Dear PASIG and Orthopaedic Section members:

We enjoyed seeing many of you at Combined Sections Meeting in Boston. Thank you for your outstanding support at our programming entitled “Evaluation, Rehabilitation and Medical Management of the HIP joint through the lifespan of the performing artist – An evolving Art”. A huge thanks to Tara Jo Manal, PASIG Vice President for her incredible programming efforts. I hope that presenters will consider submitting their case studies for publication.

We welcome our new PASIG board member, Karen Hamill who was elected to the Secretary position. Tara Jo Manal was re-elected as Vice President. Stephania Bell becomes Nominating Committee Chair, and is joined by current member, Sheyi Ojoefitimi, and newly elected member, Heather Southwick. A big thank you to Julie O’Connell who steps off as Secretary. To all of our members – get active! We need your ideas! We need you! You form a strong network that is invaluable to the performing arts community. Personally, I treasure the many friendships that I have formed through the PASIG and the help and sharing that each colleague brings to that friendship.

This month’s Citation BLAST continues our special topic series: *posterior tibialis tendon dysfunction* contributed by our newly elected PASIG Secretary, Karen Hamill. The format is an annotated bibliography of articles on the selected topic from 1996 – 2006. Special topics are targeted periodically throughout the year. If you’d like to suggest a topic or create one, please let me know.

As a reminder, each month’s citations will be added to specific EndNote libraries: 1) Ice Skating, 2) Gymnastics, 3) Music, and 4) Dance. This particular topic will be kept in a separate library, as it applies to all of our performing arts populations. The updated libraries are posted on the PASIG webpage for our members to access and download. (Information about EndNote referencing software can be found at [http://www.endnote.com](http://www.endnote.com) including a 30-day free trial).

As always, your comments and entry contributions to these Citation BLASTs are welcome. **Orthopaedic Section members, if you’d like to continue receiving these BLASTs, check us out and join the PASIG - it’s free.** Please drop me an e-mail anytime.
Performing artists have a high prevalence of injury to the lower extremity. Foot and ankle injuries make up approximately 40% of these cases. As such, performing artist clinicians should assess for posterior tibial tendon dysfunction (PTTD). It can be easily masked by a Grade 3 Inversion Ankle sprain with acute effusion or overlooked because dancers may be more likely to have flexor hallucis longus problems. PTTD is an imbalance of muscles that can result from acute or overuse injuries due to degeneration of the tendon. When left untreated, PTTD can progress to rigid flatfoot deformity and severe osteoarthritis. Diagnosis, evaluation and treatment of PTTD are discussed in the articles included in this annotated bibliography.

Karen Hamill PT


BACKGROUND: Posterior tibial tendon dysfunction (PTTD) is a relatively common problem of middle-aged adults that usually is treated operatively. The purpose of this study was to identify strength deficits with early stage PTTD and to assess the efficacy of a focused nonoperative treatment protocol. METHODS: Forty-seven consecutive patients with stage I or II posterior tibial tendon dysfunction were treated by a structured nonoperative protocol. Criteria for inclusion were the presence of a palpable and painful posterior tibial tendon, with or without swelling and 2) movement of the tendon with passive and active nonweightbearing clinical examination. The rehabilitation protocol included the use of a short, articulated ankle foot orthosis or foot orthosis, high-repetition exercises, aggressive plantarflexion activities, and an aggressive high-repetition home exercise program that included gastrocsoleus tendon stretching. Isokinetic evaluations were done before and after therapy to compare inversion, eversion, plantarflexion, and dorsiflexion strength in the involved and uninvolved extremities. Criteria for successful rehabilitation were no more than 10% strength deficit, ability to perform 50 single-support heel rises with minimal or no pain, ability to ambulate 100 feet on the toes with minimal or no pain, and ability to tolerate 200 repetitions of the home exercises for each muscle group. RESULTS: Before therapy weakness for concentric and eccentric contractures of all muscle groups of the involved ankle was significant (p<0.001). After a median of 10 physical therapy visits over a median period of 4 months, 39 (83%) of the 47 patients had successful subjective and functional outcomes, and 42 patients (89%) were satisfied. Five patients (11%) required surgery after failure of nonoperative treatment.
CONCLUSION: This study suggests that many patients with stage I and II posterior tibial tendon dysfunction can be effectively treated nonoperatively with an orthosis and structured exercises.


