Muscle Strains

• GREEN	
• **Intro:**	- Muscle / tendon strain is equivalent to ligament sprain in terms of injury type - Happens when muscle fibres are overworked, resulting in fibre tearing
• **Aetiology (risk factors):**	- ↑ incidence in athletes - Commonly occurs when there's sudden ↑ in duration, intensity, or frequency of activity **3 types of muscles at risk:** - **Two-joint muscles:** motion at one joint can ↑ passive tension, leading to overstretching injuries - **Eccentric contractions:**. common during deceleration phase, may change muscle tension & cause myofibril overload injuries - **Muscles w/ ↑ % of type II fibres:** fast-twitch muscles w/ high-speed contractions, making them more prone to injury (running & sprinting) → Hamstrings, gastrocnemius, quadriceps, hip flexors, hip adductors, ES, deltoids, & rotator cuff
• **Pathophysiology:**	- Contraction induced injury caused by extensive mechanical stress - Often occurs due to powerful eccentric contractions or over-stretching of the muscle **Muscle lesions are classified as grade I, II, & III** **Grade I (mild):** - Affect a limited number of muscle fibres - No decrease in strength - Full AROM & PROM - Px & tenderness may be delayed to the next day **Grade II (moderate):** - Nearly half of muscle fibres torn - Acute & significant px - Accompanied by swelling - Minor decrease in muscle strength **Grade III (severe):** - Complete rupture of the muscle - Tendon separated from the muscle belly or muscle belly torn in 2 parts - Severe swelling & px - Complete loss of function

Muscle Strains (cont)

- **Sx & Ssx:**
  - Swelling, bruising, or redness
  - Px at rest
  - Inability to use the muscle at all
  - Weakness of muscle or tendons
- **Management:**
  - **First phase:** protection, rest, ice, compression, elevation (PRICE) & NSAIDs
  - **Second phase:** mobilisation should occur ASAP but gradually & within limits of px, Mobs/Drops, low impact exercises
  - 3-6 weeks for the muscle fibres to recover = full ROM, pain free, & 90% strength bilaterally
  - **Third phase:** proprioceptive & endurance, SMT / STW, TrPs

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AVN / Osteonecrosis

- YELLOW

- **Intro**
  - Degenerative bone condition resulting from the death of bone cells due to disruption in the subchondral blood supply
  - Also known as AVN, aseptic necrosis, & ischemic bone necrosis
  - Typically affects the epiphysis of long bones at WB joints, w/ severe cases potentially causing subchondral bone destruction or joint collapse
  - Common sites include femoral head, knee, talus, & humeral head
  - Less common occurrences in other bones like the carpus & jaw

AVN / Osteonecrosis (cont)

• **Aetiology (risk factors):**	- 30-65 yrs - M>F - Females more at risk w/ PMHx of lupus **6 groups of risk factors:** - **Direct cellular toxicity:** chemo/radiotherapy, thermal injury, smoking - **Extraosseous arterial fracture:** hip dislocation, femoral neck fracture, iatrogenic post-surgery, congenital arterial abnormalities - **Extraosseous venous:** venous abnormalities, venous stasis - **Intraosseous extravascular compression:** haemorrhage, elevated bone marrow pressure, fatty infiltration of bone barrow due to prolonged high-dose corticosteroid use, cellular hypertrophy & marrow infiltration (Gaucher disease), bone marrow oedema, displaced fracture - **Intraosseous intravascular occlusion:** coagulation disorders (thrombophilias & hypofibrinolysis), sickle cell crises - **Multifactorial**
• **Pathophysiology:**	**1.** Reduction in subchondral blood supply **2.** Induces hypoxia **3.** Loss of cell membrane integrity **4.** Necrosis of cells (osteonecrosis)

AVN / Osteonecrosis (cont)

