

Diagnosis and Treatment of Adult Flatfoot

Clinical Practice Guideline Adult Flatfoot Panel: Michael S. Lee, DPM,¹ John V. Vanore, DPM,² James L. Thomas, DPM,³ Alan R. Catanzariti, DPM,⁴ Geza Kogler, PhD,⁵ Steven R. Kravitz, DPM,⁶ Stephen J. Miller, DPM,⁷ and Susan Couture Gassen⁸

This clinical practice guideline (CPG) is based on the consensus of current clinical practice and review of the clinical literature. The guideline was developed by the Clinical Practice Guideline Adult Flatfoot Panel of the American College of Foot and Ankle Surgeons. The guideline and references annotate each node of the corresponding pathways.

Introduction to Adult Flatfoot (Pathway 1)

Foot and ankle specialists agree that flatfoot is a frequently encountered pathology in the adult population. For the purpose of this document, adult flatfoot is defined as a foot condition that persists or develops after skeletal maturity and is characterized by partial or complete loss (collapse) of the medial longitudinal arch. Adult flatfoot may present as an incidental finding or as a symptomatic condition with clinical consequences ranging from mild limitations to severe disability and pain causing major life impediments. Adult flatfoot encompasses a wide variety of pathologic etiologies that may include a benign process reflecting continuation of a congenital problem, trauma, or a condition associated with systemic pathology.

The adult flatfoot is often a complex disorder with a diversity of symptoms and various degrees of deformity. Pathology and symptoms are caused by structural loading

changes along the medial foot and plantar arch, as well as by collapse through the midfoot and impingement along the lateral column and rearfoot (1–5). Muscles in the leg and foot tend to fatigue and cramp because of overuse (6–8). Peritalar subluxation defines the pathologic malalignment of the talus about the subtalar and midtarsal joints (9, 10). Literature on the incidence and symptomatology of adult flatfoot is limited (11–13). Ferciot (14) estimated a 5% incidence of flatfoot in all children and adults. Harris and Beath (15) studied 3,619 Royal Canadian Army recruits and found that 15% had a simple hypermobile flatfoot, 6% had simple hypermobile flatfoot with a tight heel cord, and 2% had a tarsal coalition.

Significant History (Pathway 1, Node 1)

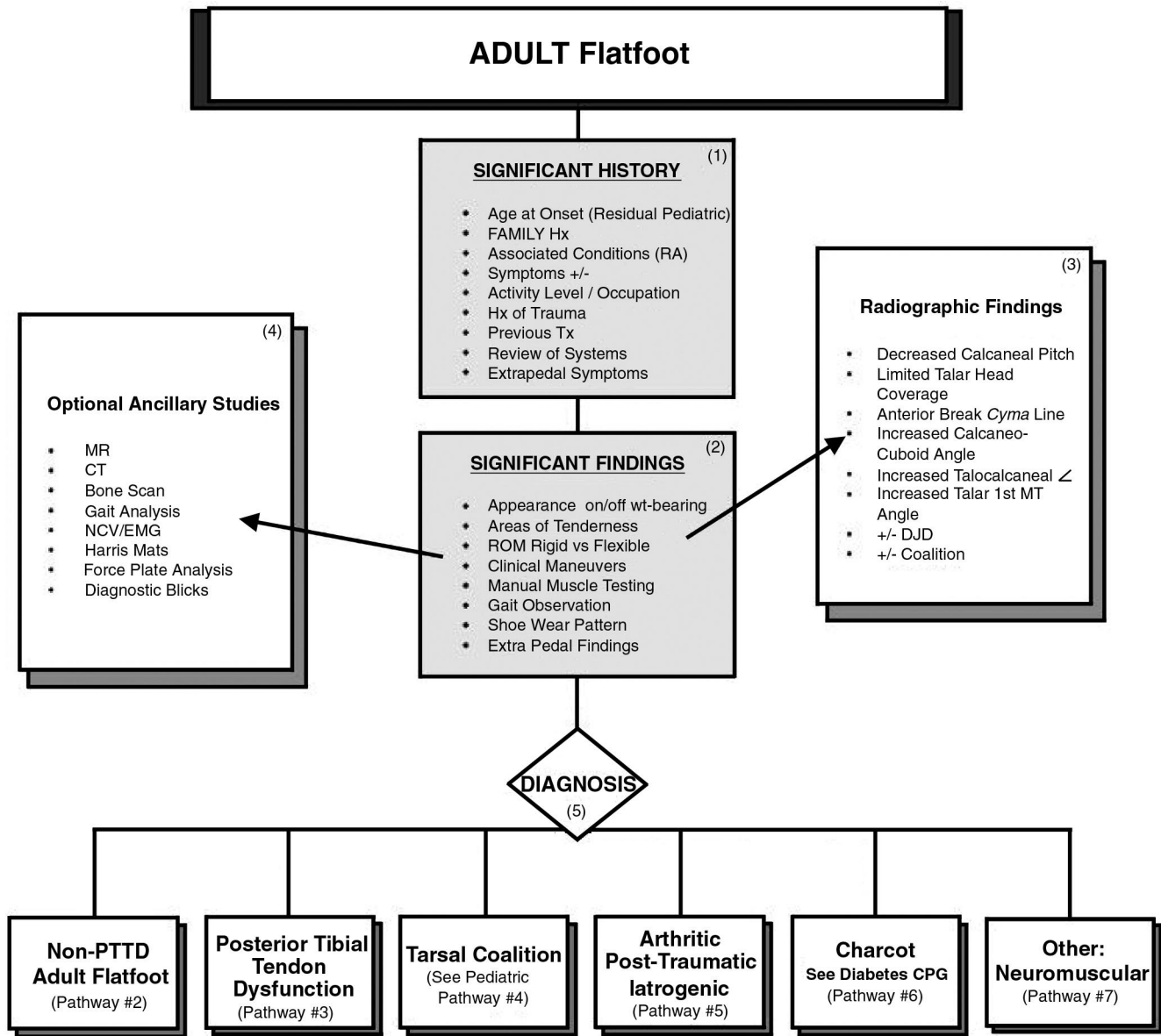
The natural history of the adult flatfoot has not been clearly defined, as evidenced by the absence of reliable studies analyzing the long-term sequelae of this condition (16). The deformity may be associated with pain, instability, and severe functional limitations or it may be of little clinical significance (17, 18).

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 (19, 20). 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. Extrapedal findings, such as knee, hip, or back pain, have also been associated with concurrent flatfoot (21).

Address correspondence to: John V. Vanore, DPM, Gadsden Foot Clinic, 306 South 4th St, Gadsden, AL 35901. E-mail: jvanore@bellsouth.net

¹Chair, Adult Flatfoot Panel, Ankeny, IA; ²Chair, Clinical Practice Guideline Core Committee, Gadsden, AL; ³Board Liaison, Birmingham, AL; ⁴Pittsburgh, PA; ⁵Springfield, IL; ⁶Richboro, PA; ⁷Anacortes, WA; ⁸Chicago, IL.

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PATHWAY 1

Significant Findings (Pathway 1, Node 2)

Flatfoot versus a normal foot (Fig 1) is readily apparent with clinical evaluation. The appearance of the foot, both on and off weightbearing, 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 (Figs 2 and 3). 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 (22, 23). Manual muscle testing and the single heel-rise test assess muscle strength and tendon function (24). Additionally, the double heel-rise test determines reducibility of rearfoot valgus (Figs 1 and 3).

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 splits, short tendo-Achilles with calf tenderness,

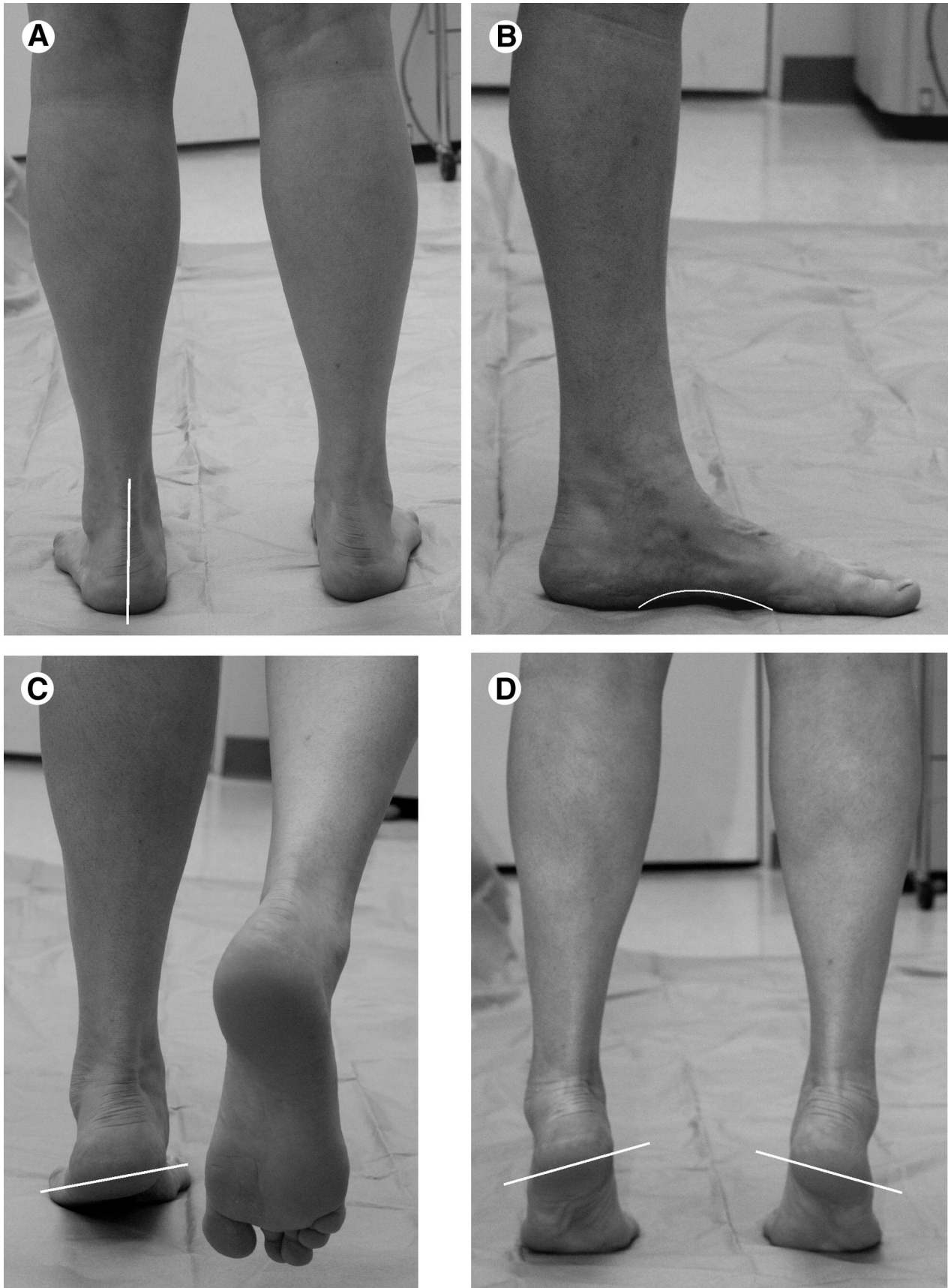


FIGURE 1 Clinical examination of the foot includes inspection of the weightbearing foot, with attention to (A) the frontal plane position of the heel and (B) maintenance of the medial longitudinal arch. The patient should be able to perform (C) single and (D) double heel rises, which show flexibility and normal supinatory potential of the foot.



FIGURE 2 Examination of the flatfoot compares the (A) nonweightbearing and (B) weightbearing arch of the foot. As the arch depresses, (C) the forefoot abducts and (D) the lesser toes become visible upon posterior observation of the foot. The relaxed calcaneal stance position is viewed standing behind the patient. A flatfoot deformity will demonstrate heel eversion that is accentuated with apparent bowing of the tendo-Achilles (Helbing sign). The too many toes sign, indicative of excessive forefoot abduction in the flatfoot, may also be noted.

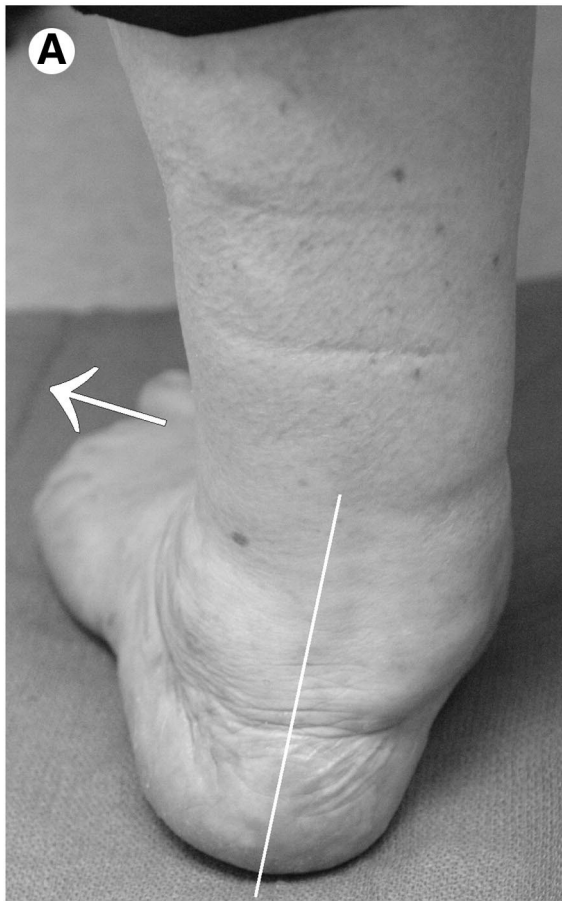


FIGURE 3 The severe flatfoot deformity shows very excessive heel valgus, (A) forefoot abduction, and (B) medial collapse of the foot. (C) The patient may be able to perform a double heel rise, but the heel shows lack of supinatory varus.

