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Congenital Deformities

Talipes Equinovarus

- Heel in equinus and hindfoot in varus, with forefoot adducted and supinated
- 1-2 per 1000 births; male twice as common, and 33% bilateral

Cause unknown:
- Postural deformity from overcrowding in uterus
- Neural tube defects (meningomyelocele)
- Genetic halted development

Clinical features:
- Obvious at birth with foot twisted inwards and soles facing each other
- Calf may be abnormally thin
- Normal babies can dorsi-flex ankle until toe touches shin – reduced ROM in affected neonates; or can be fixed deformity
- Always examine to exclude CDH or spina bifida

- Pirani Score (max 6)
  - Midfoot – lateral border, medial crease, TNJ reduction
  - Hindfoot – posterior crease, fixed equinus, empty fat pad sign.

X-ray:
- 30 degree AP film
- Kite’s angle is angle between medial border of talus and lateral border of calcaneus (normal is 20-40, reduced in talipes)
- On lateral film, the talo-calcaneal angle (between long axis of talus and lower border of calcaneus) should be 40 degrees in any position. <20 degrees shows the calcaneus cannot be tilted up into true dorsi-flexion

Conservative rx:
- Within 48 hours of birth, manipulate and strap into correct position
- Teach parents to repeat stretching while strapping in place
- Monitor (with physio) as strapping can be altered as correction obtained, or use weekly POP casts to hold.
- Manipulation should over-correct (C A V E)
  - Increase supination deformity to bring forefoot into alignment with more supinated hindfoot
  - Reduce both hindfoot and forefoot out of varus and supination
  - Dorsi-flex the foot
  - Start by correcting cavus by lifting metatarsals and increasing supination. Then correct adduction by ab-ducting the foot. Continue abduction until extreme hyper-abduction which unlocks the calcaneus and corrects hindfoot varus. Finally address equinus by Achilles tendon tenotomy (1 to 1.5 cm above incision percutaneously).
- Percutaneous Achilles tendon lengthening may be required – heals by gap healing in the young with pleuripotent cells forming new tendon as opposed to scar tissue.
Casting protocol:
- 3 weeks in cast
- 3 months in boots and bars full time
- 3-4 years night time bracing.

Surgery is usually deferred for 6 months or until child is walking, as this allows for bigger bones to aid surgery, and walking itself helps maintain correction:
- Posterior incisions (Turco, Cincinatti-Crawford, or Caroll)
- Achilles tendon and Tibialis posterior lengthened with z-divisions
- Division of posterior capsules of ankle and subtalar joints
- Lengthening of FDL and FHL sometimes needed
- Release of calcaneo-fibular ligament with complete subtalar release
- Superficial part of deltoid ligament released, but deep part preserved to maintain ankle stability
- If a K-wire is used to maintain reduction, it is removed at 6-8 weeks

Recurrent cases that fail to respond to soft tissue release, can be treated with a protracted regime using the Ilizarov frame.
- Alternative include: repeat casting for 6-8 weeks to obtain abduction, repeat tenotomy, or tibialis anterior transfer to lateral cuneiform.
**Metatarsus Adductus**
- Varies from mild curvature of forefoot to a mild club foot
- 90% resolve spontaneously, or managed using serial corrective casts.
  - Actively correctible – demonstrate by stroking lateral border of foot
  - Passive correctible – consider serial casting
  - Fixed deformity – consider surgery
- Severe deformity requires surgery:
  - Casulectomy of TMT-joints with splintage risks early arthritis
  - Variations of Dilwyn-Evans procedure (which aims to balance the lengths of the medial and lateral columns of the foot) can be combined with basal MT-osteotomies
  - Release of abductor hallucis.
- 1 in 20 risk of 2nd child being affected
- Always check for DDH – other tight/packaging deformities

**Talipes Calcaneovalgus**
- Acute dorsi-flexed foot in newborn
- Deep crease in front of ankle (cf. congenital vertical talus where crease is over midfoot)
- Flexible deformity (cf. fixed in vertical talus)
- Usually bilateral, but unilateral cases associated with CDH
- Usually due to abnormal uterine position, and correct spontaneously.
- Severe cases require serial cast correction.

**Flat foot in Children**
- Tip-toe test confirms a mobile subtalar joint and tibialis posterior function
  - Tibialis posterior will plantar flex the ankle and invert at the subtalar joint
  - Isolate by placing foot in plantar-flexed, everted position. Ask patient to invert against resistance in this position (to stop tibialis anterior masking a weakness)
- 90% of under-2s will have flexible pes planus, which gradually corrects between ages 3-5. This is due to natural joint hyper-mobility, and the broad-based gait with the mechanical axis falling slightly medial to the 1st or 2nd ray. By age 10, only 4% will have persistent flat foot.
- Assessment should include:
  - Is there a tight TA – seen if the heel is not flat during stance phase
  - Gastrocnemius tightness – assess TA in flexion and extension; tiptoe gait?
  - Family history
  - Signs of hyperlaxity
- Painful flatfoot in child:
  - Congenital vertical talus
  - Tarsal coalition
  - Tumour
  - Foreign body
  - Accessory navicular
Normal lateral x-ray of foot will show a straight line through the long axis of the talus, navicular and 1st MT. Sagging at the talo-navicular joint indicates a flat foot:
- <15° = mild
- 15 to 40° = moderate (associated with lateral subluxation of the navicular off the head of the talus)
- >40° = severe

**Vertical Talus**
- Usually bilateral with medial arch not simply flat, but most prominent part of sole resulting in a rocker-bottom appearance
- Hindfoot in valgus with talus pointing vertically
- Forefoot then abducts, pronates and dorsiflexes
- Usually a fixed deformity
- On x-ray the talus is vertical with the navicular dislocated dorsally. Always repeat lateral film with foot plantarflexed → in flexible flat foot, the navicular returns to normal position
- Treatment = surgery < 2 year old
  - Achilles tendon lengthened + capsulotomies of ankle and subtalar joints
  - Reduction of talonavicular joint with transfer of tibialis anterior tendon to neck of talus
  - Lateral structures may need to be lengthened or released
  - K-wire transfixes talonavicular joint (removed at 6 weeks)
  - 8-12 weeks POP

**Flexible flat foot**
- Normal in toddlers and usually resolves within a few years when medial arch development is complete
- Arch restored by dorsi-flexing the hallux (Jack test) perhaps by putting the FHL tendon onto a stretch which supports the medial longitudinal arch?
- Usually re-assure and no treatment needed – insoles reduce shoe-wear by changing pattern of weight bearing and do not help the feet

**Stiff Flat foot**
- Consider congenital vertical talus, an inflammatory joint disorder, tarsal coalition or a neurological disorder

**Compensatory flat foot**
- Occurs to accommodate another postural defect (e.g. tight tendo Achillis), or if the legs are externally rotated & the body weight falls anteromedially (Charlie Chaplin)

**Accessory Navicular:**
- Tenderness over medial prominence – with x-ray showing an extra ossicle
- Symptoms relate to a bursitis and pressure over the area
- Bone scanning is used to exclude infection or osteoid osteoma, but can also identify a “hot” accessory navicular.
- Excision by shelling out ossicle from Tibialis Post tendon, and then hitching it to the navicular, by looping suture around it and then passing it through 2 drill holes in the navicular, tying it on the dorsal surface of the navicular → this hitches up the medial longitudinal arch.

**Kohler’s Disease**
- AVN of navicular.
**Tarsal Coalition**

- Can result in a variety of partial or complete unions that represent a failure of segmentation of the tarsal bones: talo-calcaneal, calcaneo-navicular and talo-navicular.
- Incidence 1%; bilateral in 50% of those affected.

- Painful stiff flat foot accompanied by spasm of extensor tendons and peroneal tendons, as patients try to compensate and correct overall hindfoot alignment (*peroneal spastic flatfoot*)

- Autosomal dominant condition, with an initial fibrous syndesmosis maturing into cartilage and later into a bony bar.
  - Usually happen in puberty or early adolescence, as the cartilaginous interface matures into a rigid bony structure
  - Symptoms may only start with trauma that fractures bar, or from abnormal tarsal stress

- Treatment:
  - Initial 6 weeks conservative mx with plantigrade walking POP cast, as that the condition may be an incidental finding and not the root cause of symptoms
  - Calcaneo-navicular bars can be excised with a piece of muscle (EDB) interposed to prevent recurrence in children
  - In adults – also need to shorten the lateral column
  - Talo-calcaneal bars are more difficult, and it may be wiser to wait until after puberty to perform a triple arthrodesis.

