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Foot Kinematics During a Bilateral Heel Rise Test in Participants With Stage II Posterior Tibial Tendon Dysfunction

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Abstract

STUDY DESIGN—Experimental laboratory study using a cross-sectional design.

OBJECTIVES—To compare foot kinematics, using 3-dimensional tracking methods, during a bilateral heel rise between participants with posterior tibial tendon dysfunction (PTTD) and participants with a normal medial longitudinal arch (MLA).

BACKGROUND—The bilateral heel rise test is commonly used to assess patients with PTTD; however, information about foot kinematics during the test is lacking.

METHODS—Forty-five individuals volunteered to participate, including 30 patients diagnosed with unilateral stage II PTTD (mean $\pm$ SD age, 59.8 ± 11.1 years; body mass index, 29.9 ± 4.8 kg/m²) and 15 controls (mean $\pm$ SD age, 56.5 ± 7.7 years; body mass index, 30.6 ± 3.6 kg/m²). Foot kinematic data were collected during a bilateral heel rise task from the calcaneus (hindfoot), first metatarsal, and hallux, using an Optotrak motion analysis system and Motion Monitor software. A 2-way mixed-effects analysis of variance model, with normalized heel height as a covariate, was used to test for significant differences between the normal MLA and PTTD groups.

RESULTS—The patients in the PTTD group exhibited significantly greater ankle plantar flexion (mean difference between groups, 7.3° ; 95% confidence interval [CI]: 5.1° to 9.5°), greater first metatarsal dorsiflexion (mean difference between groups, 9.0° ; 95% CI: 3.7° to 14.4°), and less hallux dorsiflexion (mean difference, 6.7° ; 95% CI: 1.7° to 11.8°) compared to controls. At peak heel rise, hindfoot inversion was similar ($P = .130$) between the PTTD and control groups.

CONCLUSION—Except for hindfoot eversion/inversion, the differences in foot kinematics in participants with stage II PTTD, when compared to the control group, mainly occur as an offset, not an alteration in shape, of the kinematic patterns.

Keywords

ankle; flat foot; medial longitudinal arch; posterior tibialis

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The University of Rochester Research Subjects Review Board and The Ithaca College All College Review Board for Human Subjects Research approved the protocol for this study.

The heel rise test is used to assess foot and ankle muscle function for individuals with a wide spectrum of conditions, but, in particular, foot conditions.^{4,9} Specifically, the heel rise test is recommended for individuals with posterior tibial tendon dysfunction (PTTD).^{30,34} Weakness of the posterior tibialis muscle is thought to contribute to the inability to perform a heel rise task or abnormal kinematics during a heel rise task.⁴¹ The normal combined action of the posterior tibialis and triceps surae muscles is thought to produce ankle plantar flexion with inversion during a heel rise task.^{15,23,33} Clinically, an abnormal heel rise test is observed when the individual cannot perform a heel rise or performs the heel rise with hindfoot eversion (fails to invert on rising), suggesting that the posterior tibialis muscle no longer is acting to invert the hindfoot.^{22,29} Although there are anecdotal descriptions of abnormal kinematics of the foot in individuals with PTTD,^{22,29} there are no quantitative studies that examine foot kinematics during a heel rise task.

A variety of in vivo foot kinematic methods have been proposed to measure abnormal movements in individuals with foot problems.^{18,27,31,37–39} For example, some have proposed measuring 3 of 5 metatarsals of the forefoot separately,²⁷ while others treat all the metatarsals as a single forefoot segment.^{31,38} In vivo studies of skin-mounted and bone-mounted markers define skin artifact errors and influence this segmentation. Errors in tracking the movement of the calcaneus, representing the hindfoot, are low (average across a gait cycle, $\pm 2.6^\circ$),³² supporting the widespread use of the hindfoot relative to the tibia to measure hindfoot inversion/eversion and ankle plantar flexion/dorsiflexion.^{18,27,31,37–39} Measurements of forefoot motions vary considerably, depending on segmentation.^{18,27,31,37–39} Measurements of the first metatarsal have the advantage of being sensitive to changes in the height of the medial longitudinal arch (MLA).^{17,39} Further, an in vitro study estimated low (less than $\pm 2.3^\circ$) bone-tracking errors specific to the first metatarsal.⁴⁰ Tracking first metatarsal relative to the hindfoot segment, while not providing specific information about joint movement (eg, talonavicular or calcaneocuboid joints), does identify individuals with a pronated foot posture¹⁷ and PTTD.³⁹ Further, in vitro studies have demonstrated a strong relationship ($r^2 = 0.85$) between foot kinematic measurements and posterior tibialis tendon length using the hindfoot and first metatarsal.¹¹ Given the large changes in foot posture documented in previous studies of walking,^{31,39} the changes in foot movements associated with PTTD are expected to be much larger than measurement errors.

Studies of walking trials suggest that the current clinical focus on hindfoot kinematics during the heel rise test may be less significant than first metatarsal kinematics. Ness et al³¹ and Tome et al³⁹ noted significantly greater hindfoot eversion, first metatarsal dorsiflexion, and first metatarsal abduction during walking. The mechanisms that are thought to contribute to this offset in foot kinematics during walking include (1) failure to invert the subtalar joint,² (2) decreased ligamentous support (eg, spring and deltoid ligaments),^{1,8,12} and (3) decreased muscular control.⁴¹ Because in vivo foot kinematic methods are not specific to a joint (eg, talonavicular or calcaneocuboid joints), the specific mechanisms at play are not revealed by these methods. Nevertheless, the differences between controls and individuals with PTTD were reflective of a shift in foot kinematics toward a more pronated foot (greater hindfoot eversion, first metatarsal dorsiflexion, and first metatarsal abduction), but not a change in the shape of the kinematic pattern. For example, for the individuals in the control group, foot kinematic patterns for the MLA showed a lowering of the arch during the first 70% of the stance phase of walking, followed by rising after 70%.^{19,31,39} This kinematic pattern was the same for the PTTD group. However, for the individuals with PTTD, this foot kinematic pattern occurred relative to a lower MLA position.^{31,39} In contrast to the ligament and muscle changes noted above, other passive supporting structures, such as the plantar fascia, are typically less affected in individuals with PTTD.^{1,8,12} Preservation of the plantar fascia in a pronated foot may be important for the transfer of Achilles tendon force to the metatarsal heads^{10,14} and raising the MLA during late stance

through the windlass mechanism.¹⁹ The lack of involvement of the plantar fascia in many patients with PTTD may explain why rising of the MLA during late stance was preserved even in participants with severely abnormal foot posture.^{18,31,39} During a heel rise task, the foot kinematics associated with PTTD may take advantage of similar mechanisms.

