Comparison of Foot Kinematics Between Subjects With Posterior Tibialis Tendon Dysfunction and Healthy Controls

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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° and 8.0°, 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°.

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. J Orthop Sports Phys Ther 2006;36(9):635-644. doi:10.2519/jospt.2006.2293

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1, 11, 25, 26, 29, 35 Posterior tibialis tendon dysfunction (PTTD) is believed to be a primary contributor to the onset and development of adult acquired flatfoot deformity.1, 11, 25, 29, 35 Persons most commonly affected by PTTD are white females, 45 to 65 years of age, that are overweight and hypertensive.11, 25, 26 Although PTTD is viewed as an overuse injury, the exact cause of PTTD is unknown.11, 25, 26 Avascularity of the tendon and metabolic disease associated with diabetes have been correlated to tendon dysfunction and may contribute to PTTD.1, 11, 25, 26 Regardless of the etiology, PTTD is viewed as a progressive disorder.19, 28 To track progress of PTTD, clinical signs and symptoms of 4 stages of the disease are widely used.19, 28 Stage I PTTD is characterized by pain and swelling of the medial aspect of the foot and ankle. No changes in tendon length or foot posture are associated with symptoms. Stage II PTTD is characterized by weakness in inversion and difficulty completing a heel raise, indicating compromise of the tendon and muscle. Associated with muscle weakness are rearfoot valgus and forefoot abduction deformity. However, clinically the foot is...
supple, allowing the clinician to return the foot to a normal posture. In stage III PTTD, the foot deformity noted in stage II has become fixed or inflexible. Myerson et al\textsuperscript{19,29} included stage IV PTTD distinguished from stage III by valgus angulations of the talus and early degeneration of the ankle joint.

Progression from stage I to stage II PTTD is associated with abnormal foot postures, indicating a decreased function of the posterior tibialis muscle and breakdown of supporting foot ligaments. Because synergistic muscles, such as the flexor digitorum longus, are unable to adequately compensate for the decreased contribution of the posterior tibialis muscle, abnormal foot postures develop.\textsuperscript{14,30,31} Persisting abnormal foot postures may also contribute to breakdown of secondary ligamentous supports (eg, spring ligament, plantar fascia, and deltoid liga-

ment),\textsuperscript{2,12} further magnifying the flatfoot deforma-
tion.\textsuperscript{23,25,28,43} These deviations point to the importance of early recognition and management of adult acquired flatfoot deformity in preventing disease progression and protecting secondary ligamentous supports.\textsuperscript{25}

In vitro studies attempting to simulate gait mechanics suggest that the posterior tibialis muscle may play a key role in preventing collapse of the medial longitudinal arch (MLA), contribute to rearfoot inversion, and adduct the forefoot.\textsuperscript{30,31,37} Despite EMG analysis suggesting inconsistent activation patterns in the posterior tibialis muscle during gait,\textsuperscript{32} the muscle is thought to play a significant role in support of the MLA after foot flat and rearfoot inversion after heel off.\textsuperscript{3,36} While the static clinical presentation of a decreased MLA, together with increased rearfoot eversion and forefoot abduction, provide some indication of the disease progression, limited data exist regarding the dynamic interrelationship of segmental foot motion during gait in individuals with PTTD. Although the rearfoot has been primarily implicated in kinematic alterations found in subjects with PTTD,\textsuperscript{17,18,30} there is evidence to suggest even greater influence on forefoot function with tendon attenuation. Using a multsegment foot model applied to a single case of a subject with PTTD and a longstanding lacerated tendon, Rattanaprasert et al\textsuperscript{31} found that rearfoot eversion/inversion was surprisingly similar to a group of 10 healthy controls. However, forefoot motion with respect to the rearfoot demonstrated alterations in motion consistent with loss of the MLA and increased forefoot abduction. These limited data (n = 1) suggest more involvement of the forefoot than the rearfoot. Interestingly, brace and orthotic designs primarily focus on the rearfoot and MLA control, assuming forefoot abduction will consequently also improve.\textsuperscript{3} A better understanding of the abnormal kinematics displayed by subjects with stage II PTTD will assist in the development of exercises and brace/orthotic designs targeted at decreasing strain on the posterior tibialis tendon and protecting secondary ligamentous supports.

