The Foot

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The foot has two key functions:

1. Providing stability and balance to the body when standing to support the weight of the body
2. Acting as a rigid lever to push off when a person is walking or running, as well as to allow one to be mobile enough to adapt to uneven ground and absorb shock during mobilization

Brief Discussion on Arches of the Foot

There are four main arches in the foot to consider. Two are longitudinal arches running proximally to distal and two run from medial to lateral. The arches of the foot act as a spring, enabling the foot to bear weight, as well as absorb shock, during movement:

1. The medial longitudinal arch, which runs from the calcaneus to the head of the first metatarsal, includes the bones in between; the talus; the navicular; the first, second, and third cuneiforms; and the first, second, and third metatarsal heads.

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The summit is the midtarsal joint. It is the higher of the two longitudinal arches and confers significant flexibility to the foot. The plantar calcaneonavicular ligament (spring ligament) confers significant support. The main supporting tendon is the tibialis posterior.

2. The lateral longitudinal arch runs from the calcaneus to the fourth and fifth metatarsal heads and includes the cuboid and the fourth and fifth metatarsal bones. This arch is flatter than the medial one. In the standing position, it lies flat to the ground. It is supported by the long plantar and plantar calcaneocuboid ligaments.
3. The metatarsal arch extends in the coronal plane from the first to the fifth metatarsal heads with the summit at the second and third metatarsals.
4. The transverse arch of the foot is also located in the coronal plane. It is an incomplete arch consisting of the metatarsal bases, the cuboid, and the three cuneiform bones. Its peak is the base of the second metatarsal acting as the keystone. This arch is disrupted as part of traumatic Lisfranc disruptions.

Biomechanics and Movements of the Foot

The biomechanics of the foot and ankle are important to the normal functioning of the lower extremity. The main function of the foot is mechanical, i.e., to transfer loads from the leg to the ground. With appropriate neuromuscular coordination, the human foot, as a final link, remarkably adapts both in static and dynamic situations to a variety of ground terrains. Inadequate distribution of these forces could lead to abnormal stress and the eventual breakdown of soft tissues. The foot has got both dynamic and static supports. The static structures include the bones, joint surface congruity, ligaments, and fascia. The dynamic components include the tendons and muscles.

Muscle activity is not necessary to support the fully loaded foot at rest. The maintenance of the arch in the static foot is attributed to ligamentous and osseous support. In the static stance position, the plantar aponeurosis takes up approximately 60% of the stress of weight-bearing and the beam action of the metatarsals approximately 25%. The ability of the plantar aponeurosis to absorb stress increases as the aponeurosis becomes taut with toe extension. This mechanism has been described as the windlass effect [1].

The foot is made up of 28 bones and approximately 44 articulations. Consequently, most movements of the foot involve a large number of joints, and the movement of the individual joints in each movement is difficult to describe. The movement of the ankle joint is triplanar (occurring simultaneously in the sagittal, coronal, and horizontal planes) with predominant movement in the sagittal plane, i.e., plantar flexion (extension) and dorsiflexion (flexion). Subtalar is part synovial and part syndesmosis. The subtalar joint is part and parcel syndesmotic. The anterior syndesmotic part is separated from the posterior syndesmotic part by a funnel-shaped channel called the sinus tarsi, which is more or less horizontal and runs in a posteromedial to anterolateral direction. The subtalar joint is often regarded as a major contributor to the pronation and supination range of

motion, but results show that the contribution of the subtalar joint (24%) is less than that of the talonavicular joint (44%).

Pronation involves simultaneous abduction, dorsiflexion, and eversion. Similarly, supination involves simultaneous adduction, plantar flexion, and inversion. Pronation and supination are rear-foot movements when the foot is not weight-bearing [2]. During weight-bearing, these movements are constrained, depending on the magnitude and distribution of the ground reaction force acting on the plantar part of the foot.

When the whole foot is rotated internally about a longitudinal axis such that the sole faces medially, the foot is inverted; the opposite is eversion, which occurs in the subtalar joint. When the anterior part of the foot moves on the posterior part on a vertical axis, the foot is said to be adducted, while the opposite movement is called abduction. Both adduction and abduction occur at the talonavicular joint. The standard range of movements for dorsiflexion is 30°, and plantar flexion is 50°. At the subtalar joint, there is 20° of inversion and 10° of eversion.

Flatfoot

When discussing about flatfeet, it is best to take an approach broadly, dividing the topic into congenital and acquired pathologies. It also works to divide the topic into a younger population and an older/acquired population.

Three main congenital flatfoot pathologies exist: congenital vertical talus, flexible pes planus, and calcaneovalgus:

Congenital Vertical Talus

Congenital vertical talus is a rare foot deformity wherein patients present with an irreducible dorsal dislocation of the navicular on the talus. Half of the cases are bilateral with an unknown etiology, and 50% have associations with neurologic/neuromuscular conditions, congenital abnormalities, or chromosomal/genetic defects. Common conditions of association include arthrogryposis and myelomeningocele. There may be a positive family history in a fifth of cases with inheritance in an autosomal dominant fashion.

Congenital vertical talus manifests at birth with a rigid, fixed equinus hindfoot deformity. The hindfoot is in valgus due to the pull of a tight tendo Achilles, as well as tight posterolateral capsular structures of the ankle and the subtalar joint. The midfoot is pulled into dorsiflexion by tight anterior tendinous structures. The posterior tibialis tendon and the peroneus tertius are dislocated and act as dorsiflexors rather than plantar flexors. This gives the foot a convex/rocker bottom plantar surface, as well as deep creases in the dorsum. The talar head is palpable in the medial plantar arch, and the navicular is dislocated laterally and gives the midfoot fixed dorsiflexion.

A thorough family history may be positive for congenital vertical talus. A neurological exam is important as the patient may require imaging of the neurological axis. Plain radiographs have shown a vertical talus, the calcaneum in equinus, and a dorsally dislocated navicular. The lateral talocalcaneal angle is increased (Fig. 39.1).

Three lateral views are necessary (maximum dorsiflexion, maximum plantar flexion, and neutral) to show a rigid deformity. The anteroposterior (AP) radiograph demonstrates an increased talocalcaneal angle greater than 40°. A less severe form exists in the shape of an obliquely orientated talus, and imaging (forced plantar flexion radiograph) distinguishes it from a vertical talus (Fig. 39.2).

Management has trended away from surgery to serial manipulation and casting in the form of reverse Ponseti technique, followed by percutaneous Achilles tenotomy and fixation of the talonavicular joint [3]. Initially, the foot is pushed into plantar flexion and inversion/adduction. This is done progressively, with weekly cast changes with the final cast placing the foot into extreme equinovarus. K-wire stabilization of the talonavicular joint and Achilles tenotomy follows. Percutaneous capsulotomy may be necessary to achieve full reduction prior to K-wire fixation. A period of bracing follows as per the normal Ponseti regimen. More invasive

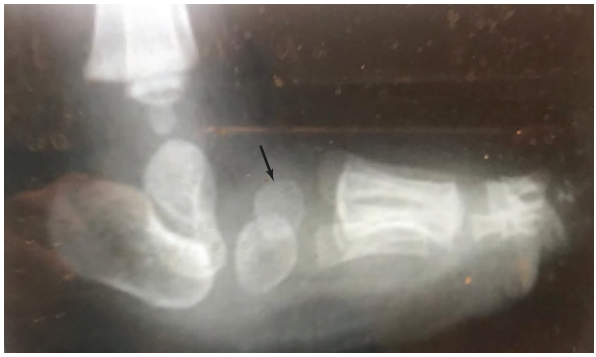

Fig. 39.1 Lateral view of the foot showing a vertical position of the talus and an increased talocalcaneal angle. The arrow is pointing at the navicular, which is dorsally dislocated

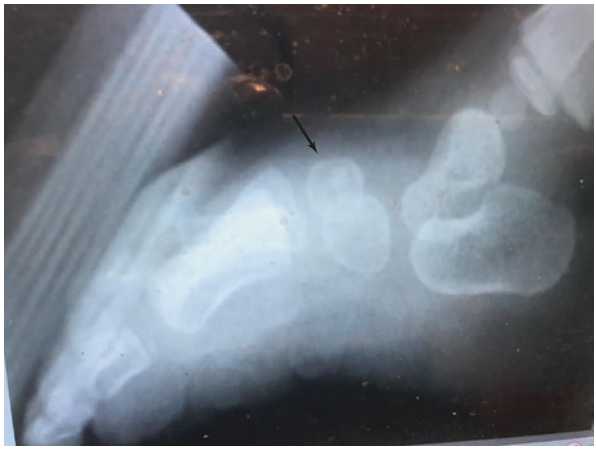

Fig. 39.2 Forced plantar flexion lateral view showing the navicular (arrow), which is still dorsal dislocated, distinguishing it from an obliquely oriented talus

procedures may be necessary and are performed at 6–12 months old, including tendon lengthening, spring ligament reconstruction, and tibialis anterior tendon transfer to the talar neck. Recalcitrant cases need more invasive open procedures, with fusion being an option in later life.

Calcaneovalgus

Calcaneovalgus is a congenital flexible deformity that is due to intrauterine malpositioning. Here the foot is in extreme dorsiflexion at the hindfoot, commonly with the dorsum of the foot flat, touching the anterior aspect of the tibia (Fig. 39.3).

The foot is also everted and abducted. It is passively correctable and thus can be differentiated from congenital vertical talus. It has preponderance in firstborn children and females. Plain radiographs, taken on the foot in plantar flexion, will show a line drawn through the talus lining up with the first metatarsal to differentiate it from congenital vertical talus. Management involves passive stretching with the addition of serial casting if necessary. Parent reassurance is important in these cases.

Flexible Pes Planus

Flexible pes planus is a relatively common finding in the pediatric population. But a lack of understanding may lead to these patients engaging with an orthopedic surgeon. In this group of patients, during standing, the foot is flat with the heel in valgus. Upon tiptoeing, the arch reconstitutes, hence the term flexible flatfoot.

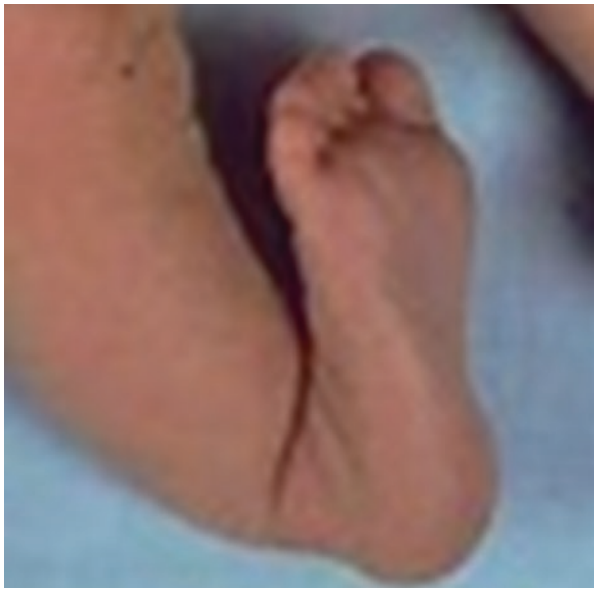

Fig. 39.3 Calcaneovalgus. Showing dorsum of the foot flat almost touching the anterior aspect of the tibia

Children are born with flexible flatfeet and develop the medial longitudinal arch during the first decade of life. The development of the medial longitudinal arch is a combination of osseous, ligamentous, and neuromuscular interplay, and inappropriate or pathological processes in each of these have theorized the development of flexible flatfoot. By the age of two, the medial arch has formed, and by the age of ten, the flexible flatfoot usually disappears. It is deemed a normal variant of childhood development. This condition has been found to be familial and bilateral. Unilateral flatfoot should warrant further investigation.