Theoretically, the inability to perform a heel rise test may occur from failure to stabilize the midfoot, resulting in a flexible foot or lever during the test. Less hindfoot inversion may lead to less bony stability in the midfoot,² contributing to dorsiflexion of the first metatarsal relative to the hindfoot. Posterior tibialis muscle weakness may lead to decreased hindfoot inversion and the inability to heel rise, or hindfoot eversion during the heel rise.^{16,41} This has led to the current clinical recommendations to focus on the presence of hindfoot eversion at peak heel rise as a sign of advancing PTTD.^{22,29} However, abnormal MLA lowering reflected by first metatarsal dorsiflexion may be equally important as a clinical sign. Currently, no defined variables from the forefoot have been identified for use clinically when evaluating a heel rise task. A current clinical guideline²⁶ suggests that both unilateral and bilateral heel rise tests be used in individuals with PTTD; however, the interpretation focuses on the presence of hindfoot eversion not midfoot stability. A description of in vivo foot kinematics provides a basis for understanding foot movement during a heel rise test and the potential for development of more defined clinical criteria for both the forefoot and hindfoot.

The purpose of this study was to compare foot kinematics of the hindfoot, first metatarsal, and hallux in patients with stage II PTTD (PTTD group), compared to individuals with a normal MLA height (control group). The primary hypothesis was that participants with stage II PTTD would demonstrate greater hindfoot plantar flexion, hindfoot eversion, and first metatarsal dorsiflexion, compared to those in the control group, during the heel rise task. Hallux dorsiflexion was expected to be lower in participants with stage II PTTD compared to those in the control group. The secondary hypothesis was that the range of movement (ROM) from standing to peak heel rise, measured using 3-dimensional motion analysis techniques, would not differ between patients with stage II PTTD and those in the control group. Previous studies noted that the kinematic patterns over the stance phase of walking for individuals with PTTD were essentially similar to those in control subjects (eg, normal arch rising/lowering, normal hindfoot eversion/inversion), but offset toward a pronated foot posture. Therefore, the ROM, during the heel rise task, was expected to be similar for each kinematic variable between the participants with PTTD and controls.

METHODS

Participants

A TOTAL OF 30 PARTICIPANTS WITH stage II PTTD and 15 controls volunteered for this study. Participants with unilateral PTTD were referred by an orthopaedic surgeon and were clinically classified as stage II. The stage II PTTD classification required participants to have 1 or more signs related to PTTD, including (1) tenderness to palpation of the posterior tibial tendon, (2) swelling of the posterior tibial tendon sheath, and (3) pain during single-limb heel rise. In addition, the individuals with PTTD were required to have 1 or more signs of flexible flatfoot deformity, including excessive nonfixed hindfoot valgus deformity during weight bearing and/or excessive first metatarsal abduction. Excessive hindfoot valgus and first metatarsal abduction were based on visual comparisons between the involved and uninvolved side. Because the inclusion criteria relied on side-to-side comparisons, all participants in the PTTD group were required to have unilateral involvement. Participants were excluded if their foot pain prevented them from ambulating greater than 15 m.

The control participants were asymptomatic and required to fall into the range of age, gender, and body mass index (BMI) of the first 15 participants of the PTTD group (TABLE 1). Control participants were admitted if they had no history of foot and ankle problems and an arch height index comparable to a previous study of healthy participants.^{5,43} The arch height index is described as the ratio of dorsum height at 50% of foot length, divided by the foot length from the heel to the base of the first metatarsal head.^{5,43} Greater values indicate a higher MLA. A normal arch height index was defined as equal to or greater than an average ($\pm$ SD) value reported in a previous study (0.34 ± 0.03).⁵ Because this study included participants both with and without flat-foot, values equal to or above the average are theorized to be more representative of how clinicians define a normal MLA. All participants were informed of the experimental procedures and signed a consent form approved by The University of Rochester and Ithaca College Institutional Review Boards.

Kinematic Measurements

A 5-segment foot kinematic model, that included the tibia, calcaneus (hindfoot), first metatarsal, second to fourth meta-tarsals, and hallux, was used to measure foot movement.¹⁷ The kinematics of the second to fourth metatarsal segment were measured but not utilized in this study. To track movement, sets of 3 infrared-emitting diodes (IREDs) were mounted on rigid thermoplastic platforms. The platforms were then attached using double-sided adhesive tape to the skin over each bony segment of interest (FIGURE 1). Six infrared cameras (Optotrak Motion Analysis System; NDI, Waterloo, Ontario, Canada), synchronized with force plate (model 9286; Kistler Group, Winterthur, Switzerland), were used to collect kinematics (60 Hz) and force (1000 Hz) data with the Motion Monitor, Version 7.24 software (Innsport Training Inc, Chicago, IL). Anatomically based coordinate systems were established for each segment using digitized bony landmarks consistent with a previous study.¹⁷ The resulting segment *x*-axes for the first metatarsal and hallux were aligned with the shaft of the first metatarsal and hallux, respectively. The vertical axes were the perpendicular to a plane formed by the *x*-axis and an arbitrary point at the same height as the distal first metatarsal and hallux digitized points. This has the effect of aligning the first metatarsal and hallux coordinates systems with the sagittal-plane orientation of the first metatarsal (ie, declination angle) and hallux. The tibia vertical axis was aligned with a vector from the lateral malleolus to the fibula head. The tibia anterior/posterior axis was perpendicular to a plane formed by the *y*-axis and medial malleolus. The hindfoot segment *x*-axis was aligned with a line from the posterior heel to the tip of the second metatarsal. The *y*- and *z*-axes for the hindfoot were aligned with the laboratory reference frame. The posterior heel digitized point, tracked relative to the hindfoot, was used to estimate heel height. Kinematic data were smoothed using a fourth-order, zero-phase-lag Butterworth filter with a cut-off frequency of 6 Hz. To calculate relative joint angles, a Cardan angle *z*-*x*-*y* sequence of rotations was used as suggested by Cole et al.⁶ The joint angles calculated included the hindfoot with respect to the tibia (hindfoot inversion/eversion and ankle plantar flexion/dorsiflexion), first metatarsal with respect to the hindfoot (first metatarsal plantar flexion/dorsiflexion), and hallux with respect to the first metatarsal (hallux plantar flexion/dorsiflexion). The errors associated with this approach are expected to be similar to studies comparing bone- and skin-mounted markers, which were reported as $\pm 2.3^\circ$ for the first metatarsal⁴⁰ and $\pm 2.6^\circ$ for the calcaneus.³²

