

Tibialis Posterior Tendon Dysfunction

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Dysfunction of the tibialis posterior tendon evolves through a series of stages. The pain symptoms, clinical signs, and roentgenographic changes for each of these stages are characteristic. This staging system permits clarification and individualization of dysfunction, expected pathologic changes, and surgical treatment. The importance of the tibialis posterior tendon in normal hindfoot function and its treatment when injured are now being properly appreciated.

Afflictions of the tibialis posterior tendon (TPT) are now being recognized and treated with increased frequency and success.^{7,8,11,12} It is important to be aware of this problem and its stages of presentation in order to make a proper diagnosis. This article reviews the evolving concepts of presentation and formulates a plan of rational care.

DIAGNOSTIC AND RADIOGRAPHIC CONSIDERATIONS

The stages of TPT dysfunction are quite distinct (Table 1). Each stage will be discussed with regard to pain symptoms, physical findings, and roentgenographic changes.

STAGE I—TENDON LENGTH NORMAL

Many TPT problems remain unrecognized because pain symptoms are only mild to moderate. The patient may, on multiple oc-

casions, have only an aching along the medial aspect of the ankle that is exacerbated by physical activity and has probably modified his or her activities to be less strenuous. Although it may be difficult for patients to localize the discomfort, with specific questioning they will point along the course of the TPT from a few centimeters proximal to the tip of the medial malleolus to its major attachment at the undersurface of the navicular. The onset of pain will have been gradual, and only infrequently can an inciting episode be recalled. Occasionally, a young athlete will remember a twisting episode with subsequent persistent pain.

On examination, the points of maximal tenderness will be detected along the tendon from just before it passes around the medial malleolus to its navicular insertion. When localized, the site of tenderness corresponds well to areas of TPT pathologic changes. Swelling is best appreciated by viewing the standing patient from a posterior vantage. Fullness of the region just inferior to the medial malleolus is evident when compared with the unaffected foot. The alignment of the hindfoot-forefoot will still be normal at this stage.

In the authors' experience, manual testing for weakness has been essentially useless. It has been suggested² that testing the TPT with the hindfoot in eversion and the forefoot in abduction with toe flexors relaxed will eliminate the synergistic action of the tibialis anterior and flexor digitorum longus, thus allowing determination of the strength of the TPT. However, because the TPT power is so strong, and probably because of substitution

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TABLE 1. Changes Associated With Various Stages of TPT Dysfunction

	<i>Stage 1</i>	<i>Stage 2</i>	<i>Stage 3</i>
TPT condition	Peritendinitis and/or tendon degeneration	Elongation	Elongation
Hindfoot	Mobile, normal alignment	Mobile, valgus position	Fixed, valgus position
Pain	Medial: focal, mild to moderate	Medial: along TPT, moderate	Medial: possibly lateral, moderate
Single-heel-rise test	Mild weakness	Marked weakness	Marked weakness
"Too-many-toes" sign with forefoot abduction	Normal	Positive	Positive
Pathology	Synovial proliferation, degeneration	Marked degeneration	Marked degeneration
Treatment	Conservative, 3 months; surgical, 3 months with synovectomy, tendon debridement, rest	Transfer FDL* for TPT	Subtalar arthrodesis

* Flexor digitorum longus.

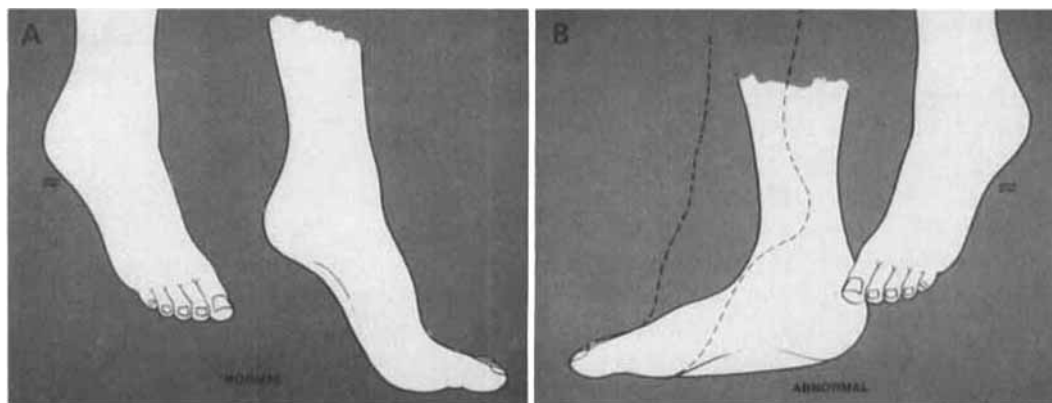
by the other extrinsic muscle-tendon units in spite of efforts to negate them, this method of testing has not been accurate when correlated with surgical findings.

