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Ankle ROM and stiffness measured at rest and during gait in individuals with and without diabetic sensory neuropathy

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Abstract

Introduction—The purpose of our study was to examine the relationship between ankle dorsiflexion (DF) range of motion (ROM) and stiffness measured at rest (passively) and plantar loading during gait in individuals with and without diabetes mellitus (DM) and sensory neuropathy. Specifically, we sought to address three questions for this at-risk patient population: (1) Does peak passive DF ROM predict ankle DF ROM used during gait? (2) Does passive ankle stiffness predict ankle stiffness used during gait? (3) Are any of the passive or gait-related ankle measures associated with plantar loading?

Methods—Ten subjects with DM and 10 age and gender matched non-diabetic control subjects participated in this study. Passive ankle DF ROM and stiffness were measured with the Iowa Ankle ROM device. Kinematic, kinetic and plantar pressure data were collected as subjects walked at 0.89 m/s.

Results—We found that subjects with DM have reduced passive ankle DF ROM and increased stiffness compared to non-diabetic control subjects, however, subjects with DM demonstrated ankle motion, stiffness and plantar pressures, similar to control subjects, while walking at the identical speed, 0.89 m/s (2 mph). These data indicate that clinical measures of heel cord tightness and stiffness do not represent ankle motion or stiffness utilized during gait. Our findings suggest that subjects with DM utilize strategies such as shortening their stride length and reducing their push-off power to modulate plantar loading.

Keywords

Ankle ROM; Stiffness; Diabetes; Gait; Loading

1. Introduction

Abnormal plantar loading due to foot floor interaction [1] is thought to contribute to the development of foot ulcers in individuals with diabetes mellitus (DM) where the estimated incidence is up to 20% of all individuals with DM [2]. Inability to heal foot ulcers or prevent recurrence contributes to progression of the local pathology, leading to the high rate of amputation seen in individuals with DM. Over 50% of non-traumatic lower limb

amputations are performed on individuals with DM [3]. Along with grave consequences affecting health and functional abilities [4], foot ulcers and amputation are often harbingers of personal and financial hardship. Factors contributing to increased loading on the plantar aspect of the foot and thus the potential development of foot ulcers are therefore of considerable interest.

Recent studies implicate limited passive dorsiflexion (DF) range of motion (ROM) and increased passive ankle stiffness as key factors contributing to increased plantar loading [5–10]. Limited ankle DF ROM and/or increased stiffness may result from the effects of longstanding hyperglycemia on connective tissue [11] and are hypothesized to restrain forward progression of the tibia on the fixed foot during the stance phase of walking. This, in turn, is believed to result in prolonged and excessive weight bearing stress under the metatarsal heads.

Evidence supporting the postulated relationship between ankle flexibility and plantar loading is limited, predominantly coming from studies demonstrating improved ulcer healing following Achilles tendon lengthening surgery [5,6,8]. These studies reported 9–18° improvement in DF ROM with concomitant 27–46% reduction in forefoot plantar pressure 2–7 months following tendo-Achilles lengthening [5,9]. However, after surgery, subjects may modulate foot pressures by altering their gait using different approaches, such as preferentially loading the non-operated limb, changing walking strategy and reducing walking speed. In these circumstances, factors other than intervention would influence the reported relationship between ankle DF ROM and plantar loading. Further, longitudinal studies of subjects who have undergone tendon Achilles lengthening demonstrate a disconnect between passive ankle DF ROM and loading [9]. Following this procedure, improvements in ankle DF ROM are maintained 7 months post-operatively, however, plantar loading increase to values close to those measured before Achilles tendon lengthening.

Cross-sectional studies seeking to examine the relationship between mechanical properties of the ankle and plantar loading indicate that subjects with reduced passive ankle ROM sustain significantly higher plantar pressures during walking [10,12,13]. However, these studies do not control walking speed, which can influence both ankle motion [14,15] and plantar loading during gait [16–18]. Walking speed thus emerges as a confounding factor, rendering interpretation of purported results difficult.

