Diabetic Foot Disorders
Multisystem disease with reduced insulin synthesis or peripheral resistance to its action by failed intra-cellular uptake. 50% of costs of diabetes associated with foot complications.

Pathogenesis
- **Sensory neuropathy** – loss of protective pain sense results in repetitive trauma in areas of high mechanical pressure
  - Diagnosis by absent sensation with 5.07 (10g) Semmes-Weinstein monofilament at 6-7 sites on the foot.
  - Or cannot feel 4.65 (4.5g) Semmes-Weinstein monofilament pressed against foot beneath 1st metatarsal head, until it bends (Saltzman 2004)
- **Autonomic neuropathy** – loss of sudomotor control results in dry, cracked skin. Fissures are portals of entry for bacteria and infection.
- **Motor neuropathy**
  - Intrinsic musculature weakness leads to clawing and increase pressure under the metatarsal heads as well as tips of toes.
  - Mononeuropathy and entrapment neuropathy most commonly affects common peroneal nerve leading to a foot drop
- **Idiopathic gastrocnemius contracture** can lead to increased forefoot pressure and ulceration
- **Neuropathic pain** from injury to peripheral nerve fibres or to CNS, even in combination with reduced sensation.
- **Impaired healing:**
  - Nutritional factors
  - Adequate wound oxygenation ← negative impact from oedems / CVI
  - Defects in Type X collagen
  - Altered gene expression that regulate osteoblast differentiation
  - HbA1c levels should be >7%

Diabetic Foot Ulcers
- **Modified Wagner-Meggitt classification**

<table>
<thead>
<tr>
<th>Depth</th>
<th>Grade</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>0 / A</td>
<td>At risk – prior ulceration, or neuropathy with deformity</td>
<td>No ischaemia</td>
</tr>
<tr>
<td>1 / B</td>
<td>Superficial ulceration without infection</td>
<td>Ischaemia without gangrene</td>
</tr>
<tr>
<td>2 / C</td>
<td>Deep ulceration exposing tendons or joints ± infection</td>
<td>Partial (e.g. forefoot) gangrene</td>
</tr>
<tr>
<td>3 / D</td>
<td>Extensive ulceration with exposed bone. Invariable deep infection or OM</td>
<td>Complete foot gangrene</td>
</tr>
</tbody>
</table>
Negative pressure vacuum dressings for chronic refractory ulcers

Off-loading with total contact casts (gold standard), removable splints, CAM walkers, or accommodative footwear with pressure relieving insoles.

Total contact casts:
- Reduce plantar pressure by distributing body weight over a larger weight-bearing surface area
- Reduce shear and vertical plantar pressures at ulcer edges
- Reduce oedema
- Change every 5-14 days to avoid skin abrasions from ill-fitting as swelling changes
- Initial non-weight bearing
- Contra-indicated in active infection, poor skin quality or arterial insufficiency.
- Average ulcer healing in 39 days (Myserson 1992, Matricali 2003, Trepman 2005)

Consider TA or gastrocnemius lengthening in “at-risk” patients or once forefoot ulcers have healed, if evidence of limited ankle dorsiflexion with neuropathy.

**Charcot Foot**

- Hypertrophic arthropathy in patients with peripheral neuropathy, associated with good peripheral circulation
- Initially most common cause was syphilis, but now diabetes.
- Usually more frequent in diabetes history > 10 years. 30% bilateral

- **Neurotraumatic theory** = microfracture from repetitive trauma in the absence of protective pain sensation, which gradually worsens.
- **Neurovascular theory** = loss of vasomotor control leads to hyperaemia and bony resorption with weakening.

- Acute Charcot foot is often confused with infection or cellulitis, as there is swelling, warmth and erythema. However, a decrease in erythema may be seen with Charcot as opposed to cellulitis.
- Chronic Charcot foot presents with collapse of the medial longitudinal arch, rocker-bottom deformity and bony prominences.
- X-ray changes are often late after 3 weeks, and include localised osteopaenia, peri-articular fragmentation, bone resorption, new bone formation, joint destruction or subluxation.
- Indium-111 WBC-scan more specific than sensitive compared with MRU, in distinguishing between osteomyelitis and Charcot.

Schon Classification by site
- I = 60% of cases occur at mid-foot and stable deformity (valgus or rocker-bottom)
- II = 20-30% at hindfoot with progressive deformity
- III = ankle (A), calcaneus (B)
- IV = multiple regions
- V = rare in forefoot, but associated with infection and ulceration.
<table>
<thead>
<tr>
<th>Eichenholtz Stage</th>
<th>Clinical</th>
<th>X-ray</th>
<th>Management</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>At risk</td>
<td>No destruction</td>
<td>Internal fixation of fractures with protective off-loading (×2 expected healing time). Accommodative footwear in long-term</td>
</tr>
<tr>
<td>1</td>
<td>Fragmentation</td>
<td>Erythema, swelling, warmth</td>
<td>Subchondral fragmentation and osteopaenia</td>
</tr>
<tr>
<td>2</td>
<td>Coalescence</td>
<td>Decreased erythema, swelling and warmth</td>
<td>New bone formation and coalescence</td>
</tr>
<tr>
<td>3</td>
<td>Consolidation</td>
<td>Resolution of oedema, and residual deformity</td>
<td>Joint subluxation, destruction</td>
</tr>
</tbody>
</table>

- Fragmentation: Erythema, swelling, warmth
- Subchondral fragmentation and osteopaenia
- Elevation, off-loading, total contact casts. Exclude infection, gout, or RA.
- Coalescence: Decreased erythema, swelling and warmth
- New bone formation and coalescence
- Accommodative orthoses ± surgical reconstruction to provide a stable, plantar-grade foot that is “braceable” or “shoe-able”, without bony prominences.

- Consolidation: Resolution of oedema, and residual deformity
- Joint subluxation, destruction