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Kinematics and Kinetics of Single-Limb Heel Rise in Diabetes Related Medial Column Foot Deformity

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Abstract

Background—Diabetes-related medial column foot deformities contribute to high plantar pressure, joint instability, ulceration and amputation. Impaired foot function may be an early indicator of foot structural incompetence and contribute to deformity progression. This study examines the ability of single-limb heel rise multi-segmental kinematics and kinetics to identify midfoot and hindfoot dysfunction in those with diabetes-related medial column foot deformity.

Methods: Single-limb heel rise foot kinematics and kinetics were examined in adults with diabetes mellitus and peripheral neuropathy with and without medial column foot deformity and age-, weight-matched controls.

Findings: Hindfoot relative to shank plantarflexion, peak and excursion, were reduced in both diabetes groups compared to controls ($P < 0.017$). Controls' initial forefoot relative to hindfoot position was plantarflexed 31 degrees and plantarflexed an additional 13 degrees during heel rise. The initial forefoot relative to hindfoot position for the diabetes group without deformity was similarly plantarflexed as controls (34 degrees) while the diabetes deformity group was less plantarflexed (lower arch position: 23 degrees, $P < 0.017$). During the heel rise task both diabetes groups demonstrated less ability to plantarflex the forefoot relative to the hindfoot compared to controls (2 and 5 degrees respectively, $P < 0.017$). Ankle plantarflexion power was reduced in the diabetes deformity group compared to controls ($P < 0.017$).

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Interpretation: The single-limb heel rise task identified movement dysfunction in those with diabetes mellitus and peripheral neuropathy. Failure to plantarflex the forefoot relative to hindfoot may compromise midfoot joint stability and increase the risk of injury and arch collapse.

Keywords

Plantarflexion; midfoot; kinematics; kinetics; diabetes

1. Introduction

Approximately 26 million Americans have diagnosed or undiagnosed diabetes mellitus (DM).(CDC 2011) Peripheral neuropathy (PN) of the sensory, motor and autonomic systems is a particularly debilitating complication of chronic DM, occurring in 30% of individuals with diabetes who are over 40 years old.(CDC 2011) PN is a key factor associated with the development of adult-acquired neuropathic foot deformity, contributing to high plantar pressure, joint instability, ulceration and lower extremity amputation. We believe that deformities occurring along the medial longitudinal arch or column of the foot (calcaneus, talus, navicular, cuneiforms, and metatarsal 1 through 3) are the most severe because they often lead to ulcer formation and amputation.

The mechanisms that initiate and result in the progression of foot deformity in those with DM and PN, placing them at high risk of ulceration, unstable and unable to bear weight, are largely unknown. Although sensori-motor neuropathy is a required component in the development of adult-acquired foot deformity, it is not sufficient, as not all individuals with DM and PN go on to develop foot deformity. The currently accepted cascade of deformity development includes, in addition to PN, an injury that can be minor and unperceived. (Sinacore 2001) We speculate that medial column deformities often begin as a result of pathology to structures that support the medial longitudinal arch [e.g. intrinsic foot muscles, (Cheuy et al. 2013) plantar fascia(Bolton et al. 2005), ligaments, and bone(Hastings et al. 2013a)] or development of limited joint mobility, particularly the talocrural joint that produces a deforming force at the arch.(Mueller et al. 1989) It is plausible that injury or damage to foot structures, critical in forming and supporting the medial longitudinal arch, is first apparent as changes in the kinematics and kinetics of the foot during weight-bearing activities and that repetition of abnormal joint kinematics and kinetics plays a role in deformity progression.

Previous examination of walking kinematics and kinetics in individuals with DMPN have inconsistently identified abnormal sagittal plane excursion between the hindfoot and shank and midfoot on hindfoot and ankle power during the stance phase of walking. (Deschamps et al. 2013;Hastings et al. 2014;Rao et al. 2007;Sawacha et al. 2009;Turner et al. 2007) The inconsistency in measuring differences in inter-segmental foot motion and ankle power during walking may be, in part, related to weaker expression of kinematic abnormalities during the walking task. Walking is a general lower extremity task, allowing compensation by the hip and knee for foot limitations. In addition walking is a task that can be completed without a maximal effort and at a slower speed which may not provide a sufficient challenge to the musculoskeletal system.

