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DECOMPRESSION WITH THE AID OF INSOLES IN THE TREATMENT OF DIABETIC NEUROPATHIC ULCERS

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Thirty-seven out of 38 neuropathic ulcers in 21 diabetic patients healed during relief of external pressure obtained by properly fitted interchangeable insoles. The time required for healing was 1 to 12 months (mean 3.6 months). The presence of mild occlusive arterial disease did not influence the course of healing. Gross infection, which occurred in eight patients could be controlled by immobilization, antibiotics and, in the presence of pus, by radical surgical drainage.

Key words: diabetes; foot wear; insoles; neuropathic ulcers; skin perfusion pressure; strain gauge plethysmography

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The term neuropathic ulcer is used for the special type of lesion that may occur in a neuropathic foot regardless of the cause, e.g. diabetes mellitus, tabes, syringomyelia, hereditary sensory radicular syndrome or leprosy. Other terms employed are malum perforans, perforating ulcer of the foot, trophic ulcer and plantar ulcer. The lesion is caused by impaired sensory perception, either peripheral or central in origin, in combination with increased mechanical stress on a limited skin surface. The patient has no warning mechanism and is unaware of where and how he places his foot and he does not perceive trauma. As the patient is usually able to walk without any handicap, the lesion shows very little tendency to spontaneous healing.

The vast majority of neuropathic ulcers in our patients are found in diabetic patients. The ulcer is typically found on the feet where the normal architecture is lost, e.g. under a prominent metatarsal head (Figure 1) or on the pulp or the tip of a rigid toe. Penetration to tendons and bones is not uncommon. A callosity surrounds the lesion indicating the increased mechanical stress.

Decompression of the ulcers can be achieved by carefully manufactured insoles. In most cases this therapy is effective as will be described in the present study.

MATERIAL AND METHODS

Patients

In a 3-year period (1.10.1971 to 31.8.1974) 38 neuropathic ulcers in 21 diabetic patients were treated. The series is consecutive including diabetic patients with a pain-free ulcer in a

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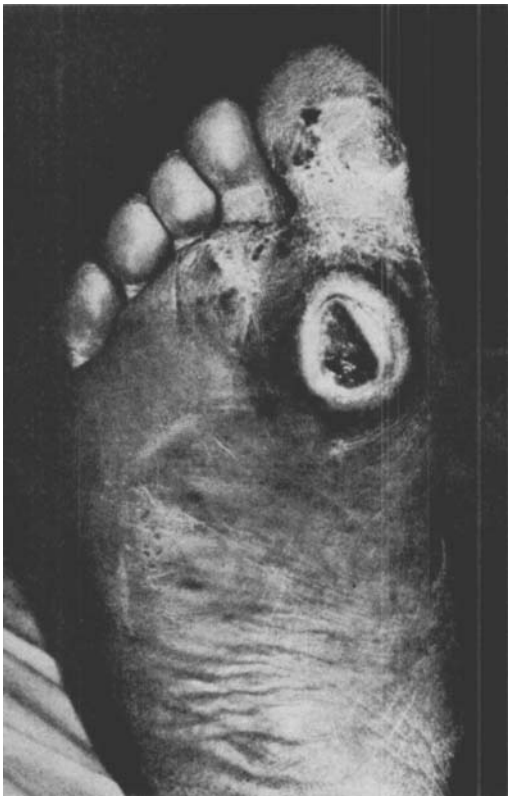

Figure 1. Neuropathic ulcer.

skin area exposed to abnormally high external pressure as evidenced by a callosity and in whom severe occlusive arterial disease could be excluded.

The localization of the ulcers is shown in Figure 2 and the duration before therapy in Table 1. The series includes four relapses of a healed ulcer and six new ulcers, i.e. ulcers with a new localization which occurred in the follow-up period of 3 to 35 months (mean 16.3 months). Ten patients were men and 11 were women. Their ages ranged from 48 to 74 years (mean 59.9 years). Diabetes of more than 10 years duration was noted in 11 patients and diabetes of 5–10 years duration in five patients. Ten patients required insulin and in 11 patients the diabetes was controlled by diet and peroral hypoglucaemics. The blood sugar in most patients was satisfactorily controlled.

