



Complete Summary

GUIDELINE TITLE

Diagnosis and treatment of adult flatfoot.

BIBLIOGRAPHIC SOURCE(S)

Lee MS, Vanore JV, Thomas JL, Catanzariti AR, Kogler G, Kravitz SR, Miller SJ, Gassen SC. Diagnosis and treatment of adult flatfoot. J Foot Ankle Surg 2005 Mar-Apr;44(2):78-113. [223 references] [PubMed](#)

GUIDELINE STATUS

This is the current release of the guideline.

COMPLETE SUMMARY CONTENT

SCOPE
METHODOLOGY - including Rating Scheme and Cost Analysis
RECOMMENDATIONS
EVIDENCE SUPPORTING THE RECOMMENDATIONS
BENEFITS/HARMS OF IMPLEMENTING THE GUIDELINE RECOMMENDATIONS
IMPLEMENTATION OF THE GUIDELINE
INSTITUTE OF MEDICINE (IOM) NATIONAL HEALTHCARE QUALITY REPORT
CATEGORIES
IDENTIFYING INFORMATION AND AVAILABILITY
DISCLAIMER

SCOPE

DISEASE/CONDITION(S)

Adult flatfoot, including:

- Flexible flatfoot (non-posterior tibial tendon dysfunction [non-PTTD])
- Posterior tibial tendon dysfunction (adult acquired flatfoot)
- Tarsal coalition
- Iatrogenic, post-traumatic, or arthritic deformity
- Charcot foot (acute, chronic: stable foot, unstable foot)
- Neuromuscular flatfoot

GUIDELINE CATEGORY

Diagnosis
Treatment

CLINICAL SPECIALTY

Family Practice
Orthopedic Surgery
Podiatry

INTENDED USERS

Physicians
Podiatrists

GUIDELINE OBJECTIVE(S)

To provide recommendations for the diagnosis and treatment of adult flatfoot

TARGET POPULATION

Adults with known or suspected flatfoot

INTERVENTIONS AND PRACTICES CONSIDERED

Diagnosis

1. Comprehensive history and review of symptoms
2. Range of motion evaluation
 - Flexibility testing using the Hubscher maneuver (Jack test)
 - Manual muscle testing
 - Single heel-rise test
 - Double heel-rise test
3. Gait observation
4. Radiographs (dorsoplantar, lateral, oblique views)
5. Optional ancillary studies
 - Magnetic resonance imaging (MRI)
 - Computed tomography
 - Bone scan
 - Gait analysis via video or computer studies
 - Nerve conduction velocities/electromyography studies
 - Ink print mats and Harris mats
 - Computerized force plate analysis
 - Local anesthetic diagnostic injections
 - Radiographs with the foot in the neutral position

Treatment

Flexible Flatfoot (Non-Posterior Tibial Tendon Dysfunction [Non-PTTD])

1. Activity modifications
2. Weight loss
3. Orthoses
 - Foot orthoses
 - Ankle-foot orthoses (AFO)

4. Immobilization
5. Antiinflammatory medications
6. Physical therapy
7. Foot wear modifications
8. Surgical management
 - Osteotomy
 - Arthroereisis
 - Fusions
9. Adjunctive soft tissue procedures
 - Soft tissue procedures
 - Tendo-Achillis lengthening (TAL)/Gastrocnemius recession

Posterior Tibial Tendon Dysfunction (PTTD)

1. Patient education
2. Orthoses
3. Immobilization
4. Antiinflammatory medications
5. Physical therapy
6. Shoe modification
7. Surgical management
 - Synovectomy
 - Tendon transfer
 - Osteotomies
 - Arthroereisis
 - Tendo-Achillis lengthening/Gastrocnemius recession
 - Isolated rearfoot fusions
 - Medial column fusion
 - Triple arthrodesis
 - Pantalar arthrodesis
 - Deltoid repair
 - Total ankle replacement
 - Supramalleolar osteotomy

Tarsal Coalition

1. Footwear modifications
 - Arch supports
 - Orthoses
2. Activity modifications
3. Appropriate weight reduction
4. Immobilization
5. Antiinflammatory medications
6. Surgical management
 - Resection of coalition
 - Arthrodesis
 - Realignment osteotomy

Iatrogenic/Post-traumatic/Arthritic Flatfoot

1. Activity modifications
2. Weight loss

3. Orthoses
4. Immobilization
5. Antiinflammatory medications
6. Physical therapy
7. Shoe gear modifications
8. Surgical management
 - Osteotomies
 - Arthrodesis
9. Adjunctive procedures
 - Tendo-Achillis lengthening or gastrocnemius recession
 - Tendon transfer

Acute Charcot Foot

1. Absolute restriction on weight-bearing (crutches, wheelchair)
2. Immobilization with splint, cast, or removable cast
3. Pharmacologic therapy
4. Bone stimulation

Chronic Charcot Foot (Foot Stable)

1. Bracing
2. Extra depth shoes
3. Custom moulded shoes
4. Multiple density insoles
5. Orthoses

Chronic Charcot Foot (Foot Unstable)

1. Supportive measures
2. Therapeutic footwear
3. Patient education
4. Periodic evaluation to prevent recurrence

Adult Neuromuscular Flatfoot

1. Activity modifications
2. Assistive devices
3. Bracing
4. Physical therapy
5. Orthoses
6. Shoe gear modifications
7. Medications
8. Surgical management
 - Tendon transfers
 - Realignment osteotomies
 - Arthrodesis

MAJOR OUTCOMES CONSIDERED

Clinical response to treatment

METHODOLOGY

METHODS USED TO COLLECT/SELECT EVIDENCE

Searches of Electronic Databases

DESCRIPTION OF METHODS USED TO COLLECT/SELECT THE EVIDENCE

Not stated

NUMBER OF SOURCE DOCUMENTS

Not stated

METHODS USED TO ASSESS THE QUALITY AND STRENGTH OF THE EVIDENCE

Expert Consensus

RATING SCHEME FOR THE STRENGTH OF THE EVIDENCE

Not applicable

METHODS USED TO ANALYZE THE EVIDENCE

Review

DESCRIPTION OF THE METHODS USED TO ANALYZE THE EVIDENCE

Not stated

METHODS USED TO FORMULATE THE RECOMMENDATIONS

Expert Consensus

DESCRIPTION OF METHODS USED TO FORMULATE THE RECOMMENDATIONS

Not stated

RATING SCHEME FOR THE STRENGTH OF THE RECOMMENDATIONS

Not applicable

COST ANALYSIS

A formal cost analysis was not performed and published cost analyses were not reviewed.

