Flatfoot in the Adult

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Abstract

Flatfoot in the adult has long been a poorly understood "wastebasket" diagnosis, often used to unfairly deny asymptomatic individuals equal employment opportunities in our society. Now that flatfoot has been classified into a variety of congenital and acquired conditions, the parameters for assessment have been well defined, and rational treatment protocols have been established. Clearly, if the foot painlessly supinates/inverts to become a rigid lever for push-off and pronates/everts to absorb stress during stance, then it "functions normally" no matter what the height of the arch. However, the biomechanically offset position of pes planus with excessive heel valgus coupled with rigidity or instability can alter the connected interplay of the bones of the foot and weaken the entire kinetic chain of the lower extremity. Careful clinical and radiographic evaluation, coupled with a thorough understanding of the anatomy and biomechanics of the foot, will allow accurate evaluation and appropriate treatment.

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Pes planus in the adult is commonly seen in orthopaedic practices but is often disregarded as a sign of significant underlying pathologic change. While we now understand that asymptomatic flexible flatfoot should be left untreated, there is a whole subgroup of congenital and acquired flatfoot deformities that require careful evaluation and prompt treatment to avoid progressive deterioration of ambulation.

Biomechanics of Flatfoot

In the normal gait cycle, initial weight-bearing commences with heel-strike. Internal rotation of the tibia, eversion of the calcaneus, and associated unlocking of the talonavicular and calcaneocuboid joints allow progressive measured pronation of the foot to decelerate the impact of weight-bearing. When foot-flat is reached, the weight-bearing axis passes through the anterior superior iliac spine down through the patella to the middle of the foot at the level of the second metatarsal. As the foot moves toward push-off, the tibia externally rotates, and the calcaneus actively inverts and locks the transverse tarsal joints via the pull of the posterior tibial tendon, thus solidifying the longitudinal arch and locking the foot in supination to act as a rigid lever for gait.[1]

Depending on the severity of a flatfoot deformity, the excursion of all these motions is variously increased, resulting in more stress on the supporting ligamentous and muscular structures. The weight-bearing axis is shifted medially during stance phase, thus potentially disrupting the entire kinetic chain of the lower extremity.[2] Internal femoral rotation, internal knee rotation with an increased Q angle, and increased lateral patellar pressure can all result from this abnormal alignment. The deformity of the abducted everted foot can be severe enough so that the anterior tibial and Achilles tendons may exert an evertor force. Increased pronation causes longitudinal rotation of the first metatarsal, changing the orientation of the first metatarsophalangeal joint in relation to the floor. This may well predispose to the development of hallux valgus.[3]

Congenital Disorders

Asymptomatic Flexible Flatfoot

Individuals with asymptomatic flexible flatfoot should be reassured that no treatment is indicated and should be further counseled that treatment has not been shown to in any way prevent the development of future problems.

Symptomatic Flexible Flatfoot

Patients with symptomatic flexible flatfoot deformities will complain that their feet tire easily and become painful with prolonged standing. The standing alignment of both lower

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extremities should be assessed from the pelvis down, including the relationship of the foot to the floor, the degree of heel valgus, and the performance of multiple single- and double-leg heel rises while active inversion of the subtalar joint is observed. With the foot in the dependent position, the range of motion of the ankle, hindfoot, midfoot, and forefoot and the presence of any painful areas should be carefully documented. Special attention should be given to assessing the degree of tightness of the Achilles tendon.

Radiographic Evaluation

The talus–first metatarsal angle measured on a lateral weight-bearing film of the foot is the standard for assessing pes planus (Fig. 1).[4] The sag in the longitudinal arch may take place at the talonavicular joint, the naviculocuneiform joint, or a combination of both. Zero degrees is considered normal. An angle of up to 15 degrees is considered to represent a mild deformity; 15 to 30 degrees, a moderate deformity; and greater than 30 degrees, a severe deformity.

The talonavicular relationship is a reliable measure of the extent of pronation on weight-bearing. Hyperpronation, which is different from pes planus, is assessed by evaluating the degree of medial deviation of the nose of the talus in the cup of the navicular on a weight-bearing anteroposterior (AP) film of the foot. Normally, the articular surface of the talus does not migrate medially out of the cup of the navicular by more than 6 to 7 degrees on weight-bearing. Over 7 degrees is considered hyperpronation (Fig. 2).[4]

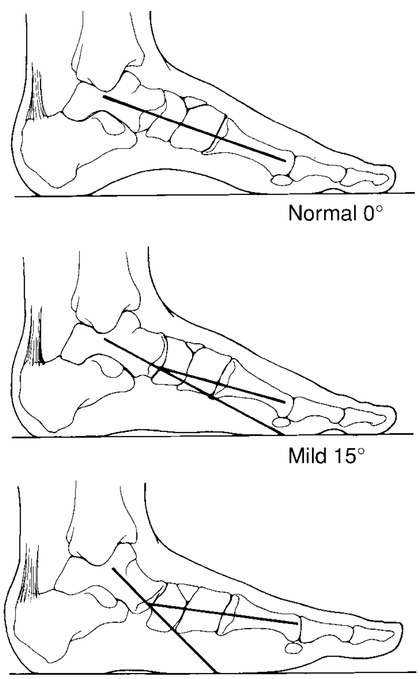


Fig. 1 The lateral talus–first metatarsal angle. On a standing radiograph, the angle would be 0 degrees in a normal foot, up to 15 degrees in a foot with a mild deformity, 15 to 30 degrees in a foot with a moderate deformity, and more than 30 degrees in a foot with a severe deformity.