• **Clinical & physical exam presentation:**	**Non-traumatic cases:** - Mechanical px w/ variable onset & severity - Difficult to localise - Normal physical exam in early disease (causing delay in Dx) - **Focused Hx considerations:** recent trauma, steroid use, autoimmune disease, Sickle cell, alcoholism, tobacco use, manual labour, change in gait, connective tissue disorders, insidious onset px, decreased ROM **AVN of the hip:** - Early stages often asymptomatic - Hip & groin px - Late-stage. progression indicated by px at rest - **Associated Ssx:** referred px in buttock & thigh, stiffness, changes in gait **AVN of the knee:** - Acute onset knee px while WB & at night - Typical responses in Hx: osteoporosis or osteopenia, no recent trauma - **Physical exam findings:** px w/ palpation over medial femoral condyle, decreased ROM **AVN of the talus:** - Associated w/ polyarticular disease & trauma - Complaints of px & difficulty ambulating beyond expected recovery time post-trauma
• **Diagnosis:**	**MRI findings:** - Osteosclerotic changes - Decreased bone resorption due to disrupted osteoclast function - Low on T1 (fat is white) - High on T2 (fat is dark)
• **Complications:**	**Postoperative complications:** - Surgical site infection - Prosthesis malfunctions - Neuromuscular compromise

AVN / Osteonecrosis (cont)

• **Management:**	- Pharmacological therapy in early stages - Surgery - Exercises to maintain joint mobility & strengthen muscles around - Later in therapy implement endurance & coordination training - Post-surgery & recovery full conservative care
• **Ddx:**	- OA - Osteoporosis - Osteomyelitis - Neoplastic bone conditions - Inflammatory synovitis - CRPS - Soft tissue trauma

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Bursitis

• **YELLOW**

• **Intro:**	- Swelling or inflammation of a bursa - Bursae are found near bony provinces & between bones, muscles, tendons, & ligaments (approx. 150 facilitate MSK movement) - Bursitis causes the bursa to enlarge w/ fluid, resulting in px w/ movement & pressure - Not all forms of bursitis are due to 1° inflammation, some result from swelling due to a noxious stimulus
• **Aetiology (risk factors):**	- Overuse of the joint - Repetitive strain: picking up & lifting heavy loads - Trauma: falling / bumping against things - Pressure: "student's elbow" & "housemaid's knee" - Bacterial infection: unattended wound (causing septic bursitis) - Other inflammatory disease: e.g. Gout (crystals can form in the bursa & cause inflammation) - Immunocompromised individuals: diabetes, rheumatological disorders, alcoholism, or HIV, are at risk of septic bursitis

Bursitis (cont)

• **Pathophysiology:**	- Bursa is a synovial lining sac - Collapses upon itself until triggered, leading to irritation & filling with synovial fluid - Px occurs when the inflamed bursa is compressed against bone, muscle, tendon, ligaments, or skin - Not all bursitis is linked to an overt inflammatory process - Subacromial bursa examination shows ↑ inflammatory mediators
• **Clinical presentation:**	- ↓ ROM due to px in involved joint - Px with AROM, but not w/ PROM in some cases - Two forms of bursitis: acute & chronic **Acute:** - Caused by trauma, infection, or crystalline joint disease - Pts experience px on palpation of bursa - Px w/ FX, but no px w/ EXT in certain types (e.g. prepatellar & olecranon bursitis) **Chronic:** - Often results from inflammatory arthropathies & repetitive pressure/overuse - Often painless - Bursa has had time to expand to accommodate increased fluid, resulting in significant swelling & thickening of the bursa
• **Physical examination:**	- Evaluate skin for trauma, erythema, & warmth - Temperature increase of 2.2°C over affected bursa compared to unaffected indicative of *septic bursitis* - Deep bursitis may not show tenderness or obvious skin changes - Normal ROM in septic bursitis
• **Diagnosis:**	- **Plain radiography:** recommended w/ Hx of trauma, concern for foreign body, or fracture causing swelling or px - **MRI:** for evaluating deeper bursa - **US:** helpful in differentiating cellulitis from infectious bursitis - **Bursa fluid punction:** can rule out infections

Bursitis (cont)

