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Deep Posterior Compartment Strength and Foot Kinematics in Subjects With Stage II Posterior Tibial Tendon Dysfunction

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ABSTRACT

Background: **Tibialis posterior muscle weakness has been documented in subjects with Stage II posterior tibial tendon dysfunction (PTTD) but the effect of weakness on foot structure remains unclear. The association between strength and flatfoot kinematics may guide treatment such as the use of strengthening programs targeting the tibialis posterior muscle.** ***Materials and Methods:*** **Thirty Stage II PTTD subjects (age; 58.1 ± 10.5 years, BMI 30.6 ± 5.4) and 15 matched controls (age; 56.5 ± 7.7 years, BMI 30.6 ± 3.6) volunteered for this study. Deep Posterior Compartment strength was measured from both legs of each subject and the strength ratio was used to compare each subject's involved side to their uninvolved side. A 20% deficit was defined, a priori, to define two groups of subjects with PTTD. The strength ratio for each group averaged; 1.06 ± 0.1 (range 0.87 to 1.36) for controls, 1.06 ± 0.1 (range, 0.89 to 1.25), for the PTTD strong group, and 0.64 ± 0.2 (range 0.42 to 0.76) for the PTTD weak group. Across four phases of stance, kinematic measures of flatfoot were compared between the three groups using a two-way mixed effect ANOVA model repeated for each kinematic variable.** ***Results:*** **Subjects with PTTD regardless of group demonstrated significantly greater hindfoot eversion compared to controls. Subjects with PTTD who were weak demonstrated greater hindfoot eversion compared to subjects with PTTD who were strong. For forefoot abduction and MLA angles the differences between groups depended on the phase of stance with significant differences between each group observed at the pre-swing phase of stance.** ***Conclusion:*** **Strength was associated with the degree of flatfoot deformity observed during walking, however, flatfoot deformity may also occur without strength deficits.** ***Clinical Relevance:*** **Strengthening programs may only partially correct flatfoot kinematics while other clinical interventions such as bracing or surgery may also be indicated.**

Key Words: Biomechanics; Tendinopathy; Gait; Kinematics; Motion Analysis; Foot And Ankle

INTRODUCTION

Posterior tibial tendon dysfunction (PTTD) is characterized by swelling and pain along the course of the posterior tibial tendon that can lead to adult acquired flatfoot deformity.[16] The onset of a flexible flatfoot deformity coupled with signs of tendinopathy are the hallmarks of Stage II dysfunction.[16] There is variability in the published data on flatfoot deformity that occurs during walking in subjects with Stage II PTTD.[21,26,31] It is proposed that changes in the muscles or ligaments that control foot kinematics may explain this variability. The association between muscle strength and foot kinematics is not yet fully understood, and weakness identified in subjects with Stage II PTTD may be associated with variability in the flatfoot deformity.

Current data describing flatfoot kinematics (hindfoot eversion, forefoot abduction, and a lower medial longitudinal arch (MLA)) collected using both in-vitro and in-vivo methods provide evidence of the role of the PT muscle. The result of simulating weakness (cutting the tibialis posterior tendon) in a controlled in-vitro environment includes changes in foot kinematics towards a flatfoot deformity.[17,22] Similar changes in foot kinematics when walking have been observed in subjects with Stage II PTTD compared to matched controls.[26,31] Also, studies using magnetic resonance imaging (MRI) to investigate muscle morphology in subjects with PTTD have found signs of muscle atrophy.[27,35,36] Weakness of the PT muscle is a component of PTTD and may be associated with flatfoot deformity. Yet, few studies[1] have examined

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tibialis posterior muscle strength, and no studies have associated flatfoot deformity and tibialis posterior muscle strength.

The clinical assessment of strength in the deep posterior compartment is sensitive to weakness of the tibialis posterior muscle observed in subjects with PTTD due in part to the muscle architecture present. The deep posterior compartment of the leg is made up of three muscles: the tibialis posterior, the flexor digitorum longus (FDL), and the flexor hallicus longus (FHL). The tibialis posterior is the largest of these muscles making up, on average, 57% of the physiological cross sectional area of the entire deep posterior compartment.[9,11,12] In addition to size, the tibialis posterior muscle's inversion moment arm crossing the talocrural and subtalar joint is estimated at two times that of the FHL, and ~10% greater than the FDL.[9,10] Due to differences in the size and moment arms of the deep posterior compartment muscles, the FDL and FHL may have difficulty compensating for a weak tibialis posterior muscle at the ankle joint. Therefore, despite the FDL and FHL being synergists of the tibialis posterior muscle, isometric forefoot adduction and subtalar inversion isometric tests are sensitive to weakness of the tibialis posterior muscle in subjects with Stage II PTTD.[13] Additionally, the characteristics of the tibialis posterior muscle architecture suggest an association between strength and foot kinematics may be present and warrants further study in subjects with Stage II PTTD.