The majority of patients are asymptomatic and are managed with reassurance to parents and patients of its normal variant nature, as well as its resolution with time. Symptomatic patients need to be investigated to rule out underlying pathology, which may require further intervention. Commonly if the deformity is flexible, there is a complaint of planteromedial pain and occasionally sinus tarsi pain, both of which may be activity related. Orthotics can be trialed in these patients, although the improvement in this group over time may be natural progression to normal feet with maturation. Indeed, there is no strong evidence to suggest benefit with orthotics. A proportion of these patients have a flexible flatfoot with a tight Achilles heel cord (pes equinovalgus). This may benefit from physiotherapy or rarely surgery if conservative measures fail. Rigid flatfoot should bring about concern regarding tarsal coalition (Fig. 39.4a and b).

Clinical examination should demonstrate that the foot is flat on standing but an arch reconstitutes when the foot is hanging down or on tiptoes. Jack's test would reveal medial arch reconstitution (Fig. 39.5).

On standing, the hindfoot is in valgus (Fig. 39.6), and a Silfverskiold test may reveal a tight heel cord. Assess subtalar motion to confirm if the deformity is rigid.

Asymptomatic flatfeet are managed by reassurance and watchful waiting. However, painful flatfeet not responding to rest, activity modification, physiotherapy, and simple over-the-counter pain killers may need operative intervention. Releasing a tight heel cord may help (orthotics may actually cause more pain in this subset of patients). Further operative intervention may be needed in combination with soft tissue and osteotomy procedures. A medial displacement calcaneal osteotomy corrects a valgus heel. A lateral calcaneal lengthening osteotomy is also a viable alternative option. Arthroereisis has some

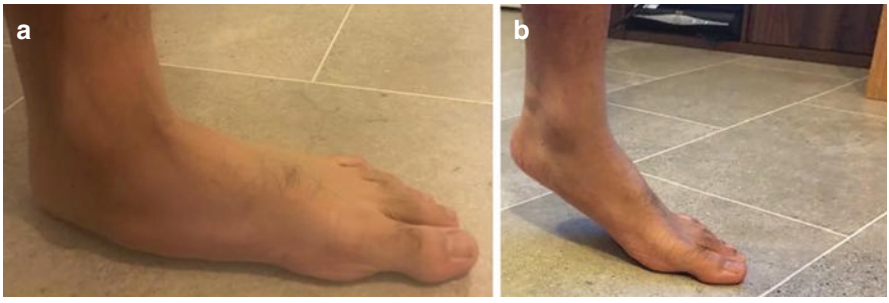

Fig. 39.4 (**a**) Showing fixed flatfoot. (**b**) Fixed flatfoot does not develop a medial arch of tiptoeing

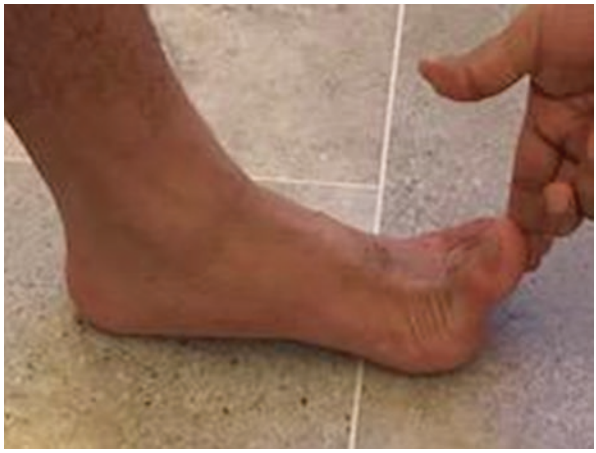

Fig. 39.5 Jack's test. Showing medical arch reconstitution on hyperextending the big toe, indicating a flexible flat foot

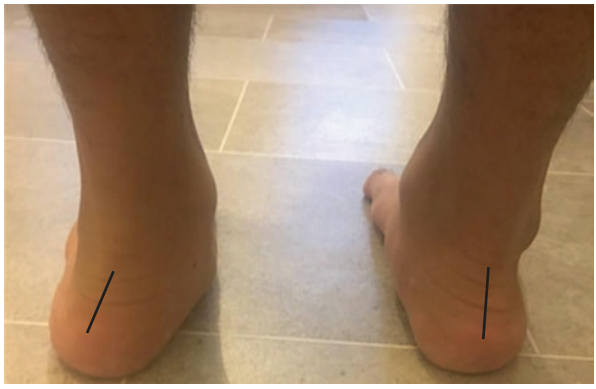

Fig. 39.6 Showing left heel valgus on standing

advocates—limiting motion by an implant in the sinus tarsi to avoid midfoot collapse [4]. Late or salvage options would include fusion.

Acquired flat foot disorders develop later in life. Examples of this include posterior tibialis tendon insufficiency (PTTI) or tarsal coalition.

Posterior Tibialis Tendon Insufficiency

Adult flatfoot deformity is most commonly caused by posterior tibialis tendon (PTT) insufficiency. This is a complex and progressive condition that commonly presents late. It mainly affects women between their fourth and sixth decades. Risk factors include obesity, diabetes, age, and inflammatory disorders. There are a proportion of patients who experience a traumatic event, but in the majority of patients, it is a degenerative process.

To understand the process, we must revisit the anatomical and biomechanical functions of the foot in relation to PTT. The muscle originates in the interosseous

membrane and adjacent tibia and fibula to become tendinous in the distal part of the calf. The tendon passes around the medial malleolus to the plantar surface of the foot. It eventually goes on to insert into multiple different parts—the navicular tuberosity and medial cuneiform, the middle three metatarsals and the plantar aspect of the remaining tarsal bones and the anterior portion of the sustentaculum tali.

The tendon is the primary dynamic support for the medial arch of the foot. Biomechanically, the tendon pull lies medial and inferior to the subtalar joint axis and posterior to the ankle joint axis. Thus, it functions to supinate and adduct the hindfoot to cause inversion and to plantar flex the ankle. During normal gait, the muscle stabilizes the foot in stance. It locks the transverse tarsal joint producing a rigid lever for toe off. There is a well-documented watershed zone of relative hypovascularity proximal to the navicular insertion, which may be a cause of degeneration and failure. This may also become pathological in patients with inflammatory disorders or those suffering from overload in obesity. The spring ligament is the primary dynamic stabilizer of the medial longitudinal arch and the talonavicular joint. It may become affected in late disease and become incompetent. It may also be involved in acute trauma. This may need to be addressed as part of operative management.

Patients present initially with medial ankle or midfoot pain in the distribution of the tibialis posterior tendon occasionally into the calf. Progressive deformity of the foot with loss of the medial longitudinal arch then occurs with lateral-sided pain due to subfibular impingement, which is a late symptom.

Physical examination is key in appreciating the progression of the disease to appropriately stage as well as to guide intervention. Inspection in early disease may be entirely normal, with only subtle swelling medially along the course of the tendon during the initial synovitis stage. Subsequently, there is progressive collapse of the medial longitudinal arch and forefoot abduction. Inspection from behind would show a valgus hindfoot with the abducted forefoot showing ‘too many toes’ when viewed from behind.

The patient is then asked to perform a single heel raise—watching for movement into eversion and reconstitution of the arch. This is not possible with an incompetent tendon and indicates middle to later stages of the disease.

The patient can be seated in the next part of the assessment. The tibialis posterior tendon is palpated along its length, and strength can be assessed by measuring resistance against inversion and plantar flexion. The tendo Achilles can be tight in the later stages of the disease as it shortens with increasing hindfoot valgus. This can be tested through the Silfverskiold test. Placing the heel into neutral alignment assesses the degree of fixed forefoot varus. The lateral side of the ankle should be palpated for signs of subfibular impingement.

The radiographic imaging series should consist of a weight-bearing AP and a lateral of the foot and a weight-bearing view of the ankle mortise. A lateral radiograph demonstrates the collapse of the medial arch with a negative tarso-first metatarsal angle (normally 0–10°) and a decreased calcaneal pitch (normal 17–32°) and a decreased medial cuneiform-floor height. An AP radiograph shows progressive uncovering of the talar head (greater than 15%). Late-stage disease will demonstrate

subtalar arthritic changes on the lateral radiograph and ankle arthritic changes and talar tilt on the AP mortise view. Magnetic resonance imaging (MRI) is the gold standard in assessing tendon pathology.

Treatment and management depend on the stage of disease at presentation with late presentation necessitating operative intervention being common. Johnson and Strom [5] classified PTT pathology, and Myerson [6] added a fourth stage. The classification system helps guide treatment.

In stage 1 disease, there is tenosynovitis without any deformity as tendon length remains the same. There may be pain on stressing the tendon and subtle swelling along the course of the tendon. The patient is able to perform a single heel raise. Immobilization in walking plaster cast or boot is first-line treatment. After a period of immobilization, conversion to custom orthosis (medial heel lift and longitudinal arch support) is required. Physiotherapy for tendon strengthening can also be started. Failure of nonoperative management would require tenosynovectomy of the tendon.

Stage 2 disease describes progression of the pathology to a flatfoot deformity with irreversible tendon elongation and dysfunction. The key to this stage is the flexibility of the deformity. The flatfoot deformity couples with a flexible hindfoot deformity. In the later parts of this stage, the forefoot becomes abducted. The patient is unable to do a single heel raise. There may be mild lateral sinus tarsi pain, as well as medial pain. Initial management, as with all later stage disease, is use of an ankle-foot orthosis, which includes a medial post to stop the collapse of the valgus hindfoot. A full length longitudinally supporting orthotic is also necessary.

Surgical intervention for stage 2 deformities involves tendon transfer of either flexor hallucis longus (FHL) or flexor digitorum longus (FDL) into the navicular to reconstruct the defunctioned PTT. The flexor digitorum longus may be preferred as it is immediately adjacent to the PTT, as well as synergistic in its line of pull and firing at similar times in the gait cycle. If an equinus contracture is present, it is corrected with a gastrocnemius recession. Spring ligament reconstruction or plication is increasingly being used by surgeons as an adjunct to treatment.

