2008

The Effect of Stage II Posterior Tibial Tendon Dysfunction on Deep Compartment Muscle Strength: A New Strength Test

Jeff Houck
George Fox University, jhouck@georgefox.edu

Candace Nomides

Christopher Glenn Neville

Adolph Samuel Flemister

Recommended Citation
Published in Foot & Ankle International 29(9): 895-902, 2008 http://fai.sagepub.com/

The Effect of Stage II Posterior Tibial Tendon Dysfunction on Deep Compartment Muscle Strength: A New Strength Test

Jeff R. Houck, PhD, PT; Candace Nomides, MS, DPT; Christopher Glenn Neville, MS, PT; Adolph Samuel Flemister, MD
Rochester, NY

ABSTRACT

Background: **The purpose of this study was to compare isometric subtalar inversion and forefoot adduction strength in subjects with Stage II posterior tibial tendon dysfunction (PTTD) to controls.** ***Materials and Methods:*** **Twenty four subjects with Stage II PTTD and fifteen matched controls volunteered for this study. A force transducer (Model SML-200, Interface, Scottsdale, AZ) was connected with a resistance plate and oscilloscope (TDS 410A, Tektronix, Beaverton, OR) to the foot. Via the oscilloscope, subjects were given feedback on the amount of force produced and muscle activation of the anterior tibialis (AT) muscle. Subjects were instructed to maintain a plantar flexion force while performing a maximal voluntary subtalar inversion and forefoot adduction effort. A two-way ANOVA model with the factors including, side (involved/uninvolved) and group (control/PTTD) was used.** ***Results:*** **The PTTD group on the involved side showed significantly decreased subtalar inversion and foot adduction strength (0.70 ± 0.24 N/Kg) compared to the uninvolved side (0.94 ± 0.24 N/Kg) and controls (involved side $= 0.99 \pm 0.24$ N/Kg, uninvolved side $= 0.97 \pm 0.21$ N/Kg). The average AT activation was between 11% to 17% for both groups, however, considerable variability in subjects with PTTD.** ***Conclusion:*** **These data confirm a subtalar inversion and forefoot adduction strength deficit by 20% to 30% in subjects with Stage II PTTD. Although isolating the PT muscle is difficult, a test specific to subtalar inversion and forefoot adduction demonstrated the weakness in this population.**

Level of Evidence: II

Corresponding Author:
Jeff R. Houck, PhD, PT
Ithaca College - Rochester
Physical Therapy
1100 South Goodman
Rochester, NY 14620
E-mail: jhouck@ithaca.edu

INTRODUCTION

Posterior tibial tendon dysfunction (PTTD) is characterized by insidious onset of swelling and pain along the posterior tibial (PT) tendon that can lead to adult acquired flatfoot deformity.[12] Subjects have progressed to Stage II when signs of tendinopathy are coupled with a flexible flatfoot deformity.[12] Progressive loss of muscle function in Stage II PTTD is suggested by abnormal gait patterns and difficulty with heel rise tasks, with little direct evidence of alterations in the muscle.[4,21,30] Yet, new treatments for subjects with Stage II PTTD are targeting the effects of exercise on muscle/tendon recovery.[4,16] Documenting muscle recovery is beneficial when assessing the effects of these new treatments on the PT muscle.[28] Yet, because all deep compartment muscles (flexor hallicus longus (FHL), flexor digitorum longus (FDL) and PT) work synergistically to contribute to subtalar inversion, it is not always clear which muscle to attribute weakness to. Therefore, the challenge was to design a test that is sensitive to PT muscle weakness to mark the progression and/or recovery during the course of PTTD.

A forefoot adduction strength test may be sensitive to PT weakness due to muscle properties and joint mechanics. A key measure of the ability of a muscle to produce force is physiologic cross sectional area (PCSA).[18] The PCSA takes into account muscle anatomic cross sectional area and the pennation angle of muscle fibers, providing a measure of the muscle fibers in parallel. A review of in-vitro[3,7,25,31] and in-vivo[8] estimates of the PCSA of leg muscles suggests the PCSA of the PT muscle is two to four times that of the FDL and FHL (Table 1). In addition to muscle PCSA, in-vitro studies suggest the moment arm of the PT muscle is significantly larger than the FDL and FHL for subtalar inversion.[6,10,14] Unfortunately, the moment arms of the deep compartment muscles at the talonavicular joint and other forefoot joints are not well understood.[6] Flemister et al. demonstrated that forefoot abduction/adduction movements were associated with moment arm estimates of greater than

Table 1: Comparison of Physiologic Cross Sectional Area (PCSA) (cm^2) of deep compartment muscles from the literature **(Mean (%))**