Studies of foot kinematics provide evidence of the impact of PTTD on foot structure during walking. Despite considerable variability across studies, the presence of flatfoot kinematics, including excessive hindfoot eversion, forefoot abduction, and a lower medial longitudinal arch, are observed in subjects with Stage II PTTD compared to controls.[21,26,31] The normal progression of loading the foot, beginning with the heel and progressing to the toes, changes foot kinematics across the stance phase. Evidence suggests the role of the tibialis posterior muscle to control foot kinematics is greatest at the end of stance, during push-off when load is transferred through the forefoot.[14,22] The association between muscle weakness and flatfoot kinematics is unexplored in subjects with Stage II PTTD and may depend on the phase of stance.

The purpose of this study was to determine the effect of deep posterior compartment muscle strength on foot kinematics in subjects with Stage II PTTD. It was hypothesized that weakness in subjects with PTTD would be associated with greater hindfoot eversion, forefoot abduction, and a lower MLA compared to matched controls and subjects with PTTD who were strong. These differences would depend on the phase of stance.

MATERIALS AND METHODS

Thirty subjects with a diagnosis of Stage II PTTD and 15 matched control subjects volunteered for this study. Matching for the control group was done using body mass index (BMI) and age due to their known effect on foot structure and gross function during walking.[4,28] Control subjects were required to have a normal foot structure as defined using the arch height index and hindfoot measure while standing. All control subjects were required to have an arch height index greater than or equal to normal (0.340) as reported by Butler et al.[25] In resting standing position control subjects were required to have between two degrees of inversion and two degrees of eversion to be classified as having a normal foot structure.[7] These measures ensured control subjects would not demonstrate flatfoot deformity but could serve as a comparison group that displayed normal foot postures.

The inclusion criteria for classification of Stage II PTTD required subjects to have one or more signs related to tendinopathy including (1) palpable tenderness of the posterior tibial tendon, (2) swelling of the posterior tibial tendon sheath, and/or (3) pain along the course of the PT muscle or tendon while completing a single limb heel-rise. Additionally, one or more signs of flexible flatfoot deformity were required for classification of Stage II PTTD. These included excessive non-fixed hindfoot eversion deformity during weightbearing, excessive forefoot abduction, or demonstrated loss of height in the MLA.

Signs of flatfoot deformity were based on comparisons from the involved to the uninvolved side. This then required that all subjects in the PTTD group had unilateral involvement. The un-involved side may have also demonstrated signs of flatfoot deformity in some subjects but was not painful and did not demonstrate the same severity of flatfoot deformity (Table 1). 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 fifteen meters. All subjects were required to have sensate feet to ensure their safety with walking. Subjects with other foot conditions, such as plantar fasciitis, were also excluded from the current study. All PTTD subjects were required to be at least 40 years of age to restrict the study to only those with the typical degenerative onset of PTTD. All subjects were informed of the experimental procedures and signed a consent form approved by The University of Rochester and Ithaca College University Research Subject Review Boards.

Isometric test of ankle inversion and foot adduction strength

Deep posterior compartment strength was used as a primary independent variable to define groups; specifically, strength was used to divide the PTTD group into a weak and strong group. Based on pilot data, it was expected that PTTD subjects could be separated into two strength groups using a cut-off of 80% strength in the ratio of affected to unaffected side. The cut-off of 80% was supported by pilot data but represented a theoretical point at which weakness in the deep posterior compartment could no longer provide dynamic support to control foot structure. Posterior tibial tendon dysfunction subjects with greater than 80% strength in the ratio of affected to unaffected side were considered a "strong" group while those with less than

Table 1: Subject Classification Variables for Subjects with Stage II PTTD and Matched Controls.

	PTTD Total	PTTD Strong	PTTD Weak	Controls	*p* value
Subjects	$n = 30$	$n = 14$	$n = 16$	$n = 15$	
Age (years)	58.1 ± 10.5	57.9 ± 11.4	58.2 ± 10.0	56.5 ± 7.7	0.88
Height (cm)	167.2 ± 8.7	162.3 ± 8.4	$171.6 \pm 6.5^{\beta,\gamma}$	164.9 ± 7.3	0.004
Weight (kg)	86.0 ± 17.4	80.2 ± 14.6	91.1 ± 18.5	83.2 ± 10.8	0.134
BMI	30.6 ± 5.4	30.4 ± 5.2	30.8 ± 5.7	30.6 ± 3.6	0.98
Sex	19 F, 11 M	10 F, 4 M	9 F, 7 M	14 F, 1 M	0.06§
AHI @ 10%	0.330 ± 0.02	$0.341 \pm 0.02^{\alpha}$	$0.321 \pm 0.02^{\beta,\gamma}$	0.376 ± 0.03	<0.001
HF eversion Involved	9.8 ± 4.2	$8.6 \pm 5.0^{\alpha}$	$10.9 \pm 3.1^{\beta}$	1.6 ± 1.7	0.03
HF eversion Un-Involved	8.9 ± 4.5	$10.0 \pm 5.2^{\alpha}$	$8.0 \pm 3.8^{\beta}$	—	
Duration of Symptoms (mo)*		11.0 ± 12.1	10.0 ± 8.8		0.82

