

# An Analysis of Dynamic Forces Transmitted Through the Foot in Diabetic Neuropathy

JONATHAN E. SHAW, MRCP  
CARINE H.M. VAN SCHIE, MSC  
ANNE L. CARRINGTON, PHD

CAROLINE A. ABBOTT, PHD  
ANDREW J.M. BOULTON, FRCP

**OBJECTIVE** — Biomechanical studies in diabetic neuropathy have clearly demonstrated abnormal foot pressures, but information on other aspects of gait is limited. This study aimed to investigate and describe the forces transmitted through the foot during walking in diabetic subjects with varying degrees of peripheral neuropathy and to determine if abnormalities in these forces might contribute to the risk of plantar ulceration.

**RESEARCH DESIGN AND METHODS** — Subjects from the following groups were included: healthy control subjects (C); diabetic control subjects (D); subjects with diabetic neuropathy (DN); subjects with previous neuropathic ulceration (DNU); and subjects with Charcot neuro-arthropathy (CH). Gait analysis was performed as subjects walked over a Kistler force plate. Peak forces were measured (as percent body weight) in the vertical and horizontal planes. Comparisons were made between all of the groups and between each diabetic group and a healthy control group matched for walking speed.

**RESULTS** — There were 181 subjects studied. In comparison with that of the speed-matched controls, the mean peak vertical force was higher in each of the diabetic groups, especially in the most neuropathic subjects (DNU, 113 vs. 110%,  $P < 0.01$ ). This increase was entirely due to higher forces during heel contact (DNU, 111 vs. 106%,  $P < 0.001$ ). The single peak force occurred during heel strike (rather than during foot push-off) in 23–38% of footsteps of healthy and diabetic control subjects but in 53–73% of footsteps of neuropathic subjects. There was also a trend for higher peak medial forces (CH, 6.2 vs. 5.5%,  $P < 0.05$ ).

**CONCLUSIONS** — Diabetic neuropathy is associated with a change in the time pattern of the forces transmitted through the foot and an increase in the vertical forces through the heel. The magnitude of the changes is small in absolute terms, but these changes may contribute to the risk of plantar foot ulceration.

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**B** biomechanical changes and abnormalities are now well recognized in diabetic neuropathy (1). Most of the work has centered on elevated foot pressure and has demonstrated its important role in neuropathic foot ulceration (2,3). There is limited literature, however, on other aspects of gait analysis in diabetes, in particular on the

forces used during walking. It might be expected that both the impairment of proprioception and the reduction in motor strength that result from neuropathy would interfere with normal gait. Furthermore, because pressure equals force divided by area, changes in the forces applied through the foot during walking may be an impor-

tant component in pressure-induced foot ulcers. Earlier work suggested that neuropathy leads to reduced ankle plantar flexion strength (4,5). In a preliminary study, we demonstrated the presence of lower peak vertical forces (6). However, because walking speed was not controlled for in these studies and neuropathic subjects walked more slowly than control subjects, it was not possible to determine whether gait changes were related to walking speed or were directly caused by neuropathy.

Therefore, this study examined, in a relatively large group of subjects, the effect of neuropathy on the forces applied through the foot during walking.

**RESEARCH DESIGN AND METHODS** — The study was approved by the local ethics committee. Consecutive patients attending the Diabetes Centre and foot clinic were invited to take part. Patients were included if they fell into one of four categories: diabetic control subjects without neuropathy (D); diabetic neuropathic patients without a history of foot ulceration (DN); diabetic neuropathic patients with a history of foot ulceration (DNU); and diabetic patients with Charcot neuro-arthropathy (CH). Healthy control subjects (C) were recruited from people accompanying patients and from hospital staff. There were 181 subjects recruited, and full demographic details are shown in Table 1.