PTT tenosynovitis is a recognized entity no longer confused with an ankle sprain. Three possible causes are (1) overuse or age related (mechanical in cause, true stage I disease), (2) seronegative spondyloarthropathies (clinical suspicion, hematologic analysis), and (3) rheumatoid arthritis (deformity may be owing to ligamentous or capsular destruction). The PTT has a hypovascular zone 40 mm proximal to the insertion of the tendon and 14 mm in length. Pain often is localized to this portion of the tendon (primarily in stage I disease). Ultrasound is an inexpensive and accurate method to assist in the diagnosis of this condition and may replace MR imaging as more experienced ultrasonographers appear. The initial management of PTT tenosynovitis includes tendon rest and nonsteroidal anti-inflammatory medication and physical therapy. Surgical synovial debridement is performed early (6 weeks) in patients with enthesopathies (seronegative disease). This procedure may be delayed 3 months in patients with true stage I disease. At surgery, the undersurface of the tendon must be inspected for longitudinal split tears, and these must be repaired with nonabsorbable suture, burying the knots. The excursion of the tendon should be checked intraoperatively. Patients with stage I disease should be evaluated carefully for preoperative structural deformity to choose the appropriate surgical procedure and prevent failure of isolated tenosynovectomy.


Posterior tibial tendon insufficiency is the most common cause of acquired adult flatfoot deformity. Although the exact etiology of the disorder is still unknown, the condition has been classified, on the basis of clinical and radiographic findings, into four stages. In stage I, there is no notable clinical deformity; patients usually present with pain along the course of the tendon and evidence of local inflammatory changes. Stage II is characterized by a dynamic deformity of the hindfoot. Stage III involves a fixed deformity of the hindfoot and typically also a fixed forefoot supination deformity but no obvious evidence of ankle abnormality. In stage IV, ankle involvement is secondary to long-standing fixed hindfoot deformities. The initial treatment of patients in any stage should be nonoperative, with immobilization, a nonsteroidal anti-inflammatory drug, and perhaps an orthotic device. Corticosteroid injections continue to be controversial. When nonoperative management fails, the treatment options consist of soft-tissue procedures alone or in combination with osteotomy or arthrodesis. Stage I insufficiency is generally treated with debridement and tenosynovectomy. Soft-tissue transfer does not appear to correct the underlying deformity in stage II disease; however, there is growing interest in joint-sparing operations that attempt to compensate for the underlying deformities with osteotomies or arthrodeses, supplemented with dynamic transfers to replace the insufficient posterior tibial tendon. Subtalar, double, or triple arthrodesis is the procedure of choice for stage III disease, frequently in conjunction with heel-cord lengthening. Tibiocalcaneal arthrodesis or pantalar arthrodesis is most commonly used to treat stage IV disease.

The legs of fifty cadavera were dissected to identify accurately the structures that attach to the tibia at the site of symptoms of medial tibial stress syndrome and that could potentially contribute to this condition. The origins of the soleus, the flexor digitorum longus, and the tibialis posterior muscles as well as that of the deep crural fascia were measured. The average sites of attachment and the ranges of attachment were determined for each structure. The soleus, the flexor digitorum longus, and the deep crural fascia were found to attach most frequently at the site where symptoms of medial tibial stress syndrome occur, while in no specimen was the tibialis posterior found to attach at this site. The data support recent reports that the soleus is probably the major contributor to traction-induced medial tibial stress syndrome. The data also contradict the contention that the tibialis posterior may contribute to this particular condition.


Posterior tibial tendon dysfunction, once thought to be a rare clinical entity, has been observed to be a major cause of acquired flatfoot deformity in adults. Several risk factors have been identified, ranging from inflammatory conditions to obesity. A physical examination using a series of tests, including the single-limb rise, first-metatarsal rise sign, and the "too-many-toes" sign, used in combination with selected radiographic imaging techniques, allows classification of the severity of disease. This staging system then serves as the basis for formulating the treatment options, which include nonoperative as well as operative alternatives. Conservative treatment involves rest, anti-inflammatory medication, orthotic devices, and modifications to shoes. Operative options are numerous and include primary tendon repair, tendon transfer, osteotomies, and arthrodesis.