		Study Results					
		Study 1[31]	**Study 2[8]**	**Study 3[7]**	**Study 4[3]**	***Study 5[25]**	**Mean % (range)**
Muscle	**PT**	20.8 (66.7)	36.8 (56.4)	22.6 (53.3)	36.2 (60.1)	16.0 (54.2)	58.1 (53.3–66.7)
	FHL	5.3 (17.0)	19.3 (29.6)	13.7 (32.3)	14.1 (23.4)	9.0 (30.5)	26.6 (17.0–32.3)
	FDL	5.1 (16.3)	9.1 (14.0)	6.1 (14.4)	9.9 (16.5)	4.5 (15.3)	15.3 (14.0–16.5)
	Total PCSA	31.2 (100)	65.2 (100)	42.4 (100)	60.2 (100)	29.5 (100)	100

Posterior tibialis, PT; Flexor hallicus longus, FHL; Flexor digitorum longus, FDL.
* This data is from anatomic cross-sectional area which does not take into account pennation angle.

2 cm for the PT muscle, with muscle shortening occurring during forefoot adduction.[6] In support of the importance of forefoot movement, an in-vivo study noted that subtalar inversion and forefoot adduction tasks have greater task specificity than 3 other tasks for isolating the PT muscle from other leg muscles.[15] Therefore, because of PCSA, joint moment arms and task specificity, changes in PT muscle function are expected to overshadow the synergistic roles of the other deep compartment muscles when performing a strength test with a subtalar inversion and forefoot adduction task.

Another key element of strength testing of the PT muscle is co-contraction of the anterior tibialis (AT) muscle. Ankle plantarflexion is advocated to inhibit the synergistic contribution of the AT muscle when attempting to detect weakness.[13] Ankle plantarflexion is thought to minimize the contribution of the AT muscle on subtalar inversion and forefoot adduction either through task specificity or by placing the muscle in a lengthened, and presumably mechanically disadvantaged position.[13] However, no studies were found that investigated the AT muscle activation of subjects with PTTD during an ankle plantarflexion, subtalar inversion and forefoot adduction strength test. Effectiveness of this clinical strategy to detect PT muscle strength partly relies on the ability to minimize the contribution of the AT muscle, suggested by low activation during maximal efforts.

The purpose of this study was to compare isometric subtalar inversion and forefoot adduction strength in Stage II PTTD subjects to controls. The subtalar inversion and forefoot adduction task was hypothesized to be sensitive to PT muscle weakness because of muscle properties, joint mechanics and task specificity in subjects with Stage II PTTD. Ankle plantarflexion was expected to minimize the activation of the AT muscle. However, the potential for subjects with PT muscle weakness to compensate with AT activation was anticipated. A goal of nominal AT activation of less than 10% was set a priori for both subjects with Stage II PTTD and controls.

MATERIALS AND METHODS

Subjects

Twenty-four subjects with Stage II PTTD (18 females, 6 males) and 15 controls (12 females, 3 male) volunteered after giving informed consent. The subjects with PTTD were representative of the population, who frequently are middle aged or older, female, and overweight (Table 2). The control subjects were matched for age and body mass index of the first 10 PTTD subjects enrolled. The control subjects were required to have no known foot pathology and be free of lower extremity pain for the last 6 months. Inclusion criteria for the PTTD group required signs of 1) tendon pathology and 2) unilateral flexible flatfoot deformity. Signs of tendon pathology included pain and/or swelling along the medial ankle. Signs of flatfoot deformity were determined by clinical assessment with any one of the following, 1) greater unilateral hindfoot eversion, 2) greater forefoot abduction and/or 3) lower medial longitudinal arch height. To verify the subjects with Stage II PTTD on average had abnormal foot posture, the arch index was used (Table 2).[32] The arch index is the height from the floor to the top of the foot at 50% of foot length divided by truncated foot length. Truncated foot length is defined as the distance between the most posterior aspect of the heel to the center of the first metatarsol phalangeal joint. The reliability and validity of this method has been reported in a previous study.[32] Exclusion criteria included inability to walk greater than 15 meters or a history of previous foot or ankle pathology. A statistical power analysis suggested that sample sizes above 10 subjects per group were required to achieve 80% power with an expected effect size of 0.30 N/Kg and standard deviation of ± 0.25 N/Kg.