Experimental studies investigating structural and functional predictors of regional plantar loading reveal that subjects who tend to walk with larger dynamic ankle ROM experience higher forefoot plantar pressures [19]. These findings, albeit from non-diabetic individuals, highlight the importance of dynamic ROM; however they do not relate dynamic ROM to available (passive)ROM. The relationship between dynamic ROM utilized in gait and passive ROM thus emerges as an important potential factor contributing to plantar loading but has not been elucidated in individuals with DM.

The purpose of our study was to examine the relationship between ankle DF ROM and stiffness measured at rest (passively) and plantar loading during gait in individuals with and without DM and neuropathy. Specifically, we sought to address three questions: (1) Does peak passive DF ROM predict ankle DF ROM used during gait? (2) Does passive ankle stiffness predict ankle stiffness used during gait? (3) Are any of the passive or gait-related ankle measures associated with plantar loading? Compared to the control group, we hypothesized that subjects with DM would demonstrate lower passive DF ROM and higher stiffness. Due to differences in impairments, we hypothesized that passive ankle DF ROM and stiffness would predict ankle DF ROM and stiffness during gait in individuals with DM

but not in non-diabetic individuals. We expected that passive ankle DF ROM and passive ankle stiffness would be associated with plantar loading in subjects with DM but not in non-diabetic individuals.

2. Methods

All procedures were approved by the Institutional Review Board at the University of Iowa Hospitals and Clinics. Subjects were recruited from the Diabetic Foot Clinic with the following inclusion criteria: diagnosis of DM, no current or history of previous ipsilateral foot ulcer, great toe or transmetatarsal amputation, absence of ipsilateral or contralateral Charcot neuroarthropathy. Presence of neuropathy was documented using 5.07 Semmes–Weinstein monofilaments. All subjects in the DM group showed loss of protective sensation [20]. Subjects in the non-diabetic control group were matched in age and gender to subjects with DM and were screened for lower extremity pain or musculoskeletal pathology or history thereof in the last 6 months. Ten subjects with DM (mean age: 56 ± 11 years; mean body mass: 96.6 ± 31.2 kg; mean height: 1.74 ± 0.1 m; M:F ratio: 6:4) and 10 non-diabetic control subjects (mean age: 54 ± 8 years; mean body mass: 76 ± 14.8 kg; mean height: 1.71 ± 0.09 m; M:F ratio: 6:4) participated in this study. The study and control groups did not differ in age ($p = 0.52$), body mass ($p = 0.09$) or height ($p = 0.53$). Subject characteristics documented in the DM group included: average duration of DM (20 ± 11 years), type of DM: 80% Type 2 DM and glycemic control (most recent HbA1C: $8.1 \pm 1.2\%$).

2.1. Passive testing

Passive ankle ROM and stiffness at specific torque levels [21,22] was measured using the Iowa Ankle ROM device, which has been shown to be valid and reliable [23]. Detailed description of the device and methods are provided in ref. [23]. Briefly, subjects were positioned supine with the knee extended; their leg was supported by a base plate and a foam block; and secured by Velcro straps. The sole of their foot was positioned so contact was maintained with a translucent Plexiglas foot plate throughout testing. The axis of rotation of the device was then adjusted in the antero-posterior and superior/inferior directions to approximate the ankle axis of rotation determined by palpation of the distal tips of the medial and lateral malleoli [24]. Torques of 15, 20 and 25 N m was applied using a hand held force gauge and resultant angular kinematics were measured using a digital inclinometer. Fig. 1 shows the apparatus and setup. The inclinometer was referenced to the tibial crest and then mounted on the foot plate which was parallel to the sole of the foot. Three cycles of testing were recorded at each force level. Next, the knee was flexed to approximately 20° to simulate the magnitude of knee flexion attained during gait [25] and ROM testing was repeated at the three force levels. Passive ankle stiffness was calculated as the slope of the resultant curves over the 15–25 N m intervals.

2.2. Gait testing

Kinematic and kinetic data were recorded as subjects walking along 10 m walkway at 0.89 m/s (2 mph) in their customary footwear. In designing this study, we decided to allow all subjects to wear their customary footwear and control walking speed. Since almost all walking is performed in footwear, we thought the continuance of this practice in our study best reflected real life situation and would confer external validity to the study. Previous studies have documented walking velocities between 0.77 and 1.26 m/s in subjects with DM [12,26,27], therefore 0.89 m/s (2 mph) was chosen as a comfortable walking speed, representative of preferred walking speed in subjects with DM. Walking speed was controlled in this study because the self-selected walking speeds in subjects with DM and neuropathy are typically significantly slower than non-diabetic controls [12,28,29]. Walking

speed influences ankle kinematics and kinetics [14,15] and also differentially affects plantar pressures in different regions of the foot [16–18].

