PRESSURE-RELATED HYPEREMIA IN HEELS OF PERSONS WITH AND WITHOUT DIABETES MELLITUS

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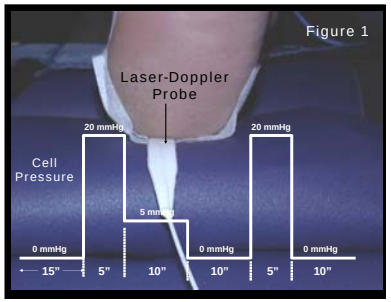
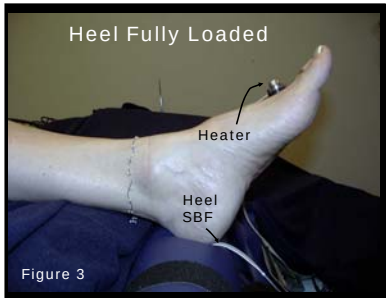
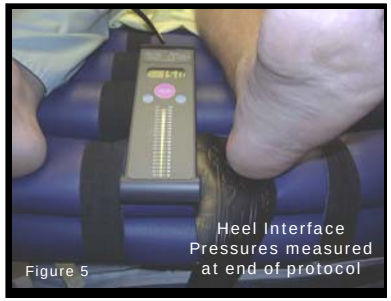
Background

Vulnerability of the heel to ulceration in bed-bound persons is related to pressure-induced blood flow decreases. Periodic pressure reduction is a clinical strategy to help prevent ulcers by allowing blood flow repayment during intervals of off-loading. The magnitude and duration of the resulting hyperemia is related to the duration and magnitude of the prior interval of ischemia. Previous work has shown that if healthy individuals lie supine with their heels in contact with a controllable support surface that allows the heel to be either partially or completely off-loaded, hyperemia features depend on the pressure-relief magnitude during offloading[1-2]. Similar affects can be shown with graded localized pressure[3-4]. In the case of supine lying, if off-loading is characterized by the magnitude of interface pressure between heel and support surface during pressure-relief, an inverse relationship between hyperemia and relief pressure is demonstrated, with the greatest hyperemia occurring with complete off-loading (zero interface pressure). If relief pressure is greater than zero, some blunting of the hyperemic response is observed. But, in healthy persons, whether off-loading is partial or complete, average heel blood flow (over a complete load-offload cycle) results in a net heel blood flow that exceeds the apparent flow deficit during the loading interval[2]. This finding is consistent with the concept that in healthy persons, hyperemia, during pressure-relief, more than compensates for flow deficits during pressurization. But, as these previous results strictly apply to normal physiological hyperemic response capacity, effects that a reduced hyperemic reserve may have are unclear. Herein we report on preliminary observations regarding the possible impact of the diabetic condition on the general features of heel loading and partial and complete pressure-relief hyperemia.







Assessment Parameters and Data Analysis

Heel hyperemic responses were assessed by two measures. During the first five minutes after pressure relief to either 5 mmHg or 0 mmHg, the area under the SBF curve was calculated and the ratio of this area to the corresponding five minute pre-load baseline was determined (***Figure 6***). This parameter is denoted as A_R. In addition, the peak SBF during the first five minutes of pressure relief was determined and the ratio of it, to the five-minute average SBF during baseline, was calculated. This parameter is denoted as Q_R. For the heat response on the foot dorsum, the peak SBF that occurred during a four minute heating cycle was determined and a ratio of its value to a four-minute average SBF prior to heating was determined. This parameter is denoted as H_R. Statistical analyses to test for overall differences of AR and QR within and between groups were done with a general linear model (GLM) for repeated measures. Follow-up tests of SBF responses (A_R and Q_R) were done by analysis of variance. A p-value < 0.05 was considered statistically significant.