METHOD OF GUIDELINE VALIDATION

Internal Peer Review

DESCRIPTION OF METHOD OF GUIDELINE VALIDATION

Not stated

RECOMMENDATIONS

MAJOR RECOMMENDATIONS

The recommendations for the diagnosis and treatment of heel pain are presented in the form of an algorithm with accompanying annotations. Algorithms are provided for [Adult Flatfoot](#); [Adult Flexible Flatfoot \(Non-Posterior Tibial Tendon Dysfunction \[PTTD\] Flexible Flatfoot\)](#); [Posterior Tibial Tendon Dysfunction \(Adult Acquired Flatfoot\)](#); [Tarsal Coalition](#); [Iatrogenic/Post-traumatic/Arthritic Adult Flatfoot](#); [Charcot Foot](#); and [Adult Neuromuscular Flatfoot](#). Annotations follow.

Significant History (Pathway 1, Node 1)

The natural history of the adult flatfoot has not been clearly defined, as evidenced by the absence of reliable studies analyzing the long-term sequelae of this condition. The deformity may be associated with pain, instability, and severe functional limitations or it may be of little clinical significance.

The evaluation of adult flatfoot requires a pertinent patient history that includes onset of the deformity, timing of symptoms, and severity of past and current symptoms (with particular regard to arch and rearfoot pain). A family history of flatfoot deformity may be elicited. Associated conditions such as rheumatoid arthritis, seronegative arthropathies, hypertension, or diabetes may be significant in the patient with adult flatfoot. Occupation, activity level, and obesity may also be contributory factors. Footwear, history of trauma, and previous treatment are significant. A pertinent review of systems should be performed. Extrapodal findings, such as knee, hip, or back pain, have also been associated with concurrent flatfoot.

Significant History (Pathway 1, Node 2)

Flatfoot versus a normal foot (refer to figure 1 of the original guideline document) is readily apparent with clinical evaluation. The appearance of the foot, both on and off weight-bearing, will help define its deformed or compensated condition. Physical examination reveals 1 or more of the following characteristics: depression of the medial longitudinal arch, everted or valgus heel in relaxed stance, and abduction of the forefoot relative to the rearfoot (refer to figures 2 and 3 of the original guideline document). Areas of tenderness may be localized with careful palpation of the foot, ankle, and leg; particular attention should be paid to the posterior tibial tendon, lateral rearfoot, and plantar fascia.

Range of motion evaluation differentiates the flexible from rigid flatfoot and identifies the degree of abnormal motion that may be present. Flexibility can also

be assessed by using the Hubscher maneuver (Jack test) to determine if the deformity is reducible. Manual muscle testing and the single heel-rise test assess muscle strength and tendon function. Additionally, the double heel-rise test determines reducibility of rearfoot valgus (see figures 1 and 3 of the original guideline document).

Gait observation may show an increased angle of gait, delayed or absent supination of the foot, or decreased propulsion. Footwear patterns also can provide valuable information. Extrapedal manifestations may include genu valgum, shin splints, short tendo-Achilles with calf tenderness, peroneal muscle spasm, medial knee tenderness, leg length discrepancy, and torsional problems. Frontal, sagittal, and transverse plane changes can be assessed in regard to deformity as well as compensation.

Radiographic Findings (Pathway 1, Node 3)

Radiographic evaluation of the adult foot in the angle and base of gait allows assessment of the degree of deformity. Routine radiographs may include standing anterior-posterior (AP) (dorsoplantar), lateral, and oblique views, as well as Harris-Beath views if a tarsal coalition is suspected. Consideration should also be given to ankle radiographs if ankle valgus is a concern. A number of radiographic criteria are used in the assessment of the foot structure (see figure 4 of the original guideline document):

- Calcaneal pitch is decreased in a flatfoot deformity and may approach 0 degrees or become less than 0 degrees in the presence of a rocker-bottom deformity.
- The degree of talar head coverage by the navicular and an increased calcaneocuboid abduction angle are useful parameters to assess the degree of pronation and abduction of the forefoot on the rearfoot.
- The *cyma* line is the radiographic visualization of the talonavicular and calcaneocuboid joints on the lateral radiograph. The joints should be visualized as a continuum, whereas in a pronated foot type, the talonavicular joint space is positioned further anterior than the calcaneocuboid joint space.
- A talocalcaneal angle is formed by the long axis of the rearfoot and the midtalar line. This angle is increased in pronated feet on both the AP and lateral views.
- The talar first metatarsal angle, measured on both AP and lateral views, increases with the degree of pronation.
- Findings consistent with degenerative arthritis of 1 or more joints are significant and may be secondary to a longstanding flatfoot deformity or may represent a primary cause of the flatfoot.
- The hindfoot alignment view provides coronal plane evaluation of the hindfoot in relation to the distal tibia.
- Radiographs may show a tarsal coalition.

Optional Ancillary Studies (Pathway 1, Node 4)

Although not routinely indicated for evaluation of adult flatfoot, the following tests can provide additional information that may better define the condition and aid in treatment selection:

- Magnetic resonance imaging (MRI)
- Computed tomography
- Bone scan
- Gait analysis via video or computer studies
- Nerve conduction velocities/electromyography studies
- Ink print mats and Harris mats
- Computerized force plate analysis
- Local anesthetic diagnostic injections
- Radiographs with the foot in the neutral position

Diagnosis (Pathway 1, Node 5)

Information obtained from the initial evaluation and diagnostic tests is correlated into a diagnosis. The differential diagnosis of the adult flatfoot includes the following: adult flexible flatfoot (non-posterior tibial tendon dysfunction [non-PTTD]) (Pathway 2); PTTD (Pathway 3); tarsal coalition (Pathway 4); arthritic, posttraumatic, or iatrogenic deformity (Pathway 5); Charcot foot (Pathway 6); and neuromuscular flatfoot (Pathway 7).