Method

Basically the external pressure to the ulcer is simply reduced by distributing the pressure to a greater skin area, in most cases to areas proximal and bilateral to the ulcer. This is obtained by an interchangeable insole, in which a flat excavation corresponding to the size of the ulcer is created. The insole is made of light thermoplastic, washable materials (Plastozote®, Rubazote®, Hindafoam®). It is important that the excavation in the insole is slightly greater in size than the ulcer, so that the callous rim is also relieved from pressure (Figure 3 (3)). Distally, the edge of the excavation is levelled or removed (Figure 3 (4) and Figure 4). This arrangement allows healing of the ulceration without restricting the walking activity of the patient. The size of the excavation in the insole is diminished during the course of healing, and new insoles are required from time to time.

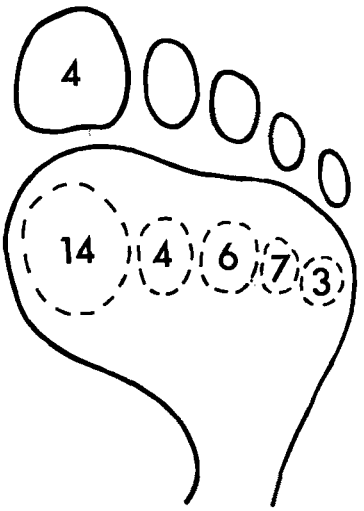

Figure 2. Distribution of 38 neuropathic ulcers.

Partial insoles are often used initially (Figure 4), especially in the case of major discharge from the ulcer. Then the excavation must be large enough for a dressing which should be arranged without wrinkles (ETE® compress). In narrow, deep ulcers a slim wick is placed for drainage. Tight plugging of the ulcer must be avoided. Later a complete insole is manufactured, preferably after imprints in a plastozote model sole on which the patient has walked for a week or two. When the wound has healed, possibly after a period of several months, prophylaxis is achieved by a leather covered insole with a suitable resid-

Table 1. Duration of the ulcers.

1– 7 years	3
3–12 months	5
1– 3 months	3
< 1 month	20
Unknown	7

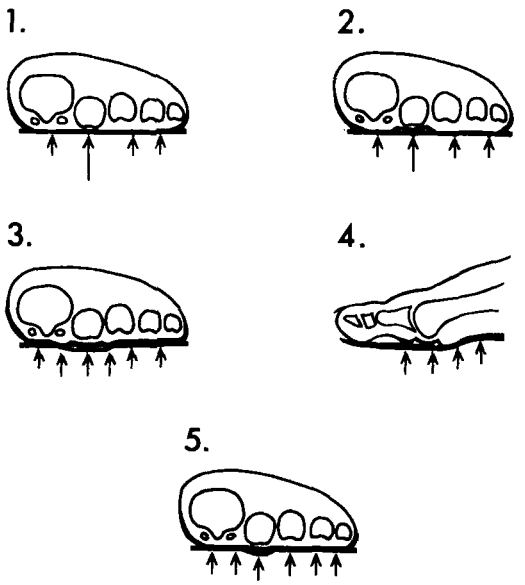

Figure 3. Protrusion of a metatarsal head (1) causing ulceration in the neuropathic foot (2). The insole has an excavation large enough to relieve pressure from the ulcer including the callous rim (3). Distally the edge of the excavation is levelled (4). Prophylaxis is achieved by a suitable small excavation after healing of the ulcer (5).

ual excavation (Figure 3 (5)). In our hospital, if no complications occur, the ulcers are treated by chiropodists who trim the callosities and manufacture and adjust the insoles*.

