Generalized Conditions

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1 Neuropathic Foot

Take-Home Message

- The neuropathic foot is most commonly a sequela of diabetes but can also result from upper motor neuron (UMN) lesions and hereditary motor-sensory neuropathies (HSMN).
- Surgical treatment of UMN disorders often include Achilles lengthening, tibialis anterior transfer, and toe flexor release.
- Surgical treatment of HSMN requires a thorough evaluation into what drives the pes cavovarus deformity and its flexibility.

Definition

- Mechanical changes in the foot which develop as a result of a disturbance in the normal sensory and motor innervation of joints.

Etiology

- Diabetic neuropathy: most common cause of neuropathic foot (see chapters Paediatric hip conditions and Paediatric feet conditions).
- Sequelae of upper motor neuron (UMN) disorders: most commonly secondary to traumatic brain injury, stroke, and spinal cord injury.

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C. Mauffrey, D.J. Hak (eds.), *Passport for the Orthopedic Boards and FRCS Examination*, DOI 10.1007/978-2-8178-0475-0_42

- Hereditary motor-sensory neuropathies (HMSN): inherited progressive peripheral neuropathy (e.g., Charcot-Marie-Tooth disease). Specific etiology depends on the particular variant. For CMT it is autosomal dominant with a duplication of chromosome 17.
- Can also be caused by chemotherapeutic agents and certain infectious diseases such as HIV.

Pathophysiology

- Diabetic neuropathy (see chapters Paediatric hip conditions and Paediatric feet conditions):
- Upper motor neuron disorders:
  - Disruption of UMN pathways can lead to paralysis, muscle imbalance, and acquired spasticity, which ultimately may cause deformity.
  - Secondary problems are contractures, calluses, pressure sores, joint subluxation, hygiene issues, shoe-wear difficulties, and dissatisfaction with personal experience.
  - The most common deformity is equinovarus caused by overactivity of gastrocnemius-soleus complex and relative overactive tibialis anterior.
- HMSN, depends on the specific type:
  - For CMT the basis is an abnormal myelin sheath protein.
  - Leads to motor imbalance and bilateral → first ray is plantarflexed due to relative unopposed pull of peroneus longus → forefoot cavus and compensatory hindfoot varus → symmetric pes cavovarus deformity (Fig. 1).
  - Intrinsic wasting → overpull of extrinsic musculature → claw toe deformity (Fig. 2).
  - Variable sensory deficits → can lead to recurrent ulceration, infection, and arthropathy.
  - Forefoot-driven hindfoot varus: deformity corrects with Coleman block test. Concomitant intrinsic hindfoot varus: deformity does not correct.

Treatment

Diabetic Neuropathy **(see chapters Paediatric hip conditions and Paediatric feet conditions):**

UMN disorders:

Nonoperative Care

- Physical therapy, stretching, maintenance of joint range of motion. Other modalities include splinting, serial casting, oral muscle relaxants, phenol and lidocaine nerve blocks, and botulinum type A toxin.
- Phenol block have proven history with longer-lasting effect and are less expensive than botulinum toxin. However, botulinum toxin is easy to deliver since it needs only an injection into the muscle belly rather than precise injection around nerve.

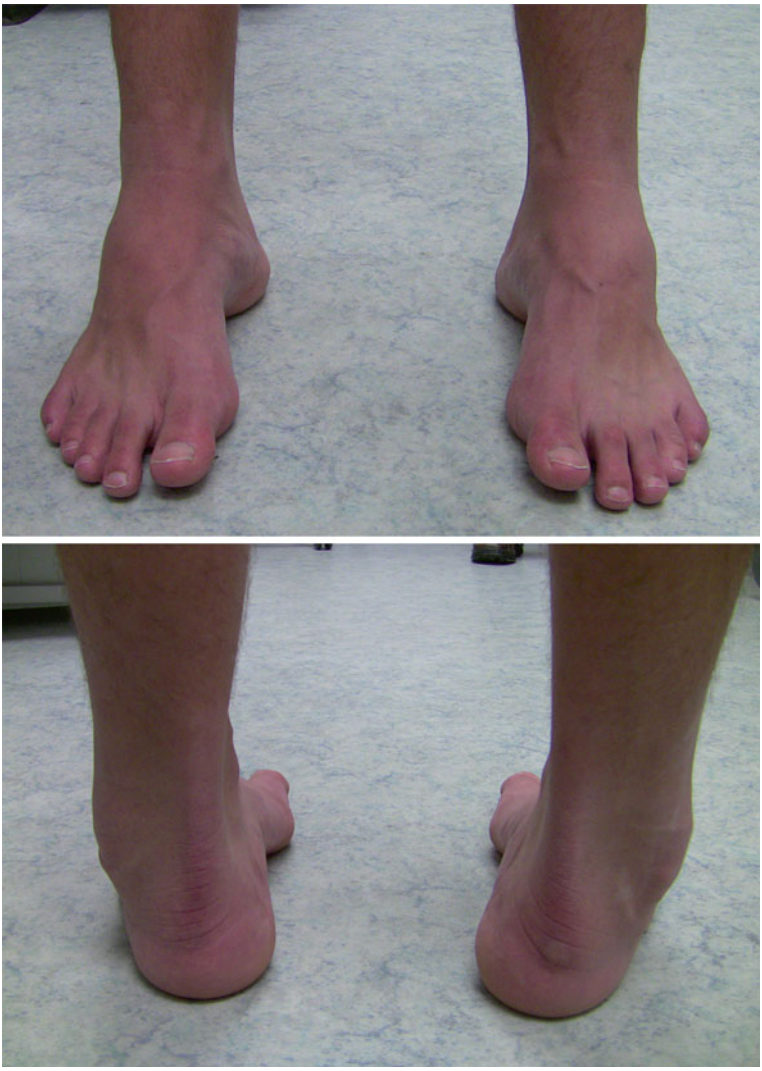

Fig. 1 *Anterior view* of a patient with HSMN with bilateral cavus feet. Note the ability to see the heel from this view with the elevated arches. The *posterior view* clearly demonstrates the varus of the hindfoot

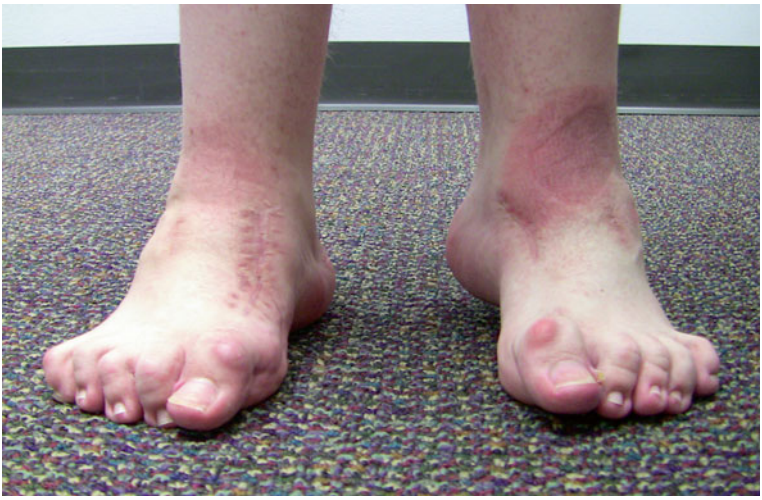

Fig. 2 Multiple claw toes and significantly claw hallux are seen in more advanced stages of HSMN. Claw hallux and claw toes in young patients should always increase the suspicion of a neurologic disorder

Surgical Treatment

- Address equinus deformity with open Z-lengthening or percutaneous lengthening.
- Varus deformity addressed with split anterior tibialis tendon transfer (SPLATT) to lateral cuneiform or cuboid or total anterior tibialis tendon transfer to lateral cuneiform. Release of toe flexors often required.

Hereditary motor-sensory neuropathies (HMSN)

Flexible Deformity (hindfoot can be passively manipulated):
Nonsurgical management:

- Not currently recommended given progressive pattern of disease
- Surgical management:
- Forefoot driven: closing wedge dorsiflexion osteotomy of first metatarsal (Fig. 3), release of plantar fascia, transfer of peroneus longus into peroneus brevis at level of distal fibula.
- Hindfoot driven: in addition to abovementioned procedures, include lateral calcaneal slide and/or closing wedge osteotomy (Fig. 4).
- Clawed hallux can be surgically treated with Jones procedure (arthrodesis of interphalangeal joint and transfer of EHL to the first metatarsal).
- Consider posterior tibial tendon transfer to dorsum (lateral cuneiform) or lengthening of the tendon to restore balance.

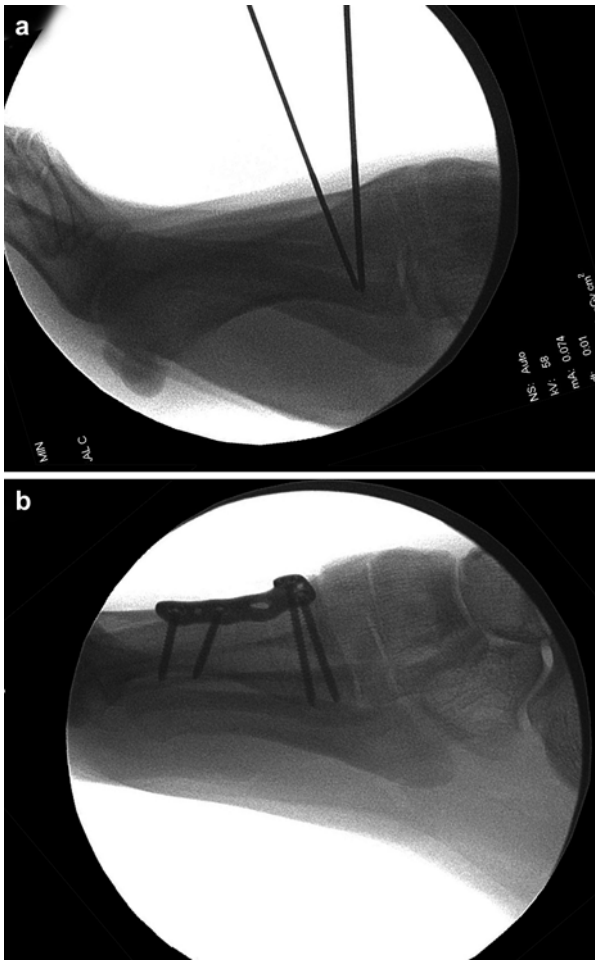

Fig. 3 Dorsiflexion osteotomy to correct the plantarflexed first ray is performed by marking the osteotomy with two K-wires (**a**). Following resection of the wedge, the osteotomy is closed and fixated with resultant elevation of the first ray (**b**)

Fixed Deformity (hindfoot cannot be passively manipulated):
Nonsurgical management:

- Attempted with locked-ankle, short-leg ankle-foot orthosis with a lateral T-strap.
- Rocker sole can improve gait and decrease energy expenditure.

Surgical management:

- Triple arthrodesis usually required for hindfoot correction. Posterior tibialis tendon transfer through the interosseous membrane can correct equinus contracture and dorsiflexion weakness.
- Must address imbalance of tendon forces even in the setting of an arthrodesis to prevent recurrence.
- Dorsiflexion osteotomy of first metatarsal, release of plantar fascia.
- Forefoot correction is performed according to the guidelines outlined previously.