Procedures

As shown in previous studies, the definition of neutral or zero position of the foot joints strongly affects the measurement of foot kinematics.^{17,35,36} Surprisingly, determining the subtalar neutral (STN) position has repeatedly shown adequate between- and within-session reliability.^{17,35,36} Consistent with these studies, the STN position was adopted to standardize the neutral alignment of the foot.^{17,39} In brief, from a relaxed standing posture

participants were asked to move their hindfoot into eversion and inversion, resulting in the raising and lowering of their arch. The examiner manually palpated the talonavicular joint until the mid position was judged to have been achieved. The participant was asked to hold this position, while a 1-second trial was collected. This procedure was repeated 2 times. The mean of 2 STN trials was then used as the zero joint position for all foot kinematic angles. Because the goal is to describe foot kinematic angles from a common zero or reference foot posture, both the hindfoot and forefoot are expected to be in a similar position when in STN. However, when participants with PTTD were placed into STN, the first metatarsal head intermittently lifted off the ground, which clinically reflects forefoot varus. To assure that the zero position is not influenced by this foot posture (ie, is similar between subjects), an adjustment to the first metatarsal position was calculated in all subjects. The adjustment calculated the angle of the first metatarsal segment as if the head were on the floor. The details of this method are published in a previous study.¹⁷ The goal of achieving a common first metatarsal position when in STN is supported by an equal average first metatarsal angle (declination angle), in the laboratory coordinate system, between participants in the control group (mean $\pm$ SD, $-25.1^\circ \pm 3.3^\circ$) and the PTTD group (mean $\pm$ SD, $-25.1^\circ \pm 5.6^\circ$) in this study. Between-session reliability collected on 6 participants using the described foot kinematic methods and procedures resulted in a standard error of the measurement of less than 2.0° for all variables, similar to published studies.^{17,35,36}

Once the STN position was established, participants completed the heel rise task. At a comfortable pace, participants were instructed to rise up on their heels and return to the starting position repeatedly. During each repetition, participants were encouraged to rise up as high as they could. Participants stopped once they completed at least 5 heel rises over a 5- to 15-second interval (range, 5–8 repetitions) to minimize discomfort and achieve peak performance (peak heel height). Finally, participants were allowed fingertip-to-fingertip contact with an examiner, to assist with balance during the heel rise task. Only bilateral heel rise tasks were performed, because many participants with PTTD are unable to perform a single heel rise task.

Because of the lack of controls on the heel rise task, vertical ground reaction force was assessed in a subset of participants, to understand the influence of “fingertip” support and compensations with the opposite limb. To assess if load from side to side was equivalent, the vertical ground reaction force (normalized to subject body mass) was assessed bilaterally in a subset of 16 participants with PTTD and all those in the control group. The summed right and left vertical ground reaction force was at least (lowest values) 89% and 83% of body weight at peak heel rise for the control and PTTD groups, respectively. The average near body weight values were 98% and 96% for the control and PTTD groups, respectively, which suggests that the effect of the “fingertip” support during the heel rise was minimal. The participants with PTTD applied from 28% to 50% (average $\pm$ SD, $40\% \pm 8\%$) of their body weight on the involved side, while those in the control group applied 37% to 58% (average $\pm$ SD, $49\% \pm 5\%$) of body weight on the measured side. Because of an unexpected prevalence for the involved side to be the left in the individuals with PTTD, the left side was tested as the involved side in 10 and the right side in 5 individuals in the control group. Data suggest subtle compensation with the uninvolved side (average, 9% of body weight) in the PTTD group.

Heel height was measured in the PTTD and control participants to determine if overall performance was similar between groups. Heel height was expected to show a dependence on truncated foot length (distance from back of heel to first metatarsal head). Therefore, a priori normalized heel height was anticipated as a covariate to adjust for differences in foot kinematics attributable to foot length.

Data Analysis

The ankle plantar flexion/dorsiflexion data were used to identify the start and end of a heel rise cycle (FIGURE 2). Each heel rise cycle was interpolated to 100% cycle at 1% intervals (101 points). For each subject the 3 trials with the highest peak ankle plantar flexion angle were selected and averaged to gain a representative pattern for each subject. Time normalization to percent cycle allowed for participants with diverse speeds, and hence, heel rise cycle time to be averaged. The heel rise cycle was subsequently divided into 2 phases, the preparation phase and heel rise phase (FIGURE 2). The preparation phase on average ($\pm$ SD) ended at $32\% \pm 6\%$ of the heel rise cycle, determined by visually assessing each pattern. To assess the primary hypothesis, foot kinematic angles at the mid point of the preparation phase (specific to each subject) and point of peak ankle plantar flexion were analyzed (FIGURE 2). To address the secondary hypothesis, the ROM for each foot kinematic angle was the difference between the foot kinematic angle at the mid point of the preparation phase and angle at peak ankle plantar flexion.