Values expressed as means ± SD. *p* values represent comparisons between PTTD groups and Control group using a one-way ANOVA. FF, forefoot; HF, hindfoot; PTTD, posterior tibial tendon dysfunction; AHI, arch height index. $^{\alpha}$, Denotes a significant difference (pairwise comparisons $p < 0.05$) between Control and PTTD strong group. $^{\beta}$, Denotes a significant difference (pairwise comparisons $p < 0.05$) between Control and PTTD weak group. $^{\gamma}$, Denotes a significant difference (pairwise comparisons $p < 0.05$) between PTTD strong and PTTD weak group. *, Represents duration of reported symptoms at time of testing. Four subjects (two in each group) reported symptoms starting greater than 5 years ago but were unable to report a date. These subjects were not included in the data. §, Results of Fisher exact statistical test to compare groups.

80% strength were considered a "weak" group. Therefore, it was anticipated that three groups would be defined for inclusion in this study, including: a group of Stage II PTTD subjects who demonstrated *strong* deep posterior compartment muscle strength, a group of Stage II PTTD subjects who demonstrated *weak* deep posterior compartment muscle strength, and a matched *control* group.

To measure deep posterior compartment muscle force, a device was used that has been previously shown to be sensitive to weakness of the deep posterior compartment in subjects with Stage II PTTD.[13] Briefly, data from a force transducer was viewed using an oscilloscope (TDS 410A, Tektronix, Beaverton, OR) as part of the strength set-up to record and display maximum isometric force recorded from the deep posterior compartment. The oscilloscope visually displays force from a maximum isometric ankle inversion and foot adduction strength effort. The force transducer (Model SML-200, Interface, Scottsdale, AZ) was connected in line with a resistance plate. The plate provided resistance to maximum efforts of ankle inversion and foot adduction. Calibration of the force transducer with known weights suggested low errors ($r^2 = 0.997$, root mean square error = ±1 N) when predicting force from voltage outputs. Stabilization of the leg was achieved with the subject sitting in a chair and supporting the ankle using an ankle stirrup brace attached to vertical supports. Padded supports fixed to the resistance plate allowed subjects to push from their resting foot position into forefoot adduction and ankle inversion.

To assess the potential for the anterior tibialis muscle to mask the decrement in ankle inversion and foot adduction, electromyographic feedback was used. A surface electrode (DE-2.1, Delsys, Inc., Boston, MA) was placed over the skin of the anterior tibialis muscle. The surface electrode was connected to a 2 channel EMG system (Bangoli-2 EMG System, Delsys, Inc., Boston, MA) for gain adjustments of 1 to 10K. An oscilloscope (TDS 410A, Tektronix, Beaverton, OR) was used to visually display force and surface electromyography from the anterior tibialis muscle. The force and electromyography readings were read directly from the oscilloscope. The digital display of the oscilloscope sampled data greater than 1000 Hz.

Procedures for isometric strength testing

Subjects were instructed to maintain plantarflexion force while performing a maximal voluntary ankle inversion and foot adduction effort. Subjects performed five to seven practice sub-maximal efforts and three maximal efforts on the involved and uninvolved sides. Rest periods between maximal efforts were included to avoid fatigue (rest time approximately 2 to 3 minutes as needed). If the anterior tibialis EMG rose, subjects were instructed to push downward on the ball of their foot, increasing the plantarflexor force and inhibiting the anterior tibialis muscle. Prior to testing, a maximum voluntary effort in dorsiflexion against manual resistance was recorded. For the proposed study, peak force was normalized to body mass and averaged across the three maximal efforts. This procedure was repeated with the unaffected leg so ratios between the affected and un-affected side could be calculated. For the control group a leg was randomly assigned at enrollment into the study to be the "involved" leg. The "involved" leg was used for the kinematic testing and also was compared to the contralateral leg to calculate the strength ratio.

Table 2: Test Re-Test Reliability for 11 Control and 11 Posterior Tibial Tendon Dysfunction (PTTD) Subjects.