Instrumentation

To capture maximal isometric efforts, a force transducer (Model SML-200, Interface, Scottsdale, AZ) was connected in series with a resistance plate and oscilloscope (TDS

Table 2: Sample, foot posture and self-report scores (Mean±SD)

	Control ($n = 15$)	PTTD ($n = 24$)	p value
SAMPLE			
Age (years)	55 ± 8	61 ± 10	NS
Height (cm)	166 ± 11	168 ± 10	NS
Mass (kg)	77 ± 10	$84 \pm 17.$	NS
Body Mass Index	28 ± 5	30 ± 5	NS
Gender	F = 13, M = 2	F = 18, M = 6	
Side Involved		L = 14, R = 10	
FOOT POSTURE			
Arch Index	0.35 ± 0.03	0.31 ± 0.03	<0.001
SELF REPORTED SCORES			
Foot Function Index (%)*			
Pain		17 ± 11	
Disability		36 ± 19	
Activity Limitation		37 ± 17	
Short Musculoskeletal			
Function Assessment (%)*			
Function		24 ± 14	
Mobility		19 ± 12	
Bothersome		23 ± 19	

Posterior Tibial Tendon Dysfunction, PTTD; Female, F; Male, M; Left, L; Right, R.
* Higher scores indicate worse function.
NS = not significant.

410A, Tektronix, Beaverton, OR). Calibration of the force transducer with known weights suggested low errors ($r^2 = 0.997$, root mean square error = ±1 N). The resistance plate was mounted on ball bearing tracks to allow for movement in the medial/lateral direction. Thus, force exerted against the resistance plate was detected by the force transducer and displayed on the oscilloscope. A small pad, covered in moleskin, that fit the general shape of the medial forefoot was used to distribute pressure over the skin, allowing subjects to exert maximal efforts against the resistance plate with little discomfort. To assist subjects in maintaining a consistent ankle plantarflexion and leg position, an air stirrup brace (Aircast, Inc.) was utilized (Figure 1). However, the air stirrup brace was not rigidly fixed to the uprights, thus requiring subjects to achieve proximal stabilization through active muscle control. The role of the air stirrup brace was to assist the subject maintain a constant ankle plantarflexion angle and foot posture. Inability to invert the hindfoot or raise the medial longitudinal arch of the foot invalidated the test. The air stirrup brace was set so the heel was approximately 10 cm above the resistance plate, resulting in varying degrees of plantarflexion from 30 degrees to 45 degrees, depending on foot size. Because the ankle plantarflexion angle has little influence on PT muscle length, the influence of ankle plantarflexion on isometric strength was assumed to be negligible.[6,14,20]

Visual feedback of the amount of force exerted and muscle activity of the AT muscle for each effort was provided via an oscilloscope. After cleaning and abrading the skin, a surface electrode (DE-2.1, Delsys, Inc., Boston, MA) was placed in a standardized location (one hand width distal to the tibial tuberosity) over the skin of the AT muscle. The surface electrode was connected to a 2-channel EMG system (Bangoli-2 EMG System, Delsys, Inc., Boston, MA) and the gain was adjusted from 1 to 1000. The force and muscle activity were displayed on the oscilloscope to provide feedback to the examiner and subjects (Figure 1). Readings were taken directly from the oscilloscope which has a digital display that samples data at 1000 Hz. Peak to peak EMG amplitudes in volts were recorded for the maximum voluntary contraction (MVC) and strength testing trials (Figure 1).

Procedure

The procedures included determining 10% AT activation and peak isometric subtalar inversion and forefoot adduction. Prior to performing a maximum inversion and forefoot adduction effort, three maximal ankle dorsiflexion efforts were recorded using manual resistance. As a benchmark,