In stage 2, there are progressive deformities that need management on a case-by-case basis. Stage 2A presents with hindfoot valgus without significant forefoot abduction. After soft-tissue management, protection of the reconstruction involves bony osteotomy. This commonly is a calcaneal osteotomy with medial displacement to reconstruct the excess valgus and realign the tendo Achilles. Stage 2B disease involves significant uncoverage of the talar head and forefoot abduction. This may also require a lateral column lengthening procedure such as an Evans opening wedge osteotomy. In stage 2C disease, forefoot supination means a loss of the tripod mechanism of the foot with a relative dorsiflexion of the first ray. This can be managed with plantar flexion opening wedge osteotomy to plantar flex the first ray or with a first tarsometatarsal joint fusion.

Stage 3 disease involves rigid deformities of the hindfoot and the forefoot with degenerative changes of the subtalar joint. Accommodative orthotics are needed for the fixed deformity. Fusion is the mainstay of treatment at this stage, but this may be done on a case-by-case basis, ranging from isolated subtalar fusion to

double (talonavicular and subtalar) or triple arthrodesis. Fusion may need to be corrective in severe deformity or on rare occasions be combined with a corrective osteotomy.

Stage 4 disease involves tibiotalar degenerative disease and deltoid ligament compromise. Again with fixed deformities, fusion operations are required. All stage 3 operative interventions are available in order to correct the deformity if ankle pain and degeneration are not significant. This would be coupled with tendo Achilles lengthening and deltoid ligament reconstruction. Despite this, the disease may progress, in which case a tibiotalocalcaneal arthrodesis will be necessary.

Peroneal Flatfoot/Tarsal Coalition

The term peroneal flatfoot is a historical misnomer and is used to describe patients with a rigid flatfoot deformity. This group of patients commonly present in adolescence. Due to the rigid deformity, there is a shortening of the peroneal muscles and an eversion deformity, hence the term peroneal/spasmodic flatfoot, although this is not a neurological or neuromuscular problem. This is mostly a congenital issue, although posttraumatic/degenerative or infectious causes may be an underlying etiology. Its incidence may be underreported.

There are two main types of congenital tarsal coalition:

1. Calcaneonavicular—this is a connection between the anterior process of the calcaneus and the lateral aspect of the navicular. It commonly presents at 8–12 years old.
2. Talocalcaneal—this is a connection of varying areas between the talus and calcaneus. It may affect all or one of the calcaneal facets (commonly the middle). It presents at 12/15 years old.

Pathophysiologically, this is believed to be a disorder or a failure of mesenchymal segmentation manifesting as a coalition at birth. This is initially a fibrous coalition and may remain so. A proportion will proceed to a cartilaginous coalition (synchondrosis) or ossification to a synostosis. Patients present with rigid deformities, including flattening of the longitudinal arch, abduction of the forefoot, and a valgus hindfoot. Subtalar motion is hindered during gait.

The pathognomonic radiographic findings are as follows: calcaneonavicular (Fig. 39.7) 45° internal oblique view demonstrates the coalition.

Lateral view of the foot shows an elongated anterior process of the calcaneum or an “Ant eater nose sign” (Fig. 39.8).

Talocalcaneal—Lateral Radiograph demonstrating talar beaking and the or a “C” sign due to the medial outline of the talar dome and the posteroinferior aspect of the sustentaculum tali (Fig. 39.9).

A Harris view may show the coalition or an irregularity of the middle facet. Computed tomography (CT) and 3D reconstruction may be of benefit to further visualize the size and extent of the coalition and plan operative management. It may

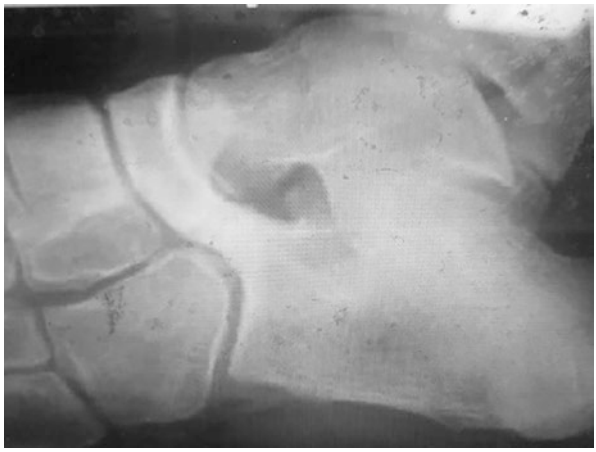

Fig. 39.7 Oblique view of the foot showing a bony bridge between the calcaneus and the navicular (calcaneonavicular coalition)

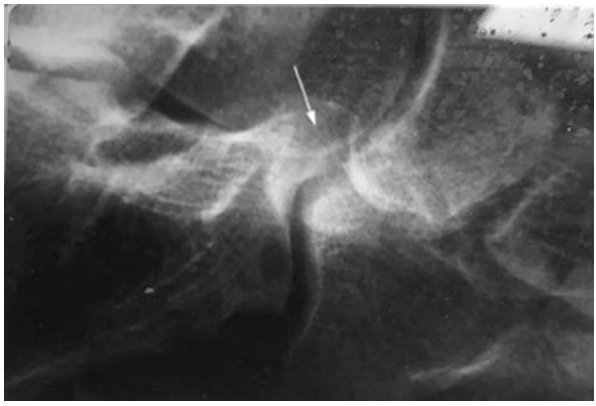

Fig. 39.8 Oblique view of the hindfoot, the arrow pointing toward the elongated process of calcaneum "Ant eater nose sign"

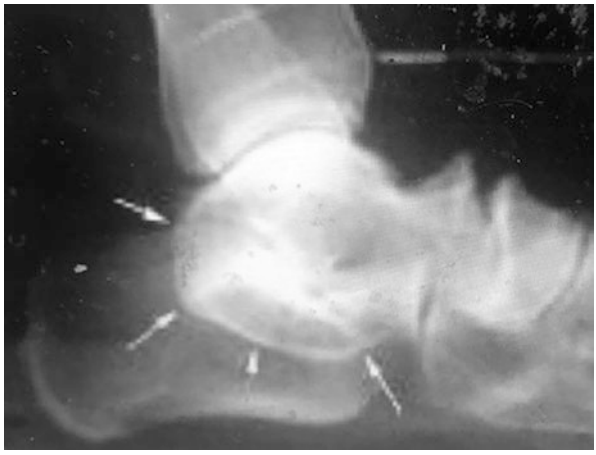

Fig. 39.9 Showing talar beaking and the "C" sign of talonavicular coalition (bony continuity of the inferomedial border of the talus with the sustentaculum tali)

also be useful in diagnostic uncertainty. MRI has recently gained popularity in 3D imaging as it demonstrates fibrous coalitions (synostosis) or cartilaginous coalitions. It also has the benefit of not exposing adolescents to ionizing radiation. Both MRI and CT may show multiple coalitions at different points in up to 20% and can be bilateral in 25–50% [7, 8].

Management of tarsal coalition is initially nonoperative. Immobilization with orthotics or casts for up to 6 weeks is successful for a proportion of patients. There is an unknown role for medial arch support of in-shoe orthotics as they may cause pain in these rigid flatfeet. Surgical management in case of failure of nonoperative management or refractory cases begins with coalition resection and interposition graft. Interposition material can be local (extensor digitorum brevis in calcaneonavicular and split flexor hallucis longus in talocalcaneal), fat graft, or bone wax. There may be a need to correct any underlying deformity commonly to correct any hindfoot valgus (calcaneal osteotomy +/− lengthening). Talocalcaneal coalitions of greater than 50% (involving the posterior facet) may do better with primary arthrodesis than with resection. Triple arthrodesis is reserved for advanced cases with associated degenerative changes or failed resections.

Miscellaneous Causes for Heel Pain

Heel pain can have multiple causes, and on occasions multiple etiologies may exist. Careful history and examination are needed to diagnose and offer appropriate treatment. In this subsection, we look at some of the common causes for heel pain and will go into greater depth in later sections come larger topics.

Plantar Fasciitis

The deep fascia of the sole, the plantar aponeurosis, is arranged in three divisions, which cover and provide partial origin to the superficial muscles of the sole. The middle division is much thicker than the others and ends distally in five digital processes. Passive hyperextension of the toes therefore renders it taut (windlass mechanism) [1]. In plantar fasciitis, inflammation of the plantar fascia, usually in the central to medial subcalcaneal region, occurs and patients present with heel pain. The nature of the lesion is obscure, and the discomfort starts with no obvious reason and is aggravated by weight-bearing. In some, it can be incapacitating to such an extent that they hobble around with the heel off the ground. Plantar fasciitis can be associated with metabolic diseases such as hypothyroidism, gout, inflammatory conditions like rheumatism, or ankylosing spondylitis. Many patients are overweight, and their work involves prolonged standing.

Clinically, apart from tenderness at the attachment of the plantar fascia to the calcaneus, the foot looks normal. The point of tenderness is just forward to the medial plantar tubercle of the calcaneum over the attachment of the central and medial plantar fascia and the flexor digitorum brevis.

A lateral radiograph of the calcaneus may show a calcaneal spur. In the absence of symptoms, the spur is left well alone.

Management

The patient should be advised that they have a chronic soft-tissue condition and that recovery may be protracted. Empirically, it appears that most cases will respond to physiotherapy, nonsteroidal anti-inflammatory drugs (NSAIDs), night splints, and local steroid injections. Static daily exercises are performed to stretch the tendo Achilles, and night splints are worn to keep the ankle in 10° of dorsiflexion and to keep the tendo Achilles stretched while in bed [9, 10]. Usually, the condition becomes tolerable in a couple of months and may take anywhere from 6 months to a year to resolve. Weight reduction in the obese is recommended, as well as treating general conditions like hypothyroidism and gout. Extracorporeal shock wave therapy (ESWT) may be of benefit [11]. Local platelet-rich plasma (PRP) injections can be tried [12].

If nonoperative measures fail, then partial central and medial plantar fasciotomy is considered. If the tendo Achilles is tight, then the gastro soleus fascia is released in the calf.

Calcaneal Stress Fracture

Although metatarsal bones are the commonest sites of stress fractures, one must consider calcaneal stress fractures in the differential for heel pain in active patients or those at risk of insufficiency. Pain worsens during weight-bearing and diminishes during rest. MRI may be needed if radiographs are inconclusive. Management involves protected weight-bearing and rest.

Subtalar Arthritis

The key in subtalar arthritis is diagnosing using a pertinent history from the patient:

1. Previous trauma to the talus or calcaneum can progress to talocalcaneal joint degeneration.
2. Progressive foot pathologies such as acquired flatfoot due to PTT pathology eventually progress to hindfoot arthritis.
3. Inflammatory arthropathies like rheumatoid arthritis could involve subtalar joint.