Onodera et al (Onodera et al. 2011) examined foot kinematics in individuals with DMPN during a more challenging foot-specific task: stair ascent and descent. Using a single foot segment model, foot on shank dorsiflexion and plantarflexion were limited during ascent and plantarflexion was limited on stair descent. The ability to detect group differences with the stair climbing task as well as the inconsistency in observed kinematic impairments during walking suggests that a kinematic and kinetic assessment of a challenging foot-specific task, using a multi-segmental foot model, may improve detection of foot impairments.

The single-limb heel rise test is commonly used to identify and assess plantarflexor muscle performance (Lunsford & Perry 1995) and plantarflexor muscle performance has been shown to be a good predictor of function including stair climbing and walking performance. (Shaffer et al. 2000) The normal completion of the heel rise task requires a complex interaction of both active and passive structures. The ankle plantarflexor muscles contract to raise and invert the heel. Calcaneal inversion, tension on the plantar fascia, and contraction of the intrinsic foot muscles assist the midfoot and forefoot joints in becoming a rigid lever so that the plantarflexor force is transmitted along the length of the foot, allowing the heel to continue to rise. (Blackwood et al. 2005) Failure of the foot to become a rigid lever during loading and propulsion results in abnormal forces being transmitted through the supporting structures including muscle, tendon, ligament, and midfoot joints and can result in injury and deformity.

The single-limb heel rise test also has been used to identify foot and ankle muscle dysfunction in individuals with adult-acquired medial column deformity as a result of posterior tibialis tendon dysfunction. (Chimenti et al. 2014; Houck et al. 2009; Johnson & Strom 1989) Kinematic examinations of the heel rise task, using a multi-segmental foot model, have identified an inability of the forefoot to plantarflex, remaining in a dorsiflexed position throughout the task. (Chimenti et al. 2014; Houck et al. 2009) To our knowledge, ankle plantarflexor power during the single-limb heel rise task has not been reported for those with posterior tibialis tendon dysfunction although ankle plantarflexor power has been reported to be reduced in individuals whose posterior tibialis tendon is transferred to the dorsum of the foot to correct a traumatic foot drop. (Hastings et al. 2013b)

The lack of consistent kinematic and kinetic deficits at the ankle and foot during walking in those with DMPN, coupled with previous use of the single-limb heel rise test to identify individuals with posterior tibialis tendon dysfunction led us to consider the use of the single-limb heel rise test in individuals with DMPN and medial column deformity. Application of a multi-segmental kinematic foot model to the single-limb heel rise task would allow measurement of intersegmental foot motion, particularly forefoot relative to hindfoot. Repetition of abnormal midfoot motion could play a critical role in the initiation and progression of medial column foot deformity and provide treatment direction. The purposes of this study were to examine 1) hindfoot relative to shank and forefoot relative to hindfoot sagittal plane kinematics and 2) speed and kinetics of a single-limb heel rise in individuals with DMPN with and without medial column deformity and controls. We hypothesized that individuals with DMPN would present with deficits in plantar flexion kinematics and kinetics when compared to a group of healthy control participants while performing a single-

limb heel rise. We further anticipated that individuals diagnosed with DMPN who have medial column foot deformity would have greater deficits than individuals without deformity. Such information would demonstrate the progressive nature of the disease and the associated foot deformity.

