

Provided for non-commercial research and education use.
Not for reproduction, distribution or commercial use.



This article appeared in a journal published by Elsevier. The attached copy is furnished to the author for internal non-commercial research and education use, including for instruction at the authors institution and sharing with colleagues.

Other uses, including reproduction and distribution, or selling or licensing copies, or posting to personal, institutional or third party websites are prohibited.

In most cases authors are permitted to post their version of the article (e.g. in Word or Tex form) to their personal website or institutional repository. Authors requiring further information regarding Elsevier's archiving and manuscript policies are encouraged to visit:

<http://www.elsevier.com/copyright>



Contents lists available at ScienceDirect

Gait & Posture

journal homepage: www.elsevier.com/locate/gaitpost

Relationships between segmental foot mobility and plantar loading in individuals with and without diabetes and neuropathy

Smita Rao^{a,*}, Charles L. Saltzman^b, H. John Yack^c

^a Department of Physical Therapy, New York University, 380 2nd Ave, 4th Floor, New York, NY 10010, United States

^b Department of Orthopedics, University of Utah, Salt Lake City, UT, United States

^c Graduate Program in Physical Therapy and Rehabilitation Sciences, The University of Iowa, Iowa City, IA, United States

ARTICLE INFO

Article history:

Received 28 July 2009

Received in revised form 12 October 2009

Accepted 25 October 2009

Keywords:

Diabetes

Foot

Segmental

Loading

ABSTRACT

The purpose of our study was to examine dynamic foot function during gait as it relates to plantar loading in individuals with DM (diabetes mellitus and neuropathy) compared to matched control subjects. Foot mobility during gait was examined using a multi-segment kinematic model, and plantar loading was measured using a pedobarograph in subjects with DM ($N = 15$), control subjects ($N = 15$). Pearson product moment correlation was used to assess the relationship between variables of interest. Statistical significance and equality of correlations were assessed using approximate tests based on Fisher's Z transformation ($\alpha = 0.05$). In individuals with DM, first metatarsal sagittal plane excursion during gait was negatively associated with pressure time integral under the medial forefoot ($r = -0.42$ and -0.06 , DM and Ctrl, $P = 0.02$). Similarly, lateral forefoot sagittal plane excursion during gait was negatively associated with pressure time integral under the lateral forefoot ($r = -0.56$ and -0.11 , DM and Ctrl, $P = 0.02$). Frontal plane excursion of the calcaneus was negatively associated with medial ($r = -0.57$ and 0.12 , DM and Ctrl, $P < 0.01$) and lateral ($r = -0.51$ and 0.13 , DM and Ctrl, $P < 0.01$) heel and medial forefoot pressure time integral ($r = -0.56$ and -0.02 , DM and Ctrl, $P < 0.01$). The key findings of our study indicate that reductions in segmental foot mobility were accompanied by increases in local loading in subjects with DM. Reduction in frontal plane calcaneal mobility during walking serves as an important functional marker of loss of foot flexibility in subjects with DM.

© 2009 Elsevier B.V. All rights reserved.

1. Introduction

With over 7% of the population in the United States affected by diabetes mellitus (DM), DM has emerged as a significant health problem [1]. In individuals with DM, chronic hyperglycemia results in increased protein glycosylation in multiple organ systems which manifests clinically as neuropathy, atherosclerosis and increased connective tissue stiffness. The ensuing compromises in sensation, and mobility, when combined with repetitive mechanical loads have been linked to the development of plantar ulcers in individuals with DM [2]. Plantar ulcers occur in approximately 15% of all individuals with DM [3]. Foot ulcers account for considerable morbidity and mortality, and 9% of all health care costs incurred by people with diabetes are attributable to the management of DM and its complications [3]. Factors contributing to plantar loads during routine functional activities such as walking, are therefore of considerable interest.

During walking, normal foot mobility allows the foot to transition from a flexible structure that dissipates impact as it contacts the ground to a rigid structure that allows for efficient propulsion during push off [4]. Restricted joint mobility in the small joints of the foot has been postulated to limit the ability of the foot to deform, to hinder forward progression of body weight [5] and contribute to the development of abnormal plantar loads at susceptible sites [5–8].

