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Changes in gait associated with acute stage II posterior tibial tendon dysfunction

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Abstract

The purpose of this study was to examine differences in gait mechanics between patients with acute stage II PTTD and healthy volunteers. Hindfoot and midfoot kinematics, plantar foot pressures and electromyographic (EMG) activity of the posterior tibialis, gastrocnemius, anterior tibialis and the peroneals were measured in five patients with acute stage II PTTD. Kinematics and kinetics were compared to a database of 20 healthy volunteers. EMG and plantar pressure data were obtained from five healthy volunteers. Hindfoot moments and powers were also calculated. The center of pressure excursion index (CPEI) was calculated from the plantar pressures. Significant differences were observed between the two groups, which confirmed clinical observations. Limited hindfoot eversion and increased midfoot external rotation occurred during the first and third rockers. The EMG data suggested that tendon dysfunction in the posterior tibialis is associated with compensatory activity, not only in its antagonists (the peroneals), but also in the anterior tibialis and the gastrocnemius. These data suggest that non-operative treatment of patients with PTTD should consider minimizing the activity of the posterior tibialis as well as the peroneals, the anterior tibialis and the gastrocnemius.

Keywords: Posterior tibial tendon dysfunction; Flatfoot deformity; Gait; Plantar pressure; EMG

1. Introduction

Posterior tibial tendon dysfunction (PTTD) is the most common cause of adult acquired flatfoot deformity. This dysfunction typically begins with a paratenonitis of the tendon which may lead to synovitis, intrasubstance tearing and tendinosis. A total rupture of the tendon is possible, however, not prevalent. In the early stages of PTTD, the hindfoot is flexible. As the pathologic process progresses, the hindfoot becomes rigid, and the subtalar, midfoot and tibiotalar joints may develop arthritis. PTTD is usually characterized into four stages. Patients presenting with stage I PTTD exhibit paratenonitis with pain over the posterior tibial tendon (PTT), typically just posterior and inferior to the medial malleolus. There is generally no loss of strength, radiographs are usually normal and the hindfoot is flexible. In stage II PTTD, the PTT elongates and intrasubstance tears develop, leading to a unilateral flexible flatfoot deformity [1]. Patients typically cannot perform the ''single heel rise test'' in the affected limb and degenerative arthritis of the hindfoot may develop. In stage III PTTD, the hindfoot is rigid and degenerative arthritis can be advanced in the subtalar and midfoot joints. Further postural changes take place, which include a varus forefoot and calcaneofibular impingement [1]. Stage IV PTTD includes the rigid hindfoot with the addition of a valgus talus in the ankle mortise. This is thought to lead to degenerative arthritis at the tibiotalar joint [2].

Despite the wealth of information on the classification and operative [2–10] and non-operative [2,11–13] treatment of patients with PTTD, there are few studies examining the effects of PTTD on gait [14–16]. Changes in step length, cadence, velocity and maximum sagittal

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ankle joint powers at push-off were observed pre- and post-operatively in patients with stage II PTTD [15], but the differences between healthy and PTTD gait were not studied and a foot specific marker set was not used. Increased plantarflexion and eversion of the hindfoot throughout the gait cycle was observed and increased dorsiflexion and restricted eversion in late stance was measured in the forefoot when patients with stage II PTTD were compared with healthy individuals [14]. Reduced moments at the hip, knee and ankle were observed in a similar population using a full body marker set [16]. To date, a comprehensive motion analysis study, including a segmental foot model of kinematics and kinetics, electromyographic activity and plantar foot pressures comparing patients with stage II PTTD with healthy volunteers has not been completed. The purpose of this study was to examine the differences in foot and ankle mechanics between patients with acute stage II PTTD and healthy asymptomatic volunteers.

2. Materials and methods

Five women that presented to a podiatrist with acute stage II PTTD (age 69 ± 7 years, range 60–75 years, body mass index (BMI) 29 ± 1 kg/m^2) were included in this study. The patients' kinematics were compared with those from a database of 20 healthy asymptomatic volunteers previously collected in our laboratory (age 46 ± 14 years, range 27–65 years, BMI 25 ± 4 kg/m^2, 10 men and 10 women). EMG and plantar pressure data were compared to data from five healthy asymptomatic volunteers (age 27 ± 4 years, range 24–34 years, BMI 24 ± 3 kg/m^2, two men and three women). The patients with acute stage II PTTD had difficulty performing a single heel rise test and there was tenderness over the PTT. Intrasubstance tear of the tendon was confirmed by a musculoskeletal radiologist with a MRI. This study was approved by the Institutional Review Board and written informed consent was obtained.

2.1. Kinematics and kinetics

Three-dimensional foot and ankle gait mechanics were collected to quantify changes that were observed clinically in acute stage II PTTD. A custom 11 marker three-segment model (Fig. 1) was used. This biomechanical model defined local coordinate systems in the lower leg, calcaneus and midfoot [17]. The midfoot segment was defined by markers placed on the proximal and distal ends of the first and fifth metatarsals. The calcaneus was defined by three markers placed on the calcaneus. The tibia was defined with markers placed on the medial and lateral malleolus and anterior along the long axis of the tibia. A ten camera Real-Time ExpertVision System (Motion Analysis Corporation, Santa Rosa, CA) was used to collect data at 120 Hz. A static reference position was collected with the patients in a