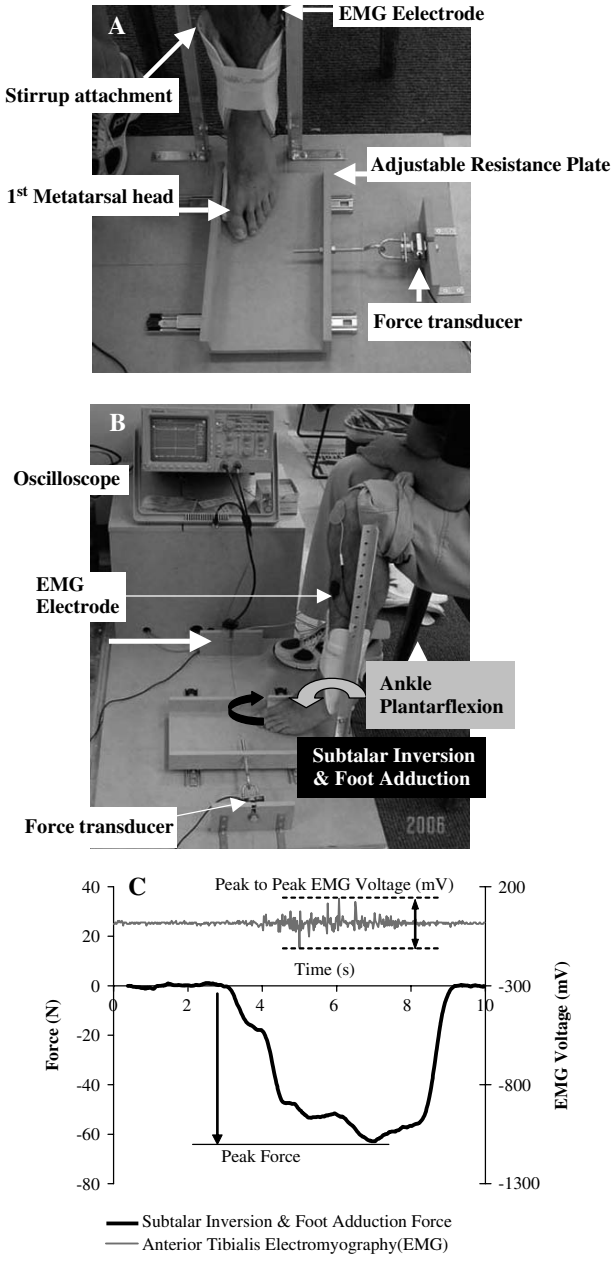

Fig. 1: **A**, Picture of foot position. **B**, Picture of subject setup in the instrumentation system for isometric subtalar inversion and foot adduction strength testing. **C**, Display of peak force (converted from volts) and electromyography (EMG).

10% of the highest peak to peak EMG signal of three MVC trials for the AT muscle was used as a target for examiners and subjects during the trials. To perform the ankle inversion and forefoot adduction effort, the subject's foot was placed in the ankle stirrup, the foot plate was adjusted to align the metatarsal head with the medial malleoli, which placed the subject in a neutral or slightly abducted foot position (Figure 1). After being properly positioned, subjects performed five practice submaximal efforts and five maximal efforts. During each effort, subjects were instructed to maintain a plantarflexion moment while performing a subtalar inversion and forefoot adduction effort. Subjects were required to maintain neutral foot posture throughout the test. Visually this meant maintenance of the medial longitunal arch as judged by the examiner. This eliminated efforts that involved simply dragging the foot across the foot plate with proximal musculature. During the submaximal efforts, if AT muscle activity was high (exceeded 10% of MVC), subjects were coached to plantarflex more, and/or push medially with their forefoot, diminishing the contribution of the AT muscle. These commands were individualized to each subject, with the goal of minimizing AT activation. Once the practice trials were completed, visual feedback of the force and verbal encouragement were used to motivate subjects to exert a maximal effort. Peak force and peak to peak EMG amplitude for each maximum subtalar inversion and foot adduction effort was read from the oscilloscope and manually recorded. For all subjects the sequence of which side was tested was randomized.

Between session reliability

Eleven subjects from the control group volunteered to perform the test again on a separate day. The second session occurred less than a week from the first session. This interval of time was sufficiently short to expect the same isometric strength measures for both sessions. All the procedures above were followed during each session. To minimize the chance for examiner bias, the examiner was not allowed to review the results of session one prior to performing the second session. One examiner tested all subjects.

Analysis of strength and EMG data

The first analysis addressed reliability, while the second addressed the comparison of isometric strength between the controls and PTTD subjects. To assess the reliability of the subtalar inversion and foot adduction peak force, an intraclass correlation statistic (model 3, 1) was applied to the session one and session two data. For the first hypothesis, the dependent variable was the peak isometric subtalar inversion and foot adduction force of the 3 maximal efforts. All force values were normalized to body mass. A two-way mixed effects ANOVA model was used to test for differences between groups (between groups factor = PTTD vs. control) and sides (within groups factor = involved vs. uninvolved). For the control subjects, the right side was considered the involved side and the left the uninvolved side. Preliminary analyses suggested no side to side differences in control subjects justifying this approach. A significant interaction, indicating lower peak isometric force of the involved side of the PTTD subjects compared to the uninvolved side of subjects with PTTD and controls (both sides) was consistent with the main hypothesis. In the presence of an interaction, pairwise comparisons were used to test for differences across groups and sides. Additionally, to mirror clinical practice, where weakness is commonly assessed by comparing to

the opposite side, the ratio of involved to uninvolved peak isometric subtalar inversion and foot adduction force normalized to mass was compared between the Stage II PTTD subjects and controls using a Student's t-test.

Second, to assess whether the average AT muscle activation during the peak isometric subtalar inversion and foot adduction test varied, the dependent variable was the peak to peak EMG activation normalized to MVC (% MVC). Similarly, a two-way mixed effects ANOVA was used to test for differences between groups (between groups factor = PTTD vs. control) and sides (within groups factor = involved vs. uninvolved). A significant interaction, indicating higher % MVC on the involved side of the PTTD subjects compared to the uninvolved side of the PTTD subjects and controls (both sides) would suggest compensation with the AT muscle. In the absence of an interaction, a main effect for group, with the PTTD group showing higher AT muscle activation than the controls would indicate substitution not unique to muscle weakness. For all analyses (two-way mixed effects ANOVA's and Student's t-test), an alpha level of 0.05 was used for significance.