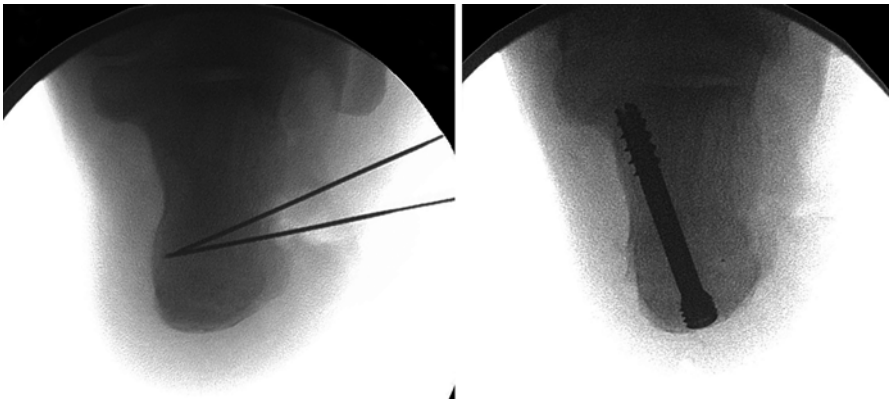

Fig. 4 Lateral closing wedge osteotomy of the calcaneus in a patient who had concomitant intrinsic hindfoot varus (failure to correct with Coleman block). This is performed in addition correction of the plantarflexed first ray and is not a substitute for a dorsiflexion osteotomy of the first metatarsal

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2 Rheumatoid Foot

Take-Home Message

- Rheumatoid arthritis is a chronic autoimmune disease that results in polyarthropathy that commonly involves the forefoot.
- Typical deformity includes dorsal and valgus toe deviation, claw toe deformity, and pes planovalgus.
- Conservative treatment involves proper shoe and orthotic wear and immune-modulating drugs under the direction of a rheumatologist.
- Surgical treatment of the forefoot includes first MTP arthrodesis, lesser MT head resections, osteoclasis of interphalangeal joints, and extensor brevis tenotomy, while midfoot or triple arthrodesis is needed for pes planovalgus deformity in the rheumatoid foot.

Definition

- Chronic, symmetrical polyarthropathy that most commonly presents in the third and fourth decades and is more prevalent in women

Etiology

- Autoimmune disease with a genetic predisposition
- Cell-mediated immune response against soft tissues, cartilage, and bone

Pathophysiology

- ESR, CRP will be elevated, and RF titers positive (most commonly IgM).
- Chronic synovitis leads to ligament and capsular laxity and cartilage and bony erosion.
- Forefoot involvement very common:
  - Complaints of forefoot swelling, poorly defined pain, and eventually deformity.
  - Incompetence of joint capsules and lateral ligaments causes toes to subluxate or dislocate dorsally and deviate into valgus (Fig. 5).
  - Contracture of the intrinsic musculature exacerbates claw toe deformity.
  - Plantar fat pad migrates distally and atrophies, causing metatarsalgia and forming keratoses.
  - As lesser toes deviate, hallux valgus occurs, and transfer metatarsalgia worsens (Fig. 6).
- Midfoot and hindfoot less commonly and less severely involved:
  - Midfoot/hindfoot arthrosis often results in pes planovalgus deformity that can be midfoot driven (tarsometatarsal joints are subluxated with a congruent hindfoot) or hindfoot driven (transverse tarsal and subtalar joint is subluxated with normal midfoot).
- Tibiotalar joint is also commonly involved and may be caused by chronic subtalar joint malalignment.

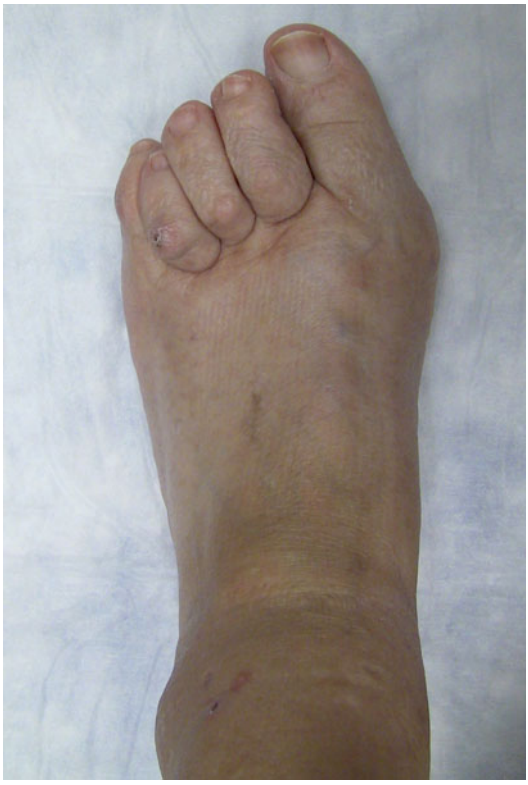

Fig. 5 Clinical photograph of a patient with RA with swelling of the digits and characteristic valgus deviation of all digits with claw toes

Radiography

- Can have significant midfoot and hindfoot arthrosis (talonavicular joint is characteristic)
- Typically has diffuse osteopenia, symmetrical joint space narrowing, and lack of osteophyte formation (which easily differentiates RA from osteoarthritis) (Fig. 7)

Treatment

- Vasculitis and soft tissue fragility is common, requiring diligent care of the soft tissues regardless of treatment.

Conservative

- Rest, NSAIDs, immune-modulating drugs under the direction of rheumatologists, toe taping, orthoses, careful use of corticosteroid injections to help symptoms related to synovitis, and patient education

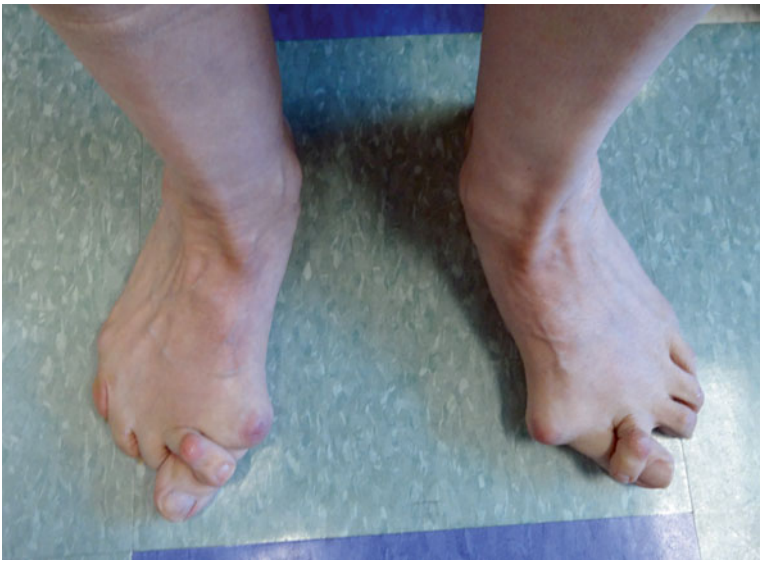

Fig. 6 In severe cases, the joint laxity that occurs results in significant hallux valgus with overlap of the second toe over the first

Surgical

- Should discuss use of immune-mediating pharmacologic therapies with rheumatologist prior to surgery → while most medications can be continued (prednisone, methotrexate, plaquenil), the newer biologic agents (such as TNF inhibitors) should be discontinued.
- "Rheumatoid forefoot reconstruction" for deformity correction:
  - First MTP arthrodesis, lesser metatarsal head resection with pinning of lesser MTP joints, closed osteoclasis of interphalangeal joints versus PIP arthroplasty (silicone arthroplasty not recommended) through the use of three dorsal incisions. Extensor brevis tenotomy and Z-lengthening of extensor longus tendons may be necessary (Fig. 8).
- Pes planovalgus: Midfoot driven, realignment midfoot arthrodesis. Hindfoot-driven and fixed deformity, triple arthrodesis
- Tibiotalar arthrosis: Ankle arthrodesis is treatment of choice, ankle arthroplasty emerging as more reliable technique (though it is associated with increased risk of wound complications).

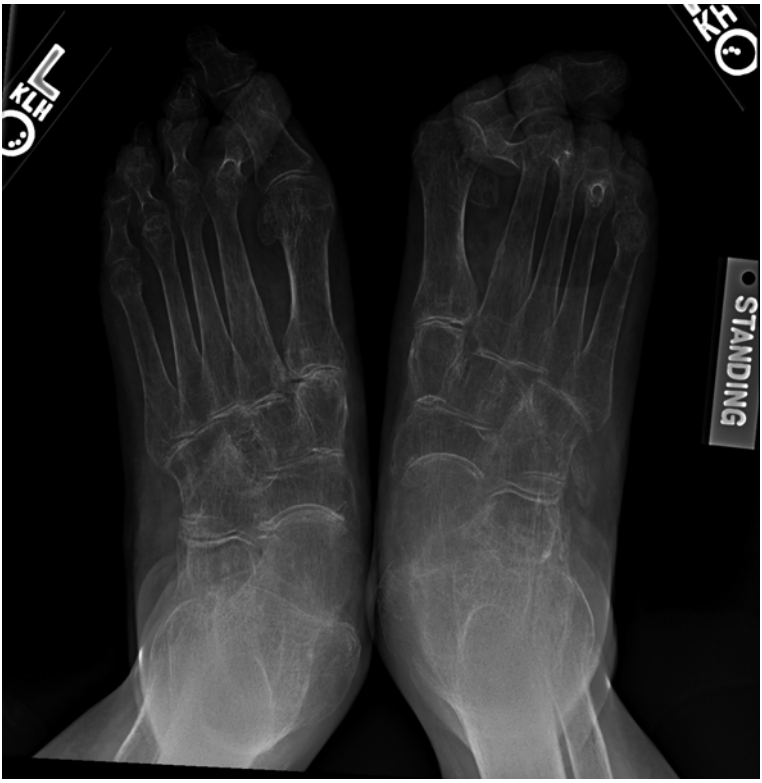

Fig. 7 Typical radiographic appearance of a patient with RA. Note the multiple joint involvement with symmetric joint space narrowing, osteopenia without osteophyte formation with associated pes planus deformity. The typical dislocation of the lesser MTP joints can be noted as well

Complications

- Wound complications common following surgical treatment.
- Current literature controversial whether patients on immunosuppressive therapies have significantly increased infection rates.
- Late recurrence of deformity has been reported and some consideration for joint sparing lesser toe surgery has been considered. However, no long-term data to support joint sparing treatment to date.