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 (25, 26).

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 (Fig 4):

- Calcaneal pitch is decreased in a flatfoot deformity and may approach 0° or become less than 0° 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 (27).
- 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) (28).
- Computed tomography (29–32).
- Bone scan (29, 33).
- Gait analysis via video or computer studies (34).

- Nerve conduction velocities/electromyography studies.
- Ink print mats (35) and Harris mats (36).
- Computerized force plate analysis.
- Local anesthetic diagnostic injections.
- Radiographs with the foot in the neutral position (37).

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 [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 (ie, flexible vs rigid). Flexible flatfoot in the adult may present as unilateral or, more commonly, as bilateral (38). It is frequently associated with a short or contracted Achilles muscle-tendon complex (39). In its late stages of progression, degenerative arthritis may occur, leading to loss of flexibility or ankylosis (37, 40). 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 plantarflexes on the calcaneus, which simultaneously everts and plantarflexes. Subtalar joint pronation unlocks the midtarsal joint, making it unstable and leading to various degrees of transverse plane abduction (41, 42). The tarsometatarsal and other joints may also be affected (2). 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 (eg, genu valgum, obesity, wide base of gait) (39).

Significant History (Pathway 2, Node 1)

Patients with adult flexible flatfoot may present with postural symptoms as well as weakness and fatigue in the

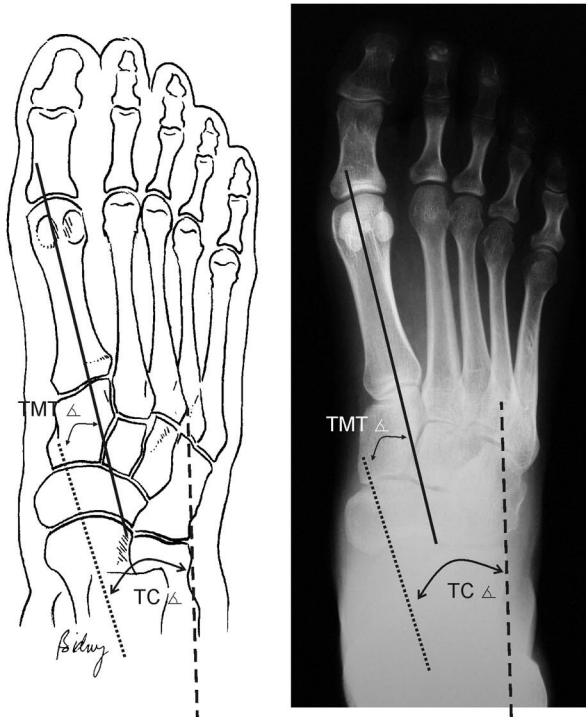
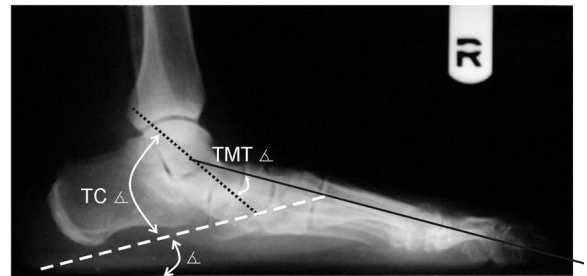
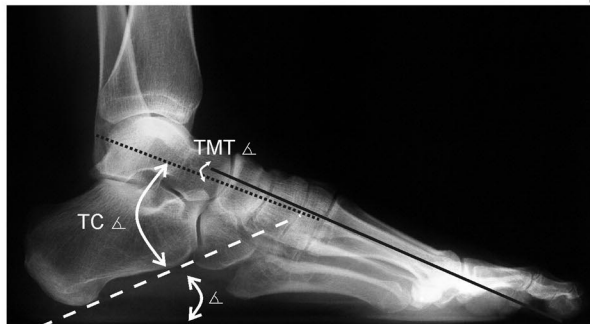
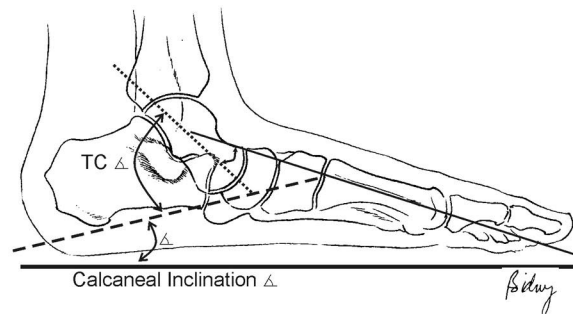
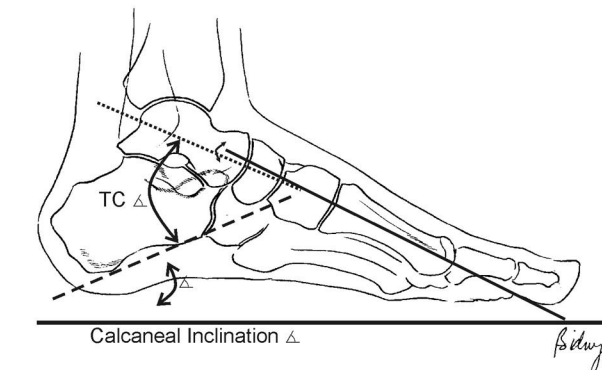
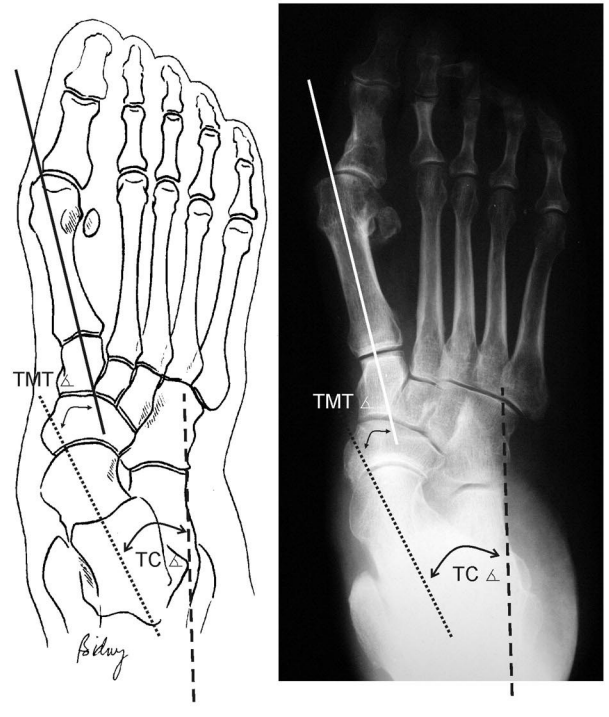
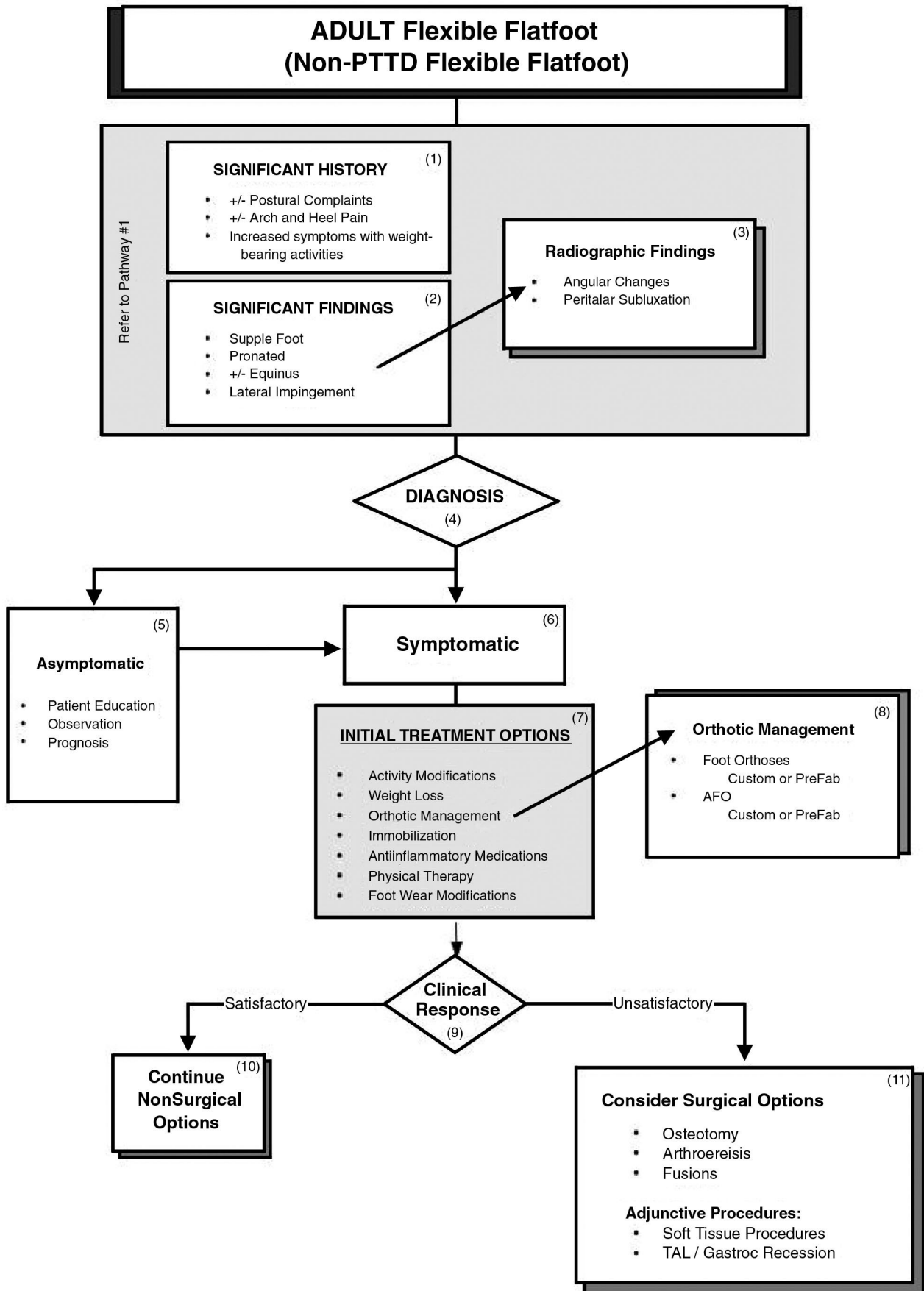
A**B**

FIGURE 4 Radiographic examination of the weightbearing foot allows for a more quantitative analysis of the flatfoot deformity. (A) Normal foot: line diagram with corresponding AP and lateral radiographs showing normal osseous relationships. Talocalcaneal angles may be drawn on both the AP and the lateral standing radiographs (dotted line, midtalar line; dashed line, longitudinal axis of hindfoot; solid line, first metatarsal bisection). The talocalcaneal angle (TC) increases with the degree of pronation. The talar first metatarsal angle should be nearly parallel on both the AP and lateral weightbearing radiographs. The AP radiograph shows good talar head coverage, whereas the lateral radiograph shows a normal calcaneal pitch or inclination angle with good height to the medial longitudinal arch. (B) Adult flatfoot: Deformity is shown on both the AP and the lateral radiographs, which show markedly increased talocalcaneal angles, decreased calcaneal pitch with depression of the medial longitudinal arch, and poor talar head coverage. Deviation of the talar first metatarsal angle is quite marked on the lateral film, showing a plantarflexed talus or talar ptosis. (Diagrams drawn by, and provided courtesy of, Maria Bidny, DPM, Hillside, MI.)



PATHWAY 2

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 (38). Arch, heel, and lateral foot pain may be primary complaints, with symptoms exacerbated by weightbearing activities (eg, 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 in [Pathway 1](#). These findings include various angular changes of the foot, peritalar subluxation, and degenerative arthritis in more advanced stages ([Fig 4](#)).

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 (43). 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 ([Fig 5](#)) (44–55).

Clinical Response ([Pathway 2](#), Node 9)

Nonsurgical therapy should be continued in patients who experience a favorable clinical response (Node 10). Surgical

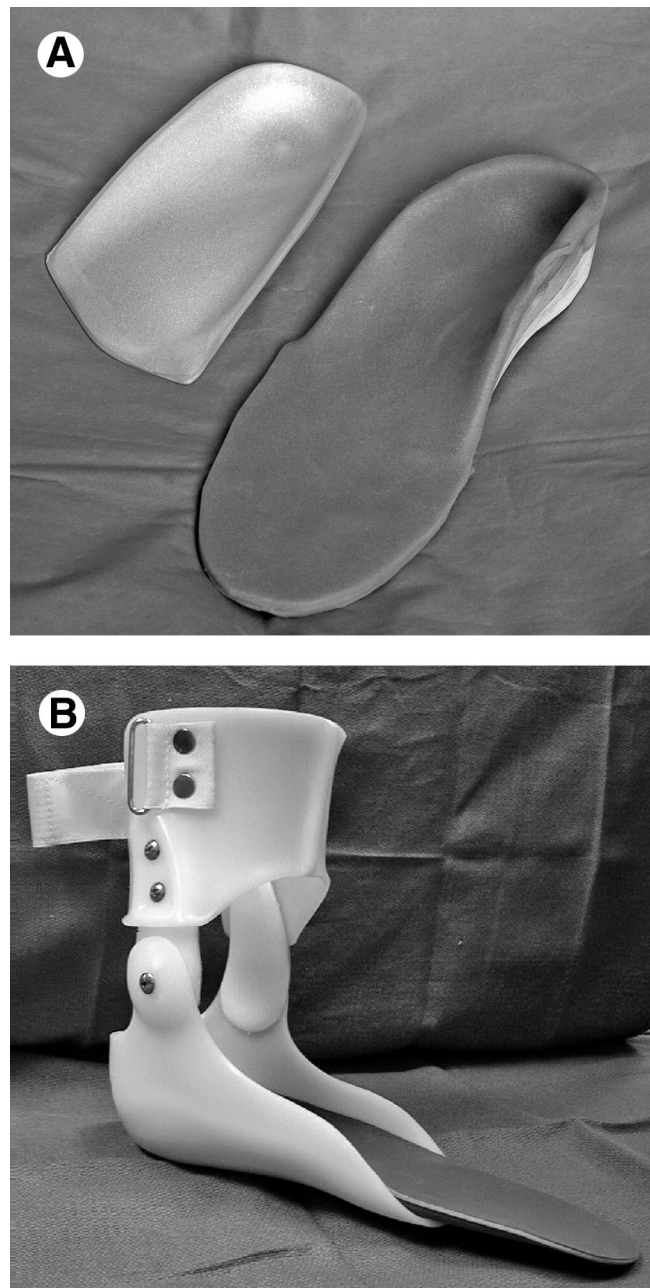


FIGURE 5 Orthotic management of flatfoot is accomplished with foot orthoses, which often include custom-made devices to accommodate the unique topography and degree of deformity present in the individual. The orthotic device may possess features of (A) a medial arch flange or deep heel seat, or (B) a full AFO (ankle-foot orthosis) that will support the entire ankle-tarsal complex.