Adult Flexible Flatfoot (Non-PTTD) (Pathway 2)

Adult flexible (non-PTTD) flatfoot is generally a progression of a pediatric condition characterized by partial or complete loss of the medial arch. There are many terms used to describe the flexible flatfoot. The designation of flexible refers to the general qualitative stiffness properties of the foot evident during dynamic loading and/or physical examination (i.e., flexible vs. rigid). Flexible flatfoot in the adult may present as unilateral or, more commonly, as bilateral. It is frequently associated with a short or contracted Achilles muscle-tendon complex. In its late stages of progression, degenerative arthritis may occur, leading to loss of flexibility or ankylosis. Additionally, peroneal spasm may result with rearfoot arthritis.

Abnormal pronation of the rearfoot during weightbearing has been associated with collapse of the longitudinal arch in the adult flexible flatfoot. The talus adducts and plantar-flexes on the calcaneus, which simultaneously everts and plantar-flexes. Subtalar joint pronation unlocks the midtarsal joint, making it unstable and leading to various degrees of transverse plane abduction. The tarsometatarsal and other joints may also be affected. Causative factors of pronation include the following: compensated forefoot varus, compensated flexible forefoot valgus, equinus, congenital talipes calcaneovalgus, torsional abnormalities of adduction or abduction, muscle imbalance, ligamentous laxity, neurotrophic feet, and anything that contributes to a medial shift in weightbearing (e.g., genu valgum, obesity, wide base of gait).

Significant History (Pathway 2, Node 1)

Patients with adult flexible flatfoot may present with postural symptoms as well as weakness and fatigue in the foot or leg. Flexible flatfoot in the adult may manifest as a bilateral (more common) or unilateral condition with an onset of symptoms later in life and not attributable to PTTD. Arch, heel, and lateral foot pain may be primary complaints, with symptoms exacerbated by weightbearing activities (e.g., running, walking, hiking).

For further discussion, see Pathway 1.

Significant Findings (Pathway 2, Node 2)

By definition, the adult flexible flatfoot is supple, although it is not always completely reducible in its later stages. The heel may assume a valgus position during weightbearing. Areas tender to palpation might include the sinus tarsi, talonavicular joint, plantar arch and heel, posterior tibial tendon, anterior tibial tendon, and anterior or posterior tibia. Significant rearfoot eversion may result in subfibular impingement pain. Equinus is often present, and evaluation of the gastrosoleal complex is warranted. Clinical maneuvers as described in Pathway 1 may be performed to assess flexibility.

For further discussion, see Pathway 1.

Radiographic Findings (Pathway 2, Node 3)

Radiographic findings consistent with flatfoot have been described above in Pathway 1. These findings include various angular changes of the foot, peritalar subluxation, and degenerative arthritis in more advanced stages (see figure 4 of the original guideline document).

Initial Treatment (Pathway 2, Node 7)

For asymptomatic flatfoot, treatment entails patient education, discussion of the prognosis, and observation of the condition (Node 5).

In the symptomatic flatfoot (Node 6), nonsurgical therapy is directed at resisting the deformity and limiting uncontrolled pronatory compensation. Correction of the deformities should not be anticipated with this line of treatment. Initial treatment options for adult flexible flatfoot include one or more of the following: activity modifications, weight loss, orthotic management, immobilization, and footwear modifications. Antiinflammatory medications and physical therapy may also be beneficial. Orthotic management (Node 8) encompasses a broad spectrum of devices that includes foot orthoses or ankle-foot orthoses (AFO), either prefabricated or custom molded (see figure 5 in the original guideline document).

Clinical Response (Pathway 2, Node 9)

Nonsurgical therapy should be continued in patients who experience a favorable clinical response (Node 10). Surgical management should be considered if nonsurgical options fail to provide adequate relief from pain, if there is progression of deformity or instability, or if there is failure to return to acceptable function.

Surgical Intervention (Pathway 2, Node 11)

Various surgical techniques have been described for the treatment of adult flexible flatfoot. These may include osteotomy, arthroereisis, arthrodesis, and adjunctive soft tissue procedures (see table 1 of the original guideline document). Procedure

selection should be based on clinical and radiographic findings, degree of arthritis, patient age, and activity level.

Soft Tissue Procedures. For the correction of symptomatic flexible flatfeet with minimal deformity, soft tissue procedures can be considered. These procedures may include the Kidner posterior tibial tendon advancement, the flexor digitorum longus tendon transfer, the Young tenosuspension, reconstruction of the spring ligament, and the medial arch reconstruction combination.

Osteotomy. Various osteotomies for correction of the adult flatfoot have been recommended and studied. The main advantage of periarticular osteotomies is preservation of joint function while improving structural alignment.

The Evans calcaneal osteotomy was developed to lengthen the lateral column and realign the midtarsal joint by reducing forefoot abduction (see figure 6 in the original guideline document). This procedure has also been shown to plantarflex the first metatarsal and reduce talocalcaneal subluxation. Cadaver studies have demonstrated increased pressures through the calcaneocuboid joint following lateral distraction; however, these increases in joint pressure have not been found to be significant in a flatfoot model.

A posterior calcaneal displacement osteotomy may be indicated for reduction of significant rearfoot valgus (see figure 7 of the original guideline document). The rationale for performing this osteotomy includes restoring the gastrosoleal complex as a heel invertor, increasing supinatory ground reactive forces, and decreasing medial arch load. Recent studies have shown good clinical results with this osteotomy. However, investigators have noted that the osteotomy alters motion and joint contact characteristics within the ankle joint, which may predispose this joint to premature arthrosis. In cases of more significant deformity, double calcaneal osteotomies (Evans and posterior calcaneal displacement osteotomy) have been performed concurrently to achieve adequate correction (see figure 8 of the original guideline document). Other calcaneal osteotomies have been described but are less frequently used.

