

Contribution of Plantar Fascia to the Increased Forefoot Pressures in Diabetic Patients

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OBJECTIVES — Secondary to peripheral neuropathy, plantar hyperpressure is a proven risk factor for foot ulceration. But limited joint mobility (LJM) and soft tissue abnormalities may also contribute. The aim of this study was to evaluate the relationships among thickness of plantar fascia, mobility of the metatarso-phalangeal joint, and forces expressed under the metatarsal heads.

RESEARCH DESIGN AND METHODS — We evaluated 61 diabetic patients: 27 without neuropathy (D group), 19 with neuropathy (DN group), and 15 with previous neuropathic foot ulceration (DNPU group). We also examined 21 control subjects (C). Ultrasound evaluation was performed with a high resolution 8- to 10-MHz linear array (Toshiba Tosbee SSA 240). The foot loading pattern was evaluated with a piezo-dynamometric platform. First metatarso-phalangeal joint mobility was assessed with a mechanic goniometer.

RESULTS — Diabetic patients presented increased thickness of plantar fascia (D 2.9 ± 1.2 mm, DN 3.0 ± 0.8 mm, DNPU 3.1 ± 1.0 mm, and C 2.0 ± 0.5 mm; $P < 0.05$), and significantly reduced motion range at the metatarso-phalangeal joint (D $54.0 \pm 29.4^\circ$, DN $54.9 \pm 17.2^\circ$, DNPU $46.8 \pm 20.7^\circ$, and C $100.0 \pm 10.0^\circ$; $P < 0.05$). The evaluation of foot-floor interaction under the metatarsal heads showed increased vertical forces in DN and DNPU and increased medio-lateral forces in DNPU. An inverse correlation was found between the thickness of plantar fascia and metatarso-phalangeal joint mobility ($r = -0.53$). The thickness of plantar fascia was directly correlated with vertical forces under the metatarsal heads ($r = 0.52$).

CONCLUSIONS — In diabetic patients, soft tissue involvement may contribute to the increase of vertical forces under the metatarsal heads. Changes in the structure of plantar fascia may also influence the mobility of the first metatarso-phalangeal joint.

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In diabetic patients, peripheral neuropathy induces foot deformities and changes in the walking pattern that are responsible for the development of areas of high plantar pressure (1–3).

Mechanical stress has been recognized to have a central pathogenic role in the onset of diabetic neuropathic ulcers (4). Among the factors that may contribute to increase mechanical stress, limited

joint mobility (LJM) of the ankle joint is associated with increased peak forefoot pressures, and therefore with risk of ulceration (5,6). Atrophy and weakness of tibialis anterior and vastus lateralis muscles as well as atrophy of the intrinsic muscles have been recognized to induce important changes in the performance of the ankle movements and in the biomechanical properties of the diabetic neuro-

pathic foot (3). Another well-known cause of increased forefoot pressure is the increased thickness of the Achilles tendon; the efficacy of its surgical lengthening on forefoot pressure release has been investigated and reported in literature, even if the results still show some discrepancies (7,8). Structural changes in the forefoot of diabetic neuropathic patients, in terms of bone and muscle density, joint angle and arthropathy, and plantar soft-tissue thickness, have also been revealed and may contribute to mechanical stress (9). It is not well known, however, whether and to what extent plantar fascia contribute to the increased plantar pressure and therefore to the risk of ulceration. Soft tissue alterations are common in diabetic patients (5,10), and plantar fascia should be included among those tissues that may change their physiology and biomechanical function in the presence of chronic hyperglycemia. Studies by Sharkey et al. (11) on cadavers revealed that the plantar fascia, despite being a passive structure, actively contributes to counteract the ground reaction forces (GRFs) acting on the metatarsal heads in almost the same way as digital flexor muscles do.

Several authors have studied the correlation between rupture of the plantar fascia and decrease in forefoot pressure (12) and also the relationship between changes in the thickness of plantar fascia and concurrent changes in the height of the longitudinal arch (13). Thus, the aim of our work was to focus on plantar fascia alterations in the presence of diabetes. We have described plantar fascia abnormalities in diabetic patients with and without peripheral neuropathy and evaluated the relationship among the thickness of plantar fascia, GRFs expressed under the metatarsal heads, and metatarso-phalangeal joint mobility.

RESEARCH DESIGN AND METHODS

Patient recruitment

A total of 61 patients were recruited from the outpatient clinic of the Metabolic Dis-

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Abbreviations: GRF, ground reaction force; LJM, limited joint mobility; NDS, neuropathy disability score; VPT, vibration perception threshold.

