Available online at www.sciencedirect.com
ScienceDirect
Clinical Biomechanics 22 (2007) 592–598
CLINICAL BIOMECHANICS
www.elsevier.com/locate/clinbiomech

The effect of flatfoot deformity and tendon loading on the work of friction measured in the posterior tibial tendon

Kenichiro Arai, Stacie I. Ringleb, Kristin D. Zhao, Lawrence J. Berglund,
Harold B. Kitaoka, Kenton R. Kaufman *

Biomechanics Laboratory, Division of Orthopedic Research, Mayo Clinic, 200 First Street SW, Rochester, MN 55095, USA

Received 28 February 2006; accepted 17 January 2007

Abstract

Background. There is limited information regarding the mechanical factors contributing to the progression of posterior tibial tendon dysfunction. Therefore, an investigation of the mechanical forces on the posterior tibial tendon may improve our understanding of this pathology.

Methods. The gliding resistance and excursion of the posterior tibial tendon in the retromalleolar region was measured in seven cadaveric lower limbs in the coronal, transverse, and sagittal planes. These data were used to calculate the work of friction and to characterize the effect of different tendon loading levels (0.5, 1.0, and 2.0 kg) in the intact and flatfoot conditions.

Findings. Flatfoot deformity significantly increased the excursion of the posterior tibial tendon ($P < 0.05$), increased forefoot and hindfoot range of motion in the coronal and transverse planes ($P < 0.05$) and the work of friction in the coronal and transverse planes ($P < 0.05$), but not in the sagittal plane. There was a significant increase in the work of friction between 0.5 and 2 kg ($P < 0.05$) in all three planes of motion.

Interpretation. The motions in the coronal and transverse planes have a greater effect on the work of friction of the posterior tibial tendon than sagittal plane motion in the flatfoot condition. This study suggests that aggressive treatment of early stage PTT dysfunction with bracing designed to limit coronal and transverse motions, while permitting sagittal motion should be investigated further. Such bracing may decrease the potential of progressive deformity while allowing for more normal ambulation.

Keywords: Foot; Flatfoot; Posterior tibial tendon dysfunction; Tendon; Biomechanics

1. Introduction

Posterior tibial tendon (PTT) dysfunction has been recognized as the most common cause of acquired flatfoot deformity in adults (Myerson and Corrigan, 1996; Augustin et al., 2003). Patients who are not effectively treated during the early stages of PTT dysfunction risk progression to a severe deformity of the foot, which can lead to disability. Consequently, PTT dysfunction has received increased attention in the literature in the past two decades and represents a challenging problem for orthopedic surgeons and other clinicians.

Numerous intrinsic and extrinsic risk factors have been proposed as the underlining cause of PTT dysfunction including tendon and joint disorders, preexisting medical conditions, and anatomic variations. Tendon and joint disorders such as pre-existing tenosynovitis (Cozen, 1965), trauma (Woods and Leach, 1991), and degenerative changes (Mosier et al., 1998; Mosier et al., 1999; Holmes and Mann, 1992) have been considered as possible contributing factors. Some experts have suggested that medical conditions such as obesity, hypertension, and diabetes (Holmes and Mann, 1992) or inflammatory systemic arthropathy (Myerson et al., 1989) may also be predisposing factors in the

---

* Corresponding author.
E-mail address: Kaufman.Kenton@mayo.edu (K.R. Kaufman).

0268-0033/$ - see front matter © 2007 Elsevier Ltd. All rights reserved.
doi:10.1016/j.clinbiomech.2007.01.011

development of PTT dysfunction. Other etiologies, such as abnormal talar architecture (Anderson et al., 1997), the relation of the posterior tibial tendon to the accessory navicular (Chen et al., 1997), a distinct area of hypovascularity in the PTT (Frey et al., 1990; Petersen et al., 2002), impingement of the PTT (Park et al., 2005), and ipsilateral valgus gonarthrosis (Sobel, 1992) have been attributed to the cause of PTT dysfunction in anatomic studies. Additionally, some patients who have pes planus may develop dysfunction of the PTT, which may lead to the development of a rigid flatfoot deformity (Mosier et al., 1999; Kettelkamp and Alexander, 1969; Funk et al., 1986; Dyal et al., 1997). Some specialists have suggested that flexible flatfoot deformity could lead to overuse injury, microtrauma and degeneration of the PTT as a result of the chronic mechanical overload (Jahss, 1982; Dyal et al., 1997; Mosier et al., 1999).

