The management of posterior tibial tendon dysfunction (PTTD), also referred to as adult-acquired flatfoot deformity, has evolved substantially since it was first recognized as a distinct entity in the 1950s.1,2 The management of this complex disorder is controversial, and different philosophic approaches to treatment are advocated by leaders in the field.

Historically, surgical management was reserved for the later stages of the disease and focused on definitive deformity correction with triple arthrodesis to establish a stable and plantigrade foot. Adjacent joint arthrosis of the midfoot and the ankle tended to develop in such rigidly corrected feet.3-5 Surgeons now focus on recognizing and diagnosing this disease at an early stage and use both nonsurgical and surgical techniques to maximize foot function and preserve range of motion. This can be accomplished by using joint-sparing surgical procedures, such as tendon transfers, calcaneal and midfoot osteotomies, and ligament reconstructions. This chapter focuses on the current concepts and treatments used in the management of PTTD in its earlier stages.

Abstract

The management of posterior tibial tendon dysfunction in adults has evolved substantially, and controversy persists regarding a specific recommended algorithm for treatment. The current focus is on early diagnosis and treatment of this disorder with joint-sparing surgeries, such as corrective osteotomies and tendon transfers, when nonsurgical modalities have been exhausted. It is helpful to be familiar with the pertinent pathophysiology and diagnostic pearls associated with posterior tibial tendon dysfunction, its treatment options, pertinent literature, and technique tips for the procedures currently being used.

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The dysfunction of the posterior tibial tendon that precipitates the deformity is likely multifactorial. Believed to be an overuse and age-related phenomenon, posterior tibial tendon degeneration typically occurs in the areas of maximal anatomic stress. Vascular studies of the posterior tibial tendon also have demonstrated that the retromalleolar region is a relatively hypovascular zone. In this region, the tendon experiences the most mechanical stress between the motion segments of the ankle and hindfoot complex.

After a tendon injury has occurred, an inflammatory cascade is initiated that increases the local metalloproteinase activity and leads to further tendon injury.

Congenital flatfoot is often asymptomatic; however, greater stress, which is likely the result of medial column deficiency and increased subtalar motion, may result in increased stress on the posterior tibial tendon as patients age. This structural predisposition may lead to a propensity for long-term tendon degeneration and dysfunction.

Medical comorbidities, including obesity, systemic steroid use, diabetes mellitus, fluoroquinolone use, and inflammatory conditions such as rheumatoid arthritis and seronegative spondyloarthropathies also can contribute to tendon rupture or dysfunction. In some cases, progressive adult planovalgus deformity can be caused primarily by a failure of the talonavicular capsuloligamentous complex rather than the posterior tibial tendon.

**Diagnosis and Physical Examination**

The physical examination begins by observing a patient with suspected PTTD. With the patient standing, the position of the affected extremity is evaluated from multiple angles to assess overall limb alignment, the height of the midfoot arch, the degree of midfoot abduction, and heel alignment. Medial talar head prominence and the presence of any skin changes on the plantar aspect of the foot also should be noted. While viewing both extremities from the front, the examiner should look for pathologic midfoot abduction, which is seen with the “too many toes sign,” and also for the degree of heel valgus (Figure 1).

To assess the maintenance of the medial arch during clinical examination, this chapter’s authors have developed and use a simple manual method (“fingometer” method) that is shown in Figure 2. If there is a loss of medial arch height but the hindfoot alignment remains neutral or physiologic, then the cause may be advanced midfoot arthritis and collapse, not PTTD. The patient should then be observed during dynamic gait to allow the examiner to assess the cadence of his or her gait, the condition of the arch of the foot during stance, and the presence or the absence of heel inversion during toe-off.

With the patient standing, the traditional single- and double-stance heel rise tests are performed to assess posterior tibial tendon function. The examiner should observe the ability to invert the hindfoot with a single-heel rise and compare this with the contralateral hindfoot during a double-heel rise. Difficulty or pain with multiple repetitions may be a sign of early tendon pathology. Other foot and ankle pathology, such as Achilles tendon dysfunction and painful midfoot arthritis, can lead to false-positive results with this test.

