment Syndrome (cont)

• **Aetiology (risk factors):**	- M>F (7.3:0.7) - Majority of cases result from trauma, w/ tibial shaft fracture being the most common - More common in males <35 yo, possibly due to larger muscle mass & high-energy trauma involvement - Pts w/ bleeding diathesis (e.g. hemophilia) are at high risk - ACS reported w/o trauma in paediatric leukaemia cases - Pts w/o fractures at high risk of complications & delayed treatment - Other causes:** soft tissue injuries, burns, vascular injuries, crush injuries, drug overdoses, repercussion injuries, thrombosis, bleeding disorders, infections, improperly placed casts or splints, tight circumferential bandages, penetrating trauma, intense athletic activity, & poor positioning during surgery - In children, supracondylar # of the humerus & ulnar/radial # are associated w/ compartment syndrome
• **Pathophysiology:**	- Caused by ↓ intracompartmental space OR ↑ fluid volume, making the surrounding fascia non-compliant - ↑ compartment pressure impairs hemodynamics, disrupting the equilibrium between venous outflow & arterial inflow - Elevated compartment pressure leads to reduced venous outflow, ↑ venous capillary pressure - If intracompartmental pressure surpasses arterial pressure, arterial inflow ↓, causing tissue ischemia - Reduced venous outflow & arterial inflow result in ↓ tissue oxygenation, potentially leading to irreversible necrosis - Normal compartment pressure is <10mmHg, while reading of 30mmHg or higher indicates ACS
• **Clinical presentation:**	- Can occur within few hours to up to 48h after trauma - Px is severe & disproportionate to the injury; may be felt as a burning sensation or deep ache - Initially, px may only occur w/ passive stretching but can be absent in advanced cases - Paresthesia, hypoesthesia, or poorly localised deep muscular px may be present - The **"5 P's"** (px, pulselessness, paresthesia, paralysis, & pallor) are classic signs, but they're typically late findings - Paresthesia may occur earlier - In some cases, a pulse may still be present, even in a severely compromised extremity

Acute Compartment Syndrome (cont)

• **Physical examination:**	- Earliest objective physical finding is the **tense*, or** 'wood-like' **feeling int he involved compartment*** *Focus on neurovascular exam.** - Observe skin for lesions, swelling, or colour change - Palpate compartment, noting °C, tension, & tenderness - Check pulses in the affected area - Evaluate two-point discrimination & sensation - Assess motor function - Due to potential rapid progression, serial exam should be performed to monitor changes over time
• **Diagnosis:**	- Radiographs are recommended if # suspected - Measurement of intracompartmental pressure (not required), can aid in Dx - Normal pressure: 0-8mmHg - Abnormal: exceeding 30mmHg indicates compartment s. & necessitates intervention - Pressure within 10-30mmHg of diastolic blood pressure suggests inadequate perfusion & relative ischemia, prompting clinical attention - **DUS** can be used to detect occlusion or thrombus - Elevated CPK levels may suggest muscle breakdown from ischemia, damage, or rhabdomyolysis - Pre-operative studies: CBC & coagulation studies
• **Complications:**	- Px - Contractures - Rhabdomyolysis - N. damage & associated numbness &/or weakness - Infection - Renal failure - Death
• **Management:**	- Immediate surgical consult - Keeping extremity at heart level
• **Ddx:**	- DVT - Cellulitis - Peripheral vascular injury

link text

Chronic (Exertional) Compartment Syndrome

• **RED**

Chronic (Exertional) Compartment Syndrome (cont)

• **Intro:**	- Often a Dx of exclusion characterised by ↑ pressures in a muscular compartment , leading to ischemia & px - CECS inv/ recurrent, reversible ischemic episodes after activity cessation, leading to predictable ↓ in fascial compartment pressures - Rare condition w/ delayed diagnosis, resembling ACS - Requires surgical emergency intervention through fasciotomies to prevent irreversible m. ischemia & neurovascular injury - Usually occurs in the LL but can also affect forearm, thigh, or hand
• **Aetiology (risk factors):**	- Relatively common among young adult athletes (running, endurance training, soccer, field hockey, & lacrosse) - Anterior compartment most commonly affected (70% of cases), then deep posterior - B limb in 37-82% of symptomatic cases - 20-25 yo, M>F, & often B - Associated w/ sports like running or skating, & higher activity intensities - Can result from overuse injuries, repetitive mechanisms causing tissue degeneration, scar formation, & military training - Pts w/ decreased fascial elasticity may be at risk for nerve entrapment & quicker rises in pathological pressures
• **Pathophys-iology:**	- Has multiple etiologies - Muscle compartment swelling during exercise, leading to increased pressure within musculofascial compartments - Rise in pressure compromises blood flow, causing px, motor weakness, & paresthesia, in corresponding neurovascular distributions - Specific Ssx depend on the affected compartment: e.g. anterior & lateral compartment involvement in the LL may present w/ px & tingling on the dorm of the foot - Increased intracompartmental pressure results in reduced myocyte oxygenation, leading to myonecrosis & neurological damage

Chronic (Exertional) Compartment Syndrome (cont)