• **Management:**	**Bursitis w/o infection:**- Most often self-limiting - RICE - NSAIDs - Injections - Mobs - Gradual ↑ in exercise - Immobilising is a risk towards adhesive capsulitis **Septic bursitis:** - Antibiotics - Aspiration (needle) - NEVER inject w/ steroids - Surgical removal of bursa (in case of tuberculous bursitis) - Surgical incision & drainage
• **Ddx:**	- OA - RA - Can mimic other conditions in specific locations (e.g. shoulder - rotator cuff / labral tear) - Pathologies can coexist w/ or precipitate bursitis (e.g. gout) - Ischial bursitis can mimic sciatica (sitting-induced px distinguishes it from sciatica) - Trochanteric bursitis differs from ITB syndrome, w/ tenderness in IT band more distal compared to proximal location of trochanteric bursa - Iliopsoas bursitis can resemble arthritis, overuse injuries, synovitis, labral tears, or AVN - Knee bursitis typically doesn't cause effusion, aiding in differentiation from other knee pathologies - Retrocalcaneal bursitis may initially resemble achilles tendinitis, enthesopathy, px from bone spurs, or plantar fasciitis

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Calcific Tendonitis

• **GREEN**

• **Intro:**	- Self-limiting disorder characterised by deposition of calcium in the tendon / muscle - Leads to px & reduced ROM

Calcific Tendonitis (cont)

• **Aetiology (risk factors):**	- 30-50yrs - F>M - Occupational risk (construction, agriculture, certain sports) - Metabolic conditions (e.g. diabetes) - Mechanical stress: repetitive microtrauma or overuse of tendons - Vascular factors: poor blood supply (reduced clearance of metabolic waste) - Genetic predisposition
• **Pathophysiology:**	- Repetitive trauma → tendon degeneration → calcification - Tendon necrosis → intracellular calcium accumulation - Active process mediated by chondrocytes arising from metaplasia → calcium deposition - Phagocytosis of metaplastic areas reforms normal tendon
• **Clinical presentation:**	- Px w/ or w/o loss of ROM - Stiffness, usually after periods of inactivity (morning) - Swelling - TTP
• **Physical examination:**	- Limited ROM - Crepitus - Muscle weakness - Warmth & redness - Palpable calcium deposits
• **Diagnosis:**	- **X-ray:** identify calcifications in the tendon or adjacent soft tissue - **US:** visualise extent & characteristics of calcifications & assess tendon thickness - **MRI:** may be used to evaluate soft tissue involvement & inflammation
• **Complications:**	- Chronic px - Tendon rupture - Compression of adjacent structures - Bursitis - 2° OA - Functional impairment - Psychosocial impact - Recurrence

Calcific Tendonitis (cont)

• **Management:**	- Pts w/ chronic calcific tendonitis often don't respond to conservative care - Anti-inflammatory NSAIDs (ibuprofen) - Injections - Surgery
• **Ddx:**	- Adhesive capsulitis - Tendinopathy - Bursitis - Arthritis - Ossifying tendinitis

Osteoarthritis

• **GREEN**

• **Intro:**	- Non-inflammatory, degenerative joint disease - Characterised by loss of articular cartilage & marginal hypertrophy of bone - Accompanied by px & stiffness that is aggravated by prolonged activity - Most prevalent type of arthritis
• **Aetiology (risk factors):**	- Higher age - F>M - Hx of joint trauma - Obesity - **1° OA:** most common subset of OA; absence of predisposing trauma or disease - **2° OA:** occurrence w/ pre-existing joint abnormality; Ssx - Modifiable environmental factors: repetitive movements, obesity, metabolic syndrome, smoking, vitamin D deficiency, muscle weakness, low bone density - Commonly affects hands, knees (most common), feet, facet joints, & hips

Osteoarthritis (cont)

