Acquired flatfoot deformity caused by dysfunction of the posterior tibial tendon is a common clinical problem. Treatment, which depends on the severity of the symptoms and the stage of the disease, includes nonoperative options, such as rest, administration of anti-inflammatory medication, and immobilization, as well as operative options, such as tendon transfer, calcaneal osteotomy, and several methods of arthrodesis.

**Anatomy and Pathophysiology**

The posterior tibial muscle forms part of the deep posterior compartment of the calf. It originates from the proximal third of the tibia and the interosseous membrane and passes immediately posterior to the medial malleolus, where it changes direction acutely. A groove in the posteromedial aspect of the distal part of the tibia holds the posterior tibial tendon but is not deep enough to keep the tendon from bow-stringing or dislocating after an injury. The flexor retinaculum, which is adjacent to the medial malleolus, tethers the tendon and keeps it in the groove, preventing dislocation. Distally, this retinaculum blends with the sheath of the posterior tibial tendon and the superficial deltoid ligament. The posterior tibial tendon does not have a mesotenon, and there is an area of relative hypovascularity immediately distal to the medial malleolus that may contribute to degenerative changes of the tendon. The posterior tibial tendon divides anterior to the tuberosity of the navicular. An anterior slip, which is in direct continuity with the main tendon, inserts onto the tuberosity of the navicular, the inferior aspect of the capsule of the medial naviculocuneiform joint, and the inferior surface of the medial cuneiform. A second slip attaches to the plantar surfaces of the middle and lateral cuneiforms and the cuboid as well as to the bases of the corresponding metatarsals.

The posterior tibial tendon passes posterior to the axis of the ankle and medial to the axis of the subtalar joint. Because of its insertion on the navicular and the plantar aspect of the mid-part of the tarsus, it acts to plantar flex as well as to invert the middle part of the foot with an action that occurs primarily across the transverse tarsal joint (the talonavicular and calcaneocuboid joints). The posterior tibial tendon therefore functions predominantly to invert the midfoot and to elevate the medial longitudinal arch rather than having a specific effect on the hindfoot. However, the tendon does support the calcaneus and the rest of the hindfoot indirectly by means of its action within the pulley posterior to the medial malleolus as well as its relationship with the deep deltoid ligament, the talonavicular capsule, and the spring (calcaneonavicular) ligament.

With dysfunction of the posterior tibial tendon, the medial longitudinal arch collapses, the subtalar joint everts, the heel assumes a valgus position, and the foot abducts at the talonavicular joint. This excessive movement around the talus suggests that the loss of function occurs as a result of dysfunction of the posterior tibial tendon at the site of its insertion on the navicular or through secondary stretching of the spring ligament. The valgus angulation of the heel is probably a result of the loss of secondary soft-tissue supportive structures, including the deltoid ligament, the talonavicular capsule, and the spring ligament. Once the heel assumes a more valgus position, the Achilles tendon begins to act as an evertor of the calcaneus as it is then lateral to the axis of rotation of the subtalar joint.

Dysfunction of the posterior tibial tendon therefore results in a reversal of its primary actions, with relative internal rotation of the tibia and talus and flattening of the medial longitudinal arch. Secondary changes that may develop over time include an equinus deformity of the ankle, a more horizontal orientation of the subtalar joint, and valgus angulation of the heel. The associated...
contracture of the Achilles tendon causes more sagittal-plane motion to occur at the subtalar joint rather than at the tibiotalar joint. As the deformity progresses, this change eventually causes the calcaneus to impinge against the fibula, creating pain in the lateral part of the foot and ankle.

The clinical deformity associated with dysfunction of the posterior tibial tendon reflects the loss of support from the spring, deltoid, and talocalcaneal interosseous ligaments as well as from the talonavicular capsule and the plantar fascia. Normally, the deltoid ligament has a substantial stabilizing effect on the tibiotalocalcaneal joint complex. As the deformity progresses, the deltoid ligament becomes attenuated and the talus begins to tilt in the ankle joint, leading to valgus deformity of the tibiotalar joint. Deland et al. demonstrated, in a cadaveric model, that no abnormality can be seen radiographically when there is dysfunction of the posterior tibial tendon alone and that a static deformity occurs only when there is also associated ligamentous damage. Although one cannot extrapolate these data directly to the clinical situation, it is important to recognize that dysfunction of the posterior tibial tendon usually is associated with changes in the ligamentous support of the hindfoot.