The single-heel-rise test is very good for testing TPT strength. The patient is asked to rise up on the ball of the foot while the other foot is held off the ground (the patient may use a door or wall for balance). The normal sequence for a single-heel rise is as follows. First, the TPT is activated, which inverts and locks the hindfoot, thus providing a rigid structure. Next, the gastrosoleus muscle group pulls up the calcaneus and the heel rise is completed. With elongation of the TPT, however, the initial heel inversion is weak and the patient either rises up incompletely without locking the heel or does not get up on the ball of the foot at all (Figs. 1A and 1B). In the Stage 1 condition, the patient usually will be able to get up on the ball of the affected foot and will be able to tell that it is more painful and somewhat weaker than the other side. At this early stage, there will not be

much in the way of secondary deformity, and overall forefoot-hindfoot alignment will be unaffected. On routine standing roentgenograms the changes will be minimal. If the diagnosis is in question, magnetic resonance imaging (MRI) may demonstrate if tendon degeneration is present.

STAGE 2—TENDON ELONGATED, HINDFOOT MOBILE

From Stage 1 to Stage 2 the change in symptoms evolves over several months to years. During Stage 1, when pain is mild to moderate, patients have often seen a physician but were not given a specific diagnosis. Often these patients have been told to live with the discomfort. In Stage 2, the pain increases in severity and distribution, is present even after cessation of weight bearing, and is significantly troublesome. The patient has already applied for a handicapped parking sticker and actively wants relief. Pain is located along the TPT for a greater length. The



FIGS. 1A AND 1B. The single-heel-rise test. (A) Normal. The TPT inverts the hindfoot and the patient rises on the forefoot. (B) Abnormal. Ability to rise on the forefoot is decreased or absent. Instead, the patient just rolls to the outside of the foot. (By permission of Mayo Foundation.)

tendon has been disrupted, and secondary changes are developing. Swelling with tenderness is still present inferior to the medial malleolus when viewed posteriorly.

The single-heel-rise test becomes even more abnormal because the tendon is weakened. Another helpful diagnostic sign is that of "too many toes," *i.e.*, seeing more toes than normal from this view (Fig. 2). In this test, the patient is asked to assume a comfortable knee-leg alignment toward a wall. From