The presence of neuropathy was defined as a peroneal nerve conduction velocity (NCV)  $<40$  m/s or at least two out of the following three abnormal quantitative sensory tests (with skin temperature maintained at  $>30^{\circ}\text{C}$  using a warmed heating pad): 1) vibration perception threshold (VPT)  $>20$  V at the great toe; 2) pressure perception threshold (PPT)  $>1$  g (monofilament 4.17) over the plantar surface of the foot; and 3) warm thermal perception threshold (TPT)  $>2^{\circ}\text{C}$  on the dorsal surface of the foot. These limits correspond to the mean  $\pm 2$  SDs of normal healthy subjects in our own laboratory. Patients were excluded if they were older than 75 years or had active foot ulcers, evidence of peripheral vascular disease (ankle brachial pressure index  $<0.85$  or symptoms of intermittent claudication), signifi-

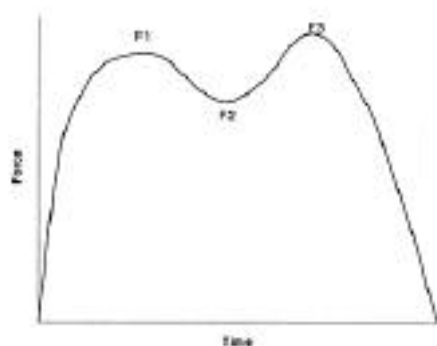
From the Department of Medicine, Manchester Royal Infirmary, Manchester, U.K.

Address correspondence and reprint requests to Dr. Jonathan E. Shaw, International Diabetes Institute, 260 Kooyong Rd., Caulfield, Victoria 3162, Australia. E-mail: jshaw@idi.org.au.

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**Abbreviations:** C, healthy control subjects; CH, diabetic patients with Charcot neuro-arthropathy; D, diabetic control subjects without neuropathy; DN, diabetic neuropathic patients without a history of foot ulceration; DNU, diabetic neuropathic patients with a history of foot ulceration; GRF, ground reaction force; NCV, nerve conduction velocity; PPT, pressure perception threshold; TPT, warm thermal perception threshold; VPT, vibration perception threshold.

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



**Figure 1**—A typical plot of vertical ground reaction force against time, during a single footstep. Two peaks separated by one trough are shown. F1, heel contact peak; F2, trough; F3, push-off peak. In this case, F3 is the single peak force.

cant orthopedic or neuromuscular disease (other than diabetic neuropathy), or were otherwise unable to perform gait trials. NCV in the common peroneal nerve was assessed using the Medelec electrophysiological system (Medelec, Old Woking, U.K.), using surface electrodes (7). VPT was measured using the Neurothesiometer (Horwell, Nottingham, U.K.) at the great toe (8). The mean of three readings at variable speeds of voltage increase was taken. PPT was assessed using a set of monofilaments (Hansens Disease Center, Carville, LA) bending with 1, 10, and 75-g force respectively on three different sites on the plantar surface of the foot. TPT was measured using a thermoesthesiometer (VU, Amsterdam) operating on the Peltier principle and using two-alternative forced-choice methodology (9).

Gait analysis was performed with the Vifor system (Lic Orthopaedics, Norrköping, Sweden). A Kistler force plate (Kistler Instruments, Winterthur, Switzerland) that measures the ground reaction force (GRF) is embedded in an 8-m walkway. The GRF is the total instantaneous

resultant force between the foot and ground. Its site of action can be only crudely determined as heel or forefoot according to the timing of the force and the known contact of the foot with the ground at that part of the gait cycle. The output of the plate is connected to a computer, and a visual record is made in both sagittal and frontal planes by two video cameras. Two photo cells, one on either side of the force plate, are used to measure walking speed. At the completion of each walking trial, the system generates summary statistics of the peak GRFs in each of the vertical, forward, backward, medial, and lateral directions, along with speed, ground contact time, and timing of the peak vertical force. Subsequent digitization of the instantaneous force vector, as recorded on each frame of the video (25 frames/s) throughout each footstep on the force plate, allows a more detailed analysis of the forces. All force data are expressed as percentage of body weight. All the summary data were analyzed, and from digitization, the two peaks (occurring during heel contact and push-off) and the trough value were obtained (Fig. 1). For each subject, three trials were performed for each leg, and as in other studies, subjects wore their own shoes (6,10). The mean values for each leg were treated separately, so that, for example, 10 subjects would provide 20 sets of data.

After familiarization with the walkway and measurement of "normal" step length, each subject was positioned so that while walking along the walkway, the target foot would naturally land on the force plate. The issue of whether or not to control walking speed during gait trials was addressed by allowing all subjects to perform the trials at their own speed; and then a subgroup of healthy control subjects performed multiple trials across a range of speeds. For comparison with each diabetic group, single trials were selected from healthy control

subjects to match on a two-to-one basis the speed from the mean of three trials for each leg from each diabetic subject. Thus, for each diabetic patient, two sets of mean values of gait parameters (peak vertical force, walking speed, etc.) were obtained (one for each leg). Two speed-matched control trials were then selected for comparison to each set of mean values.

