Effects of Support Surface Relief Pressures on Heel Skin Blood Perfusion

Harvey N. Mayrovitz, PhD; Nancy Sims, RN, LMT, CLT; Martha C. Taylor, BSN, RN, CWOCN; and Lori Dribin, PhD

ABSTRACT

OBJECTIVE: To investigate the effect of pressure-relief magnitude on heel blood flow.

DESIGN: 12 healthy subjects (5 male, 7 female; 21 to 43 years of age) lay on a support surface for 50 minutes with 1 heel on the end cell of the support surface. Cell pressure was computer controlled to vary cyclically at 5-minute intervals between a constant 20 mm Hg during loading and 10, 5, and 0 mm Hg during off-loading. Heel skin blood perfusion was monitored by laser Doppler probes on the heel and foot dorsum. Average skin blood perfusion during each 10-minute cycle and the hyperemic response after pressure relief were determined absolutely and relative to baseline.

SETTING: University research center

RESULTS: An inverse relationship was found between relief pressure and heel skin blood perfusion over each pressurization-relief cycle and during the hyperemia phase. Full-cycle average skin blood perfusion associated with release to 0, 5, and 10 mm Hg were 34.1 ± 7.5 arbitrary units (AU), 26.4 ± 7.5 AU, and 9.3 ± 3.3 AU, respectively (P <.001).

CONCLUSIONS: The reduced average skin blood perfusion is attributable to blunting of hyperemia when relief pressure is too high. When it corresponded to an interface pressure near diastolic pressure, little, if any, functional pressure relief or hyperemia is realized. Suitable relief pressures are likely dependent on an individual's diastolic blood pressure and the net tissue forces acting on heel blood vessels. This suggests that lower blood pressures need lower pressure-relief levels. It is suspected that if depressed vascular responsiveness and/or diminished hyperemic reserve is also present, even lower relief pressures are needed.

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Pressure ulcers due to sustained unrelieved or inadequately relieved pressure are an important clinical, humanitarian, and economic problem.[1-3] Pressure-dependent blood flow changes play a major role in the skin breakdown process, with the greatest breakdown frequency at sites of bony prominences. The heel is particularly prone to such effects,[4] in part because of its relatively lower resting blood perfusion level[5] and higher amount of surface pressure when under load.[6-9] Local blood flow decreases during heel loading[5] and flow recovery after unloading are involved in the breakdown process.[10-12]

Previous work has shown that when the pressure supporting the heel was cycled at different rates, the average blood flow over complete cycles was significantly greater when the level of pressure was zero (full release) when compared with a nonzero-pressure value (partial release).[13] However, because only 2 levels of pressure relief were investigated, the blood flow effects of intermediary levels of pressure relief are unknown. The present study sought to characterize the flow responses of the heel to 3 separate pressure-relief levels when the heel was supported with a uniform load magnitude and duration.

METHODS

Subjects

Twelve volunteers (7 female and 5 male), randomly drawn from the medical school student and staff population, were tested after signing an approved institutional review board consent form. All subjects were free of lower-extremity vascular disease

Harvey N. Mayrovitz, PhD, is Professor of Physiology; Nancy Sims, RN, LMT, CLT, is Research Associate; Martha C. Taylor, BSN, RN, CWOCN, is Research Associate; and Lori Dribin, PhD, is Professor of Anatomy, at the College of Medical Sciences, Nova Southeastern University, Ft Lauderdale, FL. Submitted November 16, 2001; accepted February 22, 2002.

verified by pretest ankle-brachial pressure indices (1.13 ± 0.02 [mean ± SEM]). None were taking medications that would impact vascular reactivity. Demographic features of the overall group were age, 29.8 ± 3.1 years (range, 21 to 43 yr); height, 66.4 ± 1.2 inches; and weight, 148 ± 7 pounds (range, 125 to 195 lb). All subjects were normotensive, with systolic, diastolic, and mean blood pressures of 107 ± 7 mm Hg, 67 ± 2 mm Hg, and 80.3 ± 2.6 mm Hg, respectively. No subject had diabetes or any other notable medical history.