Etiology

The etiology of dysfunction of the posterior tibial tendon is varied, ranging from inflammatory synovitis to degenerative rupture and, occasionally, to acute trauma. A number of investigators have identified an increased prevalence of rupture of the posterior tibial tendon in middle-aged obese women. Holmes and Mann reported that forty (60 per cent) of sixty-seven patients who had a rupture of the posterior tibial tendon had a history of hypertension, obesity, diabetes, a previous operation or trauma about the medial aspect of the foot, or treatment with steroids. Although acute trauma rarely plays a substantial role in ruptures of the posterior tibial tendon and although only a few patients can recall a specific history of a sprain or another injury about the foot and ankle, the tendon is at risk of injury because of its close proximity to the medial malleolar sulcus. For example, an acute rupture of the posterior tibial tendon occasionally has occurred in association with a closed fracture or dislocation of the ankle.

There appears to be an association between dysfunction of the posterior tibial tendon and the injection of steroids. Jahss reported that three of ten patients who had a rupture of the posterior tibial tendon had received multiple injections of corticosteroids around the medial aspect of the foot months to years earlier. Holmes and Mann reported that the injection of corticosteroids about the posterior tibial tendon or the oral intake of corticosteroids was associated with an increased likelihood of rupture of the posterior tibial tendon.

It is likely that most ruptures of the posterior tibial tendon are due to an intrinsic abnormality or a failure of the tendon rather than to extrinsic trauma. A rupture may result from the acute angulation of the tendon as it passes posterior to the medial malleolus, from the relative hypovascularity of the tendon just distal to the medial malleolus, or from degeneration associated with an inflammatory disorder. Neither the flexor digitorum longus nor the flexor hallucis longus angulates as acutely behind the medial malleolus as does the posterior tibial tendon, and the former two tendons rarely rupture. Frey et al. showed that the posterior tibial tendon has a zone of hypovascularity that begins one to 1.5 centimeters distal to the medial malleolus and extends one centimeter farther distally. It is in this area where degenerative changes and ruptures most often are found. Dysfunction of the posterior tibial tendon also has been linked to seronegative inflammatory disorders, such as ankylosing spondylitis, Reiter syndrome, and psoriasis. Myerson et al. reported on a group of seventy-six patients, forty-seven of whom had inflammation or rupture, or both, of the posterior tibial tendon in association with a seronegative inflammatory disorder and manifestations of enthesopathy. Whereas Anzel et al. suggested that ruptures of the posterior tibial tendon are common in patients who have rheumatoid arthritis, other authors have found no strong relationship between rheumatoid arthritis and ruptures of the posterior tibial tendon. It is my impression that flatfoot deformity associated with rheumatoid arthritis is more likely to be caused by synovitis of the talonavicular and subtalar joints.

Diagnosis

History

In the early stages of dysfunction of the posterior tibial tendon, most of the discomfort is located medially along the course of the tendon and the patient reports fatigue and aching on the plantar-medial aspect of the foot and ankle. Swelling is common if the dysfunction is associated with tenosynovitis. As dysfunction of the tendon progresses, maximum pain occurs laterally in the sinus tarsi because of impingement of the fibula against the calcaneus. With increasing deformity, patients report that the shape of the foot changes and that it becomes increasingly difficult to wear shoes. Many patients no longer report pain in the medial part of the foot and ankle after a complete rupture of the posterior tibial tendon has occurred; instead, the pain is located laterally. If a fixed deformity has not occurred, the patient may report that standing or walking with the hindfoot slightly inverted alleviates the lateral impingement and relieves the pain in the lateral part of the foot.

Examination of the Patient

First, both feet should be examined with the patient standing and the entire lower extremity visible. The foot
should be inspected from above as well as from behind the patient, as valgus angulation of the hindfoot is best appreciated when the foot is viewed from behind. Johnson described the so-called more-toes sign: with more advanced deformity and abduction of the forefoot, more of the lateral toes become visible when the foot is viewed from behind. The single-limb heel-rise test is an excellent determinant of the function of the posterior tibial tendon. The patient is asked to attempt to rise onto the ball of one foot while the other foot is suspended off the floor. Under normal circumstances, the posterior tibial muscle, which inverts and stabilizes the hindfoot, is activated as the patient begins to rise onto the forefoot. The gastrocnemius-soleus muscle group then elevates the calcaneus, and the heel-rise is accomplished. With dysfunction of the posterior tibial tendon, however, inversion of the heel is weak, and either the heel remains in valgus or the patient is unable to rise onto the forefoot. If the patient can do a single-limb heel-rise, the limb may be stressed further by asking the patient to perform this maneuver repetitively.

With the patient seated, the strength of the posterior tibial muscle is evaluated by asking the patient to plantar flex and invert the foot against resistance. During the test, the examiner should hold the hindfoot of the patient in plantar flexion and inversion and the forefoot in abduction. This eliminates the synergistic action of the anterior tibial muscle and allows the strength of the posterior tibial muscle to be quantified more accurately.