RESULTS

The between session reliability of the normalized peak isometric subtalar inversion and foot adduction test was high (Table 3).

There was a significant interaction between group and side ($p = 0.003$), that resulted from a lower peak isometric force of the involved side of the PTTD group. Normalized peak isometric subtalar inversion and foot adduction force for the PTTD group was significantly lower ($p = 0.001$) at 0.70 ± 0.24 N/kg compared to the uninvolved side at 0.99 ± 0.24 N/Kg. The involved side for the PTTD group was also significantly lower (involved $p < 0.001$ and uninvolved $p = 0.001$) as compared to both sides of the control group (Table 4). A post hoc analysis revealed a mild significant correlation ($r = -0.55$, $p < 0.001$) between age and normalized peak isometric subtalar inversion and foot adduction force. Because the groups showed a small difference in age of 5.5 years (Table 2), that was close to significant (Table 2, $p = 0.07$), the analysis was repeated with age as a covariate. With age entered as a covariate the significant interaction remained ($p = 0.012$) and no pairwise comparisons were changed. The ratio of the involved to the uninvolved side was also statistically ($p = 0.003$) lower in the PTTD group compared to the controls. Post hoc analysis suggested that the ratio of the involved to the uninvolved side was not related to age ($r = -0.263$, $p = 0.10$) so no further analysis was pursued.

The EMG activation of the AT muscle did not demonstrate a significant interaction ($p = 0.314$) or main effect for group ($p = 0.324$) (Table 4). However, the high standard deviations in the PTTD group compared to the control group caused us to examine AT activation more closely. In the PTTD group the percent of subjects with less than 10% MVC of the AT muscle was 54% and 46% on the involved and uninvolved sides respectively. Similar percentages were observed in the control group with 60% of subjects on both sides achieving less than 10% MVC of the AT muscle. The highest AT muscle activation of the control group on either side was 27% MVC. In contrast, three subjects in the PTTD group used an AT activation that was higher than 27% MVC on both sides.

DISCUSSION

The findings of this study suggest an isometric subtalar inversion and forefoot adduction weakness in subjects with Stage II PTTD as compared to controls. The results of this study show a subtalar inversion and forefoot adduction strength deficit of 20% to 30% in subjects classified with Stage II PTTD. While isolated testing of the PT muscle is difficult, a specific test based on muscle properties (Table 1), joint mechanics and task specificity was sensitive to weakness of the PT muscle. However, co-activation of the AT muscle was high despite targeting lower co-activation levels. It is assumed that pain intensity (which was not assessed in this study) during the testing may

Table 3: Reliability of isometric strength tests of control subjects ($n = 11$)

	Trial 1	Trial 2	ICC Value-model 3, 1 (95% CI)
Variable			
Subtalar Inversion and Foot Adduction Normalized Force (N/Kg)*100			
Right side	97 ± 24	99 ± 25	0.87 (0.56–0.96)
Left side	97 ± 23	98 ± 22	0.91 (0.71–0.98)
Ratio of left/right	104 ± 30	100 ± 22	0.76 (0.32–0.93)

CI = Confidence Interval. ICC = ?

Table 4: Average (SD) values for subtalar inversion and foot adduction isometric strength

	Control ($n = 15$)	PTTD ($n = 24$)	p value[#]
Variable			
Normalized Force (N/Kg)*100			
Involved Side*	99 ± 24	70 ± 24	0.005[$]
Uninvolved Side**	96 ± 21	94 ± 24	NS[$]
Ratio of left/right side	106 ± 31	77 ± 25	0.008
Anterior tibialis muscle Normalized EMG (% MVC)			
Involved Side*	12.5 ± 9.5	14.7 ± 15.0	NS
Uninvolved Side**	11.3 ± 8.5	17.1 ± 13.7	NS

[#] p values are for pair wise comparisons between control and PTTD groups.
[$] p values are the result of performing the analysis with age entered as a covariate.
* Right side for controls.
** Left side for controls.
NS = not significant.
EMG = electromyography.
MVC = Maximum voluntary contraction.

have also influenced maximum efforts. Subject positioning and proximal stabilization may also influence the specific strength test proposed. The reliability data (Table 3) suggest consistency in control subjects; however, consistency in subjects with PTTD needs to be tested to improve clinical interpretation.

