CHAPTER **4**

PLANTAR PLATE DYSFUNCTION

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INTRODUCTION

The surgical management of second metatarsophalangeal (MTP) joint instability remains a dilemma to the foot and ankle surgeon despite numerous articles and research devoted to detailing the anatomy and surgical correction of this pathology. Although several authors believe that the plantar plate is the primary static stabilizing structure of the lesser digit, few have advocated its direct repair.[1] The development of chronic subluxation and dislocation at the lesser MTP joint level has been attributed to several conditions including hallux valgus and rheumatologic joint disease, however, it is likely the etiology is due to a mechanical phenomenon secondary to excessive weight-bearing stresses.[1-5] The diagnosis of plantar plate dysfunction (PPD) requires a high index of clinical suspicion and a detailed history and physical exam. Numerous operative techniques, both soft tissue and osseous, have been proposed for the correction of this deformity, however the flexor tendon transfer continues to be the procedure of choice in light of inconsistent outcomes and a significant incidence of postoperative stiffness.[2-8] This paper will provide a discussion of plantar plate pathology including the pathoanatomy and the authors' current surgical approach to, and indications for direct repair as the primary procedure for the correction of second MTP joint instability.

DEFINING PLANTAR PLATE DYSFUNCTION

PPD is attrition or frank rupture of the plantar plate leading to plantar MTP joint instability with sagittal and frequently transverse plane subluxation and dislocation. Several classification schemes have been introduced into the literature to describe the stages of MTP joint instability.[5,9] We have divided plantar plate dysfunction into stages based on subjective and objective clinical findings and the presence or absence of a positive Lachman's test (Figure 1).

In 1995, Yu and Judge introduced the phrase predislocation syndrome (PDS) to describe the early prodromal phase of plantar plate dysfunction.[9] Subjectively, patients with PDS may describe a dull aching or throbbing pain, or perceive the sensation of fullness beneath the MTP joint and digit. As Yu notes, the magnitude of pain and the physical examination findings are significantly disproportionate. Typically, there is no history of prior injury or trauma and patients may describe a highly active lifestyle, including walking, running, or aerobics. Early clinical presentation may include significant pain and little, if any malalignment or instability present at the MTP joint. Exquisite point tenderness is localized plantarly, just distal to the MTP joint at the insertion of the plantar plate apparatus into the proximal phalanx base. Mild edema secondary to capsular distention and synovitis may be present. Although the Lachman's test will be negative indicating intact periarticular structures, the dorsal–plantar translational stress will produce significant discomfort. This category of patients will likely respond well to conservative measures of therapy,

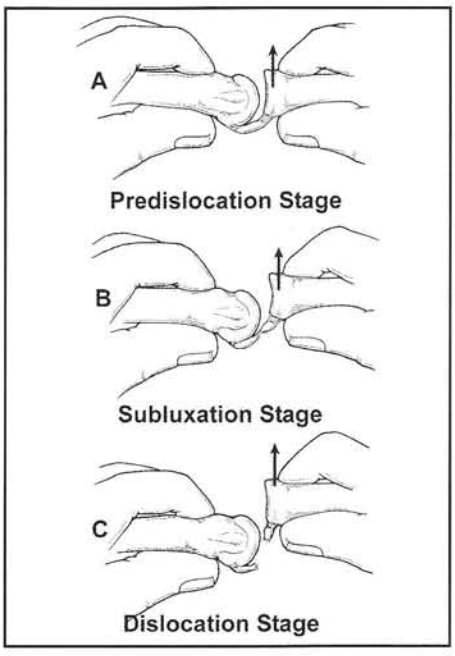


Figure 1.

including crossover digital strapping, orthoses, nonsteroidal-antiinflammatory drugs, physical therapy, and modifications in exercise regimens.

As the deformity progresses joint subluxation and instability become apparent as plantar plate dysfunction ensues. In this stage, the clinical symptoms are intensified and the plantar plate demonstrates laxity, attenuation and partial rupture indicated by a positive Lachman's test. Additional clinical features include the onset of dorsal joint pain with moderate edema and capsular distention resulting from the accelerating inflammatory cascade. As the syndrome evolves, patients may discern a gradual dorsal elevation and loss of toe purchase or hammering of the affected digit resulting from MTP joint destabilization. Arthrosis may be seen on radiographs, resulting from dorsal impingement of the proximal phalanx and the metatarsal head. The transition from PDS to PPD may occur within a relatively short time frame of weeks to months, and the onset of contralateral symptoms is not infrequent.