The role of magnetic resonance imaging in the evaluation of the patient with posterior tibial tendon dysfunction is discussed. Considerations for the proper positioning of the patient and optimal technique to obtain appropriate images of the posterior tibial tendon and associated joint abnormalities are highlighted. Cases are presented to show the effectiveness of magnetic resonance imaging in different clinical situations. The treatment algorithm for posterior tibial tendon dysfunction should include magnetic resonance imaging as a diagnostic tool when appropriate.


Posterior tibial tendon dysfunction is the most common cause of acquired flatfoot deformity in adults. Although this term suggests pathology involving only the posterior tibial tendon, the disorder includes a spectrum of pathologic changes involving associated tendon, ligament, and joint structures of the ankle, hindfoot, and midfoot. Early recognition and treatment is the key to prevention of the debilitating, long-term consequences of this disorder. Conservative care is possible in the earliest stages, whereas surgical reconstruction and eventually arthrodeses become necessary in the latter stages. The purpose of this article is to review the symptoms, physical examination, radiological examination, classification, and treatment of posterior tibial tendon dysfunction.

The posterior tibial tendons (PTTs) of 16 patients with PTT dysfunction and 10 age-matched healthy subjects were examined ultrasonographically, using a 10-MHz linear-array transducer. Normal PTTs appeared hyperechoic (more echogenic) and oval, with an average diameter of 7.8 mm x 3.7 mm at the medial malleolar level. Degenerated PTTs appeared hypoechoic (less echogenic) and swollen (9.8 mm x 5.0 mm). Peritendinitis presented as a hypoechoic rim on the longitudinal sonogram (along the long axis of the tendon) and a "target sign" (hyperechoic central structure with a hypoechoic halo) on the transverse sonogram (at the right angle to the long axis of the tendon). Complete rupture of the PTT revealed an empty tibial groove at the level of the medial malleolus on the transverse sonogram and a wavy fibril pattern over the distal end on the longitudinal sonogram. Compared with the operative findings or the results of the magnetic resonance imaging, ultrasonography was sensitive and specific in diagnosing tenosynovitis and complete rupture of the PTT.


The authors overview tibialis posterior dysfunction (TPD) with emphasis on a criteria-oriented surgical protocol for management of the various clinical and pathologic stages of TPD. Subluxation of the peroneal tendons is also detailed with respect to diagnosis and treatment.


OBJECTIVE: To study posterior tibialis tendon dysfunction using an in vitro model of the foot and ankle during the heel-off instant of gait. BACKGROUND: Previous studies have concentrated primarily on the effect of posterior tibialis tendon dysfunction on the kinematics of the hindfoot and the arch. METHODS: The specimens were loaded using a custom designed axial and tendon loading system and the location of the center of pressure was used to validate heel-off. Arch position, hindfoot position and plantar pressure data were recorded before and after the posterior tibialis tendon was unloaded. These data were recorded with the ligaments intact and after creating a flatfoot deformity. RESULTS: Unloading the posterior tibialis tendon caused significant posterior movement in the center of pressure for the intact and flatfoot conditions. This also resulted in a medial shift in the force acting on the forefoot. The forefoot loads shifted medially after a flatfoot was created even when the posterior tibialis tendon remained loaded. The spatial relationships of the bones of the arch and the bones of the hindfoot also changed significantly for the intact specimen, and to a lesser extent after a flatfoot. CONCLUSIONS: The posterior tibialis tendon plays a fundamental role in shifting the center of pressure anteriorly at heel-off. Posterior tibialis tendon dysfunction causes posterior shift in the center of pressure and abnormal loading of the foot's medial structures. This may be the reason that posterior tibialis tendon dysfunction leads to an acquired flatfoot deformity. Conversely, flatfoot deformity may be a predisposing factor in the onset of posterior tibialis tendon dysfunction. This tendon also acts to lock the bones of the arch and the hindfoot in a stable configuration at heel-off, but a flatfoot deformity compromises this ability.