Treatment

Treatment of symptomatic flexible flatfoot should usually be conservative, including the use of shoe modifications and orthotics that will support the arch and prevent hyperpronation. In a small group of individuals, the deformity is so severe and the pain so great that normal shoe wear is excluded and routine weight-bearing activities become profoundly difficult. Consideration should then be given to osteotomies that will biomechanically realign the weight-bearing forces of the foot while maintaining motion at the joints of the hindfoot.

The most reliable osteotomies involve displacement derotation of the calcaneus to a more favorable position. A 1-cm opening-wedge osteotomy of the anterior process of the calcaneus 1.5 cm proximal to the calcaneocuboid joint will correct pes planus and hyperpronation without fusion (Fig. 3).[5] Osteotomy through the body of the os calcis, which derotates it out of valgus and positions the contact portion of the heel more in line with the weight-bearing axis, can also be used (Fig. 4).[6] Both procedures can be combined with Achilles-tendon lengthening if indicated.

Fig. 2 With hyperpronation, there is excessive medial deviation of the talus in the cup of the navicular on a standing AP film. In the normal foot, the surface of the talus does not migrate medially out of the cup of the navicular by more than 6 to 7 degrees.

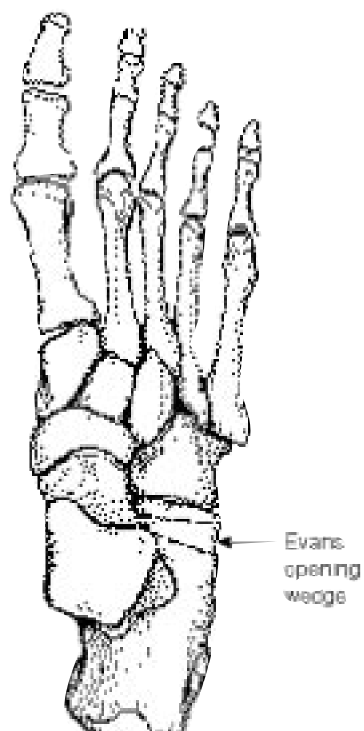


Fig. 3 The Evans anterior calcaneal osteotomy helps restore and stabilize the longitudinal arch by elongating the lateral column of the foot.

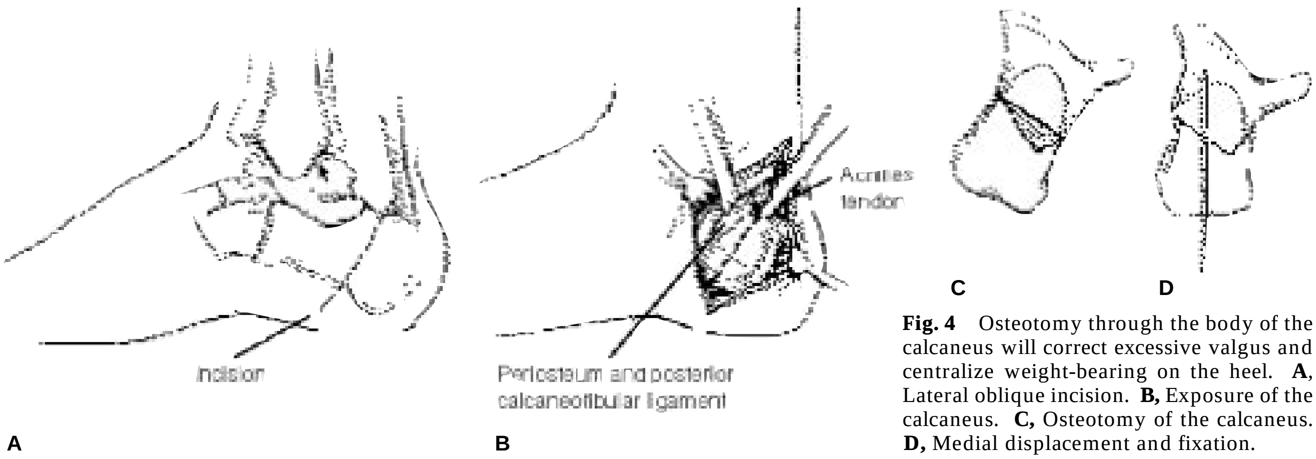


Fig. 4 Osteotomy through the body of the calcaneus will correct excessive valgus and centralize weight-bearing on the heel. **A,** Lateral oblique incision. **B,** Exposure of the calcaneus. **C,** Osteotomy of the calcaneus. **D,** Medial displacement and fixation.