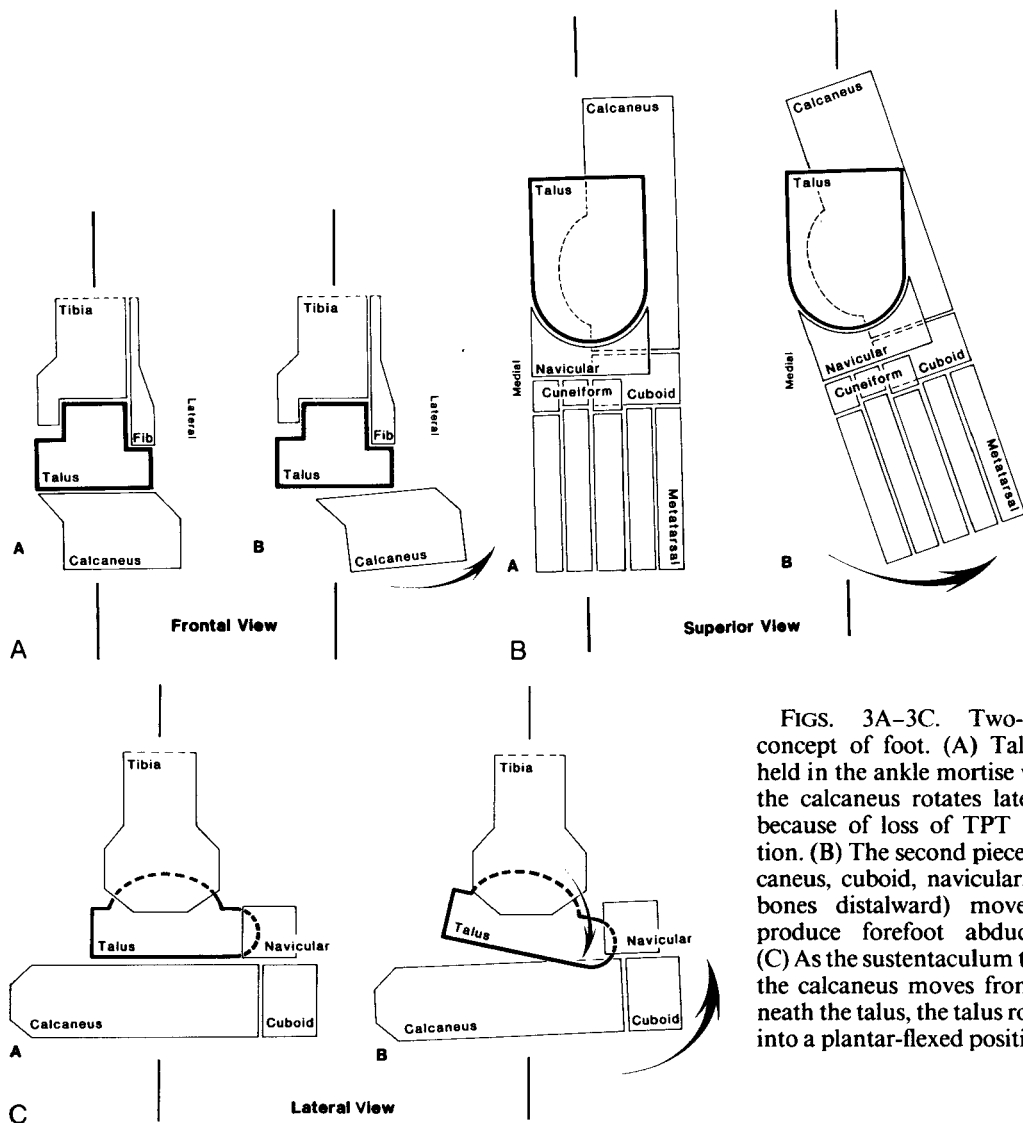


FIG. 2. "Too many toes." This patient had right TPT dysfunction. Four toes show on the right with forefoot abduction, but only two toes show on the normal left foot. (From Johnson.⁸ By permission of J. B. Lippincott.)

a direct posterior midline vantage, the examiner counts the number of toes on each foot that are visible laterally. As the heel goes into increased resting eversion and the forefoot goes into abduction, too many toes are seen on the affected side. The number of extra toes seen is a recordable measurement of the degree of deformity and is surprisingly reproducible.

Changes on routine standing roentgenograms now become evident. To understand these changes, it is useful to think of the foot as consisting of only two pieces (Figs. 3A–3C). One piece, the talus, is fixed in the ankle mortise. It can move only in flexion–extension, not in a varus–valgus plane. The second piece of the foot is everything else, *i.e.*, the calcaneus, cuboid, navicular, and bones distalward move as a unit with motion being in a varus–valgus plane through the subtalar joint. With elongation of the TPT, the second piece rotates from beneath the talus laterally to produce a hindfoot in valgus and a forefoot in abduction. When the head of the talus is left unsupported, the talus flexes.

The changes on routine roentgenograms are then predictable. On the anteroposterior (AP) view, the forefoot will be abducted in relation to the hindfoot. Also, the navicular will have subluxed off the head of the talus and



FIGS. 3A-3C. Two-piece concept of foot. (A) Talus is held in the ankle mortise while the calcaneus rotates laterally because of loss of TPT function. (B) The second piece (calcaneus, cuboid, navicular, and bones distalward) moves to produce forefoot abduction. (C) As the sustentaculum tali of the calcaneus moves from beneath the talus, the talus rotates into a plantar-flexed position.

the angle between the long axes of the talus and the calcaneus will be increased. In a lateral view, there will be sagging at the talus-navicular joint and divergence of the long axis of the talus from the long axis of the calcaneus.

If the diagnosis is in question, MRI¹ can show a tendon discontinuity as well as a proximal balling-up of the tendon (Fig. 4). At first, it might be thought that a tenogram would be

helpful.⁵ In the authors' experience, however, this is not the case. Either the diagnosis is so evident that it is not necessary or in cases of difficult diagnosis the tenogram is equivocal.