With the patient seated, the examiner begins palpating the pertinent anatomic areas. The length of the posterior tibial tendon is palpated, noting areas of tenderness, swelling, warmth, and any palpable defects in continuity. The lateral hindfoot, the sinus tarsi, and the distal fibula are palpated because tenderness in these areas can develop in more severe and chronic cases as a result of subfibular impingement.

Next, passive range of motion of the affected ankle and hindfoot should be assessed and compared with the contralateral side. Hindfoot motion can be
measured as the angle defined by the position of the second metatarsal shaft in relation to the long axis of the tibial shaft. The foot should be brought from a fully abducted and everted position to the fully inverted and adducted position. A decreased arc of hindfoot motion can indicate advanced osteoarthritis, which may preclude treatment with joint-sparing procedures. Passive ankle motion is assessed with the knee extended and flexed to 90°, and the Silfverskiöld test is used to determine the degree of gastrocnemius muscle contracture. This test specifically measures gastrocnemius contracture by alternately relaxing and incorporating the muscle because of its origin proximal to the knee joint; maximal ankle dorsiflexion is assessed in each state. Assuming that the hindfoot valgus is flexible, it is important to hold the heel in a corrected neutral position to accurately gauge the degree of equinus contracture. It also is important to check for fixed forefoot varus deformity during this portion of the examination.

The final component of the examination should focus on posterior tibial tendon power and motor function. The foot is positioned passively in a maximally plantarflexed and inverted position to isolate the tendon. The patient should be asked to hold this position when the foot is released while keeping the toes relaxed. If there is an inversion lag or the patient cannot maintain this position against resistance, then there may be attenuation or stretching of the tendon. Tendon power should then be further assessed along its entire range of excursion by testing against resistance at the neutral position and the fully everted position. For clinical grading, 5/5 is defined by full power against the entire range of passive tendon excursion, and 5−/5 is for less than full power compared with the unaffected side. With the presence of an inversion lag, the power is defined as 4/5, which is further subclassified as 4−, 4, or 4+ depending on tendon power. The ability to maintain inversion 20° beyond the midline against resistance is graded as 4+, and the inability to maintain inversion beyond the midline is graded as 4−. By carefully assessing and documenting these examination findings on serial office visits, interval improvement or deterioration can be followed.

**Imaging**

A standard series of standing AP, lateral, and oblique radiographs of the foot as well as AP and mortise views of the ankle are useful in evaluating a patient with symptomatic flatfoot deformity. For comparison, additional radiographs of the contralateral side should be obtained in patients with congenital flatfoot. If there is any proximal deformity of the hip or the knee, full-length standing radiographs should be obtained. Measurements of the foot are taken in both the AP and lateral planes to quantify the findings associated with this deformity. On the lateral radiograph, the important parameters are the calcaneal pitch, the lateral talocalcaneal angle, the lateral talometatarsal angle, and the medial cuneiform height (Figure 3). On the AP radiograph, the important parameters are the talometatarsal angle and the percentage of talar head uncoverage (Figure 4). The techniques for performing these measurements can vary greatly, and no standard has yet been established.

It is common for patients to bring previous imaging studies, such as MRIs and ultrasound studies, when presenting for examination. Although these studies can provide additional information regarding pathology in the soft tissues and joints, the findings do not usually dictate the treatment approach. Consequently, the routine use of MRI for PTTD is not necessary unless there
is concern for additional pathology that may potentially alter the surgical plan, such as possible attenuation of the deltoid ligament or peroneal tendon involvement. Standing clinical photographs are routinely obtained, and the patient may be videotaped while walking to document the degree of clinical severity.

**Classification**

PTTD is best understood as a syndrome with a spectrum of clinical presentations ranging from recent onset tendinitis with minimal deformity to long-standing tendon dysfunction with advanced deformity. In 1989, Johnson and Strom21 proposed a general classification system for PTTD that is based on clinical and radiographic findings.

Patients with stage I PTTD have swelling and pain along the posterior tibial tendon and do not have progressive planovalgus deformity. The tendon pathology is usually restricted to tenosynovitis, without attenuation or elongation of the tendon. With regard to the function of the posterior tibial tendon, these patients have no inversion lag and are able to perform an isolated single-stance heel rise. However, repetitive stresses may reveal slight weakness compared with the contralateral side.