Rigid Flatfoot

Symptomatic rigid flatfoot usually presents in the third or fourth decade of life. A tight Achilles tendon limits ankle dorsiflexion, thus putting increased stress on the transverse tarsal joints during gait. With skeletal maturity, the increased weight and activity level may lead to breakdown of the midfoot and a resultant rocker-bottom deformity.[2]

The patient presents with painful pes planus, decreased range of motion at the joints of the midfoot and hindfoot, and decreased ankle dorsiflexion. Weight-bearing radiographs demonstrate loss of the longitudinal arch with a sag at the talonavicular joint. Beaking of the talus and arthritic changes in the surrounding midfoot joints may be present. In the late stages, a full rocker-bottom deformity is present, which must be differentiated from peroneal spastic flatfoot secondary to tarsal coalition.

Initial treatment should consist of shoe modifications along with use of custom-made, total-contact, semirigid orthoses to distribute weight-bearing forces. Rigid orthotics and off-the-shelf appliances uniformly do not conform to these feet and may worsen the pain. Triple arthrodeses plus Achilles-tendon lengthening should be reserved for patients with severe disabling pain resistant to all conservative treatment.

Peroneal Spastic Flatfoot

Peroneal spastic flatfoot is usually secondary to a tarsal coalition. The problem usually occurs during adolescence, but it may become symptomatic in adults after increased levels of activity or trauma.

The coalition is secondary to a genetic mutation that causes failure of segmentation of the primitive mesenchyme. It presents in an autosomal-dominant pattern with a 4:1 male-female prevalence. Approximately 50% of patients have bilateral involvement. Talocalcaneal and calcaneonavicular coalitions are the most common, but talonavicular and calcaneocuboid coalitions have been reported.[7]

The usual clinical presentation is a painful, stiff flatfoot with spasm of the peroneal tendons. This spasm may well be adaptive shortening aimed at protecting irritability in the hindfoot complex. The foot is fixed in eversion, and attempted inversion exacerbates the spasm.

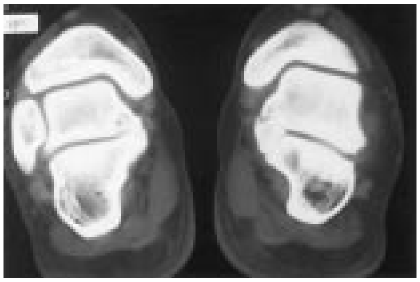
Oblique radiographs usually will reveal the coalition. The lateral radiograph may show talonavicular beaking secondary to restricted motion, which causes impingement. Computed tomography (CT) remains the best imaging technique for depicting the architecture of the problem (Fig. 5).

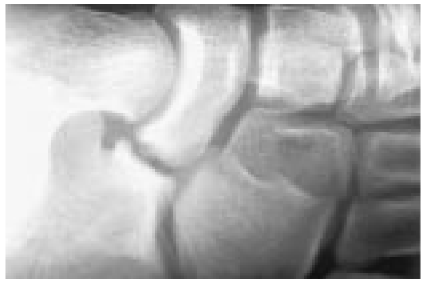
The initial treatment is a short-leg walking cast, which often relieves the symptoms after 6 weeks. Persistent or recurrent pain is best managed surgically. Talonavicular coalitions without associated arthritic changes can be excised through a dorsolateral incision. Even in adults, a large rectangular block of bone should be excised, with interposition of the extensor brevis musculature to prevent recurrence. Associated talonavicular beaking is not a contraindication to excision.

The best treatment of talocalcaneal coalition in adults seems to be subtalar arthrodesis (Fig. 6).[2] Resection of an isolated middle-facet coalition involving less than 50% of the joint space with soft-tissue interposition is reserved for children, adolescents, and young adults. Arthritis in adjacent joints associated with a symptomatic talocalcaneal or calcaneonavicular coalition requires a full triple arthrodesis.

Peroneal Spasm Without Coalition

A small subgroup of patients may present with peroneal spasm in the absence of tarsal coalition. This condition is termed the sinus tarsi syn-





A B

Fig. 5 Examples of coalition. **A,** A CT scan of a bilateral solid talocalcaneal coalition in a 25-year-old patient. Coalition usually takes place at the level of the sustentaculum tali. **B,** An oblique radiograph shows a fibrous talonavicular coalition in a 22-year-old patient.

drome and can be caused by infection, tumor, rheumatoid arthritis, or occult trauma to the transverse tarsal or subtalar joints.

The initial treatment depends on the underlying cause. For most patients, a short-leg walking cast for 6 weeks, nonsteroidal anti-inflammatory drugs (NSAIDs), and / or a corticosteroid injection into the subtalar joint is usually efficacious. If these measures fail to alleviate the symptoms, arthrodesis may be indicated.

This is an extremely rare indication for fusion. There should be significant disability that grossly interferes with routine weight-bearing activities and sports. The risk-benefit ratio for fusion performed to correct this problem must be carefully discussed with the patient.

Fig. 6 Subtalar arthrodesis performed in 5 degrees of valgus in this patient stabilized the hindfoot and eliminated pain.