The arterial condition of the limbs was determined by measurement of the distal *systolic blood pressure* on the ankle and on the first toe (or second toe) by strain gauge technique (Gundersen 1972, Nielsen et al. 1972). The distal *perfusion pressure* was measured on the dorsal side of the foot by isotope technique or on the heel by photoelectric technique (Holstein & Lassen 1973, Holstein et al. 1976). According to the criteria given by Nielsen & Munkgaard Rasmussen (1973) 10 patients had normal distal blood pressures, i.e. no arterial occlusions, and 11 patients suffered from occlusive arterial disease of varying degree. Six of these patients had intermittent claudication. Severe occlusive arterial disease suggestive of ischemia as a cause of slow healing ulcers was excluded by accepting only ulcers in limbs where all distal blood pressures were above 40 mmHg (Lassen & Holstein 1974).

* The above principles have been taught at the school for chiropodists in Copenhagen since 1969.

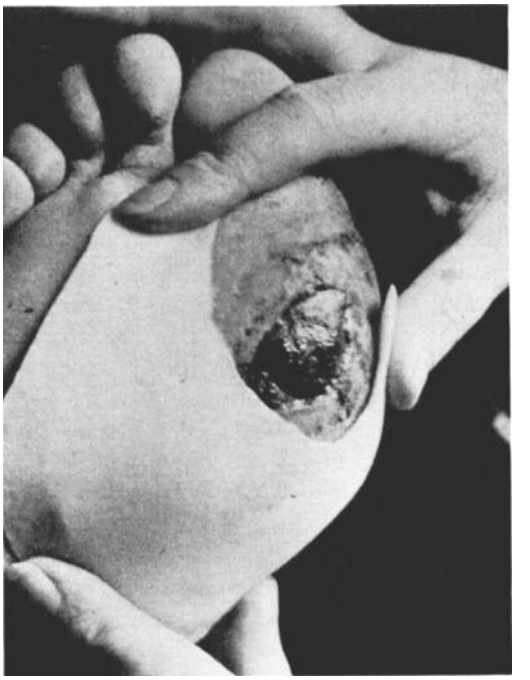

Figure 4. Partial washable insole.

The severity of the neuropathy was determined by clinical examination with the exception of the vibration sense which was measured by biothesiometry (Steiness 1957). Table 2 shows the neurological findings.

Table 2. Evidence of neuropathy.

		No. of patients
Reflexes:		
Knee and ankle reflexes present		1
Knee reflex present, ankle reflex absent		5
Knee reflex absent, ankle reflex absent		15
Sensation in the toes:		
Intact		3
Vibration sense	absent	12
	decreased	6
Position sense	absent	2
Perception of pinprick	absent	13
	decreased	5
Perception of temperature	absent	9
	decreased	9
Perception of light touch	absent	2
	decreased	5

RESULTS

In only one case was healing of the ulcer not obtained. The ulcer, 16 mm in diameter, was localized under the first metatarsal head and had persisted for 7 years when the treatment was initiated. Three years later the patient died. At this time the ulcer was 5 mm in diameter. No local complications of the ulcer occurred during the period of treatment and the patient had no sign of occlusive arterial disease. The other 37 ulcers healed in an average of 3.6 months (range 1–12 months). The time required for healing of the ulcers could not be related to the duration of the diabetes or the severity of the neuropathy. Nor did the presence of mild occlusive arterial disease influence the healing time which was 3.6 months (range 1–11 months) against 3.5 months (range 1–12 months) in diabetics without occlusive arterial disease.

Eight patients had deep infection as a complication to the ulcer when first admitted to the hospital. In five patients the infection was cured by high doses of appropriate antibiotics given systematically. In three patients the deep infection resulted in osteitis. Long-term antibiotic treatment cured one case. In two other cases control of the infections was achieved by local but radical surgical drainage combined with long-term antibiotic treatment.

The four relapsed ulcers healed after the treatment was recommenced, and the six ulcers occurring at new sites healed after suitable adjustment of the insoles.

DISCUSSION

Lesions on the feet in diabetic patients were classified by Oakley (1954) as neuropathic, septic, ischaemic or due to a combination of these causes. This classification gives the key to the proper therapy and should replace the non-specific term "diabetic gangrene" which is still commonly used.