A table elsewhere in this issue shows conventional and Système International (SI) units and conversion factors for many substances.

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eases Department at the University of Rome "Tor Vergata." They were divided into three groups: 27 diabetic patients without neuropathy (D group), 19 diabetic patients with neuropathy (DN group), and 15 diabetic patients with previous neuropathic ulcers (DNPU group). A total of 21 healthy volunteers (control [C]) were also examined for comparison. The groups of patients and healthy volunteers were recruited homogeneously as for sex, age, BMI, and occupation. The level of neuropathy was assessed by using the neuropathy disability score (NDS) and the vibration perception threshold (VPT). NDS (14) is the product of scoring ankle reflex (0 = normal, 1 = present with reinforcement, 2 = absent) plus vibration, pin-prick, and temperature sensation (cold turning fork) at the great toe (0 = normal, 1 = abnormal). VPT was assessed by using a biothesiometer on the tip of the great toe. The mean of three readings was considered.

For the purpose of this study, the presence of neuropathy in the participating diabetic patients was defined by NDS >5 V and VPT >25 V (15). All patients in the D group had NDS ≤4 V and VPT <25 V. Those subjects with a history of previous neuropathic foot ulcers up to 3 months before the study but without active ulcers were included in the DNPU group. All previous ulcers were localized at the metatarsal heads. If present, callosities were removed before measurements.

Exclusion criteria. The exclusion criteria included: 1) age >70 years; 2) history of peripheral vascular, neurological (other than those of diabetic etiology), musculo-skeletal, or rheumatic disease; 3) any major or minor amputation; 4) peripheral vascular diseases with ankle brachial index <0.85; and 5) Charcot neuroarthropathy and hallux rigidus caused by previous traumas.

The study was approved by the local ethical committee, and all patients gave their informed written consent before the screening, in accordance with the principles of the Helsinki Declaration.

Experimental set-up and measurement protocol

For ultrasound examination, we used a high-resolution 8- to 10-MHz linear array (Toshiba Tosbee SSA 240). All exams were performed by rheumatologists experienced in musculo-skeletal ultrasound technique. The thickness of plantar fascia

was examined in both feet in a comparative manner. Plantar fascia was measured under slight tension in the axial and sagittal planes. Thickness was examined for the whole length of the plantar fascia. Measurements (expressed in mm) were taken at one-fifth of the length, starting from the calcaneous insertion, on the medial aspect of the foot. This is a point of the plantar region of the foot that is not in contact with the floor, and therefore it is not influenced by GRF.

The foot loading pattern was evaluated through a piezo-dynamometric platform, which is widely described elsewhere (16). Briefly, the piezo-dynamometric platform was obtained by rigidly fixing a dedicated pressure platform (designed and constructed at the Biomedical Engineering Laboratory of the Italian National Institute of Health, Rome, Italy) onto a commercial force platform (Bertec, Worthington, OH) in order to allow the resultant GRF to be transmitted unaltered from one platform to the other. The resulting compound instrument simultaneously measures, for each sample, the resultant GRF (vertical and horizontal components, free moment, and location of center of pressure) and pressure distribution throughout the foot-floor contact area. By applying an ad hoc mathematical model, it also estimates the local GRF components acting on elementary areas. Thus, each subarea of interest, of any shape and dimension, can be wholly characterized from a dynamic point of view.

The platform was inserted at a level in the middle of a wooden walkway that was

long enough to guarantee acquisition "at regimen" (17–19). Patients were trained to walk barefoot on the walkway at their natural speed, and to step on the active surface of the platform with one foot only, without looking down at the platform. Six consecutive footsteps were acquired for each foot, and mean values were entered for statistical analysis.

Metatarsal joint motion was assessed by means of a general-purpose, long-arm mechanical goniometer. The total range of motion at the first metatarso-phalangeal joint was measured as follows: with the patient supine and with the subtalar joint in neutral position, the maximal ranges of dorsal flexion and plantar flexion were determined from the tip of the toe to the heel along the medial side of the foot. For the sake of accuracy, the range of motion was recorded for both feet of each patient by the same operator.

Statistical analysis

One-way ANOVA was performed on the raw data of each group to obtain comparisons between couples of variables in multiple groups. For post hoc multiple comparisons of the means, a Bonferroni test was used for equal variance. Linear regressions were computed between plantar fascia thickness and forces, plantar fascia thickness and LJM, and forces and LJM.