Accessory Navicular and Flatfoot

Accessory tarsal naviculars are present in 4% to 14% of the population. They are often bilateral, and the vast majority are asymptomatic. The three common types are classified on the basis of their radiographic appearance.[8,9] Type 1 is a small sesamoid-type bone within the substance of the posterior tibial tendon just proximal to its insertion into the tarsal navicular. Type 2 is a nugget of bone measuring approximately 1 cm that is connected by a cartilaginous synchondrosis to the main body of the navicular. In type 3, it is postulated that the accessory bone has fused to the main bone with an osseous bridge, resulting in a cornuate navicular.

The linkage of accessory navicular formation and pes planus has been present in the literature since the 1920s, but new studies cast doubt on this association. It was thought that the accessory navicular medializes the insertion of the posterior tibial tendon, which interferes with tarsal mechanics, resulting in a weakened, painful flat foot. While many accessory naviculars have been reported to be associated with pes planus, the vast majority appear in feet with a normal arch and are not a causative factor in the production of flatfoot.[9,10]

Accessory naviculars present as a conspicuous mass on the medial side of the midfoot. They often will not be accommodated by the normal shoe last, and local friction will cause pain. Modification of the shoe and use of an orthotic device will often relieve this simple problem. In more advanced cases, a bursa will form and may require excision.

Type 2 accessory naviculars have a tendency to become symptomatic after foot or ankle trauma. The injury disrupts the synchondrosis, and the tension and sheer forces from the posterior tibial tendon prevent healing. The accessory navicular is best seen on an external oblique view of the foot. A bone scan will show increased radionuclide uptake in the presence of a recent disruption of the syndesmosis. This finding will help differentiate this type of accessory navicular from other pathologic conditions in the midfoot. A CT scan can be helpful if the anatomy of the synchondrosis is not well seen on plain films.

Initial care should be conservative, including rest, NSAIDs, and a short-leg cast for 3 to 6 weeks. Cartilage has poor healing potential, however, and a painful nonunion often develops despite conservative care. In this situation, excision is best carried out through a medial incision splitting the posterior tibial tendon and en masse excision of the excess portion of the navicular that protrudes posteromedially, together with the accessory navicular (Fig. 7). Transfer and advancement of the posterior tibial tendon as described in the Kidner procedure is unnecessary and has not been shown to be associated with any improvement in postoperative results. A short-leg cast is used to immobilize the foot for 6 to 8 weeks. Physical therapy is then started to “reeducate” the tendon.

Fig. 7 Type 2 accessory navicular. **A,** Clinical appearance. **B,** Radiographic appearance preoperatively. **C,** Postoperative appearance. The accessory navicular should be excised flush with the medial border of the medial cuneiform and the medial talus.

Excision can be expected to provide good to excellent results, with a return to full activity. In rare cases, posterior tibial tenosynovitis develops after the excision; this responds quite well to a simple tenolysis.

Acquired Flatfoot

Posterior Tibial Tendon Tenosynovitis Syndrome

First described in 1969, posterior tibial tendon tenosynovitis syndrome has become the most common tendon problem encountered in the foot and ankle and the leading cause of acquired flatfoot deformity. As always in orthopaedics, the anatomy and the clinical findings are the critical foundations that support the treatment rationale.

The tendon has a fourfold origin from the posterior surface of the shaft of the fibula, the lateral posterior condyle of the tibia, the upper two thirds of the posterior tibial shaft, and the interosseous membrane. It then courses down the tibia behind the medial malleolus to divide into three components just proximal to the tuberosity of the navicular. The anterior component inserts into the tuberosity of the navicular and the first cuneiform. The middle component passes deep into the sole of the foot to the second and third cuneiforms and the cuboid and continues to insert into the second, third, fourth, and fifth metatarsals. The posterior component inserts on the anterior aspect of the sustentaculum tali.

The normal excursion of the posterior tibial tendon is 2 cm. It is innervated by the tibial nerve (L5-S1), and its function is to invert the subtalar joint and cause adduction of the forefoot. It also stabilizes the hindfoot by inverting the heel. Acting in concert with the flexor digitorum longus and the flexor hallucis longus, it dynamically restrains the longitudinal arch. The integrity of the static restraints of the arch (the spring ligament and the plantar calcaneonavicular ligament) is also maintained by the posterior tibial tendon.

The tendon is opposed mainly by the evertor function of the peroneus brevis and less so by that of the peroneus longus (Fig. 8). The primary site of synovitis and/or rupture is between the medial malleolus and the navicular, which may be a critical zone of hypovascularity.

Chief Complaints and Presentation

The patient complains of a progressive ache and swelling along the medial aspect of the foot and ankle, with collapse of the longitudinal arch as time passes.[11,12] There may be a history of an acute traumatic episode and a gradual decrease in endurance. Sports participation is difficult or impossible. The earlier it is in the course of the disease, the less the disability.

In the early stages (3 months to about 1 year), there is swelling and pain to palpation along the posterior tibial tendon sheath, primarily from the medial malleolus to the navicular. The patient can invert the heel and toe actively, and there is no longitudinal arch defect. The patient can do repeated single-leg toe rises, but they are painful. All joints are supple.