Investigation of the mechanical forces on the PTT under various loading conditions in the intact and flatfoot conditions may provide insight into the cause of the progression of PTT dysfunction. In healthy individuals, tendon friction is limited because of the smooth interface between the tendon and pulley (e.g., the medial malleolus) and the lubrication provided by the synovial fluid in the tendon sheath. However, in a pathological state such as trauma to the tendon or pulley, tendon repair, and/or anatomic deformity, friction may increase. Repetitive exposure to increased friction and attrition of the tendon may be one factor causing cumulative trauma and leading to tendon degeneration or rupture (An et al., 1993; Zhao et al., 2001).

A method to measure gliding resistance at the tendon–pulley interface was developed and validated using the flexor tendons of the hand (Uchiyama et al., 1997). A significant increase in the gliding resistance between the flexor digitorum profundus and the A2 pulley of the hand was measured before and after tendon repair (Coert et al., 1995). This technique was also applied to the PTT, with the ankle in neutral, maximum dorsiflexion, and maximum plantar flexion in the intact and simulated flatfoot conditions (Uchiyama et al., 2000). A significant increase in gliding resistance was observed after flatfoot deformity was created, however physiologic motions were not considered and the gliding resistance in all three planes of motion was not measured.

The purpose of this study was to examine the gliding resistance of the PTT in the retromalleolar region and the excursion of the PTT while the foot was moved passively in all three planes of motion. These data allowed us to calculate the work of friction and to characterize the effect of PTT loading levels and the flatfoot condition. Our hypothesis was that the work of PTT friction is higher in the flatfoot condition, and is higher with increasing tendon loading levels.

2. Methods

Seven fresh-frozen cadaveric lower extremities were studied. X-rays were taken and any specimen with osteoarthritis greater than grade 1 was eliminated. When the specimen was prepared for testing by a foot and ankle surgeon, the specimen was examined for deformity and hindfoot stability. No specimens were tested with ankle instability or visible deformities. The mean specimen age was 67 years (range 35–93 years) and all were male. The tibia and the fibula were transected 30 cm proximal to the plantar aspect of the foot and the soft tissues were removed from the proximal portion of the tibia and fibula. The specimens were potted in an acrylic cylinder with polymethylmethacrylate (PMMA) and mounted in a custom testing apparatus. The proximal and distal portions of the PTT were exposed and ring tension transducers (diameter 1.5 cm) were attached to measure the gliding resistance caused by the PTT wrapping around the bony pulley at the medial malleolus. At the distal end, a 1.5 cm section of the PTT was removed from its first main insertion to the navicular to accommodate the sensor without altering the length of the tendon. The transducer was then attached to the tendon and anchored to the navicular. The tendon was