The purpose of this study was to compare rearfoot inversion, MLA, and forefoot abduction kinematics during the stance period of gait between subjects diagnosed with PTTD and matched asymptomatic control subjects. We hypothesized that subjects with stage II PTTD would demonstrate greater rearfoot inversion, MLA angle, and forefoot abduction during specific phases of stance. During loading response and preswing the differences between the PTTD group and controls were expected to be larger than during midstance and terminal stance, when muscle activation of the posterior tibialis is less consistent.\textsuperscript{32}

\section*{Materials and Methods}

\subsection*{Subjects}

Fourteen subjects with PTTD (12 female, 2 male) and 10 control subjects (7 female, 3 male) participated. The control subjects were matched for age, gender, and body mass index as closely as possible with the intervention group. Subjects with unilateral PTTD were referred by a local orthopaedic surgeon and were clinically classified as having stage II PTTD. The inclusion criteria for a stage II PTTD classification required subjects to have 1 or more signs related to posterior tibialis tendon dysfunction, including (1) palpable tenderness of the posterior tibialis tendon, (2) swelling of the posterior tibialis tendon sheath, and (3) pain during single-limb heel raise, and 1 or more signs of flexible flatfoot deformity, including (1) excessive nonfixed rearfoot valgus deformity during weight bearing and/or excessive forefoot abduction. Excessive rearfoot valgus and forefoot abduction were based on visual comparisons from the involved to the uninvolved side. This led to the inclusion criteria that all subjects in the PTTD group were required to have unilateral involvement. Subjects were excluded if they had a history of pain or pathology in the foot or lower extremity that prevented them from ambulating greater than 15 m.

The subjects with PTTD were compared to an asymptomatic control group matched for age, gender, and body mass index. Additionally, these subjects were required to have no history of foot and ankle problems and a normal arch index. The arch index is described as the ratio of dorsum height at 50\% of the foot length, divided by the foot length from the heel to the base of the distal first metatarsal head.\textsuperscript{45} A larger index indicates a higher arch. For this investigation, a normal arch was defined as equal to or greater than 1 standard deviation higher (greater height of the MLA) than average as reported by Williams et al.\textsuperscript{15} The arch index was chosen because it is easy to apply and is correlated with radiographic measures of foot posture.\textsuperscript{15} Prior to testing, subjects
were informed of the experimental procedure and signed a consent form approved by the Research Subjects Review Board at the local University of Rochester and Ithaca College. Group characteristics are presented in Table 1.

### Kinematic Measurements

A 5-segment foot model (tibia, rearfoot, medial forefoot, lateral forefoot, and the hallux) was established using sets of 3 noncollinear infrared emitting diodes (IREDs). For this investigation, the medial segment of the forefoot was defined by the first metatarsal, and the lateral segment included the second through fourth metatarsals. The medial segment was used to derive MLA angle changes during gait and the lateral segment was used to define forefoot abduction with respect to the calcaneus. The representation of foot function using this model enabled delineation of rearfoot, midfoot, and forefoot motions that were considered indicative of posterior tibialis tendon function.

The IRED markers were first mounted on rigid thermoplastic platforms, and then attached to the tibia, calcaneus, first metatarsal, second through fourth metatarsals, and hallux by means of double-sided adhesive tape. Anatomical landmarks were digitized by a single examiner (JMT) to facilitate the transformation of the IRED data to local anatomically based coordinate systems for each segment (Figure 1). Euler angles, representing 3 sequential rotations (z-x-y) about the anatomical axes were used to describe 3-dimensional joint kinematics. Motion of the distal-most foot segment was then calculated relative to the adjacent proximal segment, based on the Euler rotation sequence of flexion/extension, inversion/eversion, and adduction/abduction, respectively. Thus, rearfoot eversion was described as rotation of the calcaneal coordinate system about its anterior/posterior axis with respect to the tibial coordinate system. Forefoot abduction-adduction was described as rotation of the second-through-fourth-metatarsal coordinate system about its inferior/superior axis with respect to the calcaneal coordinate system.