RESULTS — There were no significant differences between groups for sex, age, BMI, or diabetes duration. Mean values are reported in Table 1. Patients and con-

Table 1—Baseline patient characteristics

	Group			
	C	D	DN	DNPU
Patients	21	27	19	15
Sex (M/F)	13/8	19/8	10/9	10/5
Age (years)	56.6 ± 11.8	52.7 ± 12.7	53.7 ± 10.4	57.3 ± 9.6
BMI (kg/cm ²)	25.0 ± 3.1	25.3 ± 3.4	27.0 ± 4.9	27.5 ± 4.1
Smoke history (yes/no)	9/21	15/27	8/19	7/15
Type 1/type 2 diabetes	—	8/19	9/10	3/12
Diabetes duration (years)	—	15.1 ± 9.3	19.4 ± 9.3	16.9 ± 8.6
HbA _{1c} (%)	—	7.5 ± 1.5	7.8 ± 1.8	7.3 ± 2.4
NDS	0	0	6.95 (6–8)*	7.9 (7–9)*, †
Retinopathy‡	—	24/0/3	9/4/5	5/3/7
Nephropathy§	—	26/1/1	16/2/0	15/0/0
VPT big toe (V)	—	14.7 ± 5.7	31.5 ± 7.6*	39.9 ± 13.3*, †

Data are n or means ± SD. *P < 0.05 vs. D; †P < 0.05 vs. DN; ‡data are absent/background/proliferative; §data are absent/microalbuminuria/macroalbuminuria.

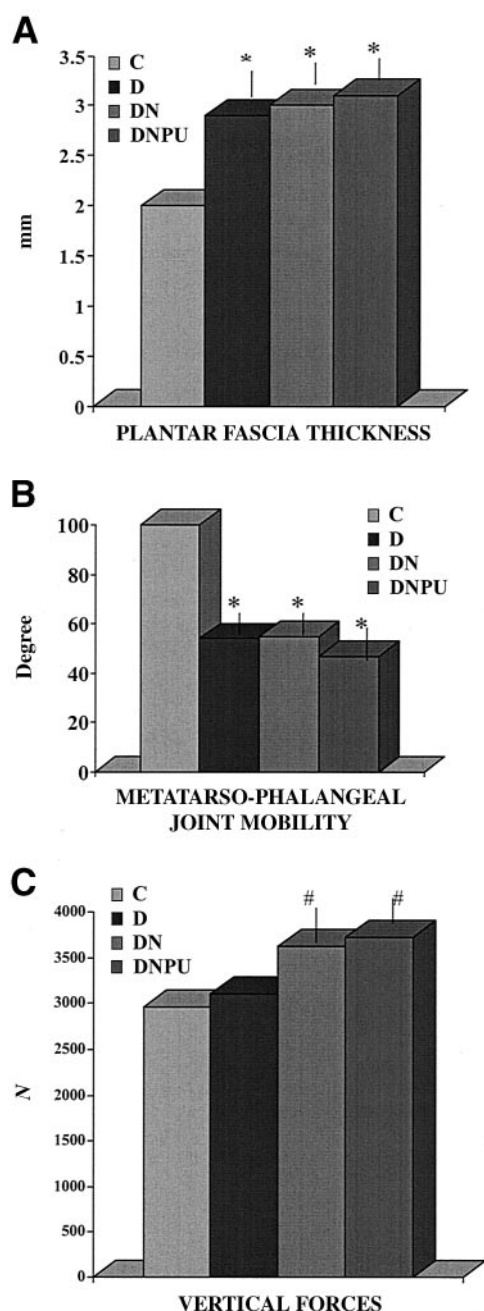


Figure 1—Mean values of plantar fascia thickness (A), metatarso-phalangeal joint mobility (B), and vertical forces under the metatarsal heads (C). * $P < 0.05$ vs. C; † $P < 0.05$ vs. C and D.

control subjects were homogeneous in terms of their occupation.

Diabetic patients showed increased thickness of plantar fascia throughout the length of the structure. Measurements taken next at the calcaneus insertion showed a statistically significant increase in all patient groups in comparison with control subjects, as shown in Fig. 1A) (2.9 ± 1.2 , 3.0 ± 0.8 , 3.1 ± 1.0 , and

2.0 ± 0.5 mm for the D, DN, DNPU, and C groups, respectively; $P < 0.05$).