Four factors specific to muscle function including 1) PCSA, 2) moment arms, 3) muscle activation and 4) pain may contribute to the subtalar inversion and forefoot adduction test being sensitive to PT weakness in subjects with PTTD. The PCSA is a key morphologic measure of muscle that reflects the ability of a muscle to produce force. Of the deep compartment muscles (FDL, FHL and PT), the PT muscle accounts for 60% of the PCSA (Table 1). A study of subjects with PTTD just prior to surgery (an average of 40 months of failed treatment) documented fatty infiltration in 3/12 PT muscles and deficits in muscle volume of 2% to 17% compared to the uninvolved side.[30] This study supports the influence of PTTD on muscle volume, and hence, PCSA. Also, in vitro studies document moment arms in cadaver subjects but not in subjects with PTTD or foot pronation.[6,10,14] Nikki et al. documented the failure of the PT muscle to raise the foot to the same position after inducing flatfoot.[19] One explanation for this decreased effectiveness of the PT muscle is a change in moment arms at either the subtalar or talonavicular joints. Studies of subjects after immobilization suggest weakness is a combination of activation failure and muscle atrophy.[26] Some degree of activation failure may also exist in subjects with PTTD. Also, pain during the isometric strength test may inhibit maximal efforts, contributing to activation deficits. Collectively, these studies suggest that the subtalar inversion and foot adduction isometric strength deficit of 20% to 30% observed in subjects with PTTD is due to a combination of PT muscle weakness and/or pain. Using a subtalar inversion and foot adduction isometric strength test may provide an early indication of PT muscle weakness in subjects with PTTD.

The greater activation of the AT muscle by the PTTD group on the involved side suggests difficulty isolating the PT muscle during the task. The average AT activation was not statistically different between the control and PTTD groups. However, only 60% of the subjects in the control group and 40% to 50% of the subjects in the PTTD group achieved the target of less than 10% activation. The 3 subjects in the PTTD group with activation greater than 30% MVC showed severe weakness (ratio involved/uninvolved side ranged from 0.50 to 0.60), suggesting they may have been attempting to substitute for a weak PT muscle. For this analysis, AT activation was presumed to have the potential to mask weakness of the PT muscle. However, the amount of AT activation did not correlate to the deficit in PT isometric strength ($r = -0.31$, $p = 0.14$). This lack of correlation, and the observation that those with the highest AT activation demonstrated significant weakness (ratio involved/uninvolved side ranged from 0.50 to 0.60), suggests AT activation may not mask weakness for this task. Because EMG only indicates neural drive and not mechanical effects, this is possible. For example, the AT muscle is significantly lengthened in the test position decreasing its ability to generate force. Also, the AT muscle may not have

a significant moment arm for forefoot adduction, neutralizing the mechanical effect of its activation. For example, the AT moment arm for rearfoot inversion has been estimated as 20% of the moment arm for the PT muscle.[14] Nevertheless, less AT activation would indicate a more isolated measure of the PT muscle, which was not achieved across subjects with PTTD in this study. Other strength tests that use a dorsiflexed foot position, such as isokinetic testing, are speculated to induce even higher AT activation.

Clinical significance

Recent studies are exploring the potential for exercise to alter the course of PTTD. Alvarez et al. reported an observational study that demonstrated promising outcomes using an aggressive exercise regimen in subjects with Stage I and II PTTD.[4] Others reported the design of a clinical trial that is underway to distinguish the benefits of eccentric versus concentric exercise for subjects with PTTD.[16] These exercise programs partially base their approach on the outcomes observed with active treatment of the Achilles tendon.[1,2] However, unlike Achilles tendinopathy, PTTD has been associated with damage to major foot ligaments[5,9] and abnormal walking mechanics.[11,22,23,27] Further, in-vitro studies suggest that the effectiveness of the PT muscle in controlling foot posture partially depends on the integrity of foot ligaments.[19] Because of this dependence of the effectiveness of the PT muscle on foot ligaments, restoring the strength of the PT muscle may have positive but limited therapeutic benefits. To assess the effect of exercise programs, such as those proposed above, methods to quantify PT muscle recovery are needed. This study presents one testing method that appears sensitive to PT muscle weakness. However, future research is needed to determine test properties such as a clinical meaningful change and responsiveness to improve clinical interpretation. Clinicians are also advised that isolating the PT muscle fully from AT muscle activation is difficult, with a high percentage of subjects exceeding 10% activation.

