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# The Flexible Flatfoot in the Adult

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The adult acquired flatfoot deformity is characterized by flattening of the medial longitudinal arch with insufficiency of the supporting posteromedial soft tissue structures of the ankle and hindfoot [1]. Although the etiology of this deformity can be arthritic or traumatic in nature [1–5], it is most commonly associated with posterior tibial tendon dysfunction (PTTD) [2]. By one estimate, PTTD affects approximately 5 million people in the United States [6]. The clinical presentation of adult flatfoot can range from a flexible deformity with normal joint integrity to a rigid, arthritic foot.

Johnson and Strom [7] described three stages of PTTD, with an additional stage added by Myerson [8]. Stage I consists of painful synovitis of the tendon. Nevertheless, tendon length and function are maintained so there is no deformity. With stage II disease, there is progressive tendon dysfunction and a flexible flatfoot deformity develops. Stage III involves a rigid deformity with stiffness and often arthritis of the midfoot and hindfoot. Finally, stage IV consists of tibiotalar valgus, usually with associated arthritic change [1].

The prevalence and increased awareness of PTTD has sparked a recent trend toward surgical interventions that involve joint preservation techniques [6,9]. This article focuses on the anatomy, diagnosis, and current treatments of the flexible flatfoot deformity.

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One author has a financial interest in the Prostop arthroereisis device (Arthrex Inc., Naples, FL).

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## Anatomy and biomechanics

The relevant anatomy of the flexible flatfoot deformity not only includes the posterior tibial tendon (PTT), but also the spring ligament, deltoid ligament complex, and the articular relationship of the talonavicular and subtalar joints. The PTT has its origin on the posterior tibia, fibula, and interosseous membrane (Fig. 1). It courses immediately posterior to the medial malleolus and inserts on the medial midfoot [1,10]. In an 11-specimen cadaver dissection, Bloome and colleagues [11] demonstrated that the PTT has three consistent bands: anterior, middle, and posterior. The anterior band represents 65% of the tendon width and inserts on the navicular tuberosity, inferior naviculocunieform joint capsule, and the inferior medial cuneiform. The posterior band comprises 20% of the tendon width, originates 2–3 mm proximal to the anterior band, and helps form the acetabulum pedis. The middle band represents approximately 15% of the PTT and is the tarsometatarsal extension with insertions to the middle/lateral cuneiforms, cuboid, and second to fourth metatarsals. The investigators also found variations of insertions to the spring ligament (4 of 11), fifth metatarsal base (7 of 11), flexor hallucis brevis (9 of 11), peroneus longus (4 of 11), and abductor hallucis (5 of 11) [11].

The vascular supply of the PTT consists of branches of the posterior tibial artery. Proximal to the medial malleolus, the PTT has vessels in the synovial sheath, which arise from muscle branches. Distally, the insertion has a periosteal blood supply. In between, there is a zone of relative hypovascularity, which often corresponds to the site of attritional rupture and extends approximately 14–15 mm distal to the medial malleolus [12].

The spring ligament complex cradles the plantar medial aspect of the talar head extending from the anterior margin of the sustentaculum tali to the plantar medial aspect of the navicular [1]. It is comprised of a superomedial

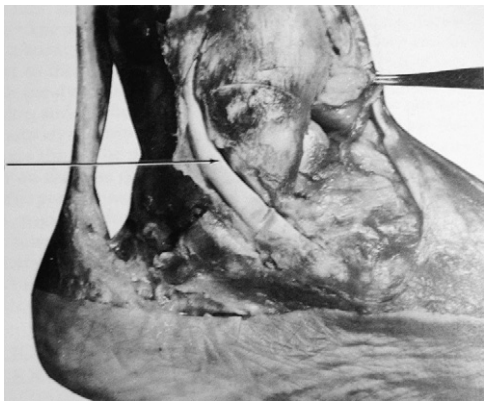

Fig. 1. Anatomy of the PTT. Arrow points to the posterior tibial tendon. (*From* Sarrafian S. Anatomy of the foot and ankle: descriptive, topographical. 2nd edition. Philadelphia: JB Lippincott; 1993. p. 217; with permission.)

calcaneonavicular portion that lies medial to the PTT and blends with the deltoid, which is wider and stronger than the inferior portion. During surgery, this ligament is often found to be torn [13]. The inferior calcaneonavicular ligament is narrower and lies at the plantar most aspect of the acetabulum pedis [14]. A third component, or articular portion, is located between the two bands and articulates with the inferior aspect of the talar head.

The deltoid ligament complex has been described as having six separate ligaments and deep superficial layers [15]. The deltoid prevents external rotation of the talus in the mortise and prohibits abduction of the ankle [16,17]. The anterior portion of the deltoid has a confluence of fibers with the spring ligament complex [10,13,14].

Brodsky [18] stressed the importance of understanding the biomechanical functions of the foot to comprehend the complex progressive deformity associated with adult acquired flatfoot. In normal gait, the tibialis posterior muscle produces inversion of the hindfoot. It is also an adductor of the forefoot at the midtarsal joint opposing the action of the peroneus brevis. The posterior tibial muscle functions during the stance phase of gait. After heel contact, the muscle acts as a shock absorber for the subtalar joint, which limits hindfoot eversion by eccentric contraction [18].