Kinematic data were recorded at 60 Hz using an active marker system (Optotrak, NDI, Waterloo, Canada). Three infra-red markers were placed in a non-collinear arrangement on the subject's foot (over shoe), leg and thigh segments. Kinetic data were collected at 240 Hz using a forceplate embedded in the walkway (Kistler Inc., NY). Plantar pressure data were collected at 50 Hz using pressure sensitive insoles (Pedar, Novel Inc., Minneapolis, MN).

Kinematic and kinetic data were processed using Kingait (Mishac Kinetics, University of Waterloo, Canada) software. Kinetic data were low pass filtered at 8 Hz, kinematic data were low pass filtered with a cut-off frequency of 6 Hz using a fourth order Butterworth filter. Anatomical coordinate systems were established using standard criteria [30]. Sagittal ankle kinematics and kinetics were calculated. Peak dorsiflexion achieved during gait was obtained using sagittal ankle kinematics. Duration of second rocker was identified. Ankle stiffness during second rocker was defined using the method defined by Davis and DeLuca [31]. The magnitude of plantar loading was quantified as the peak pressure sustained under the metatarsal head region in the forefoot during stance.

2.3. Data analysis

A two-sample *t*-test was used to assess differences between the two groups ($\alpha = 0.05$). In addition to the hypotheses outlined above, associations between subject characteristics and passive measures were examined to rule out the influence of subject characteristics on passive measures. Pearson product moment correlation (*r*) was used to assess the relationship between variables of interest. Statistical significance ($H_0: \rho = 0$) and equality of correlations ($H_0: \rho_1 = \rho_2$) were assessed using approximate tests based on Fisher's *Z* transformation ($\alpha = 0.05$).

3. Results

3.1. Passive testing

Subjects with DM showed significantly lower peak passive dorsiflexion and higher passive ankle stiffness compared to age matched control subjects (Table 1).

Peak passive ankle dorsiflexion was not associated with peak ankle dorsiflexion attained during gait in either group ($H_0: \rho = 0$, $p = 0.67$ and 0.98 , DM and control groups, respectively, $H_0: \rho_1 = \rho_2$, $p = 0.49$; Fig. 2). Passive ankle stiffness was not significantly associated with ankle stiffness during second rocker ($H_0: \rho = 0$, $p = 0.84$ and 0.17 , DM and control groups, respectively, $H_0: \rho_1 = \rho_2$, $p = 0.20$; Fig. 3).

Glycemic control and duration of DM showed fair association with ankle stiffness (Table 2, electronic addendum).

3.2. Gait testing

Peak dorsiflexion attained during gait, ankle stiffness during second rocker and peak plantar pressure at the forefoot did not differ between groups as they walked at identical speed (Table 1). Subjects in the control group showed trends towards higher peak plantarflexor moment and peak ankle power generation in the sagittal plane (Fig. 4).

Passive ankle stiffness, peak passive dorsiflexion, peak dorsiflexion used in gait and ankle stiffness used in gait did not show a significant relationship with peak forefoot plantar pressures in either group (Table 3). Stride length was associated with plantar pressure in both groups. Stride length was associated with ankle stiffness during second rocker and total

ankle ROM utilized during gait in subjects with DM but not in non-diabetic control subjects (Table 3).

4. Discussion

The main findings of our study demonstrated that subjects with DM have reduced passive ankle DF ROM and increased passive stiffness compared to non-diabetic control subjects. To our knowledge, this is the first reported study showing both increased stiffness and reduced dorsiflexion ROM in subjects with DM. However, in spite of differences in passive ankle DF ROM and stiffness, subjects with DM demonstrated ankle motion, stiffness and plantar pressures, similar to control subjects, while walking at the same speed, 0.89 m/s (2 mph). While neither passive nor dynamic DF ROM or ankle stiffness were significantly associated with plantar loading, stride length was positively associated with plantar pressure in both groups.

