Comparison of Changes in Posterior Tibialis Muscle Length Between Subjects With Posterior Tibial Tendon Dysfunction and Healthy Controls During Walking

Johnson and Strom\textsuperscript{15} developed a classification scheme used for posterior tibial tendon dysfunction (PTTD) that includes 3 stages. These stages were defined using clinical measures of strength and observations of abnormal foot postures exhibited by subjects with PTTD. These abnormal foot postures characterize the flatfoot deformity common to PTTD and are clinically thought to be associated with a lengthened posterior tibialis (PT) tendon.\textsuperscript{15} Stage I is categorized by mild weakness, no foot deformity, and normal tendon length. Stage II is indicative of a lengthened tendon, with various degrees of progressing hindfoot (HF) eversion (or valgus positioning), forefoot (FF) abduction, and lowering of the medial longitudinal arch (MLA). Stage III includes progression of stage II deformities into a fixed foot posture, with marked degeneration and lengthening of the tendon.\textsuperscript{15} Alterations in PT muscle architecture have been documented in sub-

\textbf{OBJECTIVE:} To compare posterior tibialis (PT) length between subjects with stage II posterior tibial tendon dysfunction (PTTD) and healthy controls during the stance phase of gait.

\textbf{BACKGROUND:} The abnormal kinematics demonstrated by subjects with stage II PTTD are presumed to be associated with a lengthened PT musculotendon, but this relationship has not been fully explored.

\textbf{METHODS:} Seventeen subjects with stage II PTTD and 10 healthy controls volunteered for this study. Subject-specific foot kinematics were collected using 3-D motion analysis techniques for input into a general model of PT musculotendon length (PTLength). The kinematic inputs included hindfoot eversion/inversion (HF Ev/Inv), forefoot abduction/adduction (FF Ab/Add), ankle plantar flexion/dorsiflexion (FF Pf/Df), and ankle plantar flexion/dorsiflexion (Ankle Pf/Df). To estimate the change in PTLength from neutral the following model was used: $PTLength = 0.401(\text{HF Ev/Inv}) + 0.270(\text{FF Ab/Add}) + 0.137(\text{FF Pf/Df}) + 0.057(\text{Ankle Pf/Df})$. Positive values indicated lengthening from the subtalar neutral (STN) position, while negative values indicated shortening relative to the STN position. A 2-way analysis of variance (ANOVA) model was used to compare PTLength between groups across the stance phases of walking (loading response, midstance, terminal stance, and pre-swing). Also, a 2-way ANOVA was used to assess the foot kinematics that contributed to alterations in PTLength. The Short Musculoskeletal Functional Assessment Index and Mobility subscale were used to compare function and mobility.

\textbf{RESULTS:} PTLength was significantly greater (lengthened) relative to the STN position in the PTTD group compared to the control group across all phases of stance, with the greatest between-group difference in PTLength occurring during preswing. The greater PTLength in subjects with PTTD compared to controls was principally attributed to significantly greater HF Ev/Inv during loading response ($P = .014$) and midstance ($P = .015$). During terminal stance and preswing, each kinetic input to estimate PTLength contributed to lengthening (main effect, $P = .03$ and $P = .01$, respectively). Subjects with PTTD with abnormally greater PTLength reported significantly lower function ($P = .04$) and mobility ($P = .03$) compared to subjects with PTTD with normal PTLength during walking.

\textbf{CONCLUSIONS:} The greater PTLength, as determined from foot kinematics, suggests that the PT musculotendon is lengthened in subjects with stage II PTTD, compared to healthy controls. The amount of lengthening is not dependent on the phase of gait; however, different foot kinematic inputs contribute to PTLength across the stance phase. Targeting these foot kinematics may limit lengthening of the PT musculotendon. Subjects with excessive PT lengthening experience a decrease in function. \textit{J Orthop Sports Phys Ther} 2007;37(11):661-669.

doi:10.2519/jospt.2007.2539

\textbf{KEY WORDS:} foot kinematics, pronation, tendinopathy, walking

\begin{figure}[h]
\centering
\includegraphics[width=\textwidth]{figure1.png}
\caption{Figure 1: Schematic diagram of the musculoskeletal system showing the relationship between foot kinematics and PTLength.}
\end{figure}
joints with PTTD, suggesting that changes in muscle length may also be present in subjects with PTTD. Lengthening of the PT musculotendon (muscle and tendon) is assumed to be linked to abnormal foot kinematic motion.

