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Peak foot pressures influence the healing time of diabetic foot ulcers treated with total contact casts

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Abstract—The purpose of this article is to describe the progression of ulcer healing using total contact casts (TCC) and to examine explanatory variables that may influence ulcer healing. We prospectively studied 25 diabetics with grade I (Meggitt-Wagner) plantar ulcers. All subjects received weekly contact cast changes with wound assessment. Following healing, all subjects were fitted with prescription shoe gear. Mean duration of casting until healing was 38.8±21.3 days. Individuals with pressures over 99 N/cm^2 took longer to heal (33.1±13.0 vs. 53.4±31.4 days, P=0.05) and had longer ulcer duration prior to treatment (52.7±37.2 vs. 180.7±145.0 days, P=0.02). Subjects with wound size greater than 8 cm^2 took longer to heal (50.2±26.2 vs. 29.9±10.6 days, P=0.02). We conclude that subjects with high plantar pressures and wounds greater than 8 cm^2 took significantly longer to heal when uniformly treated with TCC.

Key words: *diabetes mellitus, foot, neuropathy, pressure, ulceration.*

INTRODUCTION

Neuropathic foot ulcerations are one of the most common precursors to lower limb amputation among persons with diabetes; therefore, effective management of these wounds should have a substantial impact on amputation prevention. Most ulcers result from constant or repetitive pressure applied to the foot while walking. Persons that are insensate due to diabetic peripheral neuropathy lack the innate sensory feedback (e.g., pain) necessary to protect the foot from skin breakdown (1). In order for a neuropathic ulcer to heal, repetitive pressure must be reduced or eliminated by external mechanisms or devices.

Total contact casts (TCC) are one of the most frequently cited methods in the medical literature to facilitate healing of neuropathic ulcers (2–8). By molding casting material to the foot and leg with minimal padding, weight-bearing forces are spread out along the entire surface of contact, thus substantially reducing vertical force per unit area (9). Additionally, the nearly complete elimination of motion in TCC is thought to substantially reduce plantar shear forces.

While there have been numerous studies espousing the efficacy of TCC in the treatment of diabetic foot wounds, we have been unable to identify any reports in the medical literature that prospectively incorporate plantar pressure measurements, sensory and assessments, degree of glucose control, and wound surface area prior to and during total contact casting. In addition, previous studies that describe TCC outcomes often fail to identify the healing rates of the entire cohort and neglect to provide an explanation for individuals that had protracted courses of therapy, adverse events, or never healed their wounds. In reporting the results of only a portion of the study population, data and conclusions from some of these reports may have been biased. The purpose of this study is to describe the progression of ulcer healing using

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Table 1.
Study group characteristics.

n=	Male:Female	Age (±SD) years	Type I: Type II Diabetes Mellitus	Mean Duration of DM (±SD) years
25	17:8	52.4±11.6	4:21	13.8±7.8

TCC and to examine explanatory variables that may influence ulcer healing.

METHODS

Twenty-five consecutive diabetic subjects with neuropathic foot ulceration were selected for study. All subjects were previously treated by their primary care physicians prior to referral. The diagnosis of diabetes mellitus was confirmed using World Health Organization criteria (10). Descriptive data for these subjects are listed in **Table 1.** All ulcers were classified using the Meggitt-Wagner grading system. All wounds were grade I in depth; that is, they exhibited full thickness skin loss, but did not involve tendon, capsule, or bone (11,12). Location of ulcers is shown in **Figure 1.** All subjects had loss of plantar sensory protective threshold, using the Semmes-Weinstein monofilament wire according to the method and criteria described by Birke (13) and vibration perception threshold (VPT) using the technique described by Young and colleagues (14,15). Vascular status was evaluated by measuring systolic ankle-brachial, and toe-brachial, pressure indices (16,17). We used the EMED® electronic measurement system (Novel gmbh Electronics, Minneapolis, MN) to record mean peak plantar foot pressures (18). The system measures pressures at a resolution of approximately 4 pixels per cm^2 over the entire surface of contact. The location and value of the largest (peak) focal pressure was averaged from three recorded midgait steps on the EMED pressure platform.