The Cotton opening wedge osteotomy through the medial cuneiform has been promoted as a valuable adjunctive procedure to plantarflex the medial column. Correction of deformity is achieved with a structural allograft or autograft.

Arthroereisis. Several implant devices have been designed to limit pronatory motion by blocking movement between the talus and calcaneus. In the adult, arthroereisis is seldom implemented as an isolated procedure. Because of the long-term compensation and adaptation of the foot and adjunctive structures for flatfoot function, other ancillary procedures are usually used for appropriate stabilization. Long-term results of arthroereisis in the adult flexible flatfoot patient have not been established. Some surgeons advise against the subtalar arthroereisis procedure because of the risks associated with implantation of a foreign material, the potential need for further surgery to remove the implant, and the limited capacity of the implant to stabilize the medial column sag directly.

Arthrodesis. Arthrodesis of the medial column, including the naviculocuneiform joint(s) and/or metatarsocuneiform joint, may be used when medial column collapse is noted. More recently, medial column arthrodesis techniques have been

implemented as adjunctive procedures. Isolated talonavicular joint arthrodesis provides very powerful correction of the subluxated talar head. The reduction in subtalar joint range of motion after talonavicular joint arthrodesis has limited the use of this procedure.

Calcaneocuboid joint distraction arthrodesis has been described for the correction of the flatfoot deformity with significant forefoot abduction. Indications are similar to those of the Evans osteotomy, but the arthrodesis technique is thought to avoid the increased calcaneocuboid joint pressures/arthrosis that have been theorized in adult patients after Evans osteotomy. Autogenous bone grafting is recommended for distraction arthrodesis because of higher nonunion rates. Double arthrodesis (talonavicular and calcaneocuboid joints) may also be used for correction of the flatfoot deformity and may provide less subtalar joint arthrosis than the isolated talonavicular joint arthrodesis.

Isolated subtalar joint and triple arthrodesis provide viable options for flatfoot correction (see figure 11 in the original guideline document). A fixed forefoot varus necessitates the use of triple arthrodesis. Additionally, triple arthrodesis is more often implemented in the rigid flatfoot with rearfoot arthrosis in the subtalar and midtarsal joints. Subtalar joint arthrodesis is more frequently indicated in the flatfoot with a reducible deformity without midtarsal joint arthrosis or fixed forefoot varus. Union rates with subtalar joint arthrodesis have been reported to be high.

Pantalar arthrodesis is used in longstanding deformities with rearfoot and ankle degenerative changes. Often, the ankle arthrosis is a result of a valgus tilt of the talus within the mortise. In situations in which the midtarsal joint has been spared of degenerative changes, tibiototalcalcaneal arthrodesis may be warranted. Retrograde intramedullary nail fixation may be used in these cases.

Adjunctive Soft Tissue Procedures. The shortened Achilles muscle-tendon complex may be lengthened in conjunction with reconstructive flatfoot surgery. Common techniques include gastrocnemius recession, gastrocnemius-soleus recession, and Achilles-tendon lengthening. The relationship between flexible flatfoot and contracted Achilles muscle-tendon complex is well established, and the loss of muscle strength after lengthening has been shown to be insignificant.

Posterior Tibial Tendon Dysfunction (Pathway 3)

PTTD is the most common cause of the adult acquired flatfoot. Dysfunction of the posterior tibial tendon is typically a unilateral condition caused by pathologic changes within the tendon. The deformity is usually progressive and results in a flexible to rigid flatfoot, depending on the stage of the condition.

Significant History (Pathway 3, Node 1)

PTTD is common in women aged 45 to 65 years. Patients usually present without a specific history of trauma. Symptoms are typically preceded by overuse activity. Patients with PTTD may also present with a preexisting flatfoot deformity, family history of flatfoot deformity, or other systemic conditions.

Significant Findings and Classification (Pathway 3, Node 2)

Patients with PTTD may present with symptoms at any stage of this progressive condition. Numerous PTTD classification systems have been developed. The classification originally described by Johnson and Strom and later modified was used in developing this guideline. Additionally, the guideline panel further divided stage 2 PTTD into early (stage 2A) and late (stage 2B). Significant findings are discussed in this document, according to the stages of the deformity.

Stage 1 (Pathway 3, Node 3)

In stage 1 PTTD, the foot may have an unchanged appearance with no deformity. Pain and edema along the medial aspect of the rearfoot corresponding to the posterior tibial tendon may be present. This is usually indicative of tenosynovitis or early tendinosis. Clinical assessment may demonstrate increased warmth, edema, and tenderness along the course of the tendon. Weakness is usually absent in stage 1 PTTD. Patients are typically able to perform a single heel raise without difficulty, although this maneuver may reproduce symptoms. Radiographs are usually unremarkable, whereas MRI and ultrasound studies may show tenosynovitis.

Stage 2A (Pathway 3, Node 4)

Stage 2 PTTD encompasses a wider spectrum of pathology. Stage 2A is usually characterized by medial rearfoot pain, edema, and tenderness along the course of the posterior tibial tendon, and mild valgus of the heel, with or without lowering of the medial longitudinal arch. There may be some abduction of the forefoot on the rearfoot (too many toes sign). Although patients with stage 2A PTTD may be able to perform a single heel raise, they are more likely to have difficulty and pain completing this maneuver. Subtalar joint motion is supple with increased eversion. Radiographs typically reveal a flatfoot deformity with increased talo-first metatarsal angle, peritalar subluxation, and increased calcaneocuboid abduction angle. MRI and ultrasound studies may reveal tenosynovitis, tendinosis, or attenuation of the posterior tibial tendon (see figure 9 in the original guideline document).

