# Plantar Pressures Are Elevated in the Neuroischemic and the Neuropathic Diabetic Foot

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**OBJECTIVE** — Clinical observation has noted that diabetic neuropathic ulcers occur frequently on the plantar surface, whereas neuroischemic ulcers seem to occur often on the foot margins. The reason for this difference in the site of ulceration is unknown, but it may be related to differences in pressure loading. The aim of the study was to compare vertical in-shoe foot pressures measured during walking (using the F-SCAN system) in four groups of patients whose degree of neuropathy was measured by vibration perception threshold (VPT).

**RESEARCH DESIGN AND METHODS** — Subjects included 14 neuroischemic diabetic patients (VPT 29.3 ± 13.5 V) with history of ulceration on the margins of the foot, 18 patients with neuropathy alone (VPT 38.7 ± 12.7 V) and previous history of ulceration on the plantar surface, 10 diabetic control patients (VPT 9.9 ± 2.7 V), and 15 nondiabetic control subjects (VPT 7.0 ± 0.5 V).

**RESULTS** — When compared with the other three groups, neuroischemic patients had higher foot pressures when measured as mean peak pressures and highest peak pressures under four areas of the foot: medial and lateral forefoot, hallux, and heel. Furthermore, when measuring the maximum pressures developed at any point under the plantar surface, the neuroischemic patients also had the most elevated pressures (757.6 ± 135.9 kPa), significantly higher than those found in the neuropathic group (482.8 ± 68.6 kPa, $P = 0.04$) and in both diabetic control patients (310.2 ± 34.7 kPa, $P = 0.008$) and nondiabetic control subjects (365.1 ± 49.8 kPa, $P = 0.007$).

**CONCLUSIONS** — Despite having increased plantar pressures and a comparable degree of neuropathy, the neuroischemic patients did not have a history of ulceration on the plantar surface. These observations may have relevance to different mechanisms of ulcer formation in the neuroischemic and neuropathic foot.

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Diabetic ulcers can occur anywhere on the foot. However, clinical observation has noted that diabetic neuropathic ulcers frequently occur on the plantar surface, whereas neuroischemic ulcers seem to occur often on the foot margins (1). These different trends in distribution may be related to differences in pressure loading. Previous research has shown that the neuropathic foot with a history of ulceration is characterized by increased peak plantar pressure (2) at the ulcer sites (3). However, plantar pressures have never been measured in neuroischemic patients. It is possible to hypothesize that plantar pressures in neuroischemic feet may be lower than in neuropathic patients. Most of the foot pressure studies (static or dynamic) have been performed barefoot (2–4), but it is also important to measure the peak plantar pressures within the shoe during normal walking conditions (5,6).

The aim of this study was to evaluate vertical in-shoe foot pressures during walking in diabetic patients with both neuropathy and ischemia. The plantar pressures measured in the neuroischemic group were then compared with those in diabetic patients with neuropathy alone, in diabetic patients without neuropathy or ischemia, and in nondiabetic subjects.

## RESEARCH DESIGN AND METHODS

### Patients

Consecutive patients attending the Foot Clinic and the Diabetic Clinic at King's College Hospital who fulfilled the study entry criteria were recruited in the following four groups. *1*) Group A consisted of 14 neuroischemic patients. The criteria for entry into the study were a history of ulceration anywhere on the margins of the foot (all patients in this group reported ulcers located on the forefoot margins) and peripheral ischemia, which was defined as absent foot pulses accompanied by an ankle-brachial pressure index $<0.8$ units. Neuropathy was defined by the absence of ankle reflexes accompanied by abnormal vibration perception threshold (VPT) $>20$ V, measured with a Biothesiometer (Ohio Medical Instruments, Vickers, OH). *2*) Group B consisted of 18 neuropathic patients with a previous history of plantar foot ulceration surrounded by callus with foot pulses palpable bilaterally and/or ankle-brachial pressure index $\geq 1$. Peripheral neuropathy was also defined as in Group A. *3*) Group C consisted of 10 diabetic control patients with present ankle reflexes and palpable foot pulses. The mean VPT was 9.9 ± 2.7 V, and their pressure index was $\geq 1$. *4*) Group D consisted of 15 nondiabetic control subjects of whom none had a family history of diabetes. Clinical details are given in Table 1. The VPT was age-matched and was not significantly different between control sub-

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**Abbreviations:** VPT, vibration perception threshold.