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 (39).

TABLE 1 Relative corrective ability of flatfoot procedures

Surgical Procedure		Type of Correction Able to Be Achieved					
		Equinus	Medial Column Sag	Forefoot Abduction	Rearfoot Eversion	Peritalar Subluxation	Ankle Valgus
Osteotomies	Evans	o	++	++++	++	*	*
	PCDO	o	+	+	+++	++	+
	Double Calcaneal	o	++	++++	++++	+++	+
	Cotton	o	+++	o	+	+	?
Arthrodesis	Medial Column	o	++++	+	+	+	*
	Talonavicular	o	++	+++	+++	++++	*
	CC Distraction	o	++	++++	++	+++	*
	Double (MTJ)	o	++	+++	+++	++++	*
	Subtalar Joint	o	+	+	+++	++	*
	Triple	o	++	+++	++++	++++	*
	Pantalar	+	++	+++	++++	++++	*
	Ankle	+	+/o	+	+++	++	*
	TTCA	+	+	+	++++	++	*
	Arthroeresis	Arthroeresis	o	+	+	++	+
Soft Tissue Procedures	TAL	++++	+	+	++	+	*
	Gastroc Recession	+++	+	+	++	+	o

Scale, 0 to +++++.

Abbreviations: CC, calcaneocuboid; MTJ, midtarsal joint; PCDO, posterior calcaneal displacement osteotomy; TAL, tendo-Achillis lengthening; TTCA, tibiotalocalcaneal arthrodesis

*Indirect.

Surgical Intervention (Pathway 2, Node 11)

Various surgical techniques have been described for the treatment of adult flexible flatfoot. These may include osteotomy, arthroeresis, arthrodesis, and adjunctive soft tissue procedures (Table 1). 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 (39, 56–65).

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 (Fig 6) (66–69). This procedure has also been shown to plantarflex the first metatarsal and reduce talocalcaneal subluxation (66, 70–72). 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 (73–76).

A posterior calcaneal displacement osteotomy may be indicated for reduction of significant rearfoot valgus (Fig 7). 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 (70, 77–84). Recent studies have shown good clinical results with this osteotomy (79, 80, 85–88). 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 (89–93). In cases of more significant deformity, double calcaneal osteotomies (Evans and posterior calcaneal displacement osteotomy) have been performed concurrently to achieve adequate correction (Fig 8) (94–97). Other calcaneal osteotomies have been described but are less frequently used (98–101).

The Cotton opening wedge osteotomy through the medial cuneiform has been promoted as a valuable adjunctive procedure to plantarflex the medial column (102–105). Correction of deformity is achieved with a structural allograft or autograft.

Arthroeresis. Several implant devices have been designed to limit pronatory motion by blocking movement between the talus and calcaneus (106–118). In the adult, arthroeresis 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

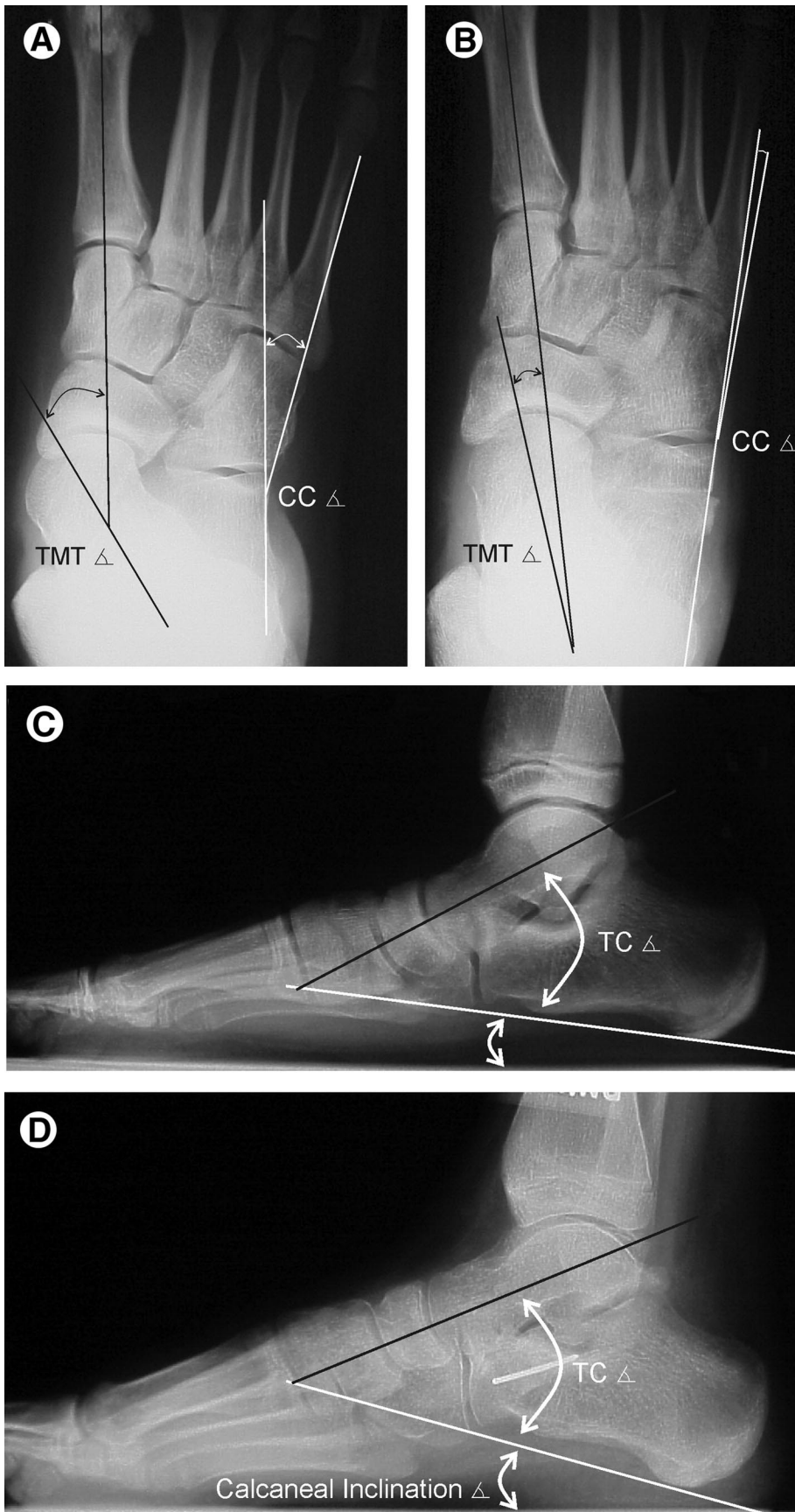


FIGURE 6 (A) This preoperative weight-bearing AP radiograph of flatfoot deformity shows increased calcaneocuboid (CC) and talo-first metatarsal (TMT) angles. An Evans calcaneal osteotomy with bone graft lengthening was performed, and (B) the postoperative weight-bearing AP radiograph shows reduction of fore-foot abduction and reduction of respective calcaneocuboid and talo-first metatarsal angles. Lateral (C) preoperative and (D) postoperative radiographs show similar improvement in biomechanical relationships.

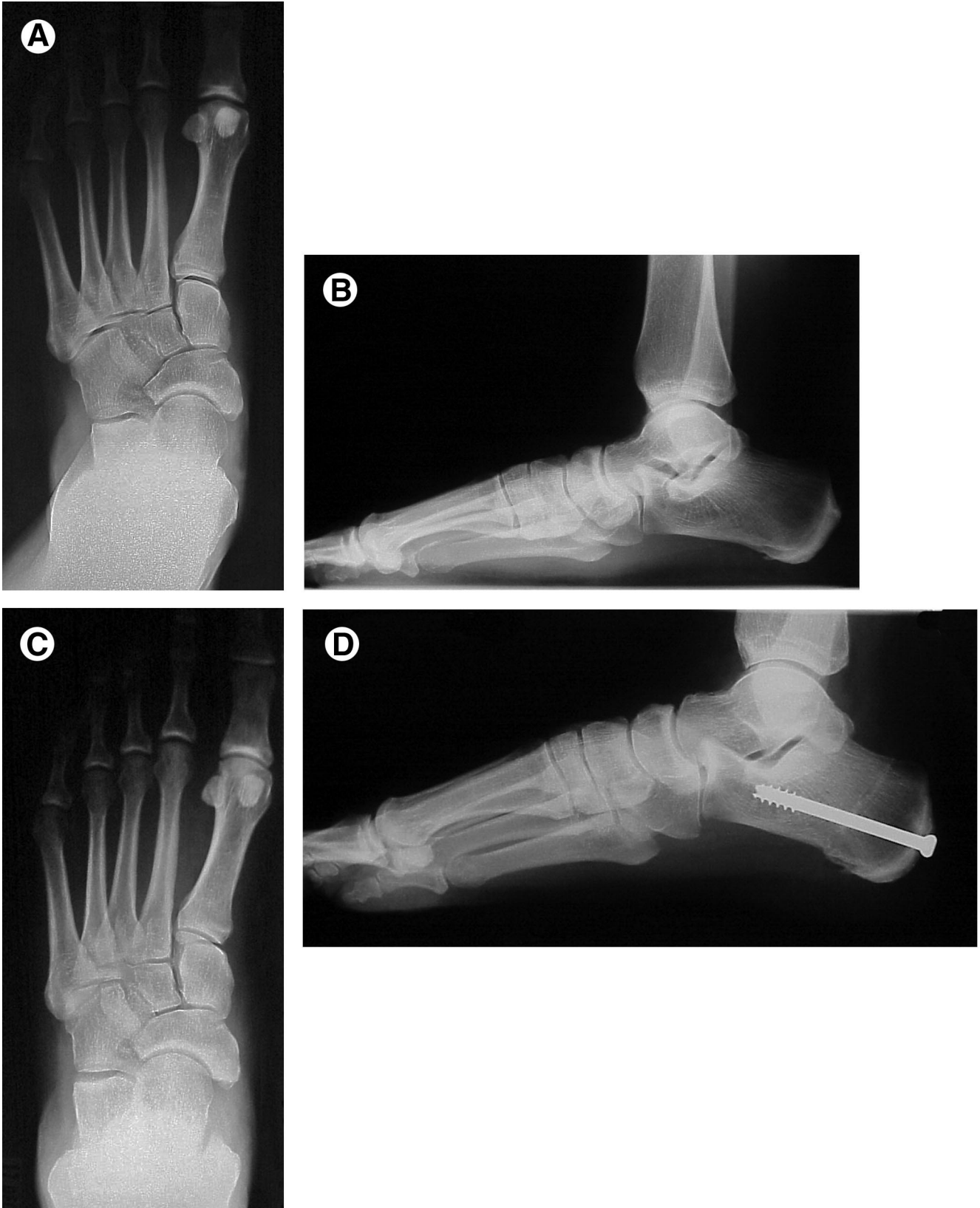


FIGURE 7 The posterior calcaneal osteotomy involves medial displacement of the posterior fragment to place the leg axis through the foot. Preoperative weightbearing (A) AP and (B) lateral radiographs show a flatfoot deformity with low calcaneal inclination or pitch and depression of the medial longitudinal arch. Forefoot abduction is limited. Postoperative weightbearing (C) AP and (D) lateral radiographs show a posterior calcaneal osteotomy with medial displacement. Improvement of talocalcaneal relationships can be appreciated as well as improved calcaneal inclination and arch height.



FIGURE 8 Multiple calcaneal osteotomies may be performed, as in this case, which included both an Evans-type procedure and a posterior calcaneal displacement osteotomy. (A) The preoperative weightbearing lateral radiograph of the flatfoot deformity shows a low calcaneal inclination angle, prominent talar declination, and depression of the longitudinal arch. (B) The postoperative weightbearing lateral radiograph shows the usefulness of performing an Evans bone graft lengthening of the lateral column combined with a posterior calcaneal osteotomy with medial displacement. Improvement of all biomechanical parameters is seen.

appropriate stabilization (37). 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 (119).

Arthrodesis. Arthrodesis of the medial column, including the naviculocuneiform joint(s) and/or metatarsocuneiform joint, may be used when medial column collapse is noted (120, 121). More recently, medial column arthrodesis techniques have been implemented as adjunctive procedures (80, 122, 123). Isolated talonavicular joint arthrodesis pro-

vides very powerful correction of the subluxated talar head (123–127). The reduction in subtalar joint range of motion after talonavicular joint arthrodesis has limited the use of this procedure (58, 127, 128).

Calcaneocuboid joint distraction arthrodesis has been described for the correction of the flatfoot deformity with significant forefoot abduction (63, 129). 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 (69, 74, 76, 130). Autogenous bone grafting is recommended for distraction arthrodesis because of higher nonunion rates (69). 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 (128, 131, 132).