Studies using both in vitro and in vivo methods report abnormal foot kinematics that characterize the flatfoot deformity associated with PTTD. Abnormal foot kinematics observed in vitro include excessive HF eversion, FF abduction, and lowering of the MLA when simulating loss (eg, releasing tension) of the PT muscle. In vivo studies confirm that these same abnormal foot kinematics occur during walking. Whether these abnormal foot kinematics reflect lengthening of the PT tendon or a combination of length changes in the tendon and muscle are not clear. Recent in vitro studies suggest a strong link between foot kinematics (excessive HF eversion, FF abduction, and lowering of the MLA) and PT musculotendon length (PTLength), consisting of the tendon and muscle length combined. However, PTLength in vivo, during walking, has not yet been explored.

Estimating PTLength in vivo during walking is challenging because the PT tendon crosses several ankle and foot joints, making the development of subject-specific estimates difficult. Muscle modeling using moment arms and joint excursion would require estimates specific to each joint crossed by the PT tendon. Current in vivo foot kinematic models do not provide the necessary joint angles or moment arms at these joints. In contrast, Flemister et al developed a general model to determine the change in PTLength from a neutral position based on 24 different foot positions measured in vitro. This general model was highly predictive of PTLength, accounting for 85% of the variance in predicting length and leaving ± 2.0 mm unexplained. The foot kinematic model used by Flemister et al was identical to the model used in a previous in vivo study. Thus a similar foot kinematic model applied during walking using subject-specific foot kinematic variables may provide a good estimate of changes in PTLength from a neutral position. A total excursion of 17.5 mm was observed from the neutral position in the in vitro study (8.3 mm of lengthening and 9.2 mm of shortening), suggesting that larger amplitude movements may result in low relative errors (errors less than 12%). However, the changes in PTLength that normally occur across the stance phase of walking are currently unknown. The large differences in foot kinematics observed in subjects with PTTD compared to controls are hypothesized to be linked with differences in PTLength between groups that are larger than the expected errors using this model.

Using subject-specific foot kinematics to estimate PTLength provides the ability to determine the contribution of abnormal foot kinematics (HF eversion/inversion [Ev/Inv], FF plantar flexion/dorsiflexion [Pf/Df], FF abduction/adduction [Ab/Add], and ankle Pf/Df) to PTLength. How separate abnormal foot kinematics combine to influence musculotendon length is unclear. For example, during late stance the hindfoot is inverting, suggesting a shortening of the PT musculotendon, while the forefoot is abducting, suggesting a lengthening. The general model of PTLength provides a means to determine the relative effect of separate abnormal foot kinematics. Treatments (bracing and exercises) that limit abnormal foot kinematics that lengthen the PT musculotendon are hypothesized to show the greatest clinical benefit. For example, in vitro data suggest controlling abnormal HF eversion and FF abduction, not MLA height, may produce the largest reduction in PTLength. However, subject-specific foot kinematics during walking were not utilized in this analysis.

While biomechanical measures like PTLength are useful for new brace design and documenting changes in foot function, their association with self-report scales is unexplored. Theoretically, PTLength may increase as a person progresses from stage I to stage II PTTD. In fact, subjects with more severe stage II disease are expected to present with more foot deformity and demonstrate worse function than those with less severe stage II disease. If self-report scales of functional status indicate lower function in subjects with a lengthened PT musculotendon, this supports the meaningfulness of PTLength as a clinical marker of disease progression in subjects with stage II PTTD.

The primary purpose of this study was to compare PTLength during the stance phase of walking between subjects diagnosed with stage II PTTD and matched asymptomatic healthy controls. The dependent variable was a change in PTLength relative to subtalar neutral (STN), with positive values indicating lengthening and negative values shortening. Our primary hypothesis was that subjects with stage II PTTD would demonstrate a greater PTLength (more positive) across all phases of stance, compared to healthy controls. If our primary hypothesis was supported, 2 secondary purposes were planned a priori. These included (1) to compare which foot kinematics (HF Ev/Inv, FF Ab/Add, FF Pf/Df, and ankle Pf/Df) contributed to PTLength in the PTTD group compared to the controls, and (2) to determine if PTLength was associated with self-reports of function in subjects with stage II PTTD. Our secondary hypotheses were that (1) HF eversion and FF abduction would be significant contributors to PTLength in the PTTD group compared to controls, and (2) that those subjects in the PTTD group with a lengthened PT musculotendon would report a decrease in function compared to subjects with PTTD, with a PTLength comparable to that of controls.