		Trial 1	Trial 2	ICC Value – model (3,1) (95% CI)
Control	Right side	70.3 ± 13.4	71.4 ± 13.7	0.87 (0.50-0.97)
	Left side	70.9 ± 16.2	71.5 ± 13.6	0.95 (0.81-0.99)
PTTD	Involved side	51.3 ± 13.7	53.7 ± 16.8	0.94 (0.77-0.98)
	Uninvolved side	58.7 ± 18.1	64.2 ± 16.1	0.97 (0.90-0.99)

Units are in Newtons. CI, confidence interval.

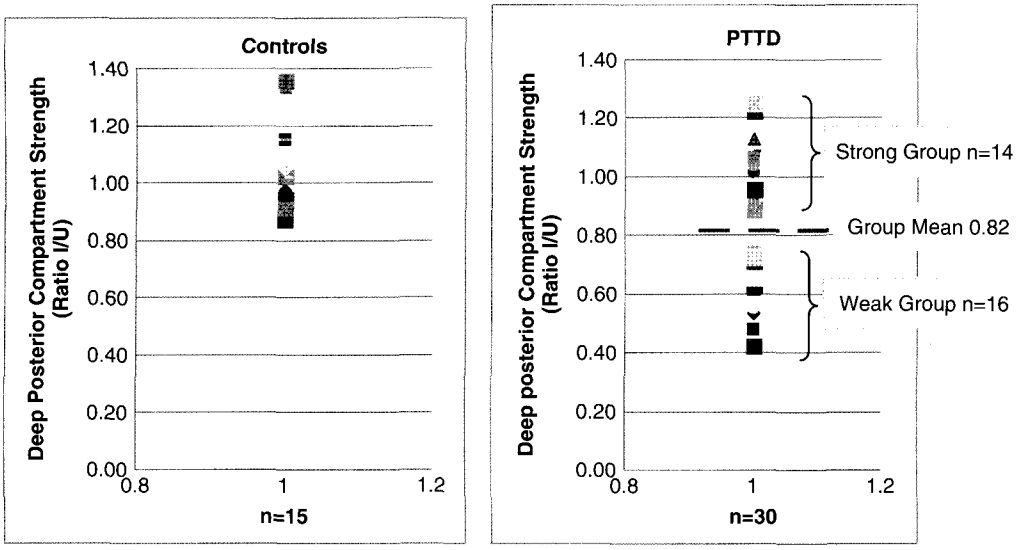

Fig. 1: Deep Posterior Compartment Strength Ratio for each subject in the control and PTTD groups. The PTTD group demonstrates a bimodal distribution with a 20% deficit used as a cut-off to define the two groups: weak and strong.

Preliminary reliability of healthy controls using the procedures in this study was completed previously and was considered good (ICC range, 0.76 to 0.91). Further, the reliability of isometric forefoot adduction and subtalar inversion strength from controls ($n = 11$) and subjects with PTTD ($n = 11$) using the methods described above was included as part of this study (Table 2) and ranged from 0.87 to 0.97. Analysis of all pilot data suggested a ratio of involved to uninvolved would serve to define two groups of PTTD subjects (weak, strong) based on a cut-off of 80%. Using this cut-off, the three groups in this study included the control group with an average strength ratio of 1.06 ± 0.1 (range, 0.87 to 1.36), the PTTD strong group with an average ratio of 1.06 ± 0.1 (range, 0.89 to 1.25), and the PTTD weak group with an average ratio of 0.64 ± 0.2 (range, 0.42 to 0.76) (Figure 1).

Kinematic measurements

A previously described five-segment kinematic model which included the tibia, calcaneus (hindfoot), first metatarsal (medial forefoot), second through fourth metatarsals (lateral forefoot), and navicular tuberosity was used.[31] The tibia, hindfoot, and medial forefoot segments were tracked by placing three IREDs on a thermoplastic molded platform on the skin overlying the segment. A single IRED was used for the navicular tuberosity. The lateral forefoot segment was tracked by placing an IRED at the base and head of the second metatarsal and a third IRED on the head of the fourth metatarsal. A previous in-vitro study suggested good repeatability and validity of the first metatarsal IRED set,[34] suggesting similar results were possible for other foot bones.