Isolated subtalar joint and triple arthrodesis provide viable options for flatfoot correction (Fig 11) (133–137). 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 (138–140). Subtalar joint arthrodesis is more frequently indicated in the flatfoot with a reducible deformity without midtarsal joint arthrosis or fixed forefoot varus (141–143). Union rates with subtalar joint arthrodesis have been reported to be high (144).

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, tibiotalar calcaneal 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 (22, 83, 103, 145–150). 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 (151).

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 (152, 153). 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 (154).

Significant Findings and Classification (Pathway 3, Node 2)

Patients with PTTD may present with symptoms at any stage of this progressive condition. Numerous PTTD clas-

sification systems have been developed (24, 155, 156). The classification originally described by Johnson and Strom (24) and later modified (157) 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 (24). 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 (156).

Stage 2A (Pathway 3, Node 4)

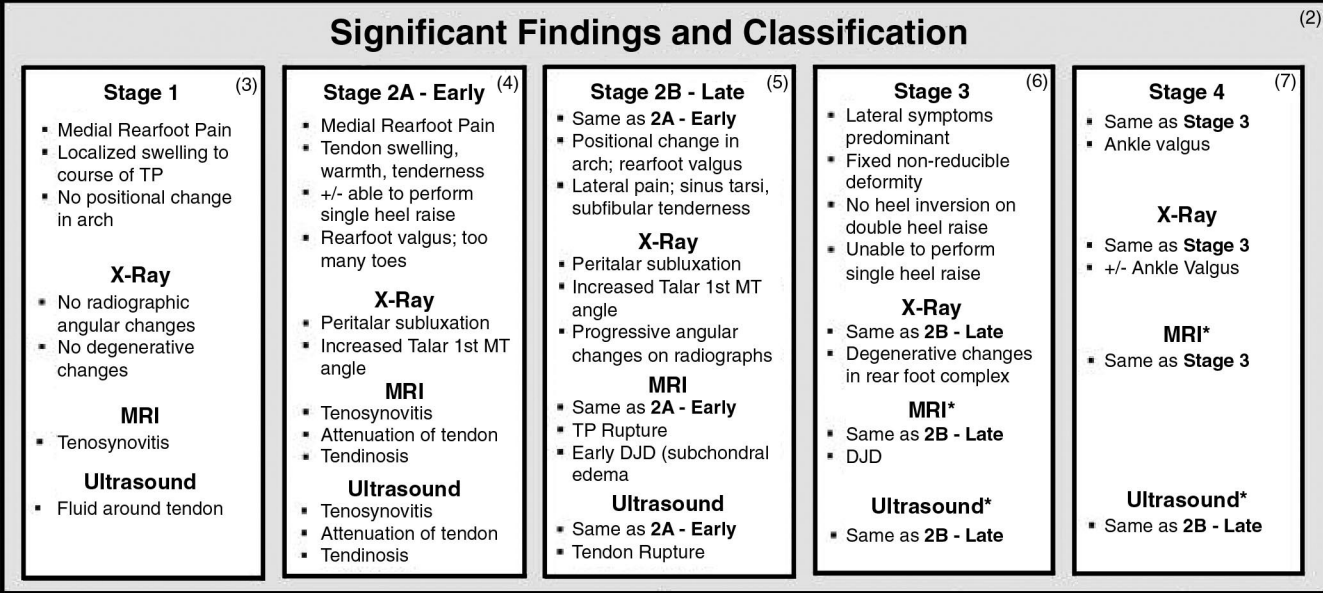
Stage 2 PTTD encompasses a wider spectrum of pathology. Stage 2A is usually characterized by medial rearfoot pain, edema, and tenderness along the course of the posterior tibial tendon, and mild valgus of the heel, with or without lowering of the medial longitudinal arch (24). There may be some abduction of the forefoot on the rearfoot (too many toes sign) (24). 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 (Fig. 9) (156) (Fig 9).

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-

Posterior Tibial Tendon Dysfunction ADULT Acquired Flatfoot

- Significant History** ⁽¹⁾
- Middle Aged Female
 - Unilateral Acquired Deformity
 - No History of Trauma



* These diagnostic tests are not essential to establish the diagnosis

DIAGNOSIS ⁽⁸⁾

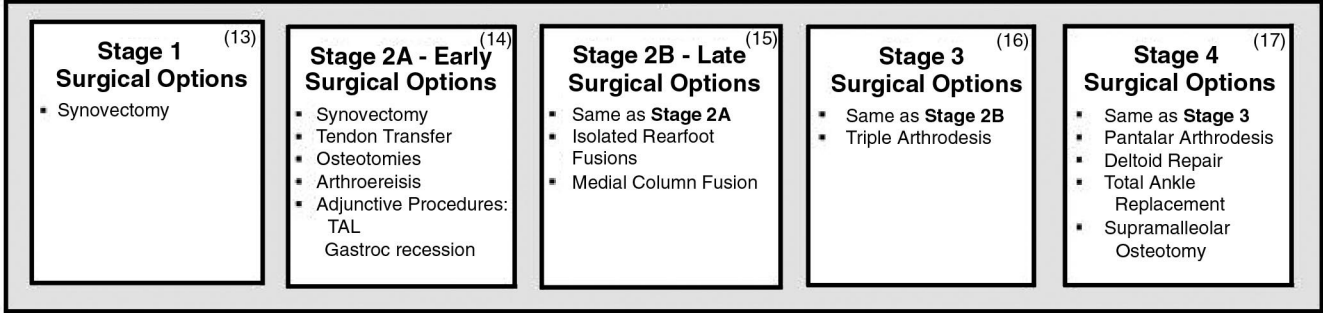
- Initial Treatment Options** ⁽⁹⁾
- Patient Education
 - Orthotic Management
 - Immobilization
 - Antiinflammatory Medications
 - Physical Therapy
 - Shoe modifications

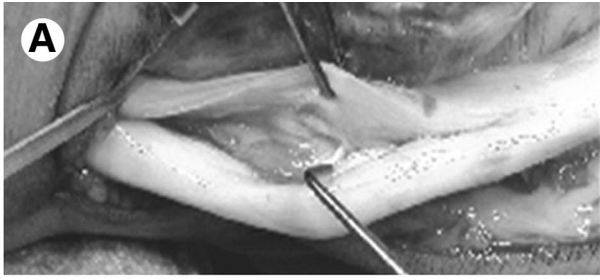
Surgical Management may be an Initial Treatment Option in more advanced Stages due to inherent disability.

Continue NonSurgical Options ⁽¹¹⁾

Clinical Response ⁽¹⁰⁾

Consider Surgical Options (BELOW) ⁽¹²⁾





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 (Fig 3). 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 (Fig 10). 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 (Fig 5). Modifications in footwear, such as an extended medial counter or medial heel wedge, may be help-

ful. 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 (153, 158).

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 (68, 78, 80, 82, 85, 87, 97, 159, 160). 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

FIGURE 9 (A) Adult-acquired flatfoot deformity is often the result of posterior tibial tendon dysfunction with pathologies involving the integrity of the tendon with attenuation or rupture. (B) MRI (shown here) or other imaging modalities may be used to evaluate the integrity of the posterior tibial tendon. This patient's preoperative weightbearing (C) AP and (D) lateral radiographs of flatfoot deformity reveals degenerative changes at the first and second metatarsocuneiform joints. Surgical treatment included tarsometatarsal arthrodesis and tendon repair, as shown in postoperative weightbearing (E) AP and (F) lateral radiographs.



well as double calcaneal osteotomy, may be considered for the stage 2B deformity (94, 97). Medial column osteotomies (eg, 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 (62, 129, 131, 161–164) (Fig 9). Selection of a single joint arthrodesis is based on the type and degree of deformity (Table 1), 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 (165, 166).

Arthroereisis procedures have also been recommended as an option in stage 2 PTTD (114, 167). Additionally, tendon transfers have been used extensively to augment these osseous procedures (80, 168–171). 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 (140, 143, 153, 172–175). 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 (Fig 10). 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 asymp-

tomatic, 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 (Fig 11), surgical treatment is more likely to include arthrodesis in the adult patient (Node 9) (Fig 12).

For further discussion, see Pathway 4 in the clinical practice guideline on pediatric flatfoot (176).

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 under-correction of many different deformities, such as talipes equino varus, pes cavus, metatarsus adductus, pes planovalgus, or Achilles tendon lengthening (177). 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 (Fig 13) (178).

Arthritic conditions such as rheumatoid arthritis may also result in a flatfoot condition (179). In such cases, patients will often relate an insidious onset, with deformity and pain progressively worsening.

FIGURE 10 (A) Posterior tibial tendon dysfunction may progress resulting in severe deformities as demonstrated by the heel eversion of the relaxed calcaneal stance position of this individual. Preoperative weightbearing (B) lateral and (C) AP radiographs show a large talocalcaneal angle with collapse of the medial longitudinal arch and low calcaneal pitch. The foot was reconstructed by triple arthrodesis to stabilize the entire hindfoot complex, as shown on postoperative weightbearing (D) lateral and (E) AP radiographs. The tendo-Achilles must also be evaluated and lengthened as necessary.

TARSAL COALITION

SIGNIFICANT HISTORY (1)

- +/- Symptoms
- Age: Typical Adolescent or Preadolescent
- +/- Weight Gain
- Altered Physical Activity
- +/- Trauma, Simple Sprain

Diagnostic Imaging (3)

- Standard Radiographs
- Special Radiographic Views:
 - Harris-Beath
- CT
- MRI
- Bone Scan

SIGNIFICANT FINDINGS (2)

- Variable Rearfoot Pain
- Decreased or Absent Rearfoot ROM
- +/- Peroneal Spasm
- Rigid Flatfoot

Refer to Pathway #1



INITIAL TREATMENT OPTIONS (5)

- Footwear Modifications, Arch Supports, Orthoses
- Activity Modifications
- Appropriate Weight Reduction
- Immobilization
- Anti-inflammatory Medications



Continue NonSurgical Options
(7)

Consider Surgical Options
(9)

- Resection of Coalition
- Arthrodesis
- Realignment Osteotomy

Observation &/or Orthoses
(8)

Recurrence of Symptoms

PATHWAY 4



FIGURE 11 Tarsal coalitions may present as rearfoot pain in a pronated foot or a foot that may appear normal. Examination determining limitation of subtalar or midtarsal range of motion is the crucial to diagnosis. Preoperative weightbearing (A) lateral and (B) oblique radiographs reveal osseous extension of the anterior calcaneus in this adult with a mild pronatory architecture. The patient underwent resection of the bar, as shown on postoperative weight-bearing (C) lateral and (D) oblique radiographs.

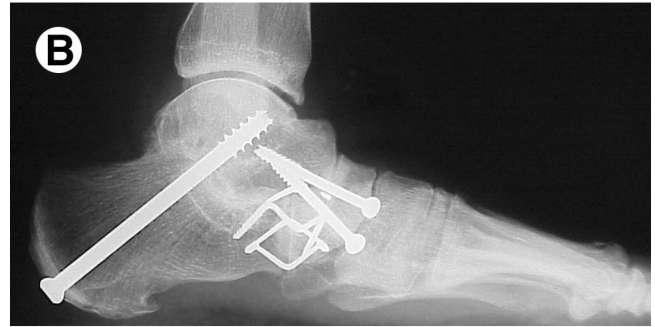


FIGURE 12 Older individuals with tarsal coalitions are generally treated with arthrodesis. (A) This symptomatic pronated foot presented as a rigid deformity, and a work-up showed a talocalcaneal coalition. The preoperative weightbearing lateral radiograph shows a pronatory architecture, with inability to visualize the middle facet of the subtalar joint. Triple arthrodesis was performed to restore normal osseous relationships through fusion of the entire rearfoot complex, shown on (B) the postoperative weightbearing lateral radiograph. (C) In another case, a preoperative lateral radiograph of a middle-aged man reveals secondary osteoarthritis caused by a calcaneonavicular bar. (D) This patient was also treated with triple arthrodesis.

ADULT Flatfoot Iatrogenic / Post-traumatic / Arthritic

SIGNIFICANT HISTORY (1)

- Family History
- Review of Systems
- Previous Surgery
- History of Trauma
- Associated Conditions: RA

Diagnostic Imaging (3)

- Internal Hardware
- Arthrosis
- Malalignment
- CT/MR

SIGNIFICANT FINDINGS (2)

- Clinical Maneuvers
- Localized Pain & Swelling
- +/- Joint Effusion
- Antalgic Gait
- Previous Trauma / Surgery

Refer to Pathway #1

DIAGNOSIS
(4)

INITIAL TREATMENT OPTIONS (5)

- Activity Modifications
- Weight Loss
- Orthotic Management
- Immobilization
- Antiinflammatory Medications
- Physical Therapy
- Shoe Gear Modifications

Clinical Response
(6)

Satisfactory

Continue NonSurgical Options (7)

Unsatisfactory

SURGICAL OPTIONS (8)

- Osteotomies
- Arthrodesis
- Adjunctive Procedures
TAL or Gasctroc Recession
Tendon Transfer

Consider Surgery, if symptoms resume

Adjunctive Measures (9)

- Orthotic Management

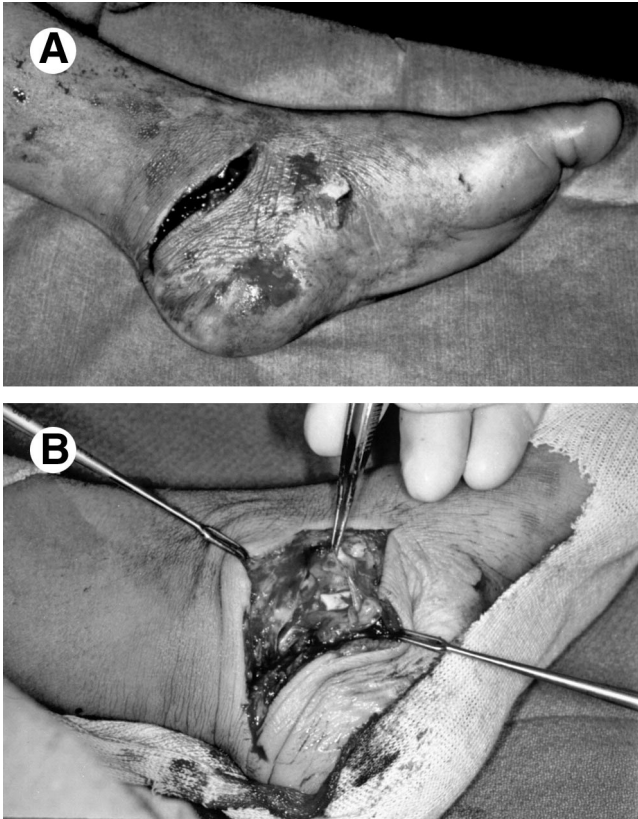


FIGURE 13 Flatfoot deformities may result from traumatic causes. (A) This clinical photograph shows a deep laceration just inferior to the medial malleolus. The tibialis posterior tendon was lacerated and the patient underwent surgical repair, as shown in this (B) intraoperative image.