Previous reports have documented that individuals with DM have reduced passive subtalar joint mobility [5,7] as a result of which, they may be expected to demonstrate reduced calcaneal eversion range of motion during walking. During walking, loss of calcaneal eversion range of motion may be expected to result in decreased forefoot mobility because calcaneal eversion is believed to “unlock” the midfoot and allow greater distal (forefoot) mobility [9]. Loss of calcaneal eversion range of motion has also been hypothesized to alter plantar load distribution patterns, reflected in increased callus formation at sites of greater load [10]. However no objective data exist examining the relationship between calcaneal eversion range of motion and plantar load distribution during walking.

In addition to the subtalar joint, limited joint mobility has been reported at the first ray in individuals with DM [6,8]. Reduced first

* Corresponding author. Tel.: +1 212 998 9194; fax: +1 212 995 4190.

E-mail address: smita.rao@nyu.edu (S. Rao).

ray mobility, attributed in part to increased plantar fascia thickness, has been reported in individuals with DM [11]. Increased plantar fascia thickness and reduced passive first ray mobility may explain the reduction in sagittal plane first ray motion noted in individuals with DM during walking. Limited sagittal plane first ray motion during walking has been hypothesized to result in increased medial forefoot loading [6,8] quantitative data elucidating this relationship are lacking.

Evidence confirming the functional consequences of limited joint mobility and increased stiffness in the foot is limited, partly due to the lack of biomechanical models that track *in vivo* segmental motion of the foot during walking. In the absence of segmental foot models, regression-based statistical models have been implemented to determine predictors of loading [12,13]. Regression-based models provide valuable insights and help identify etiological factors on the basis of how much variance they explain in the dependent variable. However, they do not shed light on the mechanisms by which abnormal plantar loads develop.

Results from recent studies that have used multi-segment kinematic models of the foot during routine functional activities have not addressed the relationship between *in vivo* foot mobility and plantar loading during walking [14–16]. Further, these studies were conducted on non-DM subjects with intact sensation, their extrapolation to individuals with DM and neuropathy may not be valid.

While segmental foot mobility during walking has been identified as an important potential contributor to plantar loads, especially in individuals with DM [6,7,11], no objective data exist characterizing the relationship between segmental foot mobility during walking and sustained regional plantar loads. These data may help elucidate mechanisms contributing to plantar load during walking in individuals with DM. The purpose of this study is to examine the relationship between segmental foot mobility and sustained regional plantar loading during walking in individuals with DM, compared with matched control subjects.

2. Methods

2.1. Subjects

All procedures were approved by the Institutional Review Board at the University of Iowa Hospitals and Clinics. Individuals with DM and neuropathy ($N = 15$) comprised the study group; non-diabetic subjects ($N = 15$) comprised the control (Ctrl) group. Inclusion criteria for subjects with DM were: diagnosis of DM based on American Diabetes Association criteria [17], no current foot ulcer, great toe or transmetatarsal amputation, and an absence of Charcot neuroarthropathy. Presence of neuropathy was documented using 5.07 Semmes–Weinstein monofilaments and vibration perception threshold of 25 V or higher [18]. The control group comprised non-diabetic subjects, without any conditions that may influence walking performance. Subjects in the control group were screened for diabetes, and matched in age and gender to subjects with DM (Table 1).

2.2. Data acquisition

Kinematic and kinetic data were acquired as subjects walked along a 10 m walkway at 0.89 m/s (2 mph) [18]. All individuals with diabetes and neuropathy walked at self-selected speed. Control subjects' speed was matched to that of the

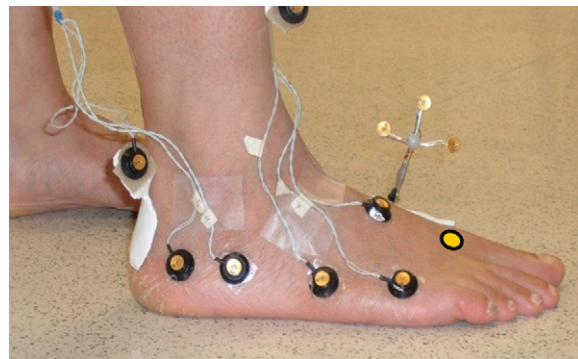


Fig. 1. Kinematic model used to track segmental foot motion during walking.

individuals with diabetes and neuropathy. Kinematic data were collected at 120 Hz using an active marker system (Optotrak, NDI, Waterloo, Canada). Kinetic data were collected at 360 Hz using a forceplate embedded in the walkway (Kistler Inc., Amherst, NY) and at 50 Hz using a pedobarograph (EMed, Novel Inc., St. Paul, MN). Five successful trials each were collected. A trial was considered successful if the subject made clean forceplate or pedobarograph contact on the tested side, without targeting.