Limitations

The ability to truly isolate the PT muscle from other synergistic muscles is difficult. The air stirrup brace may have inhibited or enhanced the contribution of the PT muscle. Siegler et al.[24] showed in-vivo that angular stiffness (degrees/Nm) increased markedly toward the end range of subtalar inversion and less so in the midrange. Because the task is isometric, the midrange stiffness is more relevant. The increased midrange stiffness of the air stirrup brace, although small, may have limited inversion motion, inhibiting the full contribution of the subtalar inverters. On the other hand, the air stirrup brace may have improved the positioning of the hindfoot in subjects with PTTD, increasing the moment arm of the PT muscle at the subtalar joint.[15,19] Both in-vitro[19] and in-vivo[15] studies document greater contributions of the PT muscle when foot position is toward neutral. Future studies may consider comparisons across different testing positions. For example, Valderrabano et al.[28] used a sidelying position when evaluating subjects with PTTD post operatively. The sidelying position also may provide greater proximal stabilization of the tibia. Further, without a direct comparison, it is unclear if isokinetic strength testing as used by Alvarez et al.[4] is sufficient to capture PT muscle weakness. Alternative positioning and stabilization strategies may also assist with inhibiting the AT muscle. Because the AT muscle is an invertor, subtalar eversion with plantarflexion may further inhibit this muscle.

Other limitations relate to reliability testing, the sample used and limb dominance. A key limitation is that the reliability testing was only performed on control subjects. Reliability and responsiveness testing of subjects with PTTD is necessary to establish the utility of this test clinically. In addition, the clinical meaningfulness of a strength deficit is not clear. Future studies focusing on repeated measurements of isometric strength over time are needed. Another issue is the sample characteristics of the PTTD and control groups. Stage II PTTD is inclusive of a wide spectrum of disease severity.[17] As new stages are defined, the PTTD group sample characteristics in Table 2 will be important to consider when comparing across studies. Also, the use of a matched control group on three key variables including gender, BMI, and age may influence the results, as demonstrated by the correlation of age and isometric strength. The effect of limb dominance was not assessed in this study. Some studies suggest that lower extremity dominance may account for some of the variance when comparing side to side.[29] However, side-to-side differences due to dominance are unlikely to fully explain the 20% to 30% differences between the PTTD subjects and controls observed in this study.

ACKNOWLEDGMENT

The authors are thankful for the funding received from the American Orthopedic Foot and Ankle Society and National Institute of Arthritis And Musculoskeletal And Skin Diseases (Grant Number R15AR054507).

REFERENCES

1. **Alfredson, H:** Chronic midportion Achilles tendinopathy: an update on research and treatment. Clinics in Sports Medicine **22**:727–41, 2003. http://dx.doi.org/10.1016/S0278-5919(03)00010-3
2. **Alfredson, H:** Chronic tendon pain–implications for treatment: an update. Current Drug Targets **5**:407–10, 2004. http://dx.doi.org/10.2174/1389450043345353
3. **Allard, P; Stokes, I; Blanchi, J-P:** Three-Dimensional Analysis of Human Movement. Edited, Champaign, IL, Human Kinetics, 1995.
4. **Alvarez, RG; Marini, A; Schmitt, C; Saltzman, CL:** Stage I and II posterior tibial tendon dysfunction treated by a structured nonoperative management protocol: an orthosis and exercise program. Foot Ankle Int. **27(9)**:723–7; **27**:2–8, 2006.
5. **Deland, JT; de Asla, RJ; Sung, IH; Ernberg, LA; Potter, HG:** Posterior tibial tendon insufficiency: which ligaments are involved? Foot Ankle Int. **27(9)**:723–7; **26**:427–35, 2005.

