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Foot progression angle and medial loading in individuals with diabetes mellitus, peripheral neuropathy, and a foot ulcer

Judy R Gelber¹, Elena J Isaac², Kathryn L Bohnert¹, Michael J Strube³, and David R Sinacore¹

¹Applied Kinesiology Laboratory Program in Physical Therapy, Washington University School of Medicine, St. Louis, MO, USA

²Our Children's House at Baylor, Baylor Healthcare. 1540 North Highway 77, Building C, Suite 10, Waxahachie, TX

³Department of Psychology, Washington University, St. Louis, Missouri

1. Introduction

According to recent estimates, approximately 285 million individuals worldwide have diagnosed or undiagnosed diabetes mellitus (DM).¹ Peripheral neuropathy (PN) of the sensory, motor and autonomic systems is a common complication, occurring in 60-70% of individuals with DM.² PN is a particularly debilitating complication of DM that places an individual at high risk for undetected foot injury, prolonged response to injury, and progressive foot deformity.³ Deformities that occur along the medial longitudinal arch or column of the foot (e.g. calcaneus, talus, navicular, cuneiforms 1 and 2, & metatarsals 1 and 2) are perhaps the most severe and debilitating, often resulting in collapse of the medial longitudinal arch, ulceration and amputation.⁴

Several biomechanical factors may influence the magnitude and duration of impact loading experienced by the foot during weight bearing activities (e.g. walking and standing). We believe that an increase in the foot progression angle (FPA) i.e., the angle formed between the longitudinal axis of the foot and the forward line of progression during walking, increases the load borne by the medial column support structures (e.g. bones, ligaments, muscles), thereby increasing the risk of injury to those structures.

The contribution of FPA to loading characteristics of the foot has been studied very little to date. FPA has been found to alter knee adduction moment in individuals with knee osteoarthritis.⁵ Additionally, there is a reported positive relationship between FPA and vertical pressure on the medial side of the foot in children with neuromuscular disease.⁶ However, we are aware of no previous studies exploring the relationship of FPA to medial loading of the foot in individuals with DM, PN and foot ulceration.

The purpose of this study was to determine the FPA and assess the relationship between FPA and foot medial column loading during walking in individuals with DM, PN, and a

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Hastings, MK¹ Program in Physical Therapy, Washington University School of Medicine, St. Louis, MO, USA. Corresponding Author Contact information, hastingmk@wustl.edu. Telephone number: (314) 286-1433.

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forefoot ulcer compared to age matched controls. Based on our observations of walking patterns in subjects with neuropathic plantar ulceration, we hypothesized that FPA would be positively correlated to foot medial column loading in all subjects.

2. Subjects

We studied 17 subjects (13 male, 4 female) with Type 2 DM, PN and a forefoot ulcer (DMPN) and 15 age-matched healthy controls (6 male, 9 female). (Table 1) Subjects were excluded if they had: 1) average midfoot pressures $>29 \text{ N/cm}^2$ (indication of midfoot bony deformity)³, 2) a gait pattern that did not include a hindfoot to forefoot and lateral to medial progression of the center of pressure line, 3) a history of a lower extremity fracture or deformity, or 4) any medical condition (e.g. dizziness) that affected balance and walking pattern.

In the DMPN group all subjects were diagnosed with DM by their primary care physician. All subjects in the DMPN group were prescribed medication (e.g. oral hypoglycemic agents, exogenous insulin or some combination) in addition to dietary therapy to maintain euglycemia. All ulcers were located on the medial plantar forefoot with 6 at the great toe, 7 at the 1st metatarsophalangeal joint and 4 at the 2nd or 3rd metatarsophalangeal joint (5 on the left foot and 12 on the right foot).

3. Methods

3.1 Peripheral Neuropathy Assessment

PN was assessed in both feet of all subjects using previously described and reliable methods.⁷ All DMPN subjects had evidence of severe PN; they had a current forefoot plantar ulcer and an inability to feel the 5.07 (10 gram) Semmes-Weinstein monofilament in at least one of 7 locations on the plantar aspect of the foot.