**Statistical analysis**

Comparisons between multiple groups were made using one-way analysis of variance followed by pair-wise comparisons using Tukey's correction for multiple comparisons. Student's *t* tests were used to compare results from each diabetic group to the matched control subjects.

**RESULTS** — There were 181 subjects studied. Demographic and neuropathy data demonstrate that the groups were matched for age, sex, diabetes type, and duration, whereas neuropathy increased across the groups (Table 1). Table 2 shows the force data for each group when walking at their own speed. The walking speed was lower in all diabetic groups than in the control group, and speed was slowest in patients with more severe neuropathy. As speed fell in the neuropathic groups, the reduction in peak vertical force was much smaller. Sixteen control subjects (12 men, mean age 48 years, mean weight 78.6 kg) performed multiple trials (mean 18) at different speeds (range 0.77–1.68 m/sec). This subgroup of control subjects did not differ from any of the diabetic groups with regard to age, weight, or percentage of men. Tables 3 and 4 show comparisons when speed-matched control trials were compared with each of the diabetic groups. In Table 3, the GRF data are shown. The peak vertical force was significantly greater in each of the diabetic groups than in the control group, and the difference was most

**Table 1**—Demographic and neurological characteristics of subjects by group

Group	n	% Men	Age	Diabetes duration (years)	% Type 1 diabetes	Weight (kg)	VPT (V)	PPT	TPT (°C)	NCV (m/s)
C	22	68	52.1 ± 10.1	—	—	77.8 ± 13.8	6 (5–9)‡	4 (4–4)‡	0.5 (0.5–0.5)‡	49 (46–51)‡
D	51	50*	52.3 ± 9.9	17.4 ± 12.8	49	74.0 ± 13.4†	13 (7–15)‡	4 (4–4)‡	0.5 (0.5–0.5)‡	44 (41–45)‡
DN	60	78	54.5 ± 10.9	19.4 ± 11.7	50	79.8 ± 14.1	24.5 (17–32)§	5 (4–6)§	6.0 (1.0–9.5)§	36 (33–39)
DNU	34	65	53.8 ± 8.8	19.9 ± 10.7	47	82.6 ± 18.9	29 (21–40)	6 (5–6)	8.5 (5.0–11.0)	32 (21–38)
CH	14	71	54.3 ± 10.0	22.2 ± 12.4	50	87.2 ± 12.0	35 (32–46)	6 (5–7)	9.5 (7.5–11.0)	29 (26–32)

Data are n, means ± SD, and medians (interquartile range). \**P* < 0.05 vs. DN; †*P* < 0.05 vs. CH; ‡*P* < 0.001 vs. DN, DNU, and CH; §*P* < 0.01 vs. CH; ||*P* < 0.01 vs. D.

**Table 2—Gait data for each group walking at their own speed**

Group	Ground contact (ms)	Speed (m/s)	Peak ground reaction force (% body weight)				
			Vertical	Forward	Backward	Medial	Lateral
C	676 ± 61*	1.31 ± 0.10§	118 ± 6.0	21 ± 2.5#	18 ± 2.5	5.6 ± 1.6	3.9 ± 1.4
D	704 ± 70†	1.15 ± 0.17	114 ± 6.8	18 ± 3.8¶	16 ± 4.3	5.6 ± 1.5†	3.8 ± 1.5
DN	717 ± 68	1.19 ± 0.21†	116 ± 9.0¶	19 ± 5.3	17 ± 5.1	6.2 ± 1.7	3.7 ± 1.7
DNU	742 ± 81	1.08 ± 0.19	113 ± 6.8	17 ± 3.7	16 ± 3.9	5.9 ± 1.7	3.5 ± 1.7
CH	767 ± 88‡	1.05 ± 0.20	114 ± 7.5	15 ± 3.3**	16 ± 2.9	6.2 ± 1.5	2.6 ± 1.0**

Data are means ± SD. \* $P < 0.01$  vs. DN, DNU, and CH; † $P < 0.01$  vs. DNU and CH; ‡ $P < 0.05$  vs. DN; § $P < 0.001$  vs. all groups; || $P < 0.01$  vs. D and DNU; ¶ $P < 0.01$  vs. DNU; # $P < 0.01$  vs. D, DNU, and CH; \*\* $P < 0.01$  vs. C, D, and DN.