Protocol and support patterns

Subjects lay on a support surface with their left heel positioned on the end cell. Pressure in the supporting cell was computer controlled and could be made to vary on a cyclic basis between a constant upper limit of 20 mm Hg and a variable lower limit of 10, 5, or 0 mm Hg (Figure 1). The overall test sequence was 50 minutes, during which time the dynamic pattern illustrated in Figure 1 was used. The first cyclic pattern was initiated after a baseline recording interval of 10 minutes, during which the heel was not loaded (0 mm Hg). Tests were conducted in a room with well-controlled ambient temperature. During the course of the experiments, room temperature varied from 23.6 ± 0.5°C at the start of the tests to 23.9 ± 0.5°C at the end.

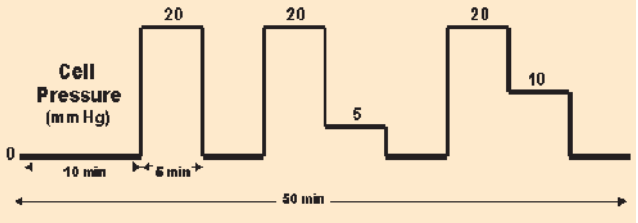
Blood perfusion

Heel skin blood perfusion (SBF) was monitored with a laser Doppler probe affixed to the heel with tape and connected to a perfusion monitor (model BPM^2; Vasamedics, Inc, St Paul, MN). The probe (P-440 Soflex; Vasamedics, Inc, St Paul, MN) is flat, thin, and has a large surface contact area. The probe was positioned where the heel made contact with the support surface. SBF was continuously monitored throughout the experimental sequence. A second probe was placed on the foot dorsum just proximal to the union of the great and second toe. The second probe monitored foot SBF and was connected to a second perfusion monitor of the same type. The foot SBF was used to judge if any systemic changes in SBF occurred during the experimental procedure. All laser Doppler data was acquired using a time constant setting of 1 second. Skin temperature was measured with a thermocouple on the foot. Average skin temperature of the group at the start of the experiment was 30.5 ± 0.6°C and 30.2 ± 0.7°C at the end, with no significant temporal changes detected. At the end of the procedure, the biologic zero of both laser Doppler measures were determined using a thigh cuff that was inflated to 40 mm Hg above systolic blood pressure for 2 minutes. The biologic zero value was subtracted from all laser Doppler raw values, which is standard procedure.[14]

Figure 1.

HEEL SUPPORT PRESSURE PATTERN

During each of the 3 cycles, the heel was supported half the time at 20 mm Hg and half the time at cell-relief pressures of 0, 5, or 10 mm Hg.



Interface pressure

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

Assessment parameters and data analysis

Two main comparison parameters were used. The first was the average SBF during each 10-minute interval in absolute terms and in relation to the zero-pressure baseline average SBF. The second parameter was the hyperemic response subsequent to pressure relief. This was determined by computing the area under the heel SBF flow response curve by integrating the response through the first, second, and fifth minute of the hyperemic response (Figure 2), then dividing by the minutes of hyperemia time to obtain an average SBF for each section of the response. Statistical analyses were performed using a full factorial general linear model (GLM) for repeated measures (SPSS, version 6.1); comparisons between pressure-relief levels were based on repeated contrasts, with a P value less than .01 considered statistically significant.