• **Pathophysiology:**	- Multifactorial & involves 3 major processes: mechanical degeneration (wear & tear), structural degeneration, & joint inflammation - Overuse & aging of the joint are believed to be the main contributors, but inflammatory processes indicated by ↑ cytokines are also present - Firstly, OA involves cartilage damage, including surface fibrillation, irregularity, & focal erosions - Then, cartilage damage prompts chondrocyte proliferation, & outgrowths can ossify, forming osteophytes - Later, subchondral bone sclerosis & bone cyst formation occur, potentially increasing joint stiffness & px - Advanced OA may lead to episodic synovitis, & in rare cases, bony erosions can occur in erosive OA
• **Clinical presentation:**	- Joint px worse w/ use & improves w/ rest - Px peaks in late afternoon or early evening, also present in the early morning **Two types of px:** - Dull, aching, throbbing px (predictable & constant over time) - Intense, unpredictable px for short periods **Classified into three stages based on px types:** - **Early OA:** sharp, predictable px limiting high-impact activities - **Mid OA:** constant px, unpredictable joint px or locking, affecting ADLs - **Advanced OA:** constant dull-aching px w/ intermittent intense episodes, limiting recreational activities **Additional joint Ssx:** - Tenderness, stiffness, crepitus - Limited ROM - Joint swelling, deformity, or instability

Osteoarthritis (cont)

• **Physical examination:**	- Bony enlargement (commonly in DIP & PIP joint of fingers & toes) - Crepitus - Effusions (non-inflammatory) - Joint line tenderness - Limited ROM due to px, swelling, or joint deformity **Specific bony enlargements:** - *Heberden's nodes:* posterolateral bony swelling of DIP joints - *Bouchard nodes:* posterolateral bony swelling of PIP joints - OA involving the base of the thumb is described as a "shoulder appearance" or "squaring"
• **Diagnosis:**	**Plain radiographs to grade OA:** - **0:** no OA - **1:** Doubtful narrowing of joint spaces &/or possible osteophytes - **2:** Definite osteophytes & possible narrowing of joint spaces - **3:** Multiple osteophytes, definite narrowing of joint space & some sclerosis & deformity of bone ends - **4:** Large osteophytes, marked narrowing of joint space, severe sclerosis & definite deformity of bone ends
• **Complications:**	- Chronic px - Long-term analgesics use - Reduced joint mobility - Decreased stability & increased fall risk - Joint malalignment - Deformity - Stress fractures - Hemarthrosis - Osteonecrosis - Joint infection - Gout & pseudogout - Depression
• **Management:**	- Exercise program - STW - Mobs/drops - SMT - NSAIDs (preferably topical)

Osteoarthritis (cont)

• **Ddx:**	- RA - Gout - Pseudogout - Septic arthritis - Hemochromatosis - Fibromyalgia - Lyme disease - Ankylosing spondylitis - Psoriatic arthritis - Neuropathic arthropathy - Parvovirus-associated arthritis

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Inflammatory arthropathies

• YELLOW

• **Intro:**	- Painful inflammation & stiffness of the joints - Inflammatory arthritis is typically associated w/ classic Ssx of inflammation - Can have various factors - inc. infectious / non-infectious factors - Inflammatory arthritis may or may not be associated w/ systemic features related to the underlying condition causing the inflammation
• **Aetiology (risk factors):**	- Increasing age - F > M (autoimmune inflammatory arthritis) - M>F (gout & seronegative spondyloarthritis) - Smoking (strongest environmental factor) - Caucasian, FHx - W/ juvenile idiopathic arthritis presenting before 10 yrs of age

Inflammatory arthropathies (cont)