The evaluation of the joint mobility at the first metatarso-phalangeal joint showed a reduction of the range of motion in all diabetic groups (54.0 ± 29.4 , 54.9 ± 17.2 , 46.8 ± 20.7 , and $100.0 \pm 10.0^\circ$ for the D, DN, DNPU, and C groups, respectively; $P < 0.05$) (Fig. 1B). The reduction was mainly due to dorsiflexion (26.1 ± 12.4 , 26.9 ± 12.2 , 22.8 ± 10.5 , and $64.2 \pm 6.4^\circ$, respectively; $P < 0.05$).

The study of foot-floor interaction under the metatarsal heads showed increased vertical forces in DN and DNPU patients and increased medio-lateral forces in the DNPU group (Fig. 1C).

Table 2 reports mean values and standard deviations of the integrals of vertical, anterior-posterior, and medio-lateral components of GRF, expressed as percent of body weight (%N) \times ms.

An inverse correlation ($r = -0.53$) between the thickness of plantar fascia and LJM was found when considering the three pathological groups together, as well as a direct correlation between the thickness of plantar fascia and vertical forces under the metatarsal heads ($r = 0.52$). No significant correlation was found between LJM and forces. Figure 2 shows the regression line and equation for the relationship between plantar fascia thickness and LJM (Fig. 2A) and for the relationship between plantar fascia thickness and forces (Fig. 2B).

CONCLUSIONS— In diabetic patients, peripheral neuropathy and, more specifically, motor neuropathy are responsible for remarkable deformities of the anatomical structure of the foot (e.g., clawtoes, pes cavus, prominent metatarsal heads, etc.) (8) and functional alterations of gait. These structural and functional changes have been recognized

Table 2—Integrals of vertical, anterior-posterior, and medio-lateral components of GRF under the metatarsals

Groups	Vertical forces	Anterior-posterior forces	Medio-lateral forces
C	$2,956.8 \pm 430.5$	257.4 ± 62.3	115.5 ± 68.7
D	$3,209.7 \pm 493.3$	264.4 ± 59.1	129.8 ± 68.6
DN	$3,625.2 \pm 695.3^{*\dagger}$	279.3 ± 73.1	139.8 ± 76.6
DNPU	$3,722.1 \pm 743.2^{*\dagger}$	301.8 ± 94.2	$185.7 \pm 114.1^*$

Data are means \pm SD. Forces are normalized with respect to body weight; integrals are expressed as %N \times ms. * $P < 0.05$ vs. C; † $P < 0.05$ vs. D.

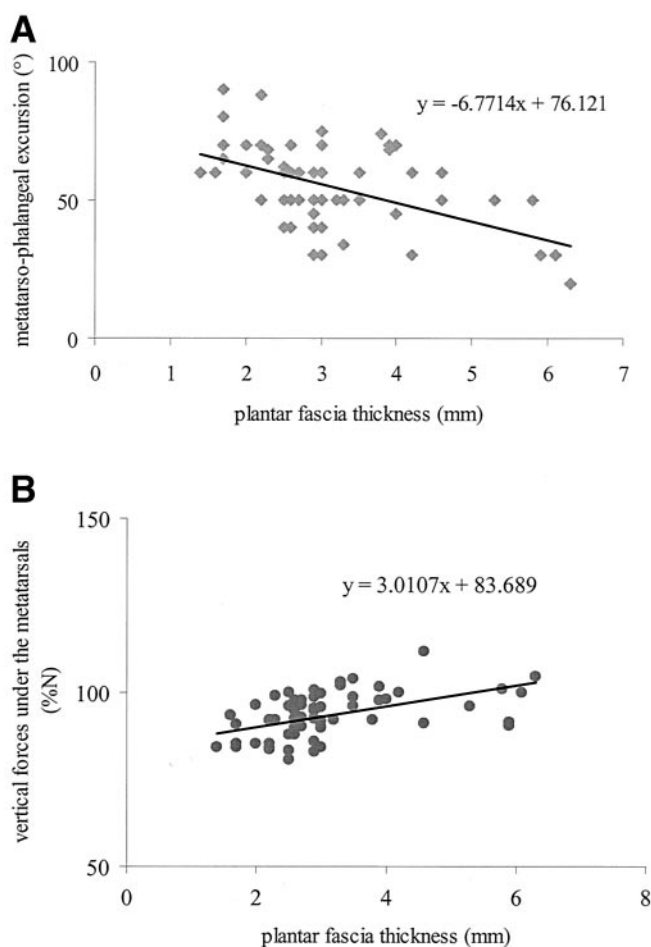


Figure 2—Scatter plot of metatarso-phalangeal joint mobility versus plantar fascia thickness (A) and of vertical forces under the metatarsal heads versus plantar fascia thickness (B).

as responsible for increased plantar pressures (7,20).