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 (Path-

way 1, Node 3). Malunited or malreduced calcaneal fractures may show a loss of calcaneal height and decreased Böhler's angle (Fig 14). 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 (eg, 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 (ie, 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.

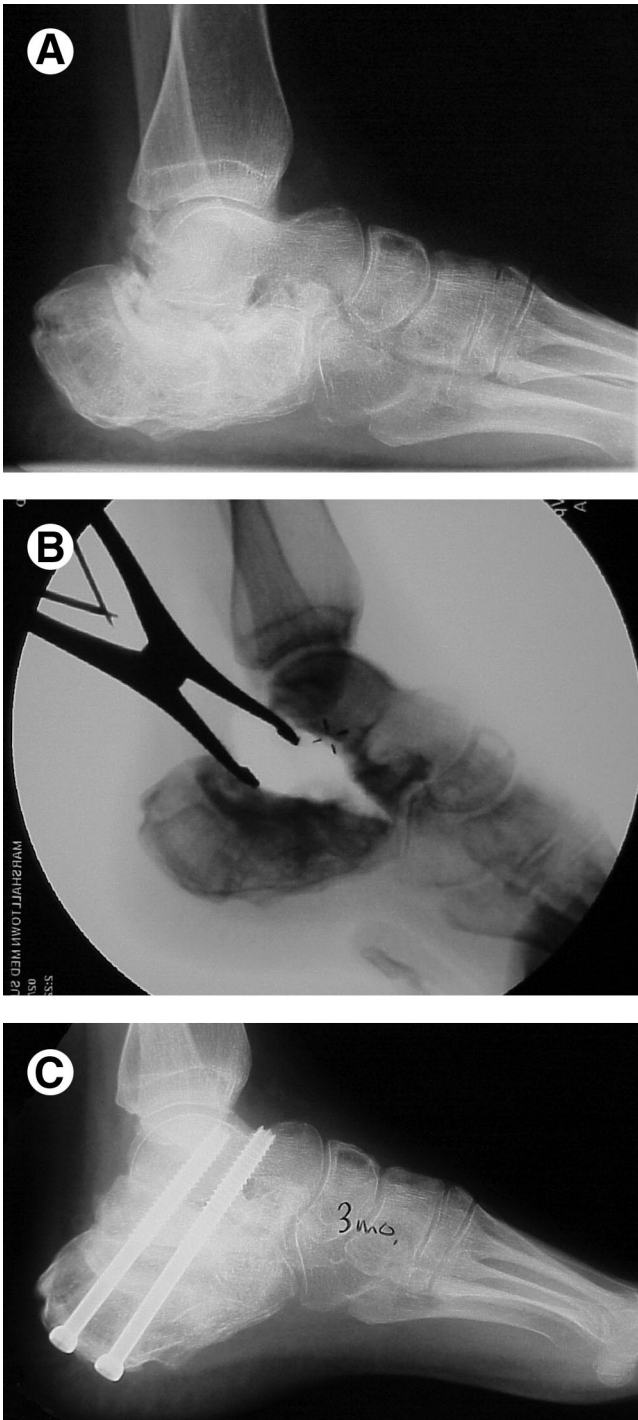


FIGURE 14 This patient sustained a joint depression calcaneal fracture, resulting in a painful flatfoot deformity, shown in (A) this lateral radiograph. The foot was reconstructed with a bone graft subtalar fusion. (B) An intraoperative image shows the technique of distracting the subtalar joint, which was followed with rigid internal fixation with 2 large cannulated screws, shown on (C) the postoperative lateral radiograph.

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 (Fig 14). When treating deformity secondary to calcaneal fractures, extra-articular 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 (180–191). 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 (Figs 15 and 16). The prevalence of Charcot foot involvement ranges from 0.15% of all patients with diabetes to 29% of patients with both diabetes and neuropathy (186).

Significant History (Pathway 6, Node 1)

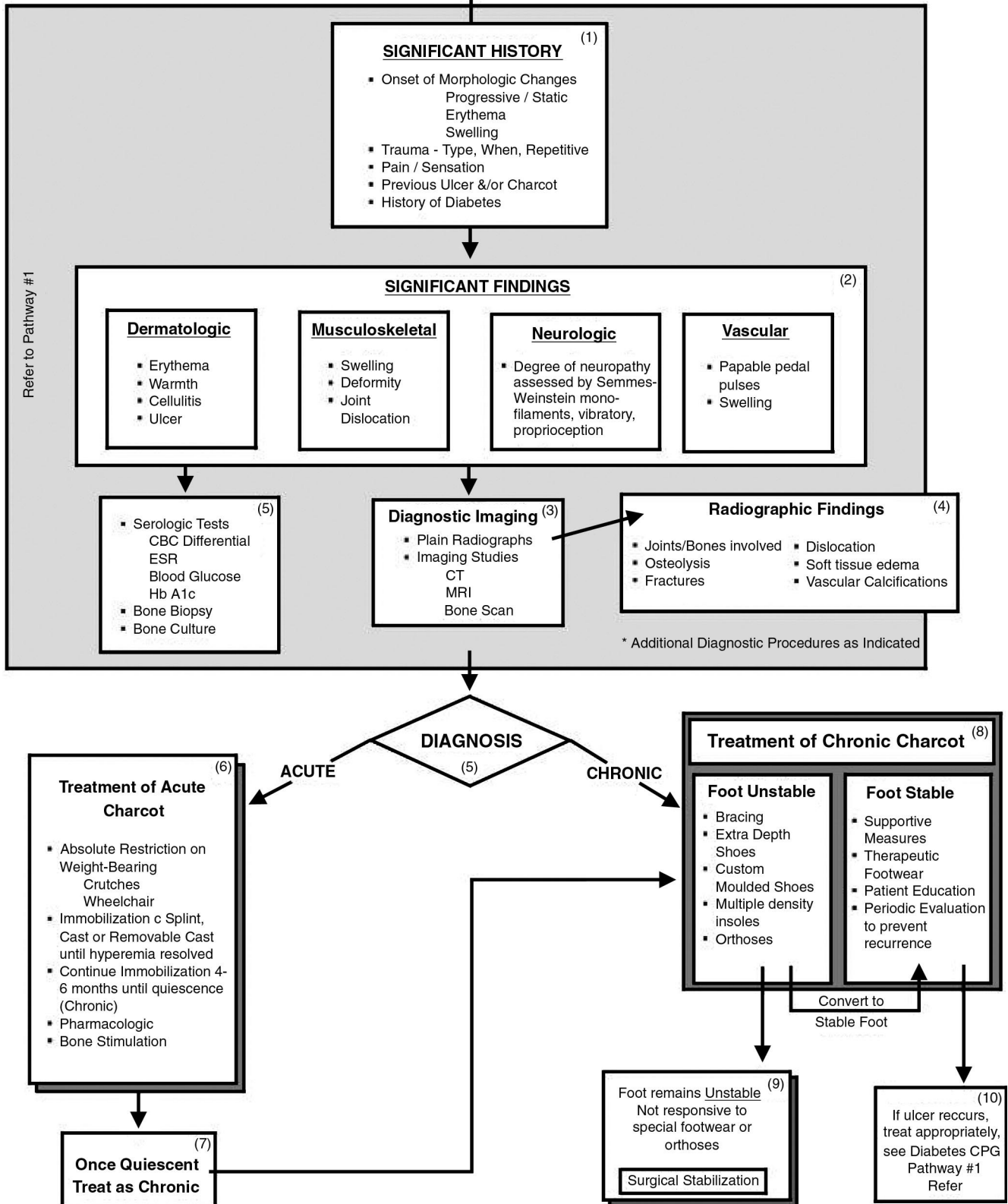
Charcot joint involvement is most often seen in obese patients with longstanding diabetes who have peripheral neuropathy. Trauma may play a role in development, although, more commonly, the repetitive stress of daily weightbearing is enough to precipitate an episode (192, 193).

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 (194–196).

Significant Findings (Pathway 6, Node 2)

The acute Charcot foot has a dramatically different presentation than the chronic Charcot foot. The acute Charcot

CHARCOT FOOT



PATHWAY 6



FIGURE 15 Charcot foot episodes result in flatfoot deformities wherein the breach occurs at the midtarsal or tarsometatarsal articulations. This patient has involvement predominantly at the Lis Franc joints, as shown on preoperative (A) AP and (B) lateral radiographs. The patient underwent Charcot reconstruction with intertarsal and tarsometatarsal arthrodesis. Postoperative (C) AP and (D) lateral radiographs show realignment of the osseous segments. The tendo-Achilles must also be lengthened as necessary.



FIGURE 16 Charcot collapse of the foot may occur at varied locations. This case shows deformity caused by a neuropathic ankle fracture dislocation with tarsometatarsal involvement, as revealed in preoperative (A) AP and (B) lateral radiographs. The patient was treated with tibiotalar calcaneal arthrodesis by using an intramedullary nail. Postoperative (C) AP and (D) lateral radiographs show impressive restoration of the pedal architecture and use of an internal bone stimulator.

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 (184, 197–202). 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) (203). 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 (182).

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 (197). 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 (201). Advanced imaging studies play a clarifying role. Bone scans, computed tomography, and MRI studies have been advocated to help differentiate the diagnosis (196, 201, 204, 205).
- 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 (190).

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 (eg, joint effusion, pathologic fracture, osteolysis, dislocation) may or may not be present. The process may occur spontaneously, following trauma, or after surgery (206–209).
- Chronic Charcot foot. The patient with chronic Charcot foot typically presents with overt deformity and resultant contiguous ulceration. The chronic Charcot foot is usu-

ally 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 (210). 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 (211). In some cases in which deformity is present, surgical management of the acute Charcot foot may be indicated.

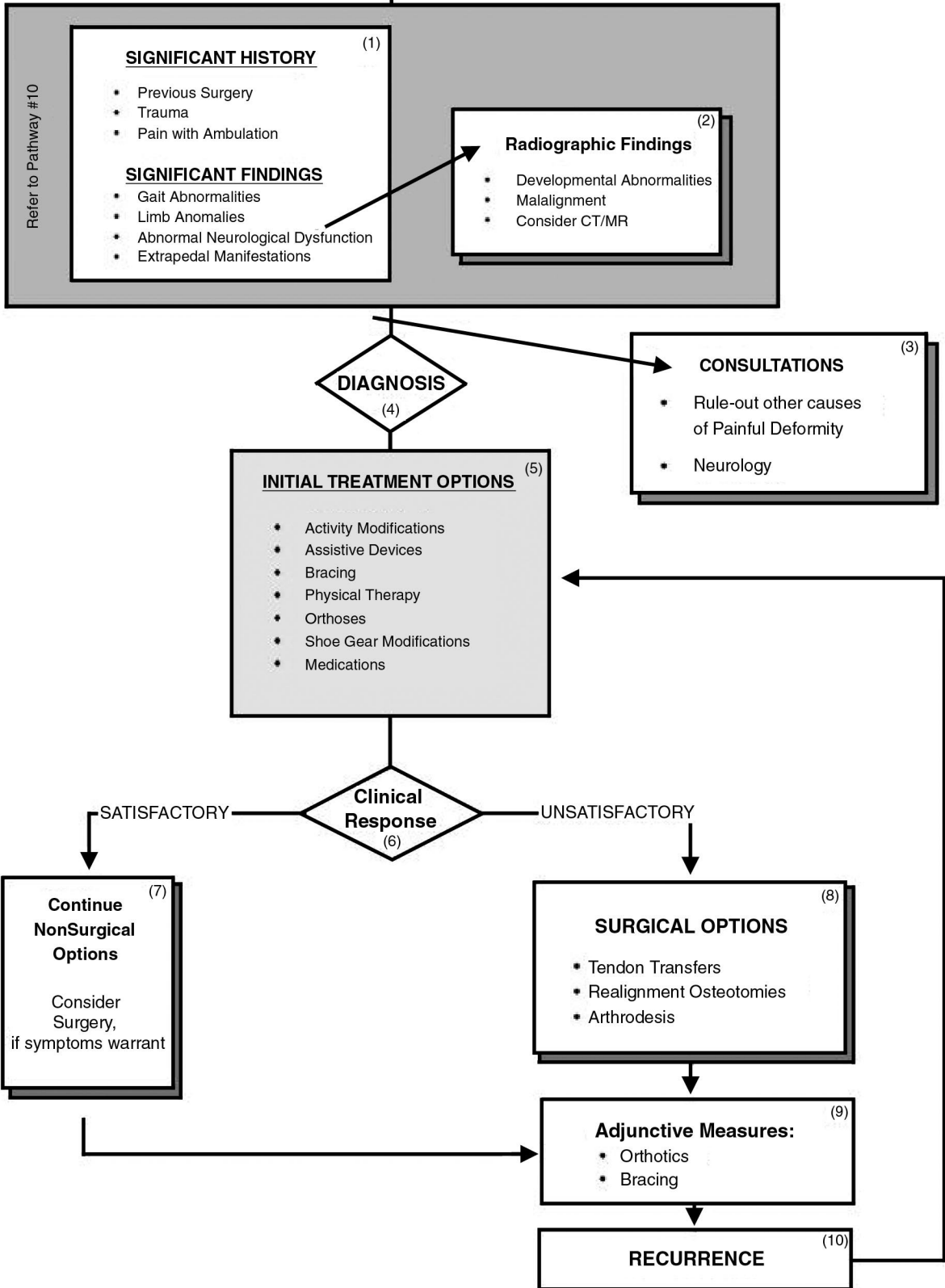
Treatment of Chronic Charcot (Pathway 6, Nodes 7–9)

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 *Diabetic Foot Disorders: A Clinical Practice Guideline* (190). 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 (212–216). These measures, combined with patient education, are important to prevent recurrence (217). In the presence of a nonhealing ulceration, isolated exostectomy may be considered (218).
- 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 (189, 219–222) (Node 9) (Figs 15 and 16).