A single IRED marker was placed on the skin overlying the navicular tuberosity. This marker in addition to digitized points on the posterior calcaneus and first metatarsal head were used to generate the MLA angle, with the navicular marker as the apex (Figure 2). The dot product of the 3-dimensional vectors from the navicular to the metatarsal head and navicular to the posterior heel was.
FIGURE 2. Navicular marker and digitized points (circles) that were used to calculate the medial longitudinal arch (MLA) angle measurement. Where \( A \) and \( B \) represent generalized 3-D vectors. In this case vector \( A = \vec{r}(\text{Methead/Navicular}) \) and vector \( B = \vec{r}(\text{Calcaneus/Navicular}) \). The dot product between these 2 vectors was used to determine the MLA angle \( \theta_{\text{MLA}} \). This resulted in a planar representation of MLA angle irrespective of foot position. A larger MLA angle indicates a decrease or lowering of the MLA, whereas a smaller, more acute angle indicates an elevation of the MLA.

Procedures

Subjects walked down a 14-m walkway to establish their mean, self-selected walking speed. Speed was monitored with the use of a timing system (Brower, Salt Lake City, UT) and maintained during testing to within \( \pm 5\% \). This resulted in an average (\( \pm \)SD) stance time of 772 \( \pm \) 83 ms for the PTTD group and 720 \( \pm \) 64 ms in the control group, and corresponds to a walking speed of 1.2 m/s and 1.3 m/s for the PTTD group and control group, respectively. Six infrared cameras (Optotrak model 3020; NDI, Waterloo, Canada), in conjunction with motion analysis software (Motion Monitor; Innovative Sports Training, Chicago, IL) were used to collect kinematic data at a sampling rate of 60 Hz. Kinematic data were smoothed using a 6-Hz cut-off frequency, fourth-order Butterworth, zero phase lag filter prior to calculating kinematic variables. An embedded force plate (model 9286; Kistler Group, Winterthur, Switzerland) was used to delineate initial contact and toe-off points of stance of the tested extremity, and data were collected at 1000 Hz. Each subject completed a minimum of 5 successful trials, which consisted of the identified self-selected walking speed and full contact of the tested foot with the force plate.

Following the collection of the walking trials, a reference (zero) subtalar neutral (STN) position was established for each subject. Previous investigations have emphasized the importance of using a reference position when comparing among subjects with varying foot postures. From their relaxed standing posture, subjects were positioned into STN, which was palpated consistent with published protocols. Determination of weight-bearing STN has shown low errors (<3°) in standing. Subjects were asked to hold this position for 3 seconds while kinematic data were collected. The mean of 2 STN trials was used as the reference position for each subject. Preliminary evaluation of the methods used in this study demonstrated intraclass correlation coefficients (ICC\(_{3,1}\)) above 0.9 within a session (\( n = 18 \)) and average differences in peak angles between sessions (\( n = 4 \)) of less than 1.8° for the tested variables.

Data Analysis

The aim of this investigation was to examine the kinematic differences between groups for the dependent variables that included rearfoot eversion, MLA, and forefoot abduction. Kinematic patterns were linearly interpolated to 100% of stance in 1% intervals over the stance period of gait. These data were then referenced to the STN (zero) position for each interval across stance. Subsequently, 5 trials for the same subject were averaged to achieve a representative pattern for each subject. The data were then averaged across stance for all subjects, generating group means and standard deviations. A 2 \( \times \) 4 mixed-design ANOVA model with the fixed factor of group (PTTD and control) and repeated factor of phase (loading response [0%-20%], midstance [21%-50%], terminal stance [51%-90%], preswing [91%-100%]) was used to assess differences in dependent measures across stance. To provide for consistency when comparing kinematic measures across stance, the ensemble averaged data were analyzed using the midpoint of each of the phases of gait. Using the midpoint of each phase prevented biases from differences in the timing of kinematic patterns from influencing the results. For each dependent variable 2 hypotheses were tested. First, the hypothesis that any differences in kinematics were group and phase dependent was tested by examining any interaction effects. In the presence of an interaction, main effects were ignored. Pairwise comparisons between groups but within a phase were pursued in the case of a significant interaction. A nominal significance level was maintained at \( P < 0.05 \).
**TABLE 2.** Means and (SD) of kinematic variables during stance.