In a previous study, we observed that significant changes in forefoot GRF components may be present in diabetic patients without neuropathy (21), thus suggesting that other modifications, independent from peripheral neuropathy, may occur.

The present study shows evidence of a significant increase in plantar fascia thickness, which may entail critical changes in the functionality of the foot (22). Plantar fascia, in fact, sustains the longitudinal arch of the foot during propulsion and assists the body in absorbing forces at the midtarsal joints during the heel strike and the load transfer from the rearfoot to the forefoot (23). When the foot is in contact with the ground, a downward force on the talus is counteracted by an upward force on the metatarsal heads, toes, and the calcaneus. The

upward force produces moments that tend to flatten the arch. The moments are countered by the horizontal forces tending to tie the forefoot and calcaneus together. These forces are generated in passive structures (plantar fascia) (23) by the active contraction of intrinsic and extrinsic muscles (24,25).

The increased thickness of the plantar fascia renders its section greater than normal, which increases its resistance to stresses in the direction of its longitudinal axis (horizontal stresses). Thus, greater upward forces should be produced at the metatarsal heads and at the calcaneus to normally flatten the foot.

In diabetic patients, hyperglycemia induces several effects in almost all tissues because of the glycosilation of proteins (26). Thus, the abnormal production of collagen renders the plantar fascia more resistant to tensile force (27,28). The stiffness of the foot is also increased by the

greater tensile force exerted by the Achilles tendon, which means greater concentric horizontal forces in the plantar fascia. However, not all of the increased forefoot pressure may be related to a shortened Achilles tendon, since its surgical lengthening does not seem to completely overcome the forefoot hyperpressure.

The locomotor pattern is altered by the reduction of mobility at the metatarso-phalangeal joint. This is partly caused by the abnormalities of the joint connective tissues (protein glycosilation), and it is partly the result of the increased tension in the plantar fascia. Because plantar fascia plays a role in foot digital stabilization (29), its intrinsic greater tensile force is confirmed by the positive correlation we found between increased plantar fascia thickness and the limited metatarso-phalangeal range of motion. The final scenario is that of a rigid cavus foot, which remains rigid during the whole walking cycle, thus facilitating the onset of high plantar pressures.

In conclusion, we have shown that diabetic patients do develop changes in plantar fascia and joint mobility, even in the absence of peripheral neuropathy. The relationship between plantar fascia abnormalities and the increased vertical forces under the metatarsal heads of the forefoot suggests that these patients already have a different pattern of plantar pressure distribution (19,21). Therefore, the above alterations may have a role in a more rigid foot, which is thus less adaptable to the floor during the foot-floor interaction. It is then reasonable to hypothesize that these changes may be critical in the onset of the mechanical stress that induces plantar hyperpressure.

References

1. Katoulis EC, Ebdon-Parry M, Lanshammar H, Vileikyte L, Kulkarni J, Boulton AJ: Gait abnormalities in diabetic neuropathy. *Diabetes Care* 20:1904–1907, 1997
2. Delbridge L, Ctercieko G, Fowler C, Reeve TS, Lequesne LP: The etiology of neuropathic ulceration in the diabetic foot. *BR J Surg* 75:1–6, 1985
3. Andersen H, Jakobsen J: Motor function in diabetes. *Diabetes Reviews* 7:326–341, 1999
4. Cavanagh PR, Ulbrecht JS, Caputo GM: New developments in the biomechanics of the diabetic foot. *Diabetes Metab Res Rev* 16 (Suppl. 1):S6–S10, 2000
5. Delbridge L, Perry P, Marr S, Arnold N,