**MATERIAL AND METHODS**

**Subjects**

Seventeen subjects with PTTD (14 female, 3 male) and 10 control subjects (7 female, 3 male) participated. Subjects with unilateral PTTD...
were referred by a local orthopedic surgeon and were clinically classified as having stage II PTTD. The inclusion criteria for classification of stage II PTTD required subjects to have 1 or more signs related to tendinopathy, including (1) palpable tenderness of the PT tendon, (2) swelling of the PT tendon sheath, and (3) pain along the course of the PT tendon during a single-limb heel raise. Additionally, 1 or more signs of flexible flatfoot deformity, while standing, were required. These included excessive non-fixed rearfoot valgus deformity, excessive forefoot abduction, or demonstrated loss of height in the MLA. Signs of flatfoot deformity were based on visual comparisons from the involved to the uninvolved side and quantified on the involved side using the arch index (Table 1), as described by Williams et al. 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 control group was chosen from an asymptomatic convenience sample based on similar age, height, and body mass characteristics to those with stage II PTTD (Table 1). Inclusion criteria were no history of foot and ankle problems (absence of tendinopathy), a normal arch index, and normal foot posture based on visual assessment during standing. 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 first metatarsophalangeal joint line. Greater values indicate a higher arch. A normal arch was defined as equal to or greater than 1 standard deviation higher than average, as reported by Williams et al. All subjects were informed of the experimental procedures and signed a consent form approved by the University of Rochester Research Subjects Review Board, and the Ithaca College All College Review Board for Human Subjects Research.

### TABLE 1

<table>
<thead>
<tr>
<th>Variables</th>
<th>PTTD Subjects (n = 17)</th>
<th>Control Subjects (n = 10)</th>
<th>P Value*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (y)</td>
<td>56.1 (11.6)</td>
<td>50.2 (6.8)</td>
<td>.11</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>169.4 (8.1)</td>
<td>165.7 (13.9)</td>
<td>.47</td>
</tr>
<tr>
<td>Body mass (kg)</td>
<td>95.4 (22.2)</td>
<td>87.5 (44.2)</td>
<td>.27</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>33.2 (7.4)</td>
<td>31.8 (3.8)</td>
<td>.52</td>
</tr>
<tr>
<td>Arch index</td>
<td>0.311 (0.036)</td>
<td>0.384 (0.026)</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Gait speed (m/s)</td>
<td>1.19 (0.22)</td>
<td>1.34 (0.22)</td>
<td>.12</td>
</tr>
</tbody>
</table>

Abbreviations: BMI, body mass index; PTTD, posterior tibial tendon dysfunction. *No significant differences using independent t test between groups with the exception of the arch index.

#### Kinematic Measurements

Kinematic data were collected using a 3-segment foot model, which included the tibia, calcaneus (hindfoot), and first metatarsal (forefoot). Sets of 3 infrared-emitting diodes (IREDs) were mounted on rigid thermoplastic platforms, then attached to the body using double-sided adhesive tape. Anatomic landmarks were digitized to establish local anatomically based coordinate systems for each segment consistent with previous studies. Kinematic data were smoothed using a fourth-order, zero-phase-lag Butterworth filter, with a cut-off frequency of 6 Hz. A Cardan angle z-x-y sequence of rotations was used, as suggested by Cole et al, to calculate the 4 angles listed below for input into a regression equation to estimate the change in PTLength from a neutral (zero) position.

