The Neuropathic Ulcer and Loads on the Foot in Diabetic Patients

I. A. F. Stokes, I. B. Faris & W. C. Hutton


To link to this article: http://dx.doi.org/10.3109/17453677508989271

© 1975 Informa UK Ltd All rights reserved: reproduction in whole or part not permitted

Published online: 08 Jul 2009.

Submit your article to this journal

Article views: 280

View related articles

Citing articles: 1

Full Terms & Conditions of access and use can be found at
http://www.tandfonline.com/action/journalInformation?journalCode=iort19
Diabetic patients often develop ulcers on the soles of their feet. These ulcers are most common under the second, third and fourth metatarsal heads. It is often stated that pressure is the cause of these lesions although there is no quantitative evidence to support this. Neuropathy, both motor and sensory, is believed to be the major predisposing factor (Oakley et al. 1956, Ellenberg 1968). The sequence of events suggested by Ellenberg (1968) is that clawing of the toes, due to weakness of the small muscles of the foot, leads to excessive loads on the region of the metatarsal heads. Callosities occur and small cracks in the skin develop and pass unnoticed because of the sensory neuropathy. These cracks develop into ulcers which may extend to involve the underlying tendon sheath and joint.

In this study we have measured the load distribution under the feet of diabetic patients and normal subjects to determine if:

1. Ulcers occur at sites of maximum load.
2. There are changes in the loading of the foot in patients who have never had ulcers which might predispose to the development of ulcers.

GROUPS STUDIED

Diabetic patients

Twenty-two patients were studied. Each of the patients’ feet was classified independently (and before any measurements were made) into one of three groups, depending upon the severity of the clinical condition. It was assumed that, because of the sensory neuropathy, each foot functioned independently, and that an ulcer or callosity on one foot did not alter the subject’s gait to the extent of abnormally loading the opposite foot. There were seven feet with ray or hallux amputations which were not included in any of the groups, although the unaffected feet of these patients were included in group 2.
The remaining 37 feet were grouped in order of the severity of clinical condition:

Group 1. Feet with penetrating ulcers (four feet) or ulcers which had healed with conservative treatment (two feet).

Group 2. The unaffected foot of patients with ulcers (six feet), ray amputations (four feet), or hallux amputations (three feet). It was assumed that most of the factors predisposing to an ulcer were present in these feet.

Group 3. Diabetic patients without ulcers, but with callosities (18 feet).

Details of the ages and duration of diabetes, along with clinical evidence of neuropathy in these patients are shown in Tables 1 and 2.

Normal subjects

This group consisted of 60 healthy persons of both sexes, aged between 16 and 65 years. Subjects in this group had no obvious foot or gait abnormalities or asymmetries, nor any history of foot complaints or treatment.

METHOD

The patients and normal subjects walked barefoot at their normal walking speed along a walkway. Recordings of load distribution under the foot were made by means of a load sensitive area in the walkway. Details of the apparatus and method have been published by Hutton & Drabble (1972) and Stokes et al. (1974). The load sensitive area consists of twelve beams, each 400 mm × 12 mm, set side by side into the walkway, flush with its surface, to make an area 400 mm × 144 mm.

The vertical component of the total load applied to each beam as the patient walks over it is transduced by means of electrical resistance strain gauges and is recorded on a 12-channel U.V. recorder. Thus, a recording, against a base of time, is made of load on strips of the sole 12 mm wide. The load sensitive area in the walkway can be rotated through 90° so as to be either longitudinal or transverse to the direction of walking. Recordings were made with the apparatus in both configurations from both feet. The averages of three longitudinal recordings and two transverse recordings were used to provide a measure of typical load distributions under the foot.

The recordings made with the beams longitudinal to the direction of walking always show a peak of loading due to the heel, and a peak which occurs during kick-off, when only the forefoot is in contact with the ground. Because we have been concerned with lesions of the forefoot, the kick-off peaks only are selected and recorded by an analogue system connected to each beam (Stokes et al. 1974), and displayed in the form of a stepped graph, which shows the distribution of peak loads across the forefoot. Typical graphs from a normal subject and a patient are shown in Figure 1.

The recordings with the beams transverse to the direction of walking were used to provide a measure of the load carried by the toes alone (except the fifth toe, which normally rests on a beam which also carries part of the ball of the foot). The position of the foot relative to the beams is recorded by means of an inked cloth placed over a piece of paper, which is positioned in register over the beams. By
Figure 1. The peak loading diagram. Typical graphs from the analogue system showing the peak load imposed by the forefoot on each beam of the apparatus. Footprints are shown in correct register. Left: a normal subject. The centre of the area (the centroid) is marked at C, and the position, t, of the highest maximum load is shown. These were used to describe foot function in analysing the results. Right: A patient with a penetrating ulcer. Note that the ulcer site corresponds to the position of the greatest maximum load.

reference to the impression of the foot made on the paper, the load recorded on individual beams can be attributed to particular areas of the foot (see Figure 1).