The first purpose of this investigation was to compare 4-foot kinematic variables, including hindfoot eversion/inversion, ankle plantar flexion/dorsiflexion, first metatarsal plantar flexion/dorsiflexion, and hallux plantar flexion/dorsiflexion angles between participants with PTTD and controls. A 2-way mixed-model analysis of variance (ANOVA) was used to individually assess each foot kinematic variable across groups (PTTD and control) and cycle points (preparation and peak heel rise). Group was treated as a fixed variable and cycle points as a repeated measure. Data were first analyzed for an interaction effect to determine differences between groups specific to either the preparation or the peak heel rise cycle point. The presence of an interaction would signal a change in shape of the kinematic pattern for that variable. In the absence of interaction, main effect for groups was analyzed to determine an offset in angular positions between the 2 groups. The presence of a main effect would indicate an offset in angular values but similarity of the kinematic patterns between groups. To assess ROM across foot kinematic variables, a 2-way ANOVA was performed, using group (PTTD and control) by kinematic variable (hindfoot eversion/inversion, ankle plantar flexion/dorsiflexion, first metatarsal plantar flexion/dorsiflexion, and hallux plantar flexion/dorsiflexion). Similarly, group was a fixed factor and foot kinematic variable was a repeated factor. For each analysis, heel height normalized to truncated foot length was used as a covariate to control for differences in heel rise performance. Significant Pearson correlation coefficients (r values ranged between 0.56 to 0.67, for all variables $P < .05$) between normalized heel height and sagittal-plane kinematic variables supported using normalized heel height as a covariate. Pairwise comparisons between groups were pursued in the case of a significant interaction for all of the ANOVA models. An alpha level of .05 was used for all statistical tests.

RESULTS

Sagittal-Plane Angles

THE SAGITTAL-PLANE FOOT KINEMATICS were significantly different between the PTTD and control groups across the heel rise cycle points (main effect). Foot kinematic angles at specific cycle points are provided in TABLE 2, and the overall kinematic patterns are provided in FIGURE 3. When using normalized heel height as a covariate, the average difference between groups, across cycle points, differed from the data shown in TABLE 2 and FIGURE 2. The data adjusted for normalized heel height showed that the PTTD group used greater ankle plantar flexion (adjusted mean difference between groups, 7.3° ; 95% confidence interval [CI]: 5.1° to 9.5°), greater first metatarsal dorsiflexion (adjusted mean difference between groups, 9.0° ; 95% CI: 3.7° to 14.4°), and lower hallux dorsiflexion (adjusted mean difference between groups, 6.7° ; 95% CI, 1.7° to 11.8°). A post hoc analysis revealed that 15 of 30

participants (50%) with PTTD failed to achieve first metatarsal plantar flexion at peak heel rise, while all of the controls (100%) achieved some amount of first metatarsal plantar flexion at peak heel rise. A Fisher exact test confirmed that the proportions of participants that failed to achieve first metatarsal plantar flexion in the PTTD group was statistically higher than for the control group ($P < .001$).

Hindfoot Inversion/Eversion

The hindfoot inversion/eversion kinematics depended on both the group and cycle point of the heel rise task (significant interaction effect). Hindfoot inversion/eversion kinematic angles at specific cycle points are provided in TABLE 2, and the overall kinematic patterns are provided in FIGURE 4. The data analyzed with normalized heel height as a covariate showed that the hindfoot inversion at the preparatory phase of heel rise for the PTTD group was significantly greater compared to the control group ($P < .001$). The data adjusted for normalized heel height showed a mean difference between groups for the preparatory phase of 7.1° (95% CI: 5.3° to 8.9°) greater hindfoot eversion. However, at the peak heel rise point, the amounts of hindfoot inversion were similar ($P = .130$) for the PTTD and control groups (mean difference between groups adjusted for normalized heel height, 1.6° ; 95% CI: -1.1° to 4.3°). A post hoc analysis revealed that 7 of 30 (23%) participants with PTTD failed to invert, while all but 1 of the controls (6.7%) achieved some amount of hindfoot inversion. A Fisher exact test suggested that the proportions of participants who failed to achieve hindfoot inversion in both groups were not significantly different ($P = .243$).

Range of Motion

The range-of-motion (ROM) values across cycle points were only significantly different for hindfoot inversion/eversion (FIGURE 5). Using normalized heel height as a covariate, there was a significant interaction between group and ROM ($P = .033$). Pairwise comparisons revealed that hindfoot inversion ROM in the PTTD group was significantly greater than that in the control group ($P = .001$). The mean difference between groups adjusted for normalized heel height was 5.2° (95% CI: 2.3° to 8.1°). All other ROM variables were similar between groups (FIGURE 4).

DISCUSSION

THE NEW FINDINGS OF THIS STUDY suggest that abnormal kinematics in individuals with stage II PTTD occur in both the hindfoot and forefoot during a heel rise task. Previous studies focusing on walking revealed that forefoot abnormalities were common in individuals with PTTD.^{18,31,39} This study confirms that similar abnormal kinematics occur during a heel rise task. Except for hindfoot inversion/eversion, differences in kinematics were primarily the result of offsets toward a flatfoot posture (greater ankle plantar flexion, first metatarsal dorsiflexion, and hallux plantar flexion in the participants with PTTD), not differences in the shape of the foot kinematic patterns (FIGURE 6). This offset in pattern is consistent with the current view that static foot posture may predict dynamic foot movement.³¹ The exception was hindfoot inversion, which depended on the heel rise cycle point. Although participants with PTTD started (preparation phase) in more hindfoot eversion than controls, they achieved a similar hindfoot inverted position as compared to controls at peak plantar flexion (FIGURE 5). This finding contrasts with current clinical observations that hindfoot eversion at peak plantar flexion during a heel rise is a typical sign of PTTD.^{22,29} The focus on first metatarsal movement may expand the interpretation of the heel rise test in patients with PTTD.

The heel height achieved in this study is reflective of heel rise performance across groups, which is similar (TABLE 2). The low ankle plantar flexion angles reported from tracking the

calcaneus as the hindfoot segment are different than clinical definitions. Clinical definitions of ankle plantar flexion use a single foot segment, including the metatarsals, not just the calcaneus. An estimate of the clinical assessment of ankle plantar flexion can be derived from the truncated foot length (heel to first metatarsal head) and heel height achieved. For example, ankle plantar flexion angle is approximately 31° (controls: $30.9^\circ = \tan^{-1}[0.11/0.184]$; PTTD: $31.2^\circ = \tan^{-1}[0.12/0.198]$), when taking truncated foot length and heel height into account. However, the covariate analysis was used to adjust for differences in performance (normalized heel height) within the PTTD group, which had the effect of reducing the differences in foot kinematics across groups. However, even when taking into account differences in normalized heel height, the participants with PTTD used different ankle and foot kinematics during the heel rise task.