The dislocation stage of PPD demonstrates complete lack of structural integrity with total joint luxation. As with the subluxation stage, Lachman's test is positive producing significant discomfort, and multiplanar instability is commonly present. Radiographs demonstrate superior and frequently medial luxation of the joint. Mild to severe MTP joint arthrosis is usually present secondary to long-standing joint malposition. Moderate edema is present obscuring the view of the extrinsic extensor tendons and is a manifestation of the underlying chronic inflammation present within the joint and periarticular soft tissue structures. Both the subluxation and dislocation stages of PPD usually require surgical reconstruction.

Although the stages described above detail the sagittal plane components of the deformity, the transverse plane element adds tremendous complexity to the overall situation. The focus of this article is the repair of the plantar plate as the primary procedure, additional adjunctive procedures (i.e. EDB/FDL tendon transfers or osteotomies) may be used to achieve the necessary multiplanar correction.

SURGICAL ANATOMY OF THE PLATE-LIGAMENT COMPLEX

A brief discussion of the plantar plate and its associated soft tissues is warranted to better understand the surgical anatomy. The components of the complex that appear to be directly involved in the pathology are the plantar plate and the collateral ligaments, which are the primary static stabilizers of the lesser MTP joint.[12] The plantar plate is a flexible, but sturdy fibrocartilaginous structure composed primarily of type I collagen which is also the central constituent found in the knee menisci. Its architecture is such that it is designed to withstand both compressive and tensile loads.[10] The plate possesses a loose origin from the metatarsal head; however it has strong attachments to the proximal phalanx base. It receives insertional fibers from the lumbrical and interosseous tendons and is contiguous with the intervening segments of the deep transverse intermetatarsal ligament. In addition, the plantar plate also supplies the most significant distal attachment of the plantar fascia.[10]

The structure of the collateral ligaments and their intimate association with the plantar plate has been well described in the literature.[10-13] The ligamentous anatomy of the lesser MTP joint is analogous to the first MTP joint and is similarly stabilized by the proper collateral ligaments (PCL) and accessory collateral ligaments (ACL)[14] (Figure 2). The PCL provide primarily transverse plane stability, and their insufficiency has been linked to crossover

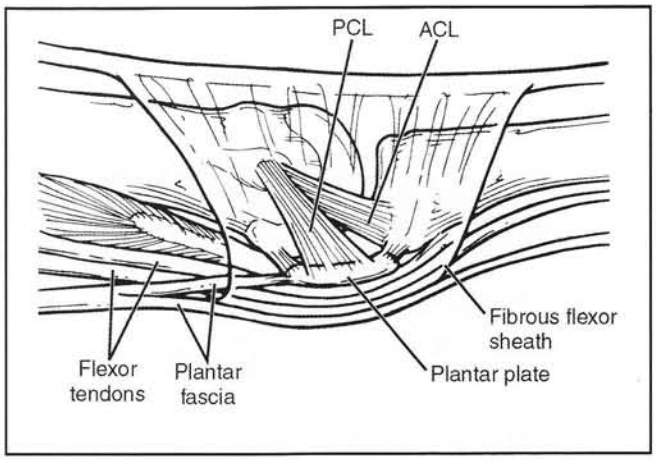


Figure 2.

toe deformities.[15] The ACL in the lesser MTP joint are anatomically comparable to the sesamoidal suspensory ligaments in their structure and orientation, providing direct attachment between the metatarsal head and the plantar plate. The plate along with the medial and lateral capsular ligaments (PCL/ACL) creates an articular unit or sling for the metatarsal head.

Plantarly, the flexor tendons are bound to the plantar plate by a specialization of the deep fascia known as the fibrous flexor sheath. This sheath serves as a retinacular structure that adheres to the outer edges of the plantar plate and attaches superiorly to the extensor hood apparatus, effectively encasing the phalanx. The flexor tendons articulate on the inferior aspect of the plantar plate that is the only separation between the tendons and the lesser MTP joint.

PATHOANATOMY OF PPD

The pathoanatomy of plantar plate derangement has been well-documented.[15-17] Yao et al has demonstrated, through the use of MRI that the focal point of plantar plate ruptures occurs directly beneath the metatarsal head adjacent to its distal attachment on the phalanx. Our surgical experience strongly agrees with this finding. Those cases that warrant surgical repair have demonstrated significant attritional changes and thinning of the plate upon intraoperative inspection making repair more difficult. The defect within the plate is often full thickness and may encompass a large percentage of the plate, depending on the degree of injury. This finding is largely, if not entirely, responsible for the sagittal plane destabilization at the MTP joint.