STAGE 3—TENDON ELONGATED, HINDFOOT DEFORMED AND STIFF

The pain may transfer to the lateral aspect of the hindfoot and be located over the sinus

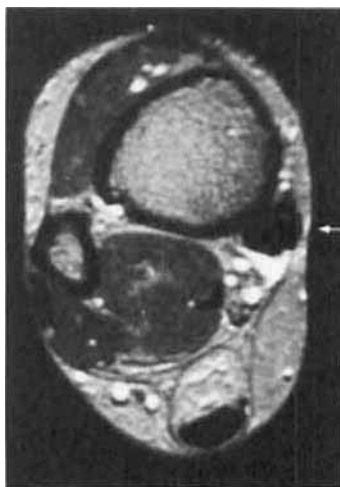


FIG. 4. MRI shows the balled-up tendon (arrow) just behind the medial malleolus. (From Alexander *et al.*¹ By permission of the American Orthopaedic Foot and Ankle Society.)

tarsi because the tendon is now completely disrupted and intrinsically less painful. This has allowed the hindfoot to go into eversion. As this occurs, the bone projection from the inferior surface of the talus at the anterior



FIG. 5. Acquired pes planus as a late result of TPT rupture. (From Johnson.⁸ By permission of J. B. Lippincott.)

margin of the posterior facet will impinge on the superior aspect of the calcaneus in the sinus tarsi. Pressing on the sinus tarsi will reproduce the patient's pain symptoms. In Stage 3, the pain also may be more suggestive of degenerative arthritis, exhibiting an activity-related sharp pain that is present long after the activity ceases.

Deformity is the most prevalent change. When viewed posteriorly, the fullness beneath the medial malleolus may not be so evident, but the hindfoot eversion and forefoot abduction are significant. The single-heel-rise test will demonstrate the absence of locking hindfoot inversion along with a diminished ability to rise on the ball of the foot. Also, too many toes will be seen. At this stage, the foot appears severely flat (Fig. 5).

The hindfoot valgus and forefoot abduction will be seen on standing AP and lateral roentgenograms, much the same as in Stage 2 but more marked. Secondary degenerative changes with joint narrowing and osteophyte formation may also appear in the subtalar, talonavicular, and calcaneocuboid joints.

If that anterior margin of the posterior facet projecting inferiorly off the talus is impinging on the superior aspect of the calcaneus, sclerosis will appear in the midsuperior calcaneus at the site of impingement. This bony condensation is called the "sinus tarsi impingement sign."

If a technetium scan is done, the delayed views will show uptake at the site of sinus tarsi impingement as well as at sites of secondary degenerative arthritis.

PATHOLOGIC AND TREATMENT CONSIDERATIONS

With an understanding of the diagnostic signs and symptoms along with roentgenographic abnormalities as presented above, the pathologic changes can be anticipated and a treatment program offered.

STAGE 1—TENDON LENGTH NORMAL

At this stage, the length of the tendon appears normal. This early presentation, how-

ever, is the most varied and hardest to describe and explain. In some instances, there is almost a pure peritendinitis. That is, when the TPT retinaculum is opened, much clear amber synovial fluid will escape.¹³ The tendon itself will look and feel essentially normal, but a luxuriant synovial proliferation will be present within the tendon sheath. In other cases, there will be minimal synovial fluid and synovial thickening; however, in this case the tendon, in a location just distal to the medial malleolus, will be firm and have an off-white color (Fig. 6A). There may be some longitudinal split tears within the tendon substance, and it may be enlarged in a bulbous configuration to almost twice the normal tendon width.¹⁷ This would be a pure tendon degeneration. Combinations of peritendinitis and degeneration may also be seen. Still, the tendon length appears normal. It is unclear whether the different presentations represent different causes, *e.g.*, peritendinitis from systemic inflammatory disease or degeneration from acute traumatic interstitial tears.

The current treatment in this situation (Fig. 6B) is to open the sheath from the TPT musculotendinous junction all the way to its insertion, leaving only a 1-cm pulley just posterior to the medial malleolus.¹⁸ Synovectomy is performed and the tendon is debrided. If the tendon is enlarged to more than 1.5 times its normal size, a wedge is removed from the substance to debulk the tendon and the gap is sutured closed.¹⁷ Small flap tears are debrided and larger tears are sutured. As the wound is closed, some betamethasone is left around the tendon. A short-leg walking cast is then applied and worn for three weeks.