A cadaver study was performed to determine the effect of the posterior tibial tendon (PTT) on the stability of the foot in simulated midstance phase of gait. Thirteen fresh-
frozen human foot specimens were used. Loads were applied axially and to each tendon. Three-dimensional positions of tarsal bones before and after tendon loading were determined with the use of a magnetic tracking device. Significant differences in tarsal bone positions were observed with application of loads to the Achilles, posterior tibial, flexor digitorum longus, peroneus longus, and peroneus brevis tendons at the metatarsotalar, calcaneotalar, and talotibial joints and in overall arch height. These tendon loads caused position changes toward arch flattening or mild pes planus deformity. Significant differences in tarsal bone positions were observed with PTT loading compared with no PTT loading in metatarsotalar, calcaneotalar, and talotibial levels as well as arch height. The PTT caused position changes toward restoring the arch alignment. These data suggest that the PTT is an important stabilizer of the arch of the foot.


BACKGROUND: Posterior tibialis tendon dysfunction (PTTD) is a common cause of foot pain and dysfunction in adults. Clinical observations strongly suggest that the condition is progressive. There are currently no controlled studies evaluating the effectiveness of exercise, orthoses, or orthoses and exercise on Stage I or IIA PTTD. Our study will explore the effectiveness of an eccentric versus concentric strengthening intervention to results obtained with the use of orthoses alone. Findings from this study will guide the development of more efficacious PTTD intervention programs and contribute to enhanced function and quality of life in persons with posterior tibialis tendon dysfunction.

METHODS/DESIGN: This paper presents the rationale and design for a randomized clinical trial evaluating the effectiveness of a treatment regime for the non-operative management of Stage I or IIA PTTD. DISCUSSION: We have presented the rationale and design for an RCT evaluating the effectiveness of a treatment regimen for the non-operative management of Stage I or IIA PTTD. The results of this trial will be presented as soon as they are available.


Posterior tibialis tendon rupture is a diagnosis that is often missed. This is thought to be secondary to nonspecific clinical findings and the lack of any laboratory or radiographic test to reliably confirm the diagnosis. We report sixteen cases of surgically confirmed posterior tibialis tendon rupture. Based on our review of these patients, the diagnosis of posterior tibialis tendon rupture should be strongly suspected in the adult patient presenting with a history of a twisting ankle injury and generalized medial ankle pain and swelling. A flexible, asymmetric pes planus and forefoot pronation deformity with absence of posterior tibialis tendon function on manual testing is seen on examination. This is associated with loss of ipsilateral heel inversion on bilateral heel rise. The patient is usually unable to perform ipsilateral single leg heel rise and has less severe pes planus of the contralateral foot. This study reviews the presentation, pathophysiology, diagnosis, and treatment of posterior tibialis tendon rupture.

We present a case report and literature review of distal intrasubstance rupture of the posterior tibial tendon with progressive pes planovalgus secondary to tendon incompetence. Three months after a severe ankle sprain, a 25-year-old basketball player presented with ankle weakness and pain. Treatment by advancement of the posterior tibial tendon to the navicular and medial displacement osteotomy of the calcaneal tuberosity restored alignment, strength, and full function.


Posterior tibial tendon dysfunction (PTTD) is a complex multifaceted condition that can affect the lower extremity. Rarely mentioned 20 years ago, today it is the subject of numerous articles, books, and is a topic at most scientific seminars relating to the foot and ankle. It is a muscle imbalance initiated by a rupture, avulsion, or chronic inflammation of the tibialis posterior tendon. With time, it progresses from a flexible to rigid flatfoot deformity. Left untreated, peritalar dislocation and degenerative joint disease may develop. This article discusses the diagnosis, evaluation, and treatment of PTTD.


Posterior tibial tendon dysfunction is a cause of painful acquired flatfoot in adults. It is associated with progressive collapse of the medial longitudinal arch, hindfoot valgus, and forefoot abduction deformities. The clinical manifestations and surgical treatment have been well documented in the literature. Epidemiologic studies have not shown any clear predisposing factors to the disease. Numerous etiologies have been proposed to explain the clinical evidence of tendon degeneration found at the time of surgery including trauma, anatomic, mechanical inflammatory, and ischemic factors. Although previously thought to be secondary to an inflammatory process resulting in acute and chronic tendinitis, more recent histopathologic evidence has revealed a degenerative tendinosis with a nonspecific reparative response to tissue injury characterized by mucinous degeneration, fibroblast hypercellularity, chondroid metaplasia, and neovascularization. These pathologic changes result in marked disruption in collagen bundle structure and orientation. This may compromise the tendon and predispose it to rupture under physiologic loads. However, it cannot be determined whether these changes precede or postdate posterior tibial tendon dysfunction. It seems that there are many contributing factors to the etiology of posterior tibial tendon dysfunction all culminating in a common disease process with resulting tendon degeneration and an insufficient repair response.