The integrity of the tendon and the location of maximum tenderness are established with palpation. Occasionally, an acquired flatfoot deformity is due not to a rupture of the posterior tibial tendon but to a tear of the spring or deltoid ligament. Therefore, the integrity of the tendon should be assessed with palpation while the patient attempts to invert the foot against resistance.

After the sites of tenderness have been located, the subtalar and ankle joints are assessed for mobility and the presence of any contracture of the Achilles tendon. With increased valgus angulation of the heel, the Achilles tendon assumes a position lateral to the axis of the subtalar and the gastrocnemius-soleus muscle group shortens over time. If the subtalar joint is mobile, the examiner should assess dorsiflexion of the ankle while passively correcting any valgus angulation of the heel, to be sure that the dorsiflexion that is present is occurring through the ankle and not through the transverse tarsal joint. The position of the forefoot is assessed with the heel held in neutral. In the early stages of flatfoot deformity, the forefoot remains plantigrade when the subtalar joint is placed in the neutral position.

As the deformity worsens, however, a fixed supination deformity of the forefoot occurs and can be identified by holding the subtalar joint reduced in a neutral position. It is important to identify any fixed supination deformity of the forefoot because such a deformity will have an effect on the method of treatment that is selected. With more advanced dysfunction of the posterior tibial tendon, rigidity of the subtalar joint may be present and may make it impossible to reduce the valgus deformity.

Radiography

Anteroposterior and lateral weight-bearing radiographs of both feet and mortise radiographs of both ankles are recommended for the evaluation of flatfoot deformity. If the deformity is present, the anteroposterior radiograph shows abduction of the forefoot at the transverse tarsal joint, with the navicular sliding laterally on the talar head. The lateral radiograph shows a decrease in the talometatarsal angle (normal angle, 0 to 10 degrees) and in the distance of the medial cuneiform from the floor (normal distance, fifteen to twenty-five millimeters) when the involved foot is compared with the contralateral foot. In patients who have an advanced deformity, subluxation or dislocation of the talonavicular joint may occur in association with degenerative osteoarthrosis of the posterior facet of the subtalar joint.

It should be emphasized that the diagnosis of dysfunction of the posterior tibial tendon is based on a thorough examination of the foot as well as plain radiographs, which confirm the extent of the deformity and the presence of osteoarthrosis. Magnetic resonance imaging is not required to make the diagnosis and does not assist in the planning of treatment. Although some authors have suggested that magnetic resonance imaging is a useful diagnostic tool in the evaluation of dysfunc­tion of the posterior tibial tendon, other authors have asserted that it is used far too frequently in clinical practice and that its clinical usefulness is questionable.

Classification

There is a continuum of dysfunction of the posterior tibial tendon, ranging from tenosynovitis to fixed deformity. Any grading or staging system of this dysfunction is therefore somewhat arbitrary. It is useful, however, to describe the dysfunction in stages in order to present treatment alternatives in a more logical manner. Johnson and Strom, in 1989, described three clinical stages of dysfunction of the posterior tibial tendon. Stage I is characterized by pain and swelling of the medial aspect of the foot and ankle. The length of the tendon is normal, and tendinitis may be associated with mild degeneration. Mild weakness and minimum deformity are present. In stage II, the tendon is torn, the limb is weak, and the patient is unable to stand on tiptoe on the affected side. Secondary deformity is present as the midfoot pronates and the forefoot abducts at the transverse tarsal joint. The subtalar joint, however, remains flexible. In stage III, degeneration of the tendon is present, the deformity is more severe, and the hindfoot is rigid. Stage IV, which was not described by John-
son and Strom\textsuperscript{7}, is characterized by valgus angulation of the talus and early degeneration of the ankle joint.

### Treatment

Non-operative management should be attempted for most symptomatic patients who have dysfunction of the posterior tibial tendon. Most patients who have tenosynovitis or stage-I disease respond to non-operative management. Patients who have more advanced (stage-II or III) disease also benefit from non-operative management, but the risk of progressive deformity must be considered when planning treatment.

**Non-Operative Treatment**

A patient who has acute tenosynovitis has pain and swelling along the medial aspect of the ankle. The patient is able to perform a single-limb heel-rise test but has pain when doing so. Inversion of the foot against resistance is painful but still strong. The patient should be managed with rest, the administration of appropriate anti-inflammatory medication, and immobilization. The injection of corticosteroids is not recommended. Immobilization with either a rigid below-the-knee cast or a removable cast or boot may be used to prevent overuse and subsequent rupture of the tendon. A removable stirrup-brace is not initially sufficient as it does not limit motion in the sagittal plane, a component of the pathological process. The patient should be permitted to walk while wearing the cast or boot during the six to eight-week period of immobilization. At the end of that time, a decision must be made regarding the need for additional treatment. If there has been marked improvement, the patient may begin wearing a stiff-soled shoe with a medial heel-and-sole wedge to invert the hindfoot. If there has been only mild or moderate improvement, a longer period in the cast or boot may be tried.