<table>
<thead>
<tr>
<th>Kinematic Variables</th>
<th>LR</th>
<th>MS</th>
<th>TS</th>
<th>PS</th>
<th>$P$ Value for Overall Differences</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rearfoot eversion</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PTTD</td>
<td>-9.6 (4.7)†</td>
<td>-10.4 (4.5)†</td>
<td>-9.0 (3.9)</td>
<td>-3.2 (4.7)</td>
<td>Group  &lt;.001</td>
</tr>
<tr>
<td>Control</td>
<td>-3.4 (4.6)</td>
<td>-5.4 (3.6)</td>
<td>-6.4 (2.9)</td>
<td>1.1 (3.5)</td>
<td>Group × phase .042</td>
</tr>
<tr>
<td>MLA</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PTTD</td>
<td>8.2 (8.7)†</td>
<td>9.8 (7.9)†</td>
<td>12.2 (6.9)</td>
<td>5.3 (8.4)†</td>
<td>Group  .007</td>
</tr>
<tr>
<td>Control</td>
<td>0.0 (3.3)</td>
<td>2.1 (4.2)</td>
<td>7.2 (4.0)</td>
<td>-4.7 (4.4)</td>
<td>Group × phase .008</td>
</tr>
<tr>
<td>Forefoot abduction</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PTTD</td>
<td>-9.1 (3.7)†</td>
<td>-10.1 (3.1)†</td>
<td>-11.4 (2.8)†</td>
<td>-9.2 (5.7)†</td>
<td>Group  &lt;.001</td>
</tr>
<tr>
<td>Control</td>
<td>-4.4 (3.4)</td>
<td>-5.7 (3.1)</td>
<td>-6.5 (2.8)</td>
<td>-0.9 (3.8)</td>
<td>Group × phase .005</td>
</tr>
</tbody>
</table>

Abbreviations: LR, loading response; MLA, medial longitudinal arch angle; MS, midstance; PS, preswing; PTTD, posterior tibialis tendon dysfunction; TS, terminal stance.

* Values are determined at the midpoint of each phase of stance.
† Denotes significant difference ($P < .05$) of pairwise comparison between groups within a phase.

### RESULTS

There were significant interactions for group and phase for rearfoot eversion/inversion ($P = .042$), MLA angle ($P = .008$), and forefoot abduction/adduction ($P = .005$) (Table 2). Because the differences between groups depended on the phase of stance, subsequent analysis focused on pairwise comparisons for each variable.

#### Rearfoot Eversion/Inversion

The PTTD group showed a consistent bias toward eversion throughout stance compared to the controls, with greater differences in rearfoot eversion during specific phases of gait. The PTTD group remained biased by 2.6° to 4.0° toward rearfoot eversion, failing to return to a STN position during terminal stance and preswing. The patterns across stance show that the subjects with PTTD moved to peak rearfoot eversion during loading response, while the control group reached peak rearfoot eversion during terminal stance (Figure 3). This led to significantly greater rearfoot eversion of the PTTD group by 5.0° to 6.2° (loading response, $P = .004$; midstance, $P = .008$) until terminal stance, at which time the PTTD group approximated the pattern of the control group ($P = .09$).

#### MLA

Subjects with PTTD maintained a significantly greater MLA angle (lower arch) compared to the control subjects throughout stance, with greater differences during specific phases of stance. Individuals in the PTTD group displayed a significantly higher MLA angle by approximately 8°, compared to the control group during loading response ($P = .009$) and midstance ($P = .011$). This difference decreased to 5.0° during terminal stance ($P = .051$), then increased to 10° during preswing ($P = .002$) (Figure 4). Although the subjects with PTTD demonstrated a change in arch angle towards arch elevation at terminal stance/preswing, they were unable to return to their STN position by 5.3°. In contrast, the subjects in the control group moved past STN by 4.7°, indicating greater arch elevation.

#### Forefoot Abduction/Adduction

The patterns indicate little movement of forefoot abduction across the first 3 phases of stance, with the PTTD subjects biased toward greater abduction. For both groups, the forefoot remained abducted relative to the STN position until preswing. From loading response to terminal stance the PTTD group demonstrated significantly greater forefoot abduction by at least 4.4°, compared to the control group (loading response, $P = .007$; midstance, $P = .003$; terminal stance, $P = .001$). These differences between the PTTD and control groups doubled (10.0°) during preswing ($P = .001$) (Figure 5).