**Flat Foot In Adults**

- Causes:
  - Idiopathic flexible deformity with ligamentous laxity
  - Disorder of tibialis posterior tendon
  - Tight TA or Gastrocnemius contracture
  - Inflammatory arthritis
  - Primary midfoot arthritis or midfoot fracture malunion
  - Calcaneus or cuboid fracture malunion
  - Tarsal coalition
  - Degenerative arthritis
  - Neuropathy or central disorder of muscle balancing (with peroneal spasm)
  - Secondary to a coronal malunion/angulation of knee, tibia or ankle

- Flat foot is the end point of collapse of the medial arch, with progressive hindfoot valgus. Peak pressures in the peritalar joints lead to increasing subluxation dorsally and laterally of the talonavicular joint (+ navicular-cuneiform and 1st MTC joints).

- The medial longitudinal arch formed by the 1st metatarsal, medial cuneiform, navicular and talus
  - Static support:
    - Spring ligament (superomedial calcaneo-navicular ligament + inferior calcaneo-navicular ligament)
- Superficial fibres of deltoid ligament which insert onto SMCN ligament
- Plantar fascia (25% of support)
- Long plantar ligament (anteroinferior surface of calcaneus to cuboid, with superficial fibres splitting to insert onto 2nd to 4th MT bases)

- Dynamic support:
  - Tibialis posterior – balanced by antagonistic actions of tibialis anterior, peroneal brevis and triceps surae
  - Long & short toe flexors – fail without an intact tibialis posterior
  - Tibialis posterior will invert the heel prior to heel raise in gait cycle. This moves Achilles tendon vector medial to subtalar joint, and its subsequent contracture will lock the bony contour of the medial arch during heel raise.

- Just 1cm lengthening of Tib Post renders it dysfunctional and no longer will lock the Chopard Joint (TN joint) to allow force to be transmitted from calf muscle to the forefoot. This force then remains within the midfoot resulting in midfoot sag and pronation. Abduction of the forefoot leads to the *too many toes* sign from unopposed action of Peroneus brevis (antagonist to Tib Post)

- Thus failure can be traumatic or degenerative affecting the osseous components (TN joint and medial cuneiform), laxity or trauma of the ligaments, and trauma or inflammatory affecting the tendons.

- Asymptomatic flexible flat foot → no treatment required or restriction of activity

- Symptomatic flexible flat foot – often related to sub-fibular impingement
  - Shoe arches and muscle strengthening exercises

- Symtomatic Stiff Flat Foot:
  - NSAIDs and good footwear
  - Immobilisation in walking plantigrade POP cast for 6-8 weeks
  - Arthrodesis for post-traumatic lesions or tarsal OA
  - If associated with Tibialis Posterior synovitis → steroid injection and splintage wth lateral iron and medial T-strap
  - Tibialis Posterior rupture → repair (young), splintage (elderly) or triple arthrodesis (severe pain).
  - NICE Guidelines – new treatment option is percutaneous sinus tarsi implant screw, to jack out the lateral side and move hindfoot out of varus. Must be done as part of a clinical trial. Usually in teenagers.

**Posterior Tibial Tendon Dysfunction (PTTD)**
- Tenosynovitis and progressive inflammation, leading to weakness or frank rupture.
- Leads to medial laxity, with hindfoot valgus and forefoot abduction.
- Separate tendon sheath behind medial malleolus with multiple insertions to adduct the hindfoot and supinate the forefoot.

- **Stage 1** = tenosynovitis
  - Pain along tendon, and maintained strength
  - Impingement in its separate fibro-osseous tunnel; worsenend by an accessory navicular
  - Repetitive micro-trauma ± hypovascular zone 4cm proximal to MM
  - Still able to perform single tiptoe
- conservative immobilisation
- Avoid steroid injection as 25% risk of tendon rupture
- Prevent progression with tenosynovectomy

Stage 2 = flexible with inflamed and elongated tendon (dysfunctional)
- Tendon pain with reduced strength and hindfoot valgus
- Foot still supple with good ROM
- “flexible” even though tendon non-functional
- Elderly – solid plastic ankle foot orthosis, or orthotic management with medial counter to support TN-J or medial heel wedge.
- Young – Tibialis posterior tendon debridement with FDL tendon transfer and extra-articular osteotomy to correct bony alignment (medial calcaneal slide or lateral column lengthening)
- 2A – can still perform heel raise
  - PTT reconstruction with FDL transfer or split Tib Anterior transfer (Cobb)
  - Also repair or reconstruct the incompetent spring ligament
- 2B – incompetent tendon
  - add in the bony calcaneal osteotomy

Stage 3 = rigid
- Subtalar arthritis with gastrosoleus tightness causing equinus and valgus
- If conservative orthoses fail, may consider selective or triple hindfoot arthrodesis
- Whereas in flexible deformity the orthosis is corrective, here in fixed deformity the orthosis is accommodative with cushioning. (e.g. solid AFO – articulated)

Stage 4 = rigid + tibiotalar joint involved
- If orthotic treatment fails (e.g. solid AFO – non articulated), will need pan-talar fusion (ankle arthrodesis and triple arthrodesis)

Pes Cavus
- Aetiology:
  - Muscle: muscle dystrophies Duchenne
  - Peripheral nerve: hereditary neuropathy HSMN I, II
  - Spinal cord: viral disease, structural polio, tethered cord, syringomelia
  - Brain: congenital Friedreich’s ataxia, CP
  - Post-traumatic: compartment syndrome Volkmann’s contractures

Clinical features
- Age 8-10
- Usually bilateral with high arches and toes drawn into clawed position
- Unilateral cavus is a red flag for a spinal pathology
- Metatarsal heads forced down ± callosities on sole
- Giving way of ankle due to varus of hindfoot.
- Tripod analogy:
  - Normal = relation between calcaneus, 1st and 5th metatarsal
  - Plantaris = then both rays are drawn towards heel
  - Cavovarus = 1st ray alone pulled towards heel
  - Calcaneus = heel is pushed plantarwards
- Calcaneo-varus = heel pushed plantarwards, and 1st ray pulled in to heel

- Initially flexible deformity with hyperextension of MTPJ, subluxation of MT-heads and clawing of toes → become fixed

- Pain on lateral aspect of foot, over 5th MT head, and even ankle instability with severe hindfoot varus.

- X-rays
  - Meary’s angle normal = 0 degrees (angle between axis of talus and 1st MT)
  - Calcaneal pitch normally 10 to 30 degrees from horizontal
  - Both increased in pes cavus

- Treatment:
  - Non-operative options such as custom made shoes, and moulded inserts treat sx, but do not alter disease progression. This will also act as a shock absorber as the rigid foot can no longer do this alone.
  - Flexible deformity can be treated with a lateral heel counter, or a medial bar and lateral T-strap to pull the heel out of varus.
  - Surgical aim is to provide a pain-free, plantigrade, supple but stable foot:
    - Soft tissue release
      - Lengthening of Achilles tendon, and posterior capsulotomy
      - Release of plantar fascia for cavus foot (in children only)
    - Osteotomies (for fixed deformity and to avoid fusion)
      - Lateral closing-wedge calcaneal osteotomy to correct varus
      - Correct cavus with 1st MT dorsiflexion osteotomy
    - Tendon transfers if joints are mobile
      - Tibialis anterior transfer from base of 1st MT to calcaneum
      - EHL from anterior surface of fibula to hallux distal phalanx, used as a sling through the neck of 1st MT, to elevate it
      - Peroneus longus runs from superior 1/3 of fibula to 1st MT-base and medial cuneiform – splitting it and attaching it to peroneus brevis will reduce pull on 1st MT and improves eversion
  - Correction of claw toes by Robert Jones tendon transfer
    - Fusion of IP joint with transfer of EHL from distal phalanx to neck of 1st MT

- Late options for fixed deformity = triple fusion (never for asymptomatic)

**Osteoarthritis**

- **Ankle**
  - Primary is rare (7%), and usually secondary to trauma (70%) or improper placement of foot onto floor
  - Pain with disability usually quite severe – as severe as hip arthritis in terms if debility (Glazebrook M 2008, JBJS-Am)
  - Options:
    - Ankle-foot orthosis to improve stability and relieve stress, with rocker-bottom sole.
Arthrodesis is the gold standard – in plantargrade position, 5° external rotation (matched with contralateral side), and 5-10° valgus
- Small movements may still be permitted via sub-talar and talo-navicular joints even after fusion, but patients will not be able to run/jump.
- However, they can walk without a limp at a normal pace; a few have returned to recreational sports levels with bilateral ankle fusions
- Arthroscopic has high union rates, as long bracing not removed early
- Open fusion if previous arthroscopic failure or if cannot enter joint
- Union rates 90-95%, with good pain relief and satisfaction
- Better for: a young patient, high demand, pre-existing stiffness, poor soft tissues, deficient bone stock, previous infection, major deformity

Arthroplasty reserved for low demand patients:
- c/i: infection, neuropathic joint, (obesity, bone loss, muscle paralysis, significant joint incongruity)
- may give better movement, without transfer-arthralgia to neighbouring joints.
- Need to have a satisfactory ROM pre-operatively (will not change post-operatively, simply preserved)
- Polyarthritic patients do well, as it prevents overload onto adjacent joints associated with fusion
  - But these patients tend to have more deformity, so beware as early failure with deformity.
  - May need to have corrective hindfoot fusions and then a staged ankle replacement.