In this study, ankle plantar flexion was an offset toward greater plantar flexion in participants with PTTD. Authors argue that the triceps surae muscle is shortened in participants with hindfoot plantar flexion due to greater calcaneal plantar flexion observed on radiographs.⁹ The data from this study confirm that participants with PTTD use greater ankle plantar flexion relative to the STN position than participants with a normal MLA ($\sim 7^\circ$). This shift toward higher ankle plantar flexion may shorten the triceps surae, resulting in a shift in the force-length relationship and functionally weaker muscle.⁹ Despite the possibility of functionally weaker plantar flexors, the participants with PTTD on average achieved similar normalized heel heights as controls. Increasing the load on the plantar flexors, as is done during a single-limb heel rise, may further reduce heel rise performance (normalized heel height). Independent of ankle plantar flexor weakness, ineffective boney contact and/or muscle control to stabilize the midfoot (measured in this study as first metatarsal dorsiflexion) may contribute to reduced heel rise ability.²

Separation of the foot into the hindfoot and first metatarsal segments provides insight into how the increase in height of the MLA contributed to the clinical definition of ankle plantar flexion of approximately 31° . The PTTD group used equal ankle plantar flexion ROM and first metatarsal plantar flexion ROM as controls to achieve a similar heel height (FIGURE 5). Although the ROM was the same between groups, first metatarsal plantar flexion occurred relative to a lower MLA position in participants with PTTD compared to the controls (FIGURE 3). Given the foot kinematic methods, it is difficult to precisely identify at which joint the first metatarsal plantar flexion ROM took place. However, for participants with PTTD, higher plantar flexion of the talonavicular, naviculocuneiform, or first metatarsal cuneiform joints is a possibility.³ The fact that the first metatarsal plantar flexion ROM occurs when the foot is increasing weight-bearing forces suggests increasing MLA height during high load. This finding is consistent with studies using a kinetic model of the foot that suggested a power generation of the medial foot at push-off during walking.²⁷ Periods of power generation suggest that active mechanisms are responsible.²⁷ The first metatarsal plantar flexion ROM, therefore, may be the result of (1) muscle control of the intrinsic muscles,²⁴ (2) flexor hallucis longus muscle,^{20,21} (3) Achilles tendon forces acting through the plantar fascia,¹⁰ and/or (4) some combination of these actions, which are retained in individuals with PTTD.

The potential influence of the plantar fascia on raising the MLA is suggested by hallux dorsiflexion. The participants with PTTD in this study used approximately 7° less hallux dorsiflexion compared to the control participants. However, the total hallux ROM was similar across groups (FIGURE 5). Previous studies have suggested that, as hallux dorsiflexion increases, so does the MLA.¹⁹ Magnetic resonance imaging studies demonstrate infrequent involvement of the plantar fascia in participants with PTTD in contrast to consistent abnormal signal (as an indication of soft tissue damage) found in the spring and deltoid ligaments.^{1,8,12} Therefore, rising of the MLA, even in participants that have a low

MLA, may be an indication of an intact windlass mechanism via the plantar fascia. Because only the movement of the hallux was collected in this study, the influence of the lateral 4 digits on the plantar fascia was not taken into account. It is possible that hallux movement alone does not fully account for plantar fascia behavior via the windlass mechanism. Unexpectedly, in this study, despite achieving comparable hindfoot inversion, first metatarsal dorsiflexion was markedly higher in participants with PTTD compared to controls. Alterations in joint axes of rotation, as noted for the first metatarsal by Glasoe et al,¹³ may be at play in altering the normal hindfoot-forefoot coupling demonstrated by Blackwood et al² in vitro. Despite equivalent first metatarsal plantar flexion ROM across groups, the PTTD group was influenced by approximately 50% of the participants who failed to plantar flex their first metatarsal beyond the STN position (FIGURE 6). In contrast, all the individuals in the control group achieved some amount of first metatarsal plantar flexion beyond the STN position. This offset toward greater first metatarsal dorsiflexion relative to the STN position may indicate altered joint axes and soft tissue support.^{18,31,39} Further research investigating mechanisms other than hindfoot inversion on first metatarsal kinematics seem warranted.

Clinical Implications

Clinically, this study demonstrates that documenting the ability to heel rise and noting the existence of hindfoot eversion at peak heel rise ignores the more commonly occurring abnormal dorsiflexion of the first metatarsal. Many participants with PTTD started in a hindfoot everted position, yet achieved hindfoot inversion at peak heel rise. This may indicate a functioning posterior tibialis muscle at 50% body weight for the bilateral heel rise. Although not common, failure to invert the hindfoot may indicate a more severe weakness.^{22,29} A single-limb heel rise test may show greater differences in foot kinematics because the load is increased from 40% to 50% body weight to close to 100%. If first metatarsal plantar flexion occurs during the test, this supports the idea that mechanisms within the foot are functioning despite starting from a flatfoot posture. Failure of the first metatarsal to achieve plantar flexion may indicate failure of coupling between the hindfoot and forefoot and/or loss of muscle control. Clinical methods that do not rely on movement analysis techniques would be beneficial to grade hindfoot and forefoot control during a heel rise test and warrant future research.

Because the alterations in foot movement are primarily offsets as opposed to alterations in kinematic patterns, foot posture may predict abnormal MLA kinematics. With the exception of hind-foot inversion/eversion, the abnormal kinematic patterns associated with the participants with PTTD were offsets, without changes in the shape of the foot kinematic patterns. A previous study of participants with a variety of foot postures, but none associated with PTTD, found that the longitudinal arch angle measured in standing was similar to the same angle measured during walking ($r = 0.97$).²⁸ Given this, measures of foot posture may reflect the offsets that are observed during movement if similar relationships hold in individuals with PTTD. Recent studies appear to support such an association.^{18,31,38,39} For example, a study that failed to use a correction or offset to align foot segments in participants with PTTD suggested similar foot kinematic angles compared to controls.³⁸ In contrast, studies that used radiographs^{18,31} or the STN position³⁹ to align foot segments documented offsets in foot kinematics, not alterations in the shape of foot kinematic patterns. Therefore, the offsets, measured statically, appear responsible for the measured alterations in foot kinematic patterns. Considered together, these studies suggest that clinical measurements of foot posture (eg, with radiographs or standardized measures) may predict foot kinematics during movement in individuals with PTTD, similar to a previous study of people free of foot pathologies.²⁸