Chao et al.\textsuperscript{6} demonstrated that when the flatfoot deformity is caused by a non-functioning posterior tibial tendon, the deformity usually is progressive. However, they showed that a molded ankle-foot orthosis or a rigid orthotic support can control the deformity and alleviate symptoms in many elderly patients who have a relatively sedentary lifestyle. They concluded, therefore, that operative treatment should be considered only for patients for whom an adequate trial of bracing has failed.

**Operative Treatment**

Many operations are available for the treatment of dysfunction of the posterior tibial tendon after a thorough program of non-operative treatment has failed. The type of operation that is selected is determined by the age, weight, and level of activity of the patient as well as the extent of the deformity. The clinical stages outlined previously are a useful guide to operative care (Table 1). In general, the clinician should perform the least invasive procedure that will decrease pain and improve function. One should consider the effects of each procedure, particularly those of arthrodesis, on the function of the rest of the foot and ankle.

### Pathological Findings

The pathological findings vary according to the underlying etiology of the dysfunction of the posterior tibial tendon. Myerson et al.\textsuperscript{4} demonstrated that patients who have a seronegative inflammatory disorder have a proliferative synovial membrane that adheres to the tendon. In these patients, the tendon often is

### Table 1

**TREATMENT OF DYSFUNCTION OF THE POSTERIOR Tibial Tendon**

<table>
<thead>
<tr>
<th>Stage</th>
<th>Characteristics</th>
<th>Non-Operative</th>
<th>Operative</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tenosynovitis</td>
<td>Acute medial pain and swelling,</td>
<td>Anti-inflammatory medication,</td>
<td>Tenosynovectomy, tenosynovectomy + calcaneal osteotomy, or tenosynovectomy + tenodesis of flexor digitorum longus to posterior tibial tendon</td>
</tr>
<tr>
<td></td>
<td>can perform heel-rise,</td>
<td>immobilization for 6-8 wks; if symptoms improve, ankle stirrup-brace; if symptoms do not improve, operative treatment</td>
<td></td>
</tr>
<tr>
<td>Rupture</td>
<td>Medial pain and swelling,</td>
<td>Medial heel-and-sole shoe wedge,</td>
<td>Débrèdement of posterior tibial tendon, flexor digitorum longus transfer, or flexor digitorum longus transfer + calcaneal osteotomy</td>
</tr>
<tr>
<td>Stage I</td>
<td>hindfoot flexible, can perform heel-rise</td>
<td>hinged ankle-foot orthosis,</td>
<td>Flexor digitorum longus transfer + calcaneal osteotomy or flexor digitorum longus transfer + bone-block arthrodesis at calcaneocuboid joint</td>
</tr>
<tr>
<td>Stage II</td>
<td>Valgus angulation of heel,</td>
<td>Medial heel-and-sole shoe wedge,</td>
<td>Rigid ankle-foot orthosis</td>
</tr>
<tr>
<td></td>
<td>lateral pain, hindfoot flexible, cannot perform heel-rise</td>
<td>stiff orthotic support, hinged</td>
<td>Triple arthrodesis</td>
</tr>
<tr>
<td>Stage III</td>
<td>Valgus angulation of heel,</td>
<td>Rigid ankle-foot orthosis</td>
<td>Tibiotalocalcaneal arthrodesis</td>
</tr>
<tr>
<td></td>
<td>lateral pain, hindfoot rigid, cannot perform heel-rise</td>
<td>ankle-foot orthosis, injection of steroids into the sinus tarsi</td>
<td></td>
</tr>
<tr>
<td>Stage IV</td>
<td>Hindfoot rigid, valgus angulation of talus</td>
<td>Rigid ankle-foot orthosis</td>
<td></td>
</tr>
</tbody>
</table>

### Pathological Findings

The pathological findings vary according to the underlying etiology of the dysfunction of the posterior tibial tendon. Myerson et al.\textsuperscript{4} demonstrated that patients who have a seronegative inflammatory disorder have a proliferative synovial membrane that adheres to the tendon. In these patients, the tendon often is...
found to be attenuated and to contain longitudinal fissures, and a chronic non-specific inflammatory infiltrate is noted histologically. In contrast, in patients who have degenerative tendinosis, the tendon sheath is edematous and thickened. Fibrous adhesions usually are present and often extend posteriorly from the inner (medial) aspect of the tendon. If a degenerative tear is present, it commonly is located one to two centimeters distal to the medial malleolus. Partial or complete rupture of the tendon occurs approximately two centimeters proximal to the tubercle of the navicular. In most instances, the scarred tendon is adherent to the tendon sheath. Longitudinal fissures commonly are found in the posterior aspect of the tendon that lies in proximity to the deeper soft tissues and tendon sheath.