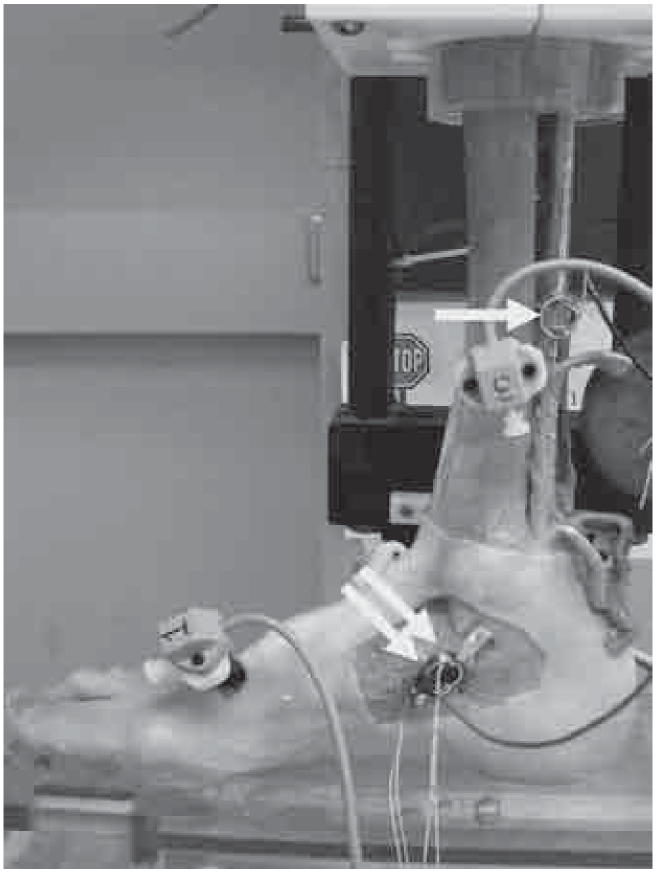

Fig. 1. Two force transducers were attached to the proximal (single arrow) and distal (double arrow) ends of the PTT. At the proximal end, a modified Krakow suture technique was used. The force transducer was attached to the distal end of the PTT using Bunnel suture technique on the proximal side of the sensor and was anchored to the navicular on the distal side of the sensor. Electromagnetic sensors are visible in the first metatarsal and tibia. An additional sensor was placed in the calcaneus on the lateral side of the foot (not visible).

attached proximally to a cable with the transducer in series (Fig. 1). The tension transducers were designed to be sensitive for low loads, with minimum inertia associated with the mass of the transducer's body. The resolution of the recorded output of the force transducer was better than 0.05% full scale output. Each sensor was calibrated with weights ranging from 0 to 4.0 kg. The relationship between the applied load and the sensor output was linear with $r^2 = 1.00$.

Static loads (0.5, 1.0, and 2.0 kg) were applied to the PTT by hanging weights on a cable wrapped around a pulley incorporating a rotary potentiometer to measure PTT excursion (Vishay Intertechnology, Inc. Malvern, PA, USA). Weights were chosen to perform a preliminary study of the effects of different loading conditions on the PTT without damaging the tendon during the 18 test conditions (i.e., three loading conditions and three motions in both the intact and flatfoot conditions). Three-dimensional kinematics of the forefoot (the first metatarsal relative to the tibia) and hindfoot (calcaneus relative to the tibia) were monitored with an electromagnetic tracking system (3Space Fastrak System, Polhemus, Colchester, VT, USA). The foot was passively manipulated, manually, in the sagittal plane (plantarflexon/dorsiflexon), coronal plane (inversion/eversion) and transverse plane (internal/external rotation). These motions were reported for each applied load through each specimen's range of motion for five cycles. Euler angles were calculated and the tendon excursion and loading were recorded using the Motion-Monitor™ (Innovative Sports Training, Inc., Chicago, IL, USA). Each specimen was tested in the intact condition and after creating a flatfoot. The flatfoot was created by sectioning the peritalar soft tissue constraints. Specifically the spring ligament, long and short planter ligaments, medial talocalcaneal ligament, talocalcaneal interosseous ligament, and tibionavicular portion of the superficial deltoid ligament were sectioned systematically for each specimen (Kitaoka et al., 1998).

2.1. Data analysis

A custom program was written in Matlab (The MathWorks, Inc., Natick, MA) to calculate the work of friction. The kinematic data were used to identify each cycle of motion. The force in the proximal sensor was subtracted from the force in the distal sensor to calculate the gliding resistance (An et al., 1993; Uchiyama et al., 1995). Gliding resistance was plotted against the PTT excursion to yield a hysteresis curve (Fig. 2). The hysteresis curve was truncated to ensure that the same range of motion was considered for each testing condition. In the coronal and transverse planes of motion, the hysteresis curves were simultaneously truncated such that 1.5 cm of PTT excursion was considered. In the sagittal plane of motion, the hysteresis curves were truncated to consider 0.3 cm of PTT motion. These curves had side by side displacement and did not follow the same tracks in every cycle. Consequently, each hysteresis was truncated individually.

