Foot ulceration in diabetes: the biomechanical factor

Introduction

The risk of developing a foot ulcer and the potential to proceed to limb amputation is a feared complication amongst people with diabetes. It has been shown that foot ulceration precedes amputation in up to 85% of lower limb amputations. ¹ For healthcare providers involved in the treatment of foot ulceration, it is often a frustrating experience, as reulceration is common after great effort and cost to achieve a healed status. This article looks at the biomechanical factors involved in the causal pathway, the healing process and the prevention of ulceration and reulceration of a foot ulcer in a person with diabetes.

Contributing factors to ulceration

Foot ulcer development in diabetes is multifactorial. Peripheral neuropathy is the main role player and it is exacerbated by peripheral vascular disease and infection. The synergy of these three contributing factors in the presence of poor glycaemic control often leads to amputation of the involved lower limb. Peripheral neuropathy has been identified as the underlying cause of foot ulceration in 60% of foot ulcers in persons with diabetes.² Distal symmetrical polyneuropathy involves motor, sensory and autonomic fibres but it is the sensory loss that predominates. In diabetes the normal gait cycle may be altered and this can also contribute to foot ulceration. Altered biomechanics and deformity of the foot in diabetes is associated with peripheral neuropathy, specifically the involvement of the motor fibres.

Biomechanics of the foot

In the normal gait pattern, the whole body is moved forward in all three directions of space (horizontal, vertical and forward) by a combination of internal and external forces. During the gait cycle the foot has to cope with vertical forces (heel strike), which reach a peak pressure involving 110-120% of the body weight. The role of the foot and lower limb as a shock absorber is therefore crucial to prevent damage to the inner structures and the skin. At heel rise (second phase), the foot becomes a rigid lever to prepare for the forward propulsion of the body.

The last phase of the gait cycle is when the heel lifts and the vertical component of force peaks again, involving approximately 120% of the body weight, and the body is propelled forward. Ambulation is not possible without friction, as an equal and opposite force must be delivered by the ground surface. In barefoot walking, a force equal to 20% of the body weight is exerted at the heel-strike and the propulsion phases.

High pressure points have long been identified as a major risk factor for plantar ulceration in people with diabetes and neuropathy.³,⁴ A peak plantar pressure of over 6 kg/cm² has the highest specificity in identifying patients at risk for ulceration.⁵ High pressure may be the result of structural changes of the soft tissue and bony structures of the feet. Many studies have looked at the causal factors for deformity and development of foot ulceration such as peripheral neuropathy, limited joint mobility and muscle atrophy.

One theory is that deformity or overextension of the metatarso-phalangeal joints and flexion of the distal interphalangeal joints is caused by an imbalance between the extrinsic and intrinsic muscles of the feet as a result of motor neuropathy (Figure 1).⁶,⁷ This finding was confirmed by Bus et al who reported atrophy in the most distal foot muscles in 73% of neuropathic patients with diabetes.⁸ Ledoux found that both intrinsic muscle atrophy and thickening of the plantar...
fascia are required for the development of claw toes in neuropathic feet of people with diabetes (Figure 2). Other researchers observed fat infiltration into the muscle tissue similar to that seen in muscular dystrophy.

Sicco et al reported migration of the fatty pad over the plantar metatarsal area as a result of the hyperextension of the metatarsophalangeal joints. This results in high pressure over the metatarsal area, as the fat pad provides cushioning and protects the skin from damage during walking (Figure 3). Findings by Robertson support structural changes in the forefoot in persons with diabetic neuropathy and history of ulcer, compared to those without.

Limited joint mobility of the first metatarsophalangeal joint, ankle joint and subtalar joints affects the foot-to-floor patterns during gait. This results in longer and greater loading of the metatarsal heads, with subsequent ulceration. Changes in the plantar fascia contribute to increased pressure over the plantar metatarsal areas in persons with diabetes, even in the absence of peripheral neuropathy. These changes may occur before the development of peripheral neuropathy due to the glycation of proteins. Shortening of the Achilles tendon and stiffening of the plantar fascia result in forefoot equinus and increased pressure under the forefoot.