2.3. Multi-segment kinematic model of the foot

A four segment kinematic model, developed in previous investigations in our lab [18] was used to assess *in vivo* segmental foot motion during walking. The foot model included the following segments: leg, calcaneus, first metatarsal, each of which represented the underlying bony segment. In addition, the lateral forefoot, defined by metatarsals two through five, was tracked (Fig. 1). These definitions, consistent with previous kinematic models [14–16,18] were used to facilitate comparison of results across studies, and target segments of interest. Each segment was tracked using three infra-red markers placed in a non-collinear arrangement. The three markers on each segment were used to define technical co-ordinate systems. Subject-specific, anatomically based local co-ordinate systems were established by defining palpable landmarks as virtual points with respect to the technical co-ordinate systems [18].

2.4. Data analysis

Kinematic data were low-pass filtered using a fourth-order butterworth filter with cut-off frequency of 6 Hz, and processed using Visual3D (C-motion Inc., MD). Motion of the distal segment was expressed relative to the proximal segment, and was calculated using Euler angles with the following sequence of rotations: sagittal, frontal and transverse. Peak motion as well as excursion (peak to peak range of motion) were computed for the following segments: first metatarsal relative to the calcaneus, lateral forefoot relative to the calcaneus and calcaneus relative to the tibia. Processed data were time normalized to 100% stance. For kinematic data, stance phase mean was subtracted from pattern to correct for systematic offsets [14,18].

Kinetic data were low-pass filtered using a fourth-order butterworth filter with a cut-off frequency of 8 Hz. Ankle joint plantarflexor moment and power were computed using an inverse dynamics approach.

Plantar pressure data were processed using Novel Win software (Novel, St. Paul, MN). The following sub-areas of interest were defined as a percentage of foot length: heel (0–28%), midfoot (28–55%), forefoot (55–80%). Each sub-area was divided into medial and lateral (50% foot width). Plantar loading was characterized using peak pressure, pressure time integral and timing of peak loading within each sub-area.

2.5. Statistical analysis

A two-sample *t*-test was used to assess differences in kinematic and kinetic dependent variables between the groups ($\alpha = 0.05$). Pearson product moment correlation (r) was used to assess the relationship between foot mobility and sustained regional plantar loading. 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

Subjects in both groups walked with similar speed (0.89 ± 0.13 and 0.93 ± 0.11 m/s, DM and Ctrl, respectively, $P = 0.169$) and stride length (1.08 ± 0.15 and 1.12 ± 0.10 m, DM and Ctrl, respectively, $P = 0.166$).

Table 1

Demographic data from DM and control groups, expressed as mean \pm SD.

	DM	Control
N	15	15
Age	58 \pm 11	56 \pm 12
Gender F:M	5:10	5:10
Height (m)	1.77 \pm 0.11	1.75 \pm 0.10
Mass (kg)	90.6 \pm 13.8	74.6 \pm 13.3
VPT	48 \pm 5	13 \pm 6
HbA1C	8.1 \pm 1.1	
Type 2	12 (80%)	
Duration (years)	19 \pm 6	

VPT refers to vibration perception threshold; HbA1C refers to glycated hemoglobin.

3.1. Kinematics

Individuals with DM showed decreased excursion of the first metatarsal relative to the calcaneus in the frontal plane (9.9 ± 3.7 and 12.3 ± 3.2 , DM and Ctrl, respectively, $P = 0.029$) as well as transverse plane (7.1 ± 3.1 and 9.6 ± 3.6 , DM and Ctrl, respectively, $P = 0.026$). Neither peak dorsiflexion (6.5 ± 3.8 and 5.6 ± 1.9 , DM and Ctrl, respectively, $P = 0.147$) nor sagittal plane excursion (13.0 ± 2.5 and 14.7 ± 3.3 , DM and Ctrl, respectively, $P = 0.270$) of the first metatarsal relative to the calcaneus differed between DM and Ctrl groups. Trends towards reduced frontal plane excursion of the lateral forefoot relative to the calcaneus were noted in individuals with DM (13.6 ± 5.3 and 16.7 ± 5.2 , DM and Ctrl, respectively, $P = 0.062$). Individuals with DM showed reduced sagittal (12.7 ± 4.3 and 19.6 ± 4.4 , DM and Ctrl, respectively, $P < 0.001$) and frontal plane (9.5 ± 4.3 and 15.0 ± 3.9 , DM and Ctrl, respectively, $P < 0.001$) excursion of the calcaneus relative to the tibia.