3.2 Dynamic Plantar Force Assessment

Dynamic plantar force assessments were performed bilaterally on all subjects while walking barefoot, at their preferred walking speed, over an embedded EMED-ST P-2 pressure platform (sampling frequency=50Hz, 2 sensors/cm², Novel Inc., St Paul MN, USA). A two-step method of data collection was used⁸ and a minimum of two trials of each foot were recorded. The plantar pressure map was bisected longitudinally into 2 masks, each 50% of the foot's width (Percent Mask software, Novel Inc., St. Paul MN, USA). (Figure 1)

The center of pressure (CoP) line was added to the pressure map using Percent Mask software. The total number of frames during the stance phase of walking and the frame at which the CoP line crossed from the lateral half of the foot to the medial half of the foot were recorded. Stance time was calculated by multiplying the total number of frames during stance by the 20 msec constant frame duration. Duration the CoP line spent in the medial half of the foot (TSM) was calculated by subtracting the number of frames the CoP line spent in the lateral half of the foot from the total number of frames in stance and multiplying by frame duration. (total # of frames - # of frames lateral)*(20 msec). TSM for each foot was normalized for total stance time and expressed as %TSM (%TSM=TSM/total stance time).

FPA was measured as the angle formed between the longitudinal bisector of the foot on the pressure map and a line drawn parallel to the vertical border of the printed paper, using a 2 degree increment goniometer. External rotation was recorded as a positive FPA, while internal rotation was recorded as a negative FPA. A comparison of FPA measured from Emed pressure maps to those measured from an inked moleskin walking trial found little

differ between methods (inked moleskin = $15 \pm 7^\circ$; EMED = $16 \pm 6^\circ$, $p=0.41$). The two FPA methods were highly correlated ($r=0.73$).⁹

3.3 Statistical Analysis

Group characteristics of age, weight, height, BMI and the center of pressure and stance time variables were assessed using a *t* test for independent samples. Comparison of gender between groups was made with a Chi-square test. Group comparisons were made using analysis of covariance, and included sex and BMI as predictors. Comparison of side (right and left in controls and involved and uninvolved in DMPN group) were made using repeated measures analysis of variance, controlling for sex and BMI. Correlations between variables were assessed using partial correlation, controlling for sex and BMI (2 tailed). Partial correlations for each group were compared, controlling for sex and BMI, using a Z test.¹⁰

4. Results

4.1 Subject Characteristics

The DMPN and control groups were matched for age. The DMPN group weighed more than controls [mean (SD) = 100 (17) and 78(14) kg respectively, $p<0.001$] and the male to female ratios were different between groups (13:4 and 6:9, $p=0.034$]. (Table 1)

4.2 Control Subjects' Right and Left Feet

We compared average values for stance time, FPA, TSM, and %TSM for the right and left feet of control subjects. There were no significant differences in the right and left feet of controls for stance time or TSM ($p=0.51$ and 0.078 , respectively). There was a small (2 degrees) but statistically significant difference in FPA between the right and left feet of control subjects ($p=0.038$). We do not believe this difference to be clinically meaningful and it is only slightly above the precision of our goniometer. TSM% between the right and left feet of control subjects was also significant (36(15) and 47(16) respectively, $p=0.041$). In order to minimize the normal but extraneous differences that occur between right and left sides, we averaged left and right side data for control subjects: all group comparisons used these averaged values.

4.3 DMPN and Control Subjects' Stance time, FPA and Medial Loading

The DMPN group had a significantly increased stance time on the involved and uninvolved sides [943(118) and 938(145) msec, respectively] compared to controls [794(103) msec, $p=0.0029$]. (Table 2)

FPA was increased in the DMPN group on the involved and uninvolved sides [15(9) and 13(4) degrees respectively] compared to the control group [9(4) degrees], and the difference remained significant on the uninvolved side after controlling for sex and BMI ($p=0.021$).

TSM was significantly decreased on the involved side of the DMPN group compared to the uninvolved side [252(178) and 427(228) msec, $p=0.0048$]. However, there was no difference in TSM between the control group [327(112) msec] and the DMPN group ($p>0.057$).

%TSM was significantly decreased on the involved side of the DMPN group [28(19)%] compared to the uninvolved side [47(23)%], $p=0.0072$. The values for the DMPN group did not differ significantly from the control [41(12)%], $p>0.14$.

4.4 Relationship between FPA and Medial Loading

In the DMPN group, FPA was significantly correlated with TSM and %TSM on the involved and uninvolved side ($r>0.54$, $p<0.036$). (Table 3) In contrast, FPA was weakly correlated with TSM and %TSM in the control group ($r<|0.07|$, $p>0.82$).