2. Methods

2.1 Participants

Thirty-six adult participants were recruited for this cross-sectional study from diabetes clinics and volunteer databases. All participants were fully informed of the study requirements, risks, and benefits and signed an Institutional Review Board approved consent form. Participants were eligible if they had ; 1) DM, PN, and medial column deformity (DMPN+, n=12), 2) DM and PN without medial column deformity (DMPN-, n=12), or if they were 3) age- and weight-matched to the DMPN+ and DMPN- groups but did not have DM or PN. Participants in the DMPN+ and DMPN- groups could have either type 1 or type 2 DM. Diabetes was diagnosed by their physician and in all cases required oral hypoglycemic medications or exogenous insulin medication in order to control their blood glucose level. Peripheral neuropathy was defined as the inability to feel the 5.07 (10 gram) Semmes-Weinstein monofilament on at least one of six locations on the plantar surface of the foot. (Diamond et al. 1989) The DMPN+ group were determined to have medial column deformity if they met 2 of the following clinical criteria on one of their feet: 1) standing calcaneal eversion relative to the leg of 5° , (Jonson & Gross 1997) 2) standing angle between the medial malleolus, navicular, and 1st metatarsal head (medial longitudinal arch angle) $<130^{\circ}$, (Jonson & Gross 1997) 3) standing height of the inferior surface of the navicular to the ground is ≥ 24 mm, (Jonson & Gross 1997; Menz & Munteanu 2005) 4) standing dorsal foot height/truncated foot length (arch index) <263 , (Butler et al. 2008) and 5) walking barefoot medial midfoot peak plantar pressure >290 kPa (Sinacore et al. 2008) measured by EMED-ST P-2 pressure platform (Novel Inc., St. Paul, MN). If a DMPN+ participant met the criteria on both feet, the foot with the greatest medial column deformity was chosen as the involved side. The DMPN- group met one or none of the clinical criteria for medial column deformity described above. The involved side for the DMPN- and control groups was chosen to be the same as the DMPN+ participant to whom they were matched. Participants were excluded if they had lower extremity amputations that extended beyond the toes, had a current plantar ulcer, were not ambulatory, or weighed >180 kg, or had metal implants or pace makers (exclusion criteria for the magnetic resonance imaging component of a larger study).

2.3 Temporal, kinematic and kinetic data collection

Temporal, kinematic, and kinetic data for the thigh, shank and foot were captured with an 8-camera 200 Hz Vicon motion analysis system (Vicon MX, Los Angeles, California, USA). Ten millimeter reflective markers were attached to the skin with double-sided tape or to thermoplastic plates for the shank, hindfoot, and forefoot. The shank markers were located on the fibular head, tibial tuberosity, medial and lateral malleoli, and four markers were mounted to a plate and secured to the distal-lateral shank. Marker placement for the hindfoot and forefoot followed the modified Oxford multi-segmental foot model described by

Carson. (Carson et al. 2001) The hindfoot markers were located at the sustentaculum tali, peroneal trochlea, and two markers were mounted to a plate that was aligned with the bisection of the posterior calcaneus. The forefoot markers were located at the base and head of the first and fifth metatarsals and a marker between the second and third metatarsal heads. Kinetic data were collected simultaneously using a Bertec K80301 force platform (Bertec Corporation, Columbus, OH, USA).

All participants received weight-bearing lateral radiographs of their involved foot in order to measure two angles required in building the hindfoot and forefoot models used in this study. Radiographic images were imported into iSite® Picture Archiving and Communication Systems (PACS) software (Philips Healthcare Informatics, Foster City, CA) and calcaneal pitch (the angle between the line from the calcaneal tuberosity to the most plantar aspect of the distal calcaneus and the ground) and 1st metatarsal declination (the angle between the line through the longitudinal axis of the 1st metatarsal and the ground) were measured. (Hastings et al. 2011) The multi-segmented foot model was built in Visual3D software (C-Motion Inc. Germantown, MD, USA). The shank segment was defined as previously described, using landmarks as well as projected and virtual markers. (Carson et al. 2001; Hastings et al. 2013b) However, the sagittal pitch of the local coordinate system for the hindfoot and forefoot were individually set using calcaneal pitch and 1st metatarsal declination obtained from the lateral radiograph measures. (Hastings et al. 2014) Radiographs were also examined by a foot and ankle fellowship trained orthopedic surgeon (XXX) for signs of bone and joint abnormalities.

Temporal, kinematic and kinetic data were collected during a single-limb heel rise task. Participants were instructed to complete as many heel rises as possible, up to 20 repetitions, keeping the knee extended and using hands for balance only, a protocol similar to that previously reported by Lunsford and Perry. (Lunsford & Perry 1995) The task was discontinued when the participant could no longer lift the heel from the ground or was pushing through their hands to assist in lifting the heel. Kinematic compensations (i.e., slight knee flexion) were allowed during the single-limb heel rise task in our three participant groups. Heel rise task speed was calculated within the kinematic processing software as the total distance traveled during the heel rise and return along the vertical axis, divided by the time it took to complete the heel rise and return. The variables of interest included hindfoot relative to shank plantarflexion and forefoot relative to hindfoot dorsiflexion/plantarflexion at the beginning of the task, the position at peak heel rise, and the total excursion from initial to peak. Hindfoot relative to shank inversion/eversion excursion and inversion at peak heel rise were also examined. The three trials with the highest plantarflexor power production were selected for the analysis and the variables of interest for these three trials were averaged.