In the presence of neuropathy, plantar pressures are increased, not only under the metatarsal areas but also at the heels. It has been suggested that the forefoot to rearfoot pressure ratio is increased only in the most advanced neuropathic patients, supporting the concept that the equinus foot only develops in the later stages of neuropathy.

It is important for the clinician to identify these risk factors early, before ulceration occurs. A clinical examination should include a biomechanical assessment to identify any of the factors described above. It is therefore essential that patients at risk of ulceration are referred to a podiatrist with a good understanding of the structure and function of the foot. They would typically use validated risk-assessment tools to conduct a complete foot examination of the diabetic foot at risk.

**Risk classification**

A number of risk categorisation tables have been developed and validated for use by clinicians, setting international standards on diagnostic, preventative and therapeutic strategies. In validating the risk classification system developed by the International Working Group on the Diabetic Foot (IWGDF), it was found that patients at higher risk for foot ulceration have a longer history of diabetes, poorer glycaemic control, a greater degree of neuropathy and an increased plantar pressure. Patients with a history of amputation were found to have an even higher risk of further complications. Other risk factors for foot ulceration are a history of alcohol abuse and the presence of other long-term complications of diabetes, such as retinopathy and nephropathy.

These factors should be identified at the initial foot risk assessment by taking a thorough history and assessing for sensory loss, occlusive arterial disease, foot deformity, foot function and related skin changes. The modified IWGDF classification system has been validated and found to be effective at predicting diabetic foot complications (Table I).
**Biomechanical assessment**

At the initial assessment of the foot, the presence of foot deformities such as bunions, claw toes, overlapping toes and limited joint mobility of the first metatarsophalangeal joint and ankle joint in the presence of sensory loss should be noted. The presence of hallux limitus is defined as dorsiflexion of less than 50 degrees at the metatarsophalangeal joint and less than 0 degrees at the ankle. The presence of callus or erythema may indicate areas of high pressure or stress with impending breakdown. Plantar peak pressures can also be identified using ink mat (the Podotrack®) or a computerised system.

**Reulceration**

Reulceration of the foot in persons with diabetes is common, with rates varying between 35-40% over a three-year period, increasing to 70% over five years. The site of reulceration was found to be the same foot, but not the same site, implicating abnormal biomechanics in the pathway to reulceration. A study by Galea established that the 12 months following healing is the most high-risk period for reulceration, and patients should therefore be monitored regularly for early signs of reulceration in this period. The majority of ulcers were found to be on the plantar surface of the foot and on the apex of the toes. The distribution of the reulcerated lesions suggested that the biomechanical problem had not been addressed to prevent reulceration. The effectiveness of therapeutic shoes in secondary prevention was demonstrated by Edmonds et al with a reulceration rate of 26% among those wearing therapeutic shoes compared to 83% among those wearing retail shoes. Similar findings were reported by Busch et al.

**Therapeutic footwear**

For people at high risk of ulceration or those with a history of ulceration, therapeutic footwear should be prescribed to protect, improve function of and prevent injury to the feet. The effectiveness of therapeutic footwear depends largely on the patient’s usage and acceptance of the shoe. It is therefore essential that patient-centred concerns are addressed before prescribing.
footwear. Tyrell and Carter recommend that the following aspects should be considered: the patient's choice of shoes, perceptions of footwear and criteria that are significant to them.

Foot amputations and biomechanics

It is conceivable that the risk factors for amputation are the same as for ulceration, with a few modifications. Peripheral arterial disease is not considered an independent risk factor for ulceration, but it is for amputation, and it is also a predisposing factor for gangrene. Infection is a significant risk factor for amputation, as it is for extensive soft tissue necrosis, deep space abscess and osteomyelitis requiring extensive debridement. Amputation has also been associated with elevated glucose and diabetes-related co-morbidities such as nephropathy, retinopathy and cardiovascular disease.