The Z test showed that the partial correlation between FPA and TSM for the control group was significantly different from the same partial correlation for the involved side of DMPN group. All remaining comparisons of partial correlations were not significantly different. (Table 3)

4.5 Stance Time Correlations

To assess the contribution of stance time to the group differences found in the variables of interest (FPA, TSM, and %TSM), correlations of these variables with stance time were examined. With the exception of a significant correlation between stance time and TSM on the uninvolved side ($r = 0.66$, $p = 0.0080$), stance time was weakly and not significantly related to FPA, TSM and %TSM ($r = |0.38|$, $p > 0.20$). (Table 3)

5. Discussion

Our findings provide evidence that individuals with DM, PN and a forefoot ulcer located on the medial half of the forefoot, have an increased FPA during walking. Furthermore, in the DMPN group, the increase in FPA was associated with an increase in measures of foot medial column loading (TSM and %TSM). The increase in medial column loading may place the supporting structures at a higher risk of injury, even during daily walking, which can ultimately contribute to bony instability and deformity of the medial column of the foot.

FPA was 30-40% larger in our DMPN group compared to controls. The average FPA of our control group (9°) was consistent with previously reported values ($8-9^\circ$).^{11,12} Additionally, previously reported FPA in individuals with DM, PN without an active ulcer (16°) was similar to our DMPN group ($13-15^\circ$).¹³ The increased FPA in the DMPN group may be related to their BMI categorization of obese ($BMI=32 \text{ kg/m}^2$).¹⁴ Messier et al found a 60% greater FPA in obese individuals ($BMI=41 \text{ kg/m}^2$) than controls subjects ($BMI=21 \text{ kg/m}^2$).¹⁵ The contribution of BMI to FPA was assessed. BMI was not a unique predictor of FPA in our small sample. However, controlling for BMI and gender did reduce the difference in FPA between controls and the involved side in our DMPN group to non-significance. In addition to BMI, the increase in FPA may be a compensatory mechanism adopted to increase gait stability in individuals with PN. Alternatively, an increased FPA may be a compensation for limited talocrural joint dorsiflexion and plantar flexion motion, a commonly encountered impairment in individuals with DM, PN, and foot complications.¹⁶ Future study of individuals with DM and PN is warranted to understand the unique contributions of obesity, disease, and anatomical impairments to FPA.

The significant positive correlation between FPA and the medial loading variables (TSM and %TSM) in our DMPN group indicates that as the FPA increases, more time is spent in the medial column of the foot during walking. The correlation was strongest on the involved side ($r = 0.64$) and slightly weaker on the uninvolved side ($r = 0.54$). There was little correlation between FPA and medial loading variables in the control group ($r = |0.07|$). We hypothesize that in control subjects, the anatomical structure of the foot is intact and dictates how the foot is loaded regardless of the FPA. In contrast, we hypothesize that the structural stability of the foot is compromised in the DMPN group allowing FPA to impact how the foot is loaded. The relationship between structural integrity, deformity, and loading patterns in individuals with DM, PN is poorly understood and is the focus of our future investigations.

The DMPN group had a 15-16% increase in stance time compared to controls. Longer stance time was reflective of a slower preferred gait speed, common in this population.¹⁷ Differences in stance time may have influenced FPA and the temporal measure of medial column loading. We explored the contribution of stance time to our study by normalizing TSM (%TSM) and determined the correlations between stance time and our variables of interest. With the exception of a significant correlation between stance time and TSM on the uninvolved side, stance time was weakly and not significantly related to FPA, TSM, or %TSM. Given the lack of significant relationships between stance time and our measures of medial loading and the consistent findings of association between FPA and measures of medial loading when controlling for stance time, we feel confident that stance time does not play a substantial role in the group differences found in this study.

It is an interesting finding of this study that TSM and %TSM were lowest on the involved side in the DMPN group. We hypothesized that TSM and %TSM would be increased in our DMPN group. The low values for TSM and %TSM could be the result of a compensatory gait strategy to decrease loading on the plantar ulcer. Given the reduction in sensory feedback from peripheral neuropathy, the compensation might be driven by a cognitive awareness of ulcer location and development of a habitual walking pattern. This finding could also be the result of excluding individuals with DM and PN who had severe midfoot deformity (midfoot pressure >29 N/cm²). We are currently investigating the loading patterns of individuals with DM and PN who have severe medial midfoot deformity to further understand how medial loading changes as the deformity progresses.