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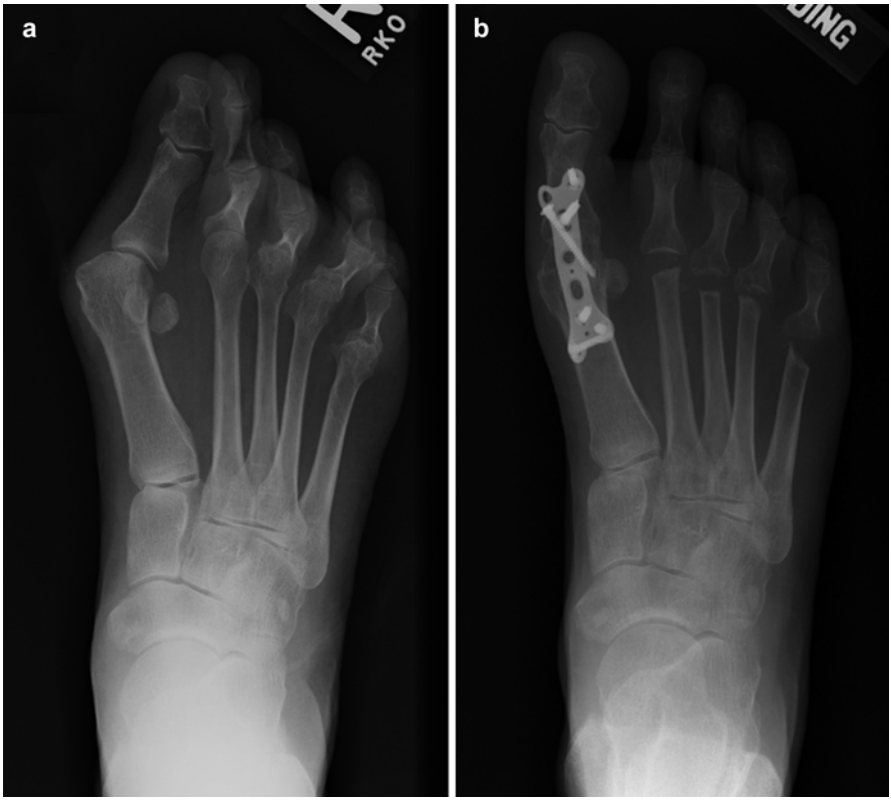

Fig. 8 Preoperative AP radiograph (**a**) of a patient with RA with clinical hallux valgus with subluxation of the lesser MTP joints. Post-op AP radiograph (**b**) demonstrating excellent alignment following first MTP arthrodesis with metatarsal head resection of joints 2–5 and osteoclysis of the PIP joints. Interposition of the extensors into the potential space created may decrease risk of late subluxation

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3 Nerve Entrapment Syndromes

Take-Home Message

- Nerve entrapment related to space-occupying mass is more likely to improve with surgical treatment than nerve entrapment without a related mass.
- The first branch of lateral plantar nerve compression between the fascia of abductor hallucis and quadratus plantae is the most common cause of nerve-related heel pain, common in running athlete.
- Superficial peroneal nerve entrapment related to chronic ankle instability and peroneal muscle herniation through fascial defect.
- Nerve entrapment syndromes most commonly cause neuropraxia type of nerve injury with nerve contusion and focal demyelination of axon sheath.

Definition

Nerve Entrapment

- Localized pressure causing nerve dysfunction.
- Tarsal tunnel syndrome → tibial nerve:
  - Boundaries – flexor retinaculum (medial); talus, calcaneus, sustentaculum tali (lateral); abductor hallucis (inferior)
  - Additional contents – tendons of tibialis posterior, flexor hallucis longus, flexor digitorum longus, posterior tibial artery, venae comitantes, numerous septa
- First branch of lateral plantar nerve (Baxter's nerve)
- Anterior tarsal tunnel syndrome → deep peroneal nerve
  - Boundaries – inferior extensor retinaculum (anterior), tibia and talus (posterior)
  - Additional contents – dorsalis pedis artery
- Superficial peroneal nerve.
- See Table 1 for symptoms and physical exam findings.

Etiology

- External compression from adjacent structures – tenosynovitis, engorged or varicose veins.
- Space-occupying mass – synovial or ganglion cyst, pigmented villonodular synovitis, nerve sheath tumors, lipomas (Fig. 9).
- Systemic disease can cause compression indirectly due to inflammatory edema – diabetes mellitus, rheumatoid arthritis.
- See Table 2 for nerve-specific etiologies.

Table 1 Nerve entrapment symptoms and physical exam findings

	Symptoms	Physical exam findings
Tarsal tunnel syndrome	Burning sensation of plantar foot, medial ankle	Positive Tinel and nerve compression tests
	Plantar foot numbness variable	Pain with dorsiflexion-eversion
	Worse with prolonged standing, walking, running	Diminished two-point discrimination
		Wasting of intrinsic musculature
		Hindfoot valgus, pes planus
First branch of lateral plantar nerve	Chronic heel pain, pain at plantar medial foot, may radiate laterally	Maximal point of tenderness at site of compression by fascia of abductor hallucis and quadratus plantae
	Symptoms similar to plantar fasciitis	
	Symptoms without weight bearing	Wasting of abductor digiti quinti
	No numbness – nerve has no sensory innervation	
Anterior tarsal tunnel syndrome	Burning pain in dorsal first webspace	Positive Tinel sign
	Vague dorsal foot pain	Diminished two-point discrimination
	Worse at night with foot in plantarflexion	Forced ankle plantarflexion reproduces symptoms
	Worse with shallow, laced shoes	Weak great toe extension
Superficial peroneal nerve	Pain and paresthesias radiating to dorsum of foot	Positive Tinel sign
	Numbness is variable	Diminished two-point discrimination
	Symptoms increase with activity	Palpable fascial defect and peroneal herniation
	May feel a bulge at lateral leg – area of muscle herniation	Forced plantarflexion and inversion reproduces symptoms
		Signs of ankle instability

Pathophysiology

- Pressure on nerve causes ischemia and neuroma formation.
- Neuroma contains bundled disorganized nerve endings within collagenous mass.
- Can result in loss of sensory and motor function.
- Pain and paresthesia replace normal sensation.

Radiography

- Weight-bearing radiographs of the foot and ankle
  - Detect bony abnormality causing or contributing to nerve entrapment.
  - Evaluate alignment of foot and ankle.
  - Rule out other source of symptoms.
- MRI – if concern for space-occupying mass
- EMG and NCV – can help confirm diagnosis but variable sensitivity

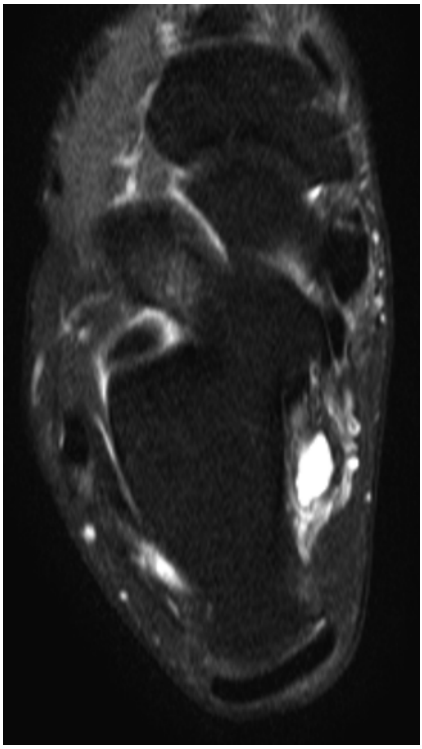

Fig. 9 Axial T2 fat-saturated image of a patient with tarsal tunnel syndrome that was noted to have a ganglion within the tarsal tunnel that required excision in addition to decompression of the nerve

Table 2 Nerve entrapment etiology

	Etiology
Tarsal tunnel syndrome	Increased nerve tension from hindfoot valgus and pes planus
	Fracture of sustentaculum tali, medial tubercle of posterior process of talus
	Accessory muscle
First branch of lateral plantar nerve	Compression between fascia of abductor hallucis and quadratus plantae
	Lateral plantar nerve injury can occur from insertion of intramedullary nail for tibiotalocalcaneal fusion
Anterior tarsal tunnel syndrome	Anterior osteophytes of tibiotalar or talonavicular joints
	Tightly laced shoes
Superficial peroneal nerve	Chronic ankle instability
	Herniation of peroneal musculature through fascial defect
	Iatrogenic injury

Classification

Seddon Classification

- Neuropraxia – nerve contusion, focal demyelination of axon sheath, no Wallerian degeneration, good prognosis
  - Most common resulting injury following nerve entrapment:
- Axonotmesis – axon and myelin sheath disruption, Wallerian degeneration, endoneurium intact
- Neurotmesis – complete disruption of nerve including endoneurium, Wallerian degeneration

Treatment

Nonoperative: first line unless a space-occupying mass is present

- Activity modification
- Medications
  - Nonnarcotic analgesics
  - Centrally acting anticonvulsants
  - Tricyclic antidepressants, selective serotonin reuptake inhibitors
  - Topically applied compounds – include local anesthetic, anti-inflammatory medication, capsaicin
- Physical and occupational therapy
- Injection of local anesthetic with or without corticosteroid medication
  - Useful for diagnosis

Operative: indicated after 3–6 months of unsuccessful conservative treatment

- Complete nerve decompression (Fig. 10)

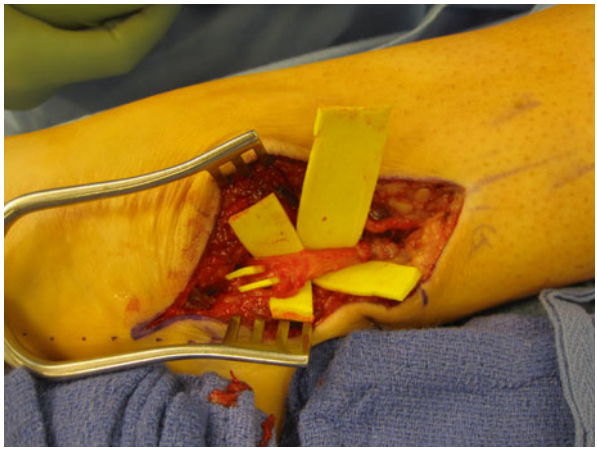

Fig. 10 Intraoperative photograph demonstrating an appropriate incision with decompression of the tibial nerve and the requisite branches. Note release of the abductor hallucis in the distal aspect of the incision, ensuring that both the medial and lateral plantar branches are adequately released

Table 3 Nerve entrapment treatment options

	Nonoperative	Operative
Tarsal tunnel syndrome	Medial heel and sole wedge if hindfoot valgus and pes planus	Identify nerve proximally
		Release deep investing fascia proximally, flexor retinaculum, deep and superficial fascia of the abductor hallucis
	Short period of immobilization with cast or boot	Assure that all branches – medial calcaneal, lateral plantar, medial plantar – are decompressed
		Release all septa
First branch of lateral plantar nerve	Heel pad	Release superficial and deep abductor hallucis fascia
	Arch support if pes planus	Remove heel spur if present
		Release part of plantar fascia if appears pathologic
Anterior tarsal tunnel syndrome	Night splint	Incise inferior extensor retinaculum
	Shoe tongue padding	Decompress both medial and lateral branch of nerve (divide 1 cm proximal to ankle joint)
		Excise bone spur if present
Superficial peroneal nerve	Lateral shoe wedge	Identify nerve distally and trace proximally to level that it pierces crural fascia (10–12 cm proximal to tip of lateral malleolus)
	Ankle brace	Partial fasciotomy
	Physical therapy for peroneal strengthening and proprioception	Test for residual tethering with intraoperative plantarflexion
		Correct concurrent ankle instability

- Removal of space-occupying mass if present:
  - Greater rate of surgical success if nerve compression secondary to space-occupying lesion

See Table 3 for nerve-specific treatment information.

Complications

- Recurrence of nerve entrapment – most commonly due to incomplete decompression
- Revision surgery – decreased success rate

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4 Pes Planus

Take-Home Message
- Adult-acquired flatfoot secondary to dysfunction of the posterior tibial tendon is the most common cause of pes planus in adults.
- Correct flexible deformity with corrective osteotomy (medial slide calcaneal osteotomy) + soft tissue reconstruction (FDL tendon transfer).
- Correct fixed deformity with arthrodesis (triple arthrodesis).
- Correct forefoot varus/supination with Cotton osteotomy (dorsal opening wedge osteotomy medial cuneiform).