marked in the neuropathic groups. Within the horizontal forces, only the peak medial force showed a consistent trend: it was greater in the neuropathic groups than in the control group. Neuropathy was associated with a change in the time pattern of forces transmitted through the foot. For both healthy and diabetic control subjects, the peak vertical force mainly occurred during push-off, but in the neuropathic patients, the peak was much more likely to occur during heel contact. The percentage of footsteps in which the single peak vertical force occurred during heel contact was similar in group D compared with speed-matched control subjects (38 vs. 25%,  $P > 0.05$ ), but it was higher in group DN (53 vs. 29%,  $P < 0.0001$ ), group DNU (55 vs. 26%,  $P < 0.0001$ ), and group CH (73 vs. 23%,  $P < 0.0001$ ). In the CH group, the peak occurred during heel contact in 83% of footsteps of CH feet and in 53% of footsteps from the non-Charcot feet. Table 4 shows a separate analysis of the peak forces during heel contact and push-off. This demonstrates that there were no significant changes in the forces transmitted through the forefoot at push-off (F3), except in the CH group, where a reduction was seen but there was a significant

increase in the peak forces during heel contact (F1). This increase was present in every diabetic group but was greatest in the more neuropathic groups. Therefore, the shift of the timing of the single peak vertical force was predominantly due to increased heel forces, rather than reduced push-off forces.

**CONCLUSIONS** — Normal walking requires both sensory input to modify learned motor patterns and muscular output to execute the desired action. The sensory input comes from visual and vestibular systems, as well as from proprioceptive information, mainly from the lower limbs. Thus, the often severe effects of diabetic neuropathy might be expected to have a measurable effect on gait patterns. Abnormalities have already been demonstrated in static postural control in a number of studies (11,12) and are thought to contribute to an increased risk of falling in diabetic neuropathy (13). Changes in the dynamic patterns of gait might also affect the forces transmitted through the foot, thereby altering pressures and increasing the risk of foot ulcers. In the only two related studies on gait analysis in diabetes of which we are aware, Mueller and colleagues (4,10) reported data from a small group of sub-

jects that suggest reduced strength of ankle plantar flexion and the adoption of a “hip strategy” of walking, whereby the leg is pulled forward from the hip rather than being pushed forward by the foot.

The interpretation of gait has often been limited by small sample sizes and failure to control for walking speed, which influences almost every gait parameter. In common with many other neuromuscular disorders, diabetic neuropathy reduces walking speed, making gait comparisons difficult with both healthy and diabetic control subjects. We chose to overcome this problem by recording gait trials in all patients at their own natural speed, and then recording multiple trials at a range of speeds in 16 normal control subjects. This allowed each group of patients to be compared with a control group accurately matched for speed. Thus, the impact of speed on other gait parameters is not a confounding factor in this study. Furthermore, by limiting the imposition of speed restrictions to the fittest and most able subjects (i.e., the healthy control subjects), we limited the possible adverse effect of walking at an “unnatural” speed.

The effects of neuropathy are mainly seen in the vertical forces. The increased

**Table 3—Gait data for each group compared with speed-matched control subjects**

	Ground contact (ms)	Speed (ms <sup>-1</sup> )	Peak ground reaction force (% body weight)				
			Vertical	Forward	Backward	Medial	Lateral
C matched to D	768 ± 86	1.15 ± 0.17	112 ± 5.5	18 ± 3.2	16 ± 2.8	5.5 ± 1.5	3.6 ± 1.6
D	704 ± 70‡	1.15 ± 0.17	114 ± 6.8‡	18 ± 3.8	16 ± 4.3	5.6 ± 1.5	3.8 ± 1.5
C matched to DN	757 ± 99	1.19 ± 0.20	113 ± 6.6	18 ± 3.7	17 ± 4.2	5.7 ± 1.5	3.7 ± 1.7
DN	717 ± 68‡	1.19 ± 0.21	116 ± 9.0‡	19 ± 5.3	17 ± 5.1	6.2 ± 1.7†	3.7 ± 1.7
C matched to DNU	798 ± 91	1.08 ± 0.18	110 ± 5.5	17 ± 3.2	15 ± 2.9	5.4 ± 1.4	3.3 ± 1.4
DNU	742 ± 81‡	1.08 ± 0.19	113 ± 6.8†	17 ± 3.7	16 ± 3.9	5.9 ± 1.7	3.5 ± 1.7
C matched to CH	807 ± 107	1.05 ± 0.21	110 ± 5.9	17 ± 3.8	15 ± 2.9	5.5 ± 1.5	3.0 ± 1.6
CH	767 ± 88	1.05 ± 0.20	114 ± 7.5*	15 ± 3.3*	16 ± 2.9	6.2 ± 1.5*	2.6 ± 1.0