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 (217, 223).

ADULT Neuromuscular Flatfoot



PATHWAY 7



FIGURE 17 Flatfeet are also associated with congenital and developmental abnormalities. This is an adult with spina bifida whose (A) clinical presentation and (B and C) radiographs show a significant flatfoot deformity. The patient has limited function of his lower extremities, requiring crutches for gait assistance and use of depth orthopedic shoes.

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 (Fig 17).

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).

References

1. Chang FM. The flexible flatfoot. Instr Course Lect 37:109–110, 1988.
2. Jones LJ, Todd WF. Abnormal biomechanics of flatfoot deformities and related theories of biomechanical development. Clin Podiatr Med Surg 6:511–520, 1989.
3. Huang CK, Kitaoka HB, An KN, Chao EY. Biomechanical evaluation of longitudinal arch stability. Foot Ankle 14:353–357, 1993.
4. Kitaoka HB, Ahn TK, Luo ZP, An KN. Stability of the arch of the foot. Foot Ankle Int 18:644–648, 1997.
5. Chu IT, Myerson MS, Parks BG. Experimental flatfoot model: the contribution of dynamic loading. Foot Ankle Int 22:220–225, 2001.
6. Gray EG, Basmajian JV. Electromyography and cinematography of leg and foot (“normal” and flat) during walking. Anat Rec 161:1–15, 1968.
7. Gray ER. The role of leg muscles in variations of the arches in normal and flat feet. Phys Ther 49:1084–1088, 1969.
8. Suzuki N. An electromyographic study of the role of muscles in arch support of the normal and flat foot. Nagoya Med J 17:57–79, 1972.
9. Ananthakrishnan DR, Ching R, Tencer A, Hansen ST Jr. Subluxation of the talocalcaneal joint in adults who have symptomatic flatfoot. J Bone Joint Surg 81A:1147–1154, 1999.
10. Hansen ST Jr. Progressive symptomatic flatfoot (lateral peritalar subluxation). In *Functional Reconstruction of the Foot and Ankle*, pp 195–207, edited by ST Hansen, Lippincott, Williams & Wilkins, Philadelphia, 2000.
11. Gould N, Schneider W, Takamara A. Epidemiological survey of foot problems in the continental United States: 1978–1979. Foot Ankle 1:8–10, 1980.
12. Page JC. Symptomatic flatfoot. Etiology and diagnosis. J Am Podiatr Assoc 73:393–399, 1983.
13. Bordin D, De Giorgi G, Mazzocco G, Rigon F. Flat and cavus foot, indexes of obesity and overweight in a population of primary-school children. Minerva Pediatr 53:7–13, 2001.
14. Ferciot CF. The etiology of developmental flatfoot. Clin Orthop 85:7–10, 1972.
15. Harris R, Beath T. Hypermobil flatfoot with short tendo Achillis. J Bone Joint Surg 30A:116–138, 1948.
16. Staheli LT. Planovalgus foot deformity. Current status. J Am Podiatr Med Assoc 89:94–99, 1999.
17. Cowan DN, Jones BH, Robinson JR. Foot morphologic characteristics and risk of exercise-related injury. Arch Fam Med 2:773–777, 1993.
18. Rudzki SJ. Injuries in Australian Army recruits. Part III: The accuracy of a pretraining orthopedic screen in predicting ultimate injury outcome. Mil Med 162:481–483, 1997.
19. Jahss MH. Spontaneous rupture of the tibialis posterior tendon: clinical findings, tenographic studies, and a new technique of repair. Foot Ankle 3:158–166, 1982.
20. Myerson MS, Solomon G, Shereff M. Posterior tibial tendon dysfunction: its association with seronegative inflammatory disease. Foot Ankle 9:219–224, 1989.
21. Botte RR. An interpretation of the pronation syndrome and foot types of patients with low back pain. J Am Podiatr Assoc 71:243–253, 1981.
22. Jack EA. Naviculo-cuneiform fusion in the treatment of flatfoot. J Bone Joint Surg 13:773–783, 1931.
23. Hicks JH. Mechanics of the foot: plantar aponeurosis and the arch. J Anat 88:25–31, 1954.
24. Johnson KA, Strom DE. Tibialis posterior tendon dysfunction. Clin Orthop 239:196–206, 1989.
25. Borrelli AH. Planar dominance. A major determinant in flatfoot stabilization. Clin Podiatr Med Surg 16:407–421, 1999.

26. Green DR, Carol A. Planal dominance. *J Am Podiatr Assoc* 74:98–103, 1983.
27. Saltzman CL. The hindfoot alignment view. *Foot Ankle* 16:572–576, 1995.
28. Yao L, Gentili A, Cracchiolo A. MR imaging findings in spring ligament insufficiency. *Skeletal Radiol* 28:245–250, 1999.
29. Deutsch AL, Resnick D, Campbell G. Computed tomography and bone scintigraphy in the evaluation of tarsal coalition. *Radiology* 144:137–140, 1982.
30. Sarno RC, Carter BL, Bankoff MS, Semine MC. Computed tomography in tarsal coalition. *J Comput Assist Tomogr* 8:1155–1160, 1984.
31. Herzenberg JE, Goldner JL, Martinez S, Silverman PM. Computerized tomography of talocalcaneal tarsal coalition: a clinical and anatomic study. *Foot Ankle* 6:273–288, 1986.
32. Smith DK, Gilula LA, Sullivan RC. Subtalar arthrosis: evaluation with CT. *AJR Am J Roentgenol* 154:559–562, 1990.
33. Maurice HD, Newman JH, Watt I. Bone scanning of the foot for unexplained pain. *J Bone Joint Surg* 69B:448–452, 1987.
34. Sneyers CJ, Lysens R, Feys H, Andries R. Influence of malalignment of feet on the plantar pressure pattern in running. *Foot Ankle Int* 16:624–632, 1995.
35. Phillipson A, Klenerman L. Footprints and arches. *J Bone Joint Surg* 75B:163, 1993.
36. Tareco JM, Miller NH, MacWilliams BA, Michelson JD. Defining flatfoot. *Foot Ankle Int* 20:456–460, 1999.
37. Miller SJ. Collapsing pes valgoplanus (flexible flatfoot). In *Principles and Practice of Podiatric Medicine*, pp 893–929, edited by LA Levy and VJ Hetherington, Churchill Livingstone, New York, 1989.
38. Henceroth WD II, Deyerle WM. The acquired unilateral flatfoot in the adult: some causative factors. *Foot Ankle* 2:304–308, 1982.
39. Mahan KT, Flanigan KP. Pathologic pes valgus disorders: Part 1. Pes plano valgus deformity. In *McGlamry's Comprehensive Textbook of Foot Surgery*, volume 1, pp 799–861, edited by SJ Miller, Lippincott Williams & Wilkins, Philadelphia, 2002.
40. Miller SJ. End-stage flatfoot. Diagnosis and conservative and surgical management. *J Am Podiatr Med Assoc* 77:42–45, 1987.
41. Waller JF. Physiology of the foot and the biomechanics of the flexible flat foot. *Ona J* 5:101–103, 1978.
42. Root ML. Planovalgus foot deformity revisited. *J Am Podiatr Med Assoc* 89:268–269, 1999.
43. Yale JF. The conservative treatment of adult flexible flatfoot. *Clin Podiatr Med Surg* 6:555–560, 1989.
44. Mereday C, Dolan CM, Lusskin R. Evaluation of the University of California Biomechanics Laboratory shoe insert in “flexible” pes planus. *Clin Orthop* 82:45–58, 1972.
45. Bleck EE, Berzins UJ. Conservative management of pes valgus with plantar flexed talus, flexible. *Clin Orthop* 122:85–94, 1977.
46. Braun S. [Orthoses for the foot]. *Rev Prat* 31:1087–1088, 1093–1094, 1097–1098, 1981.
47. Otman S, Basgoze O, Gokce-Kutsal Y. Energy cost of walking with flat feet. *Prosthet Orthot Int* 12:73–76, 1988.
48. Taylor TL. Idiopathic flexible flatfoot in the adolescent. *Clin Podiatr Med Surg* 6:537–553, 1989.
49. Blake RL, Ferguson H. Foot orthosis for the severe flatfoot in sports. *J Am Podiatr Med Assoc* 81:549–555, 1991.
50. Jay RM, Schoenhaus HD. Hyperpronation control with a dynamic stabilizing innersole system. *J Am Podiatr Med Assoc* 82:149–153, 1992.
51. Kirby KA. The medial heel skive technique. Improving pronation control in foot orthoses. *J Am Podiatr Med Assoc* 82:177–188, 1992.
52. Jay RM, Schoenhaus, HD, Seymour C, Gamble S. The Dynamic Stabilizing Innersole System (DSIS): the management of hyperpronation in children. *J Foot Ankle Surg* 34:124–131, 1995.
53. Leung AK, Mak AF, Evans JH. Biomedical gait evaluation of the immediate effect of orthotic treatment for flexible flat foot. *Prosthet Orthot Int* 22:25–34, 1998.
54. Kuhn DR, Shibley NJ, Austin WM, Yochum TR. Radiographic evaluation of weight-bearing orthotics and their effect on flexible pes planus. *J Manipulative Physiol Ther* 22:221–226, 1999.
55. Sobel E, Levitz SJ, Caselli MA. Orthoses in the treatment of rearfoot problems. *J Am Podiatr Med Assoc* 89:220–233, 1999.
56. Beck EL, McGlamry ED. Modified Young tendosuspension technique for flexible flatfoot. Analysis of rationale and results: a preliminary report on 20 operations. *J Am Podiatr Assoc* 63:582–604, 1973.
57. Howey TD, Hoversten DL. Evaluation of the Kidner procedure for prehallux. *S D J Med* 38:21–24, 1985.
58. Deland JR, Arnoczky SP, Thompson FM. Adult acquired flatfoot deformity at the talonavicular joint: reconstruction of the spring ligament in an in vitro model. *Foot Ankle* 13:327–332, 1992.
59. Mahan KT. Pes planovalgus deformity. In *Comprehensive Textbook of Foot Surgery*, ed 2, vol 1, pp 769–817, edited by ED McGlamry, A Banks, and M Downey, Williams & Wilkins, Philadelphia, 1992.
60. Prichasuk S, Sinphurmsukskul O. Kidner procedure for symptomatic accessory navicular and its relation to pes planus. *Foot Ankle Int* 16:500–503, 1995.
61. Kitaoka HB, Luo ZP, An KN. Effect of the posterior tibial tendon on the arch of the foot during simulated weightbearing: biomechanical analysis. *Foot Ankle Int* 18:43–46, 1997.
62. Bohay RD. Subtalar arthrodesis versus flexor digitorum longus tendon transfer for severe flatfoot deformity: an in vitro biomechanical analysis (letter). *Foot Ankle Int* 19:346, 1988.
63. Toolan BC, Sangeorzan BJ, Hansen ST Jr. Complex reconstruction for the treatment of dorsolateral peritalar subluxation of the foot. Early results after distraction arthrodesis of the calcaneocuboid joint in conjunction with stabilization of, and transfer of the flexor digitorum longus tendon to, the midfoot to treat acquired pes planovalgus in adults. *J Bone Joint Surg* 81A:1545–1560, 1999.
64. Deland JT. The adult acquired flatfoot and spring ligament complex. Pathology and implications for treatment. *Foot Ankle Clin* 6:129–135, vii, 2001.
65. Neufeld SK, Myerson MS. Complications of surgical treatments for adult flatfoot deformities. *Foot Ankle Clin* 6:179–191, 2001.
66. Dollard MD, Marcinko DE, Lazerson A, Elleby DH. The Evans calcaneal osteotomy for correction of flexible flatfoot syndrome. *J Foot Surg* 23:291–301, 1984.
67. Mahan KT, McGlamry ED. Evans calcaneal osteotomy for flexible pes valgus deformity. A preliminary study. *Clin Podiatr Med Surg* 4:137–151, 1987.
68. Hintermann B, Valderrabano B, Kundert HP. Lengthening of the lateral column and reconstruction of the medial soft tissue for treatment of acquired flatfoot deformity associated with insufficiency of the posterior tibial tendon. *Foot Ankle Int* 20:622–629, 1999.
69. Thomas RL, Wells BC, Garrison RL, Prada SA. Preliminary results comparing two methods of lateral column lengthening. *Foot Ankle Int* 22:107–119, 2001.
70. Jacobs AM, Oloff L, Visser HJ. Calcaneal osteotomy in the management of flexible and nonflexible flatfoot deformity: a preliminary report. *J Foot Surg* 20:57–66, 1981.
71. Sangeorzan BJ, Mosca V, Hansen ST Jr. Effect of calcaneal lengthening on relationships among the hindfoot, midfoot, and forefoot. *Foot Ankle* 14:136–141, 1993.
72. Horton GA, Myerson MS, Parks PB, Park YN. Effect of calcaneal osteotomy and lateral column lengthening on the plantar fascia: a biomechanical investigation. *Foot Ankle Int* 19:370–373, 1998.
73. Phillips GE. A review of elongation of os calcis for flat feet. *J Bone Joint Surg* 65B:15–18, 1983.