The model of PTLength developed by Flemister et al was based on a controlled in vitro experiment utilizing the same tracking of foot kinematics of a previous in vivo study. The in vitro model used by Flemister et al included tracking of the calcaneus, first metatarsal, and tibia to estimate joint angles using bone-mounted markers. For the present study, skin-mounted markers were used, but all other aspects of the kinematic tracking and modeling (anatomic landmarks and Cardan angle sequence) were identical. In the in vitro study the ankle, hindfoot (calcaneus) and forefoot (first metatarsal) were positioned in 24 distinct positions of Ankle Pf/Df, HF Ev/Inv, FF Ab/Add, and FF Pf/Df. On-line feedback was used to achieve reproducible foot positions. The 24-foot positions included isolated positions (eg, Ankle Pf/Df with neutral HF and FF positions) and combined positions (eg, ankle plantar flexion with HF eversion and FF abduction). The foot positions achieved were meant to simulate the range of positions that may occur across a variety of tasks. In each foot position the location of a weight sutured to the PT tendon was recorded and subsequently used in a regression analysis to develop a general equation to estimate the change in PTLength from the neutral position. Foot positions and corresponding positions of the weight hanging from the PT tendon were referenced to the STN position. The regression equation, PTLength = 0.401(HF Ev/Inv) + 0.270(FF Ab/Add) + 0.137(FF Pf/Df) + 0.057(Ankle Pf/Df), was the result and was used in the present analysis to estimate the change in length of the PT musculotendon from the STN position. Positive values of PTLength indicate lengthening of the PT musculotendon from the STN position, while negative values indicate shortening relative to the STN position. This equation to estimate PTLength showed a high r² value (0.85) and low standard error of the estimate of ±0.2 mm. When this equation was applied to individual specimens, the errors in PTLength varied from 1.6 to 2.5 mm. These results suggest that when comparing groups, movements that result in changes in PTLength from the neutral position of greater than 2.5 mm may yield reasonable estimates of differences in PTLength between groups.
For comparison between subjects, a reference (zero) STN position was established for each subject. This position then served as the baseline length for the PT musculotendon. Accordingly, PT\textsubscript{Length} comparisons were made to this position across the stance phase of walking. From a relaxed standing posture, subjects were positioned into STN, which was palpated consistently with published protocols.\textsuperscript{37} Determination of weight-bearing STN has shown low repeatability errors (<3°) in standing.\textsuperscript{28,35} Subjects were asked to hold the STN position for 3 seconds while kinematic data were collected. The mean of 2 STN trials was used as the reference position of each subject. Between session, reliability of the kinematic measures during gait, utilizing the STN position as a reference, was gathered from 4 subjects. Results show high intracllass correlation coefficients (ICC\textsubscript{3,1}>0.9) and low standard errors of the measurement (2.0°).\textsuperscript{35}

### Data Analysis

To address the primary purpose of this study, a 2-way mixed-effect ANOVA model was used to compare the dependent variable (PT\textsubscript{Length}) across the stance phases of walking. Group assignment served as the fixed factor, with 2 levels (PTTD and control group), and phase of stance as the repeated factor, with 4 levels (loading response [LR], midstance [MS], terminal stance [TS], and preswing [PS]). Data were analyzed by identifying the midpoint of each phase (10%, 35%, 70%, and 95% of stance), where LR occurs from 0% to 20%, MS from 20% to 50%, TS from 50% to 90%, and PS from 90% to 100% of stance.\textsuperscript{27} Two hypotheses were tested, first that PT\textsubscript{Length} would be greater in the PTTD group compared to the control group across all phases (main effect). The second hypothesis was that the difference in PT\textsubscript{Length} between groups would be phase dependent (interaction effect). To account for differences in self-selected walking speed on PT\textsubscript{Length}, the ANOVA model was repeated with walking speed as a covariate in the model. The significance level (alpha) was set at .05.

A secondary purpose was to identify which kinematic variables significantly contributed to PT\textsubscript{Length} in the PTTD and control groups. The dependent variable in this analysis was the PT\textsubscript{Length} attributed to each foot kinematic variable (HF Ev/Inv, FF Abd/Add, Ankle Pf/Df, and FF Pf/Df) of the regression equation. Examining the PT\textsubscript{Length} attributed to each foot kinematic variable is important because the regression equation takes into account a weighting factor (regression coefficient) and subject-specific kinematics, making direct (unweighted) comparisons of foot kinematics potentially misleading. Note that the ±2.5 mm error estimate reported by Flemister et al\textsuperscript{30} applies to the composite model of PT\textsubscript{Length} and not the individual kinematic PT\textsubscript{Length} contributions (HF Ev/Inv, FF Abd/Add, Ankle Pf/Df, and FF Pf/Df) used in this analysis. A 2-way mixed-effect ANOVA model was repeated for each phase of stance in which the PTTD group showed a significant difference compared to the controls in PT\textsubscript{Length} (primary purpose). Group served as the fixed factor, with 2 levels (PTTD and control group), while kinematic variables used to estimate PT\textsubscript{Length} served as the repeated factor, with 4 levels (HF Ev/Inv, FF Abd/Add, Ankle Pf/Df, and FF Pf/Df). In the event of an interaction, main effects were ignored and pairwise comparisons were completed. The significance level (alpha) was set at .05.