• **Pathophysiology:**	- Varies depending on the underlying etiology - External or self-antigens trigger an immune-mediated inflammatory response - Inflammatory cells migrate from the bloodstream into synovial membrane - Hyperplasia of synovial fibroblasts is associated with inflammatory response - Cartilage & bone damage can occur, leading to joint destruction in some cases - Infectious arthritis results from the direct invasion of the joint by infectious organisms - Infectious agents may trigger an immune response, leading to inflammatory arthritis - Autoimmune inflammatory arthropathies involve an interplay of environmental & genetic factors activating the immune system - Crystalline arthropathies involve crystals in the synovial acting as antigens, triggering a neutrophil-mediated inflammatory cascade
• **Clinical presentation:**	- Disease progression can be chronic & progressive, leading to joint damage, deformity, & disability if left untreated - Joint px - Joint stiffness - Joint swelling - Warmth & redness - Fatigue - Malaise - Low-grade fever - Inflammatory arthritis often follows a pattern of flares & remissions
• **Physical examination:**	- Inflammatory eye conditions: uveitis or iritis, common in various types of IA - Skin rashes: commonly in connective tissue diseases (systemic lupus or psoriatic arthritis) - Enthesitis: inflammation in the sites where tendons & ligaments insert into bones (common in seronegative spondyloarthropathies) - Dactylitis: swelling of an entire digit (common of certain spondyloarthropathies) - Symmetry: common in RA

Inflammatory arthropathies (cont)

• Diagnosis:

Labs:
- Elevated inflammatory markers: erythrocyte sedimentation rate (ESR) & C-reactive protein (CRP)
- Positive autoantibodies: such as rheumatoid factor (RF) & anti-cyclic citrullinated peptide (anti-CCP) antibodies in RA

Plain radiographs:
- Initially normal in early inflammatory arthritis
- May show periarticular osteopenia at the disease progresses
- Periarticular erosions are seen in inflammatory arthritis like RA
- Gout erosions are typically juxta-articular/rat-bite erosions w/ overhanging edges
- Chondrocalcinosis or CPPD deposition can be easily visualised
- Axial spondyloarthropathies can later show 'bamboo' spine & SIJ function & erosions

MRI:
- Beneficial, especially when radiographs are nondiagnostic
- More sensitive than plain film in evaluating synovitis, erosions, sacroiliitis
- Higher sensitivity than X-rays

• Complications:
- Arise from delay in treatment or mass Dx
- May lead to aggressive & permanent joint damage
- Associated conditions w/ joint damage: chronic gout, RA, seronegative spondyloarthritis
- Erosive changes in joints
- Interference w/ ADLs

• Management:
- Inflammation is reversible, while joint destruction is not
- Antibiotic therapy
- NSAIDs
- Education
- Manual therapy
- Diet
- Lifestyle modification

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Inflammatory arthropathies (cont)

- **Ddx:**
  - RA
  - AS
  - Psoriatic arthritis
  - Lupus arthritis
  - Gout
  - Juvenile idiopathic arthritis
  - Reactive arthritis
  - Spondyloarthritis

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Septic arthritis

- **YELLOW**
  - Same day referral
- **Intro:**
  - Inflammation of joints 2° to an infectious etiology
  - Usually monoarticular, however polyarticular also occurs
  - Although rare, septic arthritis is considered an orthopaedic emergency

Septic arthritis (cont)

• Aetiology (risk factors):

In children:
- Diverse casues
- Staphylococcus is the 1° pathogen
- Kingella king affects children under 2-3 yrs
- Neonates face streptococcus, staphylococcus, gonorrhea
- Adolescents: gonorrhoea concern
- Salmonella links w/ sickle cell disease
- Prolonged antibiotic use raises fungal infection risk
- Pseudomonas from puncture wounds/drug use
- Hip joint commonly affected in children

In adults:
- Staphylococcus major in adults
- Streptococcus pneumonia significant
- Gonorrhoea causes non-traumatic mono-arthritis
- Fungal/mycobacterial organisms challenging to Dx
- SCJ/SIJ infections involve pseudomonas, common in IV drug abusers
- Damaged joints, especially in RA, prone to infection w/ cartilage damage, effusions, & px

Epidemiology:
- Incidence peaks at 2-3 yrs & elderly
- M>F (2:1)

Risk factors:
- Age >80
- Diabetes mellitus
- RA
- Recent joint surgery
- Joint prosthesis
- Previous intra-articular injection
- Skin infections & cutaneous ulcers
- HIV infection
- OA
- Sexual activity (gonorrhoea)