Clinical measures of passive ankle DF ROM and stiffness obtained in this study are consistent with previous reports documenting reduced ankle DF ROM [10,26] and increased stiffness [22] in subjects with DM. Changes in ankle DF ROM and stiffness in individuals with DM have been attributed to disease-dependent as well as use-dependent processes. Disease-dependent mechanisms involve non-enzymatic glycation of collagen due to the underlying metabolic disorder [11]. Use-dependent mechanisms allude to adaptive fiber shortening within the triceps surae [29]. Qualitative changes in connective tissue stiffness as well as quantitative changes related to changes in the ratio of connective tissue to contractile tissue may contribute to increased ankle stiffness in individuals with DM.

The findings of the present investigation in regards to dynamic ankle motion, stiffness and plantar loading measures agree with previous reports of dynamic ankle DF ROM [27], ankle stiffness [12] and plantar pressures, at comparable walking speeds ranging from 0.77 to 0.89 m/s. In spite of differences in passive ankle DF ROM and passive stiffness, we found that dynamic measures of ankle DF ROM, stiffness and plantar pressure did not differ between groups while walking at a similar speed, 0.89 m/s (2 mph). However, subjects with DM walked with significantly shorter stride lengths and showed trends towards lower plantar flexor moments and peak power generation compared to non-diabetic control subjects. The study and control groups did not differ in height indicating that height did not confound stride length findings. Our results are in agreement with previous reports in which shorter stride lengths were accompanied by a decrease in the forward excursion of the center of pressure [32] as well as reduced ankle moment and power [27,29]. Reduced plantar flexor moment and reduced power at push off have also been described in subjects with DM as an attempt to provide the major positive work at push-off using their hip flexors [26]. Our results suggest that subjects with DM may utilize different strategies, such as shortened stride length and reduced push-off power, compared to non-diabetic control subjects, to achieve identical functional goals, in this case, to walk at a given speed.

Contrary to our hypotheses, passive ankle DF ROM and stiffness did not predict peak DF ROM and ankle stiffness utilized during gait. While passive ankle DF ROM varied between subjects, during gait all subjects attained approximately 10° peak DF ROM (range: 5–15°). This is consistent with the findings of Riddle [33]. Analysis of ankle stiffness revealed similar findings. These results indicate that clinical measures of passive heel cord tightness and stiffness do not represent ankle motion or stiffness utilized during gait.

To evaluate the possibility that subjects alter the contribution of the biarticular gastrocnemius muscle to ankle DF ROM and stiffness through knee flexion [34], we examined peak knee flexion attained in early stance and midstance. Our data, consistent with

previous reports [28], showed that subjects with DM and neuropathy utilize similar ranges of knee motion in early stance compared to non-diabetic controls. Further, associations between passive ankle measures obtained with the knee flexed (~15–20°) and gait-related measures were similar to those documented with the knee in an extended position. These findings argue against the role of limited passive DF on alterations in stance phase knee flexion as a potential mechanism [34] influencing the contribution of the biarticular gastrocnemius muscle on ankle stiffness or motion utilized during walking.

A second purpose of this study was to examine whether passive or gait-related ankle measures are associated with magnitude of plantar loading. We found that while neither passive nor dynamic DF ROM or ankle stiffness were significantly associated with plantar loading, stride length was positively associated with plantar pressure in both groups. Our findings underscore the role of stride length [35,36] as a mechanism influencing forefoot loading, even when walking velocity is controlled. We also found that longer stride lengths were accompanied by increased dynamic ankle motion and gait-related ankle stiffness in subjects with DM but not in non-diabetic controls (Table 3). Thus, our data suggest that ankle parameters are potentially linked with stride length in subjects with DM.

In summary, we found that in spite of differences in passive ankle DF ROM and stiffness, subjects with DM demonstrated gait-related ankle motion, stiffness and plantar pressures, similar to control subjects, while walking at the identical speed, 0.89 m/s (2 mph). These data indicate that clinical measures of heel cord tightness and stiffness do not represent ankle motion or stiffness utilized during gait. Our findings suggest that subjects with DM and neuropathy utilize strategies such as shortening their stride length and reducing the power of push-off to modulate plantar loading. Further studies are needed to explore the relationship between ankle flexibility and plantar loading in a wide spectrum of subjects with DM in whom ulcer formation may be problematic.