3.2. Kinetics and regional plantar loading

Although individuals with DM showed less plantarflexor torque (1.27 ± 0.17 and 1.40 ± 0.17 Nm/kg, DM and Ctrl, respectively, $P = 0.03$) and less plantarflexor power generation at push off (1.52 ± 0.60 and 2.51 ± 0.49 Nm/kg s, DM and Ctrl, respectively, $P < 0.01$), they sustained significantly higher plantar pressure time integral under the medial forefoot (Table 2). In addition, heel rise occurred later in individuals with DM ($66.3 \pm 12.4\%$ and $57.0 \pm 10.3\%$ stance, DM and Ctrl, respectively, $P = 0.016$), and individuals with DM showed significantly longer forefoot contact time (Table 3). Similar trends were noted with peak pressure (Table 4). There were no differences in the timing of peak pressure at the heel ($16.9 \pm 7.7\%$ and $15.1 \pm 6.4\%$ stance, DM and Ctrl, respectively, $P = 0.243$) or the forefoot ($72.7 \pm 17.3\%$ and $78.3 \pm 4.6\%$ stance, DM and Ctrl, respectively, $P = 0.112$) between the groups.

3.3. Associations

In individuals with DM, first metatarsal sagittal plane excursion during gait was negatively associated with pressure time integral sustained under the medial forefoot ($r = -0.42$ and -0.06 , DM and Ctrl, respectively, $P = 0.02$). Similarly, lateral forefoot sagittal plane excursion during gait was negatively associated with pressure time integral sustained under the lateral forefoot ($r = -0.56$ and -0.11 , DM and Ctrl, respectively, $P = 0.02$). Frontal plane excursion of the calcaneus was negatively associated with medial ($r = -0.57$ and 0.12 , DM and Ctrl, respectively, $P < 0.01$) and lateral ($r = -0.51$ and 0.13 , DM and Ctrl, respectively, $P < 0.01$) heel and medial forefoot pressure time integral ($r = -0.56$ and -0.02 , DM and Ctrl, respectively, $P < 0.01$).

Table 2

Peak plantar loading sustained during gait, under each region of the foot, in DM and control groups.

	DM	Control	P value
Pressure time integral ($[N/cm^2]s$)			
Medial heel	20.1 ± 11.1	10.3 ± 4.0	0.001
Lateral heel	16.8 ± 10.0	10.8 ± 4.2	0.019
Medial forefoot	36.2 ± 13.5	21.1 ± 9.2	0.001
Lateral forefoot	26.9 ± 6.8	19.5 ± 6.9	0.003
Pressure time integral (normalized to body weight)			
Medial heel	0.221 ± 0.106	0.138 ± 0.048	0.004
Lateral heel	0.185 ± 0.099	0.144 ± 0.047	0.072
Medial forefoot	0.410 ± 0.171	0.289 ± 0.126	0.017
Lateral forefoot	0.301 ± 0.076	0.257 ± 0.060	0.040

Table 3

Contact duration in ms (% stance time) of loading during gait, under each region of the foot, in DM and control groups.

	DM	Control	P value
Heel	701.3 ± 322.4 (59.8)	583.8 ± 220.6 (60.8)	0.102
Forefoot	1046.7 ± 424.0 (89.2)	797.5 ± 124.8 (83.1)	0.005
Hallux	681.3 ± 311.4 (58.1)	602.5 ± 194.0 (62.8)	0.201
Total	1173.3 ± 409.2	1060.0 ± 345.8	0.073

4. Discussion

We applied a multi-segment kinematic model to examine mechanisms contributing to sustained plantar loading during gait. The key findings of our study indicate that, in spite of reduced kinetic demands placed on the foot, evidenced as decreased ankle moment and power, sustained plantar loading was increased in patients with diabetes, compared to the control group. The findings of this study also identified key elements of segmental motion that may contribute to the sustained regional plantar loading. In individuals with DM, range of sagittal motion of the first metatarsal and lateral forefoot, and range of frontal motion of the calcaneus were negatively associated with the magnitude of sustained plantar loading under the respective segment. Both groups had similar standard deviations and dispersion, however only individuals with DM showed increases in sustained plantar loading associated with decreases in mobility. These findings highlight the importance of segmental foot mobility in individuals with DM and suggest possible mechanisms underlying the evolution of increased plantar loading under susceptible sites.