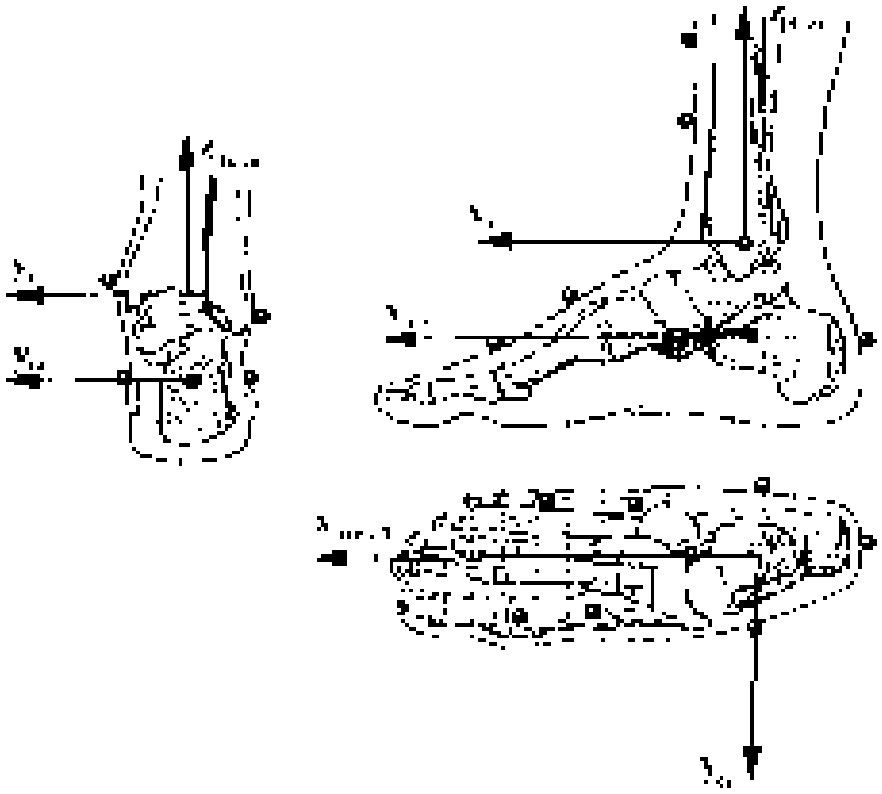

Fig. 1. Custom 11 marker, three-segment foot model, used to measure hindfoot and midfoot kinematics.

relaxed standing position, which defined the neutral position of the foot for the kinematic graphs. The motion of the hindfoot was defined as the motion of the calcaneus relative to the lower leg and the motion of the midfoot was defined as the motion of the midfoot (i.e., the segment defined by markers placed on the first and fifth metatarsals) relative to the calcaneus. Euler angles were calculated using a custom software program (Matlab 7.0, MathWorks, Natick, MA) using a 2–1′–3″ rotation sequence [18].

Kinetic data were collected at 600 Hz from four force plates (two Kistler model 9281B, Amherst, NY, and two Advanced Medical Technology Inc., model BP400600, Watertown, MA). Data from the foot marker set and from the force plates were used to calculate ankle joint moments and powers. The ankle joint center was defined as the midpoint of the markers on the medial and lateral malleolus. These data, combined with the force plate data (i.e., center of pressure and ground reaction forces) were used to calculate the ankle joint moments in a custom software program (Matlab 7.0, MathWorks) [19]. The powers were calculated by multiplying the moment by the angular velocity.

2.2. Electromyographic activity

EMG data were collected to quantify changes in muscle activity in patients with acute stage II PTTD. Five channels of EMG data were collected using commercially available EMG collection hardware (MA-300, Motion Lab Systems, Baton Rough, LA) during the entire gait cycle. Surface EMG electrodes were placed over the muscle bellies of the anterior tibialis (AT), medial gastrocnemius (gastroc), peroneus brevis (PB) and peroneus longus (PL). Placement was confirmed by

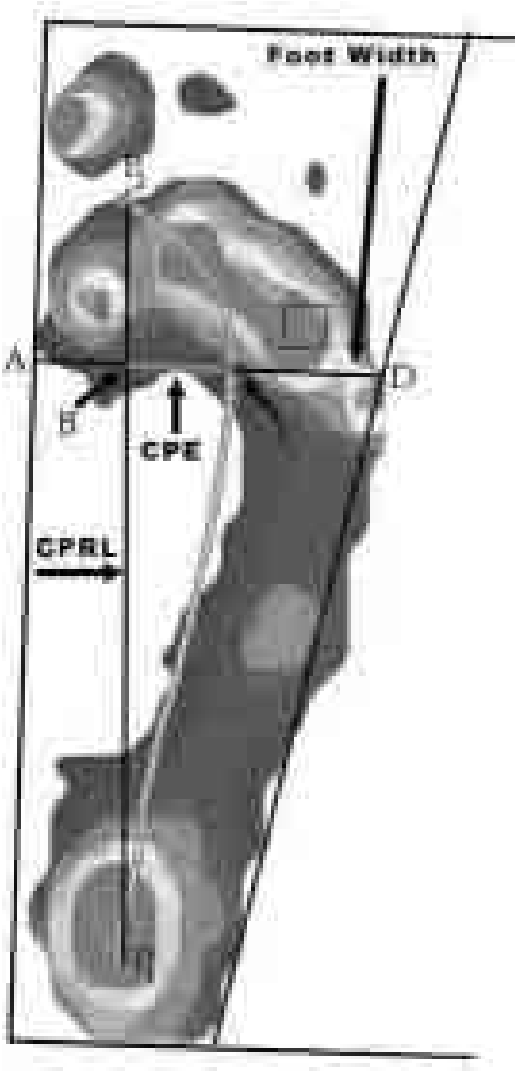

Fig. 2. Center of pressure excursion index in a healthy volunteer. The center of pressure reference line (CPRL) was drawn from the first to the last center of pressure data point (i.e., P_1 to P_2). The distance from this line (P_1P_2) to the point where the center of force trajectory is maximal was measured. This length was defined as the center of pressure excursion (CPE). The width of the foot was measured at the level of the CPE and the CPE was normalized by the foot width, defining the CPEI [24,25].