History of amputation is regarded as the best predictor of reamputation. Foot amputation causes a disturbance of the architecture and biomechanics of the foot leading to poor distribution of stress points, high peak pressures and reulceration (Figures 7 and 8). Surgery should be performed in a manner to allow for optimal function from the remaining portion of the foot or lower extremity depending on the vascular status. Armstrong proposed four categories of surgical intervention as listed in Table II.

Pedal amputations at different sites will have predictable effects on the biomechanics of the foot. Amputation of the hallux will alter the propulsion stage and the action is transferred to the second metatarsal or other lesser metatarsals, resulting in an increase of ulceration over these areas, as well as neuropathic fractures of the metatarsals. The amputation of the second metatarsal has a lesser effect on the biomechanics, but will cause the hallux to drift laterally resulting in a hallux valgus deformity and possible deformity of the lesser toes. It was found that 65% of patients who had an amputation of a hallux re-presented with reulceration, with 53% requiring further proximal amputation. Reulceration after amputation of a lesser toe was only found in 10% of patients. A partial ray amputation may result in increased pressure over the remaining metatarsal heads of the foot. The action of peroneus brevis can be lost if the amputation of the fifth metatarsal ray extends proximal to its insertion, resulting in a supinating deformity of the foot. In the case of transmetatarsal amputations, the greatest impact on the biomechanics of the remaining foot is the level at which the amputation, is performed. Care should be taken in selecting the level of amputation, as longer metatarsals will result in a greater lever arm for propulsion. A plantar flexion tendency resulting from the unopposed action of the Achilles tendon and tibialis posterior will result in overloading of the forefoot, with an increased risk of ulceration in this area.

Foot ulceration and offloading

Reducing mechanical stress over the area of ulceration is essential for healing. The most extensively studied modality for offloading is the total contact cast. Other strategies for offloading are bed rest, foams, felt, half shoes, healing sandals and other types of casts. This treatment modality is discussed fully in an article by Robins in a previous edition of this journal.

Table II: Surgical intervention in the diabetic leg

<table>
<thead>
<tr>
<th>Class</th>
<th>Goal</th>
<th>Example</th>
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<tbody>
<tr>
<td>Elective surgery</td>
<td>Relieve pain in patient without loss of sensation</td>
<td>Neuromas, bunions</td>
</tr>
<tr>
<td>Prophylactic surgery</td>
<td>Reduce the risk of ulceration in patients with loss of sensation without an open wound</td>
<td>Claw toes, exostosis, Achilles tendon lengthening, correction of previous surgical interventions</td>
</tr>
<tr>
<td>Curative surgery</td>
<td>Assist in wound healing</td>
<td>Resection of infected bone and joints, often combined with surgical flaps, skin grafts</td>
</tr>
<tr>
<td>Emergency surgery</td>
<td>Arrest or limit progression of acute infection</td>
<td>Removal of all necrotic or infected tissue, amputation at level of viable tissue</td>
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Figure 7: An illustration of a partial foot amputation resulting in an equinus deformity of the remaining foot. The ulceration over the medial malleolus is not related to the equinus deformity and pressure, but ill-fitting shoes requiring a brace to keep the shoe in place, causing friction and shear forces over the bony prominence.

Figure 8: Amputation of digits with associated reulceration over the plantar metatarsal area.
Conclusion

Better understanding of the mechanics that play a role in the ulceration process of the diabetic foot is essential in the timely detection of the foot at risk, identification of the factors that can impede the healing process and implementation of secondary measures to prevent plantar ulceration of the foot in people with diabetes. Podiatric assessment and intervention can add value to the interprofessional team effort and can provide patient-specific guidance on the ease and availability of improved style therapeutic shoes, in order to increase patient acceptability and subsequent adherence to regular use of the therapeutic footwear.

References:

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