Stage 2B (Pathway 3, Node 5)

The findings in stage 2B PTTD are similar to those in stage 2A, with the addition of lateral pain (sinus tarsi, subfibular, cuboid), more severe valgus deformity, collapse of the medial longitudinal arch, and obvious abduction of the forefoot on the rearfoot. Forefoot supinatus may be present in stage 2B. There may also be decreased subtalar joint motion. Contracture of the posterior muscle group is typical. Radiographs show an increase in the talo-first meta-tarsal angle on AP and lateral views as well as peritalar subluxation. Degenerative changes are usually absent. MRI and ultrasound findings are similar to those found in stage 2A, although they may also reveal complete tendon rupture.

Stage 3 (Pathway 3, Node 6)

In stage 3 PTTD, deformities become more severe and fixed. Significant loss of subtalar joint motion and contracture of the posterior muscle group usually occur at this stage. Lateral symptoms predominate. The patient is unable to perform a single heel-raise test and the rearfoot remains everted with a double heel-raise test (see figure 3 of the original guideline document). A forefoot varus is often noted. Radiographs of stage 3 PTTD show continued increase in angular deformities and may show degenerative changes within the rearfoot. MRI and ultrasound studies reveal similar findings to those in stage 2.

Stage 4 (Pathway 3, Node 7)

Stage 4 PTTD has ankle involvement (see figure 10 in the original guideline document). The medial soft tissue constraints, including the deltoid ligament, become attenuated. An AP view of the ankle may show valgus tilt of the talus within the ankle mortise. Degenerative changes of the ankle may develop with advancing PTTD.

Initial Treatment (Pathway 3, Node 9)

Initial treatment options for most cases of PTTD include various types of nonsurgical therapy. Stage 1 PTTD may be treated with a short-leg cast or walking boot for 4 to 6 weeks. The patient may participate in physical therapy for rehabilitation of the posterior tibial tendon muscle after cast immobilization. Antiinflammatory medications may assist in reducing inflammation. Orthoses or ankle stirrup braces may also be used in stage 1 PTTD.

Stage 2A PTTD is usually treated with orthoses. Stage 2A PTTD may involve some deformity or impending deformity, thus necessitating support of the longitudinal arch and heel. An orthotic device with a deep heel cup is usually sufficient. Patients in stage 2B PTTD may require a University of California Biomechanics Laboratory device or AFO (see figure 5 in the original guideline document). Modifications in footwear, such as an extended medial counter or medial heel wedge, may be helpful. In the acutely painful foot, immobilization may be warranted.

Stage 3 PTTD may require some type of custom AFO or supramalleolar brace. The AFO should be articulated to permit ankle joint range of motion. Unfortunately, once the deformity is fixed, the patient may have difficulty tolerating various types of AFOs. This is especially true in cases of significant rearfoot valgus deformity.

Stage 4 PTTD is usually treated with a nonarticulated AFO.

Surgical management (Node 12) may be an initial treatment option when inherent disability is present.

Clinical Response (Pathway 3, Node 10)

When nonsurgical care is rendered, the clinical response is assessed. If the patient is doing well, initial treatment may be continued (Node 11). If there is little or no improvement or if initial improvement deteriorates, surgical management may be

considered (Node 12). In cases in which the initial evaluation and treatment were performed by a primary care physician, referral to a podiatric foot and ankle surgeon is warranted.

Surgical Intervention (Pathway 3, Node 12)

Surgical management during stage 1 PTTD (Node 13) primarily consists of synovectomy. This should be considered if noninvasive therapy has been ineffective. Tendon decompression with synovectomy has been reported to provide relatively good results.

In stage 2A (Node 14), synovectomy, tendon debridement, and/or primary repair may be indicated. When tendon dysfunction is present, a tendon transfer should be considered as an adjunct procedure. Osteotomy has also been recommended when deformity is present. Calcaneal osteotomies may include posterior calcaneal displacement osteotomy or Evans calcaneal osteotomy. Posterior muscle group lengthening should be considered when equinus is present.

Stage 2B PTTD (Node 15) may require synovectomy or tendon repair in combination with a tendon transfer. Advancing deformity is common in stage 2B, necessitating some type of realignment through an osteotomy or arthrodesis. The calcaneal osteotomies described for stage 2A, as well as double calcaneal osteotomy, may be considered for the stage 2B deformity. Medial column osteotomies (e.g., the Cotton open-wedge osteotomy) may also be considered as adjunct procedures.

Isolated rearfoot arthrodesis procedures have been recommended in stage 2B. These procedures may include subtalar joint, calcaneocuboid interpositional bone block, or medial column arthrodesis (see figure 9 in the original guideline document). Selection of a single joint arthrodesis is based on the type and degree of deformity (see table 1 in the original guideline document), as well as on the goals and expectations of the patient and surgeon. Isolated rearfoot arthrodesis of any joint will subsequently affect kinematics and may contribute to adjacent joint arthrosis.

Arthroereisis procedures have also been recommended as an option in stage 2 PTTD. Additionally, tendon transfers have been used extensively to augment these osseous procedures. Stage 2 invariably results in equinus deformity that should be addressed with posterior muscle group lengthening.

Stage 3 PTTD (Node 16) usually requires an arthrodesis procedure. An isolated joint arthrodesis may be considered. However, if arthrosis is extensive and involves the entire rearfoot, triple arthrodesis is the procedure of choice. In longstanding, rigid deformities, a fixed forefoot varus often necessitates triple arthrodesis. Additionally, such patients require lengthening of the posterior muscle group to address the equinus deformity.

Stage 4 (Node 17) involves the ankle joint. A triple arthrodesis in combination with deltoid ligament repair and a medializing calcaneal osteotomy may be considered in cases in which valgus deformity of the ankle exists with no degenerative joint disease (see figure 10 of the original guideline document). A pantalar arthrodesis may be indicated when degenerative changes involve not

only the rearfoot but also the ankle joint. In addition, total ankle replacement or supramalleolar osteotomy may be used in conjunction with triple arthrodesis.