Limitations

The limitations of this study are associated with the foot kinematic methods, study design, and sample. Foot kinematic methods differ in their approach to segmenting the foot and defining foot kinematic angles which make comparisons across studies difficult.^{7,25,27,31,37,44} The size of the bones and soft tissue over the mid foot (eg, tendons) make measuring motion at these joints difficult to do using skin-mounted markers. Some studies comparing bone-mounted and skin-mounted markers suggest low average errors (less than $\pm 3^\circ$) for the hindfoot and first metatarsal.^{32,40,42} The foot kinematic data reported in this study should be viewed with respect to these errors. Because it is not possible to blind study participants to the PTTD condition, there is the potential for bias in establishing STN positions across groups. The similarity of first metatarsal angles across groups (approximately 25°) suggests that equivalence in foot position was achieved; however, without blinding, bias is not completely eliminated. The foot loading was lower (approximately 9% body weight) in the subjects with PTTD, which might have masked some differences in foot kinematics. This is a cross-sectional study, which does not allow establishing of cause-and-effect relationships. Therefore, inferences made in the discussion related to cause-and-effect relationships are speculative. This sample is large for studies of participants with PTTD.^{31,38,39} Nevertheless, larger samples are desirable. The designation of stage II PTTD was dependent on clinical evaluation methods that have not been assessed in terms of validity. Future studies may consider the reliability and validity of staging PTTD. Finally, the definition of a normal MLA used for the control group was based on a sample of 100 subjects.⁵ Alternative definitions of normal MLA may change the comparisons between groups.

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CONCLUSION

INDIVIDUALS WITH PTTD ARE MORE likely to display abnormal first metatarsal dorsiflexion as compared to abnormal hindfoot eversion at peak plantar flexion during a bilateral heel rise test. Except for hindfoot eversion/inversion, the foot kinematic differences between the participants with stage II PTTD and those in the control group mainly occurred as offsets. The amount of motion that took place between the preparation phase of heel rise and peak heel rise was similar between groups, suggesting that MLA raising and lowering occurred

similarly in both groups, though over a different range of joint movement. The one exception was for hindfoot eversion/inversion. Despite the hindfoot achieving an inverted position at peak heel rise, the first metatarsal relative to the calcaneus was significantly more dorsiflexed, which suggests that first metatarsal movement in individuals with PTTD may be less dependent on hindfoot movement.

KEY POINTS

FINDINGS

Failure of the hindfoot to invert is uncommon during a bilateral heel rise test in individuals with stage II PTTD. Failure to achieve first metatarsal plantar flexion occurred in 50% of subjects with stage II PTTD. Except for hindfoot inversion, foot kinematic ROM is normal in participants with stage II PTTD. Differences in foot movement are attributable to offsets in standing posture, in this case quantified using the STN position.

IMPLICATION

During a bilateral heel rise test, MLA kinematics may be more sensitive than hindfoot kinematics in identifying abnormal foot movement in individuals with stage II PTTD.

CAUTION

The results are specific to the severity (stage II) of PTTD, definition of “normal” MLA, and the foot kinematic methods used.

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REFERENCES

1. Balen PF, Helms CA. Association of posterior tibial tendon injury with spring ligament injury, sinus tarsi abnormality, and plantar fasciitis on MR imaging. *Am J Roentgenol* 2001;176:1137–1143. [PubMed: 11312167]
2. Blackwood CB, Yuen TJ, Sangeorzan BJ, Ledoux WR. The midtarsal joint locking mechanism. *Foot Ankle Int* 2005;26:1074–1080. [PubMed: 16390642]
3. Bluman EM, Title CI, Myerson MS. Posterior tibial tendon rupture: a refined classification system. *Foot Ankle Clin* 2007;12:233–249. v. <http://dx.doi.org/10.1016/j.fcl.2007.03.003>. [PubMed: 17561198]
4. Bowers AL, Castro MD. The mechanics behind the image: foot and ankle pathology associated with gastrocnemius contracture. *Semin Musculoskelet Radiol* 2007;11:83–90. <http://dx.doi.org/10.1055/s-2007-984418>. [PubMed: 17665354]
5. Butler RJ, Hillstrom H, Song J, Richards CJ, Davis IS. Arch height index measurement system: establishment of reliability and normative values. *J Am Podiatr Med Assoc* 2008;98:102–106. [PubMed: 18347117]
6. Cole GK, Nigg BM, Ronsky JL, Yeadon MR. Application of the joint coordinate system to three-dimensional joint attitude and movement representation: a standardization proposal. *J Biomech Eng* 1993;115:344–349. [PubMed: 8309227]
7. Cornwall MW, McPoil TG. Three-dimensional movement of the foot during the stance phase of walking. *J Am Podiatr Med Assoc* 1999;89:56–66. [PubMed: 10063775]