2.5 Statistical analyses

Participant characteristics between groups were compared using a one-way analysis of variance for continuous variables and Chi-square for discrete variables. Single-limb heel rise kinematic, temporal, and kinetic variables were analyzed using a one-way analysis of variance and a oneway analysis of covariance with heel rise speed as a covariate. The

homogeneity of regression assumption was met as indicated by a nonsignificant group x heel rise speed interaction, $P > .05$ for all dependent variables. A Bonferroni correction was applied to each variable at the comparison level (3 groups) with a corresponding significance level of $P = 0.017$. SPSS Statistics version 21 was used for all statistical analyses (SPSS Statistics Inc., Chicago, USA).

3. Results

3.1 Participant characteristics

There were no group differences in age, height, weight, and body mass index. (Table 1) The DMPN groups had DM for 17-18 years prior to study participation. The DMPN+ group had clinical measures that indicate medial column collapse including a lower medial longitudinal arch angle, lower navicular height, and higher medial midfoot peak plantar pressure than the DMPN- and control groups ($P = 0.017$). There was radiographic evidence of neuropathic Charcot arthropathy changes in the midfoot of 3 DMPN+ participants and severe osteoarthritis of the midfoot in 1 DMPN+ participant. One participant in the DMPN+ group had a 5th toe amputation on the involved side. The DMPN+ group had calcaneal eversion greater than the DMPN- group and an arch index lower than the control group ($P = 0.017$). (Table 1) Radiographic calcaneal pitch was lower in the DMPN+ group [11° (SD 5°)] compared to the DMPN- [18° (SD 5°), $P = 0.017$] but not different than controls [16° (SD 6°), $P = 0.03$]. First metatarsal declination was lower in the DMPN+ group [19° (SD 3°)] compared to the DMPN- [22° (SD 2°), $P = 0.017$] but not different than controls [20° (SD 3°), $P = 0.13$].

3.2 Kinematic, kinetic, and temporal examination of the single-limb heel rise task

Hindfoot relative to shank plantarflexion was less in the DMPN+ and DMPN- groups, compared to controls, as measured at peak heel rise ($P < 0.017$) as well as plantarflexion excursion from initial to peak ($P < 0.017$). In addition plantarflexion excursion was less in the DMPN+ group compared to the DMPN- group ($P < 0.017$). Hindfoot relative to shank inversion, peak and excursion, was lowest in the DMPN+ group compared to DMPN- and control groups but the difference was not statistically significant. (Table 2. Figures 1 and 2)

The control group began the heel rise task with 31° (SD 9°) of plantarflexion between the forefoot and hindfoot and completed the task with an additional 13° (SD 7°) of forefoot relative to hindfoot plantarflexion. The DMPN+ group's starting position of the forefoot relative to the hindfoot was less plantarflexed ($P < 0.017$ compared to DMPN- group) and both the DMPN+ and DMPN- group demonstrated less plantarflexion excursion of the forefoot on the hindfoot during the task compared to controls ($P < 0.017$). (Table 2, figures 1 and 2)

The DMPN+ and DMPN- groups completed the heel rise task slower and with less ankle power than the control group ($P < 0.017$). (Table 2 and Figure 1)