I have noted that the extent of deformity as seen clinically and radiographically does not always correspond with the degree of degeneration of the posterior tibial tendon, and I have encountered patients who have had severe unilateral flatfoot deformity in association with an intact and grossly normal posterior tibial tendon. Such patients have a rupture of the spring ligament, the talonavicular capsule, or the deltoid ligament.

Tenosynovectomy

Patients who have tenosynovitis have an acute onset of pain and swelling along the medial aspect of the foot immediately inferior to the medial malleolus. This finding is associated with aching of the foot and an inability to stand for a long time or to walk for a long distance. The area directly over the tendon is tender to palpation. Inversion of the foot against resistance is strong, and the patient is able to perform single or even repetitive heel-rises, albeit with pain. Tenosynovectomy should be considered if the patient shows no improvement after six to eight weeks of non-operative care. This intensive approach is particularly important in younger patients in whom tenosynovitis may have developed in association with a seronegative inflammatory disorder. This type of tenosynovitis appears to be particularly aggressive and is associated with an inflammatory infiltrate into the tendon that leads to rupture of the tendon unless treated expeditiously.

A tenosynovectomy may be performed with ankle-block anesthesia. A posteromedial incision is made from the musculotendinous junction to the insertion of the posterior tibial tendon. It is useful to preserve a one-centimeter strip of the flexor retinaculum adjacent to the medial malleolus to prevent subsequent dislocation of the posterior tibial tendon, which may occur if the flexor retinaculum is not preserved or re-

paired after having been opened completely.

Once the tendon sheath is opened, the tendon is inspected. When tenosynovitis is associated with a seronegative inflammatory disorder, the synovial membrane adheres to and infiltrates both the sheath and the tendon; this inflammatory tissue should be debrided thoroughly. Tenosynovitis associated with stage-I degenerative tendinosis has a less florid appearance, and bulbous swelling of the tendon associated with longitudinal fissuring (particularly on the posterior surface) typically is present. These fissures should be debrided, and the tendon splits should be repaired with 4-0 nonabsorbable sutures. Any bulbous degenerated area of the tendon should be excised, and the tendon should be thinned so that it easily passes posterior to the malleolus. After the débridement, the retinaculum immediately posterior to the medial malleolus is repaired if it was detached completely, but otherwise the tendon sheath is left open to minimize the subsequent formation of adhesions.

It is unlikely that young patients who have tenosynovitis in association with a seronegative inflammatory disorder will need another operation. Tenosynovectomy usually is sufficient treatment unless attritional tearing of the tendon already has occurred. This approach was substantiated by Teasdall and Johnson, who recommended that stage-I dysfunction of the posterior tibial tendon be treated with synovectomy and débridement of the torn tendon. Sixteen of the nineteen patients in that study reported subjective improvement, with a return of the function of the posterior tibial tendon, after having been managed with that technique.

In contrast, in older patients who have stage-I disease but in whom the degeneration of the tendon is more advanced than the symptoms suggested, it is important to consider another procedure in addition to tenosynovectomy to support the diseased tendon. If one assumes that the tenosynovitis is a manifestation of early degeneration of the tendon, then the underlying pathological process may continue to worsen after a simple tenosynovectomy. Some authors have suggested that such patients should be managed with a side-to-side tenodesis of the flexor digitorum longus tendon to the posterior tibial tendon or a calcaneal osteotomy in addition to tenosynovectomy.

**Calcaneal Osteotomy**

The use of a calcaneal osteotomy to treat valgus angulation of the heel shifts the calcaneus medially and alters the mechanical axis of the lower limb, thereby reducing the valgus thrust on the hindfoot. The medial displacement of the calcaneus redirects the pull of the gastrocnemius-soleus muscle group slightly me-
dial to the axis of the subtalar joint, increasing its varus pull on the hindfoot. Calcaneal osteotomy was performed first by Gleich, in 1893, in an attempt to displace the posterior calcaneal tuberosity both medially and inferiorly in order to restore the calcaneal pitch angle. Subsequent authors have reported on the treatment of flatfoot deformity with calcaneal osteotomy. In the osteotomy described by Rose, a triangular portion of bone is resected from the posteroinferior fragment, to create a medial ridge that passes medial to the inner side of the proximal fragment and locks the osteotomy in a stable configuration. This osteotomy creates both varus angulation and medial displacement of the calcaneus.

