

## Limb salvage in diabetics with foot ulcers

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### Abstract

The healing results in 491 ulcers in 272 diabetic patients are reported. Soft moulded insoles and shoe corrections were the main part of the therapy. There were 329 (67%) neuropathic, 87 (17%) traumatic, 44 (9%) ischaemic and 31 (6%) ulcers of other various pathogenesis. Thirty seven per cent of the ulcers were complicated with invasive infection. Within the period of observation of 18 months (3-39 months) healing was obtained in 79% of the patients (88% of the ulcers) and major amputation was carried out in 8% (4% of the ulcers). There were 21 major amputations, which in 18 cases was due to ischaemia. Thus in only 3 cases (1% of the patients) neuropathy as complicated by invasive infection caused major amputation. Fifty nine ulcers (12%) were classified as relapsing ulcers or ulcers with new localizations and were caused by severe deformity of the foot (58 cases) often in combination with neglect of prophylaxis (7 cases). Only one recurrent ulcer was caused by ischaemia. The series shows that shoe corrections and insoles are effective in treating diabetic neuropathic ulcers. Recurrent ulcerations are caused by severe foot deformity and neglect of therapy. Loss of limbs is caused by ischaemia and invasive infection.

### Introduction

The risk of amputation in the diabetic patient has been found to be increased 15 times as compared to the non-diabetic patient (Most and Sinnock 1983). Recent studies, however, suggest that the progressive destruction of the neuropathic or gangrenous foot can be stopped and the number of amputations substantially decreased (Holstein et al., 1976; Wagner, 1979; Brand, 1979; Lippmann, 1979; Runyan et al

1980; Larsen et al., 1982; Pollard and Le Quesne, 1983; Burden et al., 1983). The present study was undertaken to evaluate healing results and to identify high risk patients as regards recurrent ulceration and risk of amputation.

### Patients and methods

#### Patients

The series is consecutive consisting of 272 patients with 491 ulcers treated in Steno Memorial Hospital from 01.12.79 through 30.11.82. In 204 patients there was unilateral and in 68 bilateral ulcers. Figure 1 shows the age distribution and the treatment of diabetes. About one third were younger than 50 years, mostly insulin dependent and 48 per cent of the ulcers occurred in patients active in their jobs. Figure 2 shows the duration of diabetes mellitus, which in 24 per cent was of more than 30 years duration. Many of the patients were over-weight (Table 1), although being under-weight did not prevent ulceration.

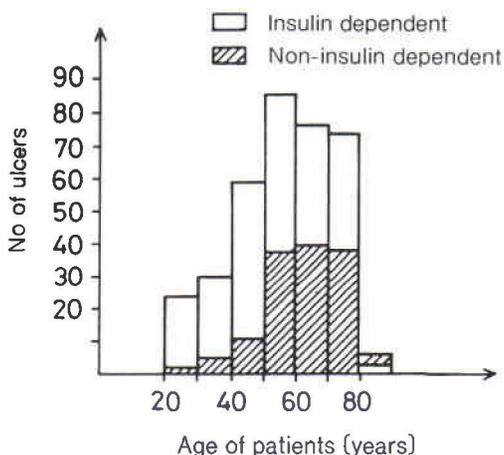


Figure 1: Treatment of diabetes and age distribution of patients.

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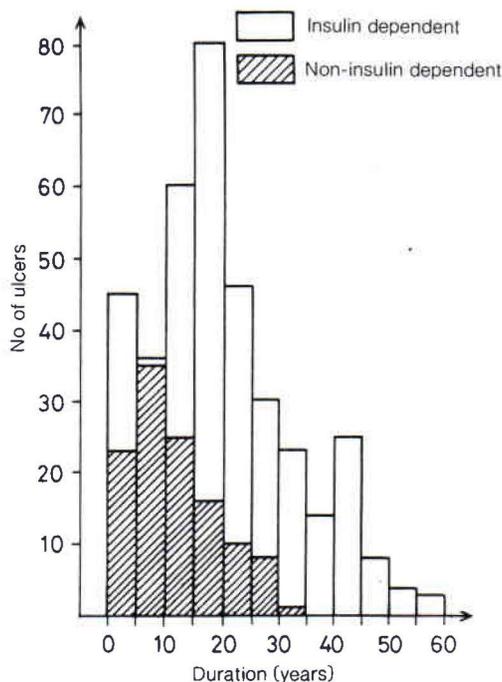


Figure 2: Treatment of diabetes and duration of the disease.