3.3 Kinematics and kinetics controlling for single-limb heel rise speed

When heel rise speed was controlled for in the analysis of the kinematic and kinetic variables, hindfoot relative to shank plantarflexion excursion remained significantly lower in the DMPN+ group compared to DMPN- and controls (adjusted means: DMPN+ = 7°, DMPN- = 13°, Control = 18°, $P < 0.017$). Forefoot relative to hindfoot initial position was less plantarflexed in the DMPN+ group compared to the DMPN- group (adjusted means: DMPN+ = -23°, DMPN- = -34°, Control = -31°, $P < 0.017$). Peak forefoot relative to hindfoot plantarflexion was reduced in the DMPN+ group compared to both the DMPN- and control groups (adjusted means: DMPN+ = -26°, DMPN- = -40°, Control = -41°, $P < 0.017$). Forefoot relative to hindfoot plantarflexion excursion was reduced in the DMPN+ group compared to controls (adjusted means: DMPN+ = 3°, DMPN- = 6°, Control = 10°, $P < 0.017$). Ankle joint power remained significantly lower in the DMPN+ group compared to controls (adjusted means: DMPN+ = 1.2 watts/kg, DMPN- = 1.7, Control = 2.0, $P < 0.017$).

3.6 Impact of Midfoot Joint Abnormalities

There were an insufficient number of study participants with radiographic midfoot joint changes (i.e. neuropathic Charcot arthropathy or osteoarthritis) to complete a meaningful comparison of those with midfoot joint changes to the other groups. However, we examined the data graphically to provide insight into the impact of midfoot joint changes on kinematics during the single-limb heel rise task. Calcaneal pitch, a radiographic measure of foot alignment, was graphed against forefoot relative to hindfoot plantarflexion excursion (Figure 3). All participants with radiographic midfoot joint changes had forefoot relative to hindfoot plantarflexion excursions during heel rise that were near or below zero degrees [mean = 1° (SD 2) °].

4. Discussion

This study used a multi-segmental foot model to examine the use of the single-limb heel rise task as a foot-specific challenge to assess foot movement dysfunction in individuals with DMPN, with and without foot deformity. The groups with DMPN, compared to controls, completed the task with less ankle power and decreased plantarflexion of the hindfoot relative to shank and forefoot relative to hindfoot. Foot movement dysfunction in the DMPN with deformity group was uniquely defined by the greatest reduction in hindfoot relative to shank plantarflexion excursion of all groups and a forefoot relative to hindfoot position at peak heel rise that was more dorsiflexed, indicating a lower arch position. These key findings suggest a potential mechanism by which kinematic and kinetic impairments increase the load on arch supporting structures increasing the risk of injury and deformity progression.

Peak ankle power during the single-limb heel rise task was reduced 40-60% in individuals with DMPN compared to controls. The DMPN+ group was significantly impaired compared to the DMPN- and control groups, even when heel rise speed was controlled. Reduction in ankle power has been previously found in those with DMPN during a walking task, however the difference is generally smaller (13 to 21%) and often the group differences are no longer apparent when walking speed is controlled. (Hastings et al. 2014; Rao et al. 2006; Raspovic

2013;Yavuzer et al. 2006) The evidence from this study implies that the more functionally demanding single-limb heel rise task enhances characterization of ankle plantarflexor power and is able to distinguish movement dysfunction unique to those with medial column foot deformity.

The DMPN+ group had significantly less hindfoot relative to shank and forefoot relative to hindfoot plantarflexion excursion during the elevation phase of the single-limb heel rise. All our participants with neuropathic Charcot arthropathy and osteoarthritic joint changes had limited forefoot relative to hindfoot plantarflexion excursion. However, there were participants in all three groups that had less than 5 degrees of plantarflexion excursion during the single-limb heel rise task. We believe this has important biomechanical and clinical implications.

We propose that the kinematic variable, excursion of forefoot relative to hindfoot plantarflexion, is a measure of the ability of the foot to transition from a flexible to a rigid lever and that hindfoot relative to shank excursion and ankle power are composite indicators of the functionality of this complex mechanism. The participants with DMPN+ and some individuals in the DMPN- and control groups showed movement impairments in these measures of foot function. Raising the heel and elevating the arch requires adequate extrinsic plantarflexor muscle force, intact joint structures that allow appropriate motion (ligaments and joint congruency), tensioning of the plantar fascia, and active contraction of the intrinsic muscles of the foot to stabilize the midfoot. We believe that individuals in the DMPN groups and some of the individuals in the control group have impairments in many of these stabilizing and dynamic structures. Our current research is measuring the dysfunction of these components so that we can better understand mechanisms underpinning diabetes and neuropathic-related foot deformity progression so that aggressive and effective treatments can be implemented to break the cycle of deformity, ulceration, and amputation.