Hindfoot
- Again more commonly secondary OA from previous trauma (particularly intra-articular calcaneal fracture)
- Symptoms of restricted movement, pain and possible lateral impingement vs. fibula.
- Talo-navicular arthritis can lead to talus falling into a plantar-medial position, resulting in hindfoot valgus and forefoot abduction to compensate alignment → acquired flat foot.
- Options:
  - Ankle-foot orthosis with the trim-line adjusted to allow 50% ankle joint movement – provides support to hindfoot while allowing smoother gait.
  - Selective arthrodesis: subtalar (50% reduced ROM), talonavicular (85%), calcaneocuboid (35%).
  - Triple arthrodesis offers close to 100% movement restriction, and therefore reduces pain and offers better union rates (less movement)
  - Subtalar joint fixed in 5° valgus, and any lateral impingement beneath fibula excised. TN & CC joints fused in neutral position to avoid any pronation-supination
**Midfoot**
- Can be either primary or secondary to a Lis-Franc fracture dislocation
- Pain
- Progressive forefoot abduction secondary to collapse
  - loss of longitudinal arch.
  - painful prominence on plantar-medial side of 1st MTC joint
- Options:
  - AFO brace cut to allow ankle movement, but with a long foot-piece of limited use
  - Intra-articular steroid injections – diagnostic & therapeutic value
  - TMT-articulations may need to be fused, correcting the abduction and dorsiflexion deformity caused by collapse and restoring the bony arch.
  - Often still have stiffness
  - Advised not to engage in high-impact sports.

**Ankle Instability**
- Medial ligament (Deltoid) is broad and strong with a superficial and deep layer – rarely affected
- The lateral ligament has two distinct components:
  - Anterior talo-fibular ligament – a horizontal thickening of ankle capsule that prevents anterior translation of the talus within the mortise
  - Calcaneofibular ligament – oblique from tip of fibula, deep to peroneal tendons and crossing the sub-talar joint to insert onto the calcaneus, to prevent inversion.
- A cavus foot results in loss of the physiologic 5° hindfoot valgus, and moves mechanical axis medial to centre of ankle joint → tensioning the lateral ligaments.
- Static stabilisers = ligaments
- Dynamic stabilisers = peroneal muscles which reflexively contract in response to sudden inversion.
- Recurrent instability associated with peroneal muscle tears and osteochondral lesions of the talus (osteochondritis dissecans)
- Treatment options:
  - Physiotherapy to strengthen and improve proprioception, can even compensate ligament laxity
  - Broström repair – direct repair of native ligament by imbrication into a small bone trough around the front of the fibula.
    - 15% failure rate associated with benign hypermobility (e.g. mild type III Ehlers-Danlos)
  - Augmented reconstruction if failure or presence of hyperlaxity using: peroneus brevis, fascia lata, hamstring tendon, or tendon allograft.
**Hallux Valgus**

- Any person who wears shoes the hallux will go into a degree of valgus

- Splaying of foot with varus of 1st MT (metatarsus prima varus), increases risk of hallux valgus at MTPJ. This causes the sesamoids to become uncovered (“subluxed”) as the intermetatarsal ligament between the 2nd MT-head and sesamoid remain a constant length. There is contracture of the lateral capsule and adductor hallucis.

- Causes:
  - Tight shoes
  - Pes Planus ??
  - Any imbalance that causes weight to fall on planta-medial aspect of hallux on ambulation → attrition of medial capsule and contracture of lateral capsule and adductor hallucis.
  - Hypermobility of 1st TMT ??
  - FHx in 60% (Hardy & Clapham 1952) in juvenile; 40% in adolescents

- Deformity involves lateral deviation of the hallux at the MTPJ, with prominence of the medial side of the 1st MT neck (bunion)
  - Results in crowding of toes ± over-riding
  - When angle >30-40°, big toe starts to pronate so nail faces medial, and sesamoids subluxes laterally.
  - Bursa or thickened skin over bunion ± osteoarthritic changes

- Patient group = women age 50-70, and usually bilateral.
  - Can occur in younger sub-group often with familial tendency

- Clinical features:
  - Deformity
  - Pain – pressure or inflamed bunion
  - Metatarsalgia
  - Deformities of lesser toes (crowding, hammer deformity)
  - Secondary OA of 1st MTPJ
  - Shoe restriction 80%

- Examination should include:
  - Correctible
  - ROM
  - Friction test = axial grind test
  - TMT instability
  - Assess for TA tightness
  - Need to ensure NV ok – 20% will note numbness, but usually 40% on Semmes-Weinstein assessment (dorsal cutaneous nerve of hallux)

- Standing x-rays:
  - Normal inter-meta-tarsal angle = 9°
  - Hallux Valgus angle at 1st MTPJ normal < 15°
  - DMAA <9° normal – to assess joint congruence and usually preserved in juvenile forms
1 = congruous (stable)
parallel and centred joint surfaces
2 = deviated (unstable)
surfaces are centred but not congruent / parallel
3 = subluxated (unstable)
neither congruent nor centred
Degree of deformity on pre-op x-ray fails to correspond with severity and AAOS-scores can have co-existing OA

- Treatment options
  - Try conservative measures first in young patient group, as they have a 20-40% recurrence rate, especially for cosmetic complaints as opposed to pain.
  - Wide and deep toe box, with soft uppers and low heel
  - **Mild** = IMT angle 9-11°, hallux valgus angle < 15-30°
    - Congruous → Chevron (risk of AVN of head of MT) or Mitchell (risks transfer metatarsalgia)
      - Chevron osteotomy is a V-shaped cut in the MT-head, allowing up to 5mm lateral displacement.
    - Incongruent → distal soft tissue re-alignment (Modified McBride: medial capsule plication, lateral capsule release & abductor hallucis tenotomy)
    - Note: combination of Chevron osteotomy with soft tissue re-alignment can strip the 1st MT head of 70% of its blood supply (Kuhn 2005). Greatest insult (45% blood supply loss) is from medial capsulotomy.
  - **Moderate** = IMT 12-18° and hallux valgus angle 30-40°
    - Congruous → SCARF + Akin osteotomy of proximal phalanx
    - Incongruent → distal soft tissue realignment + basal osteotomy (lateral closing wedge) and bunionectomy.
  - **Severe** = IMT >18°, HVA >40° → basal osteotomy

- Severe osteoarthritis changes:
  - Arthrodesis of 1st MTPJ in a slight extended position
  - Kellers removal of proximal third of proximal phalanx leaves a floppy toe

- 1st MTC joint instability
  - Assess clinically by stabilising lateral forefoot, and moving 1st ray, then assess on lateral weight-bearing film of foot by looking for dorsal angular incongruence – translation of >50% height of 1st MT-head against 2nd indicates instability
  - Lapidus procedure – fusion of 1st MTC-J in a position that corrects IMA, with combined distal soft tissue release.

- Juvenile Bunion
  - Congenital maldevelopment of joint, and not due to footwear
  - Present since adolescence
  - If associated with short thumbs, consider fibrodysplasia ossificans → short thumbs, bunion & torticollis common;
  - Associated with:
    - Metatarsus primus varus
    - Flexible flat foot (50%)
    - Long 1st metatarsal
    - Oblique 1st MTC joint
• Increased distal metatarsal articular angle (DMAA) >10°, with congruent deformity
• Spasm of abductor hallucis (e.g. in cerebral palsy)
  - Beware converting a congruent deformity into an incongruent deformity during correction, resulting in secondary arthritis
  - Closing wedge modification to Chevron Osteotomy ± proximal procedure if severe.
- **Hallux Valgus Interphalangeus** – closed wedge osteotomy of base of proximal phalanx (Akin procedure)
- Often patients will have relative long 2nd metatarsal with chronic hallux valgus, which can be exacerbated by corrective osteotomies. This can result in transfer metatarsalgia onto 2nd MTP-J, and shortening of the 2nd metatarsal may have to be considered to fully rebalance the forefoot.
- Surgery has a recurrence rate of 60%, therefore many surgeons wait until growth plate closed.