• **Clinical presentation:**	- Pts often present following sport-related activity ro exertion w/ non-specific leg px & persist after strenuous or repetitive activity - Ssx will predictably abate following activity cessation - Particular attention to pts characterisation of px during strenuous activity, well-localised to a specific compartment, & the px/Ssx disappear quickly after the cessation of activity - Pts will generally complain of discomfort described as squeezing, cramping, aching, or burning that typically begins within 15-20 minutes of activity - Discomfort resolves completely w/ rest, although the duration may vary
• **Physical examination:**	- In 70-95% of cases B px - Physical exam often unremarkable, esp. if not done during or immediately after exercise - Suspected cases should undergo pre- & post-exercise physical exams - After exercise, the affected compartment may feel tender, bulge, or be tight, & passive stretching may cause px - Focal neuro findings may inc. ↓ sensation, paresthesia, or weakness **Stryker pressure monitoring system:** - Baseline measurements w/ pt at rest - Pts then perform controlled exercise until severe Ssx occur - After 5-minute rest, compartment pressure measured again
• **Diagnosis:**	**Pedowitz criteria:** -Rule out a Dx of CECS - Resting pressure ≧ 15mmHg &/or a pressure of ≧ 30mmHg at 1 min post-exercise in any compartment, &/or; - Post-exercise pressure greater than 20mmHg at 5 min post-exercise
• **Complications:**	- Benign condition characterised by resolution of Ssx w/ rest

Chronic (Exertional) Compartment Syndrome (cont)

- **Management:**
  - Conservative treatment generally ineffective, inc. rest, activity modification, stretching, orthotics, & physical therapy
  - Non-operative modalities inc. NSAIDs, injections, gait training (forefoot strike patterns)
  - Open fasciotomy is the predominant surgical technique
- **Ddx:**
  - Initially gets misDx as shin splints or medial tibial stress syndrome (MTSS)
  - Vascular pathologies (intermittent claudication, popliteal artery impingement)
  - Tibial stress #
  - Tendon pathologies (tendinitis, tendinosis, or tendon rupture)
  - N. entrapment

link text

Fibular Nerve (Peroneal N.) entrapment

- GREEN

Fibular Nerve (Peroneal N.) entrapment (cont)

- - Branches off the sciatica n. in the distal posterior thigh & receives fibres from L4-S2 nerve roots

Intro:

- Runs down the thigh, posterior to the biceps femoris m., & crosses laterally to the head of the lateral gastroc m.
- Provides sensory innervation to the lateral leg via the lateral sural n.
- **Two branches:** *superficial* which innervates the lateral compartment of the leg, & *deep* which innervates the anterior compartment of the leg & foot dorsum
- Both have roles in foot eversion & dorsiflexion

Innervation of superficial

Motor:

- Lateral compartment
- Peroneus longus
- Peroneus brevis

Sensory:

- Anterolateral leg

Innervation of deep

Motor:

- Anterior compartment
- Tibialis anterior
- Extensor hallucis longus
- Extensor digitorum longus
- Peroneus tertius

Sensory:

- First dorsal webspace

Fibular Nerve (Peroneal N.) entrapment (cont)

• **Aetiology (risk factors):**	- Most common mononeuropathy in the LL & 3rd most common focal neuropathy overall (after carpal tunnel s. & ulnar neuropathies) - Common in traumatic injuries in young athletes (e.g. football, soccer) & following high energy trauma (car accidents) in adults - Occurs in about 16-40% of knee dislocations **Trauma or injury to the knee:** - Knee dislocation - Direct impact, penetrating trauma, or lacerations - Fibula #, esp. proximal fibula **External compression sources:** - Tight splint/cast - Compression wrapping/bandage - Habitual leg crossing - Prolonged bed rest - Positioning during anaesthesia & surgery (important to pad bony prominences) **Systemic causes:** - Diabetes mellitus - Inflammatory conditions - Anorexia nervosa **Others:** - Intramural ganglion - Peripheral nerve tumour - Iatrogenic injury following surgery to the hip, knee, & ankle
• **Clinical presentation:**	- Varies based on location, severity, & anatomic variations - Commonly presents w. weakness in ankle dorsiflexion - Classic result is *foot drop* or *catching toes* while walking - Development of acute or gradual, complete or partial - Numbness or paresthesia along lateral leg, dorsal foot, &/or first toe webspace - Possible px in traumatic cases

Fibular Nerve (Peroneal N.) entrapment (cont)

• **Physical examination:**	- Gait: significant for chronic peroneal nerve palsy w/ foot drop, high stoppage gait weakened dorsiflexors to prevent toe dragging **Localisation of lesions:** - Proximal lesions (e.g. knee dislocations) may present w/ numbness in both superficial & deep n. distributions - Upper lateral leg numbness indicates a lesion proximal to fibular head (possibly inv. sciatic n. or lumbosacral n. roots) - Lower lateral leg & dorms of the foot involvement suggests superficial peroneal n. - Altered sensation in the dorsal aspect of the first web space implicates the deep peroneal n. **Motor involvement testing:** - Assess foot eversion (superficial n.) & foot/toe dorsiflexion (deep n.) - Weakness in both suggests common Peroneal n. involvement - Proximal lesion may result in both distributions - Detailed examination of dorsiflexion ability is crucial **Tinel sign:** - Tapping along the nerve course, esp. around the fibular neck - +ve test = tingling or paresthesia distally
• **Diagnosis:**	- CT can be used to assess osseous abnormalities - MRI/US suitable fro evaluating soft-tissue sources or masses (es. in cases of traumatic knee dislocations) - Electrodiaagnostic studies (inc. NCV & EMG) are used to Dx peroneal nerve palsy - They evaluate motor & sensory axons of the peroneal n. aiding in localisation of the nerve injury - Useful in post-operative setting of a known traumatic injury for long-term management planning & pt care
• **Management:**	- Full physical therapy - Ankle-foot orthoses, even for foot-drop when surgery isn't warranted - Surgical indicators: rapid deterioration & no signs of improvement within **3 months** & open injuries w. suspected nerve laceration -Open lacerations should undergo exploration & surgical repair within 72h