Data are means ± SD. \* $P < 0.05$  vs. matched control subject; † $P < 0.01$  vs. matched control subject; ‡ $P < 0.001$  vs. matched control subject.

**Table 4—Peak and trough forces for each group compared with speed-matched control subjects**

	F1 (% body weight)	F2 (% body weight)	F3 (% body weight)
C matched to D	108 ± 6.6	80 ± 6.8	112 ± 5.9
D	110 ± 8.4*	79 ± 8.1	113 ± 6.1
C matched to DN	110 ± 8.3	79 ± 8.6	112 ± 5.8
DN	114 ± 10.8†	76 ± 11.4*	112 ± 7.1
C matched to DNU	106 ± 6.7	83 ± 7.1	111 ± 5.4
DNU	111 ± 8.0†	80 ± 8.1*	110 ± 6.2
C matched to CH	107 ± 4.9	83 ± 7.7	111 ± 6.0
CH	113 ± 8.8†	82 ± 9.5	108 ± 5.6*

Data are means ± SD. F1, peak vertical force during heel contact; F2, trough force; F3, peak force during push-off (see Fig. 1). \**P* < 0.05 vs. matched control subject; †*P* < 0.001 vs. matched control subject.

forces are evident when looking at the overall peak forces (Table 3). In addition to this, there was a major change in the timing of forces, in which the single peak force usually occurred through the forefoot during push-off in the control subjects, but mainly through the heel during heel contact in neuropathic subjects. The separate analysis of heel contact and push-off forces demonstrates that all the changes are occurring during heel contact. There was no reduction in the push-off forces (except in CH), which would be expected to reflect the strength of ankle plantar flexion. The increased vertical forces during heel contact (constituting a doubling in the additional force over body weight) presumably relate to either a faster descent of the foot towards the ground or perhaps a failure to effect a smooth vertical deceleration after the initial heel contact. Both of these could be due to sensory impairment, leading either to a need to have a more forceful ground contact to give a “normal” sensation or to unawareness of the initial heel-ground contact. Whatever the cause of the increase in heel forces, it is probably an important cause of the increased heel pressures that have been seen in other studies (14) and may contribute to plantar ulceration. High peak forefoot pressures, however, would appear to be due to altered foot shape, with consequent reduction in ground contact area, and limited joint mobility (15), with no contribution from the force. One could speculate that a faster descent of the heel is associated with a faster forefoot descent (foot slap), which might lead to an early rise in forefoot force and pressure, while not affecting peak forces and pressures occurring later in the stance phase.

This is one of the first studies to report on the horizontal or shear forces applied to the neuropathic foot. In the work by

Mueller et al. (4), the medial-lateral horizontal forces were increased in neuropathy, but the subject numbers were small, the groups were not matched for speed, and the differences were of only borderline statistical significance. Nevertheless, our results showed similar trends. It has often been speculated that shear force has a major role to play in neuropathic ulceration (16), but the data reported here seem not to support this hypothesis with regard to plantar ulceration. Although two of the neuropathic groups had slightly increased medial forces, probably indicating a more broadly based gait, the magnitude of the forces was much less than that of vertical forces. Therefore, shear forces would seem unlikely to play a significant role in plantar ulceration, unless shear forces (resulting in tissue stretching) have different effects on tissue than do vertical forces (resulting in tissue compression). However, we were examining only the interface between shoe and floor, and we were only able to measure the total resultant forces on the foot. Thus, when technologies are developed that can measure shear forces at different sites on the foot, this question can be addressed more accurately.