During mid-stance, the posterior tibial muscle contraction causes subtalar inversion, thus locking the transverse tarsal joints. This results in a rigid lever for forward propulsion by allowing the powerful gastrocnemius–soleus complex to act through the foot at the metatarsal heads. During the propulsive phase, the tibialis posterior muscle functions to accelerate subtalar joint supination and assist in heel lift. The shift of the body weight center from lateral to medial through the longitudinal axis of the foot during the propulsive phase of gait is achieved by the balanced activity of the tibialis posterior and peroneal muscles. The posterior tibialis muscle then becomes inactive shortly after heel lift, with the residual effect of acceleration during swing phase [18].

With PTTD, this balance is altered and weighted toward the peroneals with greater hindfoot eversion and ligamentous tension and stretching during stance. In mid-stance there is decreased inversion of the subtalar complex, such that the hindfoot cannot function as a rigid lever [18]. The gastrocnemius–soleus then acts at the talonavicular joint creating excessive midfoot stress, which allows increased midfoot abduction and produces a levering action at the tarsometatarsal joints. In the propulsive phase, there is less lateral column loading and a delayed heel lift that reduces the magnitude of propulsive activity [18]. The repetitive biomechanical alteration of gait results in progressive midfoot collapse, forefoot abduction, and excessive hindfoot valgus [18].

## Pathology

An acquired flexible flatfoot deformity is most often associated with PTTD. Biomechanic overloading as described above can lead to chronic

microtrauma in the tendon. Microtears occur after 1500 to 2000 cycles per hour and trigger an inflammatory response [3]. With advancing age, the tendon's elastic compliance decreases because of changes in collagen structure [3], thus creating a pathologic sequence where tendon weakening results in failure of the static stabilizers of the arch. Poor blood supply may initiate this process or may prevent an adequate healing response, resulting in chronic inflammation, tenosynovitis, and tendinosis. Both Thordarson and colleagues [19] and Deland and colleagues [20] were able to demonstrate the relationship of medial soft tissue failure in cadaver models with sequential sectioning of the spring ligament complex and posterior medial structures.

Deland and colleagues [14] reviewed MRI of patients with PTTD and aged-matched controls. They evaluated the PTT, spring ligament complex, talocalcaneal interosseous ligament, long and short plantar ligaments, plantar fascia, deltoid complex, plantar naviculocunieform ligament,and tarsometatarsal ligaments. The investigators found that ligament involvement is extensive with PTTD, with the most significant pathology in the superior and inferior medial calcaneonavicular ligaments and the interosseous ligament [14].

Developmental etiologies also may be responsible for a flexible flatfoot deformity. These include conditions associated with soft tissue laxity (Ehlers-Danlos and Marfan syndromes), accessory navicular, and neuromuscular diseases [11,21]. Extrinsic factors are less common but can result from trauma involving the medial structures in an eversion type injury. Brodsky and colleagues [4] reported two cases of traumatic rupture of the PTT in athletes, and Park and colleagues [5] described PTTD secondary to os subtibiale impingement.

Two potential mechanical causes of an acquired flatfoot deformity include medial column instability and a contracture of the Achilles tendon or gastrocnemius fascia. With the former, medial column instability results in forefoot varus and a compensatory hindfoot valgus [1]. With the latter, a tight Achilles tendon or gastrocnemius fascia results in transmission of dorsiflexion forces from the ankle to the transverse tarsal joint and midfoot. This leads to midfoot collapse and hindfoot valgus with lateral peritalar subluxation of the navicular and subfibular impingement [22,23].

## Clinical examination

The diagnosis of the flexible flatfoot is relatively straightforward. However, the subtleties of the associated pathology require a systematic approach to examination and work-up. Patients usually complain of medial ankle and hindfoot pain that radiates to the arch of the foot or proximally to the leg. As the deformity progresses, there may be a complaint of lateral or sinus tarsi pain caused by subfibular impingement. Although some

patients will attribute a nonspecific traumatic event to the pain, most patients will relate a gradual onset of the pain with loss of the medial plantar arch over recent months or years. A careful history is important and should include previous trauma, steroid use, orthotic use, diabetes, smoking, and family history of inflammatory arthropathy. Body mass index may helpful with patient counseling and surgical decision-making.

On physical examination, it is helpful to evaluate the patient in short pants with both shoes off. This allows the clinician to note the alignment of not only the foot and ankle, but also the knee. With genu valgus, an individual's center of gravity may be altered and more load may be placed on the medial ankle and PTT. Comparison of tread wear on the shoes may reveal more posteromedial wear than the opposite side. On examination of the standing patient from behind, the presence of hindfoot valgus can be noted and measured, and the "too many toes" sign can be identified (Fig. 2). The patient should be asked to perform a double leg heel rise so that the presence or absence of hindfoot inversion can be identified. Next, the patient is asked to perform a single leg heel rise on the affected side noting that inability to do so is consistent with PTTD.

Examination sitting should include assessment of ankle and subtalar range of motion. Ankle motion should be measured with the knee extended and flexed with the transverse tarsal joint locked and unlocked. This will allow the examiner to assess for Achilles tendon and gastrocnemius contractures. Palpation of the posteromedial ankle and hindfoot may reveal tenderness, swelling, or fullness. The sinus tarsi, talar dome, and navicular tuberosity should be palpated. Callus formation over the subluxated talar head may be noted. For patients who have a flexible flatfoot, reduction of the talonavicular joint and correction of the hindfoot valgus/forefoot abduction is possible. Lastly, the PTT strength is tested with resistance against the inverted and plantarflexed foot.