**Hallux Rigidus**

- **Aetiology:**
  - Primary degeneration = OA
  - Secondary:
    - Local trauma
    - Osteochondritis dissecans of 1st MT head in young
    - Gout, pseudogout in elderly
  - Equal sex distribution (in contrast with Hallux valgus)

- **Symptoms:**
  - Pain - worse on walking on slopes or uneven paths
  - Callosity on medial side of distal phalanx
  - Dorsal osteophyte over 1st MTPJ
  - Restricted dorsiflexion at 1st MTPJ
  - Compensatory hyper-extension at the IPJ
  - Increase wear on outer side of foot, as a result of rolling the foot outwards to avoid pressure on the big toe
  - *Rule out polyarthropathy of other joints*

- **X-ray:**
  - Grade 1 = dorsal osteophyte with maintained joint space
  - Grade 2 = dorsal joint space narrowing, with proliferation around dorsal spurring
  - Grade 3 = entire joint space narrowed, osteosclerosis, and large osteophytes.
  - “squaring” of 1st MT head from old osteochondritis

- **Management:**
  - Non-operative = rocker shoe
  - MUA and steroid injection used for Stage 1 disease only, with only 6 month of relief, and 1/3 needing further surgery.
  - Surgery:
    - Simple extension osteotomy of proximal phalanx in young (check there is reasonable plantar flexion before doing this)
    - Cheilectomy in older patients with excision of dorsal 20% of metatarsal head and osteophytes to restore dorsi-flexion; even 30° makes a difference in sx.
- Joint replacement with silastic prosthesis – complications include silastic synovitis, fracture of implant, and osteolysis; salvage difficult. Lifespan of replacement is only 5 years, therefore not for younger patients. Often active movement impaired and loss of passive stability, as the intrinsic muscles cannot be securely re-attached.
- 1st MTPJ arthrodesis – especially if patient engages in strenuous activity; joint fused in 10° valgus and 10-15° dorsi-flexion. Complications include non-union, and misalignment resulting in transfer of load onto IP-joint.
**Deformities of Lesser Toes**

### Claw Toes
- Flexion of IP joints with hyper-extension of MTPJ common
- Can be an “intrinsic-minus” deformity from neurological causes
  - peroneal muscle atrophy – peroneus longus and brevis both have a weak plantar flexion action in addition to eversion.
  - Polio
  - Peripheral neuropathies
  - Associated with pes cavus
- **Sx:**
  - Visible deformity
  - Bilateral commonly with restriction of activity
  - Pain under metatarsal heads, with callosities on sole and corns on dorsal surfaces of toes.
- **Treatment:**
  - Flexible
    - Metatarsal support or transverse metatarsal bar
    - Tendon transfer of flexor to extensor
  - Fixed
    - Special footwear with high toe box
    - PIPJ arthrodesis and dorsal capsulotomy of MTPJ – if no arthritic changes;
      sometimes combined with transfer of EHL to 1st MT to remove a deforming force, while maintaining its action as a forefoot stabiliser.
    - Excision of MTP-joints – if arthritic changes; common in Rheumatoid

### Hammer Toe
- Flexion of PIPJ with extension of MTPJ and DIPJ
- Corns can appear on dorsum of toe from shoe pressure
- Possible extensor dysfunction (looks similar to Boutonniere deformity)
- **Treatment:**
  - Surgery if painful or for difficulty with shoes
  - Surgery = PIPJ excision
    - Ellipse of skin with corn and underlying extensor tendon removed
    - Nibble away joint surfaces, and transfix with K-wire for 6 weeks
    - Sometimes just the head of the proximal phalanx is excised, or no K-wire used with simple splintage for 3 weeks to allow healing
    - Dorsal capsulotomy of MTPJ may be needed to allow toe to align straight with its neighbours.

### Mallet Toes
- Flexion of DIPJ, with normal straight MTPJ and PIPJ
- Painful plantar callosity as toe presses down into shoe
- **Mx:**
  - Chiropody and padding
  - Excision arthrodesis with K-wire for 6 weeks ± flexor tenotomy.
5th Toe Deformities
- Overlapping 5th toe:
  - Straighten with dorsal V/Y plasty
  - Hold in place for 6 weeks with tape or K-wire
  - If refractory/severe → Lapidus procedure to transfer long extensor tendon beneath proximal phalanx onto abductor digit minimi
- Cock-up – dislocation of 5th MTPJ with little toe sitting on dorsum of head
  - Longitudinal plantar excision and removal of proximal phalanx
  - Longitudinal wound closed transversely to pull little toe out of extension.
- Tailor’s Bunion
  - Bunionette over 5th MT-head
  - IMA > 8 degrees, and 5th MTP angle > 10 degrees is abnormal.
  - Type 1 = bunionette with prominent lateral eminence → eminence resection and lateral capsule release ± medial release
  - Type 2 = bunionette with bowed metatarsal shaft → distal chevron or metatarsal shaft osteotomy
  - Type 3 = bunionette with increased IMA → varus osteotomy avoiding proximal metaphyseal-diaphyseal junction where blood supply is tenuous
  - Elevation of the metatarsal during fixation of the osteotomy, or distal condylar resection at the 5th MT-head will address any plantar keratosis

2nd MTP Instability
- Normally stabilised by plantar plate with its firm attachment to proximal phalanx and relatively good attachment onto the 2nd MT-neck. This is predominant static block to dorsal subluxation, but up to 50% of restraint is also provided by collateral ligaments.
- Mechanical overload causes initial pathology, due to long 2nd MT, hypermobile 1st ray, hallux valgus / rigidus, equinus deformity, or reduced ankle movements.
- Initial dorsal subluxation triggers cascade of synovitis and inflammation, which causes pain and erodes and stretches the collaterals and plantar plate.
- Plantar plate attenuation or acute rupture causes progressive subluxation.
- Extension at the MTP joint results in weakening of action of EDL and EDB at the IP joints resulting in clawing. Attenuation of the collateral ligaments may also lead to medial deviation of the 2nd toe and a cross-over deformity.
- Neuralgic pain in the 2nd web space can mimic a neuroma, as the digital nerves are functionally stretched as they run under the inter-metatarsal ligament.
- Pathognomic examination is a positive anterior draw test on the 2nd MTP joint.
  - 1 mild – slight
  - 2 moderate – 50% subluxable
  - 3 severe – dislocateable.
- Treatment options:
  - Strapping or splinting with a metatarsal pad to unload MT-head
  - Stiff soled rocker bottom sole or walking cast boot
  - Steroid injections risk further rupture → protect joint for minimum 2 weeks
  - FDL flexor to extensor transfer – can lead to over stiffness
  - Metatarsal shortening osteotomy if too long (e.g. Weil’s horizontal osteotomy)
  - Extensor lengthening with plantar-condylectomy, and 3 weeks pinning
Osteochondritis Dissecans of Talus

- Unexplained pain and slight reduced ROM at the ankle in a young person
- Precipitant injury often forgotten
- X-rays – tangential views of talus/mortice in different degrees of flexion
  - Common small osteochondral fragment in antero-medial or postero-lateral corner of talar dome
- MRI or arthroscopy also useful for confirming diagnosis (CT will not show a cartilaginous defect)
- Treatment:
  - Initial restriction of activities if articular cartilage intact
  - Once softened, arthroscopic drilling can help
  - Loose bodies may need removal (arthroscopic / open)

Atraumatic osteonecrosis of Talus

- Causes:
  - (Traumatic – fracture to talar neck)
  - Inflammatory – infection, osteomyelitis
  - Neoplastic – paraneoplastic syndrome
  - Circulatory – heart failure, Sickle, Caisson's
  - Autoimmune – SLE
  - Pulmonary - COPD
  - Metabolic – Gaucher’s
  - Endocrine – Diabetic, hypercortisonism (Cushings)
  - Drugs – steroids
  - Degenerative
  - Iatrogenic – alcohol
  - Psychogenic – nil
- Pre-collapse
  - Analgesia and restriction of activity
  - Core decompression trial if still symptomatic
  - If fails → ankle arthrodesis
- Collapse:
  - Arthrodesis.

Rheumatoid Arthritis

- Stage 1 = synovitis of MTPJ, IPJ and ankle + tendon sheaths of peroneal muscles and tibialis posterior
- Stage 2 = joint erosion and tendon dysfunction
- Stage 3 = progressive deformity
- The foot can be the first presenting symptom in 17%

Forefoot

- Swelling of MTPJs are early signs
- Patient complains of uncomfortable shoes and reduced walking distance
  - Extends to entire forefoot
- Progressive weakness of intrinsic muscles + joint destruction leads to:
  - Flattened anterior arch
  - Hallux valgus
Claw toes
- Prominence of MT-heads (walking on pebbles)
- s/c nodules may ulcerate, or callosities break down

X-ray shows osteoporosis and per-articular erosion of MTPJ
- 4th and 5th toes affected first (in contrast with hand)

Stage 1 = steroid injections and attention to footwear ± synovectomy
- Wide toe box with "functional foot orthosis" with a minimum of 12mm poron insole.