Previous studies have used peak pressure and pressure time integral as a measure of plantar stress and consequent tissue trauma in patients with diabetes [19,20]. In the current study, pressure time integral, calculated as the area under the peak pressure curve, of gait was used to delineate plantar stress because it represents both, the magnitude and duration of plantar loading through stance phase. Based on Physical Stress Theory, higher pressure time integrals may be indicative of increased plantar stress [21]. In agreement with this contention, patients with diabetes demonstrated significantly greater medial forefoot pressure time integral (effect size = 1.3), which may be attributed to a combination of increased body mass (effect size = 0.93), increased contact time (effect size = 0.84) and peak medial forefoot pressure (effect size = 0.6). Pressure time integral or sustained plantar loading thus effectively captured net forefoot plantar stress during walking in patients with diabetes and neuropathy.

Recent reports have demonstrated that ankle plantarflexor power at push off may modulate forefoot plantar loading [19]. While patients in the current study attempted to reduce kinetic demands placed on the ankle at push off, reflected as reduced ankle moment and power, sustained plantar loading continued to be

Table 4

Peak plantar pressure during gait, sustained under each region of the foot, in DM and control groups.

	DM	Control	P value
Peak pressure (N/cm^2)			
Medial heel	45.9 ± 17.1	37.0 ± 8.5	0.04
Lateral heel	44.2 ± 16.3	38.2 ± 9.0	0.11
Medial forefoot	83.9 ± 34.7	65.7 ± 27.5	0.06
Lateral forefoot	62.1 ± 34.1	58.2 ± 29.3	0.37
Peak pressure (% body weight/ cm^2)			
Medial heel	$5.4 \pm 2.5\%$	$5.2 \pm 1.2\%$	0.38
Lateral heel	$5.0 \pm 2.7\%$	$5.3 \pm 1.2\%$	0.33
Medial forefoot	$9.8 \pm 4.8\%$	$9.3 \pm 4.2\%$	0.38
Lateral forefoot	$7.1 \pm 3.9\%$	$7.8 \pm 3.1\%$	0.31

high in patients with DM, compared to the control group. Taken together, these findings highlight the role of factors intrinsic to the foot, such as segmental foot mobility, in contributing to sustained plantar loading in patients with DM.

All patients in the current study had DM for greater than 10 years and also had significant neuropathy, reflected as loss of protective sensation. Due to the presence of these two factors, all the patients in our cohort may be identified as having at least two high-risk foot conditions [5]. Our cohort with DM and neuropathy was similar in age, body mass index, gender distribution, type and duration of DM compared to previous studies [5,6]. Consistent with previous studies [5,6], individuals with DM had higher BMI than control subjects. To minimize confounding due to BMI, sustained plantar loading was normalized to body weight. During walking, heel contact, which marks the beginning of foot floor interaction, was followed by rapid calcaneal eversion and plantar loading at the heel, in both groups, consistent with previous reports [14,16]. In agreement with previous reports describing the magnitude and timing of heel loading, our data showed that peak plantar loading at the heel occurs in early stance [22].

In individuals with DM, we found decreased sagittal and frontal plane excursion (eversion) of the calcaneus [18]. Several factors, including increased subtalar joint stiffness [5,7], loss of heel pad compliance [23], increased plantar fascia stiffness [11] and neuropathy [5] may contribute to the reduction in calcaneal excursion noted in individuals with DM. Decreases in calcaneal frontal plane excursion were associated with increases in sustained heel and medial forefoot loading in individuals with DM, but not in the control group. These findings support the theory that frontal plane calcaneal mobility plays a key role in mediating plantar load distribution [10] particularly in individuals with DM.

Reduction in calcaneal eversion excursion may contribute to decreased forefoot mobility because calcaneal eversion is believed to “unlock” the midfoot [9,24]. Consistent with the former contention, we found reduced frontal and transverse plane excursion of the first metatarsal relative to the calcaneus and trends towards reduced frontal plane excursion of the lateral forefoot relative to the calcaneus, in individuals with DM but not in matched control subjects. Contrary to the theory that reduced calcaneal eversion may result in a ‘locked’ midfoot, neither peak sagittal plane motion nor excursion of the first metatarsal or the lateral forefoot relative to the calcaneus differed between individuals with DM and matched control subjects. These results may suggest that frontal and transverse plane excursion may be affected before sagittal plane motion.