### DISCUSSION

The findings of this study confirm the hypothesis that abnormal kinematics associated with stage II PTTD involve the rearfoot, midfoot, and forefoot, and vary across the stance period of walking. The abnormal kinematics of greater rearfoot eversion, increased MLA angle, and increased forefoot abduction are consistent with current theories regarding posterior tibialis muscle function and loss of secondary ligamentous support. The subjects with stage II PTTD showed significantly greater rearfoot eversion, MLA angle, and forefoot abduction during specific phases of stance, with significant differences ranging between 4° to 10° compared to the control group.

The kinematic differences found at the distinct phases of stance suggest a loss of the dual function of
FIGURE 3. Rearfoot eversion/inversion patterns of the calcaneus relative to the tibia for posterior tibialis tendon dysfunction (PTTD) and control subjects during each phase of stance. Dotted lines represent ±1 SD. Subtalar neutral (STN) position is the zero reference position.

FIGURE 4. Medial longitudinal arch (MLA) angle patterns for subjects with stage II posterior tibialis tendon dysfunction (PTTD) and control subjects during each phase of stance. Dotted lines represent ±1 SD. Subtalar neutral (STN) position is the zero reference position.
FIGURE 5. Forefoot abduction/adduction patterns of the second to the fourth metatarsals relative to the calcaneus for subjects with stage II posterior tibialis tendon dysfunction (PTTD) and control subjects during each phase of stance. Dotted lines represent ±1 SD. Subtalar neutral (STN) position is the zero reference position.

The failure of the posterior tibialis in subjects with PTTD. During the first half of stance (loading response and midstance), subjects with stage II PTTD are characterized by having significantly greater rearfoot eversion and MLA angle, suggesting the lack of controlled rearfoot lowering and shock absorption in this phase of gait. In the latter part of stance (preswing), subjects with stage II PTTD are characterized by having significantly greater MLA angle and forefoot abduction when compared to the asymptomatic controls. The inability to effectively regain arch height and forefoot adduction implicates the posterior tibialis during push-off. Matching the groups for age, gender, and body mass index minimizes the potentially confounding differences between groups associated with these factors, rather than insufficiency of the tendon.

The failure of the posterior tibialis tendon to effectively control foot motion is evident through the first half of stance and is primarily a result of abnormal rearfoot and MLA angle kinematics. Theoretically rearfoot eversion and increased MLA angle move the talonavicular and calcaneocuboid joints to a more parallel position, unlocking the foot for shock absorption. Because the gait pattern for subjects with a normal-arch foot progresses from a neutral (or slightly inverted position) to eversion at foot flat, the role of shock absorption is linked to these foot kinematics. The subjects with PTTD in this study were not typical, initiating stance with approximately the same amount of rearfoot eversion that control subjects maximally obtained at terminal stance. While the healthy subjects increase rearfoot eversion and MLA angle throughout stance, the subjects with PTTD are at, or near, peak rearfoot eversion and MLA angle during loading response. This failure of a gradual shock absorption to occur may contribute to abnormal stresses on secondary ligamentous support (spring ligament, plantar fascia, intersosseous talocalcaneal ligament) as the foot is loaded.

During the terminal stance and preswing phases of gait, abnormal kinematics of the subjects with PTTD suggests a failure to position the foot effectively for push off. Rearfoot inversion and increased height of the MLA may indicate that the talonavicular and calcaneocuboid joints are in a nonparallel position, transforming the foot into a rigid lever through bony contact. The role of inverting the foot after foot flat is attributed to the posterior tibialis muscle and contralateral limb rotation. While the rearfoot does move toward inversion in the subjects with PTTD, approximating the pattern of the controls (Figure 3), MLA angle and forefoot abduction angle do not. During preswing the MLA and forefoot abduction angles (Figures 4 and 5) are markedly higher for the subjects with PTTD compared to the
controls. Although the rearfoot does move toward inversion, it fails to approximate the STN position by 4.0°. The failure of subjects with PTTD to achieve a similar degree of MLA angle and forefoot adduction to that of the controls (or as defined by STN) suggests bony contact may play less of a role in providing stability. A decreased reliance on bony stability at this stage of stance, when the foot is transferring forces from the strong plantar flexors to the floor, may indicate a high reliance on muscular and secondary ligamentous supports.