For the purpose of determining if subjects with PTTD who had greater PT\textsubscript{Length} were worse functionally, the subjects with PTTD were divided into 2 groups. Subjects with abnormal PT\textsubscript{Length} (n = 6) were identified as those with a PT\textsubscript{Length} that exceeded 2 standard deviations of the control group at the point of peak shortening in the PT musculotendon. Those subjects that were within 2 standard deviations of the control group at the point of peak shortening of the PT musculotendon (n = 10) were considered to have a normal PT\textsubscript{Length}. The peak shortening point was identified for this analysis because
shortening of the PT musculotendon is thought to play a key role in converting the foot into a rigid lever for push-off.\textsuperscript{3,14,27} PT\textsubscript{Length} at the point of peak shortening is hypothesized to be associated with functional ability in subjects with stage II PTTD.\textsuperscript{23} The average scores of the SMFA-Function Index and the SMFA-Mobility Subscale for each group were compared using a 2-sample t test ($\alpha = .05$). Subjects who reported feeling worse or much worse have reported average change scores of 10.25 points.\textsuperscript{33} Therefore, differences of 10 points were considered clinically significant differences in this analysis. SPSS software Version 13.0 was used for each analysis.

**RESULTS**

**PT\textsubscript{Length} Across Stance**

A significant difference ($P = .008$) between groups across all phases of stance (main effect) was observed in the variable PT\textsubscript{Length}. With the PTTD group demonstrating a greater PT\textsubscript{Length} compared to the healthy control group (FIGURE 1 and TABLE 2). This difference between groups was not dependent on phase of stance (group-by-phase interaction, $P = .264$). After entering walking speed as a covariate in the analysis, the main effect of a greater PT\textsubscript{Length} in the PTTD group remained. The average difference in PT\textsubscript{Length} across all phases of stance between the PTTD group and the healthy controls was 3.24 mm ($P = .008$) before controlling for gait speed, while this difference was 3.07 mm ($P = .017$) after entering speed as a covariate.

**Kinematic Contributions to PT\textsubscript{Length}**

During the LR and MS phases of stance, the contributions to PT\textsubscript{Length} from each kinematic input were dependent on group and kinematic variable (group-by-kinematic interaction: LR, $P = .009$; MS, $P = .005$). The greater PT\textsubscript{Length} in the PTTD compared to the control group was due to significantly larger contributions from HF eversion ($P = .014$), and a small ($<1$ mm) but statistically significant ($P = .01$) contribution attributed to FF dorsiflexion (TABLE 3). The greater PT\textsubscript{Length} during MS in the PTTD group compared to the controls was due to significantly larger contributions from HF eversion ($P = .015$), while a small ($<1$ mm) but statistically significant ($P = .02$) contribution was attributed to FF abduction (TABLE 3). Although HF eversion and FF abduction contributed the most to PT\textsubscript{Length} during the TS (91%) and PS (61%) phases of stance, the greater PT\textsubscript{Length} in the PTTD group was not dependent on a specific kinematic contribution (group-by-kinematic interactions: TS, $P = .28$; PS, $P = .07$). Rather, all kinematic contributions combined to predict a greater PT\textsubscript{Length} in the PTTD group (main effect: TS, $P = .03$; PS, $P = .01$) compared to controls.

**Functional Limitations and the Estimate of PT\textsubscript{Length}**

Subjects with PTTD either exhibited a PT\textsubscript{Length} that was outside 2 standard deviations of the mean of the controls at the point of peak shortening ($n = 6$; PT\textsubscript{Length} >6.5 mm) or a PT\textsubscript{Length} that was within...
2 standard deviations of the controls ($n = 10$; $PT_{\text{Length}} < 6.5$ mm). One subject with PTTD failed to complete the SMFA and was lost to follow-up. Subjects with PTTD who had abnormally greater $PT_{\text{Length}}$ had significantly higher scores (by 13 points) on the SMFA-Function Index ($P = .04$), indicating worse function, compared to PTTD subjects who had similar $PT_{\text{Length}}$ to that of the controls.