8. Deland JT, de Asla RJ, Sung IH, Ernberg LA, Potter HG. Posterior tibial tendon insufficiency: which ligaments are involved? *Foot Ankle Int* 2005;26:427–435. [PubMed: 15960907]
9. DiGiovanni CW, Langer P. The role of isolated gastrocnemius and combined Achilles contractures in the flatfoot. *Foot Ankle Clin* 2007;12:363–379. viii. <http://dx.doi.org/10.1016/j.fcl.2007.03.005>. [PubMed: 17561207]
10. Erdemir A, Hamel AJ, Fauth AR, Piazza SJ, Sharkey NA. Dynamic loading of the plantar aponeurosis in walking. *J Bone Joint Surg Am* 2004;86-A:546–552. [PubMed: 14996881]
11. Flemister AS, Neville CG, Houck J. The relationship between ankle, hindfoot, and forefoot position and posterior tibial muscle excursion. *Foot Ankle Int* 2007;28:448–455. [PubMed: 17475139]
12. Gazdag AR, Cracchiolo A 3rd. Rupture of the posterior tibial tendon. Evaluation of injury of the spring ligament and clinical assessment of tendon transfer and ligament repair. *J Bone Joint Surg Am* 1997;79:675–681. [PubMed: 9160939]
13. Glasoe W, Pena F, Phadke V, Ludewig PM. Arch height and first metatarsal joint axis orientation as related variables in foot structure and function. *Foot Ankle Int* 2008;29:647–655. <http://dx.doi.org/10.3113/FAI.2008.0647>. [PubMed: 18549767]
14. Hamel AJ, Donahue SW, Sharkey NA. Contributions of active and passive toe flexion to forefoot loading. *Clin Orthop Relat Res* 2001:326–334. [PubMed: 11764366]
15. Hansen ML, Otis JC, Kenneally SM, Deland JT. A closed-loop cadaveric foot and ankle loading model. *J Biomech* 2001;34:551–555. [PubMed: 11266681]
16. Houck JR, Nomides C, Neville CG, Samuel Flemister A. The effect of stage II posterior tibial tendon dysfunction on deep compartment muscle strength: a new strength test. *Foot Ankle Int* 2008;29:895–902. <http://dx.doi.org/10.3113/FAI.2008.0895>. [PubMed: 18778667]
17. Houck JR, Tome JM, Nawoczenski DA. Subtalar neutral position as an offset for a kinematic model of the foot during walking. *Gait Posture* 2008;28:29–37. <http://dx.doi.org/10.1016/j.gaitpost.2007.09.008>. [PubMed: 17988870]
18. Johnson J, Harris G. Pathomechanics of posterior tibial tendon insufficiency. *Foot Ankle Clin* 1997;2:227–239.
19. Kappel-Bargas A, Woolf RD, Cornwall MW, McPoil TG. The windlass mechanism during normal walking and passive first metatarsalphalangeal joint extension. *Clin Biomech (Bristol, Avon)* 1998;13:190–194.
20. Kirane YM, Michelson JD, Sharkey NA. Contribution of the flexor hallucis longus to loading of the first metatarsal and first metatarsophalangeal joint. *Foot Ankle Int* 2008;29:367–377. <http://dx.doi.org/10.3113/FAI.2008.0367>. [PubMed: 18442450]
21. Kirane YM, Michelson JD, Sharkey NA. Evidence of isometric function of the flexor hallucis longus muscle in normal gait. *J Biomech* 2008;41:1919–1928. <http://dx.doi.org/10.1016/j.jbiomech.2008.03.040>. [PubMed: 18538330]
22. Kohls-Gatzoulis J, Angel JC, Singh D, Haddad F, Livingstone J, Berry G. Tibialis posterior dysfunction: a common and treatable cause of adult acquired flatfoot. *BMJ* 2004;329:1328–1333. <http://dx.doi.org/10.1136/bmj.329.7478.1328>. [PubMed: 15576744]
23. Kulig K, Burnfield JM, Requejo SM, Sperry M, Terk M. Selective activation of tibialis posterior: evaluation by magnetic resonance imaging. *Med Sci Sports Exerc* 2004;36:862–867. [PubMed: 15126722]
24. Kura H, Luo ZP, Kitaoka HB, An KN. Quantitative analysis of the intrinsic muscles of the foot. *Anat Rec* 1997;249:143–151. [PubMed: 9294659]
25. Leardini A, Benedetti MG, Berti L, Bettinelli D, Nativio R, Giannini S. Rear-foot, mid-foot and fore-foot motion during the stance phase of gait. *Gait Posture* 2007;25:453–462. <http://dx.doi.org/10.1016/j.gaitpost.2006.05.017>. [PubMed: 16965916]
26. Lee MS, Vanore JV, Thomas JL, et al. Diagnosis and treatment of adult flatfoot. *J Foot Ankle Surg* 2005;44:78–113. <http://dx.doi.org/10.1053/j.jfas.2004.12.001>. [PubMed: 15768358]
27. MacWilliams BA, Cowley M, Nicholson DE. Foot kinematics and kinetics during adolescent gait. *Gait Posture* 2003;17:214–224. [PubMed: 12770635]
28. McPoil TG, Cornwall MW. Use of the longitudinal arch angle to predict dynamic foot posture in walking. *J Am Podiatr Med Assoc* 2005;95:114–120. [PubMed: 15778468]

29. Myerson MS. Adult acquired flatfoot deformity: treatment of dysfunction of the posterior tibial tendon. *Instr Course Lect* 1997;46:393–405. [PubMed: 9143981]
30. Myerson MS, Badekas A, Schon LC. Treatment of stage II posterior tibial tendon deficiency with flexor digitorum longus tendon transfer and calcaneal osteotomy. *Foot Ankle Int* 2004;25:445–450. [PubMed: 15319100]
31. Ness ME, Long J, Marks R, Harris G. Foot and ankle kinematics in patients with posterior tibial tendon dysfunction. *Gait Posture* 2008;27:331–339. <http://dx.doi.org/10.1016/j.gaitpost.2007.04.014>. [PubMed: 17583511]
32. Nester C, Jones RK, Liu A, et al. Foot kinematics during walking measured using bone and surface mounted markers. *J Biomech* 2007;40:3412–3423. <http://dx.doi.org/10.1016/j.jbiomech.2007.05.019>. [PubMed: 17631298]
33. Niki H, Ching RP, Kiser P, Sangeorzan BJ. The effect of posterior tibial tendon dysfunction on hindfoot kinematics. *Foot Ankle Int* 2001;22:292–300. [PubMed: 11354441]
34. Otis JC, Gage T. Function of the posterior tibial tendon muscle. *Foot Ankle Clin* 2001;6:1–14. v. [PubMed: 11385918]
35. Pierrynowski MR, Smith SB. Effect of patient position on the consistency of placing the rearfoot at subtalar neutral. *J Am Podiatr Med Assoc* 1997;87:399–406. [PubMed: 9308306]
36. Pierrynowski MR, Smith SB, Mlynarczyk JH. Proficiency of foot care specialists to place the rearfoot at subtalar neutral. *J Am Podiatr Med Assoc* 1996;86:217–223. [PubMed: 8776157]
37. Rattanaprasert U, Smith R, Sullivan M, Gilleard W. Three-dimensional kinematics of the forefoot, rearfoot, and leg without the function of tibialis posterior in comparison with normals during stance phase of walking. *Clin Biomech (Bristol, Avon)* 1999;14:14–23.
38. Ringleb SI, Kavros SJ, Kotajarvi BR, Hansen DK, Kitaoka HB, Kaufman KR. Changes in gait associated with acute stage II posterior tibial tendon dysfunction. *Gait Posture* 2007;25:555–564. [PubMed: 16876415]
39. Tome J, Nawoczenski DA, Flemister A, Houck J. Comparison of foot kinematics between subjects with posterior tibialis tendon dysfunction and healthy controls. *J Orthop Sports Phys Ther* 2006;36:635–644. [PubMed: 17017268]
40. Umberger BR, Nawoczenski DA, Baumhauer JF. Reliability and validity of first metatarsophalangeal joint orientation measured with an electromagnetic tracking device. *Clin Biomech (Bristol, Avon)* 1999;14:74–76.
41. Wacker J, Calder JD, Engstrom CM, Saxby TS. MR morphometry of posterior tibialis muscle in adult acquired flat foot. *Foot Ankle Int* 2003;24:354–357. [PubMed: 12735380]
42. Westblad P, Hashimoto T, Winson I, Lundberg A, Arndt A. Differences in ankle-joint complex motion during the stance phase of walking as measured by superficial and bone-anchored markers. *Foot Ankle Int* 2002;23:856–863. [PubMed: 12356185]
43. Williams DS, McClay IS. Measurements used to characterize the foot and the medial longitudinal arch: reliability and validity. *Phys Ther* 2000;80:864–871. [PubMed: 10960934]
44. Woodburn J, Nelson KM, Siegel KL, Kepple TM, Gerber LH. Multisegment foot motion during gait: proof of concept in rheumatoid arthritis. *J Rheumatol* 2004;31:1918–1927. [PubMed: 15468354]