This treatment method seems to be successful in stopping the inflammation and tendon degeneration that would otherwise proceed to Stage 2 changes. In a few cases, excising or reattaching the tendon was not satisfactory.⁴

It is important that the patient with Stage 1 physical findings does not progress to Stage 2 or 3 with conservative care. For three to six

months, trials of antiinflammatory agents, rest, arch supports, and perhaps medial shoe wedges or orthotics may be appropriate. Because steroid injection has been implicated in tendon weakening,³ its use is not suggested. If improvement does not occur, then the surgical treatment just outlined should be used to break the inflammation cycle. This applies also to patients with rheumatoid arthritis in whom loss of TPT function and subsequent deformity can be as disabling as some large joint involvement.

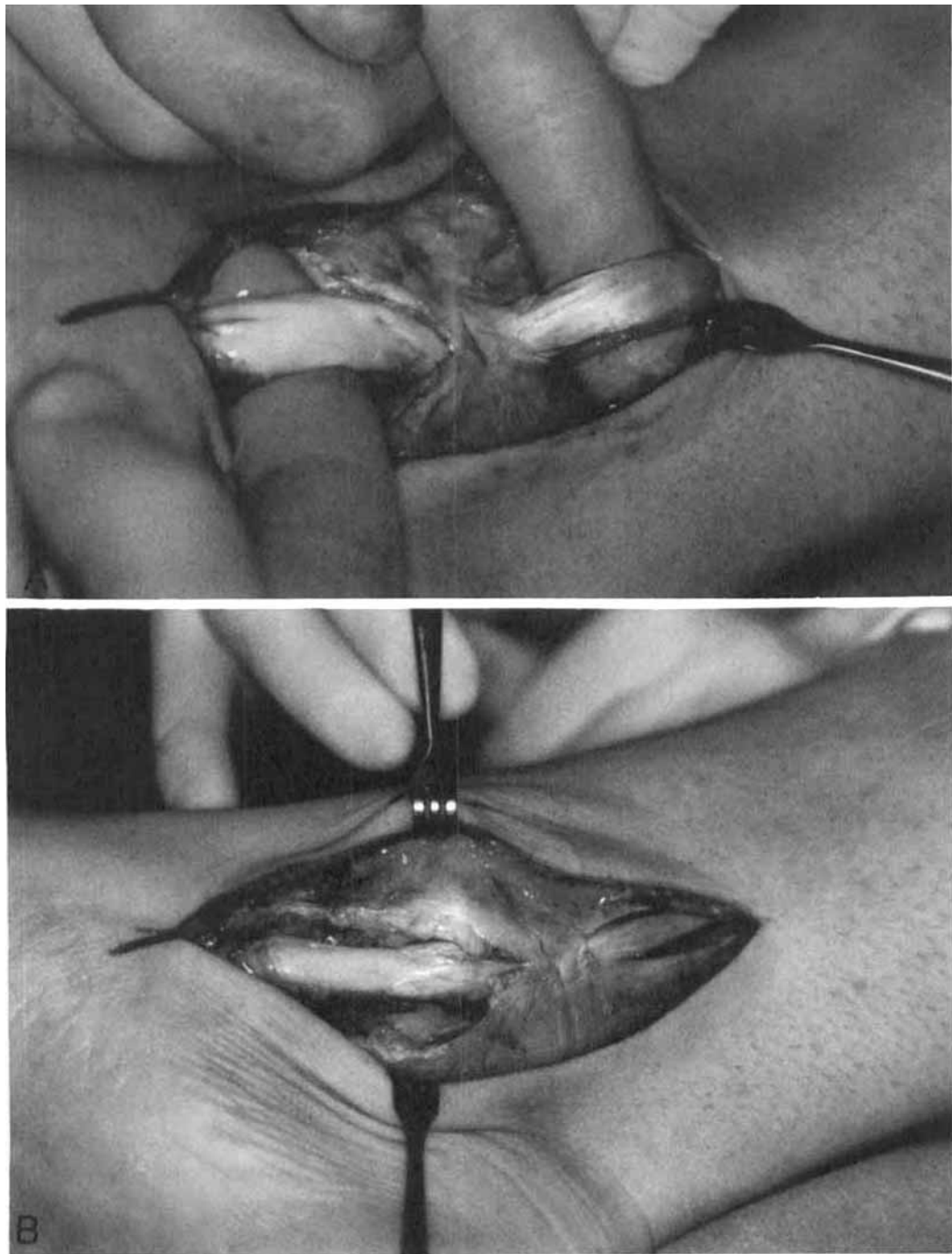
STAGE 2—TENDON ELONGATED, HINDFOOT MOBILE

In this situation, the tendon may show marked degeneration over several centimeters. The tendon is enlarged and has multiple longitudinal tears with secondary adhesions to the tendon sheath. It will be a varied yellowish white-brown color and firm in consistency. Proximal to the directly involved region, the tendon will have a peculiar white, fish-flesh appearance if the tear is old and tension has not been transmitted through the tendon for some time. In other situations, there will be a single complete transverse tear of the tendon with rounding off of the tendon ends. The tendon that remains will then have the off-white, fish-flesh appearance.