All subjects were treated with serial TCC until their ulcers healed. Casts were applied using the technique described by Kominsky (3) and were changed at weekly intervals, at which time the wound was evaluated and debrided as necessary. Wound area was calculated by multiplying the length by the width of ulcer tracings taken on acetate sheeting. Following ulcer healing, subjects were given prescription shoe gear fashioned by a certified pedorthist. Subjects were excluded if they had acute Charcot's arthropathy or a soft tissue or bone infection. Wounds were classified as infected if they displayed frank purulence and/or two or more of the following local signs: warmth, erythema, lymphangiitis, lymphadenopathy, edema, pain, and loss of function (19).

Pearson's test for correlation was used to compare ulcer duration with duration of treatment to healing. The Mann-Whitney U test was used to compare both duration of ulcer prior to treatment and duration of contact casting with maximum pressure, as well as differences between sex and long-term glucose control. For the purposes of comparison, we used 99 N/cm^2 as a cutoff point to create two dichotomous variables for

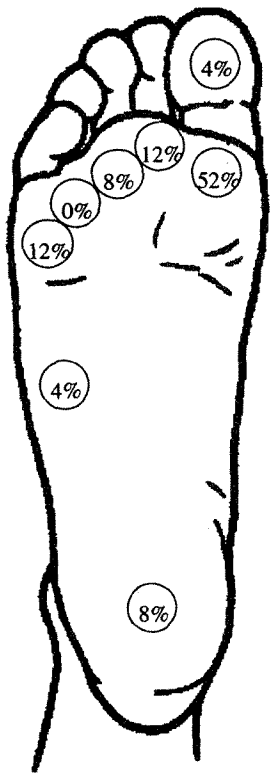

Figure 1.
Wound location.

maximum pressure and 9.0 percent as a cutoff point forglycosylated hemoglobin (20). To simultaneously control for the potential confounding variables of sex, ulcerduration, maximum plantar pressure, glycosylated hemoglobin, and wound surface area, and their effect on duration of treatment to healing, a stepwise linear regression analysis was used to model potential interactions (21).

RESULTS

The average duration of casting until healing was 38.8±21.3 days. These data and vascular and neurologic findings are summarized in **Table 2.** There was a significant association between duration of ulceration prior to treatment and duration of contact casting until ulcer healing (P=0.008). Subjects with maximum pressures over 99 N/cm^2 had a significantly longer ulcer duration prior to treatment (52.7±37.2 vs. 180.7±145.0 days, P=0.02), and took longer to heal (33.1±13.0 vs. 53.4±31.4 days, P=0.05). In every subject studied, the site of maximum plantar pressure correlated directly with the site of the neuropathic ulcer (P<0.001). Subjects with wound size greater than 8 cm^2 took longer to heal (50.2±26.2 vs. 29.9±10.6 days, P=0.02). Men took longer to heal than women (43.8±22.9 vs. 28.13±12.9 days, P=0.03), but did not have significantly higher maximum plantar pressures or larger wounds than women. Twenty-eight percent of subjects had noncompressible ankle vessels. There was no correlation, however, between noncompressible ankle vessels and prolonged healing time. Additionally, there was not a significant association between noncompressible ankle vessels and the toe-brachial index.

Mean glycosylated hemoglobin for all subjects was 9.3±2.0 percent. Subjects with glycosylated hemoglobin above 9.0 percent (n=12) had larger ulcers on initial presentation (9.2±3.9 cm^2 vs. 6.3±3.7 cm^2, P=0.04) and took longer to heal (43.5±11.2 vs. 34.5±27.4 days, P=0.02).

Using a stepwise linear regression model, we determined the relations between wound healing and the potentially confounding variables of sex, wound surface area, maximum plantar pressure, and glycosylated hemoglobin. There continued to be a strong association between time of wound healing and plantar pressure (CI=0.1 to 0.91, P<0.03) and wound surface area (CI=1.6 to 4.8, P<0.0003, r^2=0.6). Three variables that were statistically significant in the univariate analysis (sex, ulcer duration, and glycosylated hemoglobin) did not meet the criteria for inclusion in the logistic regression model after the correction for confounding.