Thus, for each patient and normal subject two transverse recordings and three longitudinal recordings of load against a base of time were made using the beams, while the relative position of the foot on the beams was found using the inked cloth impression. The information was supplemented by the analogue system's "picking off" the second peak from each longitudinal recording.

RESULTS

The results from the normal group were analysed to test if the measured parameters were influenced by the age or sex of the subjects. No significant differences were found, so the 60 normal subjects were considered as one group (Stokes, unpublished results). The results plotted on Figures 2, 3 and 4 show a mean ± 1 s.d. for each group.
Figure 2. Results obtained from the peak loading diagram. a) The position (I) of the greatest maximum load. b) The position of the centroid C of the diagram.

Groups which are bracketed together are not significantly different at the 5 per cent level.

**Peak loading diagram**

For each patient and normal subject, the peak loading diagram as shown in Figure 1 was used to make the following observations:

1. The distance (l) from the position of highest maximum load to the edge of the diagram. The normal subjects show maximum load occurring more to the medial side of the forefoot, whereas in the patients with ulcers, the maximum load has moved laterally. The deviation from normality increases with the severity of the foot condition (see Figure 2 a).

2. The position of the centroid, “C”, of the diagram (the effective ‘centre line’ of action of the foot) was found and its distance from the medial edge of the diagram was measured. The same trend is shown in that the extent of the deviation from normality increases with the severity of the foot condition (see Figure 2 b).

3. The value of the greatest maximum load was measured in each case (Figure 3). Patients with ulcers have significantly greater maximum loads than the normal group or the patients without ulcers. However, the table of bodyweights in Figure 3 shows the same difference, which suggests that the patients with ulcers have areas of greater loading on the forefoot, due primarily to their greater bodyweight.
Figure 3. The greatest maximum load (see Figure 1) and a table of bodyweight for the three groups of patients and the normal group.

<table>
<thead>
<tr>
<th>Bodyweight (Newtons)</th>
<th>Mean</th>
<th>Standard deviation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>651</td>
<td>128</td>
</tr>
<tr>
<td>Group 3</td>
<td>618</td>
<td>74</td>
</tr>
<tr>
<td>Group 2</td>
<td>794</td>
<td>152</td>
</tr>
<tr>
<td>Group 1</td>
<td>815</td>
<td>163</td>
</tr>
</tbody>
</table>

**Maximum load on the toes**

The value of the maximum load received by the toes was noted in each case, by reference to the transverse recordings. The mean value ± 1 s.d. for each group is shown in Figure 4. All three groups of patients were found to impose statistically less load on the toes than the normal group.

**Penetrating ulcers and callosities**

The position of the greatest maximum load was found in each case to correspond to the position of the ulcer in these four patients (see
Figure 4. The maximum recorded load on the toes expressed as a percentage of bodyweight.

Figure 1). This was also the case for two other patients (not included in the groups) who developed penetrating ulcers on the same foot, after "ray" amputations.

No exact correspondence was found between the position of the greatest maximum load and the position of a callosity in patients in group 3, although callosities did occur at sites of heavy loading.

DISCUSSION

The apparatus used in this study has provided a sensitive method for the study of loading on the foot. The importance of studying the foot during walking has been shown by Stott et al. (1973), who demonstrated that in normal subjects the forces under the foot were greater and more localised in walking than in standing. The study of patients while walking might therefore provide a more satisfactory method for investigating pathology of the foot.

The groups of patients studied represent a spectrum of increasing foot disorder, i.e. normal, diabetics without lesions, diabetics with lesions. The results obtained from this study support this idea. In the
Table 1. Clinical details of patients studied.

<table>
<thead>
<tr>
<th></th>
<th>Group 1</th>
<th>Group 2</th>
<th>Group 3</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Age (years)</strong></td>
<td>Mean</td>
<td>50.5</td>
<td>54.4</td>
</tr>
<tr>
<td></td>
<td>Range</td>
<td>39–66</td>
<td>40–70</td>
</tr>
<tr>
<td><strong>Duration of diabetes (years)</strong></td>
<td>Mean</td>
<td>9.0</td>
<td>13.6</td>
</tr>
<tr>
<td></td>
<td>Range</td>
<td>1–17</td>
<td>3–26</td>
</tr>
</tbody>
</table>

Table 2. Clinical evidence of neuropathy.