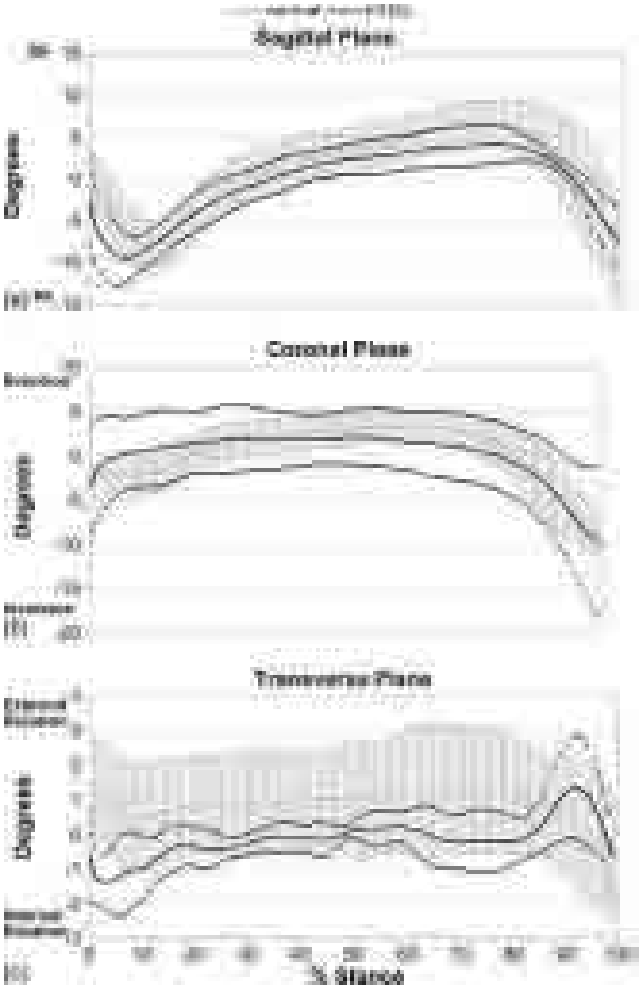

Fig. 3. Hindfoot kinematics in healthy volunteers and patients with stage II PTTD. (a) In patients with stage II PTTD the first rocker was shorter since maximum plantarflexion occurred sooner than in healthy volunteers. (b) In the coronal plane, a non-significant decrease in eversion was observed in patients with stage II PTTD. (c) Clinically insignificant changes were also measured throughout stance phase in the transverse plane.

muscle contraction against manual resistance with visual and audio feedback. Because only differences in amplitude were noted when fine wire EMG data were collected from the PB and PL [20] and the pain caused from fine wire EMG could alter gait [21], we chose to collect surface EMG from the peroneals. A fine-wire indwelling EMG electrode was placed in the posterior tibialis (PT) using the single needle technique [22]. The subjects were asked to plantarflex and invert the ankle, while manual resistance was applied in the direction of dorsiflexion and eversion to verify electrode placement using visual and audio feedback. The participants were also asked to flex their toes to confirm that the electrode was not placed in the flexor digitorum longus muscle. Additionally, electrical stimulation was used to confirm electrode placement by passing an electrical current through the wires connected to the EMG electrode using a muscle stimulator (Grass Instrument Division Model S48J Stimulator, Division of Astro-Med Inc., West Warwick, RI). The foot was positioned such that the effect of gravity was not present and the posterior tibial tendon was palpated during stimulation. If the foot plantarflexed and inverted when stimulation was applied and there was no toe flexion, proper electrode placement was confirmed.

For each muscle, a quiescent trial and maximum voluntary contraction (MVC) were collected. Quiescent data were collected while the volunteer was sitting in a chair with the muscles relaxed. Visual feedback was used to confirm that there was no muscle activity before the quiescent trial was collected. The MVC test consisted of a seven second data collection period in which the subjects actively contracted their muscle for the first four seconds, while listening to audio feedback, which increased in volume with electrical activity. In the final three seconds of the data collection period, maximal manual resistance was applied in the direction opposite to the muscle's action to increase the number of activated motor units. EMG data were bandpass filtered from 60 to 1000 Hz before they were sampled at 2400 Hz. Additionally, EMG data were time synchronized with kinematic data using an A/D card (National Instruments, Austin, TX). All data were rectified and filtered using a 6 Hz fourth order Butterworth lowpass filter. MVC levels were reported as the highest mean level of a 100 ms moving average window with 90% overlap [23]. EMG data were processed in a custom software program (Matlab 7.0, MathWorks) and reported as a percentage of MVC. If the EMG signal was less than the quiescent value, it was assumed to be zero.

2.3. Foot structure testing

Plantar foot pressure measurements were collected by a physical therapist using an F-scan plantar pressure assessment system (Tekscan, Inc., South Boston, MA). The F-scan sensor was taped to the plantar aspect of the foot and the patients walked barefoot at a self-selected pace over a level tile floor. From these data, the center of pressure excursion index (CPEI) was calculated [24,25]. Briefly, a line defined as the center of pressure reference line (CPRL) was drawn from the first to the last center of pressure data point (i.e., P_1 to P_2) (Fig. 2). The line $\overline{AD}$ was defined as the width of the

Table 1
Peak values of kinematics in healthy volunteers and patients with PTTD

Segment	Plane of motion	Foot rocker	Normal		PTTD	
			Time (% stance)	Rotation (°)	Time (% stance)	Rotation (°)
Hindfoot	Sagittal	First	$13 \pm 2^{+}$	-8 ± 3	$10 \pm 3^{+}$	-10 ± 3
		Second	76 ± 9	7 ± 3	73 ± 12	5 ± 3
		Third	100 ± 0	-8 ± 6	100 ± 0	-12 ± 3
	Coronal	First	4 ± 2	4 ± 4	2 ± 3	4 ± 3
		Second	60 ± 11	4 ± 2	50 ± 12	2 ± 3
		Third	99 ± 2	-9 ± 3	100 ± 2	-10 ± 8
	Transverse	First	12 ± 8	$0.1 \pm 2^{*}$	5 ± 3	$-2 \pm 1^{*}$
		Second	47 ± 20	$2 \pm 2^{*}$	43 ± 25	$1 \pm 0.4^{*}$
		Third	90 ± 9	1 ± 1	94 ± 2	2 ± 1
Midfoot	Sagittal	First	$19 \pm 17^{*}$	1 ± 3	$5 \pm 3^{*}$	-0.2 ± 5
		Second	33 ± 15	0 ± 2	16 ± 6	-3 ± 5
		Third	99 ± 2	$-15 \pm 6^{+}$	99 ± 1	$-8 \pm 7^{+}$
	Coronal	First	6 ± 4	$-3 \pm 2^{+}$	6 ± 4	$-6 \pm 3^{+}$
		Second	48 ± 20	3 ± 2	53 ± 17	3 ± 1
		Third	89 ± 10	$-2 \pm 2^{+}$	88 ± 7	$-4 \pm 5^{+}$
	Transverse	First	2 ± 1	$-3 \pm 2^{+}$	4 ± 2	$-1 \pm 1^{+}$
		Second	54 ± 8	2 ± 1	66 ± 16	1 ± 1
		Third	99 ± 3	$-9 \pm 3^{*}$	99 ± 2	$-3 \pm 2^{*}$

Values are reported at mean ± standard deviation. $^{+}$Indicates a notable trend ($p = 0.05$–0.02) and *indicates where the significant differences occurred ($p < 0.017$). The values in this table correspond to the mean peak value for each component of stance phase of gait. Because the peak rotations did not occur at the same percentage of the stance phase for each participant, the numbers in this table do may correspond to the peak of the mean curves in Figs. 3 and 4.

foot along the line separating the anterior third of the foot from the posterior two-thirds of the foot. The point B was defined as the intersection of the CPRL and $\overline{AD}$. The point C was defined as the intersection of $\overline{AD}$ and the center of force trajectory. The line $\overline{BC}$ was referred to as the center of pressure excursion (CPE). The CPI was then normalized by the width of the anterior third of the foot (line $\overline{AD}$), defining the center of pressure excursion index (CPEI).

$$\text{CPEI} = \frac{\overline{\text{CPE}}}{\overline{\text{AD}}} \times 100 \quad (1)$$

2.4. Data analysis

The kinematic data were separated into three rockers for analysis to provide a comprehensive comparison of the differences between kinematics in healthy volunteers and patients with stage II PTTD. Each rocker was defined using the sagittal plane hindfoot data. The end of the first rocker was defined as maximum hindfoot plantarflexion, thus identifying the end of the loading response. The end of the second rocker was defined as maximum hindfoot dorsiflexion, thus indicating the beginning of limb advancement (i.e., third rocker).