Current studies suggest that the ability of other muscles and secondary ligamentous supports to compensate for PTTD is limited. In vitro studies note that the flexor digitorum longus (FDL) and flexor hallucis longus (FHL) contribute to rearfoot inversion and MLA support, raising the possibility that these muscles may compensate for gradual loss of posterior tibialis function. Presurgical magnetic resonance imaging studies of subjects with PTTD document increases in FDL cross-sectional area, suggesting an increased contribution of this muscle to control foot kinematics. Assuming some compensation from these muscles, the abnormal kinematics associated with the subjects with PTTD suggest that these compensations were not effective. Further, magnetic resonance imaging studies of subjects with PTTD demonstrate abnormal signal in the spring ligament, sinus tarsi, and plantar aponeurosis at significantly higher rates than controls. These findings suggest increased strain on the ligamentous support of the foot in subjects with PTTD and, when combined with data from this study, support the view that (1) other muscles are limited in their ability to compensate and (2) there is an increased reliance on ligamentous support in subjects with stage II PTTD.

The study results are limited by the study design, patient population, and methods used to define foot kinematics. This is a cross-sectional study, thus cause-effect relationships alluded to in the discussion are speculative. The clinical classification of PTTD, as originally defined by Johnson and Strom, continues to be modified as new studies are completed. The inclusion criteria of an arch index equal to or greater than 1 standard deviation of a published study excluded subjects with a low MLA from the asymptomatic control group. Other foot posture measures, such as navicular height, may also be useful in categorizing subjects with PTTD. Comparisons to subjects with lower arch indexes may have resulted in less-distinct patterns attributed to the subjects with stage II PTTD. In addition, multisegment models of foot kinematics using skin-mounted markers are subject to errors. The particular IRED placements used in this study were developed specifically for tracking the medial side of the foot. Many multisegment foot models vary in their marker placement and definition of anatomic coordinate systems. Because foot kinematics are affected by these choices, comparisons across studies are challenging. The variables used in this study from the multisegment foot model were chosen to reflect the function of the posterior tibialis muscle on foot movement.

**Clinical Implications**

The goal of bracing/orthotic intervention of subjects with PTTD in stage I is to prevent the development of abnormal kinematics observed in this study (stage II PTTD). Theoretical models of disease progression from stage I to stage II suggest dysfunction of posterior tibialis muscle and involvement of secondary ligamentous support leading to clinical trials of orthotics/bracing treatments. A recent study suggests that an Arizona ankle foot orthosis (AFO) is effective in relieving symptoms of subjects with PTTD in 90% of cases over a 2-year period. Other authors reported success (67% of cases) over a 4-year period with either a molded AFO or a University of California Biomechanics Laboratory brace with medial posting. Current clinical guidelines are not specific regarding which brace or orthotic choice is optimal. The data from the current study suggest the choice of orthoses or brace should focus on the ability of the device to control rearfoot eversion, MLA angle, and forefoot abduction across the entire stance period. Future studies should address the ability of braces and orthoses in controlling abnormal foot kinematics in addition to symptom relief in subjects with PTTD.

Although there is little evidence for the efficacy of exercise for subjects with PTTD, it is a recommended intervention for subjects with stage I PTTD. Recent studies suggest that exercises which target the posterior tibialis muscle should include a forefoot adduction component combined with plantar flexion. The data from this study suggest that if exercises are to be effective, they should target muscles that play a role in controlling rearfoot eversion, MLA angle, and forefoot abduction. Although this study does not address the efficacy of exercise in influencing abnormal kinematics, it underscores the need for effective treatments that prevent progression of abnormal kinematics.

**CONCLUSIONS**

The data from this study extend previous research by documenting the abnormal kinematic patterns displayed by subjects that are classified as stage II PTTD. The findings show that abnormal kinematics occur across stance, suggesting a greater reliance on compensatory muscle and secondary ligamentous contributions to foot kinematics. These findings suggest that the posterior tibialis muscle plays a key role in controlling rearfoot, MLA, and forefoot kinematics during gait.
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