In other models of medial column deformity, in particular adult-acquired flat foot deformity as a result of posterior tibialis tendinopathy, the calcaneus is excessively everted relative to the shank.(Tome et al. 2006) Although there is a trend that the DMPN+ group has less inversion than the other two groups, the difference is small and not clinically or statistically significant. Only one participant had clinically measured calcaneal eversion in stance greater than 5°, one of the criteria for defining medial column deformity. Our clinical and kinematic data would suggest that deformity of the hindfoot in the frontal plane is not a common manifestation in medial column deformity associated with diabetes and neuropathy.

Inclusion of radiographic measures of calcaneal and 1st metatarsal pitch into the kinematic model provided insight into the relative weight-bearing alignment of the segments during the single-limb heel rise task. The forefoot relative to hindfoot segment position at the start and at the peak of the single-limb heel rise task indicate that the DMPN+ group started and completed the single-limb heel rise task in a less plantarflexed or lower arch position, which is generally considered to be more flexible and less stable. We hypothesize that coupling poor foot function with midfoot instability increases the risk of injury and deformity progression. Early identification of the movement dysfunction can assist the medical community in the implementation and development of prevention and treatment strategies

aimed at improving foot function. The level of neuropathy severity may limit restorative therapy goals and additional treatment and research should address compensatory strategies (ie foot and ankle orthoses) aimed at protecting the at risk foot from additional trauma.

The cross sectional nature of this study provides insight into foot function differences in groups that differ by severity of disease and foot deformity. The spectrum of foot deformity severity within DMPN+ group was clinically relevant. Our four participants with radiographic evidence of midfoot joint changes suggests as deformity progresses, loss of forefoot relative to hindfoot motion should be expected. However, from this current body of work we are unable to measure and determine the contribution of the multiple factors that may be associated with limited motion (e.g. limited midfoot joint mobility/stability, intrinsic muscle weakness, plantar fascia function). The use of clinical measures of alignment and plantar pressure to determine group assignment deserves additional exploration. Future research in this area should examine which measures are predictive of radiographic deformity and if the measures are responsive and indicative of deformity progression. Additional work is needed to develop reliable and valid measurement methods of foot structure integrity to determine key components leading to poor multi-segmental foot function.

Interpretation and application of study results is limited by the small sample size. However, we were able to detect foot movement dysfunction that is more prevalent in those with diabetes, peripheral neuropathy, with and without medial column foot deformity. In addition a longitudinal study is required to examine questions regarding the role foot function plays in the development and progression of foot deformity.

5. Conclusion

The single-limb heel rise task is a foot-specific challenge capable of distinguishing individuals with diabetes mellitus, peripheral neuropathy and medial column deformity from those without deformity and controls. Those with DMPN+ had kinematic and kinetic indicators of poor foot function. A focus of future research is to understand the biomechanical and anatomical contributors to acquired deformity onset and progression in order to minimize the risk of amputation and improve daily function in those with diabetes and peripheral neuropathy.

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Research Highlights

1. Single-limb heel rise foot kinematics and kinetics were examined
2. Those with diabetes and peripheral neuropathy compared to controls had:
 - a. Reduced hindfoot relative to shank plantarflexion
 - b. Reduced forefoot relative to hindfoot plantarflexion
 - c. Reduced ankle power
3. Those with medial column foot deformity were uniquely identified by
 - a. Starting the task with less forefoot on hindfoot plantarflexion

Figure 1.

Heel rise kinematics and kinetics. Shaded band represents the mean $\pm$ 1 standard deviation for the control group. Solid line is the mean $\pm$ 1 standard deviation bars for the group with diabetes, peripheral neuropathy and deformity, and the dotted line is the mean $\pm$ 1 standard deviation bars for the group with diabetes, peripheral neuropathy, without deformity.

Figure 2.

Photographs of the heel rise task demonstrating a control participant with adequate hindfoot on shank and forefoot on hindfoot plantarflexion (A), progressively decreased ability to plantarflex the hindfoot on shank and forefoot on hindfoot (B and C).

Figure 3.