### DISCUSSION

The new findings from this study support the hypothesis that a foot-kinematic-based estimate of $PT_{\text{Length}}$ (positive values indicating lengthening) suggests greater musculotendon length of the PT across stance in subjects with stage II PTTD compared to healthy controls (FIGURE 1). However, whether the differences in $PT_{\text{Length}}$ arise from tendon elongation or alterations in muscle length (sarcomere length) is unknown. The abnormal foot kinematics associated with PTTD that were applied in this study were presented in a previous study. New to this study is the identification of hindfoot eversion and forefoot abduction as the primary contributors toward a greater $PT_{\text{Length}}$ in subjects with PTTD compared to controls during the first half of stance (LR and MS), not first metatarsal dorsiflexion (a measure associated with the MLA). During the second half of stance (TS and PS) hindfoot eversion and forefoot abduction contribute the most to $PT_{\text{Length}}$, although all motions contribute to a greater $PT_{\text{Length}}$ in the PTTD group compared to controls. This suggests that footwear, orthotics, and exercise that restrict abnormal hindfoot eversion and first metatarsal abduction may be more effective at maintaining $PT_{\text{Length}}$ than treatments directed at the

### TABLE 3

<table>
<thead>
<tr>
<th>Loading response (10%)</th>
<th>Control Group (n = 10)</th>
<th>Effect Size</th>
<th>$P$ Value for Overall Differences</th>
</tr>
</thead>
<tbody>
<tr>
<td>$HF_{\text{Ev/Inv}}$</td>
<td>3.3 (2.0)*</td>
<td>12 (1.9)</td>
<td>2.1</td>
</tr>
<tr>
<td>$FF_{\text{Ab/Add}}$</td>
<td>2.4 (1.1)</td>
<td>18 (1.3)</td>
<td>0.6</td>
</tr>
<tr>
<td>Ankle $PV/DI$</td>
<td>-0.2 (0.3)</td>
<td>-0.2 (0.2)</td>
<td>0.0</td>
</tr>
<tr>
<td>$FF_{\text{Pf/Df}}$</td>
<td>0.7 (0.5)*</td>
<td>0.0 (0.8)</td>
<td>0.7</td>
</tr>
<tr>
<td>Total</td>
<td>6.2 (2.9)</td>
<td>2.8 (2.9)</td>
<td>3.4</td>
</tr>
<tr>
<td>Midstance (35%)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>$HF_{\text{Ev/Inv}}$</td>
<td>4.0 (1.8)*</td>
<td>2.2 (1.5)</td>
<td>1.8</td>
</tr>
<tr>
<td>$FF_{\text{Ab/Add}}$</td>
<td>2.3 (1.0)*</td>
<td>14 (0.9)</td>
<td>0.9</td>
</tr>
<tr>
<td>Ankle $PV/DI$</td>
<td>0.1 (0.3)</td>
<td>0.2 (0.2)</td>
<td>0.1</td>
</tr>
<tr>
<td>$FF_{\text{Pf/Df}}$</td>
<td>11 (0.7)</td>
<td>0.7 (0.9)</td>
<td>0.4</td>
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<tr>
<td>Total</td>
<td>75 (2.7)</td>
<td>4.4 (3.0)</td>
<td>3.1</td>
</tr>
<tr>
<td>Terminal stance (70%)</td>
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<tr>
<td>$HF_{\text{Ev/Inv}}$</td>
<td>3.4 (1.7)</td>
<td>2.5 (1.1)</td>
<td>0.9</td>
</tr>
<tr>
<td>$FF_{\text{Ab/Add}}$</td>
<td>2.7 (1.0)</td>
<td>15 (0.8)</td>
<td>1.2</td>
</tr>
<tr>
<td>Ankle $PV/DI$</td>
<td>0.4 (0.2)</td>
<td>0.4 (0.1)</td>
<td>0.0</td>
</tr>
<tr>
<td>$FF_{\text{Pf/Df}}$</td>
<td>2.0 (0.9)</td>
<td>1.8 (0.7)</td>
<td>0.2</td>
</tr>
<tr>
<td>Total</td>
<td>8.5 (2.8)</td>
<td>6.2 (2.1)</td>
<td>2.3</td>
</tr>
<tr>
<td>Preswing (95%)</td>
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<td></td>
</tr>
<tr>
<td>$HF_{\text{Ev/Inv}}$</td>
<td>10 (2.1)</td>
<td>-0.4 (1.4)</td>
<td>1.4</td>
</tr>
<tr>
<td>$FF_{\text{Ab/Add}}$</td>
<td>2.2 (1.4)</td>
<td>0.9 (1.1)</td>
<td>1.3</td>
</tr>
<tr>
<td>Ankle $PV/DI$</td>
<td>0.1 (0.4)</td>
<td>-0.2 (0.2)</td>
<td>0.3</td>
</tr>
<tr>
<td>$FF_{\text{Pf/Df}}$</td>
<td>0.2 (1.2)</td>
<td>-0.7 (1.4)</td>
<td>0.9</td>
</tr>
<tr>
<td>Total</td>
<td>3.6 (4.1)</td>
<td>-0.4 (3.1)</td>
<td>4.0</td>
</tr>
</tbody>
</table>