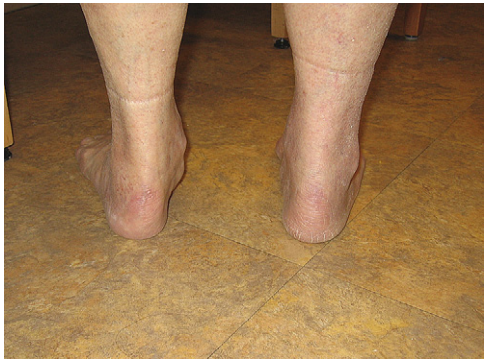

Fig. 2. Clinical photograph of a patient who exhibits the "too many toes" sign.

## Diagnostic imaging

Radiographs should be obtained on the first office visit and should include weight-bearing views of the foot and ankle. Non–weight-bearing views fail to provide valid information regarding the alignment of the foot [1,24]. The presence of joint space narrowing or varus/valgus deformity of the ankle should be noted. Selected measurements of the lateral and anteroposterior radiographs to measure the degree of the deformity have been shown in one study to be inaccurate [25]; however, Saltzman and colleagues [26] demonstrated that with careful measurement techniques, reliability is possible.

Recently, Younger and colleagues [27] studied inter- and intraobserver reliability in the evaluation of plain radiographs of patients with PTTD. On lateral radiographs they measured talar-first metatarsal angle (Meary's angle), calcaneal pitch, talocalcaneal angle, medial column height, calcaneal-fifth metatarsal height, and lateral column height. On anteroposterior radiographs they measured talar-first metatarsal angle, talonavicular uncoverage angle, and calcaneal-fifth metatarsal angle. The axis of the talar-first metatarsal angle on the lateral radiograph was the most discriminating and statistically significant radiographic parameter in patients who had symptomatic flatfoot. On the anteroposterior radiograph, the talar head uncoverage was the most statistically significant value, despite low inter- and intraobserver reliability [27].

Clinical examination and radiographs are usually sufficient to establish the diagnosis of PTTD. In certain instances, however, the use of MRI can be helpful to confirm the diagnosis, evaluate the amount of pathology in the PTT and spring ligament complex, and detect bone edema (Fig. 3) [1,14,28]. Meanwhile, CT can be helpful in the evaluation of advanced lateral impingement and the detection of arthritic change [23]. When a flexible flatfoot is associated with minor degenerative change (especially of the

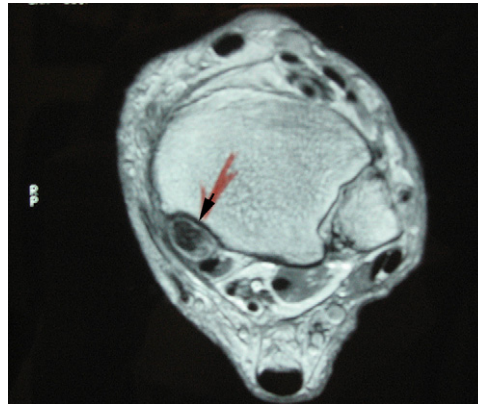

Fig. 3. Axial (proton density weighted) MRI of an attenuated PTT. Arrow points to the posterior tibial tendon.

tarsometatarsal joints), selective injection with local anesthetic under fluoroscopy can help differentiate the amount of the patient's discomfort that is attributable to the degenerative change.

In some patients, the discomfort that is caused by the chronic inflammation and tendinosis associated with PTTD cannot always be elucidated clearly on examination or imaging. In these instances, a diagnostic injection of local anesthetic into the PTT sheath can be helpful. The authors prefer to use a 1:1 mixture of 1% lidocaine and 0.25% marcaine. After the skin is sterilized, a 22-gauge needle is inserted parallel to the PTT approximately 1–2 mm above the medial malleolus and 2–3 mL of the anesthetic is injected. Care is taken to ensure that the needle is not deep within the tendon. The patient is then asked to ambulate for a few minutes after which they are re-examined.

Cooper and Mizel [29] recently reviewed 17 ankles with stage I PTTD that had a diagnostic injection and MRI of the PTT at different times. They found that although 15 of 17 ankles (88%) had positive MRI findings, all 17 ankles (100%) had relief of pain from the diagnostic injection. The investigators concluded that diagnostic injection is an accurate, sensitive, and safe diagnostic tool [29].

## Nonoperative treatment

The stage and progression of the flatfoot deformity will generally determine the degree and duration of the conservative treatment [30]. The initial treatment of the adult flexible flatfoot deformity (stage II PTTD) focuses on improving symptoms by decreasing the forces transmitted through the posteromedial hindfoot [1]. The patient should be encouraged to lose weight, modify repetitive loading activities, and use supportive shoes [1,2]. Cast immobilization or a removable walking boot with an insert that supports the longitudinal arch can be helpful with acute inflammation. A stirrup brace can be used and has been shown to assist in unloading the tendon by transferring the plantarflexion component solely to the Achilles tendon [3].

Some patients may tolerate footwar modifications, but extra deep shoes that can house adequate orthosis are often necessary. Key elements include long, rigid medial counters, soft leather uppers, high toe boxes, and shock absorbing soles to decrease the ground reaction forces [30]. A moderate rocker bottom will assist in toe off [30].