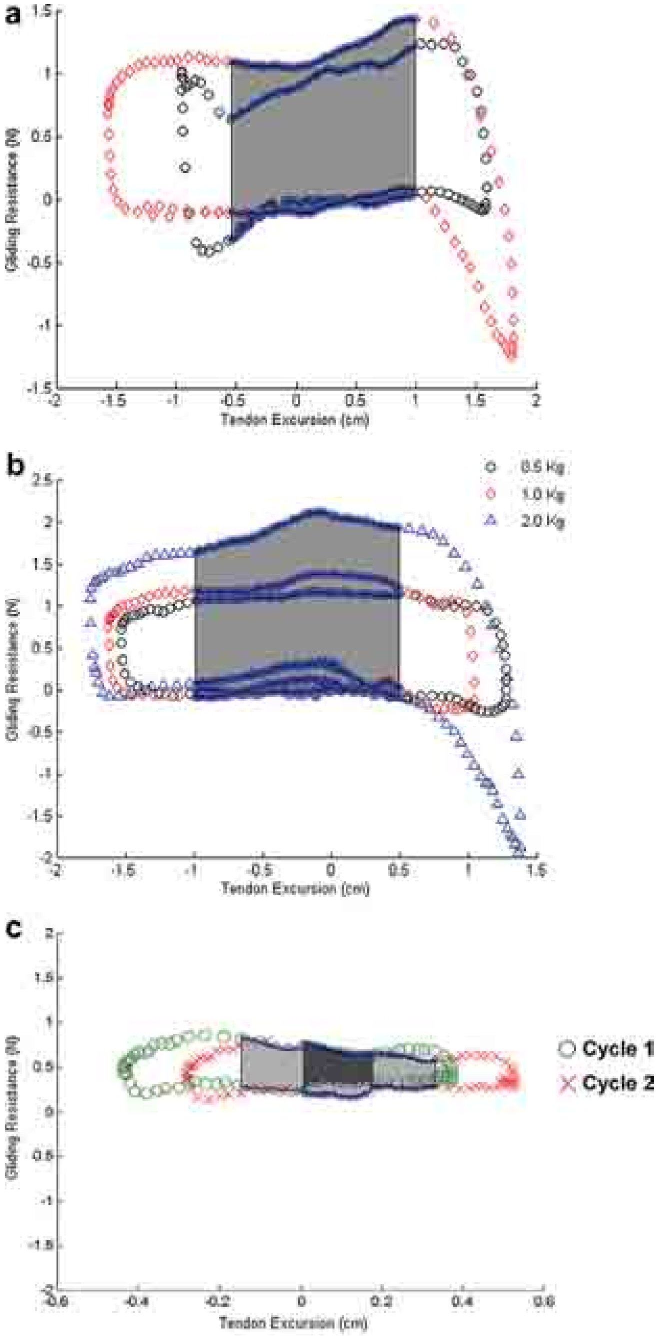

Fig. 2. (a) Sample hysteresis curves of the intact and flatfoot condition in the coronal plane. The intact condition is shown in black and the flatfoot condition is shown in red. (b) Sample hysteresis curves of the PTT loaded at 0.5, 1.0, and 2.0 kg in the coronal plane. In (a) and (b), the shaded areas represent uniform truncation of the curves for all test conditions used to calculate the work of friction. Similar curves were seen in the transverse plane. (c) Sample hysteresis curves in the sagittal plane. Both curves were truncated to consider 0.3 cm of PTT excursion, for a valid comparison.

Because work is defined as area under the curve of force vs. excursion, the area within the truncated curve was defined as the work of friction ($\int \vec{F} \cdot \mathbf{d}\vec{s}$, where $\vec{F}$ is the force and $\mathbf{d}\vec{s}$ is the differential displacement). The work of friction was calculated from these data and was used to assess the effect of flatfoot deformity and tendon loading. The relationship between the maximum tendon excursion and kinematics of the forefoot and hindfoot were also considered. The mean and standard deviation from three motion cycles was reported.

The data were not normally distributed, thus dependent variables were tested using non-parametric statistics. The Wilcoxon signed rank test was used to test differences in the tendon excursion, kinematics of forefoot and hindfoot, and the work of friction between intact and simulated flatfoot. The Friedman test was used to test differences in work of friction with each tendon loading level. P values less than 0.05 were considered to be statistically significant.