74. Cooper PS, Nowak MD, Shaer J. Calcaneocuboid joint pressures with lateral column lengthening (Evans) procedure. *Foot Ankle Int* 18:199–205, 1997.
75. McCormack AP, Niki H, Kiser P, Tencer AF, Sangeorzan BJ. Two reconstructive techniques for flatfoot deformity comparing contact characteristics of the hindfoot joints. *Foot Ankle Int* 19:452–461, 1998.
76. Momberger N, Morgan JM, Bachus KN, West JR. Calcaneocuboid joint pressure after lateral column lengthening in a cadaveric planovalgus deformity model. *Foot Ankle Int* 21:730–735, 2000.
77. Jacobs AM, Geistler P. Posterior calcaneal osteotomy. Effect, technique, and indications. *Clin Podiatr Med Surg* 8:647–657, 1991.
78. Myerson MS, Corrigan J, Thompson F, Schon LC. Tendon transfer combined with calcaneal osteotomy for treatment of posterior tibial tendon insufficiency: a radiological investigation. *Foot Ankle Int* 16:712–718, 1995.
79. Myerson MS, Corrigan J. Treatment of posterior tibial tendon dysfunction with flexor digitorum longus tendon transfer and calcaneal osteotomy. *Orthopedics* 19:383–388, 1996.
80. Catanzariti AR, Lee MS, Mendicino RW. Posterior calcaneal displacement osteotomy for adult acquired flatfoot. *J Foot Ankle Surg* 39:2–14, 2000.
81. Arangio GA, Salathe EP. Medial displacement calcaneal osteotomy reduces the excess forces in the medial longitudinal arch of the flat foot. *Clin Biomech (Bristol, Avon)* 16:535–539, 2001.
82. Den Hartog BD. Flexor digitorum longus transfer with medial displacement calcaneal osteotomy. Biomechanical rationale. *Foot Ankle Clin* 6:67–76, vi, 2001.
83. Nyska M, Parks BG, Chu IT, Myerson MS. The contribution of the medial calcaneal osteotomy to the correction of flatfoot deformities. *Foot Ankle Int* 22:278–282, 2001.
84. Hadfield MH, Snyder JW, Liacouras PC, Owen JR, Wayne JS, Adelaar RS. Effects of medializing calcaneal osteotomy on Achilles tendon lengthening and plantar foot pressures. *Foot Ankle Int* 24:523–529, 2003.
85. Guyton GP, Jeng C, Krieger LE, Mann RA. Flexor digitorum longus transfer and medial displacement calcaneal osteotomy for posterior tibial tendon dysfunction: a middle-term clinical follow-up. *Foot Ankle Int* 22:627–632, 2001.
86. Hockenbury RT, Sammarco GJ. Medial sliding calcaneal osteotomy with flexor hallucis longus transfer for the treatment of posterior tibial tendon insufficiency. *Foot Ankle Clin* 6:569–581, 2001.
87. Wacker JT, Hennessy MS, Saxby TS. Calcaneal osteotomy and transfer of the tendon of flexor digitorum longus for stage-II dysfunction of tibialis posterior. Three- to five-year results. *J Bone Joint Surg* 84B:4–58, 2002.
88. Hiller L, Pinney SJ. Surgical treatment of acquired flatfoot deformity: what is the state of practice among academic foot and ankle surgeons in 2002? *Foot Ankle Int* 24:701–705, 2003.
89. Myerson MS, Fortin PT, Cunningham B. Changes in tibiotalar contact with calcaneal osteotomy. *Trans Am Acad Orthop Surg* 61:149, 1994.
90. Fairbank A, Myerson MS, Fortin P, Yu-Yahiro J. The effect of calcaneal osteotomy on contact characteristics of the tibiotalar joint. *Foot Ankle* 16:137–142, 1995.
91. Allon SM. Effect of medial displacement calcaneal osteotomy on ankle kinematics in a cadaver model. *Foot Ankle Int* 19:574–575, 1998.
92. Michelson JD, Mizel M, Jay P, Schmidt G. Effect of medial displacement calcaneal osteotomy on ankle kinematics in a cadaver model. *Foot Ankle Int* 19:132–136, 1998.
93. Steffensmeier SJ, Saltzman CL, Brown RD. Effects of medial and lateral displacement calcaneal osteotomies on tibiotalar joint contact stresses. *J Orthop Res* 14:980–985, 1996.
94. Frankel JP, Turf RM, Kuzmicki LM. Double calcaneal osteotomy in the treatment of posterior tibial tendon dysfunction. *J Foot Ankle Surg* 34:254–261, 1995.
95. Pomeroy GC, Manoli A II. A new operative approach for flatfoot secondary to posterior tibial tendon insufficiency: a preliminary report. *Foot Ankle Int* 18:206–212, 1997.
96. Pomeroy GC, Pike RH, Beals TC, Manoli A II. Acquired flatfoot in adults due to dysfunction of the posterior tibial tendon. *J Bone Joint Surg* 81A:1173–1182, 1999.
97. Moseir-LaClair S, Pomeroy G, Manoli A II. Intermediate follow-up on the double osteotomy and tendon transfer procedure for stage II posterior tibial tendon insufficiency. *Foot Ankle Int* 22:283–291, 2001.
98. Johnston WB. A literary review of calcaneal osteotomies in the treatment of valgus deformities. *J Foot Surg* 21:139–142, 1982.
99. Marcinko DE, Lazerson A, Elleby DH. Silver calcaneal osteotomy for flexible flatfoot: a retrospective preliminary report. *J Foot Surg* 23:191–198, 1984.
100. Garelli R. Osteotomy of the calcaneum in the treatment of idiopathic valgus foot. *Ital J Orthop Traumatol* 12:53–60, 1986.
101. Weil LS Jr, Roukis TS. The calcaneal scarf osteotomy: operative technique. *J Foot Ankle Surg* 40:178–182, 2001.
102. Cotton FJ. Foot statistics and surgery. *N Engl J Med* 214:353–362, 1936.
103. Jacobs AM, Oloff LM. Surgical management of forefoot supinatus in flexible flatfoot deformity. *J Foot Surg* 23:410–419, 1984.
104. Bacardi BE, Frankel JP. Biplane cuneiform osteotomy for juvenile metatarsus primus varus. *J Foot Surg* 6:472–478, 1986.
105. Maxwell J, Nakra A, Ashley C. Use of the Maxwell-Brancheau Arthroereisis Implant for the correction of posterior tibial tendon dysfunction. *Tech Orthop* 15:183–196, 2000.
106. Subotnick SI. The subtalar joint lateral extra-articular arthroereisis: a preliminary report. *J Am Podiatry Assoc* 64:701–711, 1974.
107. Subotnick SI. The subtalar joint lateral extra-articular arthroereisis: a follow-up report. *J Am Podiatr Assoc* 67:157–171, 1977.
108. Smith RD, Rappaport MJ. Subtalar arthroereisis. A four-year follow-up study. *J Am Podiatr Assoc* 73:356–361, 1983.
109. Smith SD, Millar EA. Arthroereisis by means of a subtalar polyethylene peg implant for correction of hindfoot pronation in children. *Clin Orthop* 181:15–23, 1983.
110. Lundeen RO. The Smith STA-peg operation for hypermobile pes planovalgus in children. *J Am Podiatr Med Assoc* 75:177–183, 1985.
111. Langford JH, Bozof H, Horowitz BD. Subtalar arthroereisis. Valente procedure. *Clin Podiatr Med Surg* 4:153–161, 1987.
112. Vogler HW. Subtalar joint blocking operations for pathological pronation syndromes. In *Textbook of Foot Surgery*, pp 447–465, edited by ED McGlamry. Williams and Wilkins, Baltimore, 1987.
113. Viladot A. Surgical treatment of the child's flatfoot. *Clin Orthop*:34–38, 1992.
114. Maxwell JR, Carro A, Sun C. Use of the Maxwell-Brancheau arthroereisis implant for the correction of posterior tibial tendon dysfunction. *Clin Podiatr Med Surg* 16:479–489, 1999.
115. Smith PA, Millar EA, et al. Sta-Peg arthroereisis for treatment of the planovalgus foot in cerebral palsy. *Clin Podiatr Med Surg* 17:459–469, 2000.
116. Viladot R, Pons M, Alvarex F, Omana J. Subtalar arthroereisis for posterior tibial tendon dysfunction: a preliminary report. *Foot Ankle* 24:600–606, 2003.
117. Christensen JC, Campbell N, DiNucci K. Closed kinetic chain tarsal mechanics of subtalar joint arthroereisis. *J Am Podiatr Med Assoc* 86:467–473, 1996.
118. Kuwada GT, Dockery GL. Complications following traumatic incidents with STA-peg procedures. *J Foot Surg* 27:236–239, 1988.
119. Roye DP Jr, Raimondo RA. Surgical treatment of the child's and

- adolescent's flexible flatfoot. *Clin Podiatr Med Surg* 17:515-530, vii-viii, 2000.
120. Catanzariti AR. Medial column stabilization. *Clin Podiatr Med Surg* 8:667-692, 1991.
 121. Catanzariti AR. Modified medial column arthrodesis. *J Foot Ankle Surg* 32:80-188, 1993.
 122. Lepow GM, Sands MR. Correction of flexible pes planus deformity: medial column stabilization procedure. *Clin Podiatr Med Surg* 6:577-584, 1989.
 123. Mothershed RA, Stapp MD, Smith TF. Talonavicular arthrodesis for correction of posterior tibial tendon dysfunction. *Clin Podiatr Med Surg* 16:501-526, 1999.
 124. Harper MC, Tisdell CL. Talonavicular arthrodesis for the painful adult acquired flatfoot. *Foot Ankle Int* 17:658-661, 1996.
 125. Mann RA. Talonavicular arthrodesis for the painful adult acquired flatfoot. *Foot Ankle Int* 18:375-376, 1997.
 126. Harper MC. Talonavicular arthrodesis for the acquired flatfoot in the adult. *Clin Orthop* 365:65-68, 1999.
 127. Fortin PT. Posterior tibial tendon insufficiency. Isolated fusion of the talonavicular joint. *Foot Ankle Clin* 6:137-151, vii-viii, 2001.
 128. Thomas JL, Moeini R, Soileau R. The effects on subtalar contact and pressure following talonavicular and midtarsal joint arthrodesis. *J Foot Ankle Surg* 39:78-88, 2000.
 129. Kitaoka HB, Kura H, Luo ZP, An KN. Calcaneocuboid distraction arthrodesis for posterior tibial tendon dysfunction and flatfoot: a cadaveric study. *Clin Orthop* 381:241-247, 2000.
 130. Sands A, Early J, Harrington RM, Tencer AF, Ching RP, Sangeorzan BJ. Effect of variations in calcaneocuboid fusion technique on kinematics of the normal hindfoot. *Foot Ankle Int* 19:19-25, 1998.
 131. O'Malley MJ, Deland JT, Lee KT. Selective hindfoot arthrodesis for the treatment of adult acquired flatfoot deformity: an in vitro study. *Foot Ankle Int* 16:411-417, 1995.
 132. Mann RA, Beaman DN. Double arthrodesis in the adult. *Clin Orthop* 365:74-80, 1999.
 133. Thomas FB. Arthrodesis of the subtalar joint. *J Bone Joint Surg* 49B:93-97, 1967.
 134. Dennyson W G, Fulford GE. Subtalar arthrodesis by cancellous grafts and metallic internal fixation. *J Bone Joint Surg* 58B:507-510, 1976.
 135. Monson R, Gibson DA. Long-term follow-up of triple arthrodesis. *Can J Surg* 21:249-251, 1978.
 136. Judet J, Judet H. [Flat foot: treatment by sub-talar arthrodesis with reposition of the talus calcaneum (author's transl)]. *Presse Med* 8:3969-3971, 1979.
 137. Graves SC, Mann RA, Graves KO. Triple arthrodesis in older adults. Results after long-term follow-up. *J Bone Joint Surg* 75A:355-362, 1993.
 138. Sennara H. Triple arthrodesis: a modified new technic. *Clin Orthop* 83:237-240, 1972.
 139. Sammarco GJ. Technique of triple arthrodesis in treatment of symptomatic pes planus. *Orthopedics* 11:1607-1610, 1988.
 140. Vogler HW. Triple arthrodesis as a salvage for end-stage flatfoot. *Clin Podiatr Med Surg* 6:591-604, 1989.
 141. Mann RA, Baumgarten M. Subtalar fusion for isolated subtalar disorders. Preliminary report. *Clin Orthop* 226:260-265, 1988.
 142. Mann RA, Beaman, DN, Horton GA. Isolated subtalar arthrodesis. *Foot Ankle Int* 19:511-519, 1998.
 143. Laughlin TJ, Payette CR. Triple arthrodesis and subtalar joint arthrodesis. For the treatment of end-stage posterior tibial tendon dysfunction. *Clin Podiatr Med Surg* 16: 527-555, 1999.
 144. Easley ME, Trnka HJ, Schon LC, Myerson MS. Isolated subtalar arthrodesis. *J Bone Joint Surg* 82A:613-624, 2000.
 145. Miller OL. A plastic flatfoot operation. *J Bone Joint Surg* 9:84-91, 1927.
 146. Hoke M. An operation for the correction of extremely relaxed flatfeet. *J Bone Joint Surg* 13:773-783, 1931.
 147. Seymour N. The late results of naviculo-cuneiform fusion. *J Bone Joint Surg* 49B:558-559, 1967.
 148. Giannestras NJ. The congenital rigid flatfoot. Its recognition and treatment in infants. *Orthop Clin North Am* 4:49-66, 1973.
 149. Lovell WW, Price CT, Meehan PL. The foot. In *Pediatric Orthopedics*, pp 946-950, edited by WW Lovell and RB Winter, JB Lippincott, Philadelphia, 1978.
 150. Duncan JW, Lovell WW. Modified Hoke-Miller flatfoot procedure. *Clin Orthop* 181:24-27, 1983.
 151. Chi TD, Toolan BC, Sangeorzan BJ, Hansen ST Jr. The lateral column lengthening and medial column stabilization procedures. *Clin Orthop* 365:81-90, 1999.
 152. Churchill RS, Sfera JJ. Posterior tibial tendon insufficiency. Its diagnosis, management, and Treatment. *Am J Orthop* 27:339-347, 1998.
 153. Beals TC, Pomeroy GC, Manoli A II. Posterior tendon insufficiency: diagnosis and treatment. *J Am Acad Orthop Surg* 7:112-118, 1999.
 154. Jahss MH. Tendon disorders of the foot and ankle. In *Disorders of the Foot and Ankle*, pp 1461-1513, edited by MH Jahss, WB Saunders, Philadelphia, 1991.
 155. Mueller TJ. Acquired flatfoot secondary to tibialis posterior dysfunction: biomechanical aspects. *J Foot Surg* 30:2-11, 1991.
 156. Conti SF, Michelson J, Jahss M. Clinical significance of magnetic resonance imaging in preoperative planning for reconstruction of posterior tibial tendon ruptures. *Foot Ankle* 13:208-214, 1992.
 157. Myerson MS. Adult acquired flatfoot deformity. *J Bone Joint Surg* 78A:780-798, 1996.
 158. Funk DA, Cass JR, Johnson KA. Acquired adult flat foot secondary to posterior tibial-tendon pathology. *J Bone Joint Surg* 68A:95-102, 1986.
 159. LaFontaine JJ, Campbell NA. Calcaneal osteotomy for the treatment of posterior tibial tendon dysfunction. *Clin Podiatr Med Surg* 16:491-499, 1999.
 160. Sammarco GJ, Hockenbury RT. Treatment of stage II posterior tibial tendon dysfunction with flexor hallucis longus transfer and medial displacement calcaneal osteotomy. *Foot Ankle Int* 22:305-312, 2001.
 161. Kitaoka HB, Luo ZP, An KN. Subtalar arthrodesis versus flexor digitorum longus tendon transfer for severe flatfoot deformity: an in vitro biomechanical analysis. *Foot Ankle Int* 18:710-715, 1997.
 162. Kitaoka HB, Patzer GL. Subtalar arthrodesis for posterior tibial tendon dysfunction and pes planus. *Clin Orthop* 345:187-194, 1997.
 163. Johnson JE, Cohen BE, DiGiovanni BF, Lamdan R. Subtalar arthrodesis with flexor digitorum longus transfer and spring ligament repair for treatment of posterior tibial tendon insufficiency. *Foot Ankle Int* 21:722-729, 2000.
 164. Cohen BE, Johnson JE. Subtalar arthrodesis for treatment of posterior tibial tendon insufficiency. *Foot Ankle Clin* 6:121-128, 2001.
 165. Deland JT, Otis JC, Lee KT, Kenneally SM. Lateral column lengthening with calcaneocuboid fusion: range of motion in the triple joint complex. *Foot Ankle Int* 16:729-733, 1995.
 166. Astion DJ, Deland JT, Otis JC, Kenneally S. Motion of the hindfoot after simulated arthrodesis. *J Bone Joint Surg* 79:241-246, 1997.
 167. Fleischli JG, Fleischli JW, Laughlin TJ. Treatment of posterior tibial tendon dysfunction with tendon procedures from the posterior muscle group. *Clin Podiatr Med Surg* 16:453-470, 1999.
 168. Mendicino SS, Quinn M. Tibialis posterior dysfunction: an overview with a surgical case report using a flexor tendon transfer. *J Foot Surg* 28:154-157, 1989.
 169. Benton-Weil W, Weil LS Jr. The Cobb procedure for stage II posterior tibial tendon dysfunction. *Clin Podiatr Med Surg* 16:471-477, 1999.
 170. Mosier-LaClair S, Pomeroy S, Manoli A II. Operative treatment of the difficult stage 2 adult acquired flatfoot deformity. *Foot Ankle Clin* 6:95-119, 2001.