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

**Table 1—*Clinical details of patients***

| | *n* | Age (years) | Diabetes duration (years) | Type 1/2 | VPT (V) |
|---|---|---|---|---|---|
| Nondiabetic control subjects | 15 | 49.6 ± 11.9 | — | — | 7.0 ± 0.5 |
| Diabetic control patients | 10 | 57.6 ± 10.8 | 9.2 ± 9.7 | 4/6 | 9.9 ± 2.7 |
| Neuropathic patients | 18 | 55.4 ± 16.0 | 21.5 ± 12.7 | 8/10 | 38.7 ± 12.7 |
| Neuroischemic patients | 14 | 63.5 ± 9.8 | 17.4 ± 14.4 | 6/8 | 29.3 ± 13.5 |

Data are means ± SD.

jects and diabetic control patients nor between neuropathic and neuroischemic patients.

Clinical observation was used to assess foot deformity in the four groups. Patients with major or ray-amputations or Charcot deformity were excluded from the study.

King's College Hospital Ethical Committee approval was obtained before proceeding with the study.

## Methods

Foot pressures measurement was performed using F-SCAN system (Tekscan, Boston, MA), and the methodology of pressure measurement described below has been validated in a separate study (7).

The F-SCAN uses an ultrathin pressure-sensitive insole sensor, which consists of two layers of Mylar substrate with 960 pressure-sensing cells evenly distributed in a grid-like configuration. The insole was trimmed to fit the shoe size by cutting around its periphery to maintain the center of the insole to coincide with the center of the foot. The patients were asked to slip very carefully their foot inside the shoe.

The insole was then connected via an amplifier attached to the patient's ankle to an IBM-compatible computer with an interface board, which uses F-SCAN software for dynamic gait analysis to store and analyze the data.

## Procedure

For each recording of plantar pressure, the patient walked normally along a flat surface for 4 s, always in the same direction; this was defined as a "walk." One F-SCAN insole was used for three walks per one test in one patient. The first walk of the test was used for training only.

The recording of foot pressures was performed in standard shoes (Clarks Swing Low trainers), which were used because of the reported variation with different types of footwear (8). These trainers have a standard inlay molded Plastazote (Polyethilene) foam and a second layer of Polyurethane used to improve cushioning, with a small heel cup and arch support laminated to a woven nylon sock. The trainers were specifically ordered for the study to provide standard support and cushioning and were matched to the patient's foot size, which had been previously measured.

The patients were also given standard hosiery (thin nylon socks) because this can alter the foot pressures as well (9). Callus, which was commonly found on the plantar surface in the neuropathic group only, was not removed before taking the pressure measurements, because podiatry is known to reduce plantar pressures approximately one-third (10) in neuropathic patients. This external intervention would have added extra variability in the neuropathic group, and the standardization of the methodology would have been compromised, with the potential to artificially increase the difference between the neuropathic and neuroischemic groups.