3. Results

The kinematics and PTT excursion vs. gliding resistance curves were consistent within test conditions. The maximum tendon excursion in the coronal and transverse planes increased significantly when the flatfoot condition was created ($P < 0.05$), but did not change in the sagittal plane (Table 1). The maximum forefoot range of motion increased significantly the coronal and transverse planes ($P < 0.05$) and the maximum hindfoot range of motion increased significantly in the transverse plane between the intact and flatfoot condition (Table 2).

In the coronal and transverse planes of motion, the work of friction was calculated for 1.5 cm of PTT excursion in the coronal and transverse planes and for 0.3 cm of PTT excursion in the sagittal plane. These excursions were selected since they represented the range of steady state gliding resistance. There was a significant increase in the work of friction between 0.5 and 2.0 kg tension loading in the sagittal ($P = 0.03$ intact, $P = 0.02$ flat), coronal ($P = 0.03$ intact, $P = 0.02$ flat) and transverse ($P = 0.03$ intact, 0.04 flat) planes (Table 3). Flatfoot deformity increased the work of friction significantly in the coronal and transverse planes ($P < 0.05$), but not in the sagittal plane (Table 3).

4. Discussion

In a large epidemiological study, Gould et al. estimated that 74 million people in the United States have foot problems and one of 2.5 people or 40% (Gould et al., 1980), (about 30 million people) have been operated upon. The most common foot problem is flatfeet (pes planus) in all age categories (Gould et al., 1980) and a common cause of flatfoot in the adult is due to dysfunction of the posterior tibial tendon (Kohls-Gatzoulis et al., 2004). The etiology of PTT dysfunction is unresolved, despite its incidence rate. The long axis of the foot is at a right angle to that of the leg so that tendons crossing the ankle must turn around bony pulleys. To understand the mechanical force on the PTT it is important to understand the biomechanical characteristics of the PTT dysfunction. Specifically, because the PTT wraps around a bony pulley, it is consistently subjected to local mechanical stress (Benjamin et al., 1995). Additional factors, such as a flexible flatfoot deformity, may lead to overuse injury, microtrauma, and degeneration of the PTT as a result of the chronic mechanical overload (Mosier et al., 1999; Mann and Thompson, 1985; Jahss, 1982; Dyal et al., 1997). Despite the wealth of information suggesting physiological factors that may contribute to PTT dysfunction and its progression, there are few reports that have investigated mechanical stresses on the PTT. In this study, we investigated the gliding characteristics of the PTT in the intact foot and the flatfoot condition, applying different loads to the PTT. These data were analyzed using a novel method, considering both gliding resistance and PTT excursion, to assess the work of friction of the PTT during passive motion of the foot.

Our study, which was designed to study more realistic tendon excursions, showed that the mean PTT excursion was 0.6 cm in the sagittal plane, while the average PTT excursions in the coronal and transverse planes were more than three times larger (2.6 and 2.3 cm, respectively). Previously Hintermann and Nigg et al. measured tendon excursion of extrinsic foot muscles resulting from selected foot movement with respect to the coronal and sagittal planes axes of the foot using 15 normal fresh frozen cadavers (Hintermann et al., 1994). They mounted specimens on a

Table 1
Median (interquartile range) of the posterior tibial tendon excursion in the intact and flatfoot condition with three tendon loading levels (0.5, 1, and 2 kg)

Motion plane		Tendon load (kg)					
		0.5		1		2	
		Intact	Flat	Intact	Flat	Intact	Flat
Sagittal	Median (interquartile range)	0.64 (0.03)	0.62 (0.09)	0.61 (0.25)	0.64 (0.24)	0.54 (0.17)	0.61 (0.15)
	P-values (intact vs. flat)	0.92		0.03		0.35	
Coronal	Median (interquartile range)	2.36 (0.48)	2.98 (0.33)	2.18 (0.75)	2.88 (0.33)	2.17 (0.73)	2.65 (0.44)
	P-values (intact vs. flat)	0.03		0.03		0.02	
Transverse	Median (interquartile range)	2.04 (0.63)	2.66 (0.61)	2.12 (0.50)	2.52 (0.40)	2.07 (0.71)	2.76 (0.64)
	P-values (intact vs. flat)	0.03		0.02		0.03	

P-values indicate the difference between PTT excursion in the intact and flatfoot conditions.