The osteotomy recommended by Myerson et al., Saxby and Myerson, Fairbank et al., and Koutsoigiannis is a simpler medial displacement osteotomy of the posterior part of the calcaneus. This procedure is indicated in combination with tenosynovectomy for patients who have advanced stage-I dysfunction of the posterior tibial tendon and in conjunction with a flexor tendon transfer for those who have stage-II disease. The patient is placed in the lateral decubitus position, and an incision is made inferior and parallel to the peroneal tendons and posterior to the sural nerve. The incision extends from the superior border of the calcaneal tuberosity anterior to the retrocalcaneal space to the inferior border of the calcaneus superficial to the plantar fascia. The incision is deepened to the periosteum, and the soft tissues are retracted with a self-retaining retractor. The periosteum is reflected. A transverse osteotomy is made in the calcaneus in line with the incision in the skin with use of an oscillating saw. The cut is made at a right angle to the lateral border of the calcaneus and is inclined posteriorly at an angle of approximately 45 degrees to the plane of the sole of the foot. No wedge is removed from the calcaneus, and no attempt is made to tilt the tuberosity into varus angulation. A lamina spreader is inserted into the site of the osteotomy, and the medial soft-tissue attachments onto the calcaneus are released by spreading the device open. The lamina spreader then is withdrawn, and the posterior fragment of the calcaneal tuberosity is translated medially ten millimeters and is secured with a cannulated cancellous-bone lag screw (Fig. 1).

Another procedure, which involves lengthening of the lateral column of the foot by means of an osteotomy of the neck of the calcaneus and the interposition of a tricortical bone graft, recently has gained popularity. Evans recommended that the osteotomy be done 1.5 centimeters proximal to the calcaneocuboid joint. Other authors have reported good results with this procedure. Sangeorzan et al. investigated the effect of calcaneal lengthening on the osseous relationships of the hindfoot, midfoot, and forefoot and noted improvement in the flatfoot deformity with respect to the lateral talocalcaneal angle, the lateral talometatarsal angle, the an-
ADULT ACQUIRED FLATFOOT DEFORMITY

Figs. 7-C and 7-D: Postoperative radiographs showing a decrease in the deformities.

Figs. 7-C and 7-D: Postoperative radiographs showing a decrease in the deformities.

symptoms in the calcaneocuboid joint in adults. When lengthening of the lateral column is performed in an adult who has acquired flatfoot deformity, I think that it probably should be done through the calcaneocuboid joint itself, with use of a tricortical bone graft for arthrodesis of the joint. I believe that this procedure is indicated for a patient who has stage-II dysfunction with pain in the lateral part of the foot, a mobile subtalar joint, and no fixed supination deformity of the forefoot when the heel is held in a neutral position.

**Flexor Tendon Transfer**

Flexor tendon transfer is performed for patients who have stage-I or II dysfunction of the posterior tibial tendon in association with weakness, valgus angulation of the hindfoot, pain in the medial part of the foot, and a mobile subtalar joint. A number of authors have recommended use of the flexor digitorum longus tendon to substitute for the ruptured posterior tibial tendon. Goldner et al. recommended use of the flexor hallucis longus for the reconstruction. With that procedure, however, the dissection is more complicated, the neurovascular bundle is in closer proximity to the flexor hallucis longus, and the deficit at the donor site is more substantial. Although the flexor hallucis longus muscle is markedly stronger than the flexor digitorum longus, the proximity of the posterior tibial tendon to the flexor digitorum longus makes the latter a more suitable tendon for transfer. The transfer involves cutting the flexor digitorum longus tendon distally and rerouting it through the undersurface of the navicular through a drill-hole. The incision is made along the entire length of the posterior tibial tendon from the musculotendinous junction distally beyond the navicular in line with the first metatarsal (Fig. 2). The sheath of the posterior tibial tendon is opened, and the tendon is inspected and cut transversely adjacent to the medial malleolus, leaving a one-centimeter stump attached to the navicular distally.

Chronic rupture of the posterior tibial tendon may be associated with atrophy and fibrosis of the muscle. If the posterior tibial muscle is healthy, however, the tendon should be sutured to the flexor digitorum longus tendon at the level of the medial malleolus. When the proximal stump of the tendon is pulled in a distal direction, its normal excursion is approximately one centimeter. If there is no excursion of the musculotendinous unit when it is pulled in a distal direction, the muscle is either scarred or fibrotic, and a tenodesis to the flexor digitorum longus should not be performed.

The sheath of the flexor digitorum longus tendon is identified posterior to the posterior tibial tendon and is opened longitudinally and distally. The plane of dissection should be deep to the abductor hallucis and flexor digitorum brevis muscles. Distal dissection should allow identification of both the flexor hallucis longus and the flexor digitorum longus tendon. The flexor digitorum longus tendon should be cut as far distally as possible under direct vision (Fig. 3). It is not necessary to perform a tenodesis of the flexor hallucis longus to the stump of the flexor digitorum longus as there are multiple interconnections between the two.