Disruption or insufficiency of the lateral collateral ligament, in addition to medial dislocation of the plantar plate, have been implicated as two factors allowing the medial or transverse plane abnormalities at the lesser MTP joint.[15] In our limited experience, medial displacement of the plate and subsequent deviation of the flexor tendons is the primary event with attenuation and rupture of the lateral collateral ligament developing as a secondary occurrence. Medial and dorsal rotation of the plantar plate carries the flexor tendons medially via its association through the fibrous flexor sheath (Figure 3). This results in medial displacement of the force vector creating adduction and varus rotation of the affected digit (Figure 4). Secondary contracture of the intrinsic muscle and extensor tendons maintain and propagate the deformity.

DIAGNOSIS

The most valuable test in determining MTP joint instability is the Lachman's test or dorsal drawer test as described by Thompson and Hamilton.[7] This test is performed with the proximal phalanx held in 20°-25° of dorsiflexion relative to the metatarsal head, while a vertical or dorsal translational stress is applied in an attempt to sublux or dislocate the MTP joint. A positive test is defined as 2mm of dorsal displacement or 50% joint subluxation and is considered pathognomonic for a plantar plate rupture.[7-11] In the presence of a plantar plate rupture, exquisite tenderness is elicited plantarly and pain may prove to be a limiting factor in preventing joint luxation. However, even in the absence of gross

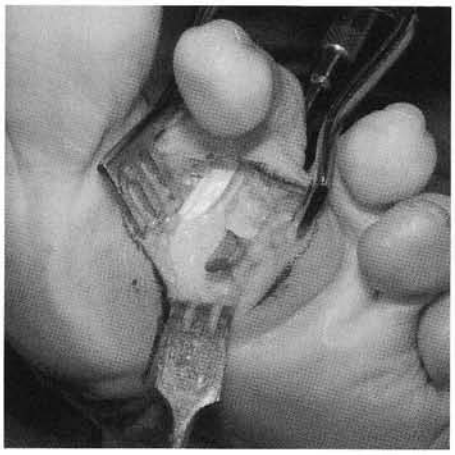


Figure 3.

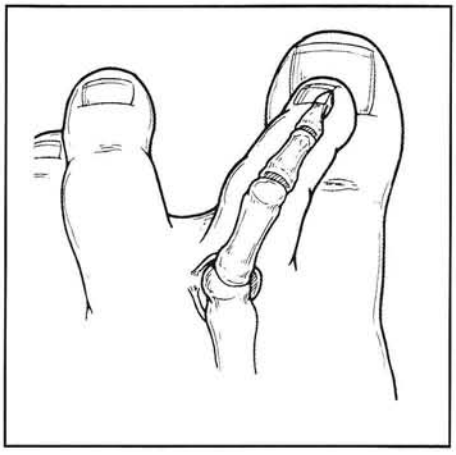


Figure 4.

joint instability, the patient's symptoms and pain may be consistently reproducible with a vertically directed stress (Lachman's test) indicative of early plate pathology. Clinically, moderate swelling may be present overlying the dorsal joint capsule creating a "fullness" and obscuring visualization of the extensor tendons which is attributed to the exuberant synovitis and periarticular inflammation seen intraoperatively. There may also be a plantar callus beneath the MTP joint indicative of the excessive mechanical overload. An unstable MTP joint may occur concomitantly with a mild to severe hallux valgus deformity, however this is not a prerequisite. The periarticular structures include the conjoined extensor tendon, collateral ligaments/MTP joint capsule and the contracture, if any, of the digital interphalangeal joints must be evaluated and each level of the deformity must be incorporated into the surgical decision making process to ensure a successful repair. Since PPD is most commonly misdiagnosed as an intermetatarsal neuroma,[2] a high index of clinical suspicion and careful physical examination are of extreme importance.