Table 2
Hindfoot and forefoot range of motion between the intact and flatfoot conditions with three PTT loading levels (0.5, 1, and 2 kg)

Motion plane		Tendon load (kg)											
		0.5				1				2			
		Hindfoot		Forefoot		Hindfoot		Forefoot		Hindfoot		Forefoot	
		Intact	Flat	Intact	Flat	Intact	Flat	Intact	Flat	Intact	Flat	Intact	Flat
Sagittal	Median (interquartile range)	45.31 (14.72)	46.09 (8.98)	60.35 (13.78)	58.18 (8.61)	44.11 (11.59)	46.33 (12.80)	50.92 (12.69)	52.61 (11.95)	44.86 (10.13)	47.07 (12.67)	53.42 (11.73)	54.69 (9.89)
	P-values (intact vs. flat)	0.50		0.74		0.02		0.02		0.13		0.50	
Coronal	Median (interquartile range)	24.20 (8.49)	25.86 (8.14)	53.62 (9.15)	56.59 (8.50)	25.21 (6.03)	25.68 (6.94)	53.54 (6.96)	61.27 (9.95)	22.45 (7.90)	27.39 (11.34)	50.31 (4.02)	56.26 (11.18)
	P-values (intact vs. flat)	0.18		0.02		0.03		0.02		0.09		0.02	
Transverse	Median (interquartile range)	12.33 (4.41)	14.22 (2.97)	34.22 (13.78)	37.32 (6.57)	11.23 (2.66)	14.59 (4.89)	32.83 (5.87)	39.83 (12.44)	12.19 (3.12)	13.99 (7.19)	32.12 (3.68)	39.70 (15.08)
	P-values (intact vs. flat)	0.18		0.24		0.02		0.02		0.02		0.02	

Significant differences were seen between the intact and flatfoot in the transverse plane in the hindfoot and in the coronal and transverse planes in the forefoot motion.

six degree-of-freedom testing apparatus and moved them in the coronal plane (9° eversion to 21° inversion) and sagittal plane (20° flexion to 30° extension). They reported that the mean PTT excursion in coronal and sagittal planes motion were 1.6 and 0.4 cm, respectively. In the sagittal and coronal plane, our study showed larger PTT excursions. These differences could be due to the differences in our experimental set up. Specifically, in the present study, the foot was not constrained by a foot plate, which may have resulted in different sagittal and coronal plane motions and a larger PTT excursion.

The effect of the increased PTT excursion with the flatfoot deformity was not considered in the work of friction calculations. The work of friction was greater in the flatfoot condition than the intact condition when the same amount of tendon excursion was considered. According to the theory of measurement of the friction force around a tendon and a pulley (An et al., 1993), the increase in gliding resistance is related to the angle of the arc of contact between the tendon and the pulley. Flatfoot deformity, one of the pre-existing conditions believed to predispose to PTT dysfunction (Kettelkamp and Alexander, 1969; Jahss, 1982; Mann and Thompson, 1985; Funk et al., 1986; Dyal et al., 1997; Mosier et al., 1999; Park et al., 2005) may increase the angle of the PTT in the retromalleolar region. This may cause an increase in the work of friction of the PTT in the coronal and transverse planes. Furthermore, the higher friction in the flatfoot condition suggests that once a patient with PTT dysfunction begins to develop a flatfoot deformity, there might be an increased potential for accelerating tendon degeneration and dysfunction because of the increased frictional force.

The results from this study, combined with knowledge gained from other studies, indicate several areas to explore in future studies in the area of PTT loading. In the present study, the work of friction increased with tendon loading and previous studies have shown that compressive forces against a pulley increase with tendon loading (An et al., 1993). While the tendon loading levels in this study were not physiologic, to prevent damage to the PTT during the 18 test conditions, the results of this study identified several proposed etiologies of PTT dysfunction that may be caused by increased loads on the PTT and will be investigated in future studies. Specifically, increased loading on the PTT may contribute to tenosynovitis seen in young athletes without inflammatory systemic disease (Supple et al., 1992), in obese individuals and in obese individuals with an existing flatfoot deformity. Young athletes with PTT tenosynovitis subject their tendons to repetitive movements with higher tendon loading than activities of daily living, which may increase the risk of injuring the PTT. Obesity is also believed to be one of the causes of PTT dysfunction (Holmes and Mann, 1992). The present study observed a significant increase in the work of friction in the coronal plane between 0.5 and 2.0 kg of loading in the intact foot, suggesting that the contribution of obesity to the development and progression of PTT dysfunction should be