Subsequent examination (1 to 1½ years later) demonstrates swelling and pain along the posterior tibial tendon sheath and marked thickening of the tendon. On toe-rise there is minimal or no varus angulation of the heel. Flattening of the longitudinal arch occurs secondary to chronic lengthening of the posterior tibial tendon and failure of the spring ligament. The patient has

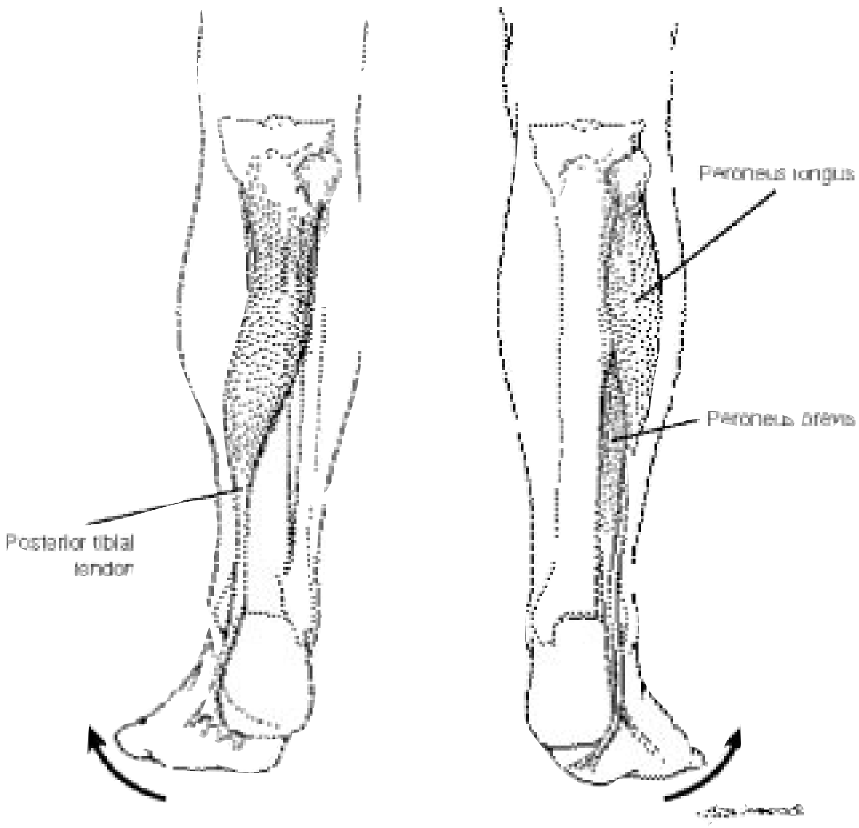


Fig. 8 **A,** The posterior tibial tendon is the prime evertor of the foot. **B,** The peroneus longus is a secondary evertor of the foot and a primary plantar flexor of the first metatarsal

difficulty doing even a few single-leg toe rises. Joint range of motion may be limited in the subtalar and transverse talar area. It is important to determine whether inversion is possible.

With a long-standing problem (duration of 1½ to 2½ years), the tendon is painful and thickened, and a gap may be palpable. There is no heel varus on toe rise. Lateral ankle pain may be present due to calcaneofibular impingement. The arch is flat. The joint range of motion is limited, and some joints may be ankylosed. There may be fixed forefoot varus.

Muscle testing is done with the foot dependent and in equinus to diminish the inversion power of the tibialis anterior. To assess the function of the posterior tibial tendon, the patient is asked to actively invert the foot. Alternatively, the evaluator can hold the patient's hindfoot in eversion, ask the patient to actively invert the heel, and then evaluate the strength in that maneuver. In younger individuals, the examiner needs to make sure that the associated pain is not from a subluxating / dislocating posterior tibial tendon.[13]

Radiographic evaluation should include weight-bearing AP and lateral views of the foot. On the AP projection, uncovering of the talo–navicular joint may be present; on the lateral projection, one looks for talonavicular, naviculocuneiform, or cuneiform–first metatarsal sag. Standing bilateral ankle radiographs should be obtained to rule out arthritis and talar tilt. Impingement of the talus against the lateral malleolus occurs in severe cases and is in itself an indication for arthrodesis. Although magnetic resonance imaging will demonstrate increased soft-tissue contrast resolution and can show inflammation, fluid, hemorrhage, and / or scar tissue, it is rarely needed, because the diagnosis is primarily made on the basis of clinical criteria.

Conservative Treatment

Conservative treatment early in the course of the disease is aimed at preventing further disability and progressive deformity and avoiding future surgical intervention. The patient with acute synovitis should be treated with rest and NSAIDs to reduce inflammation. A scaphoid pad will ease stress on the tendon. Physical therapy, including ultrasound, may also be used.

If there is no initial response to conservative care, a short-leg walking cast or brace should be used for 4 to 6 weeks. Although it may be tempting to inject a corticosteroid into the sheath, injudicious use of steroids entails a risk of further rupture.

Conservative care is also indicated in late-stage disease if the patient is not a candidate for surgical intervention. Shoe modifications alone will break down. However, the addition of a molded ankle-foot orthosis or a shoe orthosis with double metal uprights and calf and patellar-tendon-bearing containment will shift weight-bearing more proximally, thus relieving stress and pain in the foot and ankle.