Posterior tibial tendon degeneration with elongation has begun to develop in patients with stage II PTTD. These patients also exhibit varying degrees of flexible planovalgus deformity. Early in this stage, the patient may be able to perform a single-stance heel rise (although painful), but may soon lose the ability to do so. Several authors have subclassified this stage with the severity of the deformity and the presence of lateral subfibular pain, which are used as the parameters for these additional stages.22,23 For use in their practice, this chapter’s authors have informally subclassified stage II PTTD into three types based on the degree of talonavicular uncoverage and resultant abduction deformity. Types IIa and IIb are defined by less than 40% uncoverage and between 40% to 50% uncoverage, respectively.

In the treatment algorithm (Table 1), these subgroups usually can be managed with a joint-sparing procedure, such as a lateral column lengthening. Type IIC PTTD is defined as uncoverage of greater than 50%. In these patients, fusion procedures may be indicated.

In stage III PTTD, a rigid planovalgus deformity has developed. On clinical examination, these patients demonstrate severe hindfoot valgus and midfoot valgus with the too many toes sign and frequently a fixed forefoot varus deformity. There is substantial lateral pain associated with subfibular impingement. Because of the rigidity of the deformity at this stage, fusion is frequently required.

Patients with stage IV PTTD have chronic valgus loading of the ankle joint, with subsequent arthritic changes and joint erosion.24 These patients have lateral pain partially from subfibular impingement and lateral ankle joint arthritis. As the severity of the ankle deformity worsens, the deltoid ligament becomes attenuated and can rupture. Treatment of this stage will include reconstructive surgery for the foot deformity as well as the ankle deformity.

**Treatment**

**Stage I PTTD**

The mainstay of treatment of patients with stage I PTTD is nonsurgical management. This includes bracing and/
or orthotics, activity modification, and physical therapy after symptoms have largely resolved. This chapter’s authors typically begin with a period of bracing for 6 weeks to allow for diminished inflammation and improved symptomatology. The specific bracing used depends on the severity of symptoms, with a tall, controlled ankle motion (CAM) walking boot used for the most severely symptomatic patients and an off-the-shelf PTTD brace for patients with more moderate symptoms. This brace was designed to limit eversion while also incorporating a support strap that lifts the medial arch and an adjustable pneumatic bladder for additional arch support. Frequently, patients are started with the CAM boot initially and then graduated to the brace as their symptoms allow. Other braces also can be used and have been described in the literature, including standard lace-up braces, custom ankle foot orthoses, and Arizona-type braces.25,26 Shoe modification and orthotics provide adequate support and relief only in mildly symptomatic cases with minimal tendinopathy.27 Routine oral anti-inflammatory medications can be used; injectable corticosteroids are not commonly used because these have been shown to lead to further attenuation and potential posterior tibial tendon rupture.20,21

After a patient’s symptoms have resolved, formal physical therapy can be started to focus on strengthening the posterior tibial tendon and the remaining musculature of the posterior compartments. In a patient with a tight heel cord, it also is important to focus on stretching the gastrocnemius-soleus complex. Several studies have shown that these therapeutic strengthening and stretching regimens in conjunction with orthotic treatment achieve clinical benefit.28-31

Despite prolonged nonsurgical treatment with bracing and therapy, some patients remain symptomatic. In these refractory cases, alternative modalities of treatment can be considered, including shock-wave therapy or bone marrow aspirate concentrate injections. In vitro studies have shown that these modalities have beneficial effects on tenocytes.16,32,33 It is important to note that after a bone marrow aspirate concentrate injection, there is a period of increased pain, so this chapter’s authors routinely recommend 4 to 6 weeks of postinjection bracing for these patients.

For stage I PTTD, some authors have described isolated tenosynovectomy for refractory cases, but only moderate success has been reported.34,35 Although there can be success with an isolated tendon procedure, an adjunctive alignment procedure, such as arthroereisis and/or osteotomies, is more likely to optimize the chances of a positive outcome.

**Stage II PTTD**

The management of stage II begins with nonsurgical treatment that
focuses on bracing and the therapy modalities as previously outlined for stage I. After these nonsurgical modalities have been exhausted without clinical improvement, the focus of treatment shifts to surgical management. The determination of the appropriate surgical treatment is predicated on the clinical and radiographic examination as well as patient-specific medical and functional factors. The treatment algorithm used in the institution of this chapter’s authors reflects the various procedures available to correct the broad spectrum of deformity encountered.