Overall, the abnormalities of the forces used during walking demonstrated in this study are modest, and they are certainly much smaller than the changes observed in foot pressures. This indicates that despite a major sensory loss coupled with distal motor weakness, walking along a flat surface can be performed with only minor perturbations of gait. This suggests that walking along a flat surface is predominantly a learned and automatic function, or as recently pointed out (17), the additional attention required from neuropathic patients is small in these circumstances. It may be that it is only on a more challeng-

ing (and more normal) surface that greater differences can be found.

In conclusion, we have shown in a large study that vertical forces through the heel during walking are increased in diabetic neuropathy and that this might explain increased plantar heel pressures and contribute to the risk of foot ulceration.

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**References**

1. Cavanagh PR, Ulbrecht JS, Caputo GM: Biomechanical aspects of diabetic foot disease: aetiology, treatment and prevention. *Diabet Med*13 (Suppl. 1):S17–S22, 1996
2. Stess RM, Jensen SR, Mimiran R: The role of dynamic plantar pressures in diabetic foot ulcers. *Diabetes Care* 20:855–858, 1997
3. Veves A, Murray HJ, Young MJ, Boulton AJM: The risk of foot ulceration in diabetic patients with high foot pressure: a prospective study. *Diabetologia*35:660–663, 1992
4. Mueller MJ, Minor SD, Sahrman SA, Schaaf JA, Strube MJ: Differences in the gait characteristics of patients with diabetes and peripheral neuropathy with age-matched controls. *Phys Ther*74:299–313, 1994
5. Andersen H, Poulsen PL, Mogensen PE, Jakobsen J: Isokinetic muscle strength in long-term IDDM patients in relation to diabetic complications. *Diabetes*45:440–445, 1996
6. Katoulis EC, Ebdon-Parry M, Lanshammar H, Vileikyte L, Kulkarni J, Boulton AJM: Gait abnormalities in diabetic neuropathy. *Diabetes Care*20:1904–1907, 1997
7. Young MJ, Veves A, Walker MG, Boulton AJM: Correlations between nerve function and tissue oxygenation in diabetic patients: further clues to the aetiology of diabetic neuropathy? *Diabetologia*35:1146–1150, 1992
8. Young MJ, Breddy JL, Veves A, Boulton AJM: The prediction of diabetic neuropathic foot ulceration using vibration perception thresholds. *Diabetes Care*17:557–560, 1994
9. Bertelsman FW, Heimans JJ, Weber EJM, van der Veen EA: Thermal discrimination thresholds in normal subjects and in patients with diabetic neuropathy. *J Neurol Neurosurg Psychiatry*48:686–690, 1985
10. Mueller MJ, Minor SD, Schaaf JA, Strube MJ, Sahrman SA: Relationship of plantar-flexor peak torque and dorsiflexion range of movement to kinetic variables during walking. *Phys Ther*75:684–693, 1995
11. Uccioli L, Giacmini P, Monticone G, Magrini A, Durola L, Bruna E, Parisi L, Di Girolamo S, Menzinger G: Body sway in

- diabetic neuropathy. *Diabetes Care* 18:339-344, 1995
12. Simoneau GC, Ulbrecht JS, Derr JA, Becker MB, Cavanagh PR: Postural instability in patients with diabetic sensory neuropathy. *Diabetes Care* 17:1411-1421, 1994
  13. Cavanagh PR, Derr JA, Ulbrecht JS, Maser RE, Orchard TJ: Problems with gait and posture in neuropathic patients with insulin dependent diabetes. *Diabet Med* 9:469-474, 1992
  14. Sarnow MR, Veves A, Giurini JM, Rosenblum BI, Chrzan JS, Habershaw GM: In-shoe foot pressure measurements in diabetic patients with at-risk feet and in healthy subjects. *Diabetes Care* 17:1002-1006, 1994
  15. Fernando DJ, Masson EA, Veves A, Boulton AJM: Relationship of limited joint mobility to abnormal foot pressures and diabetic foot ulceration. *Diabetes Care* 14:8-11, 1991
  16. Cavanagh PR, Ulbrecht JS: Biomechanical aspects of foot problems in diabetes. In *The Foot in Diabetes* 2nd ed. Boulton AJM, Connor H, Cavanagh PR, Eds. Chichester, U.K., Wiley, 1994, p. 25-35
  17. Courtemanche R, Teasdale N, Boucher P, Fleury M, Lajoie Y, Bard C: Gait problems in diabetic neuropathic patients. *Arch Phys Med Rehabil* 77:849-855, 1996