Synovectomy

If synovitis persists despite conservative care and the tendon is intact, synovectomy is indicated. Failure to promptly decompress the inhospitable environment surrounding the tendon may well lead to progressive tissue degeneration, lengthening, and subsequent rupture of the tendon.[2]

An incision is made from 2 to 3 inches above to 2 to 3 inches below the medial malleolus along the course of the posterior tibial tendon. The posterior tibial tendon sheath is opened, leaving a 1.0- to 2.5-cm pulley behind the medial malleolus. The synovitis is debrided. All surfaces of the tendon are inspected, degenerated tissue is excised, and longitudinal rents within the tendon are repaired with 4-0 or 5-0 nylon. The sheath is then loosely closed, and a posterior mold is applied.

Crutches and immobilization are used for 2½ to 3 weeks, after which the sutures are removed. The anticipated duration of rehabilitation and the time to recovery will depend on the competence of the tendon. It is also important to prepare the patient preoperatively for a possible tendon transfer at the time of tenosynovectomy in case operative intervention demonstrates an incompetent, irreparable posterior tibial tendon.

Tendon Transfer

In patients presenting with synovitis and/or progressive deformity despite conservative care, a tendon transfer (Fig. 9) can be performed provided the foot is supple and shows no evidence of fixed hindfoot or forefoot deformity. Those individuals best