Nonoperative management involves orthotics to limit movement, NSAIDs, and targeted injections. Subtalar arthrodesis may be needed in some circumstances and may be done using minimally invasive techniques (arthroscopic subtalar fusion).

Sever's Disease

This condition is also known as calcaneal apophysitis and presents in adolescent boys between the ages of 10 and 14 years. It normally occurs just prior to physeal closure and is an overuse injury. Those who undertake running or jumping sports are more affected. It may be associated with tight tendo Achilles (especially gastrocnemius). This is a self-limiting condition, and management is supportive with activity modification, physiotherapy and stretching exercises, heel cups/pads, and NSAIDs. Further imaging is rarely needed but may be used if the diagnosis is doubtful. Lateral radiograph may show sclerosis or fragmentation of the calcaneal apophysis.

Bursae Around the Heel

Two bursae exist around the heel, which may become pathological and when inflamed may cause bursitis. The subcutaneous calcaneal bursa develops on the posterior heel pad and may become inflamed from rubbing against ill-fitting footwear.

The retrocalcaneal bursa is commonly inflamed in conjunction with insertional Achilles tendinopathy and is related to calcaneal Haglund deformity (best visible on a plain lateral radiograph) (Fig. 39.10).

These patients present with posterior heel pain and fullness on examination in the heel area. Pain on palpation of either side and anterior to the Achilles tendon is

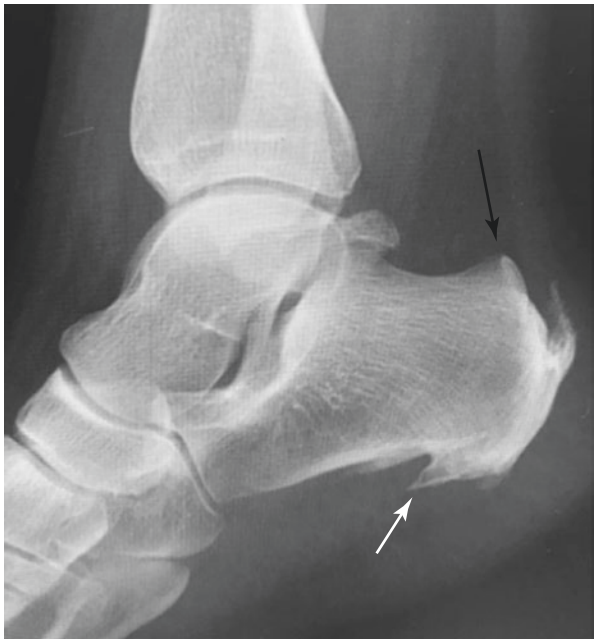

Fig. 39.10 Lateral radiograph: the black arrow points to a prominence at the posterosuperior aspect of the calcaneus (Haglund) and the white arrow to the calcaneal spur

present. Dorsiflexion of the ankle compresses the bursa inflamed by the Achilles tendon, causing pain. Nonoperative management involves NSAIDs, appropriate footwear, and possible orthotic support. There may be a role for consideration of a steroid injection under ultrasound guidance if initial conservative management fails. If associated with an insertional Achilles tendinitis, there is a risk of infiltration into the tendon and subsequent increased risk of Achilles tendon rupture. Surgical management aims to debride and remove the inflamed bursal tissue. Haglund's deformity is excised at the same time. This can all be done through a lateral approach anterior to the Achilles tendon or a direct posterior approach if managing any Achilles pathology with tendon detachment. A gastrocnemius slide should be considered, if tight.

Tendo Achilles Pathology

The tendo Achilles (TA) is a shared tendon comprising the aponeurosis confluence of the gastrocnemius and soleus muscles and the plantaris muscle. It is the largest tendon in the body, both anatomically and functionally (considering the forces that pass through it both at rest and during exercise). The tendon fibers of the soleus and the gastrocnemius rotate 90° in the Achilles tendon to allow the fibers of the gastrocnemius to insert poster laterally and the tendons of soleus to insert anteromedially. The plantaris tendon normally inserts anteromedially. The Achilles tendon inserts 2 cm distal to the posterosuperior calcaneal prominence in a crescent-like shape. When considering pathology of the TA, we differentiate between noninsertional Achilles tendinopathy (pathology 2–6 cm from the insertion) and insertional Achilles tendinitis (pathology within the last 2 cm).

Noninsertional Achilles Tendinopathy

Pathologically, this can be subdivided into inflammation of the paratenon, primary degeneration of the Achilles in the form of Achilles tendinosis due to chronic overuse or peri-tendonitis, and Achilles tendinosis, all of which are coexisting. It is frequently seen in the athletic population, especially runners or those involved in push-off sports.

Etiologically, there are both intrinsic (biomechanics, age, gender, etc.) and extrinsic factors (medication, footwear, sports, etc.). Multiple theories exist on the underlying causes, including mechanical and vascular. Mechanical injury theory suggests that physiological overload of the tendon or an accumulation of multiple overloads/microtearing with inadequate healing may occur. These would lead to tendon sheath overload (tendinitis) and the production of a degeneration spiral within the tendon itself (tendinosis). Vascular theory suggests abnormal vascularity to this are of the tendon predispose to pathology. Contradictory evidence exists with some studies demonstrating that the area is hypovascular in normal tissue but in pathological tissue there is increased blood flow.

Typically, these patients present with pain localized between 2 and 6 cm from the calcaneal insertion. Commonly, there is start-up pain during mobilization or exercise or pain at the end of exercise sessions. On examination, they can have a fusiform swelling of the Achilles tendon at this level. There is decreased range of motion due to pain with reduced plantar flexion power.

Plain weight-bearing lateral radiographs may show intrasubstance calcification and soft-tissue swelling at the level of the pain. MRI is the imaging of choice as it can demonstrate tendon thickening and disorganized tissue with intermediate high signal intensity and is useful in determining the percentage cross-section of the tendon involved.

Management initially begins with conservative strategies to manage inflammation. Rest and activity modification are augmented with eccentric strengthening exercises that show level 1 evidence of benefit. These are the mainstay of tendinopathy management. Night splints may also be of benefit, offering no load tensile stretch of the tendon. Heel lifts may be of use in these patients to reduce the tensile stretch on the pathological tendon.

Other treatments include cryotherapy, platelet-rich plasma injections to deliver growth factors and cytokines to enhance tissue repair processes, and PRP with stem cells. Extracorporeal shock wave therapy is also in use.

The aim of operative intervention is to induce mild trauma to the pathological tendon to enhance the healing response or to remove diseased tendon. Percutaneous longitudinal tenotomy may be appropriate in mild diseases. This may be augmented by stripping the anterior portion of the Achilles tendon with a large suture to reduce paratendinous adhesions. Open excision and debridement (+/− tubularization) of diseased tendon may be necessary with the flexor hallucis longus tendon being needed for transfer in patients who have a greater than 50% involvement or MRI evidence of diffuse disease.

Insertional Achilles Tendinopathy

Pain within the final 2 cm of the TA is referred to as insertional Achilles tendinopathy. Tendon degeneration occurs with loss of parallel collagen fiber structure, fatty infiltration, and loss of fiber integrity with mucoid degeneration histologically leading to tendon thickening. Mechanical overload and microtrauma are thought to be key mechanisms in the disease process. This may be different at different parts of the TA with the superficial portion of the tendon undergoing more tensile force due to the pull of gastrocnemius and soleus, while the deep portion of the tendon exhibits more compressive forces as it is impinged against the calcaneum.

Patients present with posterior heel pain and swelling. Footwear is troublesome due to compression on the pathological tendon at that level. There is swelling, which may be bony in nature to palpate. Examination demonstrates a painful range of motion with plantar flexion loss of power due to pain and pain at terminal dorsiflexion as pathological fibers are stretched.

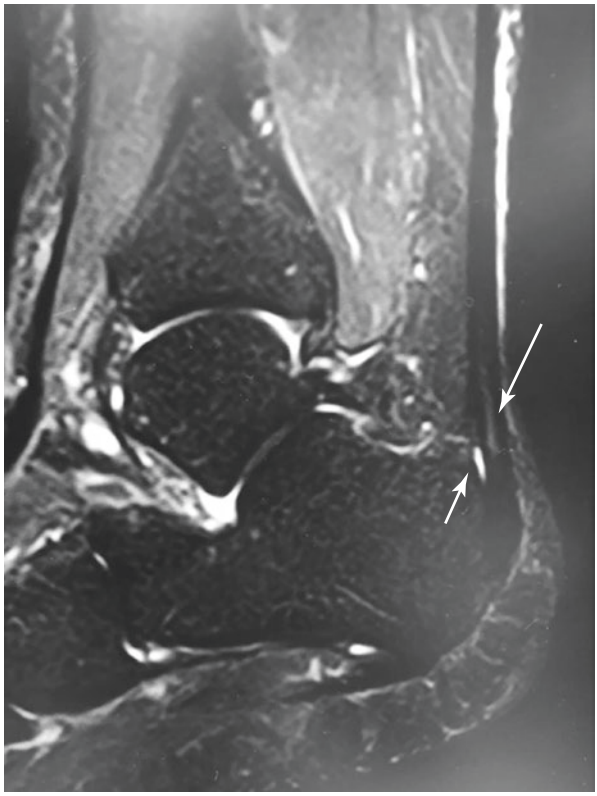

Fig. 39.11 STIR MRI images demonstrating retrocalcaneal bursitis (short arrow), along with degeneration in the tendo Achilles (long arrow), as well as Haglund prominence and calcaneal spur

Plain lateral weight-bearing radiograph imaging should be assessed for the presence of Haglund's deformity (as previously discussed). There may be calcification within the tendon and enthesophyte changes. MRI scan again is the gold standard imaging of choice to demonstrate this, as well as to signify if retrocalcaneal bursitis is occurring at the same time (Fig. 39.11).

Nonoperative management again involves eccentric exercises and progressive strengthening. This must be coupled with activity modification and rest, if appropriate. Footwear modification in the form of heel pads and heel lifts may also be used, as may night splints. ESWT has been shown to be effective at reducing pain, but intolerable pain during intervention may make this a difficult modality to adhere to and may require this to be coupled with anaesthesia. There has long been reluctance to use injections of steroid into pathological tendon for fear of promoting tendon rupture, and it is not recommended. Patients with a significant part of their pain profile arising from retrocalcaneal bursitis may benefit from focused bursal injections and from certain nonoperative management. Platelet-rich plasma has low levels of evidence showing efficacy and, along with other emerging nonoperative treatments, requires more research.

Operative treatment involves open debridement and decompression of pathological tendon. As discussed in a previous section, management of retrocalcaneal bursa and Haglund's deformity may be needed either as an initial stand-alone operation or at the

same time. The midline approach remains popular as it is extensile and allows excellent visualization of the structures. It also minimizes risk to the sural nerve and the vascular supply. A lateral approach has the benefit of not leaving the patient with a midline scar that is easily irritated by footwear. The amount of detachment of the tendon depends on the amount of pathological tendon to debride, and consideration must be taken into planned reattachment methods—suture/suture anchor and postoperative immobilization. FHL augmentation may be necessary as in noninsertional tendinopathy. Some patients may have a gastrocnemius contracture, thus necessitating gastrocnemius recession.