Tarsal Coalition (Pathway 4)

Tarsal coalition may present in the adult patient, and the evaluation and diagnosis of this developmental aberration is much the same as that for the child. This disorder is often an incidental finding to routine examination and radiographs in adult patients. Patients with tarsal coalition may be asymptomatic, display an otherwise normal examination, and not require treatment.

When tarsal coalitions in adults become painful, treatment may be required, including surgical intervention. Symptoms are often initiated by trauma. Degenerative changes secondary to tarsal coalition are common in the adult. Conservative measures (Node 5) may be used, depending on the degree of symptoms and deformity as well as activity demands. In addition to resection of the coalition (see figure 11 in the original guideline document), surgical treatment is more likely to include arthrodesis in the adult patient (Node 9) (see figure 12 in the original guideline document).

For further discussion, see Pathway 4 in the National Guideline Clearinghouse (NGC) summary of the American College of Foot and Ankle Surgeons (ACFAS) guideline [Diagnosis and Treatment of Pediatric Flatfoot](#).

Iatrogenic, Post-traumatic, or Arthritic Deformity (Pathway 5)

Significant History (Pathway 5, Node 1)

The patient with an iatrogenic, posttraumatic, or arthritic flatfoot presents a unique treatment challenge. Typically the healthy anatomy has been altered and there is a history of surgery, trauma, and/or arthritis. Severity of symptoms may vary depending on the cause and the patient's activity level. Pain may be isolated to one particular joint or region or may be global in nature. Duration and onset of pain should be determined.

The iatrogenic flatfoot may result from over- or undercorrection of many different deformities, such as talipes equino varus, pes cavus, metatarsus adductus, pes planovalgus, or Achilles tendon lengthening. A complete review of systems and family history are important in cases of suspected systemic disease.

A posttraumatic flatfoot may result from ankle fracture/dislocations, calcaneal fractures, talar neck fractures, Lis Franc fracture/dislocations, and other dislocations of the rearfoot and midfoot. Soft tissue injuries such as laceration of the posterior tibial tendon may also lead to a flatfoot deformity (see figure 13 in the original guideline document).

Arthritic conditions such as rheumatoid arthritis may also result in a flatfoot condition. In such cases, patients will often relate an insidious onset, with deformity and pain progressively worsening.

Significant Findings (Pathway 5, Node 2)

Clinical maneuvers such as the single and double heel-raise tests (previously described) may be used to test tendon function and joint range of motion of the foot and ankle. Pain on range of motion, crepitus, locking, or guarding may be noted. In many cases of iatrogenic/post-traumatic/arthritis flatfeet, the quality and quantity of joint motion are limited.

Diagnostic local anesthetic blocks are useful in determining symptomatic joints. Examination for previous incisions, lacerations, and deformity is performed. Palpation may isolate point tenderness or areas of inflammation, swelling, and joint effusions. The patient's gait is often antalgic. If the patient is already wearing orthoses or bracing devices, gait may be evaluated with and without these devices. Further weightbearing examination is performed as previously described in Pathway 1.

Radiographic Findings (Pathway 5, Node 3)

Radiographic evaluation of the iatrogenic or posttraumatic flatfoot is performed as previously described (Pathway 1, Node 3). Malunited or malreduced calcaneal fractures may show a loss of calcaneal height and decreased Böhler's angle (see figure 14 in the original guideline document). Evaluation of the distal tibia for malalignment is important, since post-traumatic ankle valgus may produce a flatfoot deformity. Lis Franc fracture/dislocations may show malalignment of the forefoot to the rearfoot. Evidence of previously placed implants or hardware may be present.

Plain radiographs are used to assess arthritic changes in the affected joints. However, in many cases, advanced imaging (e.g., computed tomography, MRI) may prove valuable in determining the extent of arthritic involvement and evaluating any changes in anatomic relationships. Erosive lesions or hypertrophic changes may exist if an arthritic process is present. MRI may also be helpful in assessing the posttraumatic foot for osseous viability (i.e., talar avascular necrosis) and for soft tissue pathology.

Diagnosis (Pathway 5, Node 4)

Once diagnosis of the causative factor(s) and the degree of deformity or arthrosis has been made, initial treatment may proceed.

Initial Treatment (Pathway 5, Node 5)

Activity modifications may be recommended, as indicated, in the initial treatment of the iatrogenic, posttraumatic, or arthritic flatfoot. If the patient is overweight, weight loss is encouraged. Over-the-counter ankle supports, custom foot orthoses, or extended leg bracing may be used. Antiinflammatory medications, corticosteroid injections, physical therapy, and footwear modifications are used in selected cases.

Clinical Response (Pathway 5, Node 6)

If clinical response to nonsurgical measures is satisfactory, these modalities are continued as indicated (Node 7). If symptoms recur or if initial treatment is unsatisfactory, surgical treatment may be warranted.

Surgical Intervention (Pathway 5, Node 8)

Osteotomies, arthrodesis, and soft tissue procedures may be used alone or in combination when surgically treating the iatrogenic, posttraumatic, or arthritic flatfoot. Posterior and/or anterior calcaneal osteotomies may be useful when performing extraarticular correction of the flatfoot deformity. Cuneiform osteotomy is beneficial when addressing first ray pathology. Distal tibial osteotomy may be an option in cases of ankle valgus.

When arthrosis or significant deformity exists, arthrodesis of the ankle, rearfoot, or midfoot may be necessary. Arthrodesis can be performed in situ with lesser degrees of deformity. However, realignment arthrodesis with or without bone grafting is commonly required. Posterior bone block distraction arthrodesis may be needed in cases of the neglected or malreduced calcaneal fracture (see figure 14 of the original guideline document). When treating deformity secondary to calcaneal fractures, extraarticular osteotomy combined with arthrodesis procedures may be used to enhance realignment of the foot and ankle.

Achilles-tendon lengthening or gastrocnemius recession may be needed to fully correct ankle equinus. Tendon transfer or ligament plications are performed as indicated.

Adjunctive Measures (Pathway 5, Node 9)

To maintain correction and prevent recurrent deformity, postsurgical orthotic or bracing management is used as needed.