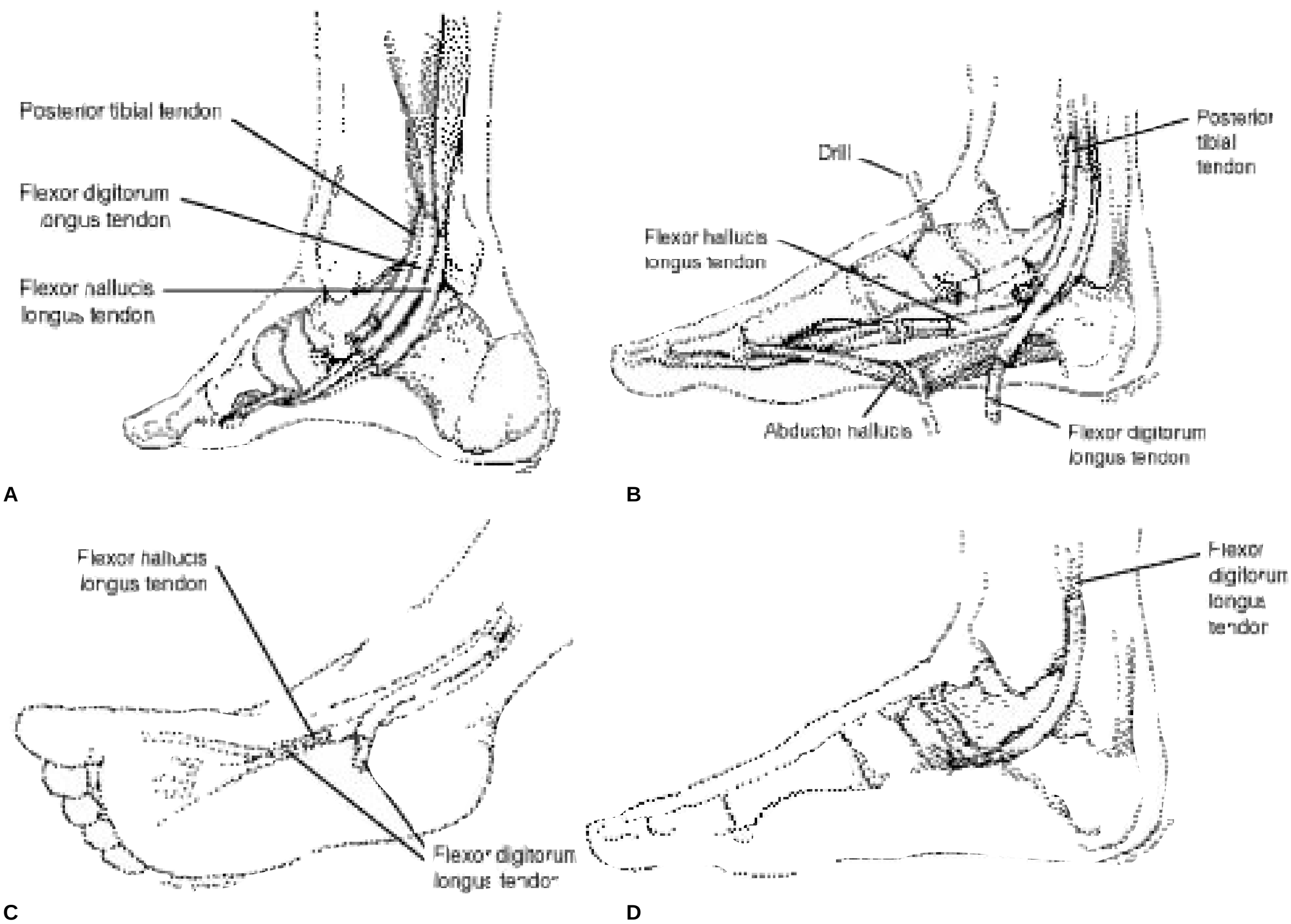


Fig. 9 Technique for tendon transfer. **A,** The incision starts just behind the musculotendinous border of the posterior tibial tendon and courses behind the medial malleolus to the base of the first metatarsal medially. Lateral **(B)** and plantar **(C)** views illustrate harvesting of the flexor digitorum longus tendon (FDL) in the midfoot as it crosses the flexor hallucis longus tendon (FHL). The distal portion of the FDL is tenodesed to the FHL to maintain adequate toe motion. A hole is drilled in the navicular. **D,** The FDL is brought up through the hole in the navicular and sutured down to itself.

suited for tendon transfer are slim, of medium to small build, under 60 years of age, active, and athletic.[2,11]

A tendon transfer is contraindicated in individuals with rigid hindfoot or forefoot deformity or lack of passive inversion of the subtalar joint. Relative contraindications are obesity, large build, sedentary lifestyle, and age over 60 to 70 years. A hypermobile foot is also a relative contraindication to tendon transfer.

The flexor digitorum longus is the tendon of choice. However, there have been reports of successful transfer of the flexor hallucis longus tendon.

The technique for tendon transfer[2,11] is as follows: The incision should extend from 10 cm proximal to the medial malleolus posterior to the tendon sheath and distally to the metatarsocuneiform joint (Fig. 9, A). The sheath is explored from the medial malleolus to the navicular to assess the quality of the tendon. If it is incompetent, one should then proceed with the transfer.

The abductor hallucis is reflected plantarward, and the master knot of Henry is released to expose the crossing of the flexor hallucis longus and the flexor digitorum longus. At that level, the two tendons are sutured together, and the proximal portion of the flexor digitorum longus is released (Fig. 9, B). If possible, the posterior tibial tendon sheath should be used. However, if it is too scarred, the flexor digitorum longus sheath can be a substitute.

A hole is drilled vertically in the navicular, and the tendon is passed through it from plantar to dorsal. The tendon is then tied to itself and the adjacent periosteum. It is important that the drill hole in the navicular be sufficiently within the midbody portion so that the tendon does not break through medially. The tendon should be sutured to itself with the foot in maximum inversion and equinus for good tension (Fig. 9, C). If the posterior tibial muscle is of good quality, it can be used to augment the tendon transfer.

An equinus short-leg cast is applied, and the patient stays non-weight-bearing for 1 month. A short-leg walking cast in neutral is then used for 1 month, after which aggressive rehabilitation is started. Arch supports may be helpful as part of the postoperative regimen.

With flexor digitorum longus transfers, the original deformity (sag in the longitudinal arch) will not be corrected, but progression may be halted, accompanied by significant clinical improvement. Adding a repair of the spring ligament and/or a calcaneal osteotomy to restore the integrity of the longitudinal arch is now considered but has not yet been subjected to a large clinical study.

If, on initial examination, the surgeon notes an avulsion of the tendon from the navicular, a tendon transfer should still be done. Attempts at reattachment of avulsed tendons do poorly in this area.[12]

Arthrodesis

Marked deformity associated with arthritis and/or fixed osseous deformity is best managed by arthrodesis. To help stabilize the hindfoot, the choices are a talonavicular arthrodesis, a double arthrodesis at Chopart's joint, a subtalar arthrodesis, and a triple arthrodesis. The more extensive the arthrodesis, the more rigid the foot and, therefore, the greater the stress transfer to distal and proximal joints. Thus, a procedure should be chosen that will involve the fewest joints and still stabilize the foot.

Talonavicular arthrodesis as an isolated procedure can be used for an unstable talonavicular joint when the remainder of the foot is supple. It is best used in an older patient with low physical demands. It is important to place the subtalar joint in 5 degrees of valgus angulation at the time of fusion to preserve maximum motion in the hindfoot.

A double arthrodesis involving the talonavicular joint and the calcaneocuboid joint is indicated in the younger patient with an unstable talonavicular joint but a stable subtalar joint.[14] There is less morbidity than is associated with a triple arthrodesis, but the procedure offers the same stability (Fig. 10).

Isolated subtalar arthrodeses are indicated in the presence of a rigid or incompetent subtalar joint accompanied by a flexible forefoot and a stable talonavicular joint. It is an excellent alternative to a double arthrodesis, and when the heel is left in 5 degrees of valgus angulation, excellent motion of the foot is preserved.

A triple arthrodesis is indicated in the presence of a fixed valgus deformity of the subtalar joint, fixed abduction of the transverse talar joints, and fixed varus deformity of the forefoot (Fig. 11). An inlay tricortical graft may be required in patients with a severe valgus deformity. Although fusion in situ was accepted in some earlier reports in the literature, failure to correct the subtalar joint to 5 degrees of valgus angulation may well put increased stress at the tibiotalar and intertarsal joints, leading to subsequent breakdown and further collapse.