A two-sample (or independent samples) *t*-test was calculated using JMP 5.1 (SAS Institute, Cary, NC). A Bonferroni correction was applied to the kinematics to preserve the experimentwise error rate caused by examining three segments of stance phase. Accordingly, a significance level of $p < 0.017$ was used for these data. A significance level of $p < 0.05$ was used for the moments, powers, EMG and plantar pressure data because only the peaks were considered when analyzing these data. A power analysis was completed when the significance level was not met using a custom program written in Mathcad (Mathsoft Engineering & Education, Inc., Cambridge, MA).

3. Results

The mean walking velocity for healthy volunteers was 1.2 ± 0.1 m/s and 1.1 ± 0.2 m/s in patients with acute stage II PTTD. There were no significant differences in walking velocities (81% power). In the sagittal plane, there was a trend ($p = 0.035$) indicating that the end of the first hindfoot rocker occurred sooner in patients with stage II PTTD (Table 1, Fig. 3a). Hindfoot kinematics also demonstrated no difference (97% power) in eversion during the second rocker (Fig. 3b). In the transverse plane, patients with stage II PTTD had increased internal rotation during first rocker ($p = 0.007$) and decreased external rotation in second rocker ($p = 0.009$) (Fig. 3c). While these differences were statistically significant, they were not considered to be clinically significant and were within the error range of the measurement technique.

Significant differences between the midfoot kinematics in PTTD patients and healthy volunteers were observed in the sagittal and transverse planes (Fig. 4). In the sagittal plane, the location of peak dorsiflexion in the first rocker occurred earlier in patients ($p = 0.01$). There was a non-significant difference ($p = 0.049$) in the value of maximum plantarflexion in the third rocker (Fig. 4a). Patients had a non-significant increase in inversion ($6 \pm 3^\circ$ versus $3 \pm 2^\circ$) in the first ($p = 0.0218$) rocker and a trend ($p = 0.05$) toward an increased midfoot range of motion ($9 \pm 3^\circ$ versus $6 \pm 2^\circ$) (Fig. 4b). Patients had restricted internal rotation ($3 \pm 2^\circ$ versus $9 \pm 3^\circ$) in the third rocker ($p = 0.0003$) when compared to healthy volunteers (Fig. 4c). Additionally, the midfoot range of motion in the transverse plane showed that the patients' midfoot range of motion was significantly

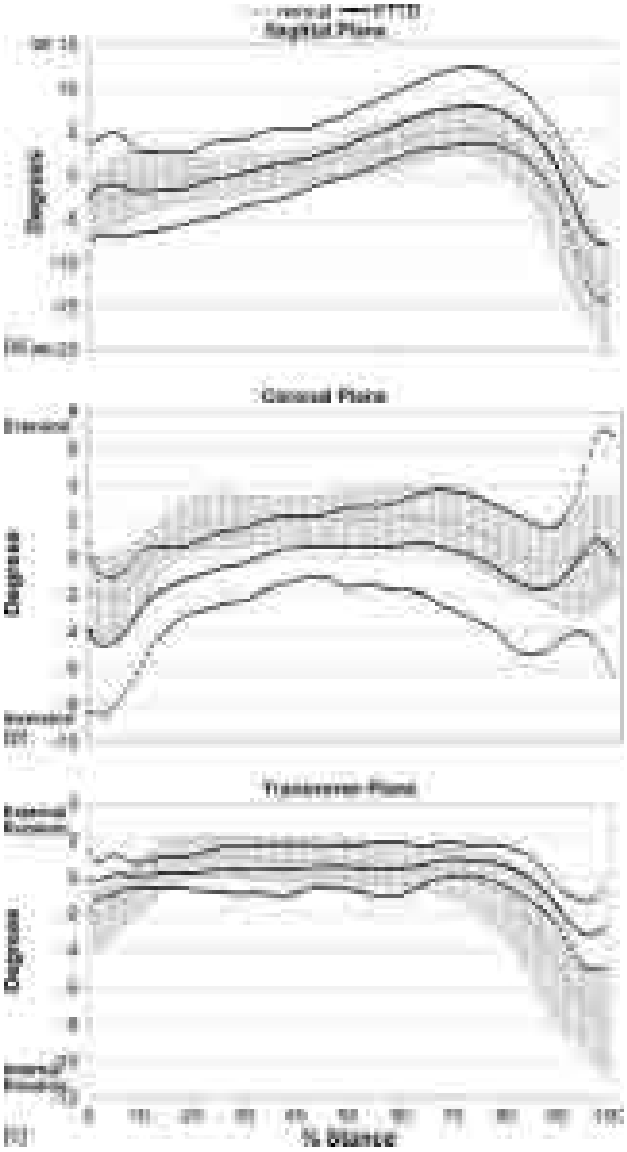

Fig. 4. Midfoot kinematics in healthy volunteers and patients with stage II PTTD. In patients with stage II PTTD: (a) plantarflexion was restricted during the third rocker, (b) inversion was higher in patients during the first rocker and (c) internal rotation was limited in the first and third rockers.