Methods

Subjects: Persons with diabetes mellitus (DM, n=13), and without DM (NO-DM, n=15) participated. For (DM vs. NO-DM) data (mean ± sem) were as follows. ABI: 1.14± 0.04 vs 1.13±0.02; Height: 67.2±0.9 vs 66.9±1.1 inches Weight: 205.2±17.4 vs 156±9.1 lbs, p<0.05; Age: 65±3 vs 55±3 yrs, p<0.05.; BP: systolic, 134.2±5.8 vs 127.7±4.8; diastolic (75.8±2.6 vs 72.4±2.4; mean, 95.2±3.0 vs 92.1±2.5 mmHg. Duration of DM was 7.5±1.5 yrs. Five subjects were on insulin; remainder on oral medication for type II DM. HbA1C for the group was 8.5 ±2.2 and their morning blood sugar level averaged 144±33 mg/dl.

Protocol: Subjects lay on a support surface with their left heel positioned on the end cell of a support surface (***Figure 1***). Pressure in this cell was under computer control, and could be made to vary between 20 mmHg and a variable lower limit of either 5 or 0 mmHg. The test sequence was initiated after supine rest of 15 minutes during which the heel was not loaded (0 mmHg, ***Figure 2***). Tests were conducted in a room with a well-controlled ambient temperature. Room temperature was 24.1±0.4 °C at the start and 24.3±0.4 °C at the end.

Blood Perfusion: Heel skin blood perfusion (SBF) was monitored with a laser-Doppler probe on the heel (***Figure 3***). The probe was at the site of contact of the heel with the support surface. A second probe, inserted in a heater, was on the foot dorsum. Heater temperature could be rapidly raised to 45° C while monitoring local SBF responses. This heat response was used to provide an index of the relative hyperemic potential for each subject. Skin temperature at non-heated sites on the foot dorsum and heel were measured with an infrared thermometer prior to the experiment start and at the end of the experiment. Skin temperatures did not differ between groups nor were there significant changes at the skin sites from start to finish. For dorsum and heel skin, temperatures were 33.1±0.3 °C and 32.3±0.4 °C respectively. Example data are shown in ***Figure 4***.

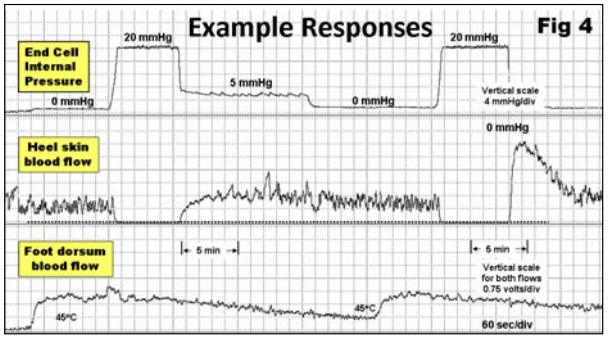
Interface Pressure: At the end of each experimental sequence, heel interface pressures (IP) were measured with a pressure sensor that was placed between the heel and the supporting cell (***Figure 5***). The cell was pressurized to the levels corresponding to those used during the test-sequence and six measurements of IP were made at each cell pressure. Averages of the six measurements were used to report interface pressures.

Results

	A_R Relief Pressure (mmHg)		Q_R Relief Pressure (mmHg)		
	5 mmHg	0 mmHg	5 mmHg	0 mmHg	H_R
NO-DM	2.6±1.4	4.8±2.8*	5.5±3.5	9.4±6.2*	37.8±16.5
DM	2.4±1.1	2.7±1.6[a]	4.3±3.2	5.4±3.5[a]	14.5±11.0[a]

Values are mean ± sd. * p<0.05 compared to 5 mmHg, [a] = p<0.05 for DM vs. NO-DM.

For A_R and Q_R there was a greater response if the heel was relieved to 0 mmHg as compared to 5 mmHg (p=0.001), There was also an interaction between group and pressure-relief magnitude for A_R and Q_R (p<0.05). As shown in the table, relief to 0 mmHg, compared to relief to 5 mmHg, showed a greater A_R and Q_R *only* in the NO-DM group. Further, for the DM group, full pressure relief was associated with significantly reduced A_R and Q_R compared to NO-DM. For H_R, which characterizes the SBF heat response on foot dorsum, a significantly reduced value was observed in DMs (p<0.05).