Many patients who have a flexible flatfoot deformity also may benefit from custom prescribed orthotics. Either a full-length or $\frac{3}{4}$-length device may be used. Most often a medial post and deep heel is used to control the hindfoot. The more rigid University of California at Berkeley Laboratory (UCBL) orthosis is designed to correct the flexible hindfoot and hold it in a neutral position. It limits motion in the subtalar joint and helps prevent subfibular impingement by exerting pressure on the lateral wall of the calcaneus and the medial longitudinal arch [1,30]. Havenhill and colleagues

[31] studied contact pressures in flatfoot cadaver specimens and then compared change with a UCBL versus a medializing calcaneal osteotomy (MCO). The UCBL and the osteotomy significantly reduced the tibiotalar contact pressure and peak pressure. The investigators concluded that both options can be used for clinical management of the flexible flatfoot. Although the UCBL can be quite helpful in correcting the flexible flatfoot deformity, some patients do not tolerate the device because of pressure over the navicular and/or difficulty fitting it in shoes.

An ankle foot orthosis (AFO) can correct deformity by stabilizing the medial to lateral movement of the hindfoot and limiting excursion of the PTT by preventing plantar flexion and pronation in the push-off stage of gait [30]. A rigid, molded AFO can be useful during the initial stage of flexible flatfoot treatment, but it can lead to compliance issues for long-term use.

Recently, the Airlift PTTD Brace (djOrtho, Inc., Carlsbad, California) has been introduced for patients who have a flexible flatfoot deformity [32]. Like other athletic ankle braces, the Airfliftbrace has a low profile, semirigid vertical shell that helps to control hindfoot valgus. In addition, the Airlift PTTD Brace incorporates an inflatable bladder underneath the hindfoot and midfoot (Fig. 4). When inflated, this bladder supports and elevates the medial longitudinal arch. Early anecdotal experience with this device has been encouraging with regard to compliance and symptomatic relief.

Physical therapy also should be considered, because it serves to strengthen the PTT and surrounding tendons, thereby providing a stronger structural support for the foot and lessening the stress on the PTT [3]. Any physical


Fig. 4. The Airlift PTTD Brace. (*Courtesy of* djOrtho, Carlsbad, CA; with permission.)

therapy regimen also should address the presence of a gastrocnemius contracture. Cryotherapy may be considered also after activity, because it decreases the release of inflammatory mediators. Iontophoresis with topical steroidal anti-inflammatory cream can be added to the treatment and has not been shown to cause an increased risk in tendon rupture [3].

Alvarez and colleagues [33] recently assessed the efficacy of a focused nonoperative treatment protocol for stage I and stage II PTTD. The investigators found only four reports [34–37] of nonoperative management of PTTD and designed a program that included the use of a short articulated AFO or foot orthosis, high-repetition exercises, aggressive plantarflexion activities, and an aggressive high-repetition home exercise program that included gastroc-soleus tendon lengthening. They studied a group of 47 patients (37 women and 10 men) over a three-year period. Patients who were unable to perform a single leg heel raise or had pain for greater than 3 months were given an AFO, and the remainder of patients were given a UCBL type orthosis. Patients underwent a four-phase intensive treatment program for 4 months with a minimum of 10 physical therapy visits. Investigators found that all patients had significant weakness of all ankle muscle groups before therapy. After treatment, 83% of patients had successful subjective and functional outcomes, and 89% of patients were satisfied with their results [33].

## Operative treatment

### *Overview*

Once nonoperative treatment measures have failed, operative intervention focuses on soft tissue balancing with tendon transfer in combination with one or more osteotomies to return the foot to a more normal anatomic position and restore normal foot biomechanics [38]. Before 1980, the adult flatfoot was routinely treated with a triple arthrodesis; thereafter, the operative focus has been on augmentation or substitution of the PTT [1]. Initial results with isolated soft tissue procedures generally were not as successful and often lead to failure caused by lack of bony realignment [1]. Currently, some type of bony realignment is recommended to augment and protect the soft tissue reconstruction.

Hiller and Pinney [9] recently conducted a large survey of foot and ankle surgeons to assess the state of practice in the approach to surgical reconstruction of stage II PTTD. In this study, a sample operative was presented to each surgeon. The investigators found that 97% of respondents reported they would employ some type of bony procedure in their surgical treatment. Eighty-eight percent described techniques that would preserve the subtalar and talonavicular joints. These techniques included an MCO in 73% of patients, a lateral column lengthening in 41% of patients, and medial column stabilization (first tarsometatarsal and/or navicular cuneiform arthrodesis)

in 15% of patients. Twelve percent of respondents reported that they would perform an arthrodesis on one or more of the hindfoot joints. Ninety-eight percent of respondents reported that they would employ some type of soft tissue procedure. Ninety-four percent of respondents would augment the PTT, 53% would formally repair the spring ligament, and 70% would address a presumed equinus contracture. The investigators concluded that most surgeons employed a combination of bony and soft tissue procedures that preserved the subtalar and talonavicular joints [9].

Soft tissue augmentation is performed to restore or recreate the function of the PTT. Whereas many surgeons prefer to transect the PTT to remove the degenerative tissue, this may not always be necessary. Valderrabano and colleagues [39] performed MRI analysis of the PTT and muscle in patients who underwent successful flatfoot reconstruction. The investigators found that even though fatty degeneration of the posterior tibial muscle was present in all patients preoperatively, there was a decrease in degeneration with increasing strength of the posterior tibial muscle and muscular size postoperatively. They also found that the recovery potential of the posterior tibial muscle was significant even after delayed repair of a diseased tendon. They concluded that the PTT should not be transected because it precludes recovery potential of the posterior tibial muscle [39].