<table>
<thead>
<tr>
<th></th>
<th>Group 1 (6 feet)</th>
<th>Group 2 (13 feet)</th>
<th>Group 3 (18 feet)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Reflexes</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Knee and ankle reflexes</td>
<td>...</td>
<td>2</td>
<td>10</td>
</tr>
<tr>
<td>Knee reflex present, ankle reflex absent</td>
<td>3</td>
<td>5</td>
<td>8</td>
</tr>
<tr>
<td>Knee and ankle reflexes absent</td>
<td>3</td>
<td>6</td>
<td>...</td>
</tr>
<tr>
<td><strong>Sensation</strong> (pinprick test)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Intact</td>
<td>...</td>
<td>2</td>
<td>10</td>
</tr>
<tr>
<td>Loss in toes only</td>
<td>1</td>
<td>5</td>
<td>8</td>
</tr>
<tr>
<td>More extensive loss in foot/leg</td>
<td>5</td>
<td>6</td>
<td>...</td>
</tr>
</tbody>
</table>

diabetic patients the position of the maximum load on the foot and the centroid of the peak load diagram are shifted laterally. Although these changes are associated with evidence of increasing neuropathy, the precise mechanism for their development is not clear, but could result from alterations in the balance between inverting and everting muscles of the foot. This could be due to weakness of the muscles or to loss of coordination because of loss of afferent impulses from tendon receptors. The trends noted probably mean that areas under the lateral metatarsal heads are carrying increased loads as the disorder progresses. This would explain the relative infrequency of ulcers under the first metatarsal head.

The increasing abnormality in loading on the foot in the three groups of patients corresponds with increasing evidence of neuropathy (Tables 1 and 2). Extensive loss of skin sensitivity was noted in all feet in group 1, and most of the feet in group 2. Deep pain sense was probably equally affected, since all the ulcers were painless.

The most striking finding is the reduction in load on the toes which is significantly present in the diabetic patients without lesions. It is
likely that this is an early result of neuropathy which causes changes in muscle balance by denervation of the intrinsic muscles of the foot. These changes have been confirmed by EMG and nerve conduction studies, the results of which will be published separately (Harrison & Faris 1974). This finding supports the sequence of events outlined in the introduction and suggests a mechanism by which load is concentrated on the metatarsal heads and the underlying tissues.

This study has demonstrated an area of heavy loading corresponding to the site of the ulcer present in the patients with neuropathic lesions. This corroborates the static and semi-quantitative results reported by Barrett & Mooney (1973). In patients whose feet have been deformed by operation, the alterations in weight distribution produced might predispose to the development of further lesions since the neuropathy is still present. In patients who have had a ray amputation we have demonstrated heavy loads on areas of the foot which have gone on to develop recurrent lesions. Such an alteration in loading is probably an inevitable sequel of ray amputations in which the toe and distal part of a metatarsal are removed, thus reducing the number of weight-bearing metatarsal heads. Transmetatarsal amputation (Wheelock 1961), which retains five weight-bearing metatarsal ends, might not have this disadvantage but we have not yet been able to test this.

The lack of exact correspondence between the position of callosities and the measured position of maximum vertical load on the forefoot indicates that there could be other factors which are important in the development of callosities.

The ability to identify the areas at risk for further ulceration gives information with which to plan preventative measures. Adequate footwear and chiropody remain essential for the care of these patients. Further studies are being carried out to test the effectiveness of splints designed to alter the loading on the foot. These might delay the development of recurrent lesions and would represent an advance in therapy.

SUMMARY

Normal subjects and diabetic patients with and without foot ulcers have been studied using an apparatus which measures the loads on the foot during walking. Diabetic patients have alterations in loading which show as a lateral shift of the highest maximum load on the forefoot and a decrease in the load carried by the toes. There is a significant pro-
gression of these changes between normal subjects, diabetic patients with deformity of the foot but no ulcer, and diabetic patients with foot ulcers. All the patients with ulcers exerted maximum loads at the site of the ulcer.

ACKNOWLEDGEMENTS

The authors wish to thank Professor L. P. Le Quesne and Dr. J. D. N. Nabarro of the Middlesex Hospital, London, for permitting us to study patients in their care. The Polytechnic of Central London provided facilities for the work, Mr. M. B. Undy of the Polytechnic gave considerable help with the statistical analysis of the results, and Mr. E. Graham did the drawings. This research is supported by a Medical Research Council grant. I.F. received a John Astor Fellowship of The Middlesex Hospital Medical School.

REFERENCES


Key words: diabetes; neuropathic ulcers; foot; load distribution; callosities; walkway

Correspondence to:

I. A. F. Stokes, M.A.
School of Engineering and Science
Polytechnic of Central London
115 New Cavendish Street
London W1M 8JS
England

I. B. Faris, F.R.A.C.S.
Department of Surgery
Perth Medical Centre
Shenton Park WA 6008
Australia