Discussion and Conclusions

In both DM and NO-DM persons, partial heel off-loading results in a reduced hyperemic response as compared to complete off-loading. But, in persons with DM there is a significantly reduced hyperemia for complete off-loading. One explanation of these results is that a diabetes-related reduced microvascular vasodilatory capacity is not exceeded during the partial relief, but is exceeded during complete pressure relief. The presence of a lesser maximum hyperemic capacity is suggested by the reduced heat response findings herein, by specific assessments of foot skin responses[5] and by numerous other studies[6-11]. Accordingly, differences in hyperemic response become unmasked only when maximum hyperemia can be established, which is only during complete off-loading.

For both groups, hyperemia, even during partial off-loading, appears to be adequate to compensate for the prior interval of ischemia. This follows since a flow area ratio (AR) of 2.0 would just be sufficient, theoretically, to compensate for the flow ischemic interval. What then accounts for the 'overcompensation" seen during complete off-loading? It has been suggested that hyperemic responses to heel loading and off-loading do not just depend on the ischemia associated with the pressure-induced flow reduction[12] . It may be that the "excess" flow serves additional physiological needs. If true, this implies that the larger hyperemia present with full pressure-relief, is in fact a needed flow response to compensate for sustained intervals of loading and off-loading. By extension, this suggests that a reduced hyperemia during complete off-loading as found in the DM group, may be problematic if widely present in the diabetic population. Further work is needed to investigate and clarify this concept.

References

1.Mayrovitz HN, Sims N. Effects of different cyclic pressurization and relief patterns on heel skin blood perfusion. Adv Skin Wound Care 2002; 15:158-164.
2.Mayrovitz HN, Sims N, Taylor MC, Dribin L. Effects of support surface relief pressures on heel skin blood perfusion. Adv Skin ound Care 2003; 16:141-145
3.Mayrovitz HN, Macdonald J, Smith JR. Blood perfusion hyperaemia in response to graded loading of human heels assessed by laser-Doppler imaging. Clin Physiol 1999; 19:351-359.
4.Mayrovitz HN, Smith J. Heel-skin microvascular blood perfusion responses to sustained pressure loading and unloading. Microcirculation 1998; 5:227-233.
5.Mayrovitz HN, Larsen PB. Functional microcirculatory impairment: a possible source of reduced skin oxygen tension in human diabetes mellitus. Microvasc Res 1996; 52:115-126.
6.Caballero AE, Arora S, Saouaf R, Lim SC, Smakowski P, Park JY, King GL, LoGerfo FW, Horton ES, Veves A. Microvascular and macrovascular reactivity is reduced in subjects at risk for type 2 diabetes. Diabetes 1999; 48:1856-1862.
7.Jaap AJ, Pym CA, Seamark C, Shore AC, Tooke JE. Microvascular function in type 2 (non-insulin-dependent) diabetes: improved vasodilation after one year of good glycaemic control. Diabet Med 1995; 12:1086-1091.
8.Meyer MF, Schatz H. Influence of metabolic control and duration of disease on microvascular dysfunction in diabetes assessed by laser Doppler anemometry. Exp Clin Endocrinol Diabetes 1998; 106:395-403.
9.Shore AC, Price KJ, Sandeman DD, Green EM, Tripp JH, Tooke JE. Impaired microvascular hyperaemic response in children with diabetes mellitus. Diabet Med 1991; 8:619-623.
10.Walmsley D, Wiles PG. Myogenic microvascular responses are impaired in long-duration type 1 diabetes. Diabet Med 1990; 7:222-227.
11.Sprigle S, Linden M, Riordan B. Characterizing reactive hyperemia via tissue reflectance spectroscopy in response to an ischemic load across gender, age, skin pigmentation and diabetes. Med Eng Phys 2002; 24:651-661.
12.Mayrovitz HN, Sims N, Dribin L. Heel skin hyperaemia: Direct compression vs. vascular occlusion. Clin Physiol Funct Imaging 2003;23:354-359.