Radiographic-measured calcaneal pitch compared to forefoot relative to hindfoot plantarflexion excursion. The diabetes mellitus, peripheral neuropathy with deformity group (DMPN+, n=12) are squares. The dark blue squares represent individuals with radiographic midfoot joint changes related to either neuropathic (Charcot, n=4) arthropathy or osteoarthritis; while red squares represent individuals with a low medial longitudinal arch without radiographic joint changes (n=8). The green triangles represent individuals with diabetes mellitus, peripheral neuropathy without deformity (n=12), and purple circles represent age- and weight-matched controls (n=12). Note the limited forefoot relative to hindfoot plantarflexion excursion of most DMPN+ participants.

Table 1
Subject Characteristics

	DMPN+ (n=12)	DMPN- (n=12)	Controls (n=12)
Age (yr) *	59 (13)	58 (8)	57 (14)
Sex (Male/Female)	7/5	8/4	8/4
Height (m) *	1.70 (0.08)	1.76 (.09)	1.74 (0.11)
Weight (kg) *	110 (24)	113 (28)	108 (30)
BMI (kg/m²) *	38 (9)	36 (9)	35 (8)
Type of DM (1/2)	1/11	2/10	
Duration of DM (years) *	17 (11)	18 (10)	
HbA1c (%) *	8.1 ^a (2.6)	7.5 (2.9)	5.2 (1.7)
Biothesiometry (volts) *	40 ^a (14)	39 ^b (15)	20 (14)
Race (Caucasian/African American)	8/4	9/3	8/4
Clinical Measures of Alignment			
Calcaneal Eversion (degrees)	6 (6)	3 (4)	4 (4)
Medial Longitudinal Arch Angle (degrees)	122 ^{a,c} (7)	150 (12)	145 (8)
Navicular Height (mm)	24 ^{a,c} (5)	37 (8)	38 (5)
Arch Index	0.288 ^a (0.055)	0.342 (0.067)	0.344 (0.031)
Medial Midfoot Peak Plantar Pressure (kPa)	273 ^{a,c} (279)	83 (76)	69 (65)

* Values are given as the mean (standard deviation) or number. Abbreviations: DMPN+, diabetes mellitus, peripheral neuropathy, and medial column deformity; DMPN-, diabetes mellitus, peripheral neuropathy, without medial column deformity.

^a DMPN+ is different than Controls ($P < 0.017$)

^b DMPN- is different than Controls ($P < 0.017$)

^c DMPN+ is different than DMPN- ($P < 0.017$)

Table 2
Heel rise kinematics and kinetics

	DMPN+ (n=12)	DMPN- (n=12)	Control (n=12)
Hindfoot on Shank			
Initial Plantarflexion (-)/Dorsiflexion (+) (degrees)	21 (7)	29 (7)	25 (8)
Plantarflexion (-)/Dorsiflexion (+) at peak heel rise (degrees)	15 ^a (8)	17 ^b (10)	4 (9)
Plantarflexion Excursion (Initial to Peak) (degrees)	7 ^{a,c} (4)	12 ^b (6)	20 (5)
Inversion at peak heel rise (degrees)	1 (7)	4 (7)	5 (8)
Inversion Excursion (Initial to Peak) (degrees)	4 (4)	6 (3)	8 (4)
Forefoot on Hindfoot			
Initial Dorsiflexion(+)/ Plantarflexion(-) (degrees)	-23 ^c (5)	-34 (9)	-31 (9)
Dorsiflexion(+)/ Plantarflexion(-) at peak heel rise (degrees)	-25 ^{a,c} (7)	-39 (12)	-44 (13)
Plantarflexion Excursion (Initial to Peak) (degrees)	2 ^a (5)	5 ^b (6)	13 (7)
Temporal and Kinetics			
Heel rise speed (cm/sec)	11 ^a (7)	10 ^b (6)	20 (5)
Peak Ankle Power (Watts/kg)	1.0 ^a (0.6)	1.4 ^b (0.5)	2.4 (0.7)

* Values are given as the mean (standard deviation).

Abbreviations: DMPN+, diabetes mellitus, peripheral neuropathy, and medial column deformity; DMPN-, diabetes mellitus, peripheral neuropathy, without medial column deformity

^a DMPN+ is different than Controls ($P < 0.017$)

^b DMPN- is different than Controls ($P < 0.017$)

^c DMPN+ is different than DMPN- ($P < 0.017$)