BACKGROUND: Degenerative pathology of the posterior tibial tendon, a common cause of foot and ankle dysfunction, frequently affects women over 40 years of age. Its etiology is still controversial. The literature reports decreased vascularization coinciding with the most common site of the lesion, near the medial malleolus. METHODS: Forty pairs of PTT obtained from human cadavers were transversally cut into six levels, from the musculotendon transition to its insertion point. In each segment, a histologic cut was made and stained with Masson's trichrome allowing viewing of the vascular structure of
the tendon under a light microscope. By using an integrating eyepiece on the microscope, vascular density was calculated. This verified any variation of the vascular concentration in the normal tendon, a possible cause of its degeneration. RESULTS: When the results were compared by side, sex, and age, no statistically significant difference was observed. When the levels were compared, no area of decreased vascularization was seen in the midportion of the tendons, the most common site of degeneration of the posterior tibial tendon. CONCLUSION: These results indicate that an area of decreased vascularity is not a factor in degeneration of the posterior tibial tendon at the medial malleolus.


BACKGROUND: Many studies have shown that lack of functional activity of tibialis posterior leads to changes in the longitudinal arch and affects the motion of the foot. A quantitative description of the affects on the motion of the foot in detail has not been reported. OBJECTIVE: To describe three-dimensional motion of the leg, rearfoot and forefoot with tibialis posterior dysfunction during stance phase of walking in comparison with normals. This study compared one case without the function of tibialis posterior with the ensemble average of 10 normals (five males, five females). METHODS: Subjects with 10, 12 mm retroreflective markers placed on their right leg, rearfoot and forefoot, performed five trials of walking at self-selected speed on a 10 m walkway. A four-camera three-dimensional motion analysis system and a synchronized force platform were used to record three-dimensional motions of the segments and force variables during stance phase of walking. RESULTS: The patterns and range of motion of the rearfoot relative to the leg, and the forefoot relative to the rearfoot demonstrated some differences between the tibialis posterior dysfunction case and normals. Most of the major differences occurred from just prior to heel-off through to toe-off, the period when a stable arch would be required. CONCLUSION: The observed differences in the three-dimensional foot motions of the tibialis posterior dysfunction case compared with normals during walking were consistent with the expected mechanical consequences of a foot without the function of tibialis posterior. The one exception was the inversion of the rearfoot which remained normal. RELEVANCE: Tibialis posterior dysfunction has been recognized as one of the significant impairments of the musculoskeletal system which affects normal walking. Dynamic investigation adds an understanding of how the muscle controls the foot during walking which is essential information for the diagnosis and the adjustment of treatment and rehabilitation for a tibialis posterior dysfunction case.


The purpose of this study was to examine differences in gait mechanics between patients with acute stage II PTTD and healthy volunteers. Hindfoot and midfoot kinematics, plantar foot pressures and electromyographic (EMG) activity of the posterior tibialis, gastrocnemius, anterior tibialis and the peroneals were measured in five patients with acute stage II PTTD. Kinematics and kinetics were compared to a database of 20 healthy volunteers. EMG and plantar pressure data were obtained from five healthy volunteers. Hindfoot moments and powers were also calculated. The center of pressure excursion index (CPEI) was calculated from the plantar pressures. Significant differences were observed between the two groups, which confirmed clinical observations. Limited hindfoot eversion and increased midfoot external rotation occurred during the first and third rockers. The EMG data suggested that tendon dysfunction in
the posterior tibialis is associated with compensatory activity, not only in its antagonists (the peroneals), but also in the anterior tibialis and the gastrocnemius. These data suggest that non-operative treatment of patients with PTTD should consider minimizing the activity of the posterior tibialis as well as the peroneals, the anterior tibialis and the gastrocnemius.