The periosteum over the navicular then is dissected, and a 4.5-millimeter drill-hole is made in the tuberosity from dorsal to plantar (Fig. 4). The tendon is passed through the drill-hole from plantar to dorsal. Before the flexor digitorum longus tendon is sutured, a vertical ellipse is removed from the talonavicular capsule. The ellipse should be approximately eight millimeters wide and extend inferiorly from the dorsal aspect of the joint toward the spring ligament (Fig. 5). Once the flexor digitorum longus tendon has been transferred and at-
tached, it is secured under moderate tension, and, finally, the shortened capsule is closed. To avoid overstretching the flexor digitorum longus muscle, the tension on its tendon is set halfway between maximum tension and complete relaxation (Fig. 6)\(^\text{39}\).

At the completion of the reconstruction, the foot should rest in a slight equinovarus position. If a tourniquet was used, a suction drain should be inserted and left in place for twenty-four hours. No weight-bearing is permitted for four weeks, after which partial weight-bearing is permitted while wearing either a below-the-knee cast or a hinged range-of-motion walker boot for an additional four weeks. By eight weeks, the patient begins walking with full weight-bearing while wearing a shoe with an ankle stirrup-brace, and physical therapy is started to strengthen the muscles of the leg (Figs. 7-A through 7-D).

**Arthrodesis**

An arthrodesis is indicated for a patient who has dysfunction of the posterior tibial tendon as well as pain in the lateral part of the foot, a rigid (stage-III) valgus deformity of the hindfoot, or a valgus deformity of the hindfoot that, when corrected to neutral, is associated with a fixed supination deformity of the forefoot. Numerous procedures have been described, including isolated talonavicular\(^\text{11,14,28}\), talonavicular and calcaneocuboid\(^\text{7}\), subtalar\(^\text{28}\), and triple arthrodesis\(^\text{19,52,55}\). According to Johnson and Lester\(^\text{28}\), it does not make much difference which procedure is performed because fusion of any one of the joints of the hindfoot effectively blocks motion of the hindfoot. They noted that it is important not to disturb the position of the calcaneocuboid and talonavicular joints when performing a subtalar arthrodesis; however, this is not always possible, particularly if the deformity is rigid and a fixed supination deformity of the midfoot and forefoot occurs when the subtalar joint is reduced into a neutral position. In their technique, a morselized bone graft obtained from the anterior part of the iliac crest is inserted into the anterior aspect of the subtalar joint. Temporary fixation was achieved with a Steinmann pin introduced percutaneously from the neck of the talus dorsally into the calcaneus.

Although triple arthrodesis has a long history of success for the treatment of neuromuscular deformities in children, fewer reports are available on its effectiveness in adults, particularly with respect to the treatment of planovalgus deformity of the hindfoot. A triple arthrodesis is indicated for a patient who has a fixed hindfoot deformity that is associated with pain in the lateral part of the foot. The goals of the triple arthrodesis are to realign the hindfoot; to establish a plantigrade weight-bearing surface; to maintain the integrity of the adjacent joints, particularly the ankle; and to avoid incisional neuromas.

The technique of triple arthrodesis should be meticulous. The use of a single incision on the lateral aspect of the foot places both the sural nerve and the lateral cutaneous branch of the superficial peroneal nerve at risk for incisional neuromas, and it does not allow visualization of most of the talonavicular joint\(^\text{1}\). Therefore, two incisions should be used. The first incision parallels the inferior surface of the peroneal tendons from the tip of the fibula over the sinus tarsi; the second incision lies medial to the anterior tibial tendon and extends from the tibiotalar joint to the naviculocuneiform joint. Laterally, the contents of the sinus tarsi are excised and the joints are denuded of articular cartilage with the use of a sharp osteotome or chisel. The correction is achieved by adequate subtalar and talo-
ADULT ACQUIRED FLATFOOT DEFORMITY

FIG. 8-C

Figs. 8-C and 8-D: Postoperative radiographs showing correction of the deformities.

FIG. 8-D

It is particularly important to identify the recess between the navicular and the cuboid, at which point all four bones (the talus, the navicular, the calcaneus, and the cuboid) may come into contact with each other. If osseous bridging can be obtained in this location, the foot will be stable even if there are radiographic signs of non-union of one of the triple joints. Jahss et al. called this concept a quadruple arthrodesis. Although they described this procedure with the use of cancellous bone graft, no internal fixation was used. Bone graft is not necessary if rigid internal fixation is used to stabilize the arthrodesis.