Although standard radiographs may be of little diagnostic value in PPD, several similarities in patients with lesser MTP joint stability have been identified. Coughlin noted that although no gross malalignment of the MTP joint may exist radiographically, all patients with joint instability exhibited excessive second metatarsal length (2>1) and that cortical hypertrophy was apparent in approximately 50% of the involved second metatarsals. As plantar plate degeneration progresses, radiographic findings may include multiplanar subluxation and dislocation of the digit. Degenerative findings may be seen resulting from longstanding joint malposition and dorsal subluxation with impaction of the proximal phalanx base on the metatarsal head. It is important to differentiate Freiberg's infarction from PPD because both may exhibit joint symptomotology and pain with associated joint arthrosis. Freiberg's infarction however, should not demonstrate joint instability, and arthrosis should be the predominating clinical finding with subchondral collapse. Although PPD may produce dorsal joint pain, plantar point tenderness at the level of the proximal phalanx base is an important differentiating finding.

Several ancillary studies have been utilized to aid in the diagnosis of plantar plate degeneration, including magnetic resonance imaging (MRI). In two separate publications, Yao et al has described MRI findings consistent with plantar plate degeneration and the associated pathology of the neighboring soft tissue structures.[16,17] MRI findings of a plantar plate rupture demonstrate an area of increased signal intensity in the plantar plate that extends beyond the immediate area of the plate attachment on the proximal phalanx base. According to Yao et al, the area of plate derangement is isointense with the synovium and joint fluid, and all plate defects occurred adjacent to the metatarsal head, near the distal attachment of the plantar plate. There is almost always concomitant distension of the MTP joint capsule and adjacent flexor tendon sheath indicative of synovitis (Figure 5). Although good success has been described with MRI, this is an expensive modality that may not render significant findings even in the face of significant pathology. In addition, several considerations should be given when ordering this study including access to a trained musculoskeletal radiologist for accurate interpretation and a facility that has the capabilities to image small joints. As with other pathology in the foot and ankle, the diagnosis of PPD is based on clinical assessment, and adjunctive tests such as MRI may be used to confirm what is already suspected.

Lesser MTP joint arthrography is an effective modality for aiding in the diagnosis of plantar plate ruptures.[16,18] The arthrogram is performed by injecting 0.5-1.0 ccs of an iodinated contrast material into the joint from a dorsal approach, under fluoroscopic guidance. Because the plantar plate is the only structure to separate the lesser

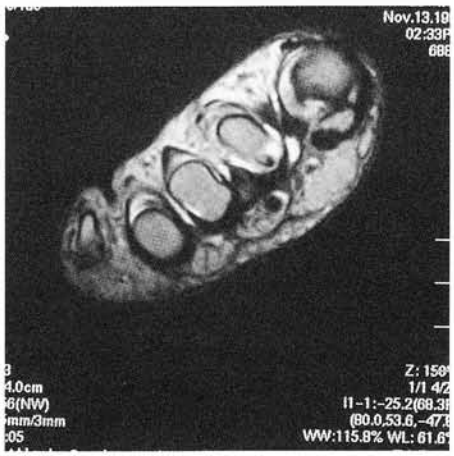


Figure 5.

MTP joint from the flexor tendons, a plate rupture may be confirmed by the extravastion of the contrast dye in the flexor tendon sheath. The primary disadvantage of this test is that it is dependent on the presence of a full-thickness defect within the substance of the plate, therefore yielding a negative result in cases of early plate degeneration.

SURGICAL TECHNIQUE

The structural significance of the plantar plate has been discused in the literature.[1,2,12,13,15] Using cadaveric studies, both Bhatia and Ford et al demonstrated that sectioning of the plantar plate creates an unstable joint construct. Several authors have stated that the plantar plate has been identified as the primary restraint to dorsal dislocation and the principal static stabilizer of the lesser MTP joint.[2,11-13]

A cadaveric study comparing flexor tendon transfers to plantar plate repair demonstrated that the direct repair of the plantar plate imparted greater dorsal–plantar stability to the MTP joint than the flexor tendon transfer, concluding that the apex of the reconstruction of the unstable lesser MPJ should be focused at direct repair of the plantar plate.[1] Although the flexor tendon transfer may restore plantarflexory stabilization to the MTP joint, it fails to address the crux of the pathology, which is alteration of the plate structure. Deland concluded that when significant involvement of the plate exists, "it does not seem likely that direct repair or reconstruction of the collateral ligaments nor the soft tissue procedures used at present would provide consistent and lasting correction."

The surgical technique for plantar plate repair may be performed concomitantly with adjunctive procedures such as interphalangeal joint arthrodesis, EHL lengthening, selective MPJ capsulotomy, flexor tendon transfer, metatarsal osteotomies and lateral MPJ capsulorraphy depending on the complexity of the deformity. The principal procedure of plantar plate repair will be described.