Definition

Pes Planus (Flat Foot)

- Loss of medial longitudinal arch.
- Hindfoot valgus (Fig. 11) with forefoot abduction (Fig. 12).
- Fixed supination of the forefoot occurs with long-standing disease (Fig. 13).

Etiology

Congenital (Flexible) Pes Planus

- Ligamentous laxity.
- Normal development of infants/children or normal adult variant.
- In many cases is asymptomatic and does not require treatment. May additionally suffer degeneration of the posterior tibial tendon during adulthood.

Acquired Pes Planus

- Insufficiency of the posterior tibial tendon with subsequent strain on the static medial stabilizers of the hindfoot. Asymmetric deformity in contrast to congenital pes planus.

Pathophysiology

Congenital Pes Planus

- Ligamentous laxity, hindfoot valgus, forefoot abduction
- Normal strength and integrity of the posterior tibial tendon

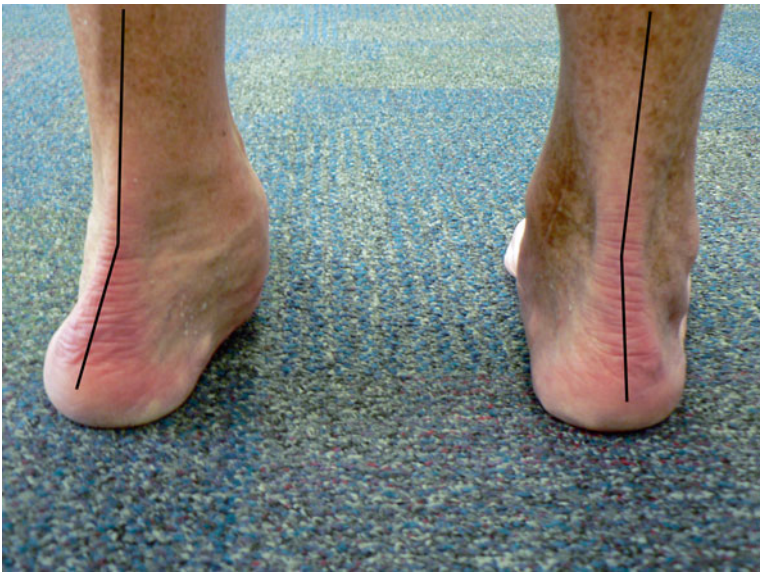

Fig. 11 The left hindfoot has significant hindfoot valgus compared to the right. This is indicative of failure of the ligamentous support of the hindfoot that occurs in stage II, III, and IV PTTD. Simple debridement of the tendon will fail once the hindfoot is in valgus

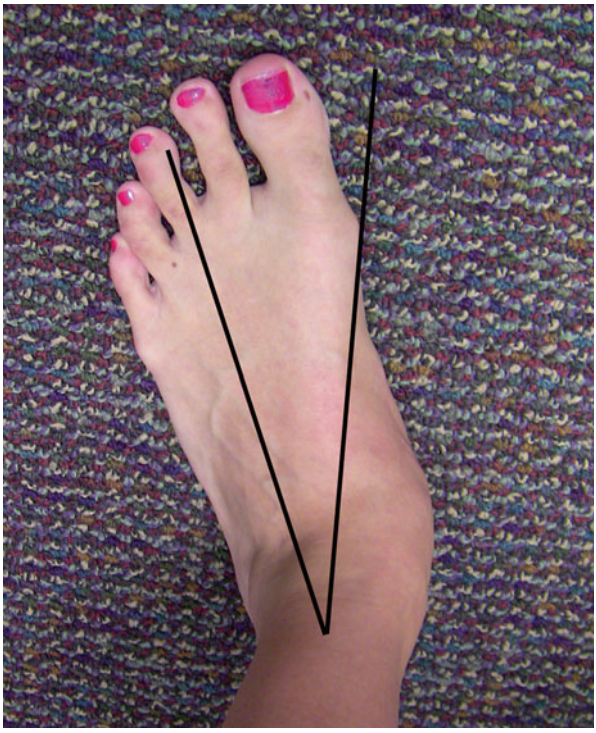

Fig. 12 The axis of the forefoot deviates into abduction relative to the axis of the hindfoot in patient with late-stage PTTD

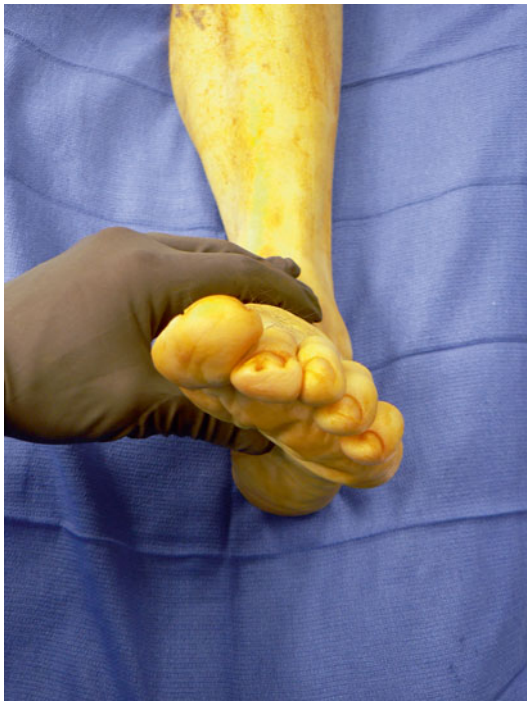

Fig. 13 Patient with fixed forefoot varus after correction of the hindfoot deformity

Acquired Pes Planus

1. Posterior tibial tendon (PTT) insufficiency – inability to perform a single-limb heel rise with associated pain and swelling (Fig. 14).
2. Loss of dynamic arch support.
3. Arch attenuation (spring ligament > talocalcaneal interosseous ligament > deltoid ligament).
4. Results in increased hindfoot valgus and forefoot abduction. More long-standing deformity results in fixed forefoot supination/varus.
5. Rigid deformity may occur with long-standing disease or arthritis.
6. Strain across the deltoid ligament may result in ankle valgus with persistent asymmetric ankle joint pressure, placing the joint at risk for ankle arthritis.

Radiography

Weight-Bearing AP/Lateral Foot/Ankle

- Talo-first metatarsal angle on AP (normal =0) – increased angle associated with flatfoot deformity.
- Talo-first metatarsal angle on lateral (normal =0) – negative angle (directed plantarward) associated with flatfoot deformity (Fig. 15).
- Talar head uncoverage – the amount of uncoverage (lack of apposition of the navicular) may direct surgical intervention (Fig. 16):
  - >50 % uncovering indicative to perform lateral column lengthening

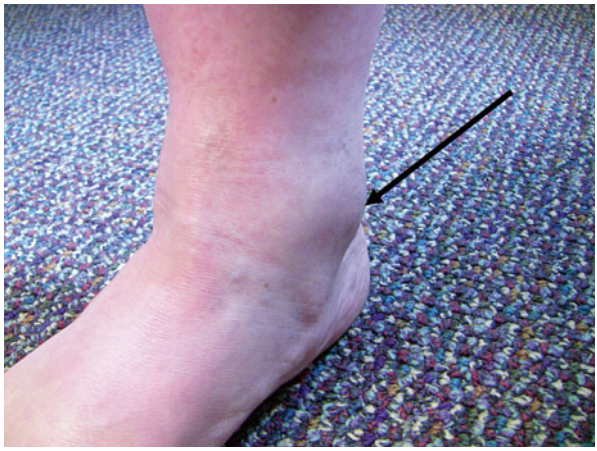

Fig. 14 Patient with complaints of posterior tibial tendon (PTT) dysfunction. Note the significant swelling along the posteromedial aspect of the hindfoot (*black arrow*). This corresponds to the course of the PTT and is typically tender to palpation

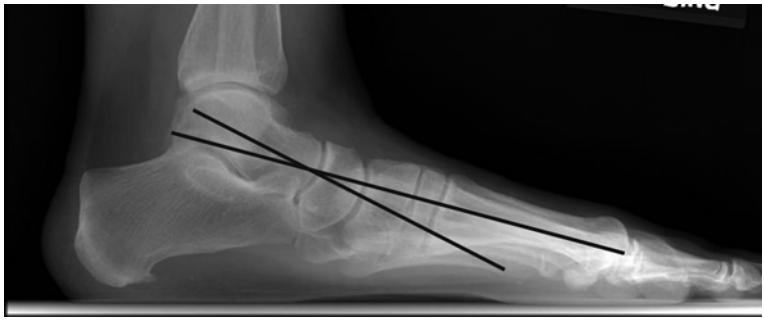

Fig. 15 Lateral weight-bearing radiograph of a patient with posterior tibial tendon dysfunction. Note the plantarflexed position of the talus relative to the first metatarsal

- Look for evidence of degenerative changes of the hindfoot – requires triple arthrodesis. Midfoot arthritis may be present and requires realignment midfoot fusion in addition to hindfoot correction.

AP/Lateral/Mortise Ankle

- Evaluate for valgus talar tilt, ankle arthritis

Ultrasound/MRI – not routinely utilized in the decision making process

Classification

- Truro classification: posterior tibial tendon dysfunction (Table 4)
- Nonoperative management for all stage II is best treated with an articulated AFO and physical therapy focused on strengthening of PTT. Stage III/IV is treated with a rigid AFO or Arizona brace.

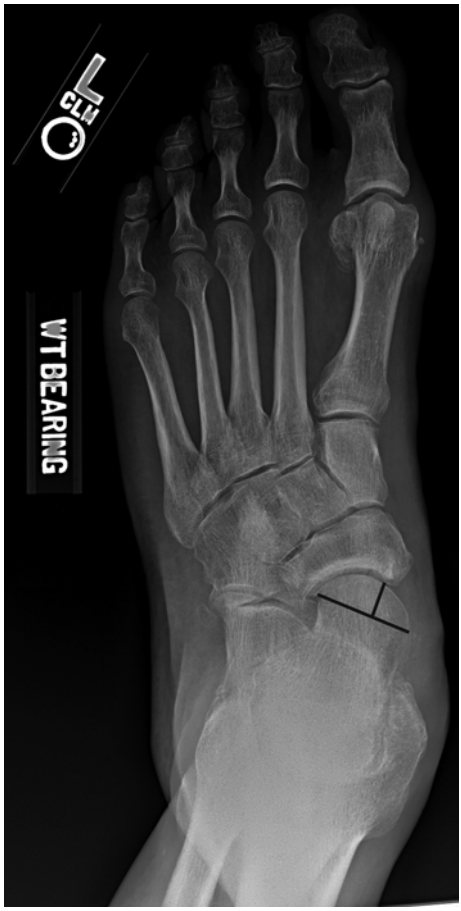

Fig. 16 AP weight-bearing radiograph of the foot in a patient with stage IIB PTTD. The navicular is laterally subluxated, resulting in greater than 30 % uncovering of the talar head

Table 4 Truro classification: Posterior tibial tendon dysfunction

Stage 1	No deformity
	Tenosynovitis
Stage 2A	Flexible hindfoot valgus
	Normal forefoot
Stage 2B/C	Flexible hindfoot valgus
	Forefoot varus >15° or fixed forefoot varus
Stage 3	Rigid hindfoot valgus
	Rigid forefoot abduction
Stage 4	Deltoid insufficiency (ankle valgus) or ankle arthritis