This algorithm does not determine the final treatment plan. Patient-specific issues, such as medical comorbidities (for example, obesity, diabetes, and steroid use) and intraoperative findings will further guide surgical treatment.

For example, a residual intraoperative equinus contracture may necessitate an adjunctive Strayer procedure for contractures of less than 15° or Achilles tendon lengthening for more severe contractures.

### Surgical Procedures

**Medial Displacement Calcaneal Osteotomy**

The medial displacement calcaneal osteotomy (MDCO) is the cornerstone procedure for reconstructing a flexible flatfoot. It shifts the mechanical pull of the Achilles tendon medially and the weight-bearing axis of the heel closer to the long axis of the tibia. These biomechanical advantages lead to improved inversion power and restoration of the medial longitudinal arch. This chapter’s authors obtain approximately 1 cm of medial translation, and fixation is achieved with one or two 6.5- or 7.3-mm cannulated, partially threaded cancellous screws. A “crushplasty” of the prominent lateral cortex is performed to optimize the lateral contour and also improve bone apposition (Figure 5).

**Flexor Digitorum Longus Tendon Transfer**

After posterior tibial tendon incompetence is demonstrated, the flexor digitorum longus (FDL) remains the optimal choice for augmentation or replacement. The FDL and the posterior tibial tendon are in-phase muscles with similar lines of pull, and the FDL can match the strength of the peroneus brevis. The transfer is rarely performed as an isolated procedure and is usually accompanied by an MDCO as well as additional corrective osteotomies. This chapter’s authors prefer to use an interference screw for FDL fixation within the navicular bone tunnel, which allows for harvesting the tendon more proximally and minimizes the need for additional dissection. The free suture ends of the whipstitch also are secured over the bone as a bridge to the posterior tibial tendon insertion to provide an additional point of fixation. The transfer is tensioned before fixation to obtain approximately 15° of plantar flexion and 15° of inversion. The posterior tibial tendon is not routinely excised if it appears salvageable, but it is instead débrided of any degenerative-appearing tendon, and the FDL transfer is used for augmentation rather than replacement (Figure 6). Outcome studies of FDL transfer with a medial displacement

### Table 1

<table>
<thead>
<tr>
<th>Procedures</th>
<th>AP Radiograph</th>
<th>Lateral Radiograph</th>
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<tr>
<td></td>
<td>TN Uncoverage (%)</td>
<td>AP T1MTA</td>
<td>Lateral T1MTA</td>
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<tr>
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<td>&lt;15°</td>
<td>&lt;10°</td>
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<td>FDL transfer and LCL</td>
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<td>&lt;50°</td>
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<tr>
<td>FDL transfer, MDCO, and lateral column lengthening</td>
<td>&gt;50%</td>
<td>&gt;50°</td>
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TN = talonavicular, T1MTA = talus-first metatarsal angle, FDL = flexor digitorum longus, MDCO = medial displacement calcaneal osteotomy.
calcaneal osteotomy have demonstrated good results, with mean maximal improvement achieved between 10 and 14 months.\(^\text{36,37}\)

**Lateral Column Lengthening**
Initially described by Evans\(^\text{38}\) in 1975 and later modified by Mosca\(^\text{39}\) in 1995, lateral column lengthening has remained a popular tool in the treatment of flexible flatfoot deformity.\(^\text{38-40}\) It is used either as the primary corrective osteotomy or combined with other osteotomies. Several adverse effects have been described with this procedure, including lateral column overload and restriction of subtalar range of motion.\(^\text{41,42}\) In addition to the traditional Evans osteotomy, other osteotomies, including stepcut and Hintermann osteotomies, have been described to achieve lateral column lengthening.\(^\text{43,44}\)

Lateral column lengthening procedures are typically reserved for severe forefoot abduction with talonavicular uncoverage of 40% to 50%. This osteotomy is performed only if the MDCO alone does not provide adequate correction. If lateral column lengthening is performed, Hintermann’s modification is used, with the osteotomy performed along the sinus tarsi, between the posterior and middle facets (Figure 7). In the experience of this chapter’s authors, the modified lateral column lengthening osteotomy achieves better graft incorporation, less restriction of subtalar motion, and no calcaneocuboid subluxation compared with Evans’ more distal osteotomy. A biplanar femoral neck allograft soaked in bone marrow aspirate concentrate is used. Internal fixation is not generally used (Figure 8). Clinical and radiographic studies (CJ Humbyrd, MD, et al, San Diego, CA, unpublished data presented at the American Orthopaedic Foot and Ankle Society annual meeting, 2012) have reported good results with this procedure.\(^\text{46}\)