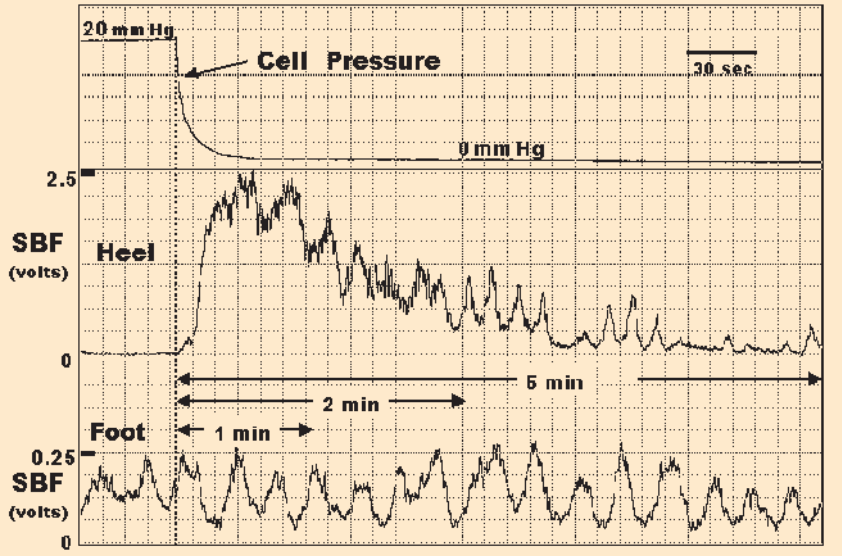
RESULTS

Interface pressures

With the end cell internal pressure set at 20, 10, and 5 mm Hg, interface pressures were 140.9 ± 8.5 mm Hg (range, 109 to 176 mm Hg), 78.6 ± 1.3 mm Hg (range, 73 to 83 mm Hg), and 44.2 ± 3.1 mm Hg (range, 39 to 60 mm Hg), respectively. This wide variation among subjects within cell settings is consistent with previous results[13] and reflects the dependence of interface pressure on multiple factors, such as foot position, body habitus, and heel shape. As a group, these interface pressure levels

Figure 2.
HYPEREMIA DURING PRESSURE RELIEF

Cell pressure is reduced from 20 to 0 mm Hg with an associated hyperemia at the previously loaded heel site. The features of the hyperemia are determined by calculating the cumulative area under SBF curve for the first, second, and fifth minutes of the postrelease response. The foot dorsum SBF was unaffected by the pressurization and release process.



indicate that the maximum support cell pressure (20 mm Hg) corresponded to a value greater than the average systolic pressure (107 mm Hg). The cell pressure of 10 mm Hg corresponded to a value slightly less than the group average diastolic pressure (80.3 mm Hg), and the support cell pressure of 5 mm Hg corresponded to a value significantly less than the group average diastolic pressure.

Features of SBF responses

Cell pressurization to 20 mm Hg caused a decrease in SBF to a level that, on average, was at or close to the biologic zero (Figure 3). This indicates that the maximum cell pressure essentially caused heel ischemia for all or most of its application. Foot dorsum SBF was not affected by either cell pressurization or pressure relief, indicating that heel SBF changes were a localized phenomena. The SBF change accompanying pressure relief depended on the relief-pressure level in a manner generally similar to that shown in Figure 3, although some variations were observed. Release to 0 mm Hg was always associated with significant hyperemia, release to 5 mm Hg normally had some hyperemia, and release to 10 mm Hg cell pressure had a marginal or absent hyperemic response. When the hyperemia was low or absent during the relief pressure, subsequent release to 0 mm Hg was always associated with a significant further flow increase, as shown in Figure 3.