Wacker and colleagues [28] studied the MRIs of 12 patients who had PTTD and found that all patients had atrophy of the PTT muscle and compensatory hypertrophy of the flexor digitorum longus (FDL) muscle. In the three patients who had complete rupture of the PTT, fatty infiltration occurred throughout the muscle. They suggested that in patients who have an intact but diseased PTT, augmentation with preservation or tenodesis of the PTT may benefit the reconstruction [28].

The spring ligament (calcaneonavicular) complex often is attenuated and contributes to the pathology of the adult flexible flatfoot. As such, reconstruction of the spring ligament is performed frequently to augment the medial soft tissue reconstruction. The attenuated complex can be imbricated with suture or advanced and secured with small suture anchors in the navicular. Choi and colleagues [40] reported using peroneus longus tendon in a cadaver model to reconstruct the spring ligament complex. They tested three different configurations and found that a superomedial/plantar passage of the tendon through the calcaneus and navicular best corrected the talonavicular deformity [40].

Although it is commonly assumed that transfer of the FDL tendon has no morbidity secondary to the intertendonous connections with the flexor hallucis longus (FHL) tendon, a recent cadaver study examined the distal anatomic relationship of these structures [41]. The following three different configurations were found: a tendon slip from FHL to FDL in 10 feet (42%); a tendon slip from FHL to FDL and another slip from FDL to FHL in 10 feet (42%); and no attachment between FHL and FDL in four feet (16%) [41].

## Flexor digitorum longus transfer

In 2001, Guyton and colleagues [42] performed a retrospective review of 26 patients who had a combined FDL transfer and an MCO. Two patients experienced early failure of fixation, and the remaining 24 patients were able to perform a single leg heel rise within one year. The study found that patients continued to improve for up to 10 months after the procedure. It also noted that while surgery improved pain and function, it did not appreciably improve the medial arch [42].

Wacker and colleagues [28] reported 3–5 year results on 44 patients who underwent FDL transfer and an MCO. They reported persistent improvement in the American Orthopaedic Foot and Ankle Society (AOFAS) hindfoot score (from 48.8 to 88.5). Guyton and colleagues [42] reported similar results; 43 of 44 patients rated their outcome with regard to pain and function as good or excellent. However, only 36 of 44 patients rated their alignment as good or excellent.

Myerson and colleagues [43] reported on a large series of 129 patients with a mean of 5.2 years follow-up. These investigators found that the procedure yielded excellent results with minimal complications; the mean AOFAS hindfoot score was 79. They reported that patients experienced improvement in symptoms for up to 14 months postoperatively, and that patients who had lower preoperative levels of daily function had the greatest benefit from the procedure. The investigators surmised that the ideal patient for a combined FDL transfer with an MCO is a patient who has a flexible flatfoot, insignificant forefoot supination, and less than 30% uncovering of the talar head on anteroposterior radiographs [43].

Brodsky [18] performed a prospective pre- and postoperative gait analysis study on 12 patients with PTT reconstruction. Six of the patients had concomitant midfoot fusion that resulted in a significant improvement in radiologic parameters. Although thesample size was limited, Brodsky found small but statistically significant increases in cadence, velocity, and maximum sagittal ankle joint power.

Sullivan and colleagues [44] recently described an alternative method for fixation of the FDL transfer. Using a cadaver model, they placed an anchor in the proximal/plantar portion of the navicular tunnel and tensioned the FDL by a passing suture through the distal/dorsal portion of the tunnel. This technique was believed to entail a shorter incision and less dissection in addition to obviating the need to disrupt the FDL distal to the knot of Henry. The investigators found identical pullout strength compared with the standard tendon to tendon repair with a navicular tunnel [44].

### *Surgical technique: flexor digitorum longus transfer*

After completion of the bony correction (MCO or LCL), a curvilinear incision is made from the posterior aspect of the medial malleolus to the medial

navicular tuberosity. The PTT sheath is opened to expose the tendon. Quite often the tendon has longitudinal tears with areas of tendinosis or detachment from its insertion on the navicular. If the tendon has minimal or no excursion, it is resected at the most proximal aspect of the incision. In cases where PTT has 1 cm or more of excursion, the tendon can be saved for tenodesis to the FDL transfer.

A capsular incision between the talar head and navicular can be made at this time to remove any redundant tissue. Then the FDL is harvested proximal to the knot of Henry. The FDL is brought proximally into the wound and a grasping stitch placed in the tendon. A bone tunnel is made from the anatomic insertion of the PTT dorsally and distally through the navicular. This is performed with a 5.5-mm cannulated drill over a guide wire if necessary. A sufficient bone bridge must be maintained to avoid fracture. Then the FDL tendon is passed from plantar to dorsal through the bone tunnel. The foot is plantar flexed and inverted while the FDL is tensioned. A 4.5-mm interference screw is placed to secure the FDL within the tunnel. Another form of fixation includes the use of heavy suture passed through the tendon and into the periosteum on either side the tunnel.

The wound is closed in layers with the foot splinted in plantarflexion and inversion. At 2 weeks postoperatively, sutures are removed and the patient is placed in a short leg cast. At 5–6 weeks postoperatively, a removable boot brace is prescribed, and weight-bearing and motion are encouraged.