Fibular Nerve (Peroneal N.) entrapment (cont)

• **Ddx:** - Peroneal tendon pathology
- Other compressive neuropathies (tarsal tunnel s., anterior tarsal tunnel s., non-specific tendinitis affecting lower limb m./t.)
- Chronic ankle px

link text

Meniscal Tears

• YELLOW

• - Lateral & medial menisci function in load transmission & shock absorption in the tibiofemoral joint

Intro: - Inner 2/3 (white zone) of the menisci is avascular, likely receiving nutrition through synovial fluid diffusion
- Peripheral 1/3 (red zone) is well-vascularised, supplied by branches of the medial & lateral vehicular arteries
- Medial meniscus is less mobile than the lateral one, firmly attached to the joint capsule & deep fibres of the MCL
- Lateral miscues doesn't connect w/ the LCL & has looser attachments w. the joint capsule
- Anterior margins of the menisci are connected by the transverse inter meniscal ligament
- Peripheral 2/3 of the menisci contain nociceptive free endings (pain perception), while mechanoreceptors are in the anterior & posterior horns, suggesting a proprioceptive function
- Posterior horn of the lateral meniscus connects to the femur via meniscofemoral ligaments & the adjacent popliteus tendon

Meniscal Tears (cont)

• Aetiology (risk factors):

- 61/100,000 in general population (USA), 9/1000 in military population
- 15% of sports injuries
- M>F
- Age >40 yo
- ACL deficient knees, esp. if ACL reconstruction is delayed beyond 1 year from initial injury
- Medial>lateral meniscal tears
- **Increased risk factors:** infantry-related duties, frequent squatting/kneeling, & participation in sports like soccer, rugby, football, basketball, baseball, skiing, & wrestling
- Traumatic impacts to the knee can lead to isolated meniscal tears or tears concomitant w/ bony lesions or damage to primary stabilising ligaments (ACL & MCL)
- Less force is required for tears in individuals w/ degenerative changes of the menisci, typically seen in adults >40 w. concomitant OA
- **Isolated meniscal tears** result from rotational or shearing forces across the tibiofemoral joint, esp. during activities w/ increased closed kinematic chain FX, heavy lifting, rapid acceleration/deceleration, change of direction, & jumping

Meniscal Tears (cont)

• **Pathophysiology:**	- Characterised by shape & location on MRI - **Horizontal** (cleavage) run parallel to the tibial plateau, associated w/ degenerative changes in people >40 w/o specific causes - **Longitudinal** run perpendicular to the tibial plateau & parallel to the meniscus axis - **Radial** run perpendicular to both the tibial plateau & the meniscus axis, originating from the inner free edge - **Complex** involve combinations of horizontal, longitudinal, or vertical tears - **Displaced** involve complete detachment or flipping of a piece still attached to the meniscal body - **Bucket-handle** are complete longitudinal tear fragments that migrate centrally - **Parrot-break** are radial tears w/ partially detached fragments - **Flap** are partially detached fragments of horizontal tears - Tears in the outer 1/3 vascular zone are "red-red"; those extending into the inner 2/3 avascular zone are "red-white", & tears within the inner 2/3 avascular zone are "white-white" - Tears in the red zone have the highest potential for spontaneous healing w/ conservative management or successful outcomes after meniscal repair
• **Clinical presentation:**	- **"POP"** sensation w/ immediate knee effusion suggests ACL tear w/ possible medial meniscal involvement - Gradual effusion over 24h indicates an isolated meniscal tear - Ssx can be insidious, featuring low-grade effusion& stiffness w/o a specific triggering event - Px commonly reported along the anteromedial or anterolateral joint line - **Additional Ssx:** locking, clicking, catching, intermittent inability to fully EXT the knee, & a sense of the knee giving way

Meniscal Tears (cont)