Calcaneal Spurs

Calcaneal spur is an outgrowth of the calcaneal tuberosity on the plantar posterior surface of the calcaneus normally arising from the medial process. It has variable anatomical associations with the plantar fascia and the muscles of the first plantar layer of the foot. It is commonly found in patients with increasing weight, elderly patients, and patients with osteoarthritis. There is also an association with patients with plantar fasciitis. Some theories exist suggesting that calcaneal spurs are an adaptive response to pathological processes and there is a relationship between heel pain and the presence of a calcaneal spur. There remain, though, patients with calcaneal spurs who exhibit no symptoms, so it is appropriate to say that they may be part of a normal spectrum/anatomical variant. Histological analysis has shown that the trabecular pattern within cadaveric spurs is vertical and perpendicular to the weight-bearing surface, which would indicate their occurrence in those who are overweight. Spurs may cause pain due to their size, fractures, and compression of the inferior calcaneal nerve, a branch of the lateral plantar nerve, and may be additive to concurrent pathologies such as plantar fasciitis. Excision is a surgical option that may be required.

Pes Cavus

Pes cavus is a broad term used to describe a foot with an abnormal high arch, which does not flatten on weight-bearing (Fig. 39.12).

It can be a sign of an underlying neurologic disorder in around 66% of cases [13]. So until otherwise proven, an underlying neuromuscular disorder has to be ruled out. Pes cavus can be present in 10% of the normal population and, in some, can be familial [14].

Biomechanics

The foot provides a stable base that helps in balance, shock absorption, load distribution and, propulsion during locomotion. The foot acts as a rigid lever in supination and a flexible coupling in pronation during various stages in the gait cycle.

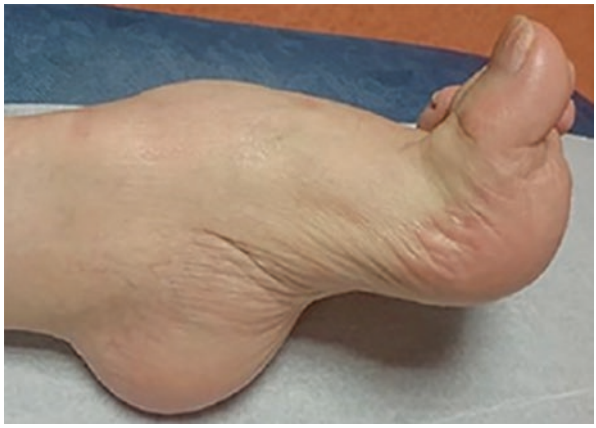

Fig. 39.12 Showing high longitudinal arch, plantar flexion of the forefoot, and cock-up deformity of the big toe

During stance, the weight-bearing stresses are transferred evenly or uniformly across the entire foot to the three points of contact (tripod), i.e., the heel, head of first metatarsal, and head of fifth metatarsal. However, in people with abnormally high arches, the size of the footprint is reduced, which increases the plantar pressure in the tripod areas for a greater proportion of the gait cycle than in normal feet, causing metatarsalgia and callus formation.

Location of the Deformity

Cavus can be located in the forefoot (anterior cavus) and posterior (hindfoot foot cavus) based on the location of the apex of the deformity or may be combined or global type [15].

Etiological Classification of Pes Cavus

1. Neuromuscular:
   (a) Muscle: dystrophies
   (b) Spinal roots and peripheral nerves: Charcot-Marie-Tooth (CMT) disease, spinal dysraphism, polyneuritis
   (c) Anterior horn cell: poliomyelitis
   (d) Central and long tract disease: Friedreich's ataxia, cerebral palsy
2. Congenital:
   (a) Idiopathic
   (b) Associated with other conditions: residual clubfoot, arthrogryposis
3. Traumatic:
   (a) Soft tissue: sequalae of compartment syndrome, burns
   (b) Bone: malunited fractures, crush injuries

Patterns of Pes Cavus

Pure pes cavus is uncommon. Here there is forefoot equinus relative to the hindfoot, which increases the height and the curvature of the medial arch. This is due to metatarsal drop (plantaris).

Pes cavo varus is more commonly seen as an abnormal high arch that is accompanied by a varus heel (medially angulated).

Equino cavo varus involves a case wherein when there is no active dorsiflexion, the ankle plantar flexes due to strong triceps surae, then the calf muscles and posterior joint capsule contract, and equinus follows.

Calcaneus varus is a dynamic deformity that is caused by the paralysis of the calf muscles, leading to excessive ankle (calcaneal) dorsiflexion and compensatory forefoot equinus (e.g., poliomyelitis).

Development of the Deformity in Neuromuscular Cavus

The development of cavus deformity is due to weakness of the tibialis anterior and peroneus brevis, overpowered by strong a peroneus longus and posterior tibialis, resulting in plantar flexion of the first ray. With the first metatarsal plantar flexed, the forefoot pronates and the medial forefoot strikes ground first. Then the subtalar joint supinates to bring the lateral forefoot to the ground. To maintain a three-point contact (Rang's tripod analogy), the hindfoot drifts into varus. Initially, the subtalar joint is flexible, and this becomes rigid with time. Recruiting extensor hallucis longus (EHL) and extensor digitorum longus (EDL) as secondary ankle dorsiflexors will lead to cock-up deformity of the hallux and claw deformity of the lesser toes due to weakness of the interossei.

Diagnosis

An accurate diagnosis is important because the underlying cause of cavus foot largely determines its future course. If the high arch is due to a neurologic disorder or other medical condition, it is likely to progressively worsen.

Clinical Presentation

Patients with pes cavus present with recurrent ankle sprains and lateral ankle pain and fifth metatarsal stress fractures due to excessive weight-bearing by the lateral foot (lateral overload). In addition, they have painful plantar calluses under metatarsal heads and plantar fasciitis.

Clinical Examination

The foot is characterized as follows:

1. Forefoot: clawing of toes and cock-up deformity of the big toe
2. Midfoot: high arch with midfoot pain and tenderness
3. Hindfoot: varus heel (initially flexible and fixed in late stages), tight gastrocnemius

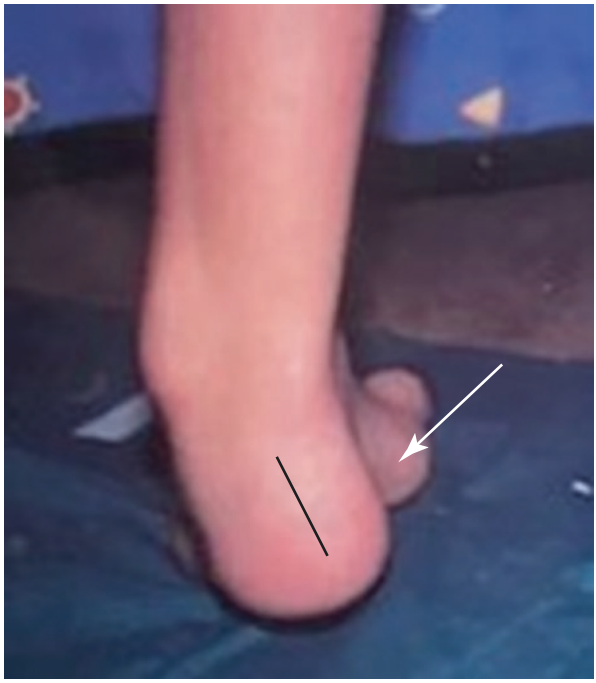

Fig. 39.13 Photograph showing the heel in varus (black line) and the white arrow pointing toward the cavus deformity of the foot

Coleman's block test [16] determines whether the hindfoot varus is forefoot driven and also establishes if the subtalar joint is mobile (Fig. 39.13).

Patient is made to stand on a raised block. If the hindfoot varus is corrected by shifting the big toe off the block (taking the first metatarsal out of the equation), then the subtalar joint is mobile (Fig. 39.14).

Peek-a-boo heel: looking from the front in the standing position shows the varus heel "peeking" around the medial ankle. Positive Silfverskiold test indicates tight gastrocnemius.

Gait is altered due to unstable base of support, increased double limb stance, and decreased single limb stance. Examination of the hands and spine may show wasting of the first dorsal interosseous muscle and scoliosis (especially in cases of CMT).

Imaging Studies

Plain Lateral weight-bearing radiographs are a key assessment as the dorsoplantar view can appear normal.

Calcaneal inclination angle (calcaneal pitch) is more than 30°.

Lateral talo-first metatarsal angle (Meary's angle) >5° with dorsal apex with break in Meary's line caused by plantar flexion of the first ray.

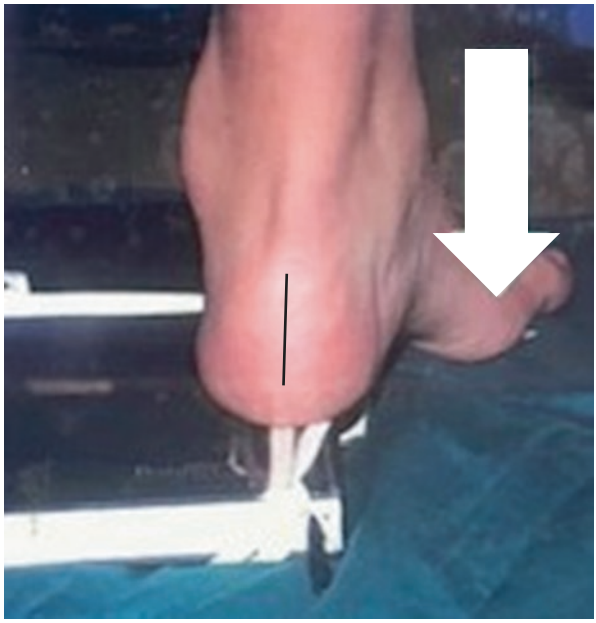

Fig. 39.14 Coleman block test showing flexible cavovarus. When the patient is made to stand on a raised block and the big toe is shifted off the block (arrow), the varus hindfoot (black line) corrects indicating a mobile subtalar joint

Cavus angle—angle of longitudinal arch is less than 150°—is formed between the calcaneal inclination axis and a line drawn along the inferior edge of the fifth metatarsal.

Management

The aim of management is to relieve pain, correct deformity, and prevent recurrence.

Nonoperative Management

Accommodative footwear includes shoes that would accommodate the high-arched foot with a wide toe box to prevent pressure over the dorsum of the toes, lateral heel wedge to prevent heel varus, and a cutout under the first metatarsal head to prevent early loading of the metatarsal. In cases of significant muscle weakness, an AFO (ankle-foot orthosis) may be needed to balance muscle forces.