The importance of an objective determination of the arterial supply should be emphasized. Ischaemia of the toes may be present even when the pedal pulses can be felt (Holstein & Sager 1973) and only methods that evaluate the most peripheral part of the circulation suffice, as was also recently pointed out by Faris (1975). *Vice versa* the blood supply may be adequate for the healing of wounds in spite of absent peripheral pulses. The results in this series demonstrate that mild occlusive arterial disease does not affect the healing of the ulcers.

In the case of deep infection, possibly with extensive necrosis of soft tissues, the clinical assessment of the distal circulation is especially difficult. We consider deep infection in a diabetic foot as an emergency case. Immobilization and proper antibiotics given in the early phase may be adequate, but the patient often comes late because pains are moderate or lacking. Then in the presence of pus radical surgical drainage is mandatory and if the blood supply is not too inadequate most limbs can be saved. In severe occlusive arterial disease, diabetic foot lesions seldom show the clear-cut characteristics of the chronic neuropathic ulcer and ischaemia is the factor which determines the localization, appearance and prognosis.

It is generally accepted that the two important factors in the pathogenesis of the neuropathic ulcer are the increased mechanical stress and the sensory neuropathy. Stokes et al. (1975) measured quantitatively the load distribution under the feet of diabetic patients walking *barefoot* on a load sensitive area. All patients with ulcers exerted maximum loads at the site of the ulcer. Diabetics without ulcers, as compared to normal subjects, had alterations in loading which showed as a lateral shift of the highest maximum load on the forefoot and a decrease of the load carried by the toes. Semiquantitative measurements *inside*

the shoes during walking were reported by Barrett & Mooney (1973) who demonstrated high frictional forces at the ulcer-shoe interface. The in-shoe testing also demonstrated an abnormal pressure distribution on the forefoot as compared to barefoot walking.

In our series there was a relative high frequency of ulcers localized under the first metatarsal head as was also found by Kelly & Coventry (1958). The lateral shift of the maximum load as demonstrated by Stokes et al. (1975) is one interesting factor probably related to motor neuropathy. But the site of the ulcer (or the callosities) is probably determined by a series of factors including also the individual abnormalities of the architecture of the foot, the pattern of the gait and the type of foot wear.

The neurological state of diabetics with neuropathic ulcers was recently studied by Barrett & Mooney (1973). The area immediately around the ulcers was painless, but this sensory disturbance did not imply the whole distribution of a particular peripheral nerve. The authors suggested that local damage to the small sensory nerve endings was an important factor in the absence of pains. In our series there were three patients with intact sensation of the toes and feet in spite of a pain-free ulcer of typical localization and appearance. This supports the theory of local nerve damage. In this instance it is notable that the mechanical factor in these three patients was extreme: one patient had bilateral pes cavus and the two other patients were obese.

The mechanical factor is the one which may be eliminated. Good results have been achieved by surgical decompression (Martin 1954, Oakley et al. 1956, Kelly & Coventry 1958, McCook et al. 1966, Catterall 1968, Ellenberg 1968). It is often pointed out that surgical therapy was undertaken when conservative measures of relieving pressure such as bed rest, use of walking casts and proper shoes had failed. However, surgical therapy such as ray resection is in principle unattractive, as it reduces the weightbearing area, possibly exposing new skin areas to intolerable external pressure. This has been demonstrated objectively by Stokes et al. (1975).

The interchangeable insole is an alternative method of eliminating the mechanical factor. Frequent adjustments of the insoles according to the imprints corresponding to the heavily loaded areas make possible a proper pressure distribution and after healing of the wound prophylaxis is established.

The four relapses in this series occurred after the patients had omitted to use their insoles. We have occasionally seen ulcers appearing after the patients had been provided with individual hand-made shoes for foot deformities and, in the literature, warnings against rigid shoe wear with relief pads as conventionally used for metatarsalgia are found (Oakley et al. 1956, Barrett & Mooney 1973). For these reasons any patient susceptible to neuropathic ulcers should benefit from individual insoles, which would also be a valuable and easily adjustable supplement to hand-made shoes.

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