The patient is placed in a supine position, and a regional block with epinephrine administered. A 4.0-5.0 cm longitudinal incision is created directly beneath the plantar plate beginning at the level of the digital flexor crease (Figure 6). Sharp dissection is carried through the subcutaneous tissue to prevent disruption of the subcutaneous compartments. The superficial slips of the plantar fascia may be encountered and incised. The neurovascular structures including the intermetatarsal nerves are well protected in the medial and lateral tissue flaps.

The next surgical plane encountered is a specialization of the deep fascia known as the fibrous flexor sheath, which encases and compartmentalizes the flexor tendons beneath the plantar plate and the proximal phalanx (Figure 7). The transverse fibers of the fibrous sheath are incised longitudinally in order to dislocate the flexor tendons from the plate complex. This incision is placed between the tendons and the edge of the plantar plate laterally in order to facilitate tissue reapproximation during closure (Figure 8). The flexor tendons can then be retracted medially, allowing direct access to the plate. The plate defect can be identified through either the plantar or the dorsal incision if it exists. The defect is excised by removing a 3-5 mm transverse section of the plate, which facilitates reefing, or advancement (Figure 9). Theoretically, a trapezoidal wedge may be used to address the transverse plane component of the deformity.

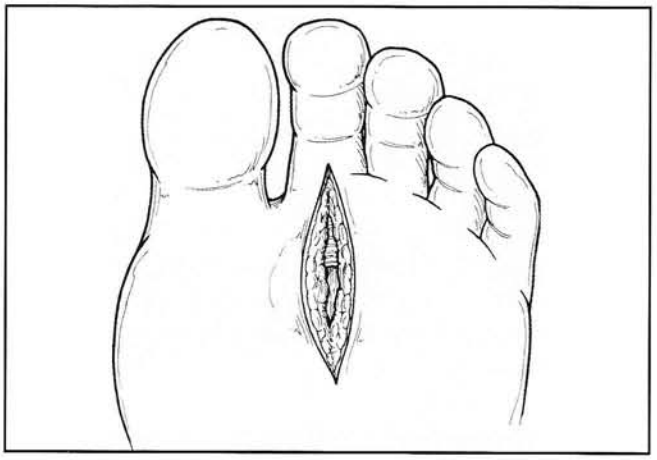
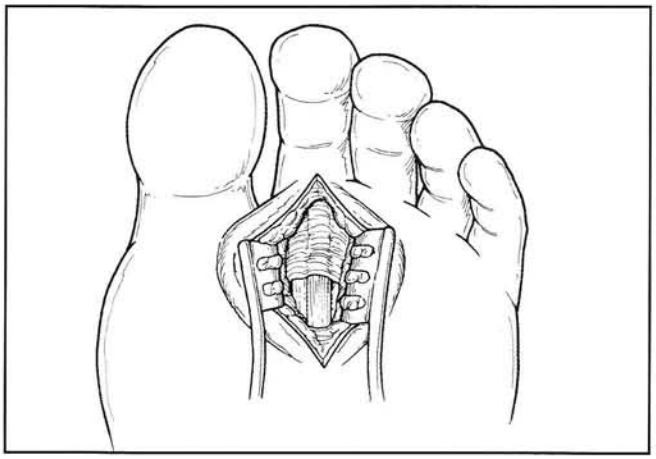


Figure 6.



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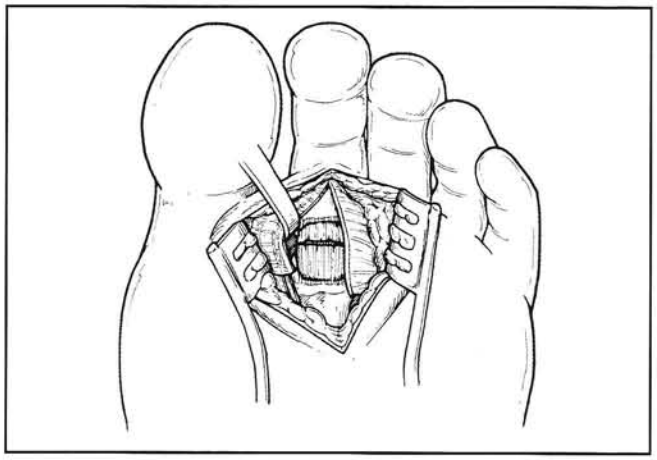