Previous amputations were recorded as follows: one above-knee, 20 below-knee and 22 on the toes or on the forefoot.

### Ulcers

Table 2 shows the duration of the ulcers before treatment in the present hospital. The duration did not, however, correlate with the healing time. The localization of the foot ulcers is shown in Figure 3, highlighting the importance of proper footwear, insoles and protection of the heel. Table 3 shows the pathogenesis, most ulcers having been caused by repetitive stress (Brand, 1979). Ischaemic ulcers were defined by the finding of a digital

Table 1. Bodyweight\* and number of ulcers.

Normal	256
Over-weight	187
Under-weight	49

\*According to weight table by Natvig (1956).  
Normal weight: Ideal weight  $\pm$  10 per cent.

Table 2. Duration of ulcers

<1 month	318	65%
1 $\leq$ 3 months	109	22%
3 months $\leq$ 1 year	44	9%
>1 year	20	4%

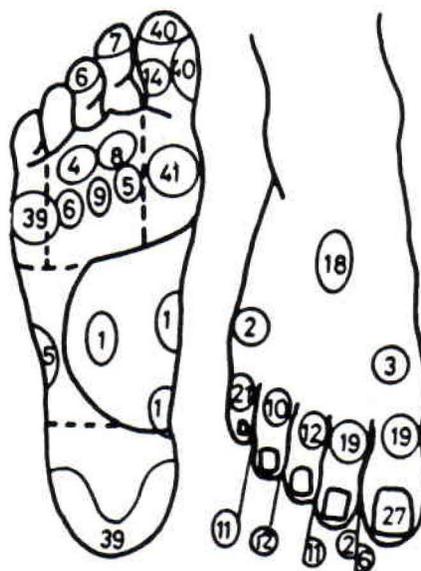


Figure 3: Localization of 439 ulcers. The drawing does not include ulcers around the malleoli and ulcers following digital amputations.

blood pressure less than 40 mm Hg. Fifty nine ulcers were classified as relapses or new ulcers in previously healed feet. Invasive infection occurred in as many as 37 per cent of the ulcers.

### Methods

Two main principles in treating plantar foot ulcers were followed: 1) Abnormal varus or valgus load present in most cases was corrected with modules made from soft materials (3-5 mm Rubazote<sup>R</sup>) and adjusted to balance the foot. 2) Abnormal pressure points were compensated for by pads of the same material. This compound insole was corrected during the course of healing resulting in a final device for permanent use, eventually covered with thin leather. Moreover, prominent structures, e.g. claw toes were protected with silicone rubber pads. Venous ulcers were treated with zinc paste bandage followed by compression stockings. More spacious shoes were prescribed whenever necessary, possibly as orthopaedic

Table 3. Pathogenesis

Neuropathic	329	(67%)
Traumatic	87	(17%)
Ischaemic	44	(9%)
Venous	27	(5%)
Miscellaneous	4	(0.8%)

Table 4. Footwear and ulcers

Unsuitable footwear	205	(42%)
Suitable footwear	283	(57%)
No footwear	3	(0.6%)

footwear (Table 4). In cases of suspected ischaemia, digital and ankle blood pressures were measured with strain gauge plethysmography.

Minor necroses were excised often by repeated small revisions without anaesthesia. Digital amputations were done during local anaesthesia in Steno Memorial Hospital whereas major amputations were done in collaborating hospitals. Invasive infection in the form of osteitis or plantar abscess was treated with surgical drainage supported by antibiotics according to cultures. Great attention was always paid to blood sugar control. The vast majority of the series (83 per cent) were treated in the out-patient clinic.