It has been demonstrated that athletes who are properly diagnosed and treated early for PTT dysfunction with aggressive conservative management typically have a much better prognosis and are able to return to activities much sooner. Conservative management of this over-use or traumatic injury is helpful, even as a stop-gap measure in anticipation of an inevitable surgical correction. For the elderly, sedentary, or high-risk patient, aggressive conservative care is a viable option in lieu of surgical intervention. If surgery is indicated, however, there are a multitude of procedures to choose depending on the pathology and its extent.


STUDY DESIGN: A 2 x 4 mixed-design ANOVA with a fixed factor of group (posterior tibialis tendon dysfunction [PTTD] and asymptomatic controls), and a repeated factor of phase of stance (loading response, midstance, terminal stance, and preswing).

OBJECTIVE: To compare 3-dimensional stance period kinematics (rearfoot eversion/inversion, medial longitudinal arch [MLA] angle, and forefoot abduction) of subjects with stage II PTTD to asymptomatic controls. BACKGROUND: Abnormal foot postures in subjects with stage II PTTD are clinical indicators of disease progression, yet dynamic investigations of forefoot, midfoot, and rearfoot kinematic deviations in this population are lacking. METHODS: Fourteen subjects with stage II PTTD were compared to 10 control subjects with normal arch index values. Subjects were matched for age, gender, and body mass index. A 5-segment, kinematic model of the leg and foot was tracked using an Optotrak Motion Analysis System. The dependent kinematic variables were rearfoot inversion/eversion, forefoot abduction/adduction, and the MLA angle. An ANOVA model was used to compare kinematic variables between groups across 4 phases of stance. RESULTS: Subjects with PTTD demonstrated significantly greater rearfoot eversion (P =.042), MLA angle (P =.008) and forefoot abduction angles (P <.005) during specific phases of stance. Subjects with PTTD demonstrated significantly greater rearfoot eversion (P <.004) and MLA angles (P <.009) by 6.2 degrees and 8.0 degrees, respectively, during loading response when compared to controls. During preswing, the subjects with PTTD demonstrated a significantly greater MLA angle (P <.002) and a forefoot abduction angle (P <.001) which exceeded that of the controls by 10.0 degrees. CONCLUSIONS: The abnormal kinematics observed at the rearfoot, midfoot, and forefoot across all phases of stance implicate a failure of compensatory muscle and secondary ligamentous support to control foot kinematics in subjects with stage II PTTD.


A new classification scheme is presented along with an algorithmic surgical approach to the treatment of posterior tibial tendon dysfunction (PTTD). This classification scheme treats as separate entities the soft-tissue and osseous components of PTTD. The soft-tissue pathology is staged from tendinitis to tendinosis to that of complete rupture. The osseous element is graded as no planar deformity, reducible planar deformity, or rigid planar deformity. The stage of soft-tissue pathology is then combined with the grade of the osseous condition, leading to a comprehensive surgical treatment plan. Based on this classification, 13 patients were retrospectively evaluated. Only those patients falling into a specific classification (2A and 2B) were included in this analysis. The American Orthopedic Foot and Ankle Society clinical hindfoot-ankle scale was utilized. The mean preoperative clinical rating was 32.8. The mean postoperative clinical rating was 88.0. Pre- and postoperative radiographic criteria were also analyzed. Overall patient satisfaction utilizing this treatment algorithm was good to excellent.


Approximately 50% of all sports injuries are secondary to overuse and result from repetitive microtrauma that causes local tissue damage. Injuries are most likely with changes in mode, intensity, or duration of training and can accumulate before symptoms appear. Intrinsic factors contributing to injuries are individual biomechanical abnormalities such as malalignments, muscle imbalance, inflexibility, weakness, and instability. Contributing extrinsic (avoidable) factors include poor technique, improper equipment, and improper changes in duration or frequency of activity. Injuries are often related to biomechanical abnormalities removed from the specific injury site, requiring evaluation of the entire kinetic chain. This article discusses common overuse injuries of the lower leg, ankle, and foot: tendinopathies, stress fractures, chronic exertional compartment syndrome, and shin splints.