When the TPT has been elongated, the flexor digitorum longus (FDL) is transferred to substitute for the TPT.^{4,6,8,12} This transfer entails detaching the FDL distally and reinserting it into the undersurface of the navicular through a drill hole (Figs. 7A–8G).

The distal portion of the FDL does not need to be tenodesed to the adjacent flexor hallucis longus tendon. The intrinsic toe flexors are so good in the foot that leaving the distal stump alone will cause no functional loss in lesser toe function later. Avoiding this tenodesis also allows a greater length of FDL to be used for transfer.

Should the FDL be left in its own sheath or rerouted through the flexor hallucis longus



FIGS. 6A AND 6B. Medial view of right foot. (A) TPT is exposed, leaving a pulley just posterior to the medial malleolus. The off-white discoloration of the tendon just distal to the medial malleolus is seen. (B) At completion of the surgical procedure, the tendon sheath has been released and synovectomy is completed.

sheath? It does not make sense to reroute a healthy tendon through a diseased tendon sheath. The FDL tendon is probably strong enough for substitution. The anatomic cross-sectional area of the FDL is only about one-third that of the TPT,¹⁵ but it seems to be holding up well clinically. Perhaps it hypertrophies.

The FDL should be pulled as tight as reasonably possible. With the ankle in equinus and the forefoot in varus, the tendon is pulled quite taut and sutured distally. (There is no anatomic rationale for this but it seems to work.)

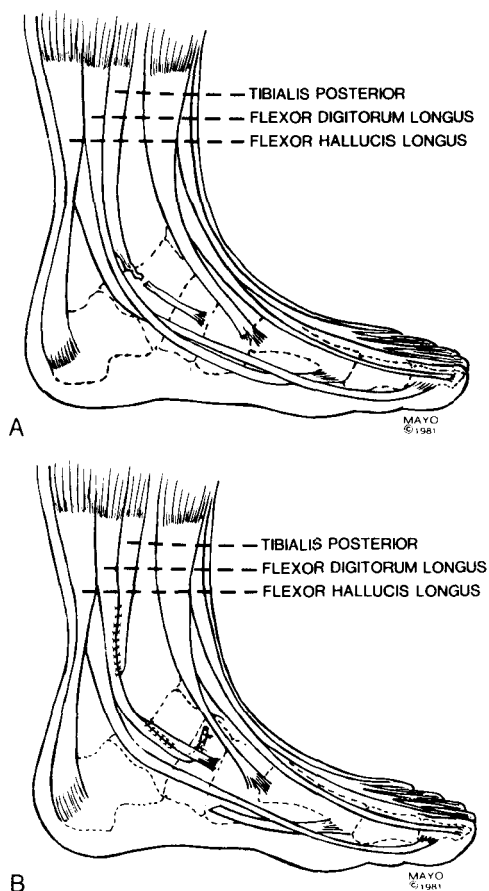
A proximal attachment of the TPT muscle should not necessarily be made to the FDL. If, when the proximal stump of the TPT is pulled, the muscle seems to be nonyielding and fibrotic, the proximal attachment is not done because the muscle is nonfunctional.

A soft-tissue static transfer is also usually unnecessary. Although some authors⁶ have advocated reefing the talonavicular capsule and the calcaneonavicular (spring) ligament, this has not been done as a part of the repair procedure. Suturing of the FDL to the under-surface of the talonavicular region may accomplish this to some degree. Static transfers alone, historically, have not been successful in maintaining the arch of the foot.

A negative exploration for a suspected TPT dysfunction has become more and more infrequent. Usually what is found at operation is more extensive than suspected clinically. It is necessary to carry the surgical dissection all the way to the TPT insertion where the tearing and elongation may be located.