The primary limitation of this study is a lack of long-term follow up and we are unable to determine whether deformity progression is related to the biomechanical differences. Future work to follow individuals throughout the course of their disease could yield important information about risk factors related to injury and foot deformity progression. Additionally, we did not assess the anatomical source of excessive FPA (e.g. tibial or femoral torsion, rotation at the knee or hip) which would assist in directing the patient in the appropriate correction of the excessive FPA during walking.

6. Conclusion

This study provides evidence that individuals with DM, PN, and a forefoot ulcer have an increased FPA and that there is a strong correlation between FPA and foot medial column loading. We believe the pattern of loading has significant clinical implications, increasing the risk of midfoot injury and medial column breakdown. Our data might suggest that one step in prevention of medial column deformities would be to train patients to decrease FPA thereby decreasing the medial loading of the foot. Such an intervention requires further validation.

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Figure 1.

A. Plantar pressure map of the left foot with a line (green) bisecting the foot through the longitudinal axis of the foot to create medial and lateral masks. Center of pressure line (aqua) has also been applied. B. The foot progression angle (orange) was measured as the angle formed between the line bisecting the foot through the longitudinal axis of the foot on the pressure map and a line drawn parallel to the vertical border of the printed paper. The vertex of the angle was placed at the intersection of the lines drawn.

Figure 2.

Plot of foot progression angle and % of total stance time spent medial for the control group, the involved side of the DMPN group, and the uninvolved side of the DMPN group.

Table 1

Subject Demographic Data, reported as mean (standard deviation)

	Age	Weight (kg)	BMI (kg/m ²)	Gender (male/female)	Duration of DM (year)	HbA1c
DMPN	56 (11)	100 (17)	32 (5)	13/4	16(10)	8.65(1.98)
Controls	55(9)	78 (14)	27 (4)	6/9	NA	NA
P values	0.83	<0.001 *	0.0036 *	0.034 *		

* denotes significant findings, $p < 0.05$

Table 2

The mean and standard deviation of foot progression angle (FPA), time spent in the medial mask of the foot (TSM), and percent of total stance time spent in the medial mask (%TSM) between individuals with diabetes mellitus, peripheral neuropathy, and a forefoot ulcer (DMPN) and the control group. P values for group and side comparisons are provided with and without adjusting for BMI and Sex.

	Control	DMPN involved	DMPN uninvolved	P values (Without Adjusting for BMI and Sex)		P values (Adjusting for BMI and Sex)		
				Involved v. control	Uninvolved v. control	Involved v. control	Uninvolved v. control	Involved v. uninvolved
Stance Time (ms)	794(103)	943(118)	938(145)	<0.001 *	0.0034 *	0.0018 *	0.0029 *	0.81
FPA (Degree)	9(4)	15(9)	13(4)	0.022 *	0.0035 *	0.26	0.021 *	0.47
TSM (ms)	327(112)	252(178)	427(228)	0.17	0.14	0.32	0.057	0.0048 *
%TSM (%)	41(12)	28(19)	47(23)	0.027 *	0.41	0.14	0.18	0.0072 *

* denotes significant findings, $p < 0.05$.

Table 3

Table of partial correlations (adjusting for BMI and Sex) and the Z test for comparison of the independent partial correlations. Foot progression angle (FPA), time spent medial (TSM), percent of stance time spent medial (%TSM), group with diabetes mellitus and peripheral neuropathy (DMPN). $\alpha=0.05$ and critical value for the Z test is 1.96 ($p<0.05$, two-tailed test). Significance levels are in parentheses.

Comparison	Controls	DMPN Involved	DMPN Uninvolved	Z Test for Comparison of Independent Correlations	
				Controls vs Involved	Controls vs Uninvolved
FPA and TSM	-0.07	0.66 *	0.54 *	2.00 *	1.59
	(0.82)	(0.0078)	(0.036)		
FPA and %TSM	-0.01	0.64 *	0.62 *	1.80	1.71
	(0.97)	(0.010)	(0.014)		
Stance time and FPA	-0.12	-0.14	0.16	-0.05	0.67
	(0.69)	(0.61)	(0.56)		
Stance time and TSM	0.38	-0.18	0.66 *	-1.35	0.90
	(0.20)	(0.53)	(0.0080)		
Stance time and %TSM	-0.16	-0.36	0.17	-0.49	0.77
	(0.60)	(0.19)	(0.55)		

* $p<0.05$.