• **Physical examination:**	- Inspection of edema, palpation of joint line, standing & supine ROM, muscle strength testing, special testing - Anteromedial & anterolateral joint line tenderness at 90° FX - Px & deficits in FX or EXT ROM may vary based on tear type & effusion extent - Deficits in open kinetic chain knee FX/EXT strength testing are unlikely - Antalgic gait or increased px w/ squatting may indicate meniscal issues due to compressive forces - **Special tests:** Thessaly test, McMurray's test, Apley's compression test
• **Diagnosis:**	- Begin w/ radiographs - AP, lateral, oblique, sunrise, & WB views to assess concomitant bony pathologies, loose bodies, & OA - Arthroscopy is the gold standard - MRI is the best mode of imaging to Dx & characterise tears
• **Management:**	- RICE - NSAIDs - Early px-free knee & ankle ROM exercise (help limit motion loss & aid edema) - Bracing/sleeves (protection & compression) **Simple tears (outer 1/3 of the meniscus) & degenerative tears:** - 4-6 weeks relative rest & physical therapy - Despite conservative management, pts w/ persistent px, swelling, & mechanical Ssx should be evaluated for surgical intervention **Surgical tears:** - Meniscal repair is preferred over meniscectomy (risk of accelerated OA) - Factors ↑ success: tears that occur in red zone of the meniscus, shorter than 2cm, vertical longitudinal tears, & acute tears **Rehab:** - First 6 weeks inc. restrictions in knee FX ROM & WB status (depending on tear & repair type) - Strengthening - Mobs

Meniscal Tears (cont)

- **Ddx:**
  - ACL injury
  - Contusions
  - ITB syndrome
  - Knee osteochondritis
  - LCL injury
  - Lumbosacral radiculopathy
  - MCL injuy
  - Medial synovial plica irritation
  - Patellofemoral joint syndrome
  - PCL injury

link text

Osgood-Schlatter Disorder (OSD)

- **GREEN**

- **Intro:**
  - Common cause of anterior knee px in skeletally immature athletes
  - Also known as *osteochondrosis* or *traction apophysitis of the tibial tubercle*
  - Common in sports like basketball, volleyball, sprinting, gymnastics, & football
  - Self-limiting & results from repetitive stress on the extensor mechanism (jumping/sprinting)
  - While benign, OSD can lead to prolonged recovery & absence from sports
- **Aetiology (risk factors):**
  - Leading cause of knee px in adolescent athletes
  - Onset typically aligns w/ growth spurts: 10-15 M & 8-13 F
  - M>F
  - 9.8% of adolescents 12-15 yo (11.4% M; 8.3% F)
  - B Ssx observed in 20-30% of pts
  - Overuse injury due to repetitive strain from patellar tendon
  - Force increases w/ higher activity levels, after rapid growth
  - **Predisposing factors:** poor flexibility of quadriceps & hamstrings, extensor mechanism misalignment

Osgood-Schlatter Disorder (OSD) (cont)

• **Pathophysiology:**	- Tibial tubercle develops as a 2° ossification centre for patellar tendon attachment - Bone growth surpasses muscle-tendon-bone-attachment, susceptible to injury from repetitive stress - Repeated quadriceps muscle contraction, esp. in sports involving running & jumping, can cause apophyseal ossification centre softening & partial avulsion, resulting in osteochondritis **Tibial tubercle development sequence:** - Entirely cartilaginous before age 11 - Apophysis forms between 11-14 - Apophysis fuses w/ proximal tibial epiphysis between 14-18 - Proximal tibial epiphysis & tibial tubercle apophysis fuse w/ the rest of the proximal tibia after age 18 - **Prevailing theory:** repeated traction over tubercle causes microvascular tears, fractures, inflammation
• **Clinical presentation:**	- Common Ssx: anterior knee px - Presentation: w/ or w/o swelling, unilateral or bilateral - Onset: typically insidious, w/o preceding trauma - Nature of px: dull ache localised over tibial tubercle - Px progression: gradually increases w/ activity - Px relief: typically improves w/ rest - Duration of relief: subsides minutes to hours after stopping activity or sport - Exacerbating factors: running, jumping, direct knee trauma, kneeling, & squatting
• **Physical examination:**	- Enlarged prominence at the tibial tubercle - Tenderness over the patellar tendon insertion site - Reproduction of px: resisted knee EXT & active/passive knee FX can reproduce px

Osgood-Schlatter Disorder (OSD) (cont)

• **Diagnosis:**	- 1° Dx clinically, radiographic evaluation typically not necessary - Consider comparing B images to help delineate normal vs abnormal in the pt **Radiographic use:** - Plain radiographs may be employed in severe or atypical presentations - Used to rule out additional conditions like fractures, infections, or bone tumours - Assessment fro avulsion injury or other traumas may necessitate radiographic evaluation **Classic findings:** - Elevated tibial tubercle w/ soft tissue swelling - Fragmentation of the apophysis - Calcification in the distal patellar tendon
• **Complications:**	- Prominence of tibial tubercle - Ongoing px - Ssx continue to adulthood if treatment isn't provided or poor compliance w/ recommended treatment
• **Management:**	- Excellent prognosis - Self-limiting but time to resolution can take up to 2 yrs until apophysis fuses - Surgery rarely indicated, low benefit & high complication risk - Relative rest & activity modification based on px levels - Participation in sports allowed if px resolves w/ rest & doesn't limit activities **Px management:** - Ice & NSAIDs - Protective knee pad recommended over tibial tubercle to prevent direct trauma - Hamstring & quadriceps stretching, & strengthening - In severe cases, short knee immobilisation might be considered **Refractory cases:** - In up to 10%, Ssx may persist >1-2 yrs beyond skeletal maturity - Ossicle excision may be performed in skeletally mature pts w/ persistent Ssx

Osgood-Schlatter Disorder (OSD) (cont)