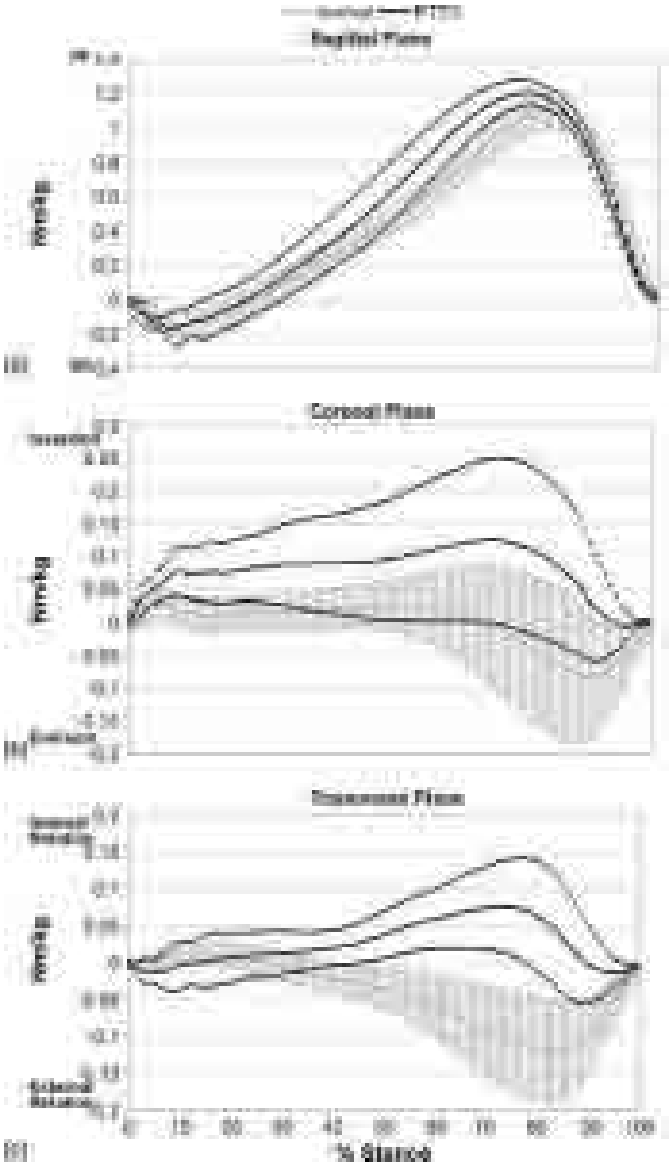

Fig. 5. Internal hindfoot moments (Nm/kg) in patients with stage II PTTD showed: (a) no change in the sagittal plane, (b) an inversion moment, while healthy volunteers had an eversion moment and (c) internal rotation moments, while healthy volunteers had external rotation moments.

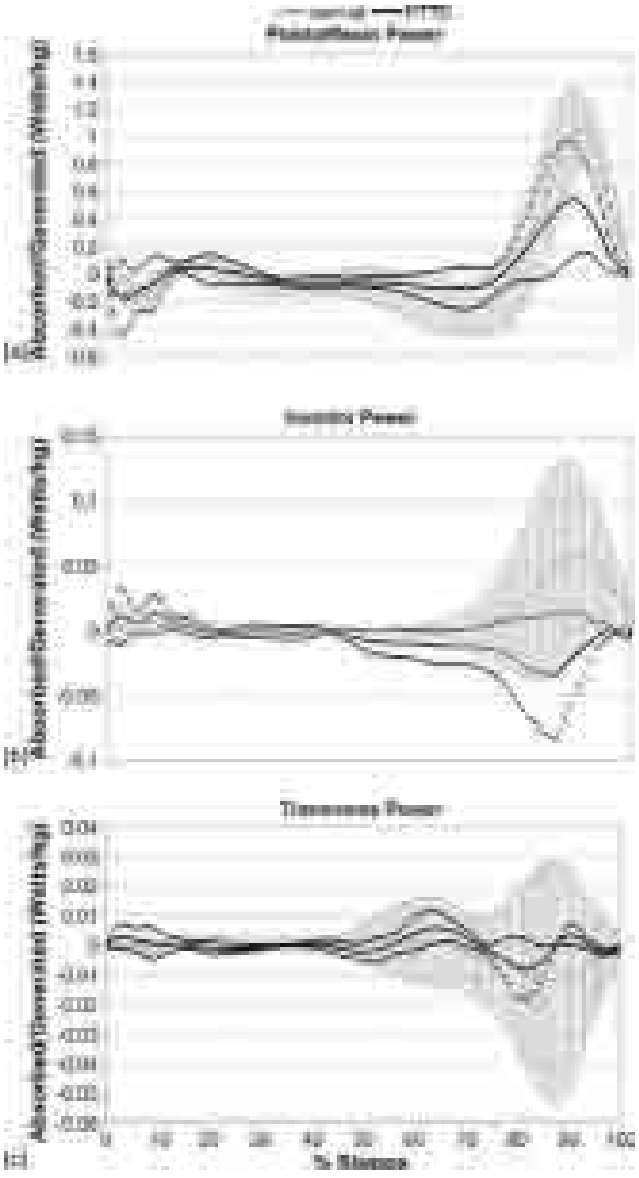

Fig. 6. There was a significant decrease in the internal hindfoot sagittal plane power (a) in patients with stage II PTTD.

decreased ($5 \pm 2^{\circ}$) when compared to healthy volunteers ($10 \pm 3^{\circ}$) ($p < 0.001$).

Stage II PTTD patients had an inversion moment in the third rocker (-0.1 ± 0.1 Nm/kg), while healthy volunteers had an eversion moment (0.08 ± 0.1 Nm/kg) ($p = 0.019$) (Fig. 5). The peak moment occurred at $91 \pm 4\%$ stance in healthy volunteers and at $75 \pm 8\%$ stance in patients with stage II PTTD ($p = 0.008$). From 30% stance to terminal stance, the ankle moments were negative (i.e., indicating an external rotation moment) in healthy volunteers and positive (i.e., internal rotation moment) in patients ($p < 0.001$). There was no significant difference in sagittal plane moments (power = 90%). Sagittal plane powers were significantly lower in PTTD patients during the third rocker ($p = 0.05$) despite the similar walking velocity (Fig. 6). There were no significant differences between healthy and PTTD patients in the coronal plane (power = 100%) or transverse plane powers (power = 56%).