Our findings demonstrated that sagittal plane excursion of the first metatarsal relative to the calcaneus explained a small but significant portion (approximately 17%) of the variance in sustained medial forefoot loading, in individuals with DM. A fair relationship was found between lateral forefoot sagittal plane excursion during walking and lateral forefoot plantar loading in individuals with DM ($r^2 = 0.31$). Previous studies using regression-based models to examine predictors of medial forefoot loading explained 47–48% of the variance in medial forefoot loading using a combination of static and dynamic factors [12,13]. Of these factors, static or structural factors accounted for 38–46% of the variance in medial forefoot loading [13,25]. The addition of dynamic variables explained an additional 10% of the variance in medial forefoot plantar loading in non-diabetic individuals [12]. However, previous studies did not include segmental foot mobility as a predictor of regional plantar loading. Based on the findings of the current study, sagittal plane excursion of the first metatarsal and lateral forefoot relative to the calcaneus may be key determinants contributing to sustained forefoot loading in individuals with DM.

The reduction in segmental mobility in individuals with DM may stem from changes in joint stiffness and increased plantar fascia stiffness [11]. Due to its attachments on the calcaneus and on the ball of the foot [26], plantar fascia strain is closely related to segmental foot mobility [27]. Increased plantar fascia stiffness may also explain prolonged forefoot contact time and consequent increase in sustained forefoot plantar loading in patients with DM.

Increased plantar fascia stiffness and forefoot loading may signal that greater bending moments are sustained at the apex of the arch [28]. The combination of increased forefoot loading and reduced frontal and transverse plane forefoot mobility introduce the possibility of increased torsional moments about the midfoot. Increases in plantar loading thus have implications for not only tissue breakdown and potential ulcer development, but also the evolution of Charcot changes at the midfoot, due to both, bending and torsional stresses.

The chief limitation of this study is the relatively small sample size. Future studies are indicated to develop comprehensive regression models that include segmental foot mobility among other predictors of plantar loading, in larger patient cohorts. Alternative approaches, including the use of a static standing offset, or other reference positions, such as subtalar neutral, may be valuable in examining absolute peak foot mobility in this population.

5. Conclusions

The key findings of our study were that, in individuals with DM, sagittal motion of the first metatarsal and lateral forefoot and frontal motion of the calcaneus were negatively associated with the magnitude of sustained plantar loading under the respective segment. Reduction in frontal plane calcaneal mobility during walking serves as an important functional marker of loss of foot flexibility in individuals with DM.

Acknowledgments

This study was supported in part by the following grants: RO1 NR07721-03 and M01 RR00059 from the General Clinical Research Centers Program, NCMRR, NIH.

Conflict of interest statement

We, the authors of this manuscript, affirm that we have no financial affiliation (including research funding) or involvement with any commercial organization that has a direct financial interest in any matter included in this manuscript except as cited in the manuscript.

References

- [1] U.S. Department of Health and Human Services and Centers for Disease Control and Prevention. National diabetes fact sheet: general information and national estimates on diabetes in the United States; 2007.
- [2] Cavanagh PR, Simoneau GG, Ulbrecht JS. Ulceration, unsteadiness, and uncertainty: the biomechanical consequences of diabetes mellitus. *J Biomech* 1993;26(Suppl. 1):23–40.
- [3] Gordois A, Scuffham P, Shearer A, Oglesby A, Tobian JA. The health care costs of diabetic peripheral neuropathy in the US. *Diabetes Care* 2003;26:1790–5.
- [4] Saltzman CL, Nawoczenski DA. Complexities of foot architecture as a base of support. *J Orthop Sports Phys Ther* 1995;21:354–60.
- [5] Mueller MJ, Diamond JE, Delitto A, Sinacore DR. Insensitivity, limited joint mobility, and plantar ulcers in patients with diabetes mellitus. *Phys Ther* 1989;69:453–9 [discussion 459–62].
- [6] Birke JA, Franks BD, Foto JG. First ray joint limitation, pressure, and ulceration of the first metatarsal head in diabetes mellitus. *Foot Ankle Int* 1995;16:277–84.
- [7] Fernando DJ, Masson EA, Veves A, Boulton AJ. Relationship of limited joint mobility to abnormal foot pressures and diabetic foot ulceration. *Diabetes Care* 1991;14:8–11.
- [8] Glasoe WM, Allen MK, Ludewig PM, Saltzman CL. Dorsal mobility and first ray stiffness in patients with diabetes mellitus. *Foot Ankle Int* 2004;25:550–5.