• **Ddx:**	- Patella tendonitis - Osteomyelitis of the tibia - Perthes disease - Synovial place injury - Infectious apophysitis

link text

Osteochondritis Dissecans (OCD)

• **YELLOW**

• **Intro:**	- Rare condition affecting the knee, categorised as a form of osteonecrosis in the subchondral bone - 1° occurs in school-aged children & adolescents, w/ manifestations of the dysfunction & px - Juvenile OCD occurs in pts w/ open growth plates, while adult OCD applies to skeletally mature pts - If left untreated, OCD can lead to degenerative changes, chronic px, & mechanical Ssx such as 'locking' & 'clicking'
• **Aetiology (risk factors):**	- Highest incidence 12-19 yo - 9.5-29 / 100,00 - M>F - 75% of affected pts have knee lesion, w/ 64% localised in the medial femoral condyle - 32% of knee lesions are found in the lateral condyle, while other cases localise to the trochlea, patella, & tibial plateau - Usually unilateral, but 7-25% of pts have B disease - Theories inc. micro-trauma, ischemia, & genetic predisposition - Pts w. extreme obesity & elevated BMI face an increased risk of developing OCD - Repetitive trauma is widely considered the 1° cause of OCD of the knee - Adult form of OCD is believed to result from vascular insult

Osteochondritis Dissecans (OCD) (cont)

• **Pathophysiology:**	- Disruption of epiphyseal vessels, leading to ischemia & necrosis at trauma site - Softening, tearing, fissuring, & erosion of hyaline cartilage follow as a consequence of the disrupted blood supply - Advancement of the affected area result in focal demineralisation & repeated shear forces, causing detachment of bone & overlying cartilage - Repetitive axial loading, esp. w/ increased valgus & varus stress, is suggested by experts as a contributing factor tot he condition - OCD lesions can introduce irregularities in the articular surface, potentially leading to degenerative arthritis
• **Clinical presentation:**	- Vague, poorly localised knee px that worsens w/ activity - Stiffness & occasional swelling may occur during or after activity as the disease progresses - Advanced stages may be indicated by **locking or catching**, suggesting the presence of a sizeable loose body in the knee - Hx of trauma, recent increase in activity level, previous knee injuries, & the presence of mechanical Ssx - Approx. 80% of pts report px when WB - Juvenile: intermittent, activity-associated px poorly localised around the anterior aspect of the joint - Adult: more likely effusion, limited ROM, or mechanical Ssx such as 'catching or locking' - Depending on chronicity of the lesion, pts may report quadriceps dysfunction & intermittent knee instability

Osteochondritis Dissecans (OCD) (cont)

• **Physical examination:**	- Genu varus, associated w/ lesions at medial femoral condyle - Genu valgus, associated w/ lesions at lateral femoral condyle - Quadriceps atrophy or weakness may be evident - Foreign body may be palpable - FX of knee during joint palpation can reveal effusion or bony tenderness along the femoral condyles - ROM may be restricted due to px, swelling, or the presence of a loose body - Antalgic gait or lateral rotation of the foot on the affected side may indicate efforts to alleviate WB px **Wilson sign:** - Identifies lesions of the lateral aspect of the medial femoral condyle - +ve test: px w/ INT ROT, relieved by EXT ROT, indicating impingement of the OCD lesion - Absence of the Wilson sign does not rule out OCD
• **Diagnosis:**	- Arthroscopy is the gold standard for assessing lesion stability - Plain radiographs used to locate the lesion, assess growth plates, & rule out other conditions - Initial radiographs may appear normal in OCD **Lesion characteristics:** - Distinct Lucent areas w/ varying density levels - Calcifications & Lucent lines may or may not be present, depending on lesion severity - B comparison **Classification:** - Lesion location can provide important prognostic info - Atypical locations like trochlea or patella may not respond effectively to conservative management **MRI evaluation:** - Useful for assessing unstable lesions presenting w/ mechanical Ssx or knee effusion - Unstable lesions on mRI may exhibit increased T2 signal, destruction of overlying articular cartilage, or multiple cyst-like foci - Gadolinium contrast may be necessary for assessing blood supply & stability uncertainties - Line of high signal intensity between fragment & underlying bone is a sensitive prognosticator

Osteochondritis Dissecans (OCD) (cont)

• **Complications:**	- If left untreated, adults often progress to arthritis - Degenerative articular changes over time - A non-union & dissociation of the bony fragment - Chronic px & mechanical Ssx - Surgical complications inc. postop infection, pneumonia, haemorrhage, & reactions to anesthesia - Venous thrombosis due to immobility
• **Management:**	- Prognosis influenced by age, location & appearance of lesion **Non-operative:** - Recommended for juvenile pts w/o a displaced fragment OR stage 1-3 disease - Immobilisation & protected WB for 4-6 weeks - Physical therapy initiated after immobilisation & continued until pain-free, achieving full ROM, strength, power, & mobility - NSAIDs for px & edema **Operative:** - Surgery recommended if conservative treatment ineffective after 3-6 months or if not suitable - 1° treatment for Ssx related to OCD in adults, stage 2 disease, or expanding lesions on radiographs - Surgical intervention warranted in juveniles w/ stage 4 disease, loose bodies, unstable lesions, or impending physeal closure

Osteochondritis Dissecans (OCD) (cont)