When the EMG from patients with acute stage II PTTD were compared with healthy volunteers, there were magnitude differences in the posterior tibialis, anterior tibialis and gastrocnemius and temporal changes in the peroneus longus and peroneus brevis (Fig. 7). In the posterior tibialis, the peak EMG activity during the second half of stance phase was significantly greater in PTTD patients than healthy volunteers ($p = 0.025$). The peak EMG value at approximately 70% of the gait cycle in the anterior tibialis was significantly greater in patients than in healthy volunteers ($p = 0.038$). The gastrocnemius had

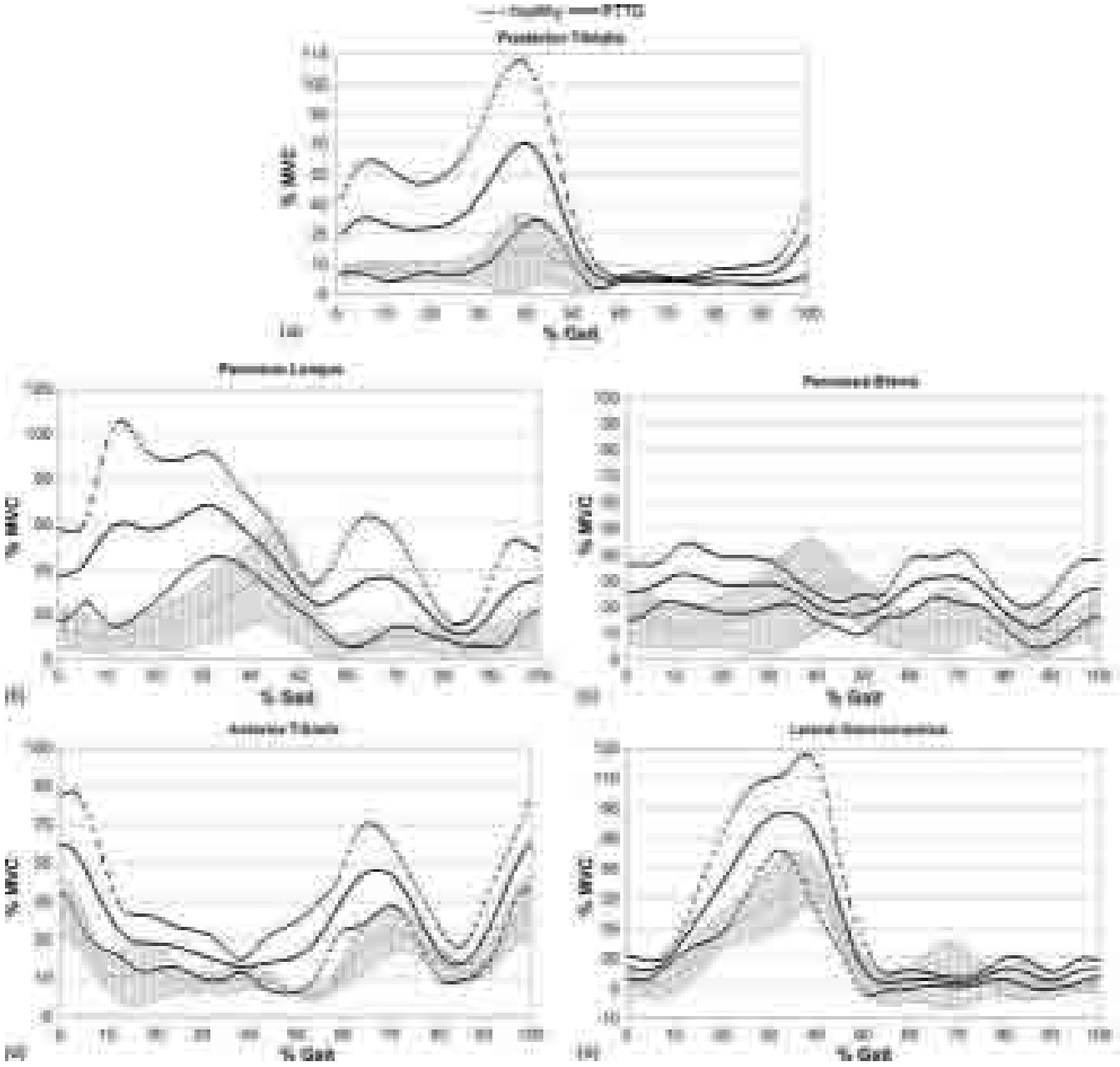

Fig. 7. EMG data of the foot and ankle muscles during gait. Patients with stage II PTTD had an increase in magnitude of the posterior tibialis (a), anterior tibialis (d), and the gastrocnemius (e). The peroneus longus (b) exhibited increased activity and magnitude and the peroneus brevis (c) had phase reversal when compared to healthy volunteers.

increased activity during stance phase in patients with PTTD ($p = 0.004$). Patients had increased and prolonged EMG activity in the peroneus longus ($p = 0.021$). Finally, the EMG activity in the peroneus brevis exhibited phase reversal between healthy volunteers and patients with PTTD and prolonged activity during swing phase ($p = 0.003$).

Plantar pressure data exhibited a medial shift of the center of pressure in patients with PTTD (Figs. 2 and 8) and a significant decrease in the CPEI ($p = 0.019$). The CPEI was $20 \pm 6\%$ in healthy volunteers and $10 \pm 4\%$ in patients with PTTD.

4. Discussion

The most common clinical observations used to diagnose stage II PTTD are the inability to perform a single heel rise and forefoot abduction (i.e., the too many toes sign) [1]. The inability to perform a single heel rise was quantified in this study by decreased hindfoot eversion, decreased plantarflexion in terminal stance, an increased inversion moment and decreased sagittal plane powers. The ''too many toes'' sign was quantified with decreased midfoot internal rotation in the third rocker. EMG data identified changes in the

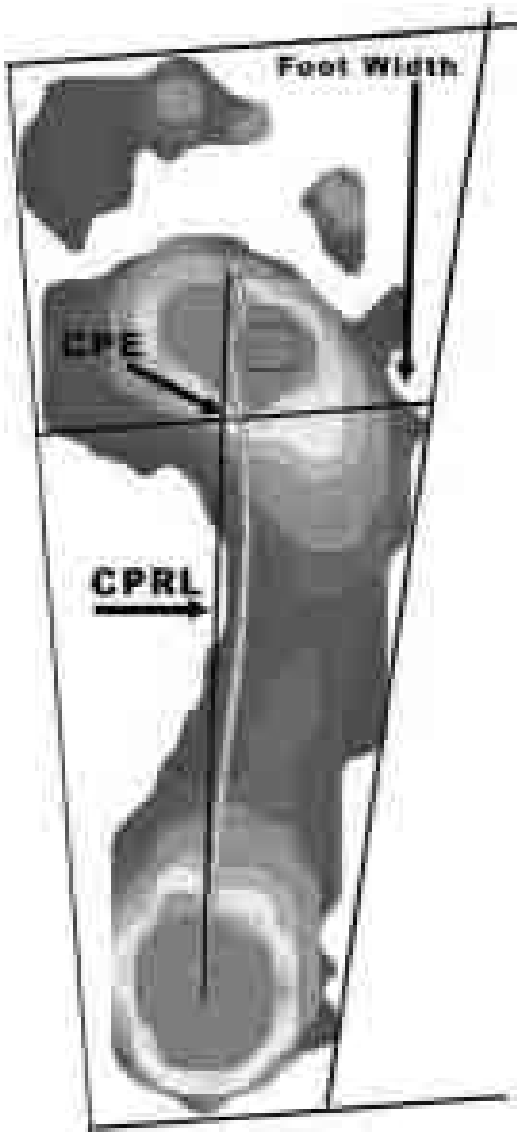

Fig. 8. Center of pressure excursion index (CPEI) data from a patient with stage II PTTD. There was a significant decrease in the CPEI in patients with stage II PTTD.