Stage 3 = treat hallux valgus and claw toes
- Otherwise excision arthroplasty ± modified Keller’s on great toe ± arthrodesis of great toe
  - Stainsby procedure is excision arthroplasty with division of EHL proximal to MTPJ, then resection of proximal 2/3 of PP with freeing up of fat pad. Stabilise with K-wire and suture EHL to FHL to ensure fat pad position restored.
- Following 1st MTP fusion, foot can be placed in –ve Camber shoe (no POP) to minimise impact on function
- Often combined with excision of MT-heads of lesser toes, which are left with a K-wire spike to hold alignment
- Wounds often look poor initially but eventually heal, with a delayed improvement of the 1st and 2nd inter-metatarsal angle

Helal osteotomy – a long oblique osteotomy of the metatarsal shaft. No fixation, but allows a prominent metatarsal head to rise up on weight bearing – surprising good results.

No need to stop steroids and methotrexate pre-operatively, but need to stop biologic agents (e.g. infliximab) up to 4 weeks prior to surgeon and only re-start 2 weeks later when wound healed – always discuss with rheumatologist

Ankle & Hindfoot
- Swelling and tenderness localise early to the medial malleolus and lateral malleolus
  - Tenosynovitis of tibialis posterior and peronei respectively
  - Lateral synovitis of ankle can occur
  - Plantar fasciitis
- Painful inversion/eversion
- Valgus hindfoot
  - Tibialis posterior rupture
  - Peroneal spasm
  - Patient weight and muscle tone
  - Ligament damage
  - Subtalar joint damage or is it the ankle?
  - Talar head dislocation - 2° problem
- Tibialis posterior can rupture unnoticed leading to tarsal erosion + drift into valgus position
  - Rheumatoid Pes Planus
    - loss of longitudinal arch
    - valgus hindfoot - single tiptoe testing fails to correct
    - abduction of forefoot - talus projects medially and weight bears, leading to ulceration and infection  → best treated early with triple fusion
- X-ray shows erosion of tarsal joints and ankle

- Treatment:
  - Splintage during synovitis + systemic medical treatment
  - Steroid injections (max 3 times)
  - Light-weight below knee calliper to restore stability
  - Synovectomy
  - Tendon replacements of no use, as persistent erosion likely
  - Arthrodesis of ankle and tarsal joints in late stages can abolish pain, favouring modest function improvement

- Ankle replacement in RA – avoid in young, over-active, heavy or pre-existing deformity. Choose a fusion instead.

**Gout**

*Podagra* defines the common presentation of tumor, rubor, calor and dolor seen in the MTP joint of the hallux. But the ankle joint can equally be affected – especially after innocuous trauma, with a clinical presentation that mimics septic arthritis. However aspirate cultures are negative, with findings of crystals. Plantar fasciitis under the heel is another common manifestation.

- Uric acid metabolism:
  - Formed by breakdown of purines, by xanthine oxidase
  - Undergoes glomerular filtration, with proximal tubule reabsorption and distal tubule secretion.
  - DT secretion blocked by ↓ dose NSAIDs
  - PT reabsorption blocked by ↑ dose NSAIDs (greater effect)

- Causes:
  - ↑ Turnover of purines (25%)
    - Myeloproliferative disorders (PCV, AML, CGL, ET, myelodysplasia)
    - Lymphoproliferative disorders (CLL)
    - Psoriasis
  - ↑ Production of purines
    - Lesh-Nyan X-linked HGPRT deficiency
    - Phosphoribosyl pyrophosphate (PRPP) synthetase deficiency
    - Glucose-6-phosphate deficiency
  - ↓ Renal excretion (75%)
    - Renal failure
    - Drugs – diuretics, pyrazinamide, ↓ dose aspirin
    - Hyperparathyroidism
    - Hypothyroidism (myxoedema)
    - Down’s syndrome
    - Lactic acidosis (alcohol, starvation, vomiting, toxaemia of pregnancy)

**Achilles Tendon**

**Peritendinitis**
- Athletes, joggers and hikers – local irritation of paratenon
- Small lump above heel may be palpable
Conservative treatment: RICE, heel raise, ultrasound
Steroid injection may precipitate rupture
Surgery for refractory pain:
- Resection of inflamed / degenerate tissue ± tendon reconstruction if too much removed.

**Acute Rupture**
- Most are over 40 years – as usually only occurs if tendon degenerate
- 20% risk of subsequent contra-lateral rupture
- Sudden calf muscle contracture + resistance by body weight → rupture
  - Pushing off in sprinters or playing squash/badminton
  - Pain like being kicked in the back of the leg or ankle
  - Unable to tip toe
  - Negative Simmond’s test (calf squeeze) – absent of chronic rupture
- Palpable gap over tendon ± swelling or fluctuance

**Risks:**
- Weekend warrior
- Fluoropquinolones
- Systemic illness with inflammatory arthritis, renal disease or hyperthyroid

**Differential diagnosis:**
- Partial tear (rare) – usually misdiagnosed because late presentation allows palpable gap to be masked by swelling
- Tear of soleus muscle – tenderness half way up calf;
  
  rx = physio + heel raise

**Treatment:**
- If seen early can try equinus cast for 8 weeks, and then a shoe with heel raise for further 6 weeks
  - Confirm diagnosis on MRI with assessment if gap closed by apposition
  - Newer treatment with initial equinus cast followed by air-cast boot offers lower re-rupture rates of 6% - early weight bearing through stimulation of tenoblasts and better alignment of scar tissue
- Surgery for high demand patients
  - Risks:
    - 4% infection
    - 3.5% vs. 12% re-rupture with surgery; 3-30% risk of complications
    - Skin / wound break down
    - Adhesions and stiffness (8-10%)
    - Insensitivity around scar (damage to sural nerve)
  - Same protocol and duration of immobilisation
  - Air-cast boot or lockable brace to allow ankle movements, but heel raises block/reduce tension on suture line
- Late presentation:
  - Reconstruction with tendon substitutes (e.g FDL)

**Heel Pain**
- Aetiology:
  - Mechanical – plantar fascia or disorder of fat pad
  - Inflammatory – RA, sero –ve spondyloarthopathy, Reiters, Ank Spond
- Neurologic – Baxter’s nerve, tarsal tunnel, diabetic peripheral neuropathy
- Localised bone lesion
- Peripheral Ischaemia

- **Differential diagnosis should include:**
  - Calcaneal stress # – if pain on squeezing heel, get bone scan
  - Tumour
  - Infection
  - Plantar fibromatosis
  - Flexor Hallucis Tendinosis

**Posterior Heel Pain**

- **Traction Apophysitis (Sever’s Disease)**
  - Occurs in boys age 10 = mild traction disorder
  - Localised to insertion of Achilles tendon
  - X-ray reports increase density and fragmentation of apophysis – but often painless heel look similar
  - Conservative rx: avoid strenuous exercise, wear heel raise, NSAIDs

- **Non-insertional Achilles tendinitis**
  - In mid-substance of tendon 2° reduced vascular supply
  - Associated with fluoroquinolones, steroids and smoking
  - Aetiology: repetitive strain, overuse or hyper-pronation of foot
  - Stage 1 = pain after activity
  - Stage 2 = pain during and after activity
  - Stage 3 – rest pain
  - May be a fusiform swelling over heel, and generally tender
  - USS better to look at paratenon, and exclude chronic rupture
    - Allows dynamic testing compared with MRI
  - Conservative:
    - Reduce activity, rest
    - NSAIDs
    - Heel raise
    - Physiotherapy – stretching, eccentric loading, muscle strengthening
    - USS therapy
  - Surgery:
    - Incise paratenon and remove adhesions
    - Longitudinal incisions of tendon to stimulate inflammation and healing
    - Excise portion of tendon; requires augmentation if > 1/3:
      - Peroneus brevis
      - FHL preferred – phasic muscle with redundancy, and close proximity
      - but reduced power of “push off”

- **Insertional Tendinitis**
  - Associated with pre-tendon bursa, retro-calcaneal bursitis / spur or Hagland deformity (bony prominence on postero-lateral aspect)
  - X-ray for bony anomalies + MRI to identify site of bursitis
  - Conservative mx
    - Activity reduction, NSAIDs and heel raise
    - New treatments include: shock-wave treatment, and Platelet-GF
  - Surgery:
- Debride bursae
- Excise any bony spurs
- Tendon detachment requires use of anchors or augmentation
- Keep in POP in neutral position or use air-cast boot

**Calcaneal Bursitis**
- Older women complain of this usually due to high heel shoes
- c/o painful bumps on the back of the heel
- Conservative treatment as above, with padding of the heel
- Surgery may improve cosmesis, but unpredictable results in respect to pain.