Table 5 Posterior tibial tendon dysfunction treatment options

	Nonoperative treatment	Operative treatment
Stage 1	Immobilization (CAM Walker)	Tenosynovectomy
	Orthotics (medial wedge and arch support) (Fig. 17)	Equinus correction (GSR/TAL)
	PT	
Stage 2A	Orthotics (articulated AFO with focused PT to strengthening PTT)	FDL transfer (Fig. 18) +
		Medial slide calcaneus osteotomy (Fig. 19)
		Lateral column lengthening (>50 % uncoverage)
		Equinus correction (GSR/TAL)
Stage 2B	Orthotics (articulated AFO with focused PT to strengthening PTT)	As per stage 2A
		Cotton osteotomy (stable first TMT) (Fig. 20) vs. first TMT fusion – arthritis or instability (Fig. 21)
Stage 3	Orthotics (AFO, Arizona brace)	Triple arthrodesis (subtalar, talonavicular, calcaneocuboid) (Fig. 22) (Some current consideration to perform isolated talonavicular and subtalar arthrodesis)
Stage 4	Orthotics (AFO, Arizona brace)	Rigid ankle: tibiotalocalcaneal arthrodesis or
		Pantalar fusion (Fig. 23)
		Flexible non-arthritic ankle: may consider deltoid reconstruction (Fig. 24) + triple arthrodesis with medial slide calcaneal osteotomy

Treatment

Congenital Pes Planus

Nonoperative: observation, heel cord stretching, orthotics (arch support)
Operative treatment: indicated for failure of nonoperative treatment

- Gastrocnemius recession (GSR) or tendo-Achilles lengthening (TAL) if equinus contracture
- Calcaneus osteotomy:
  - Calcaneal lengthening osteotomy (Evans)
  - Medial calcaneal slide osteotomy, medial cuneiform plantar closing wedge osteotomy, cuboid opening wedge osteotomy (triple C)

Acquired Pes Planus

Treatment (Table 5)

Complications

- Nonunion of arthrodesis
- Failure to achieve satisfactory correction of deformity
- Failure to achieve pain relief – 10 %

Fig. 17 Standard 3/4 length orthotic. Note the longitudinal arch support. Custom orthotics are useful if the patient has a severe deformity that would not be amenable to over the counter orthotics – however, custom orthotics are 10–20 times more expensive

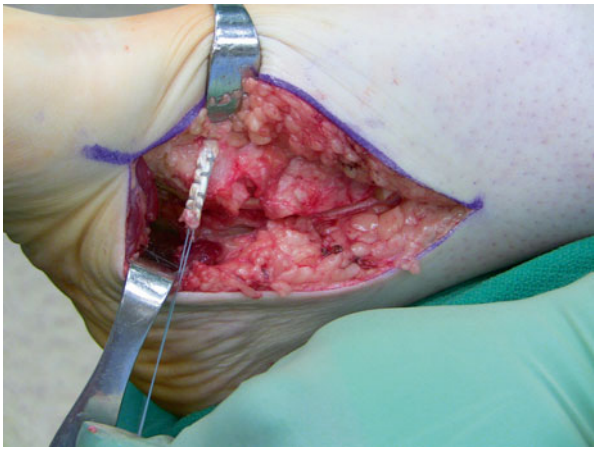

Fig. 18 Use of the FDL is the most common tendon transfer to recreate the function of the posterior tibial tendon. The tendon is placed through a drill hole in the navicular and routed from plantar to dorsal

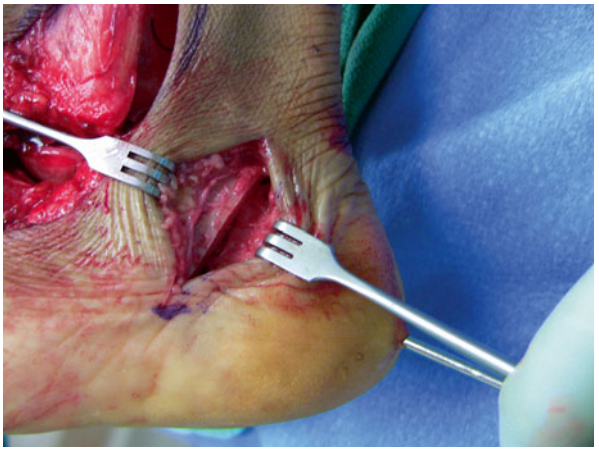

Fig. 19 Note the medial translation of the calcaneal tuberosity. A 1 cm shift is sufficient

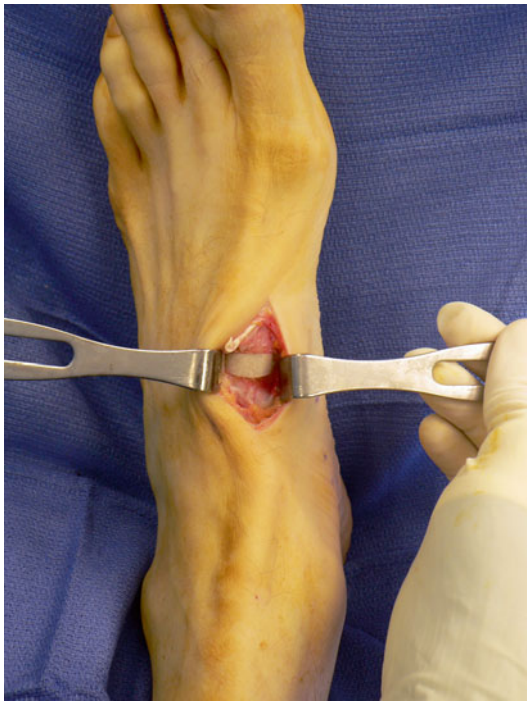

Fig. 20 Plantarflexion osteotomy of the medial cuneiform is commonly used to correct the fixed forefoot varus. Use of allograft to achieve the correction is demonstrated here

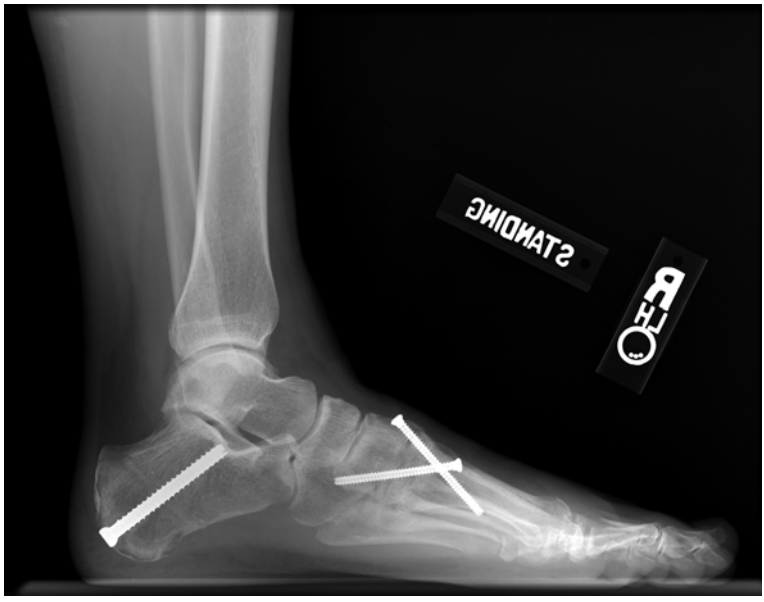

Fig. 21 Stabilization of the first tarsometatarsal (TMT) joint is critical in flatfoot reconstruction if there is instability of that joint. After joint preparation – the first MTP is dorsiflexed – which forces the first TMT joint to plantarflex secondary to the windlass mechanism. Fixation is typically performed with cross screws

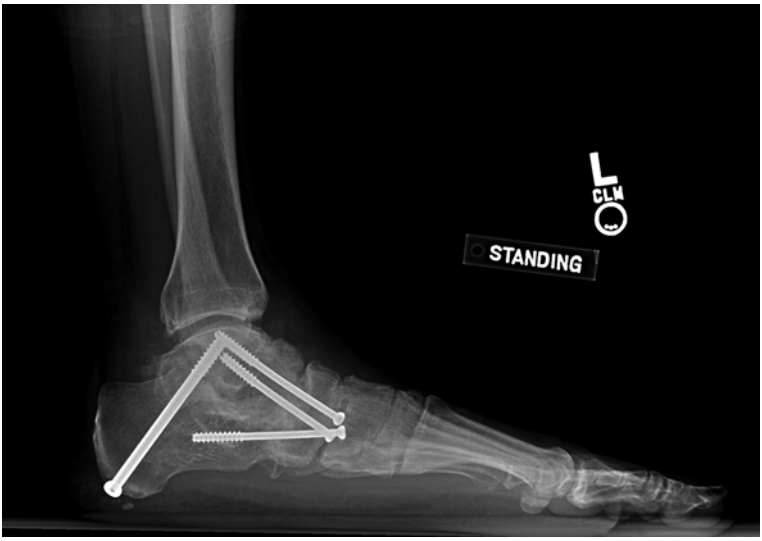

Fig. 22 Lateral radiograph of a patient who underwent a triple arthrodesis for a fixed hindfoot valgus deformity – stage III

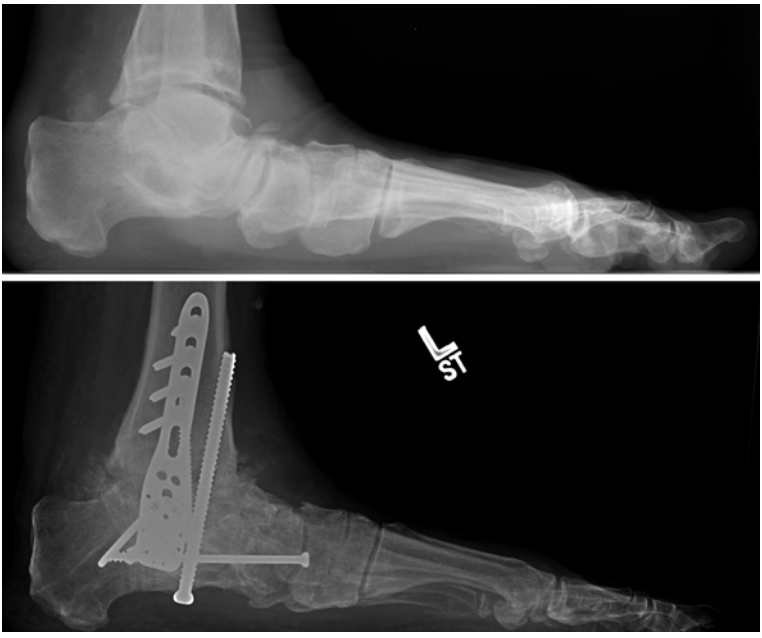

Fig. 23 Lateral radiograph of a patient with stage IV PTTD with ankle arthritis. A pantalar fusion is the gold standard in this case. Performing an ankle replacement and triple arthrodesis is possible – however, there are no long-term results of ankle replacements in this setting

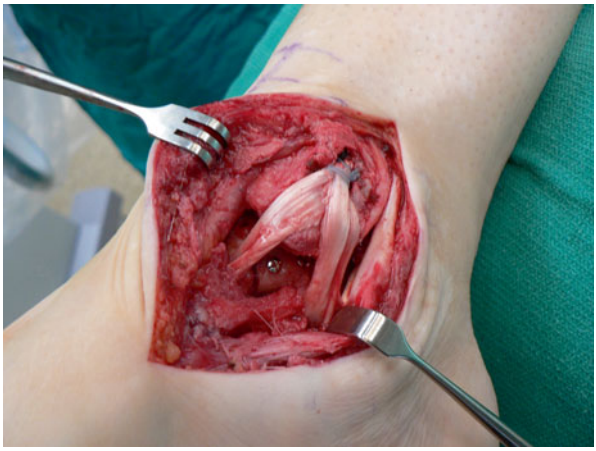

Fig. 24 Allograft reconstruction of the deltoid ligament can be performed in a patient with stage IV PTTD with ankle valgus without evidence of arthritis. Concomitant correction of the hindfoot deformity is required

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5 Pes Cavus

Take-Home Message

- Primarily progressive, eventually rigid deformity from prolonged muscle imbalance.
- Etiology primarily neurologic.
- Rigid deformities should be treated with corrective osteotomies as well as tendon transfers to remove deforming forces.