*Denotes a significant pairwise comparison ($P<.05$) between groups.
The clinical meaningfulness of a greater PTLength was supported by reports of worse function in subjects with greater PTLength (Figure 2). This finding is consistent with theories suggesting that worse function may be associated with abnormal foot kinematics and greater PTLength.

The shift toward a greater PTLength of 2.3 to 4.0 mm in the PTTD group compared to the controls indicates possible alterations in the function of the PT musculotendon. The 2.3-mm difference between groups observed at the TS phase of stance in PTLength is just below the measurement error of ±2.5 mm, and therefore should be viewed with caution. However, the differences between the subjects with PTTD and controls during the LR, MS, and PS phases are not attributable to measurement error. The PTLength differences of 3.4 to 4.0 mm in the PTTD group compared to controls represent an offset toward lengthening that begins at initial foot contact. The lengthened position at initial foot contact suggests failure to properly align the foot during swing. The difference in PTLength between PTTD and control subjects across stance are only slightly smaller than the peak-to-peak excursion in PTLength across stance phases in the control group (6.2 mm [TS] – 0.4 mm [PS] = 6.6 mm). In vitro data suggest that much greater changes in PTLength are possible, observing an average 17.5-mm change in PTLength from maximum shortening (~9.2 mm) to lengthening (8.3 mm). Although concurrent electromyography would be beneficial, studies of healthy subjects suggest consistent activation of the PT during LR and TS through PS with inconsistent activation during MS. Given the small excursion observed in PTLength across stance, the function of the PT muscle may be viewed as primarily isometric; however, verification of this hypothesis would require information on muscle fiber length. Whether the small change toward lengthening indicated by greater PTLength in the PTTD group compared to the controls should be attributed to tendon or muscle elongation is unclear. As others have, this study hypothesized that a shorter PTLength may have therapeutic effects, either through maintaining the force/length properties of the muscle or length of the tendon.

The foot kinematics primarily responsible for a greater PTLength in subjects with PTTD is hindfoot eversion and first metatarsal abduction during early stance (LR and MS), not first metatarsal dorsiflexion or ankle dorsiflexion. During LR, when the body is progressing over the heel, excessive HF eversion has on lengthening of the PT. This agrees with other clinical data that identify excessive HF eversion as a common clinical sign of stage II PTTD. Further, these data suggest that strategies to limit hindfoot eversion and forefoot abduction show the greatest promise in decreasing PTLength. Orthoses such as the Arizona Brace and the University of California Biomechanics Laboratory (UCBL) may provide control for excessive HF eversion after heel-off. Additionally, control of foot kinematics during the swing phase may be necessary given the greater PTLength observed in subjects with PTTD compared to controls at initial contact and toe-off.