## Recording of plantar pressures

A new insole was used for each subject, who was asked to walk normally on a flat and even surface for 2 min before calibration in order to reach a suitable level of insole "bedding-in." The necessity for a bedding-in process in the insole resulted from laboratory tests of cyclical loading (7) that showed that the insole should be conditioned by at least 60 s of loading (and for another 60 s if using a new insole) before calibration and that a trial walk be undertaken within a few seconds thereafter. This procedure aimed to reach the plateau of a recoverable creep and allowed the recordings to be made in the part of the creep curve that was shown to have a variability in the range of 3–4% (7,11). Also, this initial bedding-in period allowed the insole to conform to the curved shapes of the foot and enabled temperature equilibration inside the shoe, as suggested by similar research (6) showing that temperature stability was gained within 3 min of use of the insole. The temperature sensitivity of the insole reported by F-SCAN manufacturer was 1.8% for 1°C. During a three-walk pilot trial, we found 1.1°C temperature change, which was unlikely to influence significantly the level of recorded pressure.

The insole was then calibrated against each individual body weight by asking the patient to load all the body weight on one foot while the pressure distribution on the screen was checked to be even, and then the "Calibration" function was triggered. This procedure allowed for the different effects of body weight on the amplitude of pressure measurements, given that a previous study (12) suggested a significant correlation between body mass and peak plantar pressure, although the functional relationship between the two variables was found to be weak.

The recording of the first walk followed immediately and was used for training and allowing the patient to acclimatize to the recording conditions. The next two walks were recorded for analysis, and the pattern of pressure was visualized live dynamically on the computer screen.

## Analysis of plantar pressure recordings

The data were analyzed in two active windows on the screen. The gait mode displayed a two-dimensional representation of the plantar pressures as they developed sequentially from heel-strike to toe-off on the map of the foot. This was visible on the screen in video mode. The increase in pressure was seen as a change in color of the pressure map, and a graph of the pressure amplitude during gait was constructed as a function of time. The "Average" function from the screen was activated to allow a smoothing effect on the immediate surrounding cells and to reduce the possibility of artifacts as a result of impact of cell-to-cell sensitivity (7).

Peak plantar pressures were measured by selecting an area of interest under the four main areas of the foot: the big toe, the medial and lateral side of the forefoot, and the heel. The size of the area of interest was adjusted accordingly: 15 × 15 mm for the big toe and an area of 45 × 45 mm for the medial or lateral forefoot and heel. The size of the window containing the gait sequence to be analyzed was kept constant, and the box was placed centrally on the same location on the image of the foot, which corresponded to the same anatomical site such

**Table 2—*Mean peak plantar pressures (kPa) measured under hallux, lateral and medial forefoot, and heel in the four groups of subjects***

| | Hallux | Lateral forefoot | Medial forefoot | Heel |
|---|---|---|---|---|
| Patients | | | | |
| Control | 115.0 ± 12.8 | 127.3 ± 17.8 | 151.7 ± 19.6 | 134.4 ± 15.7 |
| Diabetic control | 170.0 ± 22.8 | 138.9 ± 15.9 | 139.0 ± 20.5 | 148.9 ± 22.3 |
| Neuropathic | 171.2 ± 16.5 | 182.0 ± 16.2 | 211.8 ± 20.1 | 217.3 ± 24.9 |
| Neuroischemic | 272.5 ± 35.7 | 263.9 ± 30.5 | 267.1 ± 34.5 | 245.8 ± 32.3 |
| Comparisons between groups | | | | |
| Control versus diabetic control | 0.04 | 0.63 | 0.66 | 0.60 |
| Neuropathic versus control | 0.02 | 0.03 | 0.05 | 0.02 |
| Neuropathic versus diabetic control | 0.96 | 0.017 | 0.02 | 0.07 |
| Neuroischemic versus control | 0.001 | 0.001 | 0.008 | 0.005 |
| Neuroischemic versus diabetic control | 0.02 | 0.001 | 0.004 | 0.02 |
| Neuroischemic versus neuropathic | 0.007 | 0.01 | 0.15 | 0.49 |

Data are means ± SD or *P* value.

as the big toe, medial or lateral forefoot, and heel.

The mean peak plantar pressure was obtained from the pressure-time graphs by moving the cursor along until the peak of each graph, representing the pressures developed during each step, was reached. A mean of all pressures measured in each step (three steps per walk) of each walk (two walks per test) was calculated for each patient.