171. Baravarian B, Zbonis T, Lowery C. Use of the Cobb procedure in the treatment of posterior tibial tendon dysfunction. *Clin Podiatr Med Surg* 19:371–389, 2002.
172. Hintermann B. [Dysfunction of the posterior tibial muscle due to tendon insufficiency]. *Orthopade* 24:193–199, 1995.
173. Zwipp H, Dahlen C, Amlang M, Rammelt S. [Injuries of the tibialis posterior tendon: diagnosis and therapy]. *Orthopade* 29:251–259, 2000.
174. Kelly IP, Easley ME. Treatment of stage 3 adult acquired flatfoot. *Foot Ankle Clin* 6:153–166, 2001.
175. Jarde O, Abiraad G, Gabrion A, Vernois J, Massy S. [Triple arthrodesis in the management of acquired flatfoot deformity in the adult secondary to posterior tibial tendon dysfunction. A retrospective study of 20 cases]. *Acta Orthop Belg* 68:56–62, 2002.
176. Harris EJ, Vanore JV, Thomas JL, Kravitz SR, Mendelson SA, Mendicino RW, Silvani SH, Gassen SC. Diagnosis and treatment of pediatric flatfoot. *J Foot Ankle Surg* 43:341–373 2004.
177. McLaren CA, Wootton JR, Heath PD, Jones CH. Pes planus after tibial osteotomy. *Foot Ankle* 9:300–303, 1989
178. Mueller TJ. Ruptures and lacerations of the tibialis posterior tendon. *J Am Podiatr Med Assoc* 74:109–119, 1984.
179. Masterson R, Mulcahy D, McElwain J, McInerney D. The planovalgus rheumatoid foot—is tibialis posterior tendon rupture a factor? *Br J Rheumatol* 34:645–646, 1995.
180. Finby N, Kraft E, Spyropoulos E. Diabetic osteopathy of the foot and ankle. *Am Fam Physician* 14:90–95, 1976.
181. Rosenberg JN. Diabetes mellitus with osteopathy and Charcot's arthropathy. *Proc R Soc Med* 69:705, 1976.
182. Edmonds ME. The diabetic foot: pathophysiology and treatment. *Clin Endocrinol Metab* 15:889–916, 1986.
183. Reiner M, Scurran BL, Karlin JM, Silvani SH. The neuropathic joint in diabetes mellitus. *Clin Podiatr Med Surg* 5:421–437, 1988.
184. Scartozzi G, Kanat IO. Diabetic neuroarthropathy of the foot and ankle. *J Am Podiatr Med Assoc* 80:298–303, 1990.
185. Sanders LJ, Frykberg RG. Diabetic neuropathic osteoarthropathy. The Charcot foot. In *The High Risk Foot in Diabetes Mellitus*, p 297, edited by RG Frykberg. Churchill Livingstone, New York, 1991.
186. Sanders LJ, Frykberg RG. Charcot foot. In *The Diabetic Foot*, p 149, edited by JH Bowker, Mosby Yearbook, St. Louis, 1993.
187. Frykberg RG, Kozak GP. The diabetic Charcot foot. In *Management of Diabetic Foot Problems*, pp 88–97, edited by GM Habershaw, WB Saunders, Philadelphia, 1995.
188. Klenerman L. The Charcot joint in diabetes. *Diabet Med* 13 (Suppl 1):S52–S54, 1996.
189. Schon LC, Easley ME, Weinfeld SB. Charcot neuroarthropathy of the foot and ankle. *Clin Orthop* 349:116–131, 1998.
190. Frykberg RG, Armstrong DG, Giurini J, Edwards A, Kravette M, Kravitz S, Ross C, Stavosky J, Stuck R, Vanore J, American College of Foot and Ankle Surgeons. Diabetic foot disorders. A clinical practice guideline. *J Foot Ankle Surg* 39(Suppl):S1–S60, 2000.
191. Rajbhandari SM, Jenkins RC, Davies C, Tesfaye S. Charcot neuroarthropathy in diabetes mellitus. *Diabetologia* 45:1085–1096, 2002.
192. Brower AC, Allman RM. Pathogenesis of the neurotrophic joint: neurotraumatic vs. neurovascular. *Radiology* 139:349–354, 1981.
193. Slowman-Kovacs SD, Braunstein EM, Brandt KD. Rapidly progressive Charcot arthropathy following minor joint trauma in patients with diabetic neuropathy. *Arthritis Rheum* 33:412–417, 1990.
194. Gandsman EJ, Deutsch SD, Kahn CB, McCullough RW. Differentiation of Charcot joint from osteomyelitis through dynamic bone imaging. *Nucl Med Commun* 11:45–53, 1990.
195. Shih WJ, Purcell M. Diabetic Charcot joint mimicking acute osteomyelitis in radiography and three-phase radionuclide bone imaging study. *Radiat Med* 9:47–49, 1991.
196. Palestro CJ, Mehta HH, Patel M, Freeman SJ, Harrington WN, Tomas MD, Marwin SE. Marrow versus infection in the Charcot joint: indium-111 leukocyte and technetium-99m sulfur colloid scintigraphy. *J Nucl Med* 39:346–350, 1998.
197. Cavanagh PR, Young MJ, Adams JE, Vickers KL, Boulton AJ. Radiographic abnormalities in the feet of patients with diabetic neuropathy. *Diabetes Care* 17:201–209, 1994.
198. Smith DG, Barnes BC, Totty WG. Prevalence of radiographic foot abnormalities in patients with diabetes. *Foot Ankle Int* 18:342–346, 1997.
199. Caputo GM, Ulbrecht J, Cavanagh PR, Juliano P. The Charcot foot in diabetes: six key points. *Am Fam Physician* 57:2705–2710, 1998.
200. Sella EJ, Barrette C. Staging of Charcot neuroarthropathy along the medial column of the foot in the diabetic patient. *J Foot Ankle Surg* 38:34–40, 1999.
201. Tomas MB, Patel M, Marwin SE, Palestro CJ. The diabetic foot. *Br J Radiol* 73:443–450, 2000.
202. Sommer TC, Lee TH. Charcot foot: the diagnostic dilemma. *Am Fam Physician* 64: 1591–1598, 2001.
203. Mueller M, Minor SD, Diamond JE, Blair VP III. Relationship of foot deformity to ulcer location in patients with diabetes mellitus. *Phys Ther* 70:356–362, 1990.
204. Beltran J, Campanini DS, Knight C, McCalla M. The diabetic foot: magnetic resonance imaging evaluation. *Skeletal Radiol* 19:37–41, 1990.
205. Oyen WJ, Netten PM, Lemmens JA, Claessens RA, Lutterman JA, van der Vliet JA, Goris RJ, van der Meer JW, Corstens FH. Evaluation of infectious diabetic foot complications with indium-111-labeled human nonspecific immunoglobulin G. *J Nucl Med* 33:1330–1336, 1992.
206. Kristiansen B. Ankle and foot fractures in diabetics provoking neuropathic joint changes. *Acta Orthop Scand* 51:975–979, 1980.
207. Edelman SV, Kosofsky EM, Paul RA, Kozak GP. Neuro-osteoarthropathy (Charcot's joint) in diabetes mellitus following revascularization surgery. Three case reports and a review of the literature. *Arch Intern Med* 147:1504–1508, 1987.
208. Connolly JF, Csencsitz TA. Limb threatening neuropathic complications from ankle fractures in patients with diabetes. *Clin Orthop* 348:212–219, 1998.
209. Fabrin J, Larsen K, Holstein PE. Long-term follow-up in diabetic Charcot feet with spontaneous onset. *Diabetes Care* 23:796–800, 2000.
210. Armstrong DG, Lavery LA. Acute Charcot's arthropathy of the foot and ankle. *Phys Ther* 78:74–80, 1998.
211. Guis S, Pellissier JF, Arniaud D, Turck F, Witjas T, Roux H, Mattei JP. Healing of Charcot's joint by pamidronate infusion. *J Rheumatol* 26:1843–1845, 1999.
212. Rubin, G, Cohen E. Prostheses and orthoses for the foot and ankle. *Clin Podiatr Med Surg* 5:695–719, 1988.
213. Saltzman CL, Johnson KA, Goldstein RH, Donnelly RE. The patellar tendon-bearing brace as treatment for neurotrophic arthropathy: a dynamic force monitoring study. *Foot Ankle* 13:14–21, 1992.
214. Morgan JM, Biehl WC III, Wagner FW Jr. Management of neuropathic arthropathy with the Charcot Restraint Orthotic Walker. *Clin Orthop* 296:58–63, 1993.
215. Giurini JM. Applications and use of in-shoe orthoses in the conservative management of Charcot foot deformity. *Clin Podiatr Med Surg* 11:271–278, 1994.
216. Boninger MR, Leonard JA Jr. Use of bivalved ankle-foot orthosis in neuropathic foot and ankle lesions. *J Rehabil Res Dev* 33:16–22, 1996.
217. Green MF, Aliabadi Z, Green BJ. Diabetic foot: evaluation and management. *South Med J* 95:95–101, 2002.
218. Catanzariti AR, Mendicino R, Haverstock B. Osteotomy for diabetic neuroarthropathy involving the midfoot. *J Foot Ankle Surg* 39:291–300, 2000.

219. Cheng YM, Lin SY, Tien YC, Wu HS. Ankle arthrodesis. *Gaoxiong Yi Xue Ke Xue Za Zhi* 9:524–531, 1993.
220. Cohen M, Roman A, Lovins JE. Totally implanted direct current stimulator as treatment for a nonunion in the foot. *J Foot Ankle Surg* 32:375–381, 1993.
221. Pinzur MS, Kelikian A. Charcot ankle fusion with a retrograde locked intramedullary nail. *Foot Ankle Int* 18:699–704, 1997.
222. Pinzur MS. Charcot's foot. *Foot Ankle Clin* 5:897–912, 2000.
223. Pinzur MS. Benchmark analysis of diabetic patients with neuropathic (Charcot) foot deformity. *Foot Ankle Int* 20:564–567, 1999.