## Medializing calcaneal osteotomy

An MCO is performed to correct the hindfoot valgus deformity and also redirect the coronal vector of the Achilles tendon. Using pressure sensitive film, Steffensmeier and colleagues [45] studied the effects of medial and lateral displacement on tibiotalar joint contact. They loaded cadaver specimens in three positions: neutral, 1 cm of lateral displacement of the posterior fragment, and 1 cm of medial displacement of the fragment. For an applied load equaling two times body weight, a 1-cm lateral displacement shifted the center of pressure an average of 1.06 mm laterally, whereas a 1-cm medial displacement shifted the center of pressure an average of 1.58 mm medially [45]. Regional contact parameters changed in a reproducible and statistically significant manner. Among four equal sized, para-sagittally bounded cartilage zones, lateral displacements consistently unloaded the most medial zone and increased loading of the most lateral zone; medial calcaneal displacements had the converse effect. The investigators concluded that translational calcaneal osteotomies may be used clinically to partially offload focal areas of cartilage along the medial and lateral borders of the tibiotalar joint [45].

It was initially hypothesized that plantar fascia tightening occurs with these procedures, helping to restore a more normal longitudinal arch. In 1998, Horton and colleagues [46] studied nine cadaver specimens using

a flatfoot model. They measured fractional length changes in the plantar fascia after a medial displacement calcaneal osteotomy and after lateral column lengthening through the calcaneocuboid joint. They reported that tightening of the plantar fascia did not occur with either medial calcaneal displacement or lateral column lengthening; moreover, the plantar fascia became significantly less taut with both procedures. The investigators also found that lateral column lengthening produced significantly looser plantar fascia than did medial calcaneal displacement [46].

Hadfield and colleagues [6] performed a 1-cm MCO on 14 cadaver specimens and measured the effects on Achilles tendon lengthening and changes in plantar foot pressures. They found a statistically significant decrease in average pressure over the first and second metatarsal regions and a concomitant increase in pressure over the lateral aspect of the calcaneus and the lateral forefoot.

Greene and colleagues [47] studied the anatomic relationship of the medial neurovascular structures in relation to an MCO in 22 cadaver specimens. They performed an MCO in each specimen with subsequent dissection medially to identify the medial plantar nerve (MPN), the lateral plantar nerve (LPN), the posterior tibial artery (PTA), and their respective branches. The investigators found that an average of four neurovascular structures crossed each osteotomy site (range 2–6), most of which were branches of the LPN or the PTA. The MPN did not cross in any of the specimens studied, the LPN crossed in one specimen, and the PTA crossed in two specimens. The calcaneal branch of the LPN was identified and crossed in 86% of the cadavers. A more distal second branch of the LPN (Baxter's nerve) was identified and crossed in 95% of the specimens. Each PTA distributed from zero to three branches that variably crossed the osteotomy at a point from 2% to 100% along its length. The investigators recommended that completion of the osteotomy through the medial calcaneal cortex be carefully performed in a controlled manner to reduce the risk of complications [47].

*Surgical technique: medial calcaneal osteotomy*

The patient is positioned lateral after a thigh tourniquet is placed. An oblique incision is made one finger breadth below the tip of the lateral malleolus. Sharp dissection can be performed down to the lateral calcaneal wall. Still, care should be taken with the skin edges and with the sural nerve if encountered. The direction and level of the MCO is confirmed with fluoroscopy. The osteotomy is performed with a sagittal saw and can be completed with a thin osteotome. Then the calcaneal tuberosity is displaced medially by approximately 1 cm. Distal translation also can be performed to increase calcaneal pitch. The osteotomy is secured with a 6.5-mm or 7.3-mm cannulated screw place from posterior to anterior (Fig. 5). Then the wound is closed in layers.

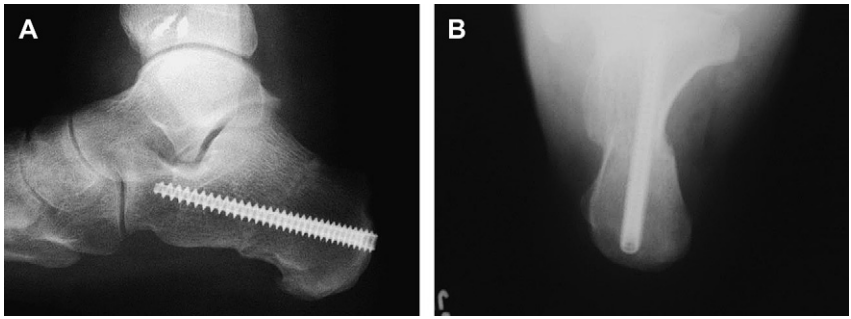


Fig. 5. (*A*, *B*) Lateral and calcaneal radiographs of an MCO.

## Lateral column lengthening

For more severe flexible flatfoot deformity with uncovering of the head of the talus, the lateral column of the foot becomes relatively shortened with respect to the medial column. The Evans procedure helps restore the medial longitudinal arch with an opening wedge osteotomy of the anterior calcaneus centered 10 mm proximal to the calcaneocuboid joint [38]. Lateral column lengthening results in loosening of the plantar fascia and may tighten the peroneus longus to increase plantar flexion of the first ray [2,38]. Lengthening of 8–12 mm is advised using a tricortical autograft or allograft wedge [38]. Some reports have shown increased calcaneo-cuboid joint pressures associated with the osteotomy and an increased risk of arthritis in the joint [38]. Distraction arthrodesis of the calcaneo-cuboid joint is another surgical option that eliminates the concern for arthritis; however, this could lead to future midfoot arthritis [38].