Overall quantitative features

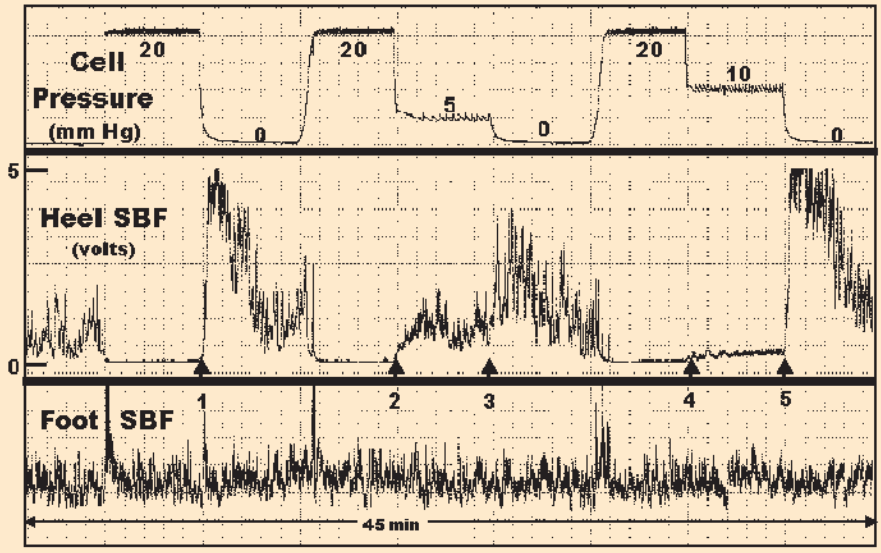
Integrated over the 10-minute baseline and calculated as standard flow units per minute, basal no-load SBF was 22.4 ± 5.7 (Figure 4). The corresponding biologic zero flow was 1.04, which is, on average, less than 5% of the basal flow. The average flow during each of the 10-minute pressurization relief cycles varied, being largest when released to 0 mm Hg (34.1 ± 7.5), least when released to 10 mm Hg (9.3 ± 3.3), and intermediary when released to 5 mm Hg (26.4 ± 7.5). Therefore, the net mean flow for the different cycles was greater than, about equal to, or less than the basal level. From a statistical point of view, analysis of variance for repeated measures showed an overall significant difference within these values (P <.001). Follow-up tests showed that, when released to 0 mm Hg, the full-cycle average SBF was significantly greater than the baseline SBF (P <.001). When released to 10 mm Hg, average SBF was significantly less than baseline SBF (P <.01). When released to 5 mm Hg, average SBF was not significantly different from either the 10-minute average baseline (P = .383) or the SBF when released to 0 mm Hg (P = .220). Expressed in relation to baseline SBF, release to 0, 5, and 10 mm Hg was associated with full-cycle flow ratios of 2.07 ± 3.2, 1.65 ± 3.5, and 0.60 ± 0.14, respectively. Overall, these were significantly different (P <.001), with follow-up tests showing that release to 10 mm Hg was significantly less than the others and showing no significant difference between release to 0 or 5 mm Hg.

Hyperemia features

The postrelease SBF was greatest when pressure was released to 0 mm Hg, intermediary when released to 5 mm Hg, and least when released to 10 mm Hg (Figure 5). In this sense, the hyperemic responses closely paralleled the findings for the average cycle results. By separating the responses into 1-, 2- and 5-minute intervals, the relative contributions of early versus full hyperemia intervals could be discerned. The first minute of hyperemia release to 0, 5, and 10 mm Hg was associated with SBF of 100.5 ± 15.6, 49.0 ± 10.7, and 22.1 ± 7.3, respectively, with the 0 mm Hg release being significantly greater than the other release levels (P <.001).

Figure 3.
OVERALL RESPONSE FEATURES

Typical differences in temporal responses to pressure relief to 0, 5, and 10 mm Hg cell pressure are illustrated for 1 subject. Vertical numbered arrows point to initiation of pressure relief. Note that at a cell pressure of 20 mm Hg (1, 2, and 4), heel SBF is at or near zero and the hyperemia during pressure relief is nearly suppressed when released to 10 mm Hg (arrow 4). Also note that when the heel is fully off-loaded to zero pressure from 5 and 10 mm Hg cell pressures (arrows 3 and 5), there is a significant hyperemia. Large spikes noted in the foot SBF tracing at the transition pressures are due to movement artifact.