Septic arthritis (cont)

• **Pathophysiology:**	- Bacterial invasion of synovial, followed by inflammatory processes - Common pathogens change throughout lifetime (viz. aetiology) - Damaged joints through RA are highly susceptible to infection - Synovium lacks limiting basement membrane → systemic infection can spread to bones - Contagious spread from osteomyelitis (hip & shoulder are prone)
• **Clinical presentation:**	**In children** *Local Ssx:* - Px, joint swelling, warmth - Limited ROM - Limp, refusal to use or move the affected joint (pseudoparalysis) *Systemic Ssx:* - Ill appearance - Fever - Tachycardia - Fussiness/irritability - Decreased appetite **In adults:** - Acute onset monoarticular (large joints) joint px - Fever (40-60% of pts), swelling, reluctance or refusal to move the affected joint - LL (hip, knees, ankles) commonly affected - Knee most affected joint
• **Physical examination:**	- Palpation may elicit px - Limited ROM - Effusions are common

Septic arthritis (cont)

• **Diagnosis:**	- Synovial WBC count - ESR & CRP **Imaging** *Plain radiographs:* - May reveal widened joint spaces - Soft tissue bulging - Subchondral bony changes (late finding) - Normal plain radiograph doesn't rule out septic arthritis *US:* - Useful for identifying & quantifying joint effusion - Aids needle aspiration *MRI:* - Sensitive for early detection of joint fluid - Reveals abnormalities in surrounding soft tissue & bone - Cartilaginous involvement
• **Complications:**	- Osteomyelitis - Chronic px - Osteonecrosis / AVN - Leg length discrepancies - Sepsis - Death
• **Management:**	- Same day referral to GP - Antibiotics - Aspiration of joints
• **Ddx:**	- Infections - CPPD - OA - Fractures - AVN/osteonecrosis - Other inflammatory arthropathies - Systemic infections - Tumour

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Tendinopathies

• **GREEN**

• **Intro:**	- Tendinopathy is an umbrella term to describe tendon px, w/ unknown cause - **Tendinosis** describes the degenerative state of tendons (more applicable)

Tendinopathies (cont)	
• **Aetiology (risk factors):**	- Not fully understood - Mechanical stressors, repetitive overloading, or toxic chemical exposure ca initiate tendinosis - Age, genetic predisposition, &/or comorbidities can increase susceptibility to healing failure leading to tendinosis
• **Pathophysiology:**	- Decreased simplistically in 3 stages, but actually occurs on a continuum **Stage 1:** - Begins w/ tendon experiencing the initial insult, stress, or injury - Causes: acute overload, repetitive stress, or chemical irritation - Linked to the death of tenocytes (tendon cells) **Stage 2:** - Characterised by failed healing of the tendon - Unclear cause, but believed to result from an altered tendon environment (steroids/NSAIDs may alter natural healing cascade) - Improper cell recruitment & a cascade of healing issues may occur **Stage 3:** - Characterised by apoptosis (death) of cells, disorganisation of the matrix, & neovascularisation - Pts often present at this stage, experiencing mechanical weakness or increased px - Neovascularisation theorised to supply neonerves, contributing to px (neurogenic inflammation)
• **Clinical presentation:**	- Identify potential stressors - Impact on ADLs - Recent changes in medication, inc. antibiotics (may influence treatment)
• **Physical examination:**	- Tenderness - Swelling - Other abnormalities - Special tests based on the specific tendon
• **Diagnosis:**	- Labs: CRP & ESR (aid in identifying inflammatory processes) - **X-ray:** if bone injury is suspected - **US:** increased spacing of fibrillar lines, reduced echogenecity, tendon thickening, neovascularisation (via colour Doppler) - **MRI:** valuable for evaluating tendinosis

Tendinopathies (cont)