• **Ddx:**

Juvenile:
- Patellofemoral syndrome
- Patellar tendonitis
- Osgood-Schlatter disease
- Sinding-Larsen-Johansson syndrome
- Fat pad impingement
- Symptomati discoid meniscus
- Symptomatic synovial plica

Adult:
- Patellofemoral px
- Knee OA
- Chondromalacia
- Patellar tendonitis
- Meniscal tear
- Fat pad impingement
- Symptomatic synovial plica

Adult w/ more severe Ssx:
E.g. atraumatic edema & mechanical Ssx
- Meniscal tear
- Osteochondral loose body
- Neoplasm

link text

Patellofemoral Pain Syndrome (PFPS)

• **GREEN**

• **Intro:**
- Also known as *Chondromalacia patella (CMP)* & *Runner's knee*
- Softening of hyaline cartilage on articular surfaces of bones
- CMP specifically refers to the softening, tearing, fissuring, & erosion of the patellar cartilage
- Can occur in any joint, but common in joints w/ trauma & deformities

Patellofemoral Pain Syndrome (PFPS) (cont)

• Aetiology (risk factors):

- F>M
- Increased Q angles in F (lateral positioning of the patella)
- No hormonal cause has been identified
- Active young adults (esp. running sports), & workers who stress their patellofemoral joint (stairs, kneeling)
- Often multifactorial
- LL malalignment & patellar maltracking play a significant role
- Foot & ankle variances: pes planus can lead to increased lateral wear of the patellofemoral joint
- Miserable malalignment syndrome, w/ femoral ante version, gene valium, & pronated feet
- Muscular weakness: vests medals & core m.
- Patellar lesions from injuries, immobilisation, or surgical procedures causing quadriceps atrophy
- Abnormal wear & tear of the patellofemoral joint's hyaline cartilage
- Iatrogenic factors: injecting chondrotoxic medications into joints

Patellofemoral Pain Syndrome (PFPS) (cont)

• **Pathophysiology:**	**Pathological process:** - Hyaline cartilage composed of chondrocytes, type 2 collagen, proteoglycans, & water - Avascular w/ nutrients diffusing from synovial fluid - Poor repair due to lack of blood supply, devoid of lymphatic & neural tissue **Factors leading to hyaline cartilage degeneration:** - Destruction by chondrotoxic substances, cytokinins, & proteolytic enzymes - Microtrauma from wear & tear - Repeated compressive stress or increased loads on patellofemoral joint - Aging-related decrease in chondrocytes, proteoglycan production, & water content - Cross-linking of collagen fibrils leads to loss of elastic properties - Superficial zone of hyaline cartilage is the 1st to degenerate in aging process **Px generation:** - Anterior fat pad & joint capsule commonly involved in generating px signals - SUbchondral bone less likely to cause px signals -**Initiation of CMP pathology:** begins w/ softening, swelling, & edema of articular cartilage
• **Clinical presentation:**	- CC: anterior knee px - Pts may report insidious onset of diffuse retropatellar or pre patellar px, exacerbated by activities stressing the patellofemoral joint - Aggravating factors: stair ascending or descending, squatting, kneeling, running, & prolonged sitting (theatre px) - Additional Ssx: effusion, quadriceps wasting, & retropatellar crepitus (not specific to CMP) - Hx evaluation: previous trauma, comorbid conditions, joint stability, foot & ankle issues, & activity levels
• **Physical examination:**	- Px is usually sharp & achy - Examine quadriceps appearance, foot & ankle orientation, & specific evaluation of the patellofemoral joint - Patella malt racking signs: increased femoral anteversion, EXT tibial torsion, lateral patella subluxation, loss of medial patellar mobility - +ve patellar apprehension test - +ve Clark's test

Patellofemoral Pain Syndrome (PFPS) (cont)

- **Diagnosis:**
  - Reliable Dx requires excluding other conditions causing anterior knee px
  - Arthroscopy is the most efficient (invasive, so non-invasive methods essential for initial Dx)
  - Plain radiographs: lower sensitivity in earlier stages
  - CT: measures TT-TG distance & detects torsional deformities of the LL
  - MRI: modality of choice fro articular cartilage, esp. T2 sequence
- **Complications:**
  - 2° to NSAID usage (e.g. GI Ssx)
  - Bracing may cause dermatological reactions
- **Management:**
  - **Min. 12 months** of conservative management before considering surgery
  - May be reversible
  - Could progress to patellofemoral OA
  - Pts often fully recover (can take months to yrs)

  Conservative 1st phase:
  - Activity modification
  - RICE
  - NSAIDs

  Conservative 2nd phase:
  - Knee + hip exercise to increase strength, mobility & function
  - Patella taping
- **Ddx:**
  - Patellofemoral OA
  - Osgood-Schlatter
  - Plica syndrome
  - Bursitis
  - Saphenous neuritis
  - Quadriceps tendinopathy
  - Patellar tendinopathy
  - Referred px from hip/back

link text

Medial Tibial Stress Syndrome (MTSS)

- GREEN
- **Intro:**
  - Early stress injury leading to tibial stress fractures
  - Known also as *shin splints*
  - Common overuse injury in athletes & military personnel
  - Involves exercise-induced px along the anterior tibia

Medial Tibial Stress Syndrome (MTSS) (cont)