Table 3
Work of friction of the posterior tibial tendon between the intact and flatfoot with three tendon loading levels (0.5, 1, and 2 kg)

Motion plane		Tendon load (kg)					
		0.5		1		2	
		Intact	Flat	Intact	Flat	Intact	Flat
Sagittal (0.3 cm of PTT excursion)	Median (interquartile range)	0.07 (0.06)	0.10 (0.08)	0.09 (0.06)	0.12 (0.06)	0.15(0.10)	0.19 (0.13)
	P-values (intact vs. flat)	0.71		0.68		0.03	
Coronal (1.5 cm of PTT excursion)	Median (interquartile range)	0.83 (0.12)	1.00 (0.56)	1.04 (0.31)	1.31 (0.41)	1.38 (0.64)	1.84 (0.73)
	P-values (intact vs. flat)	0.03		0.02		0.03	
Transverse (1.5 cm of PTT excursion)	Median (interquartile range)	0.86 (0.13)	1.00 (0.17)	1.08 (0.44)	1.19 (0.36)	1.59 (0.64)	1.62 (0.66)
	P-values (intact vs. flat)	0.02		0.02		0.03	

There was a significant increase in the work of friction between the intact and flatfoot conditions in the coronal and transverse planes.

studied. Additionally, we observed a greater increase in the work of friction as tendon loading increased in the flatfoot condition. We also know that flexible flatfoot deformity may predispose people to developing PTT dysfunction (Holmes and Mann, 1992). Therefore, it is possible that obesity in combination with a flexible flatfoot deformity may increase the risk of developing PTT dysfunction. These proposed etiologies will be modeled with physiologic tendon loading and the application of an axial load in future studies.

There are several factors that were not considered in this study. All of the specimens tested in this study were male, while the majority of patients that develop chronic PTT dysfunction are female. The reason why more women than men develop PTT dysfunction is unknown and this method should be used to investigate female limbs in a future study. Axial loads were not applied to the tibia in this study due to limitations of the experimental set up. The small, yet statistically significant increase in hindfoot and forefoot range of motion may have been caused by the lack of an axial load. Because the arch will collapse more with an axial load, we hypothesize that the work of friction will increase more with the application of an axial load. This hypothesis will be tested in future studies. Loads were not applied to adjacent tendon such as the flexor hallucis longus and flexor digitorum longus, which are located close to the PTT beneath the flexor retinaculum. Other anatomic structures, such as a tight flexor retinaculum and shallow medial malleolar groove, should be considered because they might constrict the PTT in its excursion behind the medial malleolus. The loads applied to the PTT were minimal due to the number of trials and the limitations of our force transducers. The application of an axial load, additional tendon loading, and measuring the gliding resistance using automated passive motion will be considered in future studies.

The increased work of friction observed with increased PTT loading and in the flatfoot condition suggests that PTT degeneration may be accelerated when a condition affecting the forces on the PTT occurs. Therefore, aggressive treatment in the early stages of PTT dysfunction should be considered due to a limited ability of the PTT to self-repair from tissue injury because of its avascular nature (Mosier et al., 1999; Pufe et al., 2003). Additionally, the effects of non-operative treatment limiting the coronal and transverse plane motions on the work of friction should be investigated further. This may decrease the degenerative effects caused by the increased gliding resistance, while permitting sagittal motion to allow for more normal ambulation.

5. Conclusion

This study presented a novel method, considering both gliding resistance and excursion, to assess the work of friction in the PTT during passive motion of the foot. The work of friction increased with tendon loading and was greater in the flatfoot condition than the intact condition. The work of PTT friction, which was higher in the flatfoot condition and with increasing tendon loading levels, is thought to be one of the most important etiologies of PTT dysfunction.

Acknowledgements

We gratefully acknowledge the financial support from Mayo foundation. We thank Takaaki Hirano, M.D., Ph.D. for his assistance.