• **Complications:**	- Tendon rupture (if untreated) - Contractures of the tendon, w/ reduced tendon liability - Tendon adhesions - Atrophy of muscles - Loss of functionality, even up to & including disability
• **Management:**	- Healing as long as 3-6 months - STW - SMT - Mobs/drops - RICE - NSAIDs
• **Ddx:**	- Acute compartment syndrome (ACS) - Ankle injury - Bursitis - Carpal tunnel syndrome - Gout & pseudogout - Hand infections - Reactive arthritis - Rotator cuff injuries - Soft tissue knee injury

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LL nerve entrapments / Tunnel syndromes

• **GREEN**

• **Intro:**	- Umbrella term for conditions characterised by compression or entrapment of nerves, blood vessels, or tendons within anatomical tunnels in the LL - Can lead to various Ssx inc. px, numbness, tingling, & weakness, affecting the function & sensation of the LL **Common LL tunnel syndromes inc.:** - Sciatica - Tarsal tunnel s. - Common peroneal n. entrapment - Anterior compartment s. (ACS) - Popliteal artery entrapment s. (PAES)

LL nerve entrapments / Tunnel syndromes (cont)

• **Aetiology (risk factors):**	- Age-related degeneration - Obesity - Sedentary lifestyle - Trauma - Arthritis - Tumours - Diabetes - Pregnancy - Tight clothing - Occupation-related factors
• **Pathophysiology:**	- Increased pressure within confined spaces leads to nerve or vascular compression - Inflammation, fibrosis, or space-occupying lesions contribute to compression - Repetitive use or trauma can exacerbate Ssx - Individual anatomical variations may predispose individuals to specific tunnel syndromes
• **Clinical presentation:**	- Px or discomfort in the affected LL - Numbness or tingling - Weakness in the muscles of the affected area - Altered sensation or hypersensitivity - Px may worsen w/ specific activities or movements - Swelling in the affected area - Impaired coordination or balance
• **Physical examination:**	- Tenderness or px upon palpation of the affected nerve pathway - Muscle atrophy in severe or chronic cases - Limited ROM in affected joint - Positive **Tinel's sign**: tingling or px elicited by tapping on the nerve - Positive **Nerve tension tests**: straight leg raise / slump test to assess nerve mobility & irritation - Changes in reflexes, such as diminished or exaggerated reflexes
• **Diagnosis:**	- **MRI**: provides detailed soft tissue visualisation, helping identify nerve compression & surrounding structures - **US:** useful fro dynamic imaging, assessing nerve movement during various joint positions - **CT:** may be used in specific cases, especially fro bony abnormalities contributing to nerve compression

LL nerve entrapments / Tunnel syndromes (cont)

• **Complications:**	- Nerve compression & damage - Ischemia (reduced blood supply) - Muscle atrophy & weakness - Chronic px - Motor dysfunction - Sensory abnormalities - Functional limitations - Trophic changes (skin, hair, nail condition) - Risk for 2° injuries (risk of falls) - Psychosocial impact - Surgical complications
• **Management:**	- Rest & activity modification - NSAIDS & neuropathic px medication - Injections - Occupation & ergonomic modifications - Weight management - Surgery - Pt education - SMT & STW - Strengthening & mobilising
• **Ddx:**	- Peripheral neuropathy - Lx radiculopathy - MFPD - Complex regional px s. (CRPS) - Tarsal tunnel syndrome - Morton's neuroma - Stress fractures - Compartment s. - MSK injuries (ligament sprains, tendonitis etc.) - Inflammatory arthropathies (RA etc.) - Infectious causes (cellulitis, osteomyelitis etc.) - Neoplastic conditions (tumours) - Systemic conditions (diabetes, hypothyroidism etc.) - Iliotibial band s. - Popliteal entrapment s.