- Yue DK, Turtle JR, Reeve TS: Limited joint mobility in the diabetic foot: relationship to neuropathic ulceration. *Diabet Med* 5:333-337, 1988
6. Salsich GB, Mueller MJ, Sahrman SA: Passive ankle stiffness in subjects with diabetic and peripheral neuropathy versus an age-matched comparison group. *Phys Ther* 80:352-362, 2000
 7. Armstrong DG, Stacpoole-Shea S, Nguyen H, Harkless LB: Lengthening of the Achilles tendon in diabetic patients who are at high risk for ulceration of the foot. *J Bone Joint Surg Am* 81:535-538, 1999
 8. Hastings MK, Mueller MJ, Sinacore DR, Salsich GB, Engsborg JR, Johnson JE: Effects of a tendo-Achilles lengthening procedure on muscle function and gait characteristics in a patient with diabetes mellitus. *J Orthop Sports Phys Ther* 30:85-90, 2000
 9. Robertson DD, Mueller MJ, Smith KE, Commean PK, Pilgram T, Johnson JE: Structural changes in forefoot of individuals with diabetes and a prior plantar ulcer. *J Bone Joint Surg Am* 84:1395-1404, 2002
 10. Lundbaek K: Stiff hands in long term diabetes. *Acta Med Scand* 158:447-451, 1957
 11. Sharkey NA, Donahue SW, Ferris L: Biomechanical consequences of plantar fascia release or rupture during gait. Part II. Alterations in forefoot loading. *Foot Ankle Int* 20:86-96, 1999
 12. Arangio GA, Chen C, Kim W: Effect of cutting the plantar fascia on mechanical properties of the foot. *Clin Orthop* 339: 227-231, 1997
 13. Arangio GA, Chaorong C, Salathè EP: Effect of varying arch height with and without the plantar fascia on the mechanical properties of the foot. *Foot Ankle Int* 19: 705-709, 1998
 14. Young MJ, Boulton AJM, MacLeod AR, Williams DRR, Sonksen PH: A multicentre study of the prevalence of diabetic peripheral neuropathy in the UK hospital clinic population. *Diabetologia* 36:150-154, 1993
 15. Boulton AJM, Kubrusly D, Bowser JH, Gadia MT, Quintero L, Becker DM, Skyler JS, Sensenbrenner JM: Impaired vibratory perception and diabetic foot ulceration. *Diabet Med* 3:335-337, 1986
 16. Giacomozzi C, Macellari V: Piezo-dynamometric platform for a more complete analysis of foot-of-floor interaction. *IEEE Trans Rehabil Eng* 5:137-140, 1998
 17. Macellari V, Giacomozzi C: Multistep pressure platform as a stand-alone system for gait assessment. *Med Biol Eng Comput* 34:299-304, 1996
 18. Barnett S: International protocol guidelines for plantar pressure measurement. *Diabetic Foot* 1:137-140, 1998
 19. Uccioli L, Caselli A, Giacomozzi C, Macellari V, Giurato L, Lardieri L, Menzinger G: Pattern of abnormal tangential forces in the diabetic neuropathic foot. *Clin Biomech (Bristol, Avon)* 16:446-454, 2001
 20. Cavanagh PR, Ulbrecht JS: Biomechanics of the diabetic foot: a quantitative approach to the assessment of neuropathy, deformity, and plantar pressures. In *Disorder of the Foot*. Vol. 3, 2nd ed. Jahss MH, Ed. Philadelphia, PA, W.B. Saunders, 1991 p. 1864-1907
 21. Giacomozzi C, Caselli A, Macellari V, Giurato L, Lardieri L, Uccioli L: Walking strategy in diabetic patients with peripheral neuropathy. *Diabetes Care* 25:1451-1457, 2002
 22. Hicks JH: The mechanics of the foot. I. The joints. *J Anat* 88:25, 1954
 23. Hicks JH: The mechanics of the foot. II. The plantar aponeurosis and the arch. *J Anat* 88:25-30, 1954
 24. Frankel VH: *Basic Biomechanics of the Skeletal System*. Philadelphia, PA, Lea & Febiger, 1986, p. 209-210
 25. Ker RF, Bennet MB, Bibby SR, Kester RC, Alexander RM: The spring in the arch of the human foot. *Nature* 325:147-149, 1987
 26. Barbagallo M, Novo S, Licata G, Resnick LM: Diabetes hypertension and atherosclerosis: pathophysiological role of intracellular ions. *Int Angiol* 12:365-370, 1993
 27. Crisp AJ, Heathcote JG: Connective tissue abnormalities in diabetes mellitus. *J R Coll Physicians Lond* 18:132-141, 1984
 28. Grant WP, Sullivan R, Sonenshine DE, Adam M, Slusser JH, Carson KA, Vinik AI: Electron microscopic investigation of the effects of diabetes mellitus on the Achilles tendon. *J Foot Ankle Surg* 36:272-278, 1997
 29. Pontious J, Flanigan KP, Hillstrom HJ: Role of the plantar fascia in digital stabilization: a case report. *J Am Pod Med Assoc* 86:43-47, 1996