• **Aetiology (risk factors):**	- 13-20% incidence in runners - Up to 35% in military - **Factors contributing:** significant increasing loads, volume, & high-impact exercises - **Intrinsic risk factors:** F gender, previous MTSS Hx, high BMI, navicular drop, ankle plantar FX range, hip EXT ROT range - Overuse condition, specifically a tibial bony overload injury w/ associated periostitis - **Common for:** recurrent impact exercise, such as running, jumping, & military personnel - Suggested link between vitamin D & increased risk of stress injury
• **Pathophysiology:**	- Involves accumulation of unprepared micrdamage in the cortical bone of the distal tibia - Overlying periostitis is typically present at the site of bony injury - Periostitis correlates w/ tendinous attachments of soleus, flexor digitorum longus, & posterior tibialis - Sharpey's fibers, perforating connective tissue linking periosteum to bone, play a role in the mechanical connection - Repetitive muscle traction is believed to be the underlying cause of periostitis & cortical microtrauma
• **Clinical presentation:**	- Presence of exercise-induced px along the distal 2/3 of the medial tibial border - Presence of px provoked during or after physical activity, which reduces w/ relative rest - The absence of cramping, burning px over the posterior compartment &/or numbness/tingling in the foot
• **Physical examination:**	- Presence of recognisable px reproduced w/ palpation of the posteromedial tibial border >5cm - The absence of other findings not typical of MTSS (e.g. severe swelling, erythema, loss of distal pulses, etc)
• **Diagnosis:**	- Dx through clinical & physical findings - Imaging done when uncertain about cause or to rule out other exercise-induced LL injuries - Plain radiographs are normal in MTSS & early stress fractures - *"dreaded black line"* indicates a *stress fracture* - MRI is preferred for identifying MTSS & higher-grade bone stress injuries like tibial stress fractures

Medial Tibial Stress Syndrome (MTSS) (cont)

• **Complications:**	- Px leading to decreased performance &/or time away from training/participation - May progress to tibial stress fracture - Severe tibial stress fractures may require surgical intervention
• **Management:**	- Full recovery is expected - Rest & activity modification w/ less repetitive, load-bearing exercise - **Additional therapies:** iontophoresis, phonophoresis, ice massage, US therapy, periosteal pecking, & extracorporeal shockwave therapy - **No benefit:** low-energy laser therapy, stretching, strengthening, LL braces, & compression stockings - **Slow response cases:** optimising calcium & vitamin D status & gait retraining may improve recovery & prevent further progression
• **Ddx:**	- Tibial stress fracture - Compartment syndrome - Functional popliteal artery entrapment syndrome

link text

Varicose Veins

• YELLOW

• **Intro:**	- Characterised by subcutaneous dilated, tortuous veins of ≧3mm - Age & FHx are important risk factors - Common clinical manifestations of chronic venous disease - Evaluating associated superficial axial venous reflux is crucial - Manifestations can range from limited leg discomfort to swelling & non-healing ulcers
• **Aetiology (risk factors):**	- Up to 30% of general population - F>M - **Risk factors:** F gender, multiparty, high BMI, constipation, Hx of venous thrombosis, smoking, & circulating iron levels - Both genetic & environmental factors

Varicose Veins (cont)

• **Pathophysiology:**	- Valve dysfunction leads to increased pressure in veins - Elevated pressure causes vein walls to weaken & dilate - Ironic pressure causes the vein walls to stretch & lose elasticity - Weakened walls contribute to the development of varicosities
• **Clinical presentation:**	- Leg heaviness - Itching - Cramps - Mild tenderness - Skin discoloration - Exercise intolerance - Leg fatigue
• **Physical examination:**	- Visible distended veins from thigh to ankle - Discolouration most prominent around ankle & calf - **Special test:** Trendelenburg test - assesses deep venous valve competency
• **Diagnosis:**	**CEAP classification:** - C0: no visible, palpable signs - C1: spider veins - C2: varicose veins - C3: edema - C4a: pigmentation, eczema - C4b: lipodermatoslerosis - C5: healed ulcer - C6: active ulcer - Colour duplex venous US exam is recommended for suspected venous reflux
• **Complications:**	- Venous ulcers - Pain - Poor cosmesis - DVT - PE (rare) - Superficial thrombophlebitis might be complicated w/ prolonged bleeding & px - Superficial vein thrombosis
• **Management:**	- No cure - Long-term graduated compression stockings, leg elevation, & oral px medication - Surgery (recurrence is likely)

Varicose Veins (cont)