The highest peak plantar pressure in a patient was calculated as the greatest pressure value observed under a specific area of interest during all steps of both walks.

The maximum plantar pressure developed by a patient during a gait stance was also analyzed. A standard box (10 × 10 mm) was applied automatically by a new version of F-SCAN software, which has allowed us to determine the maximum pressure developed by a patient at any point under the entire plantar area and the site where this is located.

### Statistical analysis

Data are presented as means ± SEM. Data were analyzed using analysis of variance for pressures developed in similar anatomical locations in neuroischemic patients compared with neuropathic patients and the two groups of control subjects. The significance level was set at $P = 0.05$.

**RESULTS** — The neuroischemic patients showed significantly greater mean peak plantar pressures when compared with both control groups (Table 2). When compared with diabetic patients with peripheral neuropathy, the mean peak plantar pressures were found to be significantly higher under the hallux and the lateral forefoot.

The neuropathic patients showed significantly increased mean peak plantar pressures when compared with both control groups under the lateral and medial sides of the forefoot and under the hallux when compared with nondiabetic control subjects only. However, in diabetic control subjects, pressures under the hallux and the heel were not statistically different from the neuropathic patients.

Although the diabetic control subjects showed a trend to higher mean peak pressures than nondiabetic control subjects, this was not statistically significant, except under the hallux.

Again, the highest peak plantar pressures under the main four areas of the foot were significantly elevated in the neuroischemic group when compared with the other three groups, with the exception of pressures under the heel, which were not significantly different between neuroischemic and neuropathic patients (Table 3).

In the neuropathic patients, highest peak plantar pressures were greater than in the control groups. However, the difference was not statistically significant, except for the medial side of the forefoot, where the pressures were significantly elevated in the neuropathic group versus the nondiabetic control group.

The highest peak pressures in the control subjects did not differ significantly from the pressures measured in the diabetic control patients.

The maximum pressures developed under the plantar surface by the neuroischemic patients (757.6 ± 135.9 kPa) were highest in the neuroischemic patients; and they were significantly greater than those found in the neuropathic group (482.8 ± 68.6 kPa, $P = 0.04$) and both the diabetic

**Table 3—*Highest peak plantar pressures (kPa) measured under hallux, lateral and medial forefoot, and heel in the four groups of subjects***

| | Hallux | Lateral forefoot | Medial forefoot | Heel |
|---|---|---|---|---|
| Patients | | | | |
| Control | 162.5 ± 28.4 | 156.0 ± 22.1 | 204.6 ± 37.8 | 188.2 ± 33.0 |
| Diabetic control | 190.8 ± 28.1 | 158.9 ± 17.5 | 167.0 ± 26.4 | 166.1 ± 22.8 |
| Neuropathic | 195.3 ± 19.3 | 212.0 ± 19.2 | 242.0 ± 25.1 | 240.1 ± 28.1 |
| Neuroischemic | 344.0 ± 49.2 | 331.6 ± 45.7 | 349.1 ± 49.5 | 301.0 ± 39.1 |
| Comparisons between groups | | | | |
| Control versus diabetic control | 0.48 | 0.92 | 0.43 | 0.59 |
| Neuropathic versus control | 0.32 | 0.06 | 0.03 | 0.24 |
| Neuropathic versus diabetic control | 0.89 | 0.06 | 0.06 | 0.07 |
| Neuroischemic versus control | 0.01 | 0.002 | 0.02 | 0.03 |
| Neuroischemic versus diabetic control | 0.01 | 0.002 | 0.004 | 0.007 |
| Neuroischemic versus neuropathic | 0.003 | 0.01 | 0.04 | 0.21 |

Data are means ± SD or *P* value.