Anatomic landmarks were digitized by a single examiner (CGN) to establish local anatomically based coordinate systems for each segment. For this investigation, 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 abduction/adduction as suggested by Cole et al.[5] The three digitized points used to establish an anatomic coordinate system for the calcaneus were forced to be in the transverse plane of the global coordinate system. The points used include the midpoint on the posterior heel on the floor, the tip of the second toe, and the medial side of the foot on the floor. Similarly, for the medial and lateral forefoot the z-axis is consistent with the global system. Two of the three

digitized points used to establish anatomic coordinate systems for the medial and lateral forefoot segments were the base and head of the first and second metatarsals respectively. The third point was an arbitrary point that was at the same height as the metatarsal head. The MLA angle was defined using a point on the posterior calcaneus, a point on the first metatarsal head and the single IRED on the tuberosity of the navicular. The navicular marker served as the apex of the angle and the dot product of the 3-dimensional vectors from the navicular to the metatarsal head and navicular to the posterior heel defined the MLA angle.[31]

To adjust the anatomic coordinate systems to align with a reference zero, the sub-talar neutral (STN) position was used. Previous investigations have emphasized the importance of using a reference position when comparing among subjects with varying foot postures.[30] From their relaxed standing posture, subjects were positioned into STN, which was palpated as described in published protocols.[32] Determination of weight-bearing STN has shown low errors (less than 2 degrees) in previous studies and in our laboratory.[24] Subjects were asked to hold this position for three seconds while kinematic data were collected. The mean of two STN trials were used as the reference position for each subject. Preliminary evaluation of the methods used in this study demonstrated intraclass correlation coefficients (model 3, 1) above 0.9 within a session ($n = 18$) and differences (absolute values of between-session differences) in peak angles between sessions ($n = 4$) of less than 3 degrees for the tested variables. This error estimate combines errors due to digitizing and determining the STN position.

Two banks of infrared cameras (Optotrak model 3020, Northern Digital Inc, CAN), in conjunction with Motion Monitor software Version 7.24 (Motion Monitor, Innsport Training Inc, Chicago, IL) were used to track IRED sets on each segment at a sampling rate of 60 Hz. The field of view of the Optotrak was 2.25 m^2 at a distance of two meters. The manufacturer has reported accuracy of tracking an individual IRED at ± 0.1 mm with additional studies also reporting excellent precision and repeatability using the Optotrak system.[19,29] Using a 10-N threshold of vertical forces (collected at 1000 Hz from an embedded force plate, Model 9286, Kistler, Switzerland) initial contact and toe-off points of the gait cycle were identified. Kinematic data were smoothed using a fourth order, zero phase lag, Butterworth filter with a cut off frequency of six hertz.

Procedures for foot kinematics

Subjects walked down a 10-m walkway at a walking speed constrained to be 1.0 m/s. This constraint was required to allow comparisons between groups. Average self-selected walking speed is reported to be 1.4 m/s with only minor changes (less than 0.5 J/kg/m) in gross energy cost with speeds between 1.0 to 1.5 m/s[4]. This suggests the dynamic function of the body to walk at speeds between 1.0 to 1.5 m/s is consistent. It was expected dynamic foot function would also be consistent at these speeds. This allowed comparisons between groups without the confounding effects of gait speed. During testing, speed was monitored using an infrared timing system (Brower, Salt Lake City, UT). Each subject completed a minimum of five successful walking trials, which consisted of the appropriate speed and full contact of the tested foot with the force plate. Following the collection of the walking trials, a reference (or zero) STN position was established for each subject.

Analysis

A 3×4 mixed-design ANOVA model was used to assess each kinematic variable. The two factors of the model included a between-subjects factor of group with 3 levels (controls, PTTD weak, PTTD strong). The second factor was a within-subjects factor that included 4 levels representing the mid-points of each phase of the stance phase of gait. The stance phase was defined as loading response (0% to 20%), midstance (21% to 50%), terminal stance (51% to 90%), and preswing (91% to 100%). The midpoints of each phase were used to prevent small differences in the timing of kinematic patterns from influencing the results. For each ANOVA model, if significant interactions were detected (group $\times$ phase) they were followed by pairwise comparisons and main effects were ignored. A significance level was maintained for each analysis at alpha < 0.05.

RESULTS

There was a significant difference between groups ($p < 0.001$) for the variable hindfoot eversion that did not depend on the phase of stance. On average (across all phases of stance), the control group demonstrated 2.7 ± 5.3 degrees of eversion, the PTTD strong group 7.7 ± 2.3 degrees of eversion, and the PTTD weak group 10.5 ± 5.2 degrees of eversion. Post hoc comparisons between groups revealed significant differences between each group (Table 3, Figure 2).

The amount of forefoot abduction observed depended on group and phase of stance ($p < 0.001$) (Figure 3). Due to the dependence on phase for comparing each group subsequent analysis focused on pairwise comparisons as proposed a priori. Across loading response ($p = 0.004$), midstance ($p = 0.02$), and terminal stance ($p = 0.008$) subjects with PTTD who were weak demonstrated significantly greater forefoot abduction compared to controls. There was no difference between subjects with PTTD who were strong and those who were weak or between subjects with PTTD who were strong and controls. At the pre-swing phase of stance there was a significant difference ($p < 0.05$) between each group (Table 3).