**Inferior Heel Pain**

**Calcaneal bone lesions**
- stress fractures – confirm with MRI and treat with cast boot.
- osteomyelitis
- osteoid osteoma
- Paget’s disease

**Plantar fasciitis**
- Pain under the ball of the heel; common in men age 30-60
- Pain present throughout day while weight bearing, but 1st steps are worst
  - Foot tends to curl/flex during sleep, which relaxes plantar fascia
  - Walking in the morning stretches fascia and causes pain = start up pain
- Signs:
  - Tenderness along distal edge of heel contact area where long plantar ligament attaches
  - Localised pain over medial calcaneal tuberosity (proximal fascia)
  - Pain on passive DF of toe (if not present consider Baxter’s nerve)
- If bilateral may be associated with other inflammatory disorders (e.g. gout, Reiter’s ankylosing spondylitis)
- Investigations:
  - X-ray may show heel spur – 50%, may be a traction lesion of plantar ligament of FDB
  - Rule out other causes with blood tests, bone scan, electrophysiology and USS/MRI
- Conservative rx = 90% successful if trialled for 6 months (max 1 year)
  - Begin plantar fascia and gastrocnemius stretching several times per day
    - 83% success at 12 months, 98% success at 18 months
    - high level athlete needs to take time out of training
  - wear shoes with good support or use prefabricated orthotic
  - NSAIDs ± steroid injection
    - risks heel pad atrophy or fascia rupture
    - 50% recurrence rate within 5-8 months
  - Night splints; below-knee weight bearing cast for 4 weeks
  - shock wave therapy
    - stimulates angiogenesis, bone formation and cytokine diffusion
    - disrupts calcific deposits
- Surgery is 90% effective in those who fail conservative trial
  - Release 1/3 plantar fascia origin + decompress Baxter’s nerve
  - ± removal of heel spur, and keep in POP for 4-6 weeks

**Painful Fat Pad**
- Tender directly over the fat pad, under the heel
- May follow direct blow (e.g. fall from height)
- Athletes may suffer if fat pad separates from bone, or loses its normal shock-absorbing property
- Conservative mx – heel cups, foot baths, NSAIDs

**Nerve entrapment**
- Compression of 1st branch of lateral plantar nerve in between abductor hallucis and quadratus plantae muscles
- Medial sided heel pain mimicking plantar fasciitis **but** no pain on passive dorsiflexion of toes.
- Trial of conservative measures for 6-8 months (maximum 2 steroid injections), before surgical decompression.
- Tarsal Tunnel syndrome considered later.

**Cavus Foot Pain**
- Mechanical pain from pressure on heel and MT-heads in a pes cavus
- Relieved by heel padding and building up in-step to distribute pressure away from heel.
- Osteotomy usually does not give good results.
Diabetic Foot

- Risks of diabetes to the feet:
  - Peripheral vascular disease
    - Poor skin or nail quality, with claudication
    - Smooth, cold skin with poor/absent pulses
    - Superficial ulceration under toes, and deep under heel (painful)
    - distal vessel occlusion → dry gangrene; proximal occlusion → wet
  - Damage to peripheral nerves (neuropathy – 30% prevalence in diabetics)
    - Loss of proprioception (spinocerebellar), and vibration/temperature/pain sense (spinothalamic)
    - Symptoms a consequence of loss of protective sensation
    - Motor loss manifests with high arches, claw toe (intrinsic loss) and foot drop → plantar ulceration (insensate)
    - Related to duration of diabetes and glycaemic control
    - Distal, symmetrical and progressive
    - Usually sensory with autonomic dysfunction as well
    - Gold standard assessment with 10g Semmes-Weinstein monofilament to test for sensation on applying to foot with a pressure that causes the filament to bend.
  - Reduced resistance to infection
    - Impaired leucocyte function
    - Prone to skin penetration as insensate
    - Local ischaemia can make it an immune-protected site
  - Collagen abnormality from increased glycosylation on cross-links – results in tight TA and plantar-flexed foot which overloads the forefoot.
  - Osteoporosis
    - Generalised and can result in insufficiency fractures in ankle or metatarsals

- Stages:
  - Foot at risk – patients with ischaemia, neuropathy or deformity
    - Education – footwear, foot care (moisturise), regular follow-up
  - Acute problems – critical ischaemia, ulcer, infection, Charcot
  - Healed foot – still at risk of further injury

- Neuropathic joints can be caused by (1) diabetes, (2) leprosy and (2) tertiary syphilis
  - Usually triggered by a twisting injury or fracture
  - Progressive painless joint collapse, and rapid destruction of articular surface
  - Can look similar to infection, but affect simultaneously several small joints in the absence of systemic illness

- Confirming osteomyelitis:
  - X-ray
  - Repeat at 4 weeks
  - MRI
  - Bone biopsy (gold standard – from different approach)

- Management
Treatment goals: stable and plantargrade, ulcer free and shoeable/braceable with function

Multi-disciplinary diabetic clinics with input from diabetologist, surgeon, chiropodist and orthotist

Good foot care, footwear, skin hygiene and glycaemic control essential
  - Keep nails trim, and moisturise to prevent skin cracks

Early detection by regular testing of sensation (Semmes-Weinstein hairs) and vibration (biothesiometer)

Swab any ulcers, and cover for anaerobes with Abx

X-ray changes of diabetic osteopathy
  - Peri-osteal reaction
  - Osteoporosis
  - Cortical defects near articular margins
  - Osteolysis

Treat insufficiency fractures, but keep immobilisation to a minimum

Amputation may be required at different levels.

Diabetic ulcers pathophysiology:

- Loss of protective sensation and repetitive trauma
- Loss of sweat leads to fissuring and cracks in dry skin
- Loss of adaptive gait changes to a Charcot arthropathy
- Increased glycosylation-crosslinking of collagen in heel leads to a tight TA, with reduced dorsiflexion and the heel raising off the ground earlier during gait cycle. This puts more pressure on the MT- heads for longer.
- Reduced blood flow due to atherosclerosis NOT a risk for ulcer development, but reduces healing
- Microvascular ischaemia will predispose to forefoot wet gangrene.

Treatment:

- Good glycaemic control with HbA1c < 8%
- 90% will not develop ulcers if reduced sensation to Semmes-Weinstein 5.07 filaments present
- Lanolin rich moisturiser
- Accommodative orthotic footwear with regular replacement
- Pressure relief with ulcer debridement and serial casting for fore- and mid-foot ulcers.
- Strict non weight-bearing and dressings for heel ulcers
- Vascular assessment
- Local debridement ± topical negative pressure or larvae. Consider total contact casting – to reduce oedema and distribute pressure evenly
- Investigation for osteomyelitis with plain x-rays or nuclear scans.
- If an ulcer clinically probes to bone, there is a 90% chance of OM.
- Dressing adjuncts:
  - Becalerin gel with platelet derived GF
  - copper containing dressing for granulation stimulation
  - Mesh with cultured human fibroblasts
  - Silver gels vs. Pseudomonas colonisation

Wagner classification modified by Brodsky:
- **Depth of ulcer**
  - 0 = no break in skin but foot at risk → protective & preventative (TA length)
  - 1 = superficial ulcer → pressure relief (e.g. total contact cast)
  - 2 = exposed tendon or joint → debridement & antibiotics then pressure relief
  - 3 = osteomyelitis / deep abscess → major surgical debridement (incl. amputation) and prolonged antibiotics
  - 4 = partial gangrene
  - 5 = extensive gangrene

- **Vasculopathy (A to D)**
  - Do not rely on ABPIs as false +ve in diabetic from calcification of vessels
  - Formal Doppler analysis or angiograms
  - (Transcutaneous CO2 if available)

- **Advice in diabetic trauma:**
  - Stable fractures – non-operative
  - Unstable fracture – ORIF
  - Presence of neuropathies or renal disease – more fixation and prolonged protected weight bearing.
Charcot Joint

<Bookmark>Neurotrophic theory - loss of autonomic vasomotor flow leads to "flushing out" of bone mineral with hyperaemia and increases osteoclast stimulation vs.