- [9] Blackwood CB, Yuen TJ, Sangeorzan BJ, Ledoux WR. The midtarsal joint locking mechanism. *Foot Ankle Int* 2005;26:1074–80.
- [10] Tiberio D. Pathomechanics of structural foot deformities. *Phys Ther* 1988;68:1840–9.
- [11] D'Ambrogi E, Giurato L, D'Agostino MA, Giacomozzi C, Macellari V, Caselli A, et al. Contribution of plantar fascia to the increased forefoot pressures in diabetic patients. *Diabetes Care* 2003;26:1525–9.
- [12] Morag E, Cavanagh PR. Structural and functional predictors of regional peak pressures under the foot during walking. *J Biomech* 1999;32:359–70.
- [13] Mueller MJ, Hastings M, Commean PK, Smith KE, Pilgram TK, Robertson D, et al. Forefoot structural predictors of plantar pressures during walking in people with diabetes and peripheral neuropathy. *J Biomech* 2003;36:1009–17.
- [14] Hunt AE, Smith RM, Torode M, Keenan AM. Inter-segment foot motion and ground reaction forces over the stance phase of walking. *Clin Biomech (Bristol Avon)* 2001;16:592–600.
- [15] Tome J, Nawoczenski DA, Flemister A, Houck J. Comparison of foot kinematics between subjects with posterior tibialis tendon dysfunction and healthy controls. *J Orthop Sports Phys Ther* 2006;36:635–44.
- [16] Leardini A, Benedetti MG, Berti L, Bettinelli D, Nativo R, Giannini S. Rear-foot, mid-foot and fore-foot motion during the stance phase of gait. *Gait Posture* 2007;25:453–62.
- [17] Diagnosis and classification of diabetes mellitus. *Diabetes Care* 2006;29 (Suppl. 1):S43–8.
- [18] Rao S, Saltzman C, Yack HJ. Segmental foot mobility in individuals with and without diabetes and neuropathy. *Clin Biomech (Bristol Avon)* 2007;22:464–71.
- [19] Maluf KS, Mueller MJ, Strube MJ, Engsborg JR, Johnson JE. Tendon Achilles lengthening for the treatment of neuropathic ulcers causes a temporary reduction in forefoot pressure associated with changes in plantar flexor power rather than ankle motion during gait. *J Biomech* 2004;37:897–906.
- [20] Veves A, Van Ross ER, Boulton AJ. Foot pressure measurements in diabetic and nondiabetic amputees. *Diabetes Care* 1992;15:905–7.
- [21] Mueller MJ, Maluf KS. Tissue adaptation to physical stress: a proposed “Physical Stress Theory” to guide physical therapist practice, education, and research. *Phys Ther* 2002;82:383–403.
- [22] Bryant AR, Tinley P, Singer KP. Normal values of plantar pressure measurements determined using the EMED-SF system. *J Am Podiatr Med Assoc* 2000;90:295–9.
- [23] Kao PF, Davis BL, Hardy PA. Characterization of the calcaneal fat pad in diabetic and non-diabetic patients using magnetic resonance imaging. *Magn Reson Imaging* 1999;17:851–7.
- [24] Elftman H. The transverse tarsal joint and its control. *Clin Orthop* 1960;16:41–6.
- [25] Cavanagh PR, Morag E, Boulton AJ, Young MJ, Deffner KT, Pammer SE. The relationship of static foot structure to dynamic foot function. *J Biomech* 1997;30:243–50.
- [26] Bojsen-Moller F, Flagstad KE. Plantar aponeurosis and internal architecture of the ball of the foot. *J Anat* 1976;121:599–611.
- [27] Flanigan RM, Nawoczenski DA, Chen L, Wu H, DiGiovanni BF. The influence of foot position on stretching of the plantar fascia. *Foot Ankle Int* 2007;28: 815–22.
- [28] Trepman E, Nihal A, Pinzur MS. Current topics review: Charcot neuroarthropathy of the foot and ankle. *Foot Ankle Int* 2005;26:46–63.