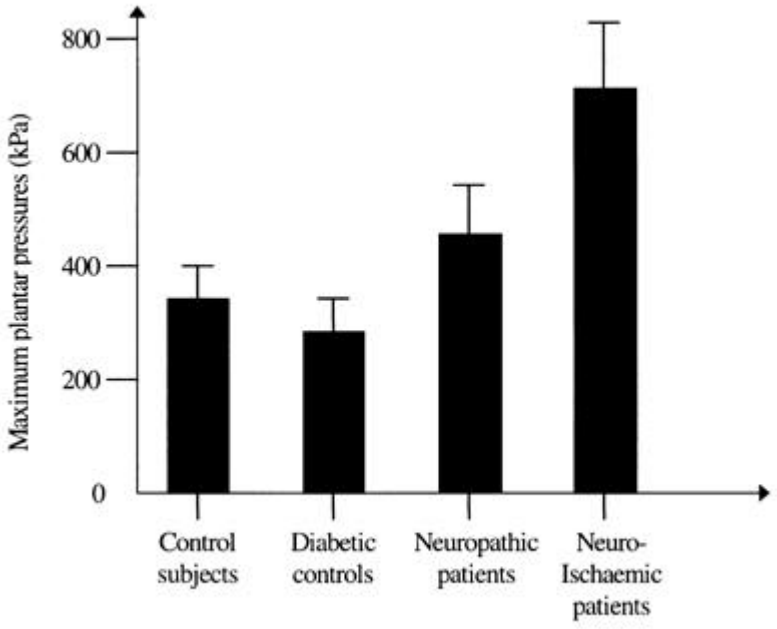


**Figure 1—***Comparison of maximum pressures developed at any point under the plantar surface in the four groups of patients: neuroischemic patients, patients with neuropathy alone, diabetic control patients, and nondiabetic control subjects.*

control patients (310.2 ± 34.7 kPa, $P$ = 0.008) and the healthy control subjects (365.1 ± 49.8 kPa, $P$ = 0.007) (Fig. 1).

The neuropathic patients had also significantly increased maximum pressures when compared with nondiabetic subjects ($P$ = 0.04) and to diabetic control subjects ($P$ = 0.03).

**CONCLUSIONS** — This study has shown that the plantar pressures were generally higher in the neuroischemic group than in the neuropathic group and in both control groups. Despite having increased plantar pressures and a comparable degree of neuropathy, the neuroischemic patients did not have a history of ulceration on the plantar surface. We believe these observations may have relevance to the mechanism of ulceration in the neuroischemic and neuropathic foot. As high plantar pressures have been implicated in the etiology of neuropathic ulceration, the hypothesis of this study was that neuroischemic patients would have lower plantar pressures, because they do not develop ulcers underneath the foot. Surprisingly, this hypothesis was not confirmed by the present findings.

The mean peak plantar pressures under all areas of interest were higher in the neuroischemic patients than in the neuropathic and both control groups. They were obtained by calculating the mean of all the peak pressures recorded in all steps of both strides. However, by such spatial and temporal averaging, the peaks of pressure could be overlooked, and it is possible that such occasional peaks are more likely to lead to ulceration. Therefore, in a further analysis, the highest peak pressure developed at any time under each of the main four areas of the foot was evaluated. Similarly, the maximum pressure developed at any point under the entire plantar area was calculated using a small sensor area to minimize spatial averaging.

The highest peak plantar pressures were noted under the neuroischemic foot. The neuropathic patients had lower pressures than the neuroischemic patients but higher than both control groups. Although the highest peak pressures in the neuropathic patients were significantly increased only under the medial side of the forefoot, this has been recognized as the area most prone to ulceration in neuropathic patients (13). The pressures measured under the other areas of the foot also showed a trend to increase but without statistical significance. When we analyzed the data to obtain maximum plantar pressures, we found that the neuroischemic patients had indeed developed the greatest levels, significantly more than all the other groups. These findings would suggest that the maximum plantar pressure measurements could be a useful parameter in detecting an increase in plantar pressures and their location in patients at risk of foot ulceration.