Operative Management

Due to the progressive nature of the condition, surgical management is preferred to ankle foot orthosis.

Soft-Tissue Procedures

Cavus deformity: plantar fascia release (Steindler's stripping releases short flexors of calcaneus) is the first procedure in the surgical management of pes cavus, along with other soft-tissue or bony procedures. Tendon transfers for plantar-flexed first ray to balance the muscle forces. For example, transfer of the peroneus longus to brevis decreases plantar flexion force on first ray without weakening eversion. For

cock-up deformity of the big toe, EHL is transferred to the neck of the first metatarsal (Jones transfer) with fusion or tenodesis of the interphalangeal joint of the big toe.

Key Points on Pes Cavus

- Pes cavus can be familial in 10% of cases.
- Rule out underlying neurological condition.
- If unilateral, then rule out spinal cord tumor/dysraphism.
- Idiopathic pes cavus is not associated with any muscle imbalance and is not progressive.
- Accommodative footwear is recommended in idiopathic/nonprogressive cases.

Congenital Talipes Equinovarus (CTEV) (Clubfoot)

CTEV, aka clubfoot, is a congenital deformity in which an infant's foot is turned inward, and in severe cases, the sole of the foot faces sideward or even upward (Fig. 39.15). Although diagnosed at birth, many cases are first detected during perinatal ultrasound. Clubfoot is more common in males. Boys are twice more affected

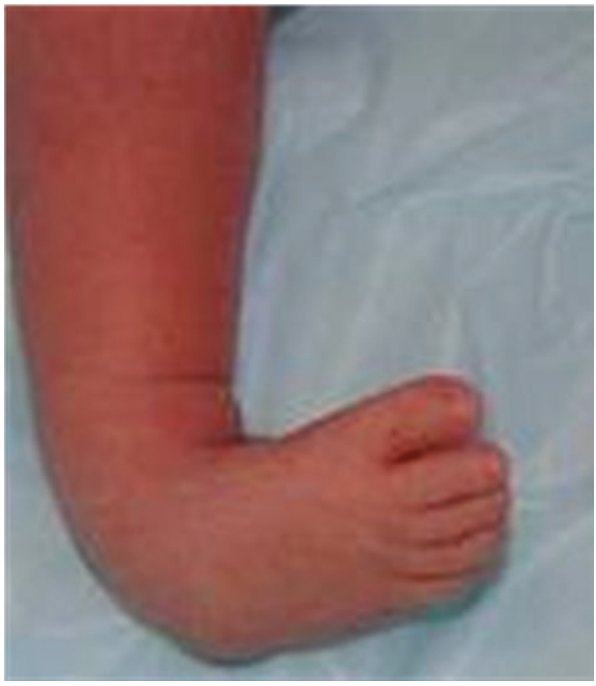

Fig. 39.15 Photograph of CTEV showing forefoot adduction and supinated, hindfoot varus, and equinus

than girls, and half of the cases are bilateral. The right foot is affected more than the left. Congenital talipes equinovarus or clubfoot deformity occurs in 1–2/1000 births.

Etiology

The etiology is unknown, but a few theories have been proposed:

1. Mechanical theory: it is one of the packaging disorders; e.g., oligohydramnios forces the foot into a deformed posture in the uterus. As the fetal position of presentation is left occipito-anterior, the right foot is more commonly forced into a deformed position.
2. Neuromuscular theory: it is due to neuromuscular imbalance, and the bony deformity is merely secondary.
3. Germ plasm defect theory: the talus never develops into normal size and shape, and all other deformities are secondary to the talar deformity.

Pathologic Anatomy

Clubfoot deformity is due to the talar neck being deviated medially and plantarward with medial rotation of the calcaneus and medial displacement of the navicular and the cuboid. The posterior and medial soft-tissue structures, i.e., Achilles tendon, tibialis posterior, flexor hallucis longus, flexor digitorum longus, joint capsules, ligaments, and fascia, are contracted. There is atrophy of the calf muscles, and the foot on the affected size is smaller.

Clinically

The mnemonic "CAVE" identifies the major findings of **C**avus, forefoot **A**dduction and supinated, hindfoot **V**arus, and **E**quinus. Clubfoot can be mild to severe, with the more severe (complex or atypical) forms being associated with other conditions (e.g., arthrogryposis, diastrophic dwarfism, prune belly, tibial hemimelia, myelomeningocele, and those derived from improper casting methods).

Management

The treatment of CTEV has evolved with time. It is divided into two main approaches: conservative and surgical.

Conservative technique primarily achieves the correction of clubfoot by slowly stretching the tight structures (viscoelasticity), allowing time for soft tissue and cartilaginous remodeling. This is achieved by serial casting.

Casting usually is started early (or until the foot reaches a length of at least 8 cm) but not later than the first month of life to make casting easier. In premature babies,

treatment is deferred for several weeks until the infant is stabilized and the foot has grown. There are numerous manipulation techniques used in CTEV (Ponseti, Kite, and French methods).

Dr. Ponseti, an orthopedic surgeon at Iowa university, in the 1940s described his "Ponseti" method following extensive anatomical studies and understanding of the flexibility of the foot, which has become popular since the 1990s. His technique involves a very specific series of short and gentle manipulation (to stretch the soft-tissue structures) and casting (to maintain the amount of correction achieved). The principles based in manipulative correction in the Ponseti method can be considered as the creep; on the other hand, the cast immobilization and the FAO wearing can be considered as stress relaxation [17–19].

In serial casting, in the first cast, the first metatarsal must be raised, which means supinating the forefoot to align the forefoot with the hindfoot and to decrease the cavus. Thereafter, abducting the foot, with counterpressure on the talar head, as a fulcrum corrects forefoot adduction and the hindfoot varus simultaneously. The calcaneus should not be touched as this might block the motion of the calcaneus, which must be free to swing out from underneath the talus and thereby to abduct, evert, and dorsiflex ("Kite's error" of manipulation, as mentioned by Ponseti, is applying counterpressure over the calcaneocuboid joint and correcting the deformities step by step, i.e., forefoot varus first, inverted hindfoot second, and the ankle equinus last rather than simultaneous correction of the hindfoot varus, inversion, and adduction). The foot must be abducted to 60–70° before correcting the equinus. Attempts to correct equinus before heel varus and foot supination are corrected will result in rocker bottom deformity.

Percutaneous Achilles Tenotomy (PAT)

Ponseti recommended tenotomy when dorsiflexion was less than 15–20°, after foot had been abducted to at least 60°. The prerequisites are as follows: talar head must be covered, the heel should be in slight valgus, and it should be possible to abduct the foot to 60°. He recommended performing PAT under local anesthesia, which can be performed as an outpatient procedure and is safe and effective. Tenotomies are usually performed between 4 and 12 weeks, the average being around 9 weeks.

Light sedation is a safe alternative to general anesthesia (GA) and is recommended when treating older infants who might struggle with local anesthesia leading to an incomplete tenotomy. PAT is followed by a further 3 weeks in a cast. After the deformity is fully corrected, next is a Denis Browne splint or a foot abduction brace, which consists of a bar equal to the length of the child's shoulders with shoes at both ends. These shoes are externally rotated to 70° (on the CTEV side) and 40° (on the normal foot, in unilateral clubfeet) with 10–15° of dorsiflexion. The brace is worn full time for 3 months and then at night and during nap times (at least 10 h a day) for 4 years.

Common Errors (Red Flags) in Manipulation Techniques Leading to Complex Deformities

Incorrect counterpressure on the talus (either too low or on a wider area). The applied abduction force applied then acts on the mid foot joints, resulting in a spurious correction.

Improperly applied cast: cast application is a skilled process. Ponseti recommended a thin cast from the groin with the knee in at least 90° of flexion and with the cast molded well behind the knee and around the heel.

Accelerated Ponseti is when the casts are changed every 5 days (rather than weekly) [20].

When Ponseti's method is used correctly, there is a 98% chance of achieving full correction. Thus, this method has become the treatment of choice for clubfoot and is now a gold standard in the management of clubfoot. Pirani's score gives a significant correlation between initial severity of the foot and outcomes [21].

Other manipulative methods described that are less commonly used are Kite and French methods.

Refractory or Resistant Clubfeet

Surgery is favored in resistant feet, usually at 6–9 months. The principle of surgery is to align the talonavicular joint and the calcaneus. Surgical release of tight structures follows an "a la Carte" approach where tight structures are released systematically. This could include Z-plasty of the Achilles tendon; fractional lengthening of FDL, FHL, or section of the tibialis posterior and abductor hallucis; posterior capsulotomies of the posterior tibiotalar joint, talocalcaneal joint, and talonavicular joint; and resection of the fibulocalcaneal ligament. The soft-tissue release is followed by casting for several months postoperatively [22, 23].

Relapse

Signs of a relapse include tightness of the heel, loss of dorsiflexion, toe walking, and forefoot turning in.

Ponseti method, followed by tibialis anterior transfer and/or tendo Achilles tenotomy or lengthening when indicated, has been successful in older children with recurrent deformity or prior surgery [24].

Other methods may be needed in treating a relapse; depending on the age of the child (3–10 years), methods like osteotomies, cuboid decancellation, wedge tarsectomy, and metatarsal osteotomy should be carefully planned. Triple arthrodesis is used for recurrent clubfoot salvage surgeries after 12 years to relieve pain and correct deformity. For neglected clubfoot, salvage procedures like triple arthrodesis/talectomy may be considered.

Key Points
- Clubfoot is a common congenital deformity that affects one in 100 live births.
- Most are idiopathic and not associated with other conditions.
- Babies should be referred early for treatment.
- The current best treatment is serial casting and bracing according to the Ponseti method.
- Results are better with manipulative methods than with surgical release.
- Recurrences can occur and are normally caused by noncompliance with bracing.

Painful Conditions of the Ankle

Sprains of Ankle Joint

Ankle sprains occur following a sudden sideways or twisting movement of the foot. An ankle sprain is an injury to the ligaments that support the ankle. When an ankle sprain happens, the ligament is stretched too far and is either partially or completely torn. The injury is common in athletes but also occur in routine daily activities such as stepping off a curb or slipping on ice.

Differentiating between a sprained ankle and an ankle fracture can be difficult, and sometimes radiographs are needed. Ottawa rules indicate radiographs only in patients if there is any pain in the malleolar zone and bony tenderness at the posterior edge of the tip of lateral malleolus, bone tenderness at the posterior edge or tip of the medial malleolus, or inability to bear weight both immediately and in the emergency department for four steps [25].

There are two broad categories of ankle sprain:

1. **Inversion ankle sprain**: the most common type of ankle sprain (90%) occurs when the foot is inverted, twisting inward. In this type of ankle sprain, the lateral ligaments are stretched too far and commonly involve the anterior talofibular ligament (ATFL) and occasionally the calcaneofibular ligament (CFL). The posterior talofibular ligament is rarely involved.
2. **Eversion ankle sprain**: the other type of sprained ankle is called an eversion injury, where the foot is twisted outward. When this occurs, the medial deltoid ligament is stretched too far. Patients will have pain on the medial side of the ankle joint.