**Cotton Osteotomy**
If residual fixed forefoot varus remains after the other corrective procedures, a dorsal opening wedge osteotomy of the medial cuneiform, also known as a Cotton osteotomy, can be performed.\(^\text{45}\) The medial cuneiform is exposed with a dorsomedial approach. The extensor hallucis longus and the dorsal neurovascular bundle are retracted and protected, and a transverse osteotomy is performed without violating the plantar cortex. An approximately 7-mm wedge-shaped graft is prepared from a femoral head allograft that is soaked in bone marrow aspirate concentrate. Internal fixation is not generally used (Figure 8). Clinical and radiographic studies (CJ Humbyrd, MD, et al, San Diego, CA, unpublished data presented at the American Orthopaedic Foot and Ankle Society annual meeting, 2012) have reported good results with this procedure.\(^\text{46}\)

**Arthroereisis**
The role of arthroereisis in the management of the adult flexible flatfoot is controversial. This chapter’s authors use this procedure in conjunction with an MDCO and FDL transfer for patients with more severe deformity; obesity; or

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**Figure 5** Intraoperative photograph (A) of a medial displacement calcaneal osteotomy after lateral ridge crushplasty, with additional fluoroscopic images demonstrating axial (B) and lateral (C) views after medial translation and fixation. The calcaneal osteotomy is slid approximately 1 cm for severe valgus and fixed with 6.5- or 7.3-mm screws.

**Figure 6** Intraoperative photograph of the flexor digitorum longus transferred into the navicular tunnel after interference screw fixation. Note the preservation of the posterior tibial tendon.
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The addition of an arthroereisis to an MDCO and FDL transfer has been shown to provide improved correction in a cadaver flatfoot model.47

Arthroereisis (Figure 9) is performed under fluoroscopic guidance with a small incision centered over the sinus tarsi. A guidewire is placed across the interval between the posterior and middle facets of the talus, and the appropriately sized implant is inserted across the lateral half of the talar neck. The implant is designed to limit pathologic eversion and block the talus from rotating plantarly and medially. Preoperatively, contralateral heel valgus is assessed and an attempt is made to replicate this degree of valgus after final implant insertion on the affected extremity. Clinical studies have thus far demonstrated promising results at midterm follow-up, but persistent lateral pain has been described; as a result, the implant sometimes must be removed.48,49 If the implant needs to be removed, the correction is often maintained because of the concomitant procedures.

Stages III and IV PTTD
In the later stages of PTTD, a rigid deformity of the hindfoot complex exists; thus, joint salvage procedures are no longer indicated. Surgical treatment of stages III and IV usually involves a triple arthrodesis. When planning and performing these fusion procedures, it is critical to ensure that the appropriate final alignment is achieved. This should be tailored to the patient, taking into consideration the patient’s contralateral hindfoot position as well as the specific characteristics of his or her gait. If no arthrosis is present, a
double-hindfoot arthrodesis with sparing of the calcaneocuboid joint may be attempted. This spared motion segment may prevent progressive degeneration of the four to five metatarsocuboid articulations. Thus far, this has led to good results without substantial compromise to union rates. The presence of valgus ankle arthritis in patients with stage IV PTTD also must be addressed with a corrective supramalleolar osteotomy, an ankle arthroplasty, or arthrodesis.

Summary
The treatment of PTTD is complex and controversial. As the focus in surgical management has shifted to earlier intervention and joint-sparing reconstructions, ongoing debate has ensued regarding the optimal approach and the specific procedures used. A comprehensive trial of nonsurgical treatment should always be used initially. When the patient’s level of dysfunction and/or pain does not improve satisfactorily with nonsurgical treatment, then the discussion should shift to selecting an appropriate surgical plan. Despite differing opinions, there is growing consensus in the need to achieve the goals of preserving motion, reestablishing alignment, and ultimately restoring patient function.

References