6. **Flemister, AS; Neville, CG; Houck, J:** The relationship between ankle, hindfoot, and forefoot position and posterior tibial muscle excursion. Foot Ankle Int. **28**:448 – 55, 2007. http://dx.doi.org/10.3113/FAI.2007.0448
7. **Friederich, JA; Brand, RA:** Muscle fiber architecture in the human lower limb. J. Biomech. **36(4)**:577 – 91; **23**:91 – 5, 1990.
8. **Fukunaga, T; Roy, RR; Shellock, FG; et al.:** Physiological cross-sectional area of human leg muscles based on magnetic resonance imaging. J. Orthop. Res. **10(6)**:928 – 34; **10**:928 – 34, 1992.
9. **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. **88(8)**:1764 – 8; **79**:675 – 81, 1997.
10. **Hintermann, B; Nigg, BM; Sommer, C:** Foot movement and tendon excursion: an in vitro study. Foot Ankle Int. **27(9)**:723 – 7; **15**:386 – 95, 1994.
11. **Johnson, J; Harris, G:** Pathomechanics of Posterior Tibial Tendon Insufficiency. Foot and Ankle Clinics **2**:227 – 239, 1997.
12. **Johnson, KA; Strom, DE:** Tibialis posterior tendon dysfunction. Clin. Orthop. **(333)**:172 – 80; 196 – 206, 1989.
13. **Kendall, HO; Kendall, FP; Wadsworth, GE:** Muscles, testing and function. Edited, Baltimore, Williams and Wilkins, 1971.
14. **Klein, P; Mattys, S; Rooze, M:** Moment arm length variations of selected muscles acting on talocrural and subtalar joints during movement: an in vitro study. J. Biomech. **36(4)**:577 – 91; **29**:21 – 30, 1996.
15. **Kulig, K; Burnfield, JM; Requejo, SM; Sperry, M; Terk, M:** Selective activation of tibialis posterior: evaluation by magnetic resonance imaging. Med. Sci. Sports Exerc. **38(4)**:746 – 52; **36**:862 – 7, 2004.
16. **Kulig, K; Pomrantz, AB; Burnfield, JM; et al.:** Non-operative management of posterior tibialis tendon dysfunction: design of a randomized clinical trial [NCT00279630]. BMC Musculoskeletal Disorders **7**:49, 2006. http://dx.doi.org/10.1186/1471-2474-7-49
17. **Lee, MS; Vanore, JV; Thomas, JL; et al.:** Diagnosis and treatment of adult flatfoot. J. Foot Ankle Surg. **44(2)**:78 – 113; **44**:78 – 113, 2005.
18. **Lieber, RL; Friden, J:** Clinical significance of skeletal muscle architecture. Clin. Orthop. Relat. Res. 140 – 51, 2001. http://dx.doi.org/10.1097/00003086 – 200102000-00016
19. **Niki, H; Ching, RP; Kiser, P; Sangeorzan, BJ:** The effect of posterior tibial tendon dysfunction on hindfoot kinematics. Foot Ankle Int. **27(9)**:723 – 7; **22**:292 – 300, 2001.
20. **Ottaviani, RA; Ashton-Miller, JA; Wojtys, EM:** Inversion and eversion strengths in the weightbearing ankle of young women. Effects of plantar flexion and basketball shoe height. Am. J. Sports Med. **21(1)**:49 – 54; **29**:219 – 25, 2001.
21. **Pinney, SJ; Lin, SS:** Current concept review: acquired adult flatfoot deformity. Foot Ankle Int. **27**: 66 – 75, 2006.
22. **Reinschmidt, C; van den Bogert, AJ; Lundberg, A; et al.:** Tibiofemoral and tibiocalcaneal motion during walking: external vs. skeletal markers. Gait & Posture **6**:98 – 109, 1997. http://dx.doi.org/10.1016/S0966-6362(97)01110-7
23. **Ringleb, SI; Bensamoun, SF; Chen, Q; et al.:** Applications of magnetic resonance elastography to healthy and pathologic skeletal muscle. J. Magn. Reson Imaging **25(2)**:301 – 9; **25**:301 – 9, 2007.
24. **Siegler, S; Liu, W; Sennett, B; Nobilini, RJ; Dunbar, D:** The three-dimensional passive support characteristics of ankle braces. J. Orthop. Sports Phys. Ther. **26**:299 – 309, 1997.
25. **Silver, RL; de la Garza, J; Rang, M:** The myth of muscle balance. A study of relative strengths and excursions of normal muscles about the foot and ankle. J. Bone Joint Surg. Br. **67(3)**:432 – 7; **67**:432 – 7, 1985.
26. **Stevens, JE; Pathare, NC; Tillman, SM; et al.:** Relative contributions of muscle activation and muscle size to plantarflexor torque during rehabilitation after immobilization. J. Orthop Res. **10(6)**:928 – 34; **24**:1729 – 36, 2006.
27. **Tome, J; Flemister, S; Nawoczenski, D; Houck, J:** Comparison of Foot Kinematics Between Subjects with Posterior Tibial Tendon Dysfunction and Healthy Controls. J. Orthop. Sports Phys Ther. **33(5)**:273 – 82; discussion 283 – 6; **36**:635 – 644, 2006.
28. **Valderrabano, V; Hintermann, B; Wischer, T; Fuhr, P; Dick, W:** Recovery of the posterior tibial muscle after late reconstruction following tendon rupture. Foot Ankle Int. **25**:85 – 95, 2004.
29. **Valderrabano, V; Nigg, BM; Hintermann, B; et al.:** Muscular lower leg asymmetry in middle-aged people. Foot Ankle Int. **27(9)**:723 – 7; **28**:242 – 9, 2007.
30. **Wacker, J; Calder, JD; Engstrom, CM; Saxby, TS:** MR morphometry of posterior tibialis muscle in adult acquired flat foot. Foot Ankle Int. **27(9)**:723 – 7; **24**:354 – 7, 2003.
31. **Wickiewicz, TL; Roy, RR; Powell, PL; Edgerton, VR:** Muscle architecture of the human lower limb. Clin. Orthop. **(333)**:172 – 80; 275 – 83, 1983.
32. **Williams, DS; McClay, IS:** Measurements used to characterize the foot and the medial longitudinal arch: reliability and validity. Phys Ther. **79(9)**:854 – 9; **80**:864 – 71, 2000.