Definition Cavovarus:

- Hindfoot varus, forefoot pronation

Etiology

- Neurologic
  - Hereditary motor and sensory neuropathies – Charcot-Marie-Tooth (typically bilateral disease)
  - Cerebral palsy
  - Stroke
  - Spinal cord lesions (typically unilateral disease)
- Traumatic
  - Compartment syndrome
  - Talar neck malunion
  - Peroneal nerve injury
- Residual clubfoot
- Idiopathic

Pathophysiology

- Muscle imbalance
  - Strong peroneus longus (PL) and posterior tibialis (PT)
  - Weak peroneal brevis (PB) and anterior tibialis (AT)
- Posttraumatic
  - Deep posterior compartment contractures (PT, FDL)
- Prolonged deformity leads to plantar fascia contraction; flexible deformity becomes rigid.

Radiography Weight-bearing AP/lateral foot/ankle, calcaneus axial

- Hindfoot varus
- First MT plantarflexion

Classification Coleman block test – block under lateral forefoot

- If hindfoot corrects – flexible hindfoot deformity, forefoot driven
- If hindfoot does not correct – rigid hindfoot deformity rigid ± forefoot deformity

Treatment

Nonoperative

- Orthotics – lateral wedge, depression for first ray
- Ankle-foot orthosis with varus correction for rigid deformities

Table 6 Cavovarus surgical treatment options

Coleman block test finding	
Flexible hindfoot	PT/PL tendon transfer + plantar fascia release + tendo-Achilles lengthening (TAL) ±1st metatarsal (MT) dorsiflexion osteotomy
Rigid hindfoot	Above procedures + calcaneal osteotomy (lateral slide vs. closing wedge)
Hindfoot/midfoot arthritis or severe deformities	Triple arthrodesis + PT/PL tendon transfer

Operative (Table 6)

Complications

- Nonunion of arthrodesis
- Recurrence of deformity
- Wound breakdown/infection

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6 Tendinopathies

Take-Home Message

- Tendinosis is a chronic noninflammatory degeneration and should not be confused with tendonitis, an acute inflammatory disease.
- Nonoperative treatment should focus on eccentric strengthening.
- Operative intervention should focus on removing diseased tendon with appropriate tendon transfers if >50 % of operative tendon is removed.

Definition

- Tendonitis – inflammation of the tendon
- Tendinosis – chronic damage to tendons with microscopic analysis showing tissue necrosis and mucoid degeneration

Etiology

- Anterior tibial (AT) – tendonitis from overuse, partial/complete rupture from trauma. May present as a mass along the anterior ankle (pseudotumor) (Fig. 25).
- Flexor hallucis longus (FHL) – tendonitis from overuse (ballet dancers) or impingement/stenosis along posterior ankle. Prolonged symptoms can lead to partial rupture.
- Peroneal tendons
  - Subluxation/dislocation from forced eversion/dorsiflexion ->disruption of superior peroneal retinaculum (Fig. 26)
  - Tendon tears caused by degeneration from tendon subluxation/dislocation, ankle sprains, ankle instability

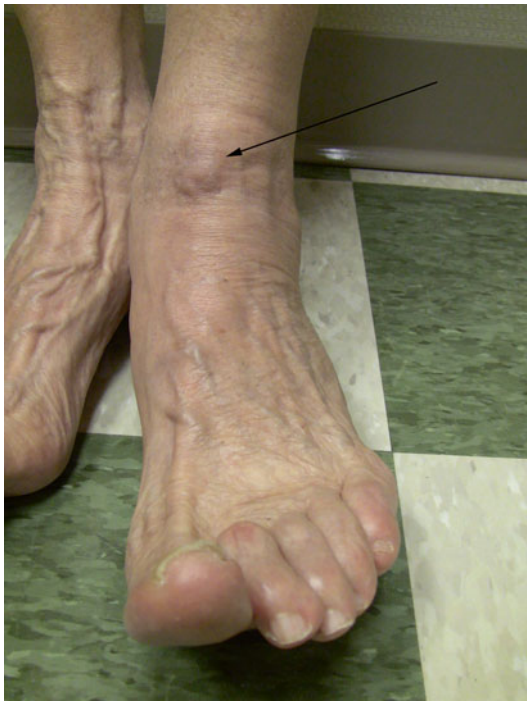

Fig. 25 Patient with a chronic anterior tibial tendon rupture. Note the extensor recruitment and the stump of the anterior tibial tendon (*arrow*) at the level of the ankle. This patient presented for evaluation of ankle mass

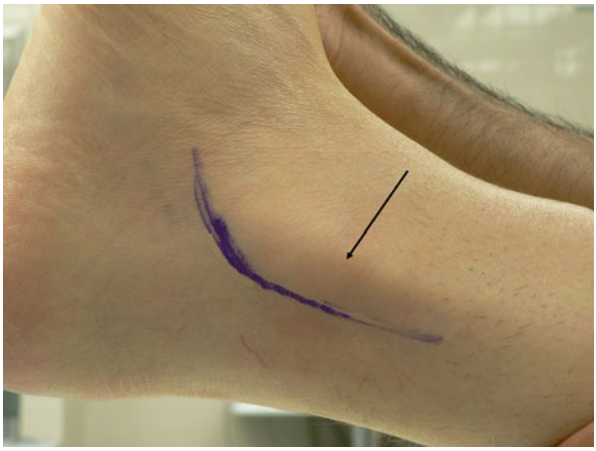

Fig. 26 Subluxation of the peroneal tendons (*arrow*). Note the tendons are displaced anterior to the posterior border of the fibula (*line*)

- Achilles
  - Insertional tendinopathy – tendinosis (cause unknown)
  - Non-insertional tendinopathy – inflammation of peritenon alone from overuse, fluoroquinolone antibiotics, mechanical imbalance
  - Rupture – "weekend warrior," sudden active plantarflexion against resistance or forced dorsiflexion in plantarflexed foot

Pathophysiology

- Tendinosis degenerative changes → collagen fiber disorientation; hypocellularity; patchy necrosis, calcification; minimal inflammatory cells

Radiography

AP/Lateral Radiographs of the Foot/Ankle

- Fracture of lateral ridge of distal fibula (peroneal subluxation/dislocation)
- Fracture of os peroneum (peroneal longus rupture)
- Superior calcaneal tuberosity bone spur and calcification at Achilles insertion (insertional Achilles tendinopathy) (Fig. 27)

MRI

- Fluid around FHL – high-signal intensity surrounding the tendon (Fig. 28)
- Achilles thickening with intermediate intrasubstance signal (non-insertional tendinopathy) (Fig. 29)

Ultrasound

- Dynamic exam peroneal subluxation/dislocation

Classification Ogden classification of superficial peroneal retinaculum (SPR) tears (Table 7)

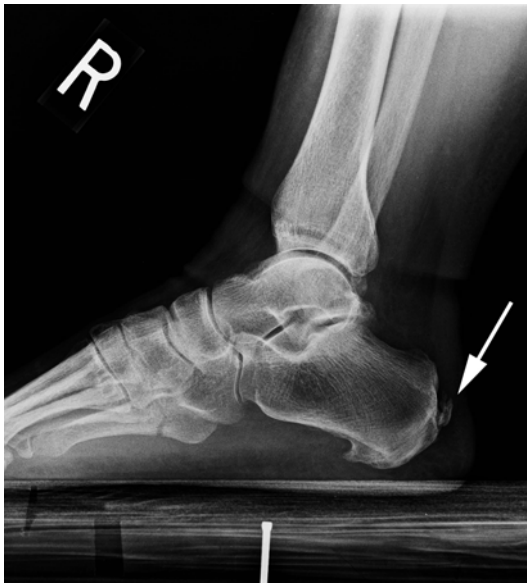

Fig. 27 Lateral radiograph demonstrating calcification of the Achilles tendon at the insertion (*arrow*)

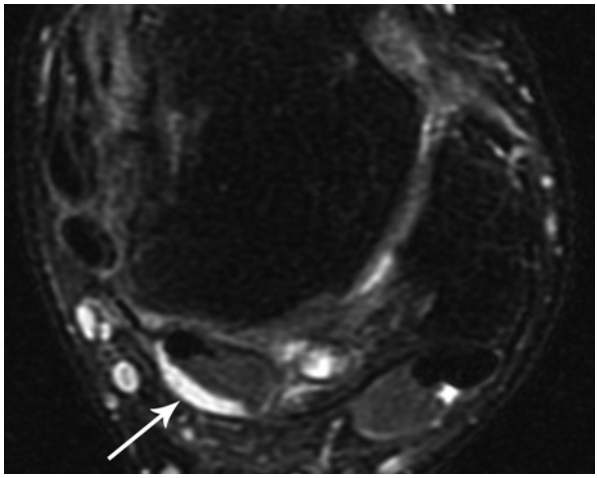

Fig. 28 Axial T2 image consistent with synovitis of the flexor hallucis longus. Note the increased signal intensity (*arrow*) surrounding the tendon

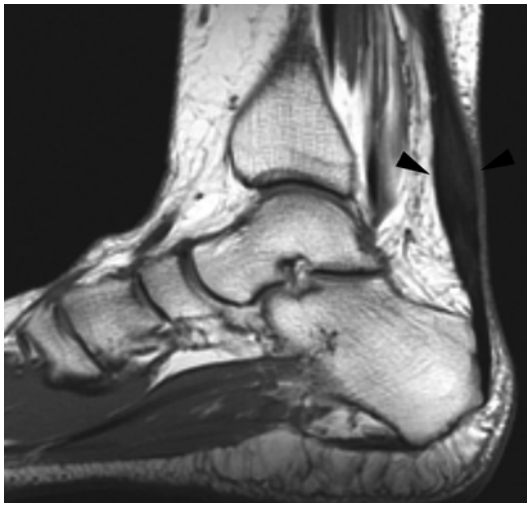

Fig. 29 Sagittal T1 imaging of a patient with non-insertional Achilles tendinosis. Note the thickening of the tendon (*arrowheads*) and the intermediate-signal intensity within the tendon consistent with degeneration

Table 7 Ogden classification of Superficial Peroneal Retinaculum (SPR) tears

Grade 1	SPR partially elevated off fibula
Grade 2	SPR separated from cartilofibrous ridge lateral malleolus, subluxation between SPR and ridge
Grade 3	Cortical avulsion SPR off fibula, subluxation under fracture
Grade 4	SPR avulsed from calcaneus