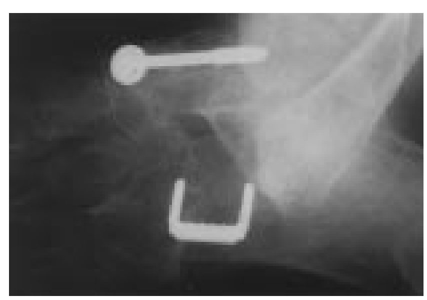


Fig. 10 In a double arthrodesis, the calcaneocuboid and talonavicular joints are fused with internal fixation.

Fig. 11 A triple arthrodesis fuses the calcaneocuboid, talonavicular, and talocalcaneal joints. Internal fixation maintains position and enhances healing.

Flatfoot and Rheumatoid Arthritis

Rheumatoid arthritis affects the foot in up to 90% of patients; with increasing duration of disease, up to 50% of these patients will develop a progressive flatfoot deformity.[15,16] The repetitive stress of weight-bearing superimposed on disease-weakened ligamentous supports leads to a progressive planovalgus deformity of the hindfoot. With the passage of time, secondary osteoarthritis may develop, accompanied by a fixed deformity. There is also evidence to suggest that a progressive valgus deformity at the hindfoot causes excessive stress at the ipsilateral knee, leading to a progressive genu valgum deformity.

Early recognition is important. Early use of shoe modifications and braces may prevent progression of the deformity. If conservative treatment fails, surgical intervention should be prompt to prevent the difficult problems associated with an end-stage deformity.

A talonavicular arthrodesis will be durable in a patient with low physical demands, but a double arthrodesis of the talonavicular and calcaneocuboid joints is advised for the more active patient with a flexible deformity.[14] An isolated subtalar arthrodesis may stabilize the foot if the disease is limited to that joint. A triple arthrodesis is reserved for a major deformity with diffuse arthritic changes.

Arthrosis

Degenerative arthritis usually is posttraumatic, but a less common primary form may develop in the joints of the hindfoot and midfoot, leading to collapse and pes planus. In mild cases, the initial presentation will include pain, swelling, limited motion, and decreased endurance. Once the joints involved have been identified, NSAIDs and shoe modifications and orthotic devices should be the initial treatment.

With intractable pain despite conservative treatment, continued progression of the deformity, or the presentation of a late deformity in an unacceptable position, surgery is indicated. Careful clinical examination, bone scanning, CT, and perhaps selected injections into the involved joints may be helpful in determining the extent of the arthritic changes. To keep proximal and distal stress transfer to a minimum, the fusion should be limited to those joints that are involved. The use of internal fixation and consideration of supplementary bone graft are thought to improve the probability of successful fusion.

Pes Planus Associated With Charcot Foot

Diabetes is now the leading cause of Charcot arthropathy, and the foot is the primary site of involvement. Ninety percent of the changes take place in the midfoot and hindfoot.[17] Repetitive minitrauma in the presence of neuropathy leads to microfractures, which cause progressive subluxation, fragmentation, and collapse into a flatfoot deformity (Fig. 12).

Fig. 12 Midfoot collapse in a Charcot foot with subluxation, fragmentation, and an abnormal plantar bony prominence

Management of this problem requires a thorough knowledge of the syndrome. Surgery is reserved for initial or late deformities in which the displacement is so severe that bracing is not a viable option.[18]

Flatfoot Associated With Neurologic Deficit

Pes planus may develop in the adult foot secondary to an acquired neurologic deficit and muscle imbalance. Head injuries, stroke, and peripheral nerve lesions are the most common causes.[19,20] Overcorrection of equinovarus (which is much more common in the neurologically impaired patient) may also result in pes planus.

Patients must be assessed individually to determine whether the deformity is supple or rigid, to identify the phase of gait during which the deformity occurs, and to seek associated osseous changes. A kinesiologic electromyographic study is often helpful.

Supple deformities may require bracing. However, surgery including tendon transfers and/or arthrodeses may be required to produce a balanced, pain-free plantigrade foot to improve shoe wear, wheelchair positioning, decreased brace wear, and ambulation.

Posterior Tibial Tendon Laceration

Laceration of the posterior tibial tendon occurs infrequently. When recognized, however, it should be promptly repaired end to end. If discovered late, especially in an active individual with no associated arthritic changes, a flexor digitorum longus transfer, as described previously, should be performed.

Plantar Fascia Rupture

Posttraumatic or spontaneous rupture of the plantar fascia may occur with subsequent limited flattening of the longitudinal arch. Pain is felt on the plantar surface of the sole just distal to the calcaneal tuberosity. In some individuals a gap may be palpated. Radiographs are usually unremarkable.

This condition is extremely painful and is associated with prolonged morbidity. With shoe modifications and a supportive orthosis, the rupture will heal in time. The flattening of the arch will be moderate at most and should not progress to symptomatic flatfoot.

Summary

The ability to care for symptomatic flatfoot in the adult is an important skill in the armamentarium of the orthopaedic surgeon. Careful clinical and radiographic evaluation, coupled with a thorough understanding of the anatomy and biomechanics of the foot, will allow accurate evaluation and appropriate treatment.

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