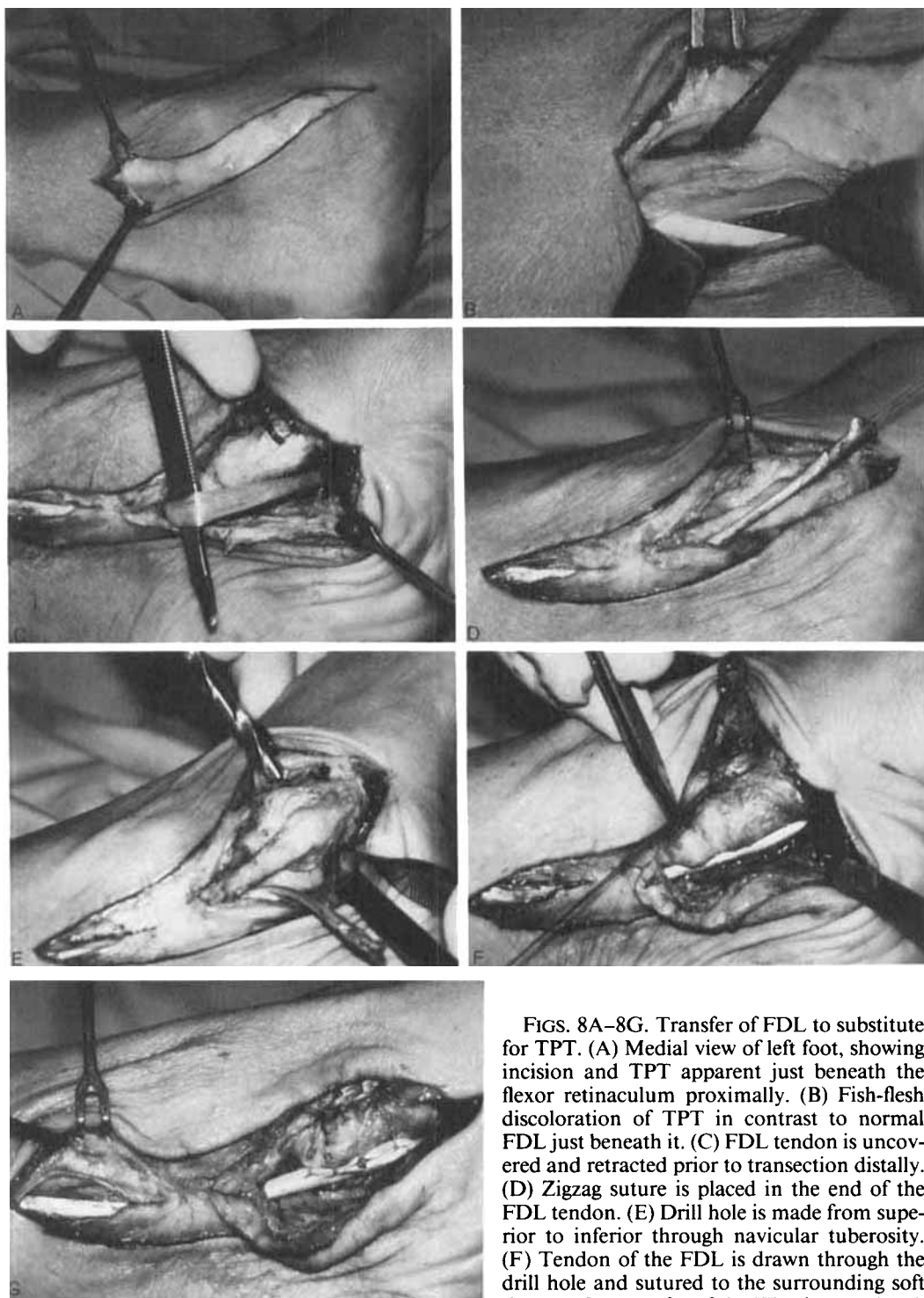
The results of transfer of the FDL for the elongated TPT have been quite good.^{4,12} Initially, resuturing or reattachment of the elongated tendon was recommended but the results were not satisfactory. Because the FDL seems to be so expendable, liberal use of this transfer seems reasonable.