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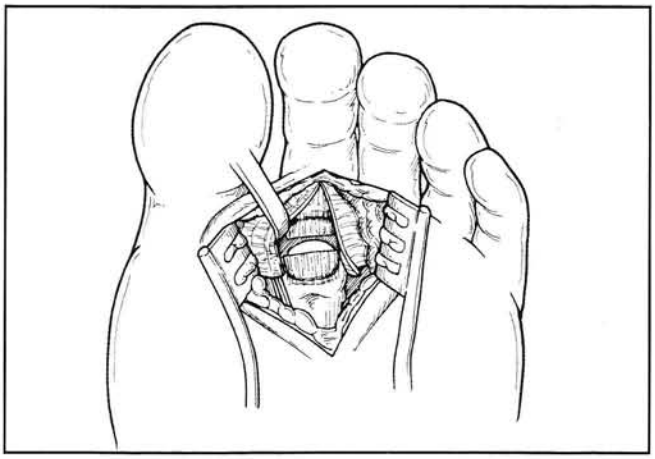

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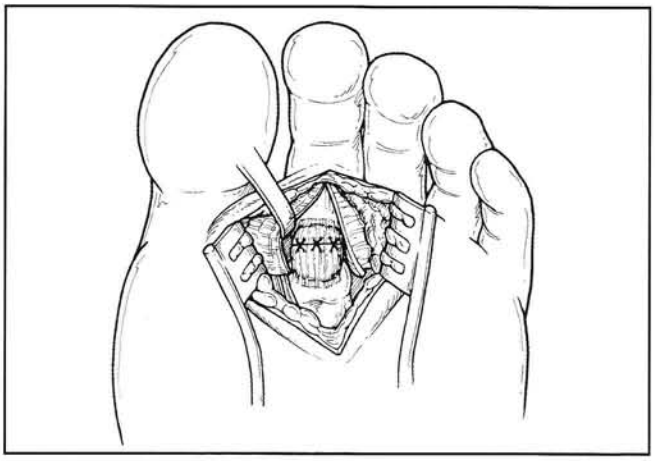

Figure 10.

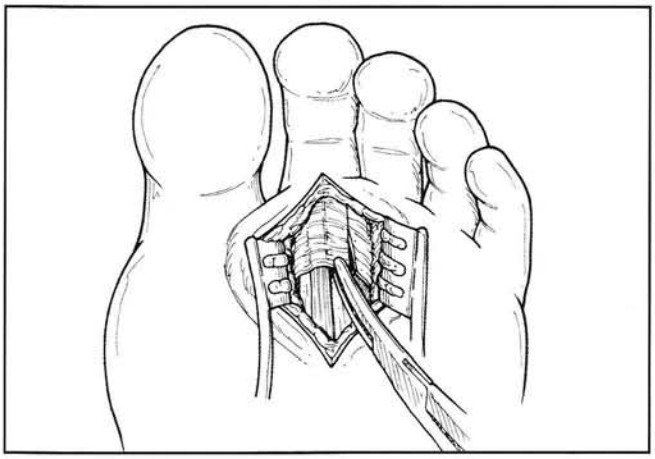

Figure 11.

With the digit held in slight plantarflexion, the plate is repaired using a 2-0 non-absorbable suture with a pulley or advancement stitch (Figure 10). The flexor tendons are then relocated and the fibrous sheath is repaired using a 3-0 absorbable suture (Figure 11). The skin and subcutaneous tissues are closed in a single layer with a 3-0 non-absorbable suture using a combination of horizontal mattress and simple interrupted stitches. If a proximal interphalangeal joint fusion is performed concomitantly, the MTP joint may be pinned in the position of correction to facilitate plate repair.

Postoperative care consists of protected weight bearing in a fracture brace, wedge surgical shoe, or other device which may neutralize bending forces at the MTP joint. If PIP joint arthrodesis and MTP joint are stabilized with a Kirschner wire, the wire may be pulled across the MTP joint at 2-3 weeks to prevent postoperative stiffness.

SUMMARY

Although lesser MTP joint instability has been a recognized clinical entity for quite some time, the role of the plantar plate has not been fully understood. Plantar plate dysfunction is a clinical diagnosis that is best determined by performing the Lachman's test. Because there are many causes of pain involving the lesser MTP joint and adjacent periarticular soft tissue structures, a high index of clinical suspicion and careful physical examination are of great importance. Long term follow up is not yet available; however in our experience direct repair of the plantar plate appears to give reliable correction for second MTP joint instability.

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