Table 8 Anterior tibialis and FHL treatment options

	Nonoperative	Operative
Anterior tibialis	NSAIDs, CAM boot or walking cast	Acute rupture – primary repair
		Chronic rupture – reconstruction with interpositional graft
Flexor hallucis longus	Activity modification	Tenosynovectomy, fibro-osseous tunnel release

Treatment

Treatment of anterior tibialis, flexor hallucis longus disorders (Table 8)
Treatment of peroneal tendon disorders (Table 9)
Treatment of Achilles tendon disorders (Table 10)

Complications

- Wound breakdown/infection
- Tendon re-rupture

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Table 9 Peroneal tendon treatment options

	Nonoperative	Operative
Peroneal tendon subluxation/ dislocation	Cast immobilization, protected weight bearing 6 weeks (acute injuries in reduced position)	Reconstruction SPR, fibular groove deepening
Peroneal tendon tear	NSAIDs, activity modification, physical therapy, lace-up ankle brace	<50 % diseased tendon – tenosynovectomy, debridement, tendon repair ± groove deepening
		> 50% diseased tendon or complete rupture 1 tendon – excision and tenodesis
		>50% diseased tendon or complete rupture 2 tendons – FHL transfer to fifth metatarsal (MT)
		Hindfoot varus – lateral closing wedge calcaneus osteotomy (Dwyer)

Table 10 Achilles tendon treatment options

	Nonoperative	Operative
Achilles insertional tendinopathy	Activity/shoe modification, heel lifts, stretching, physical therapy (eccentric strengthening), silicone sleeves/pads, NO corticosteroid injections	Debridement degenerative tendon, resection prominent superior calcaneal tuberosity, excision retrocalcaneal bursa
		If >50 % tendon detached, reattach insertion with suture anchors
		If >50 % tendon excised, FHL transfer
Achilles non-insertional tendinopathy	Activity modification, heel lifts, physical therapy (eccentric strengthening), shock-wave therapy	Mild disease – percutaneous tenotomies
		Moderate disease – debridement, tendon tubularization
		If >50 % tendon excised, FHL transfer
Achilles rupture	Acute – bracing/cast in equinus with early functional rehabilitation (no difference in re-rupture rate)	Acute – direct repair (improved satisfaction in age <45)
	Chronic – AFO	Chronic – VY advancement ± FHL transfer

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7 Diabetic Foot

Take-Home Message
- The diabetic foot is at a constant risk for recurrent ulcerations, especially due to neuropathy and peripheral vascular disease.
- Ulcer classification is essential to set a treatment strategy and determine prognosis.
- Treatment in the setting of infection should include culture-directed therapy and a thorough understanding of the different levels of amputations.

Introduction

- Diagnosis of foot ulcerations results in the greatest rate of hospital admissions in diabetics, as well as lower extremity amputations.

Pathophysiology

- Diabetic neuropathy:
  - Sensation: sensory loss begins in stocking distribution and progressive proximally. Ninety percent of patients who cannot feel the 5.07 monofilament have lost protective sensation to their feet and are at risk for ulceration.
  - Autonomic neuropathy: abnormal sweating mechanism leads to a dry foot → vulnerable to fissuring cracks → portals for infection.
  - Motor neuropathy: most commonly involves the common peroneal nerve → resultant loss of tibialis anterior function and foot drop; small intrinsic musculature also affected → claw toes and subsequent toe-tip ulcerations.
- Hypomobility syndrome:
  - Decreased range of motion in joints from excessive glycosylation of soft tissues
- Peripheral vascular disease:
  - Occurs in 60–70 % of patients who have diabetes for over 10 years, involving both large and small vessels.
  - Noninvasive vascular examination should be performed when pulses not palpable (normal waveform is triphasic); ankle brachial index of 0.45 is minimum for healing and greater than 1.3 is consistent with calcification of vessels.
  - Minimum toe pressures for healing: 40 mmHg.
  - Transcutaneous oxygen measurements of the toes greater than 40 mmHg have been found to be predictive of healing.

Table 11 Wagner grading system for diabetic foot infections

Grade	Depth of ulcer
0	Skin intact with bony deformity that leads to "at risk" foot
1	Localized superficial ulcer without tendon or bone involvement
2	Deep ulcer with exposed tendon or joint capsule
3	Extensive ulcer with exposed bone/osteomyelitis or abscess
4	Partial gangrene
5	Extensive gangrene

Table 12 Brodsky grading system: Based on ischemia

Grade	
A	Normal vascularity
B	Ischemia without gangrene
C	Partial gangrene
D	Complete gangrene

- Immune system impairment:
  - Poor cellular defenses, altered chemotaxis of white blood cells, and poor cytotoxic environment (due to hyperglycemia) to fight bacteria lead to difficulty in fighting off infection.
- Metabolic deficiency:
  - Total protein less than 6.0, WBC count less than 1,500, and albumin levels less than 2.5 result in poor healing potential.

Radiography

- Routine radiographic series of the foot and ankle should be obtained to determine the extent of bone loss and deformity. Findings can be difficult to interpret in the setting of neuroarthropathy. Bony desctruction directly over an open ulcer is highly suspicious for osteomyleitis.
- MRI can be obtained is an abscess is suspected.
- MRI has high false-positive rate in the diagnosis of osteomyelitis, particularly with concurrent Charcot arthropathy.
- WBC-labeled scan or dual-image Tc/In scan is more sensitive and specific for osteomyelitis than isolated technetium scan.

Classification Wagner classification: based on depth of ulcer (Table 11)
Brodsky classification: based on ischemia (Table 12)

Table 13 Diabetic ulcer management based on the Wagner Grading system

Grade	Treatment
0	Extra-depth shoe and pressure relief insoles
1	In office debridement, shoe modification or total contact cast if no infection
2/3	Operative debridement of all exposed bone/tendon and nonviable tissue. Dressing changes and total contact casting once wound bed is healthy
4/5	Local vs. larger amputation

Treatment

Ultimate goal is an ulcer-free, functional, plantigrade foot that can fit within a brace or shoe (Table 13).

- The Therapeutic Shoe Bill allocated money for neuropathic patients to purchase extra-depth shoes and total contact inserts (3 per year) for ulcer prevention.
- Workup should include noninvasive vascular studies and surgical revascularization if indicated and metabolic assessment with delay in surgery if possible until nutritional status is improved.
- Additional treatment includes:
  - Tendo-Achilles lengthening to offload the midfoot/forefoot if recurrent ulcerations with equinus deformity.
  - Ostectomy of bony prominences (stable deformity) or fusion if instability present.
  - Toe deformities often require joint resection or amputation.
- Diabetic foot infections are polymicrobial:
  - Should not perform superficial wound culture → deep cultures provide most accurate results.
  - Treat infections with initial broad-spectrum antibiotic coverage once surgical cultures obtained, and adjust once sensitivity returns.
  - Abscesses require surgical drainage and antibiotics.
  - Osteomyelitis is treated with antibiotics and usually surgical debridement.
- Amputation as needed at different levels with appropriate tendon transfers:
  - Transmetatarsal → lowest energy expenditure, no tendon transfer needed
  - Lisfranc → requires a transfer of peroneals to cuboid to prevent varus; Achilles lengthening
  - Chopart → requires transfer anterior tibialis to talus to prevent equinus; Achilles lengthening
  - Syme's → next lowest energy expenditure (superior to Lisfranc and Chopart with regard to amount of energy needed to ambulate)
  - Transtibial → superior results with postoperative casting for 3–5 days with conversion to rigid removal dressing

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8 Charcot Arthropathy

Take-Home Message

- Charcot arthropathy is a limb-threatening destructive process that occurs in patients with sensory, motor, and autonomic neuropathy associated with medical diseases such as diabetes mellitus.
- The Eichenholtz classification describes the evolution of the condition through time, whereas the Brodsky classification is defined by location.
- Initial treatment consists of prompt immobilization and non-weight bearing but may warrant arthrodesis or amputation with long-standing deformity.

Definition

- Chronic, progressive, noninfectious destructive process affecting bone architecture and joint alignment in people lacking protective sensation (Fig. 30)

Etiology

- In developed world, diabetic neuropathy is the most frequent cause of Charcot arthropathy.
- Other possibilities are alcoholism, leprosy, tabes dorsalis, myelomeningocele, and congenital insensitivity to pain.
- Often challenge is determining whether there is superimposed osteomyelitis.

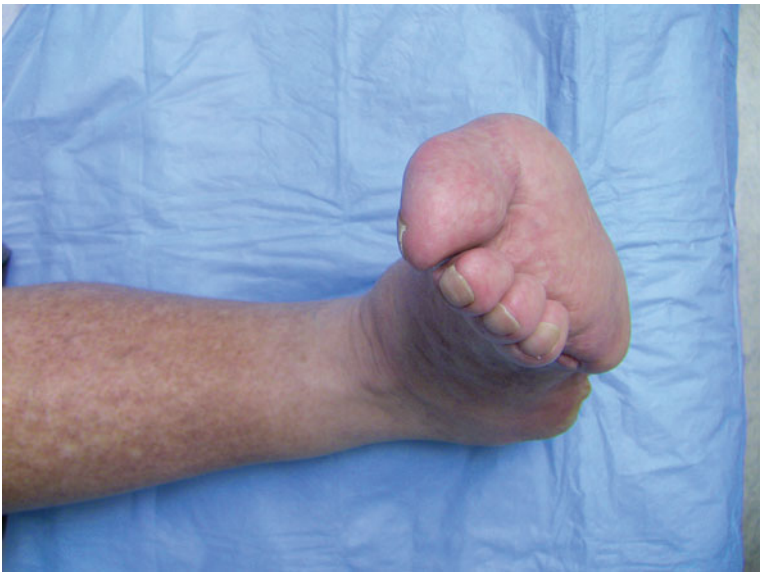

Fig. 30 Varus deformity of the ankle and hindfoot is a common deformity seen in Charcot. The deformity can be severe with some patient presenting with callous or ulcer over the fibula as it now contacts the ground

Pathophysiology Two traditional theories:

- Neurotraumatic: exaggerated overuse injury where insensate joints that cannot adopt normal protective mechanisms are subjected to repetitive microtrauma
- Neurovascular destruction: autonomic dysfunction leads to increased blood flow, resulting in osteoclast stimulation, bone resorption, and weakening

Likely results from a combination of these processes → the development of abnormal bone with no ability to protect the joint results in gradual bone fracture and subluxation of the joint (Fig. 31).

Radiography

- Radiographic appearance dependent on what stage the disease is in (see below).
- The hallmark deformity associated with this condition is midfoot collapse, described as a "rocker bottom" foot (Fig. 32).
- Technetium bone scan may be positive in all stages. Indium WBC scan may be negative for neuropathic joints and positive for osteomyelitis.
- Difficult to differentiate infection from Charcot based on MRI, although MRI allows detection of subtle changes in early stages of Charcot arthropathy.