DISCUSSION

The results of this study suggest that longer healing times are associated with high peak plantar pressures at the site of the ulceration and large wounds. Outcomes in this study were uniformly good in a population that consisted primarily of inadequately controlled diabetics with severe peripheral neuropathy, good vascular perfusion, and superficial forefoot wounds on the sole of the foot. Many of the previous works in this area do not consistently provide quantitative data on vascular perfusion, peripheral neuropathy, hyperglycemia, and plantar foot pressures. The mean time to complete healing of ulcers in our study (39 days) was very similar to the times reported in other TCC studies (35–44 days), even though most of these descriptive series only provide data for 71–90 percent of their subjects (7,8,22–24). Poor results in previous studies may be at least partially explained by the presence of peripheral arterial occlusive disease, more severe grades of diabetic ulcerations, severe hyperglycemia, or subjects with very high focal areas of pressure.

Table 2.
Ulcer characteristics.

Vibration Perception Threshold	Toe-Brachial Pressure Index*	Mean Surface Area (cm^2)	Mean Maximum Plantar Pressure (N/cm^2)	Mean Ulcer Duration (days)	Mean Time to Healing in Total Contact Cast (days)
45.8±5.2	0.68±0.14	7.7±4.0	92.7±14.3	88.5±98.4	38.8±21.3

*Mean ankle brachial index was 0.94±0.18. However, 28% of subjects did not have calculable values due to non-compressible ankle vessels.

There is a relative paucity of work in the literature that directly compares the clinical efficacy of different methods to off-load neuropathic ulcers. Mueller (23) and coworkers prospectively compared TCC and "traditional dressing treatments," which consisted of therapeutic shoe gear with insoles and walker- or crutch-assisted gait. The mean healing time was over 35 percent faster in the group treated with TCC (42 days vs. traditional treatment 65 days). And, as in our study, there were no infections or amputations in the TCC group in Mueller's study. In other descriptive case series of diabetic foot ulcerations treated in "half-shoes" and "bivalved ankle-foot orthoses," healing times were 70 days and 300 days, respectively (25,26). Laboratory pressure studies also indicate that casts are superior to therapeutic footwear (9) and removable plaster walking splints at reducing foot pressures (27) and may help explain why casts have been more popular than other modalities in diabetic foot specialty clinics.

In the presence of neuropathy, there are three main factors that influence the development and healing of foot ulcers in persons with diabetes: foot deformity, limited joint mobility, and repetitive stress. Both limited joint mobility and frank deformity have been shown to increase plantar pressures (28–30). Duckworth (31) and Boulton (32) have suggested that there is a threshold pressure at which diabetics are likely to ulcerate in the presence of peripheral neuropathy. The people in our study demonstrated a large variation of maximal foot pressures ranging from 68 to 125 N/cm^2, corresponding in each case to the site of ulceration. While our data suggest that subjects with elevated foot pressures (in the range described by Duckworth and Boulton) took longer to heal, it appears that ulceration can clearly develop in areas with relatively "normal" pressure.

The tissue damage that is a precursor for neuropathic ulcer development can involve areas of moderate to high pressures exposed to repetitive trauma from each step a person takes during the course of normal activity. Individuals with normal pressures probably are exposed to more cycles of repetitive trauma than those with very high pressures. Unfortunately, we were unable to precisely measure activity in this population. Casts probably enhance ulcer healing by decreasing foot pressures across the entire foot as well as altering cadence, stride length, and overall activity. Essentially both sides of the equation can be decreased: the magnitude of pressure and the number of repetitions.

The association between degree of glucose control and incidence of numerous diabetic complications has been well demonstrated (33). These include neuropathy (34,35), impaired leukocytic phagocytosis (36–41), and soft tissue crosslinking through production of advanced glycosylation end products (42). All of these factors may delay wound healing. Additionally, other investigators have associated poor glucose control with ulceration (43) and amputation (20). In our study, poor glucose control was strongly associated with prolonged wound healing in univariate analysis, but failed to show significance in the multivariate linear regression model. Clearly, this association calls for further investigation.

Appropriate diabetic foot care in general, and wound care specifically, is not the purview of a single medical specialty. The etiology and treatment of diabetic wounds is a multifactorial problem. This underscores the importance of a team approach toward effecting an acceptable long-term result by obtaining optimal glycemic control, off-loading the foot to heal the ulcer, obtaining adequate vascular perfusion, and educating the subject so he or she can help manage the disease and identify early warning signs of lower limb diabetes complications.

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