References

An, K.N., Berglund, L., Uchiyama, S., Coert, J.H., 1993. Biomed. Sci. Instrum. 29, 1–7.

Anderson, J., Harrington, R., Ching, R., Tencer, A., Sangeorzan, B., 1997. Foot Ankle Int. 18, 705–709.

Augustin, J.F., Lin, S.S., Berberian, W.S., Johnson, J.E., 2003. Foot Ankle Clin. 8, 491–502.

Benjamin, M., Qin, S., Ralphs, J.R., 1995. J. Anat. 187 (Pt 3), 625–633.

Chen, Y.J., Hsu, R.W., Liang, S.C., 1997. J. Bone Joint Surg. Am. 79, 1791–1798.

Coert, J.H., Uchiyama, S., Amadio, P.C., Berglund, L.J., An, K.N., 1995. J. Hand Surg. [Br] 20, 573–577.

Cozen, L., 1965. Clin. Orthop. 42, 101–102.

Dyal, C.M., Feder, J., Deland, J.T., Thompson, F.M., 1997. Foot Ankle Int. 18, 85–88.

Frey, C., Shereff, M., Greenidge, N., 1990. J. Bone Joint Surg. Am. 72, 884–888.

Funk, D.A., Cass, J.R., Johnson, K.A., 1986. J. Bone Joint Surg. Am. 68, 95–102.

Gould, N., Schneider, W., Ashikaga, T., 1980. Foot Ankle 1, 8–10.

Hintermann, B., Nigg, B.M., Sommer, C., 1994. Foot Ankle Int. 15, 386–395.

Holmes Jr., G.B., Mann, R.A., 1992. Foot Ankle 13, 70–79.

Jahss, M.H., 1982. Foot Ankle 3, 158–166.

Kettelkamp, D.B., Alexander, H.H., 1969. J. Bone Joint Surg. Am. 51, 759–764.

Kitaoka, H.B., Luo, Z.P., An, K.N., 1998. Foot Ankle Int. 19, 203–207.

Kohls-Gatzoulis, J., Angel, J.C., Singh, D., Haddad, F., Livingstone, J., Berry, G., 2004. BMJ 329, 1328–1333.

Mann, R.A., Thompson, F.M., 1985. J. Bone Joint Surg. Am. 67, 556–561.

Mosier, S.M., Lucas, D.R., Pomeroy, G., Manoli 2nd, A., 1998. Foot Ankle Int. 19, 520–524.

Mosier, S.M., Pomeroy, G., Manoli 2nd, A., 1999. Clin. Orthop., 12–22.

Myerson, M.S., Corrigan, J., 1996. Orthopedics 19, 383–388.

Myerson, M., Solomon, G., Shereff, M., 1989. Foot Ankle 9, 219–225.

Park, H.G., Sim, J.A., Koh, Y.H., 2005. Foot Ankle Int. 26, 184–186.

Petersen, W., Hohmann, G., Stein, V., Tillmann, B., 2002. J. Bone Joint Surg. Br. 84, 141–144.

Pufe, T., Petersen, W., Kurz, B., Tsokos, M., Tillmann, B., Mentlein, R., 2003. J. Orthop. Res. 21, 610–616.

Sobel, M., 1992. Am. J. Knee Surg. 5, 59–64.

Supple, K.M., Hanft, J.R., Murphy, B.J., Janecki, C.J., Kogler, G.F., 1992. Semin. Arthritis Rheu. 22, 106–113.

Uchiyama, S., Coert, J.H., Berglund, L., Amadio, P.C., An, K.N., 1995. J. Orthop. Res. 13, 83–89.

Uchiyama, S., Amadio, P.C., Coert, J.H., Berglund, L.J., An, K.N., 1997. J. Bone Joint Surg. Am. 79, 219–224.

Uchiyama, E., Fujii, T., Kitaoka, H.B., Luo, Z., Momose, T., An, K., 2000. ORS Trans. 25, 0191.

Woods, L., Leach, R.E., 1991. Am. J. Sports Med. 19, 495–498.

Zhao, C., Amadio, P.C., Zobitz, M.E., Momose, T., Couvreur, P., An, K.N., 2001. Clin. Biomech. 16, 696–701.