Effects of Support Surface Relief Pressures on Heel Skin Blood Flow in Persons with and without Diabetes Mellitus

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ABSTRACT

OBJECTIVE: To investigate the effects of pressure relief magnitude on heel blood hyperemia in persons with and without diabetes mellitus.

DESIGN: Study participants (1 group of persons with diabetes and 1 group without diabetes) lay on a support surface for 70 minutes with 1 heel on an end cell of a support surface. Cell pressure was computer controlled to be 20 mm Hg during support and 5 or 0 mm Hg during relief. Heel skin blood perfusion was monitored by laser Doppler on the heel and foot dorsum. Heel hyperemia was determined as ratios of skin blood perfusion areas during hyperemia to preloading (A_R) and peak hyperemia to mean skin blood perfusion during preload (Q_R).

SUBJECTS: 13 persons with diabetes mellitus (6 females, 7 males; age 65.2 ± 3.0 years) and no known diabetes-related complications, and 15 persons without diabetes mellitus (7 females, 8 males; age 54.7 ± 3.1 years)

SETTING: University research center

RESULTS: For the nondiabetes mellitus group, hyperemia was significantly greater with complete pressure-relief compared with partial relief ($P < .001$). In contrast, the diabetes mellitus group showed no significantly increased response to full relief, and the hyperemia achieved during full relief, measured by A_R and Q_R, was significantly less than with the nondiabetes mellitus group.

CONCLUSIONS: These results suggest that a diabetes-related reduced microvascular vasodilatory capacity is not exceeded during partial pressure relief, but is exceeded during complete pressure relief. Accordingly, differences in hyperemic response would become unmasked only when maximum hyperemia could be established during complete heel off-loading. This would suggest that a diminished hyperemia during complete off-loading, as found in the present diabetes mellitus group, may be problematic if widely present in the diabetic (or possibly older) population, under conditions in which heel loading occurs for sustained intervals.

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In bed-bound persons, the heel's vulnerability to ulceration is related to direct pressure-induced decreases in blood flow. Periodic pressure reduction is a clinical strategy to help prevent ulcers by allowing a blood flow repayment hyperemia that has a magnitude and duration most likely related to the duration of prior intervals of ischemia. Research has shown that when healthy individuals lie supine with their heels in contact with a support surface, the hyperemia that accompanies off-loading depends on whether the off-loading is partial or complete.[1,2] Similar effects can be demonstrated with graded localized pressure procedures.[3,4]

When off-loading is characterized by the magnitude of interface pressure between heel and surface during pressure relief of a supine patient, an inverse relationship between hyperemia and relief pressure exists, with the greatest amount of hyperemia occurring during complete off-loading with a 0 mm Hg interface pressure.[2] If relief pressure is greater than zero, some blunting of the hyperemic response is observed. In

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either partially or fully off-loaded healthy persons, however, the average heel blood flow over a full cycle of loading and off-loading results in a net heel blood flow that exceeds the apparent flow deficit during the loading interval.[2]

In the healthy persons studied, the hyperemia seen during pressure relief tends to compensate for flow deficits during pressurization, although the mechanism is unclear. Because these results apply only if the person is capable of a normal physiologic hyperemic response, a diminished hyperemic reserve might alter this characteristic. Therefore, the present study reports on preliminary observations regarding the possible impact of diabetes on the general features of heel loading and partial and complete pressure-relief hyperemia.

The tentative hypothesis was that the hyperemic response would be diminished in the presence of a diminished vascular reserve. Postischemic hyperemia is characterized by both a maximum flow and an integrated flow repayment that depends on the form and duration of the response. Therefore, the present study examines both of these aspects by considering peak flow and flow-time responses as parameters.

METHODS

Subjects

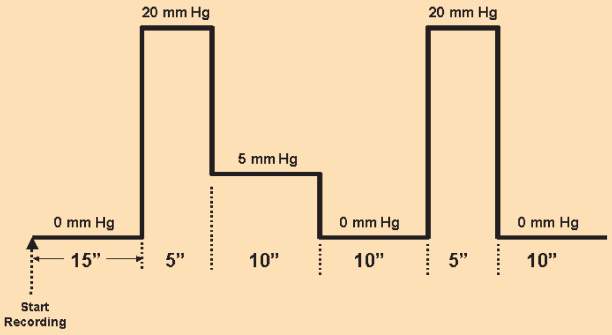
Thirteen persons (6 female and 7 male) with diabetes mellitus (DM) and no known DM-related complications and 15 persons (7 female, 8 male) without DM participated in the study. The university's review board approved the protocol, and informed consent was obtained from all participants.