Neuropathy was assessed by measurement of VPT, which has been demonstrated in the past to be a useful predictor for ulceration in neuropathic patients, as described by Young et al. (14). In our study, the degree of neuropathy in neuroischemic patients was not significantly different from that of the neuropathic patients. Therefore, some increase in the plantar pressure may have been expected considering that these neuroischemic patients did have a degree of neuropathy. However, despite having elevated plantar pressures, the neuroischemic patients did not have a history of plantar ulceration.

A possible explanation may be the patient's level of activity, an etiological factor in ulcer formation, which was considered to be essential by Cavanagh et al. (15). Furthermore, the amount and type of activity during daily living are also important when interpreting foot pressures (16). It is important to know how often and for how long an area on the plantar surface is subjected to a repetitive and high stress. In our study, it is possible that the neuropathic patients simply continued to walk on areas of high pressure, whereas the neuroischemic patients walked less, possibly because of a degree of claudication. We have not assessed formally either the level of activity or the degree of claudication. However, it has been suggested that patients with severe peripheral ischemia and neuropathy do not often have claudication (17). This may be a consequence of neuropathy and may also be related to the very distal distribution of peripheral vascular disease in diabetes (18). Correlating the levels of claudication and activity with the amplitude of foot pressures would be of interest for future research.

Callus has been implicated in the etiology of neuropathic ulceration, acting as a foreign body and increasing plantar pressure (10). However, the neuroischemic foot does not build up excess callus, possibly because of the poor blood supply (19). This is in contrast to the neuropathic foot, which, in the presence of a good blood supply, develops callus at sites of high pressure, or under bony prominences or deformities, such as the Charcot deformity. It is therefore unlikely that the high plantar pressures in the neuroischemic foot were secondary to deformity or callus formation.

An association has been described between abnormally high plantar pressures and limited joint mobility at the metatarsophalangeal and subtalar joints of the neuropathic foot (4,20,21). Joint mobility was not assessed in this study but may have contributed to the differences between the plantar pressures in the groups. This needs addressing in future studies of neuroischemic feet.

The properties of the soft tissue of the foot have just recently been considered as important in the etiology of ulceration. Thickness of the plantar soft tissue has been shown to be a useful predictor of elevated plantar pressures under metatarsal heads in walking (22). Brink (23) has shown in 10 neuropathic patients with a history of ulceration that soft tissue at the sites of previous ulceration over the metatarsal heads was significantly more rigid than at other sites in the same feet or in the control patients' feet. Brink also found that the peak plantar pressures were significantly higher at these areas of induration of the plantar pad and concluded that the induration of the soft tissue may predispose to recurrent foot ulcers in diabetic patients by decreasing the shock absorption capacity of the plantar pad. It may be that the intrinsic properties of the plantar soft tissue, such as compressibility and elasticity, are also altered as a result of peripheral ischemia. Recent work has shown that there is a loss of plantar soft tissue thickness in neuroischemic feet (24) that may increase their susceptibility to shear forces, which can thus be transmitted to deeper tissues. Age also may influence the soft tissue properties and foot structure, especially in the neuroischemic patients, who tended to be older, although the age differences among the four groups were not statistically significant. Further studies are awaited to elucidate the role of soft tissue in the diabetic foot ulceration and specifically in the neuroischemic foot.

In conclusion, neuroischemic patients who develop ulcers on the margins of the foot, rather than under the plantar surface, have been shown to have abnormally high plantar pressures. This study has also confirmed that neuropathic patients who develop ulcers on the plantar surface of the foot have increased pressures. Thus, high plantar pressures in the presence of ischemia, even associated with neuropathy, are not always associated with plantar ulceration. These findings may suggest different mechanisms of ulcer formation in the two groups of diabetic foot patients: neuropathic and neuroischemic.

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