Although abnormal MLA kinematics (FF dorsiflexion relative to the HF) contributed little to PTLength, they may indicate failure of secondary ligamentous support and decreased ability of the PT musculotendon to maintain the MLA. Studies document abnormal signal on magnetic resonance images of the spring ligament, sinus tarsi, and plantar fascia in subjects with PTTD. Failure of the PT musculotendon is thought to increase reliance on secondary ligamentous support, eventually leading to damage. Orthotic support of the MLA during gait is hypothesized to protect these ligaments. In addition, muscle control may also be enhanced through improved MLA alignment. An in vitro study demonstrated that the PT musculotendon controls MLA position only when the ligamentous support for the MLA is intact. This suggests the PT musculotendon is partially dependent.
on ligamentous support to lift the MLA. Further, Kulig et al.\(^9\) suggested that exercises performed while wearing orthotics increased the contribution of the PT muscle. Although control of abnormal first metatarsal dorsiflexion (viewed as an indication of MLA angle) in this study was found to only minimally contribute to a change in PT\(_{\text{Length}}\), protection of secondary ligamentous support and muscle control may warrant therapeutic strategies that limit abnormal MLA kinematics.

Subjects with PTTD who exhibited abnormally greater PT\(_{\text{Length}}\) reported a decrease in function and mobility. Some suggest stage II PTTD is too broad and should be subdivided into 2 substages (IIa and IIb).\(^2\) In the current study, 10 out of 16 subjects with PTTD were within 2 standard deviations of normal controls, suggesting less shift toward a greater PT\(_{\text{Length}}\). These subjects also noted higher function and more mobility on self-report scales compared to the 6 out of 16 subjects with abnormally greater PT\(_{\text{Length}}\) who fell outside of 2 standard deviations. The differences in function and mobility between these subgroups of subjects exceeded the estimated clinically meaningful change on each scale. While the groups are small, these preliminary data support the concept that markers of PT\(_{\text{Length}}\) are useful for indicating disease progression, and may be useful in defining subgroups of stage II PTTD. Whether other clinical markers of PT\(_{\text{Length}}\) that do not require 3-D movement analysis show similar results is worthy of investigation.

**Limitations**

The study results are limited by the study design, patient population, and methods used to define foot kinematics. The inclusion criteria required control subjects to have an arch index equal to, or greater than, 1 standard deviation higher than the average in a previous study.\(^2\) Comparison to subjects with lower arch heights may have made comparisons between groups less distinct.

This study is a case control design and therefore is unable to determine cause-effect relationships. The estimate of PT\(_{\text{Length}}\) is based on the correlation with identified foot kinematic variables, not a direct measure of PT\(_{\text{Length}}\). Thus, the model of PT\(_{\text{Length}}\) used is dependent on the ability of skin-mounted markers to track bone movement, which is supported by current studies.\(^28,41\) Additionally, differences in anthropometrics (foot length measures) between the current sample of subjects and the sample of cadaver specimens used to develop the equation to predict changes in PT\(_{\text{Length}}\) may influence PT\(_{\text{Length}}\) estimates.\(^10\) The reported loss in accuracy in using a general model versus a subject-specific model that includes anthropometric adjustments to predict PT\(_{\text{Length}}\) is approximately 10%.\(^10\) The foot kinematic model used to calculate angle variables included only the first metatarsal to track the forefoot. Future studies may consider the inclusion of different tracking schemes to model the kinematics of the forefoot, as well as evaluating the dependence of foot kinematics on walking speed. The PT\(_{\text{Length}}\) was lower than expected; however, the differences between the PTTD and control groups exceeded the error (±2.5 mm) by a factor of 1.2 to 1.6 during the LR, MS, and PS phases of stance. More direct measures of muscle behavior during walking are desirable, yet are difficult to achieve in vivo.

**Conclusion**

The greater PT\(_{\text{Length}}\) during gait predicted from foot kinematics is consistent with theories suggesting that the PT musculotendon is lengthened in subjects with PTTD compared to healthy controls. Specific foot kinematics contribute more to PT\(_{\text{Length}}\) than others and, therefore, may be more important to control clinically. The largest contributions to PT\(_{\text{Length}}\) were from greater HF eversion and FF abduction in subjects with PTTD compared to healthy controls. The clinical relevance of PT\(_{\text{Length}}\) was supported by preliminary data of decreased function in subjects with PTTD who exhibited greater PT\(_{\text{Length}}\).

**Acknowledgements**

We would like to acknowledge support from collaborators in the Center for Foot and Ankle Research at Ithaca College.

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