activity of the peroneals, the anterior tibialis and gastrocnemius along with increased EMG in the posterior tibialis. Patients with stage II PTTD had a shorter first rocker characterized by earlier maximum hindfoot plantarflexion (Fig. 3). The patient data indicated an insignificant increase in plantarflexion. These findings are similar to the study by Johnson and Harris that compared ten patients with stage II PTTD with a database of healthy, asymptomatic volunteers [14].

Clinical observations suggest that a dysfunctional posterior tibialis does not transmit enough force to sufficiently evert the calcaneus [1]. This observation was supported by the increased inversion moment, decrease in sagittal plane powers and decreased hindfoot eversion in patients with stage II PTTD. The change in coronal plane moments suggest that the center of pressure shifts medially, which was confirmed with the plantar pressure data (Figs. 2 and 8). The significant decrease in hindfoot sagittal plane powers was similar to the report that patients with stage II PTTD have significantly lower ankle powers pre-operatively than post operatively [15]. Tome et al. reported significantly greater calcaneal eversion in patients with stage II PTTD compared to age matched healthy volunteers [26], while this study observed an insignificant increase in hindfoot eversion. Conversely, Johnson and Harris observed increased hindfoot eversion. These differences may be due to differences in the definition of neutral position. Tome et al. referenced their data to the subtalar joint in neutral position. The definition of neutral position in Johnson and Harris' study was not reported. In the present study, a relaxed standing neutral position was obtained, without placing the subtalar joint into neutral position or supporting the arch of the foot. It is probable that we would observe a significant decrease in hindfoot eversion if our neutral position was collected in subtalar joint neutral with arch support.

The midfoot kinematics quantified a flexible flatfoot deformity and ''too many toes'' sign used to clinically diagnose PTTD [1]. Increased midfoot dorsiflexion and inversion as, a result of midfoot flattening in late stance indicating a flexible flatfoot deformity, was observed in this study (Fig. 4). Additionally, increased transverse plane moments occurred as the foot collapsed during second rocker. This change in the moment increased the hindfoot stress, which may lead to the formation of osteoarthritis that is found in stage II PTTD [1]. The ''too many toes'' sign was quantified with a decrease in internal rotation during the third rocker as well as the lack of medial shift of the center of pressure (measured from the plantar pressure data). Similarly, the study by Johnson and Harris observed increased midfoot dorsiflexion and a varus shift in forefoot kinematics at toe off [14].

Patients with acute stage II PTTD had significant changes in the electromyography activity of the muscles crossing the ankle joint. The differences in PT, PB and gastrocnemius EMG activity may be associated with a valgus deformity, while the change in activity of the peroneus longus and anterior tibialis may be uniquely associated with acute stage II PTTD. Keenan et al. collected fine-wire EMG data in patients with rheumatoid arthritis (RA) with and without a valgus deformity [27]. They found a significant increase in the magnitude of the posterior tibialis EMG, but no change in timing. These findings are similar to ours. Keenan et al. suggested a valgus deformity caused by RA may cause the posterior tibialis to work harder to support the arch. This situation may also happen in patients with stage II PTTD. However, it is also possible that the MVC in these patient populations was limited due to pain. Specifically, when the gait analysis was performed, the patients with acute stage II PTTD reported pain. Similarly, the patients with a valgus deformity in the study by Keenan et al. had a significantly greater pain score in the hindfoot than the RA patients without a valgus deformity. In RA patients with a valgus

deformity, the preoneus brevis had increased magnitude, perhaps to help minimize the flatfoot deformity, but there was no change in the EMG of the peroneus longus [27]. In the stage II PTTD patients, the peroneus brevis exhibited phase reversal (but no change in magnitude), while the peroneus longus had premature and prolonged activity. The compensatory EMG activity in the peroneals indicates that they were probably acting to minimize the acquired flatfoot deformity differently than RA patients with a valgus deformity. The change in the activation pattern of the peroneals may lead to the intrasubstance tears that are frequently present in the peroneals in stage II and stage III PTTD. In patients with stage II PTTD, the anterior tibialis had a significant increase in activity. Because the AT is an antagonist to the PL, the patients with stage II PTTD may have had to increase the AT activity to make the first ray more stable. In the patients with RA, there was no change in the AT activity. However, there was also no change in the peroneus longus activity in the RA patients with a valgus deformity. This suggests that the changes in the peroneals and the AT are related to PTTD, not just a valgus deformity. Studies with a larger patient population must be conducted to improve our understanding of these changes in the EMG data. Finally, patients with stage II PTTD had increased activity in the gastrocnemius, which commonly occurs with foot and ankle pathologies. Similarly, the gastrocnemius activity of RA patients both with and without a valgus deformity indicated that they had increased activity over healthy volunteers.

A significant decrease in the CPEI was observed in patients with stage II PTTD when compared to healthy volunteers. CPEI alone was not able to detect the differences between rectus and planus foot types, however it was postulated that when a pathology was present that the CPEI would be able to detect differences between healthy and pathologic feet [24]. This study demonstrated that the CPEI was sensitive enough to quantify the differences between plantar pressures in feet from healthy volunteers and patient with stage II PTTD.

The three-segment foot model assumed that the midfoot was a rigid structure. In an in vitro study comparing kinematics with skin markers and bone pins, the root mean square error was 3° [17]. Therefore, any differences in rotations that are less than 3° must be carefully interpreted, even when there is a statistically significant difference between the two measurements. The small sample size and the lack of age, BMI and gender matching were additional limitations to this study. A recent study concluded that aging does not affect voluntary activation of the ankle dorsiflexors during isometric, concentric and eccentric contractions [28]. Therefore, the age difference in our PTTD patients and healthy volunteers should not affect the reported differences in the EMG data. The effect of age on kinematic gait data is a subject of controversy. One study examining the difference in age found that both healthy and adult men had similar gait characteristics [29], while a similar study found significant differences between young and old women [30]. Another study reported significantly different kinematic and kinetic variables between older and younger populations, however the absolute differences were small [31]. A future study of PTTD should include a larger patient population and age matched participants.