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Stress fractures

• **RED**

• **Intro:**	- Fractures that occur due to an imbalance between the bone strength & the chronic mechanical stress placed upon the bone (→ overuse s.) - *spondylolisthesis* is also classified as stress fracture

Stress fractures (cont)

• Aetiology (risk factors):

- Abrupt increase in activity or training patterns

Intrinsic factors:

- Poor physical conditioning
- F>M
- Hormonal disorders
- Menstrual disorders
- Poor bone density
- Reduced muscle mass
- Genu valgum kness
- Short leg

Extrinsic factors:

- High-impact sports
- Abrupt increase in physical activity
- Irregular or angled running surface
- Poor footwear
- Running shoe wear older than 6 months
- Vitamin D & calcium deficiency
- Smoking

Common risk factors:

- Abrupt increase in activity
- Females
- PMHx of stress fractures

Common sites:

- Metatarsals, tibia, tarsals, femur, fibula, & pelvis (in decreasing order)
- Pelvic & metatarsal stress # common in females
- UL stress # rare but reported in gymnasts, weightlifters, & throwing sports

Sports-specific risks:

- Runners: tibia & metatarsal stress # (F may also experience pelvic stress #)
- Long-distance runners: association w/ femoral neck & pelvic injuries
- Hurdlers: patella #
- Gymnasts, female soccer players, certain American football positions & weightlifters: increased risk of spondylolysis (unique stress # related to repeated hyperEXT of the spine)

Stress fractures (cont)

• **Epidemiology:**	- Stress # are 20% of all sports injuries - Common along military - Runners (16% of injuries): tibia (23.6%), tarsal navicular (17.6%), metatarsals (16.2%), femur (6.6%), pelvis (1.6%) - F>M - Neuromuscular factors play a role (muscle loss / fatigue decreases ability to absorb forces) - Rapid weight loss, particularly muscle loss, is associated w/ stress # - Overtraining / relative energy deficiency s. contribute (esp. in females w/ disordered menstruation & hormonal imbalances) - Male endurance athletes w/ high training volumes & restricted calorie intake (low testosterone), leading to osteoporosis & stress #
• **Pathophysiology:**	- Wolff's law: applied force on a normal bone leads to remodelling for increased strength - Osteocytes: most common bone cells, orchestrate osteoclastic & osteoblastic functions - Osteocyte's dendritic network: responds to biomechanical stress, secretes mediators regulating bone activites - Cycling loading impact: compromises osteocyte singling, hinders physiological repair mechanisms - Repetitive loading effect: stimulates osteoclasts for faster resorption, outpacing osteoblasts in new bone formation - Normal remodelling cycle: takes 3-4 months
• **Clinical presentation:**	- Insidious onset of px after activity - Progressive increase in px duration post-exercise - Px present upon waking up following training activity - Important factors in Hx: recent changes in training, nutrition, intrinsic & extrinsic risk factors, PMHx, & medications

Stress fractures (cont)	
• **Physical examination:**	- Focal TTP - Occasional edema at the suspected stress fracture site - Limited clinical diagnostic tests - Px percussion & vibration - Commonly used tests: "hop test" & fulcrum test - Bony tenderness on palp, often at distal to middle third junctions I the tibia or over 3rd & 4th metatarsal shafts
• **Spondylolysis & spondylolisthesis:**	- Require high index of suspicion - *Spondylolysis* may be asymptomatic & found incidentally on Lx films - Lx extension increases px in *spondylolysis*, Stork test or single leg hyperextension test is common - *Spondylolisthesis* occurs when pars defect does not heal, leading to anterior migration of the vertebral body
• **Diagnosis:**	- First is plain radiography - CT - MRI
• **Complications:**	- Small for low-risk stress fractures - Occasional residual px - Higher for high-risk stress fractures - More likely to progress to non-union & thus require surgery
• **Management:**	- NSAIDs - Splinting - Resting / non-WB - Supplementation of vitamin D, magnesium - Potential surgery - Post-surgery / healing rehab - Strengthening /mobs

Stress fractures (cont)

- **Ddx:**
  - Cellulitis
  - Osteomyelitis
  - Tendonitis
  - Tendinopathy
  - Exertional compartment syndrome
  - Tumours (benign & malignant)
  - Nerve entrapment
  - Arterial entrapment
  - Coagulation disorders
  - Compartment s.
  - Neuropathic px
  - CRPS

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