### *Surgical technique: lateral column lengthening (arthrodesis)*

The patient is positioned in the lateral position and a thigh tourniquet used. A longitudinal incision is made dorsally over the calcaneo-cuboid joint. The sural nerve is protected, and dissection is carried down to the extensor digitorum brevis muscle belly. The extensor digitorum brevis muscle is elevated off its proximal origin to expose the anterior process of the calcaneus and its articulation with the cuboid. A sagittal saw is used to remove the articular surface of calcaneo-cuboid joint. One pin from a cervical distractor device is placed in the anterior process of the calcaneus and the other in the cuboid. Then the distractor is used to correct the abduction under image intensification. Once the talar-first metarsal axis is realigned, measurements are taken for the graft to be fashioned on the back table. A height and depth of 18 mm allows for maximal bone fill without medial or dorsal impingement. The only variable is the length of the graft. Tricortical allograft from the anterior process of the calcaneus or the femoral neck is the made and impacted into place. With removal of the distractor, the

correction is checked and confirmed using fluoroscopy (Fig. 6). A compression staple is used for solid fixation of the lateral column. After completion, soft tissue reconstruction can be performed.

## Subtalar arthroereisis implant

For patients who have more severe flexible flatfoot deformities, lateral column support of an FDL transfer/medial displacement calcaneal osteotomy may be necessary. There has been a recent increase in interest for the subtalar arthroereisis procedure, because decreased mobility, lateral column overload, and more extensive surgical exposure can be associated with a double calcaneal osteotomy. Arthroereisis involves the placement of an implant (Fig. 7) into the sinus tarsi to correct forefoot abduction and prevents lateral column shortening. By blocking nonphysiologic eversion, the implant decreases the tendency of the talus to rotate medially and plantarly [48]. The implant prevents strain on the medial tissues during the healing phase after a flatfoot reconstruction. Once healing is completed, the role of the implant is marginal, and it may be removed if it becomes symptomatic [48]. The implant is not recommended for use as an isolated procedure [49].

While Grice [50] initially described an extra-articular arthrodesis of the subtalar joint with a sinus tarsi bone graft, Subotnick [51] first described the arthroereisis procedure using a silicone block in the sinus tarsi. The arthroereisis procedure was initially more common in the pediatric population, and it continues to be used in children who have symptomatic flatfeet. Giannini and colleagues [52] recently reported the results of the procedure in 21 children. These investigators found that in combination with other indicated procedures (heel cord lengthening, modified Kidner), 95%

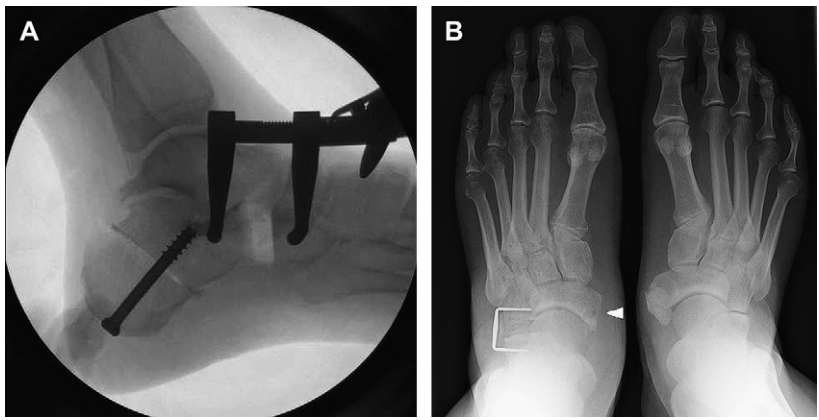


Fig. 6. (*A*) Intraoperative lateral radiograph of the distraction during an LCL procedure. (*B*) Postoperative anteroposterior radiograph after an FDL transfer, MCO, and LCL. Arrow depicts bone tunnel in the navicular.

Fig. 7. The Prostop arthroereisis device. (*Courtesy of* Arthrex, Inc., Naples, FL; with permission.)

of patients were asymptomatic at 4 years. Viladot and colleagues [53] performed arthroereisis in 21 adults in conjunction with PTT synovectomy or FDL transfer. In this study, the average AOFAS hindfoot scores improved from 47 to 82 at an average of 27 months.

Needleman [49] recently reported on 28 arthroereisis procedures combined with FDL transfer and MCO with an average 44-month follow-up. He found significant improvement of the AOFAS hindfoot score (52 preoperatively to 87 postoperatively). On a 10-point scale, the average patient satisfaction was 8.3 points, and 78% of patients said they would have the surgery again. The implant was surgically removed in 11 feet (39%) because of pain. Needleman [49] concluded that reconstructive foot and ankle surgery that included a subtalar arthroereisis resulted in favorable clinical outcomes despite the high incidence of temporary sinus tarsi pain until the implant was removed.

*Surgical technique: subtalar arthroereisis*

VanAman and Schon [48] recently described the technique of subtalar arthroereisis. The surgical decision-making guidelines are described in Table 1. The implant can be used to supplement flatfoot deformities in patients who are obese, have diabetes, use tobacco, and have noncompliance issues. Heel cord lengthening is performed when necessary.

The investigators perform the surgery as three separate procedures in the following order: MCO, subtalar arthroereisis, and FDL transfer. The overall foot deformity is assessed under anesthesia with attention given to the degree of hindfoot valgus and forefoot supination.