• **Ddx:**	- Lymphedema - DVT - Cellulitis - Dermatological disorders (e.g. stasis dermatitis)

link text

Vasculitis

• **RED**	
• **Intro:**	- Heterogenous group of over 30 different kinds of vasculitis. presenting either as a 1° process or 2° to another pathology - Clinical & pathological manifestation vary based on the affected blood vessels' type & location
• **Aetiology (risk factors):**	- Incidence of 20-40 million / year - Gender dominance depending on type of vasculitis - Giant cell arteritis is the most common form - **Risk factors:** Behcet disease (ancient Silk route), Takayasu disease (South Asian), Kawasaki disease (children <5), hepatitis B/C
• **Pathop-hysiology:**	- Unknown specific cause - Immune system activation: becomes overactive - Inflammation of blood vessels: immune system mistakenly identifies blood vessels as foreign invaders - Attack on endothelium: attacks the endothelium (inner lining of blood vessels) - Adhesion molecules & leukocyte activation: cytokines. signalling molecules in the immune system, cause changes in adhesion molecules on the endothelium → inappropriate activation of leukocytes & their adherence to the blood vessel walls - Vessel damage: combined effect of immune cells sticking to the blood vessel walls & the inflammatory response damages the vessels - Formation of immune complexes or antibodies: different forms of vasculitis may involve the formation of immune complexes or the production of antibodies targeting specific components in the blood vessels - Granuloma formation (in some cases): in certain vasculitis types. granulomas may form. contributing to tissue damage
• **Clinical presen-tation:**	- Ssx dependent on location of vasculitis - Potential organ damage from vasculitis - Fevers, unexplained weight loss - Nose bleeding, hemoptysis, hematuria

Vasculitis (cont)

• **Physical examination:**	- Upper airway disease - Ocular inflammation - Limb claudication - +ve sensory/motor neuropathy, purpura, change of pulses
• **Diagnosis:**	- Chest x-ray or high-resolution CT for respiratory Ssx - Vascular imaging (MRI, MRA, CTA, vascular US, PET) to detect large artery lesions in vasculitis cases -**Labs:** CBC, kidney & liver function, ESR, serologies, & urinalysis w/ urinary sediment
• **Complications:**	- Depend on the type of vessel involved - **Large vessel involvement:** acute MI, stroke, mesenteric ischemia, aortic s., critical extremity ischemia - **Life threatening complications of small vessels:** alveolar haemorrhage, renal failure, intestinal ischemia
• **Management:**	- Long-term survival highly depends on the Dx, response to treatment, & adverse effects of drugs - Managed w/ medication - 3 main components: remission induction, remission maintenance, & monitoring
• **Ddx:**	- Infections, neoplasms & certain drug toxicities can mimic vasculitis - Coagulopathies can present w/ similar Ssx to vasculitis

link text

Venous Thrombo-embolism (VTE)

• **RED**	
• **Intro:**	- Significant complication of hospitalisation - 3rd leading CV Dx, following heart attacks & strokes - VTE encompasses DVT & PE - Growing public health concern - Need for increased awareness among the public & healthcare providers

Venous Thrombo-embolism (VTE) (cont)

• **Aetiology (risk factors):**	- Global annual burden of VTE is in millions - Significant morbidity & mortality associated w/ VTE cases worldwide - Majority of VTE cases are hospital-related or acquired (60%) - Leading preventable cause of death in hospitalised pts - **Risk factor:** >40 yrs, obesity, varicose veins, immobility, oral contraceptive, smoking, hypercoagulability, pregnancy, & pelvic/hip/long-bone fractures - **Disease states ↑ risk:** malignancies, spinal cord injury, nephrotic s., congestive heart failure, IBD, & recent MI
• **Pathophysiology:**	- Venous thrombosis involves the formation of a clot made of platelets & fibrin within blood vessels - Clinically significant thrombi typically form in large-lumen vessels, such as deep veins in the legs, pelvis, & arms - Clots can propagate & extend proximally, leading to clinical Ssx when vascular flow is obstructed - Dislodged clots may embolise to distant sites, w/ the pulmonary vasculature being a common location - Obstruction in pulmonary vascular flow can result in impaired gas exchange, alveolar edema, & pulmonary alveolar necrosis - Chronic repetitive pulmonary embolisation can increase pulmonary vascular resistance, ultimately causing pulmonary hypertension - In the presence of cardiac abnormalities like a patent foramen ovale or atrial septal defect, paradoxical embolism may occur, leading to systemic arterial vascular involvement
• **Clinical & physical findings of DVT:**	- Unilateral limb px is a common complaint - Physical signs may include swelling, warmth, & tenderness to touch - Physical exam signs for DVT have low Dx yield

Venous Thrombo-embolism (VTE) (cont)

• **Clinical & physical findings:**	- Sudden onset of dyspnea (most common presenting complaint) - Pleuritic chest px, cough, & hemoptysis - Massive PE can lead to syncope, hypotension, & shock - Physical examination findings for PE are variable & often nonDx - Tachypnea (resp. rate >18/min) is common - In older pts, new-onset atrial fibrillation may be a presenting Ssx **Established PE physical findings:** - Tachypnea (resp. rate >18/min) is common - Rales may be present in up to 50% of cases - Tachycardia (HR >100/min) & fever occur in about 45% - Diaphoresis & S3 or S4 gallop may be audible in about 30% - Pleural friction rub may indicate peripheral PE w/ pulmonary necrosis
• **Diagnosis:**	- Chest radiography - D-dimer assay - US & serial US for DVT - CTPA & VQ for PE
• **Complications:**	- Bleeding - Heparin-induced thrombocytopenia - Warfarin-induced skin necrosis
• **Management:**	- Treatment is based on associated conditions - 1° treatment is anticoagulation
• **Ddx:**	- Localised Ssx of DVT can be similar to cellulitis, arterial insufficiency, lymphedema, & hematoma - PE: congestive heart failure, acute respiratory distress s., pneumonia, & MI

link text