These groups were subsequently designated as DM and nonDM and compared in that order. The DM group was recruited from inside and outside the university using methods that included word-of-mouth, announcements posted at the university and its Web site, and solicitations to local endocrinologists and diabetes educators inside and outside the institution. The nonDM group was recruited from university staff. All subjects were nonsmokers with no demonstrable lower extremity vascular disease, as judged by history and by pretest ankle-brachial pressure indexes (ABI). An ABI value of <1.0 was used as a threshold criterion for exclusion on the basis of possible peripheral arterial disease.[5] For the DM vs. the nonDM group, the ABI values were 1.14 ± 0.04 vs. 1.13 ± 0.02, respectively.

Average heights of both groups were similar (67.2 ± 0.9 for the DM group vs. 66.9 ± 1.1 inches for the nonDM group). However the DM group differed ($P < .05$) from the nonDM group with respect to weight (205.2 ± 17.4 vs. 156 ± 9.1 pounds) and age (65.2 ± 3.0 vs. 54.7 ± 3.1 years). The age difference resulted from an inability to recruit healthy nonDM subjects in the upper age levels. The possible impact of this age difference is addressed in the discussion.

Figure 1.
TEST SEQUENCE

The 55-minute test sequence began after the subject had been lying supine for 15 minutes, with the heel in position at the support surface end cell, but with the cell set at 0 mm Hg (fully deflated). Internal cell pressures were then altered in magnitude and duration, as shown in the figure.



Groups did not differ significantly with respect to blood pressure, but the DM group tended to have slightly higher systolic (134.2 mm Hg ± 5.8 vs. 127.7 mm Hg ± 4.8), diastolic (75.8 mm Hg ± 2.6 vs. 72.4 mm Hg ± 2.4) and mean (95.2 mm Hg ± 3.0 vs. 92.1 mm Hg ± 2.5) blood pressures. The average duration of diabetes was 7.5 ± 1.5 years, with 5 subjects on insulin and the remainder on oral medication for type 2 diabetes. The average glycosated hemoglobin (HbA_{1C}) for the group was 8.5 ± 2.2, and morning blood glucose levels averaged 144 ± 33 mg/dL.

Protocol and support patterns

Subjects were asked to lie on a support surface, positioning their left heel on the end cell of a support surface. The surface was a standard air overlay modified so that pressure in the end supporting cell was under computer control and could be made to vary between an upper limit of 20 mm Hg and a variable lower limit of either 5 or 0 mm Hg (Figure 1). The test sequence shown in Figure 1 was initiated after supine rest of 15 minutes, in which time the heel was not loaded (0 mm Hg).

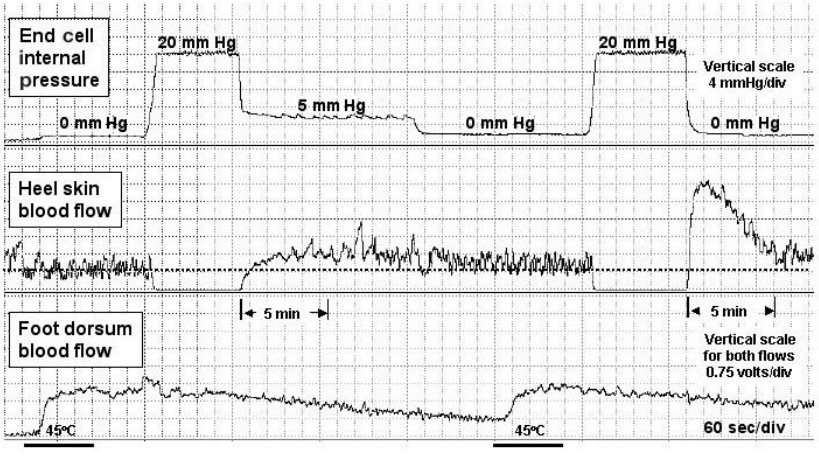
Tests were conducted in a room with a well-controlled ambient temperature. During the experiments, room temperature varied from 24.1 ± 0.4° C at the start to 24.3 ± 0.4° C at the end.

Blood perfusion

Heel skin blood perfusion (SBP) was monitored with a laser Doppler probe affixed to the heel with tape and connected to a

Figure 2.
EXAMPLE RESPONSES AND PARAMETERS

The top panel shows the actual pressures measured sequentially. The middle panel shows the heel skin blood flow (SBP) during each phase of the loading and off-loading sequence. During loading, SBP falls to zero. During off-loading, the hyperemic response depends on whether partially off-loaded (5 mm Hg) or fully off-loaded (0 mm Hg). The third panel shows the foot dorsum SBP during 2 sequential heating intervals, which are denoted by the horizontal bars. The heel and dorsum flows are shown at the same sensitivity as recorded. The differences in their resting magnitudes and spontaneous variations are related to the normal variability in perfusion at different skin sites. This is one reason that responses are usually assessed relative to the prevailing baseline perfusions.



perfusion monitor (model BPM[2]; Vasamedics, Inc, St Paul, MN). The probe (P-440 Soflex; Vasamedics) is flat and has a large surface contact area with the skin. The probe was positioned at the site of heel contact with the support surface. SBP was continuously monitored throughout the experimental sequence.