Neurotraumatic theory – trauma or repetitive trauma with loss of protection sensation
</Bookmark>

- Inflammatory processes with TNFα

❖ 3 Stages - Eichenholtz:
- 0 (at risk): any patient with diabetes and fracture, lacking protective sensation
  - at risk of inflammation with osteoclast stimulation that will impair fixation, delay union and increase infection risk (45% complication rate)
- 1 (fragmentation): acute fracture-like stage with soft tissue swelling, collapse and instability. (300-fold risk of ulcer development over bony prominence)
  - treat with rest, NWB, total contact casting and bisphosphonates
- 2 (coalescence): early coalescence with stable soft tissue envelope with reduction in swelling and erythema
- 3 (consolidation): joints coalesced and developed mature calluses and stability usually over 18 months. Resolution of oedema with residual deformity.
  - Custom made footwear

❖ Types:
- 1 = midfoot – TMTJ most common resulting in fixed deformities with plantar and medial prominences
- 2 = hindfoot – result in dislocation with wide rocker bottom
- 3 = ankle – least common but unstable and difficult to control leading to ulceration at malleoli

❖ 30% risk of future bilaterality

❖ Midfoot disease more common. Treat initially with serial total contact casting, until soft tissue volume status stable. Then change to Charcot Restrained Orthotic Walker (CROW) until Stage 3 reached clinically and radiographically.
  - Assess for bony prominences from midfoot collapse and loss of arches, treated by excision. Severe midfoot collapse leads to reversal of arch with fixed rocker-bottom deformity, which needs midfoot fusion to reconstitute the arch.

❖ Hindfoot Charcot disease more grim with early deformity, ulceration and osteomyelitis. Often develop secondary after simple ankle fractures. Early fusion with retrograde nailing may be needed, as surgical stabilisation is usually avoided in early Charcot arthropathy.

❖ Clinical presentation:
  - initially warm, swollen foot that mimics infection, but less painful.
“hot, red, swollen foot” without any ulceration in a diabetic patient
bounding pulses
Bone oedema seen on MRI without obvious collection, bone destruction or soft tissue oedema.

- Treatment strategies for Acute Charcot:
  - Immobilise and off-load
  - Accommodative, pressure relieving cast = total contact cast
  - Follow-up 1-2 week intervals – to ensure well-fitting cast, and skin checks
  - Idea is to get the patient from Stage 1 to 2 – assessed with visual checks, skin temperature probes
  - At stage 2 → CROW (walker) until stage 3
  - Based on x-ray, and at this point look at accommodative custom made shoes

- Surgical indications: severe uncontrollable deformity or deep infection
  - Stable deformity / prominences – managed with removal of exostosis
  - Unstable deformity – stabilise with internal fixation / fusion
  - Infection – resection and external fixation or amputation

- Outcomes:
  - Union rates of 70%
  - Stable foot that will fit in a shoe / brace 80%
  - Amputation not advised 1st line as there is a 30% risk of bilaterality, and increases energy expenditure → patients with comorbidities may become wheelchair bound.
Foot complaints

Paralysed Foot

- Common complaints: difficulty walking, catching toes on climbing stairs, falls/stumbles
- UMN lesions:
  - In children with Cerebral Palsy or adults following a CVA
  - Muscle imbalance leads to equinus or equinovarus deformity
  - o/e: brisk reflexes with normal sensation
- LMN lesions:
  - Commonest cause historically = poliomyelitis
  - Balanced weakness can result in flail foot which dangles from ankle
  - If knee extension affected, patient requires a calliper to walk
  - FFD results from unbalanced weakness
  - Other causes:
    - Spinal cord tumours
    - Peroneal muscular atrophy
    - Severe nerve root compression
- Peripheral nerve injuries
  - Sciatic, superficial and deep peroneal nerves most at risk
  - Foot drop is most common feature
  - Compression bandaging or pressure from the leg moving into external rotation during immobilisation affect the peroneal nerves
  - Usually combined motor and sensory defect; recovery after several months unless the nerve has been divided by trauma/surgery.
- Management:
  - Physiotherapy ± splint (anti-footdrop orthosis, AFO splint)
  - Tendon transfer of tibialis posterior through the interosseous membrane to the mid-tarsal region
  - Spastic paralysis can be treated by tendon release and transfer; but avoid over-correction.

Midfoot Pain

- Kohler's disease = childhood osteochondritis of navicular
  - Children <5 years
  - Dense fragmented bony nucleus of navicular on x-ray
  - Tender, with warm thickening over navicular
  - Spontaneous resolution without specific treatment
- Brailsford Disease = similar to Kohlers, but affects middle-aged women
- In adults with high arch, can develop a secondary ridge of bone on the dorsal surface of the medial cuneiform and 1st metatarsal
  - Either manage with shoe adjustments, or shave off surgically
Forefoot Pain

- Metatarsalgia
  - Generalised ache from foot strain related to faulty weight distribution (flat arches), undue shortening of 1st MT, or even prolonged/unusual exercise.
  - Mismatch between load on foot, internal structures, and muscular effort to resist/support load – 60% of stance phase is with maximal weight to metatarsal region.
  - Therefore treatment is to treat any deformity or use orthotics to redistribute load, and physiotherapy to improve intrinsic muscle strength to aid anterior (metatarsal) arch
  - Caused usually by problems at the MTPJ:
    - Synovitis 2° inflammatory arthritis \(\rightarrow\) synovectomy, or 1st MTPJ arthrodesis with decompression of lesser MTP-joints by excision of MT-head (Fowler's procedure), which also pulls the protective fat pad back down over the weight bearing area.
    - Plantar plate dysfunction – cystic degeneration.
    - Freiberg's infarction – AVN and collapse of MT-head of unknown cause; occasionally requires surgical debridement.
    - Subluxation of MTPJ – can result in a cock-up deformity with pressure on the MT-head.
    - Non-specific synovitis around 2nd MTPJ – “walking on a painful lump”; if footwear padding, NSAIDs, and steroid injection all fail, consider synovectomy.

- MTP Joint Pains is associated with swelling, tenderness, and are bilateral should always be investigated for manifestations of inflammatory arthritis (Rheumatoid)

- Morton’s foot = pain over plantar aspect of MT-head
  - Causes include: a prominent fibular, long (2nd) metatarsal, short 1st metatarsal, instability or traumatic disruption of 1st MTC-joint.
  - Weil’s Osteotomy – extra-articular osteotomy of distal meta-tarsal to decompress MTPJ and shorten the metatarsal, thereby relieving any plantar prominence.
Localised Forefoot Pain

- **Sesamoiditis:**
  - Pain and tenderness under 1st MT head worse on walking or passive dorsiflexion
  - Inflammation of peri-tendinous tissue around sesamoids (medial most common)
  - Acute – direct trauma or unaccustomed stress
  - Chronic – sesamoid displacement, local infection (e.g. diabetic) or AVN
  - Sesamoid chondromalacia – bipartite or fragmented appearance on x-ray

- **Rx:**
  - reduced weight bearing, pressure pad
  - local steroid injections
  - surgical excision taking care not to interrupt FHB tendon

- **Freiberg's Disease**
  - Crushing osteochondritis of 2nd MT head (rarely 3rd), with AVN and collapse.
  - Young adults and women
  - Irritable MTP joint, with a bony lump felt under MT head
  - X-ray shows widened and flat MT head, with a thick neck

- **Rx:**
  - Walking plaster or moulded sandal to reduce pressure on metatarsal head
  - Operative synovectomy, debridement and trimming if persistent symptoms

- **Stress Fracture**
  - Usually from unaccustomed activity
  - Common in post-menopausal women or those with osteoporosis.
  - Delayed presentation, with often a finding of callus on x-ray around a fine transverse fracture
  - Conservative management – rest and analgesia

- **Soft tissue lesions:**
  - Plantar wart – liquid nitrogen rx
  - Seed wart – invagination of skin; trimming or curettage
  - Hyperkeratotic skin lesion – trimming
    - Hard corn between skin and shoe
    - Soft corn between two toes
  - Try trimming and relieving pressure initially. If this fails look for any exostosis that can be excised.
  - Atrophy of plantar fat pad – elderly, treat with soft shoe
  - Lipoma

- **Morton's Metatarsalgia**
  - Women > 50 years with forefoot pain and burning/tingling in toes
  - Worsened with tight shoes
  - Tender and paraesthesias elicited by direct compression in web space
  - *Digital nerve entrapment*, but secondary nerve thickening mimics “neuroma”
  - Interdigital neuroma assessed by pressing plantarward over sole of forefoot, while squeezing in medial-lateral direction. Pain = Mulder's click

- **Ix:** LA-injection into transverse intermetatarsal ligament or USS

- **Rx:**
  - Protective padding and wider shoes
- Injection of LA-steroid
- Release of tight inter-metatarsal ligament