Charcot Foot (Pathway 6)

Charcot foot, also called neuropathic osteoarthropathy, is a pathologic condition associated with peripheral neuropathy. Diabetes mellitus is the most common cause of this disorder. Syphilis, alcoholism, leprosy, and idiopathic neuropathy are less common causes. Charcot foot is a deformity characterized by pathologic fractures, joint dislocation, and overt loss of normal pedal architecture (see figures 15 and 16 of the original guideline document). The prevalence of Charcot foot involvement ranges from 0.15% of all patients with diabetes to 29% of patients with both diabetes and neuropathy.

Significant History (Pathway 6, Node 1)

Charcot joint involvement is most often seen in obese patients with longstanding diabetes who have peripheral neuropathy. Trauma may play a role in development, although, more commonly, the repetitive stress of daily weightbearing is enough to precipitate an episode.

Typically, the patient with an acute episode of Charcot foot presents with a generally painless, swollen extremity. Regional inflammation may lead to

misdiagnosis. Charcot foot often results in a progressive flatfoot deformity that may be grossly unstable or rigid in nature. Ulceration and subsequent osteomyelitis may develop because of the resultant deformity.

Significant Findings (Pathway 6, Node 2)

The acute Charcot foot has a dramatically different presentation than the chronic Charcot foot. The acute Charcot foot exhibits significant localized or regional changes, including erythema, warmth, and swelling. The condition is normally painless, despite the presence of significant deformity. Because of increased blood flow to the area, pulses in the foot and extremity are palpable and often bounding. Radiographs may show that the architecture appears normal, with the exception of soft tissue swelling or vascular calcifications. The stress of continued weightbearing leads to joint subluxation and fractures, resulting in dislocation and deformity. The foot is clearly unstable in this phase.

In the chronic Charcot foot, deformity and/or a plantar ulcer may be the most striking feature(s). The limb may be enlarged and indurated because of osseous changes, but erythema will be absent unless there is infection or ulceration. An insensate extremity with gross deformity or instability without vascular deficit is the rule in chronic Charcot foot.

Ancillary Diagnostic Procedures (Pathway 6, Nodes 3 and 4)

Because of the differential diagnosis, identification of a Charcot process may require additional diagnostic studies as follows.

- Imaging studies. Radiographs may depict joint disorganization, osteolysis, and deformity. These findings, combined with the presence of ulceration associated with chronic Charcot foot, often produce a diagnostic dilemma regarding whether the pathology reflects infection or Charcot. Advanced imaging studies play a clarifying role. Bone scans, computed tomography, and MRI studies have been advocated to help differentiate the diagnosis.
- Laboratory testing. Serologic testing may also help in the diagnostic process. In patients with no history of diabetes and with acute signs of inflammation, useful information may be obtained through routine blood work and tests that measure complete blood cell count, sedimentation rate, uric acid, blood glucose, and glycohemoglobin.
- Bone biopsy and bone culture. In the presence of apparent osteolysis or destructive bone changes, particularly when adjacent ulceration exists, bone biopsy and bone culture may be the best method for confirming or negating the presence of osteomyelitis.

Diagnosis (Pathway 6, Node 5)

The diagnosis of neuropathic osteoarthropathy generally provides distinction between acute and chronic Charcot foot.

- Acute Charcot foot. Recognition of acute Charcot foot is often based on clinical acumen and a high index of suspicion. Profound unilateral swelling with inflammation in a diabetic patient with neuropathy is the rule. Depending

on the duration, bone changes (e.g., joint effusion, pathologic fracture, osteolysis, dislocation) may or may not be present. The process may occur spontaneously, following trauma, or after surgery.

- Chronic Charcot foot. The patient with chronic Charcot foot typically presents with overt deformity and resultant contiguous ulceration. The chronic Charcot foot is usually free of erythema and swelling, except in the ulcerated or infected foot.

Treatment of Acute Charcot (Pathway 6, Node 6)

Treatment of the acute Charcot limb must begin with absolute restriction of weightbearing that would otherwise lead to certain deformity. Avoidance of weightbearing of the extremity may be accomplished with the use of crutches or a wheelchair. Elevation and immobilization with a compressive wrap is helpful to reduce acute swelling and abate the hyperemic process. The patient may be immobilized in a splint, cast, or removable orthosis. Immobilization should be continued until quiescence (Node 6), with absence of inflammation and edema. More recently, treatment of the acute Charcot process with pharmacotherapy and/or bone stimulation has been advocated to aid in abatement. In some cases in which deformity is present, surgical management of the acute Charcot foot may be indicated.

Treatment of Chronic Charcot (Pathway 6, Nodes 7-9)

Treatment of the chronic Charcot foot varies, depending on whether instability or ulceration is present. For information on the treatment of infection and ulceration, refer to the ACFAS guideline "Diabetic Foot Disorders: A Clinical Practice Guideline". In the absence of infection or ulceration, the clinician must assess whether the foot is stable or unstable to reach a decision regarding allowance of weightbearing.

- Stable chronic Charcot foot. Patients with stable chronic Charcot foot (Node 8) may be provided with relatively simple supportive measures and therapeutic footwear, and allowed to bear weight with periodic evaluation. In some instances, extradePTH or custom-molded shoes combined with in-shoe orthoses or multiple density insoles may provide enough support to allow daily activities and prevent ulceration. These measures, combined with patient education, are important to prevent recurrence. In the presence of a nonhealing ulceration, isolated exostectomy may be considered.
- Unstable chronic Charcot foot. The unstable chronic Charcot foot (Node 8) provides a more difficult treatment challenge. Various forms of bracing - patellar weightbearing, molded AFO, adjunctive footwear - may be used. If these are unsuccessful, the foot may require surgical intervention with arthrodesis or amputation (Node 9) (see figures 15 and 16 in the original guideline document).

In summary, flatfoot deformity as a result of neuropathic osteoarthropathy is a diagnostic dilemma that deserves the full attention of the attending clinician. Recognition and timely management can prevent lifelong deformity or amputation.