The goal is to correct the heel to a neutral position without causing forefoot supination. The sinus tarsi is palpated just inferior and distal to the tip of fibula and a 1 cm incision is created dorsal to the peroneal tendons over

Table 1
Indications for use of an arthroereisis implant

| Preoperative guidelines for subtalar arthroereisis implant | Criteria |
|---|---|
| Isolated FDL transfer with subtalar arthroereisis | Able to invert past midline<br>Clinical heel valgus ($<5^\circ$)<br>Minimal abduction deformity (20%–30% talonavicular uncoverage, talo-first metatarsal angle $<20^\circ$ on anteroposterior radiograph)<br>Minimal arch collapse (talo-first metatarsal angle $<10^\circ$ on lateral radiograph) |
| FDL transfer, MCO, and subtalar arthroereisis | Unable to invert past midline<br>Moderate heel valgus ($<15^\circ$)<br>Moderate abduction deformity (30%–40% talonavicular uncoverage, talo-first metatarsal angle $21^\circ$–$40^\circ$ on anteroposterior radiograph)<br>Moderate arch collapse (talo-first metatarsal angle $11^\circ$–$20^\circ$ on lateral radiograph) |

*Adapted from* VanAman S, Schon L. Subtalar arthroereisis as adjunct treatment for type II posterior tibial tendon deficiency. Techniques in Foot & Ankle Surgery 2006;5(2):117–25; with permission.

the soft tissue sulcus. Blunt dissection with a hemostat exposes the tarsal canal (Fig. 8), and routine release of the interosseous ligament is not recommended. Then a blunt guide wire is inserted from lateral to medial, recognizing that the sinus tarsi is oriented obliquely from anterolateral to posteromedial. Resistance may be felt as the guide wire penetrates the interosseus ligament. The guide wire is advanced until it tents the medial skin just dorsal to the sustentaculum tali (Fig. 9). A relaxing puncture wound is

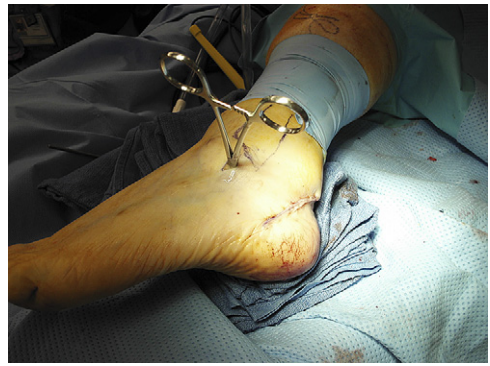

Fig. 8. Intraoperative photo depicting the incision and dissection for the arthroereisis implant.

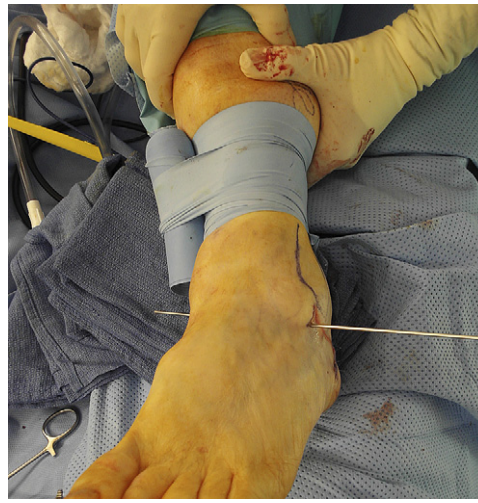

Fig. 9. Intraoperative photo depicting correct placement of the guidewire for the arthroereisis implant.

created to allow the wire to pass medially, where it is secured with a hemostat. The medial penetration point should fall within the planned FDL incision site [48].

With the guide wire in place, cannulated blunt trial sizers are advanced from lateral to medial. Under fluoroscopy, the trial implant typically crosses half of the mediolateral diameter of the talar neck and should not be proud of the lateral border. It is recommended that motion and position of the hindfoot be assessed to ensure the implant is not too small (allowing too much eversion) or too large (hindfoot overcorrected into varus). Then the implant is inserted over the guide wire until it is flush with the lateral border of the talar neck (Fig. 10) [48].

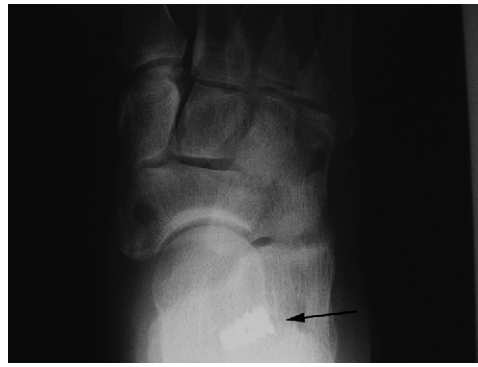

Fig. 10. Postoperative anteroposterior radiograph showing the arthroereisis implant (*arrow*) with optimal placement.

## Summary

The adult flexible flatfoot is commonly the result of PTTD, which ultimately leads to attenuation of the static stabilizers of the medial longitudinal arch. Recent advances in physical therapy regimens and lightweight bracing have improved nonoperative treatment. Past surgical approaches included selected hindfoot and midfoot fusions; however, more recent surgical procedures focus on transfer of the FDL combined with an MCO. The subtalar arthroereisis implant has emerged as a potentially beneficial treatment option to aide the correction of hindfoot valgus.

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