In laser Doppler monitoring, a low-intensity laser light signal is transmitted into the skin to a depth of 1 to 2 mm; the reflected light is used to measure local blood perfusion. The Doppler-shifted signal contains information about the speed and number density of moving red blood cells in a tissue region to a depth of 1 to 2 mm. Speed and number density information is processed to yield a parameter—perfusion—that is proportional to blood flow.

A second probe was placed on the foot dorsum, proximal to the union of the great and second toe, to monitor foot SBP using a second perfusion monitor of the same type. The pencil-type probe (Model 8F; Vasamedics) was inserted through a 19-mm diameter, circular heating element that was in contact with the foot skin. The temperature of the heating element could be rapidly raised to 45° C, while simultaneously monitoring local SBP responses. This heat response was used to provide an index of the relative hyperemic potential for each subject. Because local heating to this temperature results in a maximally vasodilated condition, the maximum flow achieved is an index of an individual's vascular reserve.[6]

Laser Doppler data were acquired using a time-constant setting of 1 second and, after analog to digital conversion, was recorded at fixed standard gain on a dedicated computer. Laser Doppler perfusion may be expressed in perfusion units or other linearly related quantities. When reporting absolute perfusion values (Figure 2), perfusion was expressed in volts with 1 volt = 100 perfusion units. At the end of the procedure, biologic zeros of both laser Doppler probes were determined using an ankle cuff inflated to 40 mm Hg above systolic blood pressure for 2 minutes. The biologic zero value was subtracted from laser Doppler raw values.[7]

Skin temperature at nonheated sites on the foot dorsum and heel were measured with an infrared thermometer prior to and at the end of the experiment. Skin temperatures did not differ between groups, and no significant changes were noted at the skin sites from start to finish. For room, dorsum, and heel skin, the overall temperatures were 24.2 ± 0.3° C, 33.1 ± 0.3° C, and 32.3 ± 0.4° C, respectively.

Interface pressure

At the end of each experimental sequence, heel interface pressures (IP) were measured with a pressure sensor placed between the heel and the supporting cell. The cell was pressurized to the levels corresponding to those used during the test sequence; 6 measurements of IP were made at each cell pressure. Averages of the 6 measurements were used to report interface pressures.

Assessment parameters and data analysis

Heel hyperemic responses were assessed using 2 measures. During the first 5 minutes after pressure relief to either 5 mm Hg or 0 mm Hg, the area under the SBP curve was calculated and the ratio of this area to the corresponding 5-minute preload baseline was determined (Figure 2). This parameter is des-

Table 1.
MAIN RESULTS

	A_R		Q_R		H_R
	Relief Pressure		Relief Pressure		
	5 mm Hg	0 mm Hg	5 mm Hg	0 mm Hg	
NonDM	2.61 ± 1.42	4.82 ± 2.82*	5.53 ± 3.48	9.42 ± 6.21*	37.8 ±16.5
DM	2.39 ± 1.08	2.78 ± 1.57[a]	4.31 ± 3.16	5.43 ± 3.49[a]	14.5 ± 11.0[a]

DM = diabetes mellitus
NonDM = nondiabetes mellitus
Values are mean ± SD
* $P < .05$ compared with 5 mm Hg relief pressure
[a] = $P < .05$ for DM vs. NonDM
A_R is the 5-minute hyperemic area divided by the 5-minute preload area.
Q_R is the maximum hyperemia divided by the preload average SBP.
H_R is the peak SBP during a 4-minute heat interval divided by the average preheat SBP.

ignated as A_R. In addition, the peak SBP during the first 5 minutes of pressure relief was determined, and the ratio of it to the 5-minute average SBP during baseline was calculated. This parameter is designated as Q_R.

For the heat response on the foot dorsum, the peak SBP that occurred during a 4-minute heating cycle was determined and a ratio of its value to a 4-minute average SBP prior to heating was determined. This parameter is designated as H_R. Statistical analyses to test for overall differences of A_R and Q_R within and between groups were done with a general linear model for repeated measures (SPSS, version 6.1). Follow-up tests of SBP responses (A_R and Q_R) were performed using analysis of variance. In all cases, a P value < .05 was considered to be statistically significant. Values in text are reported as mean ± SD unless otherwise specified.

RESULTS

Interface pressures

With the end cell internal pressure set at 20 mm Hg and 5 mm Hg, measured IP was 140.7 ± 34.1 mm Hg and 50.0 ± 19.8 mm Hg, respectively. This variation among subjects is consistent with previous results[2] and reflects the dependence of IP on multiple factors, such as foot position, body habitus, and heel shape. As a group, these IP levels indicate that the maximum support cell pressure (20 mm Hg) corresponds to a value greater than the average systolic pressure of both groups, whereas a cell pressure of 5 mm Hg corresponds to a value significantly less than average diastolic pressure of both groups.