### Results

The series was evaluated 3 months after the end of the period studied yielding a mean time of observation of 18 months (3–39 months). Out of 491 ulcers 88 per cent healed, i.e. 79 per cent of the patients (Table 5). The healing rate was statistically significant correlated with digital as well as ankle blood pressures (Table 6). Healing of ulcers was not related to their localization.

Eight per cent of the patients had major amputation below the knee, i.e. in four per cent of the ulcers. Of these 21 amputations 18 were caused by ischaemia and three by infection originating from heel necrosis. Vascular reconstruction was only attempted in one ischaemic limb and was not successful.

Table 7 demonstrates the pathogenesis of 59 cases of recurrent ulceration found in 46 patients. One was ischaemic and 58 were neuropathic, all of these occurring in severely deformed feet. Eight of the patients had neglected the prophylaxis.

Table 5. Healing rate

	Death	Still under treatment	No follow-up	Major amputation	Healing
Neuropathy	2	5	18	3	301
Trauma	1	0	0	0	86
Ischaemia	8	1	0	18	17
Venous	0	2	1	0	24
Misc.	0	0	2	0	2
Total number of ulcers 491	11 (2%)	8 (2%)	21 (4%)	21 (4%)	430 (88%)

Table 6. Healing in relation to distal blood pressure

Systolic digital blood pressure	Healing rate	
< 20 mm Hg	2/7 (29%)	P < 0.001
20–29 mm Hg	11/16 (69%)	
30–39 mm Hg	7/9 (78%)	
≥ 40 mm Hg	39/41 (95%)	
Systolic ankle blood pressure		
< 50 mm Hg	1/4 (25%)	P < 0.05
50–89 mm Hg	12/17 (71%)	
≥ 90 mm Hg	53/60 (88%)	

Table 7. Relapses and new ulcers

Neuropathic	58	}	plantar ulcer	26
			first toe	11
			digitus malleus	11
			misc.	10
Ischaemic	1			
	59			

### Discussion

The localization of the ulcers in the present series is similar to that demonstrated in leprosy patients (Languillon, 1964) the common denominator being the insensitive feet.

In the treatment of diabetic neuropathic foot ulcers two different principles are currently used. In some centres healing is obtained by application of rigid casts (Brand, 1979; Wagner, 1979; Burden et al., 1983; Jernberger, 1986) and maintained by various shoe systems. In other centres the external pressure is relieved with the aid of soft materials which are adjusted during the healing phase (Holstein et al., 1976; Faris, 1982). The authors prefer the latter principle because pressure necrosis from rigid casts is avoided and the adjusted shoes and insoles provide a proper prophylaxis against new ulcers—with a shoe cosmesis acceptable to most patients.

The literature on diabetic foot lesions is growing, but it is at present difficult to compare the results from one centre to another and an

untreated control group is not available. However, the compensation for neuropathy and the aggressive treatment of invasive infection give healing rates, which are comparable to those obtained in ulceration and gangrene in non-diabetic patients (Holstein and Lassen, 1980), and these results are confirmed in the present study.

This series demonstrates that soft insoles and proper footwear are adequate in preventing major amputation in neuropathic lesions, but these measures were not adequate in preventing new ulcers, which occurred in the severely deformed feet possibly in connection with neglect of prophylaxis. It is possible that a wider use of surgical correction of the deformities and more extensive use of orthotic measures are justified in such cases.

Major amputations were almost exclusively performed in severe ischaemia where the patients were not suitable for vascular reconstruction. However, the femoro-crural in situ by-pass (Leather et al., 1981), had not yet been introduced in Denmark during the period studied. Today a number of diabetic patients with ischaemic ulcers are effectively treated by this procedure.

The annual number of major amputations in diabetics in Denmark is slowly but steadily decreasing. From 560 in 1980 to 300 in 1986 (Ebskov, 1988). This is probably due to an increasing interest in diabetic foot problems, economic support for regular foot control and reinforcement of education at all levels. Major attention is paid to the prophylaxis since "if an ulcer develops . . . the preventative measures have failed (Faris, 1982)".

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