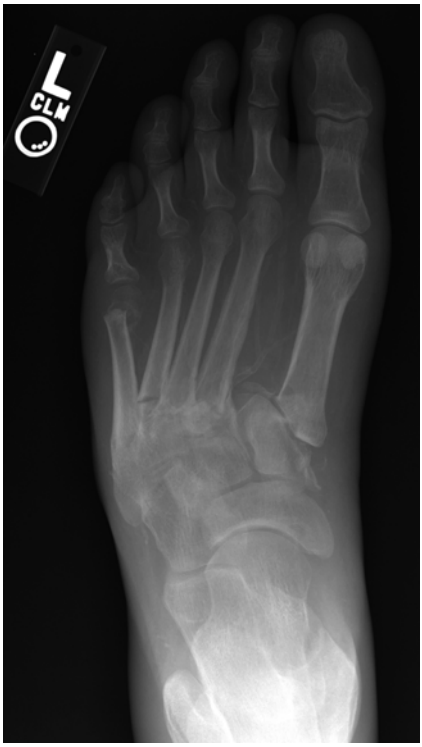

Fig. 31 AP radiograph of a patient with midfoot Charcot in Eichenholtz stage 1. The patient had 2 weeks of swelling without significant trauma. Note the fractures and dislocations through the tarsometatarsal joints

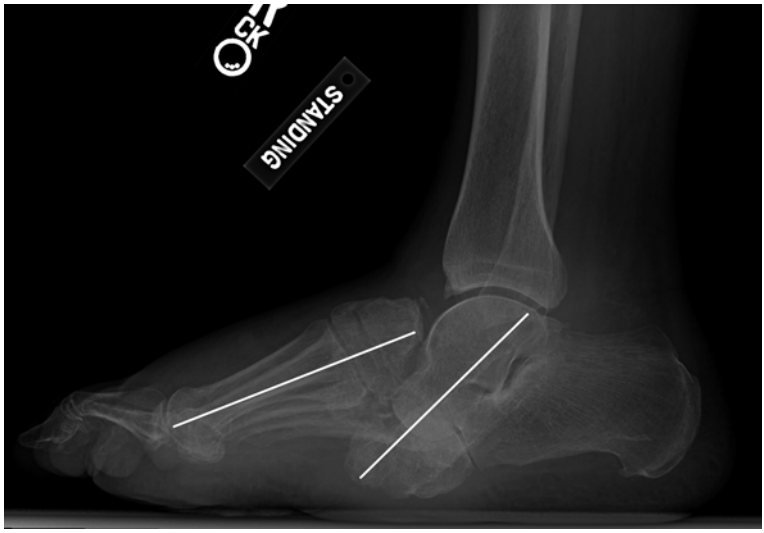

Fig. 32 Rocker bottom deformity in a patient with hindfoot Charcot. Note the severe plantarflexed position of the talus relative to the forefoot and the break through the talonavicular and metatarsocuboid joint. Plantar prominence of the cuboid risks ulceration in this patient

Table 14 Modified Eichenholtz stages for neuroarthropathy

Stage	Clinical manifestations	Radiographs
0: Pre-fragmentation	Acute inflammation, swelling, erythema with dependent rubor; confused with infection. Lack of systemic symptoms. Pain may not always be present	Normal or regional bone demineralization
1: Fragmentation (dissolution)	Acute inflammation, swelling, erythema, warmth (typically >2.0 °C in the affected foot). May have ligamentous laxity	Osseous periarticular fragmentation, joint subluxation/dislocation
2: Coalescence	Decreased local swelling, erythema, and warmth	Absorption of fine bone debris, early bone healing and periosteal new bone formation
3: Resolution	Resolved inflammation, more stable but often deformed	Consolidation and remodeling of fracture fragments, joint arthrosis, osteophytes

Table 15 Modified Brodsky classification for neuroarthropathy

Type	Location
Type 1 (most common – about 60 %)	Tarsometatarsal and naviculocuneiform joints (leads to fixed rocker bottom deformity)
Type 2	Subtalar, talonavicular, or calcaneocuboid joints (unstable, requires long periods of immobilization)
Type 3	Tibiotalar joint (late varus or valgus deformity produces ulceration and osteomyelitis of malleoli)
Type 4	Combination of joints
Type 5	Only within the forefoot

Classification Modified Eichenholtz stages: related to the degree of warmth, swelling, and erythema. Continuum from resorption and fragmentation to bone formation and consolidation that takes 6–18 months (Table 14).
Modified Brodsky classification: based on anatomic location (Table 15)

Treatment

- The goal is to achieve stage 3 (resolution) while maintaining alignment and ambulatory status and minimizing soft tissue breakdown.

Initial

- Frequent follow-up with serial radiographs and patient education on diabetic foot care.
- Immobilization and non-weight bearing → best with serial total contact casting (the aim is to achieve homogenous pressure distribution in plantar arch through gait); can transition to custom brace (AFO or Charcot restraint orthosis walker (CROW) boot) once swelling and warmth subsides.
- Some studies use bisphosphonates to help reduce osteoclastic resorption and increase the osteoblastic redeposition of bone.

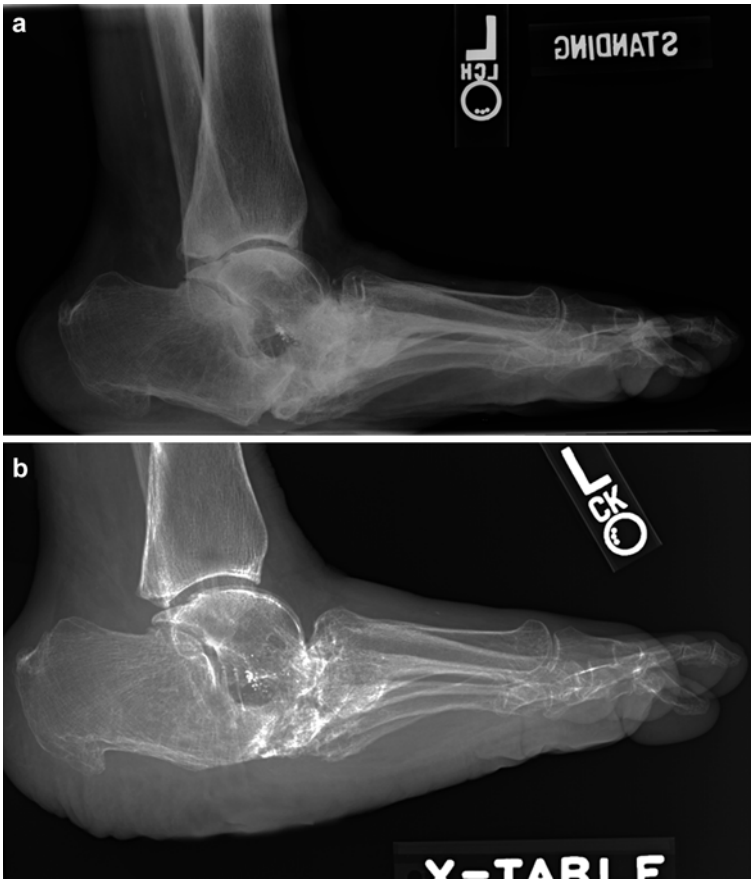

Fig. 33 Patient with a rocker bottom deformity with a large plantar prominence (**a**). The patient is in the consolidated phase and therefore exostectomy can be performed (**b**). The exostectomy removes the offending prominence, but does not correct the malalignment of the joints

Surgical

- Stable deformity with recurrent ulcers secondary to prominence → exostectomy (Fig. 33)
- Unstable/un-braceable deformity → arthrodesis with internal or external fixation (Fig. 34)
- Tendo-Achilles lengthening almost universally required
- Amputation as salvage procedure (Fig. 35)

Complications

- High complication rate (up to 70 %)
- Infection, hardware malposition, recurrent ulceration, fracture

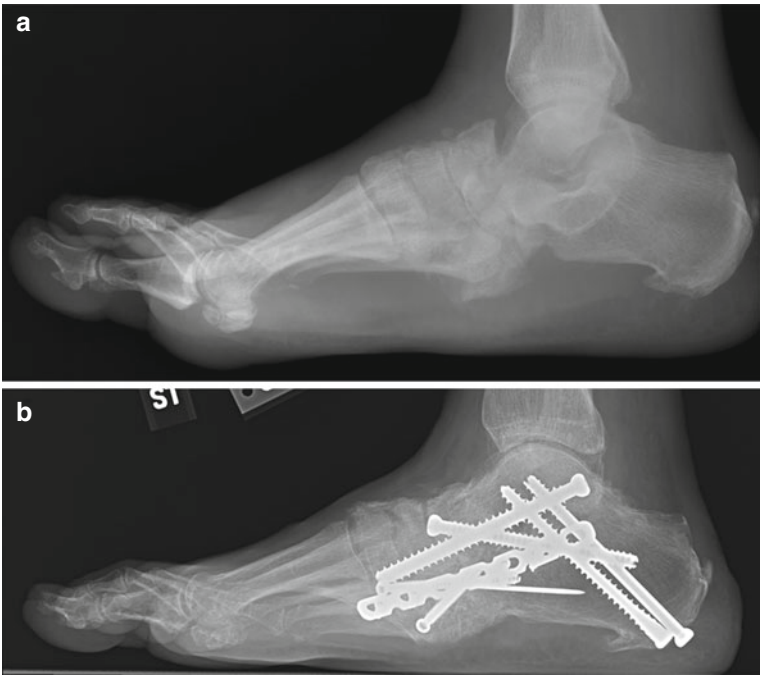

Fig. 34 Patient with hindfoot Charcot and rocker bottom deformity (**a**) treated with an extended hindfoot arthrodesis (**b**)

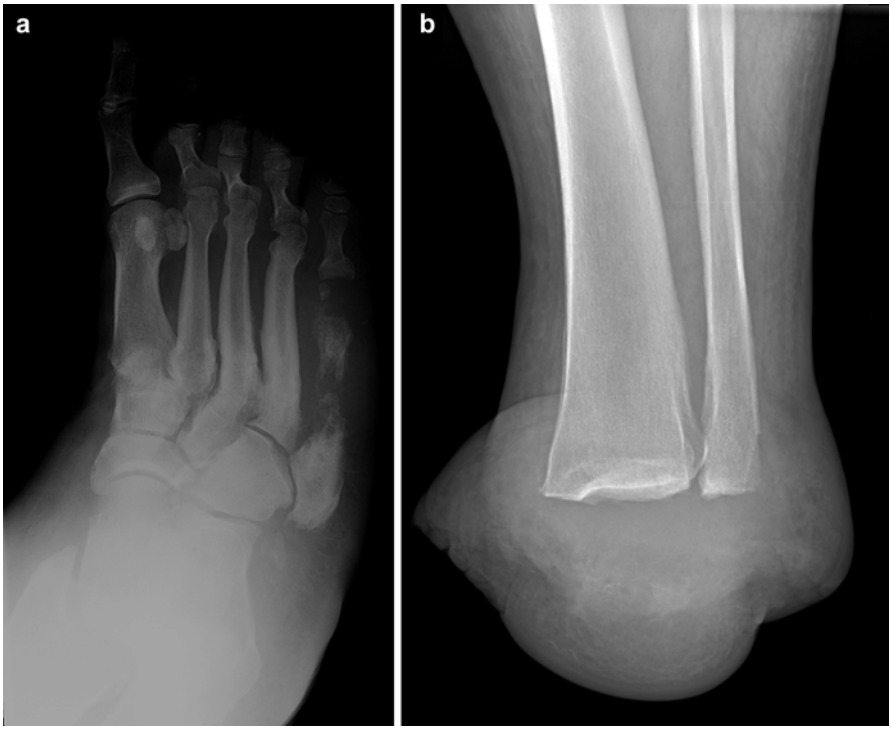

Fig. 35 AP radiograph of patient with poorly treated diabetic neuropathy with osteomyelitis of the fifth metatarsal (**a**). Reconstructive surgery was not possible and a Syme's amputation was performed (**b**)

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