Hyperemic responses following pressure relief

Overall hyperemic responses, evaluated with the general linear model, showed that the hyperemic area ratio, A_R, was significantly greater when the heel was relieved to 0 mm Hg as compared with 5 mm Hg ($P = .001$). This difference, however, was associated with a significant interaction between group and pressure-relief magnitude ($P = .017$). As summarized in Table 1, relief to 0 mm Hg, as compared with relief to 5 mm Hg, was associated with a significantly greater A_R only in the nonDM group. For the DM group, the A_R value associated with heel pressure relief to 0 mm Hg, was significantly less than for the nonDM group ($P = .029$).

For hyperemic responses characterized by Q_R, an overall significantly greater response was noted when the heel was relieved to 0 mm Hg, as compared with 5 mm Hg ($P = .001$). This difference was also associated with a significant interaction ($P = .029$) between group and pressure-relief magnitude. As summarized in Table 1, relief to 0 mm Hg, as compared with relief to 5 mm Hg, was associated with a significantly greater Q_R only in the nonDM group. The Q_R value associated with the heel release to 0 mm Hg was significantly less for the DM group than for the nonDM group ($P = .049$). For H_R, which characterizes the SBP heat response on the foot dorsum, a significantly reduced value was observed in the DM group, as shown in the last column of Table 1.

DISCUSSION

The present findings show that partial heel off-loading causes a blunted hyperemic response when compared with complete off-loading in both the DM and nonDM groups. One possible explanation for this difference is that only during complete off-loading does the diabetes-related reduced microvascular vasodilatory capacity become unmasked. Accordingly, differences in hyperemic response would become evident only when maximum hyperemia could be established during complete off-loading.

The presence of a lesser maximum hyperemic capacity in

persons with diabetes is suggested by the reduced blood flow response to heat found in the present study, as well as by other specific assessments of foot skin responses[6] and by numerous other studies. These include reduced vasodilatory responses to iontophoretically administered acetylcholine and sodium nitroprusside,[8] to local heating,[9-11] and after postischemic occlusion.[12-14]

The older age of participants in the DM group may account for some of the reduced hyperemic response. The extent to which age modifies maximal hyperemic response is not fully resolved. It has been reported that over age 40, endothelial-mediated forearm reactive hyperemia declines at a rate of 0.21% per year.[15] But an age-related decline in endothelial-independent responses, such as declines that are not predominantly mediated by endothelial function, has not been observed.[15] Comparisons between pressure-induced hyperemia in the forearm of 65-year-old subjects vs. 25-year-old subjects indicates a reduced reactive hyperemia.[16] Other research suggests that neither age nor diabetes per se are major factors in pressure-induced reactive hyperemia.[17] At the opposite extreme, a reduced postischemic hyperemia has been reported in younger (22 years) vs. older (49 years) subjects.[18] Because of the unknown impact of the group age difference, results of this study should be cautiously interpreted as possibly related to both diabetes and age.

An examination of the relative amounts of hyperemia (Table 1) shows that even during partial off-loading, hyperemia appears to be adequate to compensate for the prior interval of ischemia. Theoretically, a flow area ratio (A_R) of 2.0 would be sufficient to compensate for the interval comprised of the flow ischemic interval and the interval during which the hyperemia is occurring. On average, the "break-even" level is slightly exceeded during the partial release. However, this does not account for the fact that the nonDM group's A_R associated with full release was, on average, more than twice the break-even amount. Similar reports [1,2] have suggested that this excess flow may simply represent an overcompensation. More recently, researchers have suggested that the hyperemic response to heel loading and unloading is not dependent only on the ischemia associated with pressure-induced flow reduction.[19] Reports indicate that equal durations of flow stoppage to the heel area resulted in different amounts of hyperemia, thus being greater if the flow stoppage was produced by heel loading as compared with ankle occlusion.

It is possible that the excess flow serves additional physiologic functions. This could imply that a larger hyperemia, associated with full pressure relief, is, in fact, a needed flow response to compensate for sustained intervals of loading and unloading. By extension, this suggests that a diminished hyperemia during complete off-loading, as found in the DM group, in this study, may be problematic if widely present in the diabetic (or possibly older) population, under conditions in which heel loading occurs for sustained intervals.

Although, further work is needed to investigate and clarify this concept, the implication for clinical practice is to attempt to achieve periodic full, rather than partial, heel off-loading in all at-risk patients and to extend the duration of off-loading in those patients with suspected or demonstrated impairments to vasodilatory capacity. ●

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