The MLA angle depended on group and phase of stance ($p < 0.001$) (Figure 4). Due to the dependence on phase for comparing each group subsequent analysis focused on pairwise comparisons as proposed a priori. At loading

Table 3: Means and SD of Kinematic Compressable Pulsatile Mass. Variables Across the Stance Phase of Gait

	Phases of Gait*					
	LR	**MS**	**TS**	**PS**	***p* value for Overall Differences**	
HF Eversion						
Control	-5.1 ± 1.9	-5.5 ± 2.0	-3.8 ± 2.2	3.7 ± 2.7	Group	**<0.001**
PTTD Strong	$-10.6 \pm 2.6^{\alpha}$	$-10.5 \pm 2.5^{\alpha}$	$-8.1 \pm 2.1^{\alpha}$	$-1.5 \pm 3.3^{\alpha}$	Group × phase	0.231
PTTD Weak	$-12.3 \pm 5.9^{\beta,\gamma}$	$-13.2 \pm 5.0^{\beta,\gamma}$	$-11.5 \pm 4.8^{\beta,\gamma}$	$-5.1 \pm 6.0^{\beta,\gamma}$		
FF Abduction						
Control	-2.4 ± 1.9	-5.0 ± 2.2	-6.6 ± 2.1	1.2 ± 2.9	Group	0.002
PTTD Strong	-4.2 ± 2.7	-6.0 ± 2.5	-8.5 ± 2.5	$-3.8 \pm 4.1^{\alpha}$	Group × phase	**<0.001**
PTTD Weak	$-6.2 \pm 4.8^{\beta}$	$-8.1 \pm 4.6^{\beta}$	$-10.0 \pm 4.7^{\beta}$	$-7.3 \pm 5.5^{\beta,\gamma}$		
MLA						
Control	0.3 ± 3.1	2.2 ± 2.9	5.1 ± 3.0	1.8 ± 4.7	Group	<0.001
PTTD Strong	$6.1 \pm 4.8^{\alpha}$	$7.4 \pm 4.5^{\alpha}$	9.2 ± 4.6	$8.0 \pm 4.6^{\alpha}$	Group × phase	**<0.001**
PTTD Weak	$10.7 \pm 8.8^{\beta,\gamma}$	$12.0 \pm 8.4^{\beta,\gamma}$	$13.0 \pm 8.1^{\beta}$	$12.5 \pm 8.2^{\beta,\gamma}$		

LR, loading response; MS, midstance; TS, terminal stance; PS, pre-swing; MLA, medial longitudinal arch; FF, forefoot; PTTD, posterior tibial tendon dysfunction; HF, hindfoot. *, Values are determined at the midpoint of each phase of stance. $^{\alpha}$, Denotes a significant difference (pairwise comparisons $p < 0.05$) between Control and PTTD strong group. $^{\beta}$, Denotes a significant difference (pairwise comparisons $p < 0.05$) between Control and PTTD weak group. $^{\gamma}$, Denotes a significant difference (pairwise comparisons $p < 0.05$) between PTTD strong and PTTD weak group.

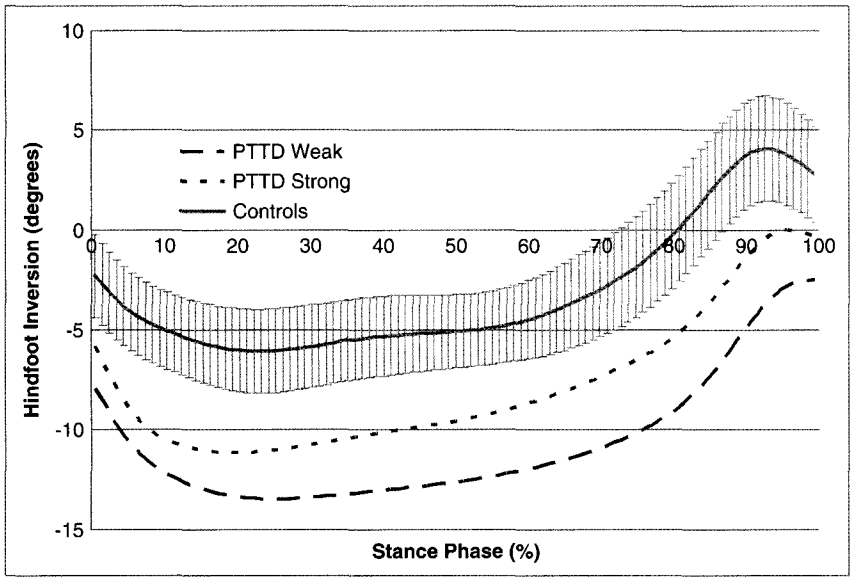

Fig. 2: Hindfoot inversion/ eversion kinematic pattern (hindfoot relative to the leg) across the stance phase of gait.

response, midstance, and preswing phases of stance there were significant differences ($p < 0.05$) between each group (Table 3). At terminal stance subjects with PTTD who were weak demonstrated a significantly greater MLA angle (lower MLA) compared to controls ($p < 0.001$). There was no difference between subjects with PTTD who were strong and those who were weak ($p = 0.07$) or between subjects with PTTD who were strong and controls ($p = 0.06$) at the terminal stance phase.