High Ankle Sprain (Syndesmotic Injury)

These injuries have a more complex recovery as they involve the ligaments above the ankle connecting the tibia and the fibula (syndesmosis), as well as on the side of the joint. Patients suffering from high ankle sprain require recovery periods of almost twice those for patients with common ankle sprains.

Grades of Sprain

The degree of symptoms tends to correlate well with the severity of ligament injury and the prognosis for recovery:

A. **Grade I ankle sprain** causes stretching of the ligament. The symptoms tend to be limited to pain and swelling. Most patients can walk without crutches but may not be able to jog or jump.
B. **Grade II ankle sprain** involves a more severe partial tearing of the ligament. There is usually more significant swelling and bruising caused by bleeding under the skin. Patients usually have pain with walking but can take a few steps (Fig. 39.16).
C. **Grade III ankle sprain** is a complete tear of the ligaments. The ankle is usually quite painful, and walking can be difficult. Patients may complain of instability or a giving-way sensation in the ankle joint. This bruising will move down the foot toward the toes in the days after the ankle sprain because of gravity pulling the blood downward into the foot.

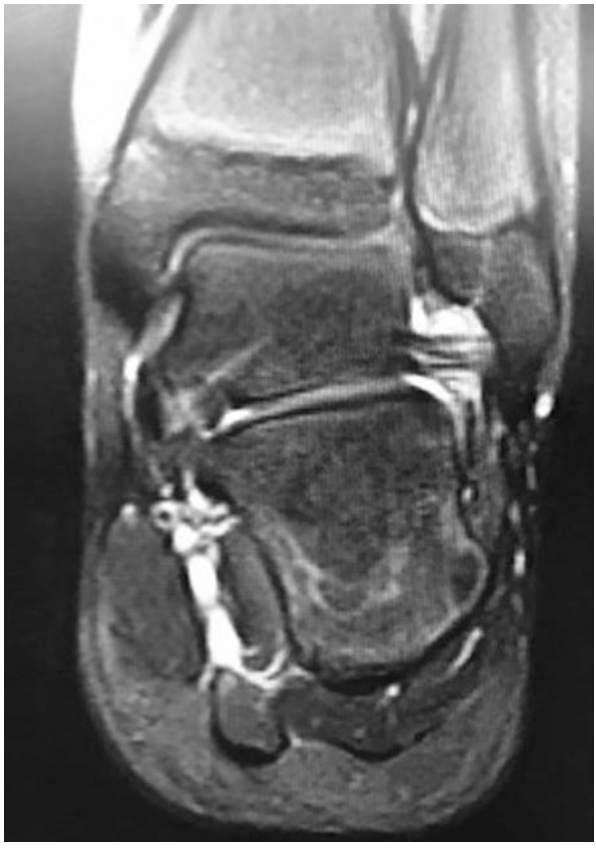

Fig. 39.16 Showing grade 2 sprain, the arrow pointing to the posterior talofibular ligament with peri-ligamentous edema

Treatment of sprained ankles is important because returning to normal activities in a timely manner is important for most patients. Rest, ice, compression, and elevation (RICE) treatments are important but should quickly progress to rehabilitation and strengthening.

Surgical treatment is reserved for recurrent symptomatic ankle instability with excessive tilt and a positive anterior drawer sign on exaination/postive stress radiographs. Anatomic procedures (modified Brostrom procedure) are usually successful.

Traumatic Arthritis of the Ankle (Anterior Impingement, Footballer's Ankle)

Anterior ankle impingement is caused by repeated strain on the anterior capsule due to repeated holding and kicking the ball onto the dorsum of the foot in plantar flexion. The classic form of impingement is referred to as "footballer's ankle." Despite the name, this can happen in many different types of sports, including soccer, football, and basketball, and in dancers. Soft tissues get entrapped between exostoses or calcified spurs, which form on the anterior lip of the tibia and the neck of the talus from repetitive traumatic avulsions of the anterior ankle capsule. The impingement incites inflammation of the surrounding synovial and capsule tissues, limiting range of motion and causing pain.

Patients present with pain and decrease in overall ankle range of motion, mostly affecting dorsiflexion, and in some an effusion may be present. Radiographs show spurs arising from the anterior distal tibia or the talus. Occasionally, an MRI is also utilized to evaluate other structures of the ankle. Management includes physiotherapy to help break down scar tissues and improve range of motion and anti-inflammatory medications to relieve pain and swelling. Steroid injection can be tried for inflammation. If patients present with effusion, then ankle immobilization may be necessary for 3 weeks. If conservative measures fail, then either arthroscopy or open surgery is performed to remove the bony spurs or exostoses that are causing the impingement.

Rupture of Tendo Achilles

Introduction

The Achilles tendon is the largest, strongest, and thickest tendon in the body, connecting the gastrocnemius, soleus, and plantaris to the calcaneus. It is approximately 15 cm long and begins near the middle portion of the calf. Contraction of the gastrosoleus causes plantar flexion of the ankle, enabling such activities as walking, jumping, and running. The Achilles tendon receives its blood supply from its musculotendinous junction and its innervation from the tibial nerve. Occasionally, the tear may be partial and usually occurs where the tendon joins the calf muscle. This

injury is managed slightly differently and usually involves resting the ankle in a boot for a few weeks. Most common site of rupture is around 2–6 cm above its insertion due to poor vascularity in this region.

Causes

The incidence of Achilles rupture is 7 out of 100,000 in the general population and 12 out of 100,000 in competitive athletes [26]. It can occur at any age but is most common in 30–50 years with a male-to-female ratio of nearly 20:1. Activities that cause forceful push off (strong contraction of the calf muscles) or lunging type movement with sudden hyperdorsiflexion can stress the Achilles tendon excessively, causing rupture. Other mechanisms by which the Achilles can be torn involve sudden direct trauma to the tendon or sudden activation of the Achilles after atrophy from prolonged periods of inactivity. Fluoroquinolone antibiotics, e.g., ciprofloxacin; previous steroid injections; chronic Achilles tendonitis; rheumatological disease; and kidney disease are known to increase the risk of tendon rupture.

Symptoms

When a rupture of the Achilles tendon occurs, one experiences a sudden pain in the heel or calf, sometimes associated with a snapping or popping sound. Patients often describe the feeling as if someone has hit them or has shot them in the back of the leg, only to turn around and find that no one is there. After rupture, there may be some swelling and bruising, and it is usually difficult to walk as it is difficult to push off the ground properly on the affected side. A partial tear of the Achilles tendon is rare, so any acute injury to the Achilles tendon should be assumed to be a complete rupture.

Diagnosis

It is usually possible to feel a gap in the tendon, usually 4–5 cm above its calcaneal insertion. The tear can occur higher up, about 10 cm above the insertion into the heel, at the site where the muscles join the tendon; this is known as a musculotendinous tear.

Special tests: Thompson (Simmonds) test, which is a calf squeeze test, will be performed. Normally if the Achilles tendon is intact, this causes the foot to point downwards, but if it is ruptured, it causes no movement. Ultrasound scan helps to confirm the diagnosis and the exact site of the rupture.

Treatment options: there are two treatment options available for Achilles tendon ruptures—nonoperative (conservative) and operative (surgical).

Nonoperative management (functional bracing), as evidence suggests, has similar results to surgery without the associated complications. Occasionally, surgery may be considered, especially in cases of delayed presentation or atypical ruptures.

The principle of functional bracing is the use of a specialized boot to keep the ankle in a fixed position while allowing the tendon to heal and the patient to try to

function normally. There is a set regime that involves initially being in a below-knee plaster cast with the foot in maximum equinus position while the patient can briefly bear weight on the toes for a couple of weeks. This then changed to a specialist boot (e.g., Vacoped), which is a below-knee boot with the ankle in 30° of equinus with Velcro straps. The boot should be worn at all times, including in bed, to ensure that your tendon is protected throughout the healing process. During the weeks following the injury, the boot will be gradually adjusted to allow the degree of equinus to be corrected from 30° (full) to 15° (mid) and 0° (neutral). Full weight-bearing is commenced at 3 weeks, and the boot is removed at 8 weeks.

As the calf pump is not effective and the patients are at a high risk for venous thromboembolism (VTE), thromboprophylaxis is advised for about 6 weeks from the injury. The total treatment time will be approximately 9 weeks. Following this, a flat shoe with a single heel raise is worn for 4 weeks.

The risks of conservative treatment include risk of rerupture (8%), decreased tendon strength, risk of clot in leg veins (deep vein thrombosis)/lungs (pulmonary embolus).

Surgical Treatment

This is not usually the preferred treatment option as the risks of complications may outweigh the benefits. Surgery may allow earlier weight-bearing with reduced rerupture rate (4%), but wound-healing problems and sural nerve injury are potential risks. However, surgery may be considered for certain patient presentations; delayed presentation (more than 2–3 weeks following injury), reruptures of the Achilles tendon with tendon gap and elite athletes for (some evidence of slightly increased push off strength).

There are two different types of surgeries: open surgery and percutaneous surgery.

During an open surgery, an incision is made in the back of the leg and the Achilles tendon is repaired. There are different techniques, and some surgeons harvest the plantaris, which is spread out and wrapped around the Achilles tendon repair, preventing adhesions and adding to the strength of the repaired tendon.

In percutaneous or minimally invasive surgery, the surgeon makes several small incisions, rather than one large incision, and sews the tendon back together through small incisions. Surgery may be delayed for about a week after the rupture to let the swelling go down. For sedentary patients and those who have vasculopathy or risks for poor healing, percutaneous surgical repair may be a better treatment choice than open surgical repair.

Chronic ruptures of the Achilles tendon are those that present 6 weeks from the injury. These are managed with open repair if the gap between the two ends are less than 2.5 cm. If the gap is more than 2.5 cm, then the surgical options are V-Y plasty of the proximal part of the tendon, turndown flap of the aponeurosis, use of peroneus brevis, flexor hallucis longus, or fresh-frozen allografts.

Outcome

The general outcome of Achilles tendon rupture is good. Kotnis et al. [27] elected to manage conservatively only those patients whose gap in full equinus was less than 5 mm. All others were managed operatively. They observed no statistically significant difference in rerupture rates between the groups. The tendon usually heals around 2 months or more and will take several more months to regain strength and flexibility. If patients have more physical work, then it may take 3–4 months to return to work. Driving a manual car should be avoided for at least 9 weeks following your injury. Return to sport is between 4 and 12 months, depending on the sport that the patient wishes to return to and depending on one's strength and ability to perform the necessary skills.