FIGURE 1.

Picture of the placement of the infrared light-emitting diodes used to track the tibia, calcaneus, first metatarsal, and hallux.

FIGURE 2.

Representative trial of ankle plantar flexion/dorsiflexion relative to time. The points used to determine the start and end of the heel rise are identified. The preparatory and rising phases were also distinguished based on ankle angle. Data were taken at 15% of the heel rise cycle and at peak ankle plantar flexion. Plantar flexion is negative.

FIGURE 3.

The average ($\pm$ SD, shown for 1 direction) sagittal plane angles for (A) hindfoot dorsiflexion/plantar flexion, (B) first metatarsal dorsiflexion/plantar flexion, and (C) hallux dorsiflexion/plantar flexion are shown across the heel rise cycle for the participants with posterior tibial tendon dysfunction (PTTD) and controls.

FIGURE 4.

The average ($\pm$ SD, shown for 1 direction) frontal-plane angle for hindfoot relative to the tibia is shown across the heel rise cycle for the participants with posterior tibial tendon dysfunction (PTTD) and controls.

FIGURE 5.

The average ($\pm$ SD) range of motion (ROM) values for hindfoot inversion, first metatarsal plantar flexion, ankle plantar flexion, and hallux dorsiflexion are shown for the participants with posterior tibial tendon dysfunction (PTTD) and controls. *Indicates statistical significance.

FIGURE 6.

A schematic sagittal plane representation of the foot illustrates the findings. (A) Control participants used a combination of ankle plantar flexion, first metatarsal plantar flexion, and hallux dorsiflexion to achieve peak heel rise. (B) The participants with posterior tibial tendon dysfunction (PTTD) used greater ankle plantar flexion, a variable amount of first metatarsal plantar flexion, and lower hallux dorsiflexion to achieve peak heel rise.

TABLE 1

Subject Characteristics *

Variable	Control (n = 15)	PTTD (n = 30)	P Value [‡]
Age (y)	56.5 (7.7)	59.8 (11.1)	.316
Height (cm)	164.9 (7.3)	168.3 (9.9)	.194
Mass (kg)	83.0 (10.8)	85.4 (17.5)	.643
Body mass index (kg/m ²)	30.6 (3.6)	29.9 (4.8)	.658
Involved side [†]	R, 5; L, 10	R, 12; L, 18	
Gender	M, 1; F, 14	M, 9; F, 21	
Static foot posture			
Foot length (cm)	24.55 (1.02)	24.95 (4.50)	.648
Truncated foot length (cm)	18.37 (0.92)	19.80 (1.83)	<.01
First metatarsal height (cm)	6.98 (0.41)	6.14 (0.61)	<.01
Arch height index	0.38 (0.03)	0.31 (0.02)	<.01
Heel rise performance			
Heel height (m)	0.11 (0.01)	0.12 (0.02)	.660
Normalized heel height (%)	59.3 (8.2)	54.0 (10.4)	.093

Abbreviation: PTTD, posterior tibialis tendon dysfunction.

* Data are means $\pm$ SD, with the exception of involved side and gender.

[†] Involved side: control group assigned proportional to PTTD group.

[‡] Between-group difference using an independent *t* test.

Table 2

Variables Evaluated During the Heel Rise Task

Variable	Control Group (n = 15)*	PTTD Group (n = 30)*	Main Effect <i>P</i> Value [†]	Interaction <i>P</i> Value [‡]
Ankle plantar flexion (–)/dorsiflexion (+)				
Preparation	8.5 (3.2)	0.2 (3.9)		
Peak heel rise	–22.1 (7.7)	–18.4 (5.6)	<.001	.407
First metatarsal plantar flexion (–)/dorsiflexion (+)				
Preparation	3.6 (2.7)	13.5 (7.7)		
Peak heel rise	–18.2 (5.9)	–4.9 (14.9)	.001	.590
Hallux plantar flexion (–)/dorsiflexion (+)				
Preparation	–3.6 (3.5)	–7.2 (6.7)		
Peak heel rise	40.1 (6.7)	28.3 (14.2)	<.01	.362
Hindfoot eversion (–)/inversion (+)				
Preparation	–2.2 (1.9)	–9.1 (3.1) [§]		
Peak heel rise	5.9 (3.2)	3.8 (4.6)	<.001	.034

Abbreviation: PTTD, posterior tibialis tendon dysfunction.

* Data are mean (SD) degrees.

[†] Main effect difference between groups using normalized heel height as a covariate.

[‡] Based on 2-way (group by portion of heel rise) mixed-model ANOVA.

[§] Significant difference ($P < .05$) between groups at preparation.