I prefer to achieve internal fixation with compression screws and have found it unnecessary to use bone graft. I have found the prevalence of non-union associated with this technique to be negligible, provided that broad surfaces of bleeding bone are present and that rigid compression fixation is achieved (Figs. 8-A through 8-D). In a recent institutional review of 143 triple arthrodeeses performed between 1986 and 1994, my colleagues and I found that only one non-union of the talonavicular joint had occurred.

Some clinicians advocate fixing the talonavicular joint first; others believe that the subtalar joint should be stabilized first. It is my belief that realigning the medial column at the talonavicular joint is the key to aligning the rest of the foot. The forefoot is adducted and plantar flexed across the talonavicular joint, and that joint is held secure with a guide pin for a cannulated screw. Talonavicular correction should result in passive correction of the subtalar joint, which ideally should be aligned and stabilized in slight valgus. When stabilizing the calcaneocuboid joint, it is very important to elevate the cuboid forcefully to prevent any inferior sag that later may create pain along the lateral surface of the foot with weight-bearing.

It is far preferable to leave the foot in slight valgus than in neutral or varus, despite the possibility that impingement may occur between the fibula and the calcaneus in some patients who have more advanced deformity. One must be careful not to overcorrect the foot by internally rotating the subtalar joint and adducting the forefoot severely at the transverse tarsal joint. In contrast, an in situ arthrodesis that leaves the calcaneus in marked valgus will place undue stress on the tibiotalar joint. On the basis of these findings, Jahss recommended adding a medial displacement osteotomy of the calcaneus to a triple arthrodesis in the presence of a fixed deformity of the hindfoot. Such a medial displacement osteotomy may normalize the weight-bearing surface of the hindfoot and improve the distribution of

FIG. 9

Radiograph of a sixty-three-year-old woman, made twenty-eight months after a triple arthrodesis, showing valgus angulation of the talus.
Figs. 10-A through 10-D: A forty-eight-year-old man who had stage-II dysfunction of the posterior tibial tendon that was associated with pain in the lateral part of the foot and a flexible hindfoot deformity. The patient was managed with bone-block distraction arthrodesis of the calcaneocuboid joint and transfer of the flexor digitorum longus tendon.

Figs. 10-A and 10-B: Preoperative radiographs. Note the abduction of the forefoot on the anteroposterior radiograph (Fig. 10-A).

weight-bearing forces across the medial part of the ankle, thereby reducing the stress on the tibiotalar joint and the deltoid ligament. This is certainly an option for the treatment of a fixed valgus deformity that is associated with valgus tilting of the ankle, but every effort should be made to reduce the hindfoot deformity intraoperatively.

Unfortunately, the results of triple arthrodesis in older adults who have flatfoot deformity are disappointing, as many of these patients have later problems with the ankle joint. Graves et al. found that triple arthrodesis in older patients eliminated or decreased the pain, but they cautioned that this procedure should be used only in a salvage attempt for fixed deformity because of the high rate of postoperative complications. With severe deformity, the hindfoot may become rigidly fixed in valgus and the talus may begin to tilt into valgus in the tibiotalar joint, perhaps as a result of attritional changes in the deltoid ligament. Triple arthrodesis fails in many of these patients (Fig. 9). Johnson and Lester were perhaps the first to recognize this fact, and they...
recommended a primary tibiotalocalcaneal arthrodesis to address the problem.

The use of an arthrodesis to lengthen the lateral column has been successful for the treatment of severe planovalgus deformity of the foot, with excellent results and long-term maintenance of correction. This procedure is indicated for a patient who has a valgus deformity that is not fixed (stage-II disease). Options for bone graft include autogenous graft from the iliac crest or allograft. While the use of allograft is far easier and involves less morbidity, it is important to advise patients of its greater potential for non-union. I prefer to use a femoral-head allograft, contouring a trapezoidal block of bone in two planes. The trapezoid is fashioned so that it is wider (twelve millimeters) dorsally and laterally and narrower medially and inferiorly. On the plantar surface, the trapezoid tapers to eight millimeters on both sides to achieve adduction of the midfoot and plantar flexion of the hindfoot (Figs. 10-A through 10-D). The lengthening arthrodesis of the calcaneocuboid joint is facilitated with use of a smooth lamina spreader (without teeth), which can be removed smoothly from the joint as the bone graft is tamped into place. Although securing the bone graft may not be necessary as it is inserted under compression, I prefer to use either a cannulated screw or a percutaneous Kirschner wire to hold it in place.

Conclusions

The options for non-operative and operative treatment of acquired flatfoot deformity in adults are numerous. Although each classification or treatment regimen has its flaws, the steps described should help the clinician to make an appropriate treatment decision.

References