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Fig. 1.
Apparatus and setup for ankle ROM testing.

Fig. 2.
Relationship between passive ankle stiffness and dorsiflexion ankle stiffness utilized during second rocker.

Fig. 3. Group mean $\pm$ S.D. for sagittal ankle power (W/kg), moment (N m/kg) and kinematics ($^{\circ}$) during stance phase.

Fig. 4.
Relationship between passive ankle dorsiflexion and dorsiflexion utilized during gait.

Table 1

Summary of passive and gait-related measures

	DM	Control	<i>p</i> -Value
Peak passive DF (°)	6.4 ± 6.9	19.3 ± 3.9	<0.001
Peak passive DF ^{KF} (°)	13.1 ± 4.2	24.1 ± 5.5	<0.001
Peak DF during gait (°)	9.9 ± 2.0	11.8 ± 2.7	0.099
Peak knee flexion in mid-stance (°)	12.5 ± 6.6	14.8 ± 8.8	0.167
Passive ankle stiffness (N m/degree)	1.5 ± 0.49	1.042 ± 0.56	0.001
Passive ankle stiffness ^{KF} (N m/degree)	1.508 ± 0.43	1.021 ± 0.47	0.04
Ankle stiffness during second rocker (N m/degree)	6.526 ± 1.3	6.161 ± 1.753	0.603
Stride length (m)	1.06 ± 0.1	1.21 ± 0.07	0.001
Peak Pressure (N/cm ²)	27.2 ± 6.1	24.6 ± 1.5	0.207
Peak plantar flexor moment (N m/kg)	1.21 ± 0.18	1.40 ± 0.25	0.06
Peak dorsiflexor moment (N m/kg)	0.19 ± 0.12	0.15 ± 0.04	0.383
Peak ankle sagittal power (N m/(kg s))	1.64 ± 0.47	2.00 ± 0.43	0.08
Walking velocity (m/s)	0.91 ± 0.07	0.92 ± 0.05	0.343

Superscript 'KF' indicates knee flexed condition.

Table 2

Summary of associations between subject characteristics and passive ankle stiffness and DF ROM

	DM		Control	
	Stiffness	ROM	Stiffness	ROM
Age	$r = -0.36, p = 0.31$	$r = 0.46, p = 0.18$	$r = 0.28, p = 0.58$	$r = 0.19, p = 0.43$
Body mass	$r = 0.22, p = 0.54$	$r = -0.15, p = 0.64$	$r = 0.41, p = 0.20$	$r = -0.42, p = 0.18$
Duration of DM	$r = 0.55, p = 0.04$	$r = 0.33, p = 0.2$		
HbA1C (%)	$r = 0.54, p = 0.08$	$r = 0.28, p = 0.42$		

Table 3

Summary of Pearson's product moment correlation to assess relationships between variables of interest in subjects with and without DM (r_1 and r_2 , respectively)

Variables of interest		DM, r_1	Control, r_2	Z
Peak passive DF	Gait DF	-0.150	-0.007	0.270
Peak passive DF ^{KF}	Gait DF	0.145	0.158	0.025
Passive stiffness	Gait stiffness	0.075	0.472	0.843
Passive stiffness ^{KF}	Gait stiffness	-0.017	0.14	0.295
Peak passive DF	Forefoot PP	-0.512	0.457	1.981
Peak passive DF ^{KF}	Forefoot PP	0.065	-0.46	1.052
Gait DF	Forefoot PP	0.134	0.317	0.362
Passive stiffness	Forefoot PP	-0.501	0.433	1.897
Passive stiffness ^{KF}	Forefoot PP	-0.273	-0.13	0.279
Gait stiffness	Forefoot PP	0.020	0.588	1.225
Stride length	Forefoot PP	0.708	0.656	0.182
AROM	Stride length	0.732	-0.403	2.577
Gait stiffness	Stride length	0.769	-0.396	2.688

Superscript 'KF' indicates knee flexed condition; PP indicates peak pressure. The test $|Z| \geq z_{0.025}$ has a critical value of 2.57 and assesses equality of the correlations ($H_0: \rho_1 = \rho_2$).