❖ Tarsal Tunnel Syndrome
- Pain and sensory changes in medial part of forefoot, unrelated to weight bearing
- Compression of tibial nerve behind and below medial malleolus
- Can be secondary to SOL (e.g. ganglion, haemangioma, varicosity)
- Pain worse at night, and relieved by standing and stamping feet
- Nerve conduction studies can be normal
- Symptoms can be mimicked by nerve tension caused by flat foot (or hindfoot valgus)

- Tarsal Tunnel lies deep to the flexor retinaculum (formed by condensation of deep fascia of leg and transverse fascia of ankle), with its floor formed by medial wall of talus, and superior aspect of calcaneus – contains tendons of Tibialis Posterior, FDL and FHL along with tibial nerve and posterior tibial artery

- Surgical decompression possible, but also ensure not trapped by the belly of adductor hallucis (rising more proximal than normal)
Skin and Nail Disorders

- Corns are hyperkeratotic lesions usually on dorsal surfaces of toes or between toes
  - Reaction to friction of local pressure from hammer deformities or clawing of toes pushing dorsal surface upwards against inside of shoe
  - Treat with felt pads, or paring of hyperkeratotic skin

- Calluses more diffuse hyperkeratotic lesions on soles of feet
  - Usually under prominent MT heads or heel
  - Common in dropped metatarsal arches, claw toes or varus/valgus heels
  - Treatment by redistributing forces by surgical correction or corrective footwear.

- Plantar Warts resemble calluses by more tender
  - On paring hyperkeratotic skin, a papillomatous “core” is revealed and dotted with fine capillaries
  - Viral lesions
  - Rx:
    - Salicylic acid plasters
    - Cryotherapy
    - Surgical excision tends to leave a painful scar at pressure site

- Foreign Body “Granuloma”
  - Thorns, splinters, shard of glass
  - Painful lump resembling a callus
  - Improves with removal of foreign body; x-ray can help identify glass

- In-growing Toe nail
  - Grows into nail fold causing local ulceration, infection and pain
  - Mx:
    - Teach patient to cut toenail straight or square
    - Use cotton pledgets under sides to lift away in-growing edges
    - Keep feet clean and dry
    - Wedge resection with chemical ablation/curettage of germinal matrix

- Overgrown Nail (Onychogryposis)
  - Hard, thick and curved
  - Chiropodist usually can help, but complete nail excision may be needed

- Undergrown Nail
  - A subungual exostosis grows on dorsum of terminal phalanx and pushes nail upwards
  - Requires debridement of exostosis.
Osteochondral Lesions of the Talus (Osteochondritis Dissecans)

- Detachment of a segment of cartilage and subchondral bone from the articular surface of the talus
  - This can be an acute event, or chronic if it leads to a non-union (the classic osteochondritis dissecans)
  - 60% posteromedial – deeper and traumatic in 70%; usually caused after plantarflexion and inversion
  - 40% anterolateral – broad and wafer like, and traumatic in 98% caused by eversion and dorsiflexion

- Berdt & Harry Classification (1959) – CT and MRI classifications very similar principles:
  - 1 = small area of subchondral bone impaction
  - 2 = partial detachment of fragment
  - 3 = complete detachment but no displacement
  - 4 = osteochondral loose body

- Symptoms:
  - Typically occurs in conjunction with ankle sprain: ecchymosis, ligament pain, swelling and limited ROM.
  - Difficult to differentiate from ankle sprain in the acute phase.
  - Chronic injury presents with activity-related pain and intermittent swelling
  - Locking is rare
  - Assess for tenderness on palpation at the interval between the talus and tibiofibular ligament (anterolateral talar dome)

- X-rays should include mortise view in plantar flexion (posterior talar dome) and dorsiflexion (anterior dome). MRI or CT usually needed to visualise.

- Medial sided injuries have greater potential for healing. Therefore up to grade 3 medial lesions and grade 2 lateral lesions can be trialled for 6 months with conservative treatment: walking boot or cast for 6 weeks followed by physiotherapy.

- Surgical options – based on arthroscopic classification for stability.
  - A = smooth and intact, but softening
  - B = rough surface
  - C = fibrillations or fissures
  - D = flap with bone exposed
  - E = loose, non-displaced
  - F = displaced

  - Acute OLT that are stable can be treated with retrograde drilling. If overlying cartilage is not completely intact, it can be debrided to a stable rim

  - Acute unstable OLT treated with internal fixation using Herbert screws, K-wires or bioabsorbable pins/screws. Medial sided lesions may require a medial malleolar osteotomy
Chronic lesions:
- 1 & 2 = drilling with curettage of any cystic lesions; large cysts may need bone grafting
- 3 & 4 = excision of loose bodies with non-viable cartilage, with debridement and drilling of base. Rarely if fragment is viable it can be internally fixed.
- Autologous osteochondral grafting (mosaicplasty)
- Autologous chondrocyte implantation (ACI) using cultured chondrocytes.

Examination of Foot & Ankle

- Observe gait and standing assessment:
  - Has the shape been the same or changed?
    - normal, cavus, planus, convexus (rocker bottom)
  - Calf wasting
  - Knee deformity (genu valgus / carus)
  - Double heel raise → single heel test (ONLY if normal double heel test)
  - Heel walking and tip-toe walking for global weakness (e.g. spinal cause)
  - Stand on edge of feet
  - Ski stance – dynamic assessment of ankle ROM
  - Coleman Block test for heel varus – if driven by a plantar flexed 1st ray, hindfoot varus will correct on the block

- Sitting examination
  - Use time to get onto couch by looking at shoes
  - One finger at a time
  - Look for scars

- Special tests:
  - Anterior ankle impingement sign – check ROM and test for pain on dorsiflexion while pressing into medial and lateral gutters
  - Instability – syndesmosis instability (direct squeeze or external rotation); anterior drawer (grade 1-3), CFL
  - Posterior impingement (footballers and dancers) – hyper-plantarflexion and jog the os calcis
  - Peroneal tendon pain – resisted eversion

- Role of subtalar joint is a torque converter – external rotation of tibia will result in heel inversion and medial elevation.

- Simmonds test for TA rupture is positive if there is a rupture.

- Silfvershiold’s test – assess for ROM in terms of ankle DF with knee straight and flexed to differentiate between Achilles tightness and Gastrocnemius tightness

- 1st TMTJ instability – more than half of the metatarsal head height dorsal translation hold the head of the 1st MT with one hand, and the heads of 2nd to 4th MT with other

- Morton’s Neuroma can present in the same way as MTPJ instability of the lesser toes – assess with a vertical Lachman’s test of the lesser MTPJ with pain
Orthotics – Basics

- Normal gait there is momentary varus in heel strike (stiff), followed by mid-stance pronation (loose foot), then a toe off as part of windlass mechanism (stiff).
  - Corresponds to normal wear pattern in show on lateral heel and medial forefoot.

- Function
  - Stability – stability, e.g. AFO
  - Corrective – flexible deformity
  - Accomodative – fixed deformity, arch support or wedges to bring floor to foot
  - Pressure relieving – e.g. total contact

- UCBL (University of California Biomechanics Lab) orthosis
  - Medial arch support with heel support to prevent rolling of chubby heels in paediatrics

- Metatarsal bar to offload MT head
  - Used in Morton’s neuroma, Freiberg’s infarction and flexible clawing

- Loss of transverse arch will increase joint reaction force onto 2nd MT head – can be managed with cutout to offload this area and load other MT heads instead.

Ankle Fractures

- It is the deep part of the deltoid (medial) ligament that provides stability in an ankle fracture.

- Laughe-Hanson classification:
  - First description is position of foot at injury
  - Second is direction of movement / trauma

  - Supination adduction – first an avulsion of fibula from stretch on lateral side (horizontal fracture line), then talus adducts and hits medial malleolus causing a shear fracture medially

  - Supination-external rotation: - tear or ATFL and then spiral fracture of fibula. As foot externally rotates there is tension on medial side tearing the deep deltoid ligament and then resulting in horizontal/avulsion fracture line

  - Pronation-abduction – transverse fracture of fibula at level of syndesmosis. Isolated medial malleolus fracture needs to be observed closely as deep deltoid ligament is not acting as a restraint. If displaced then ORIF, if undisplaced close observation.

  - Pronation – external rotation – results in deltoid injury or avulsion of medial malleolus. Then disrupts syndesmosis before force transmitted proximally through fibula to cause a spiral high fibula fracture.
Tibial pilon fractures:
- Classification based on Topliss (JBJS 2006)
- Treatment mantra = span, scan and plan