DISCUSSION

Data from this study suggest strength is associated with foot kinematics during walking in subjects with Stage II PTTD. Previous clinical reviews and guidelines have suggested two groups should be defined to appropriately categorize the clinical presentation of subjects with Stage II PTTD; however, no data were available to confirm these clinical observations.[3,18] The spectrum of strength deficits across

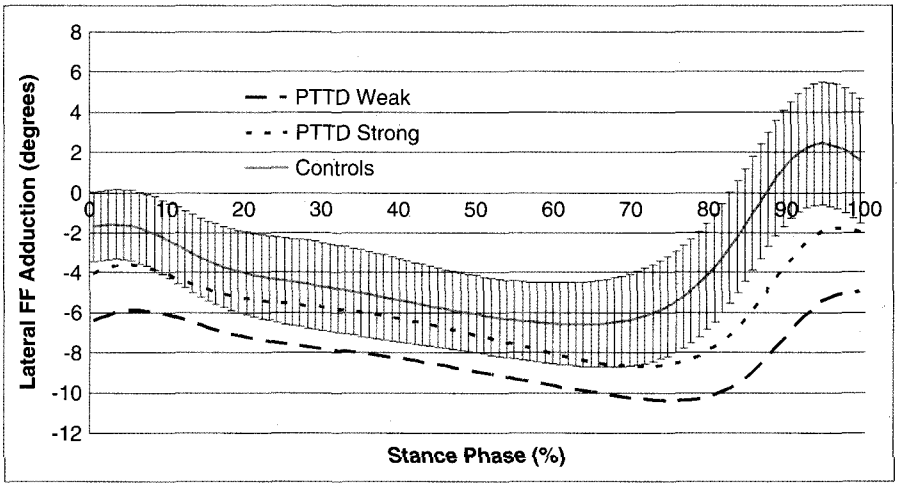

Fig. 3: Forefoot abduction/ adduction kinematic pattern (forefoot relative to the hindfoot) across the stance phase of gait.

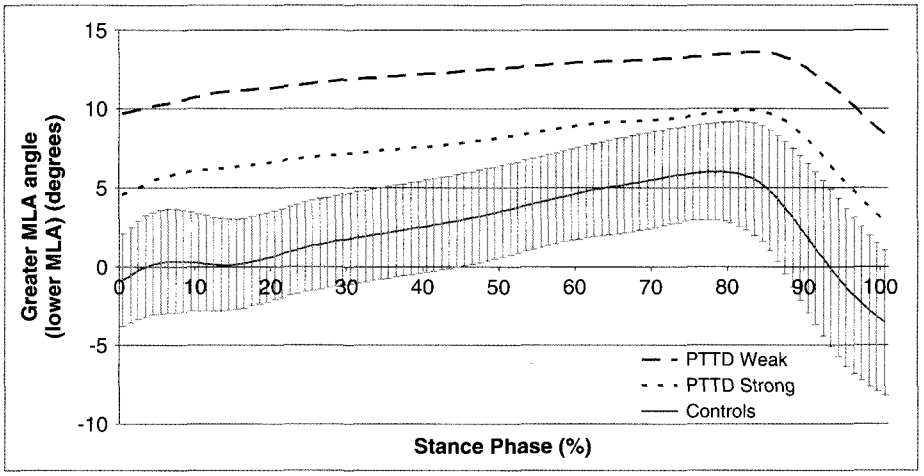

Fig. 4: Medial Longitudinal Arch angle across the stance phase of gait with greater angles indicating a lower arch.

subjects classified as having Stage II PTTD is represented in the strong and weak groups of this study. The approximately 40% difference in the isometric strength ratio was associated with specific aspects of flatfoot kinematics in the weak group as compared to the strong group. However, the strong PTTD group also showed significant differences from controls, suggesting specific aspects of flatfoot kinematics may not be associated with strength. The control group was selected based on a normal arch height making the differences in foot kinematics between the control and strong PTTD groups expected, but suggesting other factors are associated with flatfoot deformity in the absence of a strength deficit. These findings underscore the relationship between strength and flatfoot kinematics in subjects with PTTD and suggests strengthening programs may only partially correct flatfoot kinematics.