There are two signs that will indicate a complete disruption of the TPT before the whole tendon is visualized. The first is the white sign. If, when the sheath of the TPT is



FIGS. 7A AND 7B. Diagrams showing (A) the discontinuity in the TPT and the position of adjacent tendons and (B) the transfer of the FDL to substitute for the TPT. (By permission of Mayo Foundation.)

opened several centimeters proximal to the medial malleolus, the tendon appears white compared to the adjacent FDL, the tendon will be completely disrupted distally. The second is the resident sign and is only possible when practicing in a training program with residents. If, upon entering the operating room one hears the resident muttering "Tom, Dick, and Harry," followed by the statement that the patient does not have a TPT, then the tendon is torn. The reason is that the tendon has changed color and is scarred closely behind the tibia and has not yet been correctly identified.



FIGS. 8A–8G. Transfer of FDL to substitute for TPT. (A) Medial view of left foot, showing incision and TPT apparent just beneath the flexor retinaculum proximally. (B) Fish-flesh discoloration of TPT in contrast to normal FDL just beneath it. (C) FDL tendon is uncovered and retracted prior to transection distally. (D) Zigzag suture is placed in the end of the FDL tendon. (E) Drill hole is made from superior to inferior through navicular tuberosity. (F) Tendon of the FDL is drawn through the drill hole and sutured to the surrounding soft tissues. (G) Transfer of the FDL is completed.

Distally, the transferred tendon runs beneath the TPT and is sutured to it. Proximally, tenodesis of the TPT to the FDL was not done because of the fibrotic changes within the tibialis posterior musculotendinous structure.

STAGE 3—TENDON ELONGATED, HINDFOOT DEFORMED AND STIFF

The tendon changes are much the same as in Stage 2. The static supports of the foot also have been damaged, and fixed flatfoot has developed. A dynamic tendon transfer against a fixed deformity is not reasonable. Instead, a realignment followed by an arthrodesis is done. Controversy rages as to whether subtalar arthrodesis,¹⁶ talonavicular arthrodesis,¹⁶ talonavicular with calcaneocuboid arthrodesis,¹² or triple arthrodesis should be done. In fact, it probably does not make much difference. Arthrodesing any one of the hindfoot joints will effectively block hindfoot motion. It is important that the method of arthrodesis not malalign the adjacent joints.

The authors' personal preference is for an isolated subtalar arthrodesis in most cases. Based on the two-piece concept of the foot (Fig. 3), the talus and everything else distally, with the subtalar arthrodesis being the everything else, is repositioned beneath the talus. The subtalar arthrodesis should not disrupt the relative positions at the talonavicular and calcaneocuboid joints.

For the subtalar arthrodesis, a morcellated bone graft from the anterior iliac crest is inserted into the subtalar joint with temporary fixation by a Steinmann pin across the neck of the talus into the calcaneus.

An arthrodesis with ten weeks of cast immobilization and then gradual recuperation may seem a bit drastic. But the FDL tendon transfer requires six weeks of casting followed by an even longer period of recuperation. If there is any question as to which of the two should be done, based on the degree and rigidity of hindfoot change, the authors select the arthrodesis.

DISCUSSION

The tibialis posterior muscle-tendon unit is a prime stabilizer of the hindfoot. By virtue of its position posterior to the axis of the ankle joint and medial to the subtalar axis, it provides plantar flexion at the ankle and in-

version of the hindfoot. The tendon excursion is short and the muscle is powerful. Thus, elongation of the tendon will decrease its function significantly.

The recent concepts of the TPT difficulties have evolved through the efforts of multiple authors. Two particularly important contributions were the paper by Kettelkamp and Alexander⁹ clearly describing the problem, and that by Goldner *et al.*⁶ suggesting tendon transfers. Since then, the experience and variations of others have been added. It is interesting to speculate that perhaps the most efficacious pain relief aspect of the tendon transfer procedures is really the release of the TPT sheath. Earlier^{10,14,18} it was reported that release of the sheath of the TPT alone was satisfactory. Still, it is reasonable to add the strength of the FDL to substitute for the TPT and to avoid progression of the flatfoot deformity.

When this information is presented at a meeting someone will inevitably say that he or she has been in practice for 20 years and has never seen a case of TPT dysfunction. Then, a few weeks later, this same doctor surprisingly finds his or her first case. These patients are out there and can be helped.

There is probably also a Stage 4 TPT dysfunction. This is when the hindfoot has become fixed in eversion, over a number of years, to produce a valgus tilt of the talus within the ankle mortise and lateral tibiotalar degeneration. This pattern may be seen without prior history of trauma. For these patients, an arthrodesis from the tibia to the calcaneus can be done. With awareness of the TPT problem, such an extensive reconstructive procedure could have been avoided.

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