Fusion of the Ankle Joint

The goal of ankle fusion (also commonly known as ankle arthrodesis) is to relieve pain and improve function for a patient with painful end-stage ankle arthritis. Asymmetrical joint surfaces along with severe pain, swelling, instability, and stiffness due to various causes, i.e., trauma, infection, inflammatory conditions, and osteochondral defects. Advanced or end-stage ankle arthritis is degeneration of the articular cartilage of the lower tibia and the talus with exposed underlying subchondral bone and osteophytes formation. Pain typically is made worse with movements of the arthritic ankle. The goal of arthrodesis is to fuse the tibiotalar joint into one bone, which eliminates joint movements and reduces pain from the arthritic joint.

Indications

Patients who have advanced ankle arthritis that is symptomatic and who have exhausted all other options, like activity modification, analgesics, anti-inflammatories, corticosteroid injections, walking aids, special ankle braces, or ankle foot orthoses to stabilize the ankle and restrict its movements, and have failed arthroscopic ankle debridement may be a candidate for ankle arthrodesis.

Relative contraindications are insufficient quantity or quality of bone for fusion, poor blood supply of the ankle, impaired nerve function, medical conditions that increase the risk of anaesthesia, and severe limb deformity.

Surgical Techniques

Ankle arthrodesis can be done either arthroscopically or by an open technique. Two important factors for successful fusion are as follows:

1. The position of the foot in relation to the leg
2. The method of fusion

The ankle joint is responsible for the majority of plantar flexion and dorsiflexion movements. Ankle fusion decreases this movement, but the movement of the subtalar joint and the other joints of the foot remains unaffected. If the patient's ankle is fixed at 90°, then some transverse tarsal movements will provide an average of 10° of plantar flexion. In women, the ankle is held in 100° to accommodate for a heel.

With regard to the method of obtaining sound fusion, arthroscopic techniques involve making small incisions around the ankle, and any remaining cartilage within the ankle joint is removed so that there is good apposition of the bony surfaces of the tibia and the talus. The ankle is held in the most functional position with percutaneous screws, which allow the bones to fuse together.

Open Techniques

Many open techniques have been described either by the lateral or anterior approach. In the lateral approach to the ankle, the distal fibula is excised and the tibial and talar surfaces are cleared of cartilage remnants to expose the underlying bone. Tibio-talar contact surfaces are held with two screws passed obliquely from the tibia to the talus. Bone grafts may be used to aid fusion. In the anterior approach, the joint is approached from the front, and the tibio-talar surfaces are prepared, which are then compressed using screws, plates, or staples.

Postoperatively, patients are kept non-weight-bearing in a cast for 6 weeks, followed by partial weight-bearing in a walking boot. After 10–12 weeks, the ankle fusion is typically sturdy enough to allow walking out of the plastic boot and a gradual return to more vigorous activity.

Complications

Apart from the general complications of any surgery the (the risks associated with anesthesia, infection, damage to nerves and blood vessels, and bleeding or blood clots), a specific risk associated with ankle arthrodesis is nonunion (10%). Loss of motion in the ankle after a fusion causes premature wear and tear in the subtalar and other joints of the foot as these joints have to compensate for the ankle fusion. However, the literature suggests no difference in fusion times for open and arthroscopic fusion; wound complications and return to function are quicker in the latter [28].

Peroneal Tenosynovitis

The peroneal tendons act by everting and plantar flexing the foot. The peroneal tendons also provide lateral stability to the ankle during weight-bearing and protects it from sprains. They also help to pronate and stabilize the arch when walking. Peroneal tendinitis is related to the pulley action of the lateral malleolus on the peroneal tendons. The peroneal tendons and their tendon sheaths become inflamed due to various conditions like overuse, faulty footwear, inappropriate training methods, and trauma. Peroneal tendonitis is particularly common in athletes and especially runners as they are more likely to roll their feet outward,

causing friction between the tendon and bone. Other risk factors are pes cavus and muscle imbalance.

Peroneal tendonitis can be either acute, meaning it comes on suddenly, or chronic, i.e., it develops over time. Patients present with pain in the retrofibular region with tenderness over the course of the tendons. Stressing the tendon against resistance reproduces the pain overlying the tendons. Other presentations could be pain at the back of the ankle, pain that worsens during activity and lessens during rest, swelling at the back of the ankle, and instability of the ankle when bearing weight.

MRI or sonography can assist in the diagnosis of tenosynovitis, focal degeneration, and attenuation of tendons.

Treatment

Nonoperative treatment includes a rest for the foot and ankle, such as a boot or support. Anti-inflammatory medication such as ibuprofen can help relieve pain and swelling. Physiotherapy in the form of ultrasound therapy can reduce pain and swelling. and once symptoms improve, exercises that strengthen the muscles and improve proprioception can be performed. A brace for use during activities that involve repetitive ankle motion can be tried. If symptoms persist, then a local corticosteroid injection can be considered. Surgery (peroneal tenosynovectomy) is rare when treating peroneal tenosynovitis, and it is only usually considered as a last resort if nonsurgical methods fail in reducing pain.

Dislocation of Peroneal Tendons

The peroneal tendons (peroneus longus (PL) and peroneus brevis (PB)) are located on the lateral side of the leg, and their tendons are enclosed in a fibrous osseous tunnel that runs behind the lateral malleolus. The retrofibular groove is a shallow sulcus within which the peroneus brevis lies. Although PL shares the same synovial sheath with PB, it does not contact the sulcus and distally separates into its own sheaths. A tough connective tissue known as the superior peroneal retinaculum holds the peroneal tendons in place.

Traumatic Subluxation

Activities involving forced dorsiflexion inversion mechanism resulting in powerful reflex muscular contraction of the peroneal tendons results in injury to the superior peroneal retinaculum (e.g., skiing, dancing, playing basketball, playing soccer, ice skating), then the peroneal tendons move out of their place and slip over the lateral malleolus on the lateral aspect of the ankle. This condition is known as peroneal

tendon subluxation or dislocation. The peroneal tendons continue to remain in the dislocated position, or they may return to their previous position on their own. Diagnosis is confirmed by observing the subluxation or dislocation of the peroneal tendons during eversion and dorsiflexion. The superior retinaculum may be avulsed with a fleck fracture at its insertion along the lateral border of the lateral malleolus [29]. Ankle instability usually accompanies subluxation, and the ankle mortise should be checked. Acute traumatic subluxation does require a non-weight-bearing cast held in 90° for at least 4 weeks. Inadequate treatment results in chronic subluxation.

Chronic subluxation of the tendon will be accompanied by an audible click or pop as the tendon displaces and relocates into the grove. Treatment option includes repair of superior peroneal retinaculum, posterior fibular groove reconstruction using periosteal flap, rerouting the tendon below the calcaneofibular ligament, and bone block reconstruction procedures [29].

Rupture of Plantaris Tendon

The plantaris muscle is the smallest component of the tendo Achilles complex. The plantaris muscle and tendon sit roughly in the center of the calf, between the two heads of the gastrocnemius.

Around 10–20% of the population is born without plantaris muscles, but this does not affect mobility, either in the long or short term. Injuries to the plantaris muscle can either be a muscle strain or, more commonly, a plantaris muscle rupture. Plantaris muscle ruptures have also been called “tennis leg” as this injury is seen in athletes who are lunging forward, such as tennis players. Typical symptoms of a plantaris muscle rupture include sudden pain in the back of the calf, swelling or bunching of the calf muscle, bruising in the back of the leg, and cramping and spasm sensations of the calf muscles.

Diagnosis

Plantaris muscle tears can be differentiated from an Achilles tendon tear by a Simmonds test, which shows that the foot can be pointed downward following a plantaris rupture, on squeezing the calf, which is not the case with Achilles tear. Plantaris ruptures can also be confused with deep vein thrombosis (DVT). If the diagnosis is unclear, then either an MRI or an ultrasound can be helpful, which can also look out for other possible causes of calf pain.

Treatment of a plantaris muscle injury is almost always nonsurgical. The initial treatment of a plantaris injury is with the usual RICE (rest, ice, compression, elevation) treatment. If the pain is significant, patients may require a brief time of immobilization or crutch use to allow the pain to subside. Gradual increases in mobility and strength can be obtained with the assistance of a physiotherapist. With conservative treatment, symptoms will gradually resolve over the course of several

weeks, although a full recovery may take up to 8 weeks, depending on the severity of the injury.

Peroneal Palsy Due to Hematoma

Compression of the common peroneal nerve due to a traumatic hematoma within its nerve sheath following spiral fractures of the distal part tibia or inversion sprains of the ankle usually after skiing injuries is known but rare. Traction is transmitted along the nerve, which seems to be vulnerable, from the ankle to mid-thigh, where the vasa nervorum of the common peroneal nerve rupture below the sciatic bifurcation, resulting in a hematoma between the epineurium and perineurium [30]. Persistent pain, numbness, and weakness in peroneal nerve distribution, after reduction and immobilization of an ankle injury, occurring for hours or even days after the injury should raise suspicion and should alert the surgeon to investigate either with an ultrasound or MRI.

Early exploration of the nerve and its branches with prompt evacuation of the hematoma with ligation of the ruptured vasa nervorum is followed by swift and dramatic recovery [29].

Foot Drop in Leprosy

Leprosy (Hansen's disease) is a chronic infectious tropical disease (neglected tropical disease (NTD)) caused by mycobacterium leprae that attacks the nerves but is curable. There are two main forms: lepromatous, which is progressive, and tuberculoid, which is generally self-limiting because the host had reacted defensively and successfully against the bacillus. Most patients with tuberculoid will have tender and thickened nerves, which will ultimately make them lose their function with time, which can resolve spontaneously (with years) or progress to other rare types of leprosy such as borderline or lepromatous leprosy. Neuropathy in tuberculoid leprosy is an inherent effect of the host response and is not related to the proliferation of the bacilli. In lepromatous leprosy, the hematogenous spread of the bacilli and unchecked proliferation cause nodules throughout the skin, which is widespread due to poor immune response. Here the nerve damage is slower than in tuberculoid form [31]. The first sign of leprosy is usually a patch of discolored numb skin. The disease is curable but, if left untreated, causes life-changing permanent damage to the hands, feet, and eyes, leading to paralysis, blindness, ulcers, and amputations. Penetrating ulcers are treated by total contact casts, i.e., plaster boot with a rocker [32]. The common paralytic deformities of the foot seen in leprosy patients are foot drop, inversion of the foot, and claw toes due to paralysis of the dorsiflexors, namely the tibialis anterior, and the extensors and evertors, namely the peronei. Long-standing foot drop results in secondary contractures mainly of the tendo Achilles and instability of the talonavicular joint. A foot drop deformity should be corrected by a tibialis posterior transfer before

complications occur. If the tendo Achilles is tight, then the tendon is lengthened by Z-plasty.

Conclusion

The foot and ankle is a complex conglomerate of bones (28 bones), joints (31 joints), ligaments, and fasciae, which work in unison to provide a stable, functional, and effective lever arm to the body for energy-efficient locomotion. It can be affected by a multitude of pathologies, some of which have been illustrated above. To discuss all the pathologies is outside the scope of this chapter.

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