5. Conclusions

This study quantified changes in muscle activity that are associated with the clinically observed alterations in joint mechanics of patients with stage II PTTD. Changes in kinematics quantified the ‘‘too many toes’’ sign, midfoot flattening during stance (i.e., a flexible flatfoot deformity) and verified that the PT could not sufficiently evert the calcaneus. Changes in kinetics may be associated with the presence of osteoarthritis as the PTTD progresses. The altered EMG data were helpful toward understanding the complications caused by the progression of PTTD, such as intrasubstance tears in the peroneals and forefoot abduction. These data suggest that non-operative treatment of patients with PTTD should not only consider reducing posterior tibialis activity with arch support, but should also use a similar approach to reduce the activity of the peroneals, tibialis anterior and the gastrocnemius.

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References

[1] Johnson KA, Strom DE. Tibialis posterior tendon dysfunction. Clin Orthop Relat Res 1989;196–206.
[2] Myerson MS. Adult acquired flatfoot deformity. Treatment of dysfunction of the posterior tibial tendon. J Bone Joint Surg Am 1996;78:780–92.
[3] Chi TD, Toolan BC, Sangeorzan BJ, Hansen Jr ST. The lateral column lengthening and medial column stabilization procedures. Clin Orthop 1999;81–90.
[4] Funk JR, Hall GW, Crandall JR, Pilkey WD. Linear and quasi-linear viscoelastic characterization of ankle ligaments. J Biomech Eng 2000;122:15–22.
[5] Kitaoka HB, Patzer GL. Subtalar arthrodesis for posterior tibial tendon dysfunction and pes planus. Clin Orthop 1997;187–94.
[6] Mann RA, Thompson FM. Rupture of the posterior tibial tendon causing flat foot. Surgical treatment. J Bone Joint Surg Am 1985;67:556–61.
[7] Moseir-LaClair S, Pomeroy G, Manoli 2nd A. Intermediate follow-up on the double osteotomy and tendon transfer procedure for stage II posterior tibial tendon insufficiency. Foot Ankle Int 2001;22:283–91.
[8] 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;25:445–50.
[9] Kelly IP, Nunley JA. Treatment of stage 4 adult acquired flatfoot. Foot Ankle Clin 2001;6:167–78.
[10] Kelly IP, Easley ME. Treatment of stage 3 adult acquired flatfoot. Foot Ankle Clin 2001;6:153–66.

[11] Augustin JF, Lin SS, Berberian WS, Johnson JE. Nonoperative treatment of adult acquired flat foot with the Arizona brace. Foot Ankle Clin 2003;8:491–502.
[12] Chao W, Lee TH, Hecht PJ, Wapner KL. Conservative management of posterior tibial tendon rupture. In: Proceedings of the American Academy of Orthopaedic Surgeons, vol. 18; 1994–1995. p. 1030.
[13] Pomeroy GC, Pike RH, Beals TC, Manoli 2nd A. Acquired flatfoot in adults due to dysfunction of the posterior tibial tendon. J Bone Joint Surg Am 1999;81:1173–82.
[14] Johnson JE, Harris GF. Pathomechanics of posterior tibial tendon insufficiency. Foot Ankle Clinics 1997;2:227–39.
[15] Brodsky JW. Preliminary gait analysis results after posterior tibial tendon reconstruction: a prospective study. Foot Ankle Int 2004;25: 96–100.
[16] Dyrby CO, Chou LB, Andriacchi TP. A relationship between lower limb kinetics and foot mechanics during gait. In: ASME Bioengineering Conference. 2001. p. 745–6.
[17] Kaufman KR, Kitaoka HB, Hansen D, Shaughnessy WJ. Technique for measurement of foot and ankle kinematics in children. In: IEEE/EBMS 19th International Confrence; 1997.p. 2878–82.
[18] Chao EY. Justification of triaxial goniometer for the measurement of joint rotation. J Biomech 1980;13:989–1006.
[19] Kaufman KR, An KN, Chao EY. A comparison of intersegmental joint dynamics to isokinetic dynamometer measurements. J Biomech 1995; 28:1243–56.
[20] Walmsley RP. Electromyographic study of the phasic activity of peroneus longus and brevis. Arch Phys Med Rehabil 1977;58: 65–9.
[21] Young CC, Rose SE, Biden EN, Wyatt MP, Sutherland DH. The effect of surface and internal electrodes on the gait of children with cerebral palsy, spastic diplegic type. J Orthop Res 1989;7:732–7.
[22] Perotto A. Anatomical guide for the electromyographer, 3rd ed., Springfield, IL; 1994.
[23] Nilsson J, Panizza M, Hallett M. Principles of digital sampling of a physiologic signal. Electroencephalogr Clin Neurophysiol 1993;89: 349–58.
[24] Song J, Hillstrom HJ, Secord D, Levitt J. Foot type biomechanics. comparison of planus and rectus foot types. J Am Podiatr Med Assoc 1996;86:16–23.
[25] Ledoux WR, Hillstrom HJ. The distributed plantar vertical force of neutrally aligned and pes planus feet. Gait Posture 2002;15:1–9.
[26] Tome J, Flemister A, Houck J, Nawoczenski D. The effect of posterior tibial tendon dysfunction on dynamic foot motion Portland, OR: Gait and Clinical Movement Analysis Society; 2005. p. 113–4.
[27] Keenan MA, Peabody TD, Gronley JK, Perry J. Valgus deformities of the feet and characteristics of gait in patients who have rheumatoid arthritis. J Bone Joint Surg Am 1991;73:237–47.
[28] Klass M, Baudry S, Duchateau J. Aging does not affect voluntary activation of the ankle dorsiflexors during isometric, concentric, and eccentric contractions. J Appl Physiol 2005;99:31–8.
[29] Blanke DJ, Hageman PA. Comparison of gait of young men and elderly men. Phys Ther 1989;69:144–8.
[30] Hageman PA, Blanke DJ. Comparison of gait of young women and elderly women. Phys Ther 1986;66:1382–7.
[31] Nigg BM, Fisher V, Ronsky JL. Gait characteristics as a function of age and gender. Gait Posture 1993;2:213–20.