Weakness in the deep posterior compartment was associated with greater hindfoot eversion across all phases of stance, but a pre-existing flatfoot deformity may be evident in subjects with Stage II PTTD who are strong. The current study suggests that weakness was associated with approximately 3 degrees greater hindfoot eversion across stance (difference between averages across stance for the strong PTTD and weak PTTD groups). This finding is consistent with the well documented role of the tibialis posterior muscle in controlling hindfoot eversion.[14,22,23] Three degrees of eversion documented in this study is greater than previous in-vitro studies[14,22] suggesting an even larger potential role in controlling eversion than previously thought. Interestingly, the strong PTTD group also demonstrated greater hindfoot eversion relative to the control group by approximately 5 degrees (difference between averages across stance for the strong PTTD and control groups). The greater hindfoot eversion of the strong PTTD group suggests some degree of hindfoot eversion is not associated with weakness in subjects with PTTD. Previous studies have documented greater flatfoot deformity in the uninvolved foot of subjects with PTTD, suggesting a predisposition for flatfoot deformity.[8,20] Also,

recent studies postulate that flatfoot deformity may precede tendinopathy.[2,33] These hypotheses are speculation based on the cross sectional data from this study and require further study to confirm. The presence of weakness in ankle inversion and forefoot adduction can serve as a clinical sign of advanced dysfunction that is associated with worse flatfoot deformity and should be clinically managed.

Weakness is associated with greater forefoot abduction with the greatest effect (largest difference between groups) at the end of stance when high loads are on the forefoot. This is consistent with other studies documenting the increased role of the tibialis posterior at the end of stance.[14,22] Weakness is not the only factor associated with forefoot abduction with subjects in the PTTD strong group also demonstrating greater forefoot abduction compared to controls. Control subjects returned to a neutral forefoot abduction posture at the end of stance while PTTD subjects who were strong remained almost four degrees abducted and PTTD subjects who were weak remained over seven degrees abducted (Figure 3). This failure to return to a neutral forefoot posture is consistent with data suggesting excessive medial loading observed when simulating either a flatfoot deformity or tibialis posterior weakness.[14] Excessive forefoot abduction observed in this study of subjects with PTTD who are weak or strong suggest clinical interventions should target forefoot correction at the end of stance when body weight is over the forefoot. This may be most important in subjects who demonstrate clinical weakness of the deep posterior compartment.

The role of the tibialis posterior muscle to control the MLA angle is evident in the dynamic movement of the MLA across the stance phase. Significant differences between all three groups, at three of the four stance phases were observed. The control group maintained an arch angle near neutral across all phases of stance with an excursion of approximately nine degrees from peak arch lowering to peak arch rising at the end of stance (Figure 4). The PTTD groups demonstrated greater MLA angles indicating a flatfoot posture consistent with previous studies.[21,31] The PTTD weak group exhibited the greatest MLA angle across all phases of stance and a more limited excursion with approximately 4 degrees from peak arch lowering to raising (Figure 4). Weakness may be associated with failure to dynamically control the MLA and a limited excursion. A lower MLA that remains low throughout stance underscores the importance of supporting or correcting foot kinematics with surgery or orthotics. The spring ligament may also provide support for the MLA[15] and data indicates a higher prevalence of spring ligament damage in subjects with Stage II PTTD.[6] Subjects with Stage II PTTD who are strong may demonstrate a lower MLA due to the loss of spring ligament support.

Other factors may contribute to the strength measures used in the current study. The quantification of force output requires both the neural drive and force capacity of the muscle and tendon unit. This study did not seek to differentiate these factors although the use of direct measures of muscle activation in the deep posterior compartment muscles using EMG could be considered for future work. Historically, PTTD is thought to be a disorder predominantly seen in females with samples in most studies greater than 80% female.[21,26,31] The current sample included 19 females (~63% of sample) and 11 males. Interestingly, the weak group consisted of seven males and nine females. This imbalance may raise the question that perhaps males, although less likely to have PTTD, may be at greater risk for developing weakness or severity of the disorder. This study defined a control group based on normal arch height while the PTTD strong group had no strength deficit but evidence of flatfoot deformity. A combination of muscle strength and ligament integrity may be necessary for normal arch height and comparisons in future studies to an asymptomatic flatfoot control group may isolate the effect of strength in a PTTD weak group.

Deep posterior compartment strength is associated with greater flatfoot deformity in subjects with Stage II PTTD. Flatfoot deformity in subjects with PTTD who are strong suggests that factors other than strength may also play a role in flatfoot deformity. A pre-existing flatfoot posture may be present without a strength deficit due to congenital flatfoot posture or spring ligament damage. The use of strengthening programs may only partially correct flatfoot kinematics in subjects with Stage II PTTD since other factors also contribute to foot posture during walking.

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