Neuromuscular Flatfoot (Pathway 7)

The neuromuscular flatfoot is the result of any one of a number of conditions that generally cause weakness or overactivity of intrinsic and extrinsic musculature. There are numerous causes of the adult neuromuscular flatfoot, including the expression of congenital or hereditary disorders, cerebral vascular accidents, posttraumatic responses, and iatrogenic responses (see figure 17 in the original guideline document).

Significant History (Pathway 7, Node 1)

Focused history includes previous trauma, surgery, pertinent family history, and any neurologic deficits, as well as pain and dysfunction during ambulation.

Significant Findings (Pathway 7, Node 1)

Typical physical findings of neuromuscular flatfoot include gait abnormalities, limb anomalies, and abnormal neurologic findings. Deformities may be flexible or rigid in nature. Extrapedal manifestations may also be recognized.

Radiographic Findings (Pathway 7, Node 2)

As previously described in this guideline, radiographic findings consistent with flatfoot include various angular changes of the foot, peritalar subluxation, and degenerative arthritis in more advanced stages.

Diagnosis (Pathway 7, Node 4)

When formulating a diagnosis and treatment plan for the neuromuscular flatfoot, it is important to determine whether the disorder is progressive or static and whether it has a central or peripheral etiology. A neurology consultation may be warranted (Node 3).

Initial Treatment (Pathway 7, Node 5)

Initial treatment options may include 1 or more of the following: activity modifications, medication, footwear modifications, physical therapy, orthotic devices/AFO, and bracing. Progressive disorders should be monitored to assess for worsening of condition and the need for treatment modification.

Clinical Response (Pathway 7, Node 6)

Once initial treatment options have been implemented, the clinical response is assessed and monitored. If the patient is doing well, initial treatment may be continued (Node 5). If little or no improvement is achieved or if initial improvement deteriorates, the treatment plan should be modified (Node 7) or surgical management may be considered (Node 8).

Surgical Intervention (Pathway 7, Node 8)

Surgical treatment for neuromuscular flatfoot, which is aimed at establishing optimal stability and improving gait, encompasses tendon transfers, osteotomies, and arthrodesing procedures. Generally, static or less progressive disorders

respond to tendon transfers and osteotomies, whereas more progressive disorders are more commonly treated with arthrodesis.

Adjunctive Measures (Pathway 7, Node 9)

After surgery, treatment may be enhanced with adjunctive forms of therapy such as foot orthoses, AFO, leg bracing devices, and assistive devices.

Recurrence (Pathway 7, Node 10)

Neurologic disorders and their clinical sequelae are often progressive or recurrent, requiring periodic monitoring, reassessments, and modifications of the treatment plan (Node 6).

CLINICAL ALGORITHM(S)

Algorithms are provided for:

- [Adult Flatfoot](#)
- [Adult Flexible Flatfoot \(Non-Posterior Tibial Tendon Dysfunction \[PTTD\] Flexible Flatfoot\)](#)
- [Posterior Tibial Tendon Dysfunction \(Adult Acquired Flatfoot\)](#)
- [Tarsal Coalition](#)
- [Iatrogenic/Post-traumatic/Arthritic Adult Flatfoot](#)
- [Charcot Foot](#)
- [Adult Neuromuscular Flatfoot](#)

EVIDENCE SUPPORTING THE RECOMMENDATIONS

TYPE OF EVIDENCE SUPPORTING THE RECOMMENDATIONS

The type of evidence supporting the recommendations is not specifically stated.

This clinical practice guideline is based on the consensus of current clinical practice and review of the clinical literature.

BENEFITS/HARMS OF IMPLEMENTING THE GUIDELINE RECOMMENDATIONS

POTENTIAL BENEFITS

- Appropriate diagnosis and treatment of adult flatfoot
- Various osteotomies for correction of adult flatfoot have been recommended and studied. The main advantage of periarticular osteotomies is preservation of joint function while improving structural alignment.

POTENTIAL HARMS

- Investigators have noted that the osteotomy alters motion and joint contact characteristics within the ankle joint, which may predispose this joint to premature arthrosis.

- Some surgeons advise against the subtalar arthroereisis procedure because of the risks associated with implantation of a foreign material, the potential need for further surgery to remove the implant, and the limited capacity of the implant to stabilize the medial column sag directly.

IMPLEMENTATION OF THE GUIDELINE

DESCRIPTION OF IMPLEMENTATION STRATEGY

An implementation strategy was not provided.

IMPLEMENTATION TOOLS

Clinical Algorithm

For information about [availability](#), see the "Availability of Companion Documents" and "Patient Resources" fields below.

INSTITUTE OF MEDICINE (IOM) NATIONAL HEALTHCARE QUALITY REPORT CATEGORIES

IOM CARE NEED

Getting Better
Living with Illness

IOM DOMAIN

Effectiveness
Patient-centeredness

IDENTIFYING INFORMATION AND AVAILABILITY

BIBLIOGRAPHIC SOURCE(S)

Lee MS, Vanore JV, Thomas JL, Catanzariti AR, Kogler G, Kravitz SR, Miller SJ, Gassen SC. Diagnosis and treatment of adult flatfoot. J Foot Ankle Surg 2005 Mar-Apr;44(2):78-113. [223 references] [PubMed](#)

ADAPTATION

Not applicable: The guideline was not adapted from another source.

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FINANCIAL DISCLOSURES/CONFLICTS OF INTEREST

Not stated

GUIDELINE STATUS

This is the current release of the guideline.

GUIDELINE AVAILABILITY

Electronic copies: Available in Portable Document Format (PDF) from the [American College of Foot and Ankle Surgeons Web site](#).

Print copies: Available from the American College of Foot and Ankle Surgeons, 515 Busse Highway, Park Ridge, IL 60068-3150; Web site: www.acfas.org.

AVAILABILITY OF COMPANION DOCUMENTS

None available

PATIENT RESOURCES

None available

NGC STATUS

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