However, if the full 5-minute release intervals were considered, the corresponding hyperemia values were 60.8 ± 14.3, 50.50 ± 13.8, and 17.5 ± 5.0, for which there was no significant difference between releases to 0 or 5 mm Hg—both were significantly greater than release to 10 mm Hg (P <.001).

DISCUSSION

Assessment approach

The main findings of this study relate to the effects of dynamic heel support patterns that differed with respect to support pressure-relief magnitude. Impacts of the different pressure-relief levels were assessed via a priori established figure of merit: the relative SBF in the 3 different 10-minute cyclic intervals in relation to 10 minutes of zero-pressure baseline. The rationale of this approach is based on the fact that clinical utility depends on overall perfusion effects, which depend on pressure-relief levels.

Main findings

The data show that for a constant magnitude and duration of heel loading, the average perfusion over full pressurization-relief cycles and the hyperemia during relief diminish with increasing pressure-relief levels. As a consequence of this inverse relationship between relief pressure and heel SBF, the full-cycle perfusion was found to be more than, equal to, or less than basal flow, depending on the pressure-relief level. The fact that complete heel off-loading during a pressure-relief phase yielded a greater relative perfusion when compared with partial off-loading is likely due to the fact that the amount of partial off-loading used in this investigation blunted the normal hyperemic response magnitude. Because hyperemia magnitude during pressure relief contributes most to the full-cycle average perfusion, blunting of this hyperemia would clearly account for the findings. When pressure was released to 5 mm Hg, the net perfusion was near that for the unloaded heel. This is explained on the basis of partial blunting of the normal full hyperemia, which is in excess of that needed to fully repay the prior interval of ischemia, as shown by the release to zero pressure. However, the blunting was especially severe when the support cell pressure was at 10 mm Hg. At this cell pressure level, it is likely that the compression pressure acting on the blood vessels in the heel tissue was near or, in some cases, above the arterial diastolic blood pressure. Little, if any, pressure relief would be realized under these conditions and, consequently, the net average flow would be less than during baseline. The combined results demonstrate the important role of the pressure-relief level in dynamic surfaces targeted for use in pressure ulcer prevention. Based on the present findings and related studies on external pressure effects on localized leg blood flow,[15-17] one may speculate that a suitable pressure-relief level (other than to zero) is likely dependent on the relation between an individual's diastolic blood pressure and the effective tissue forces acting on heel blood vessels. This would suggest that lower blood pressures need lower pressure-relief levels—a concept that may be worth keeping in mind when dealing with patients who may fall into the relatively hypotensive category.

The present results are strictly applicable when normal

Figure 4.
FULL-CYCLE AVERAGE HEEL SKIN BLOOD PERFUSION

Values are SBF (arbitrary units, AU) over each full 10-minute interval. For the 3 pressure-relief intervals, the heel was resting on a cell with an air pressure of 20 mm Hg for 5 minutes prior to off-loading to either 0, 5, or 10 mm Hg. Release to 10 mm Hg was associated with an SBF that was significantly less than all other SBF values (P <.01).

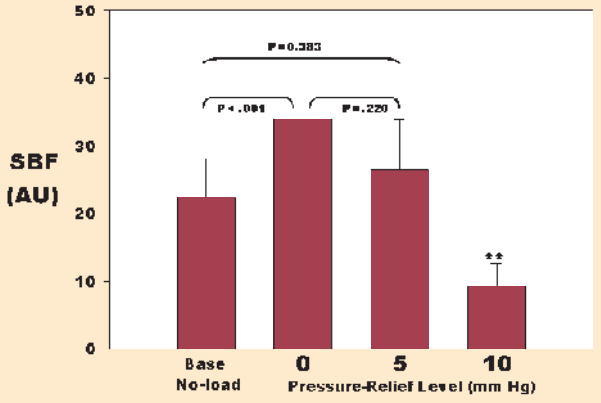
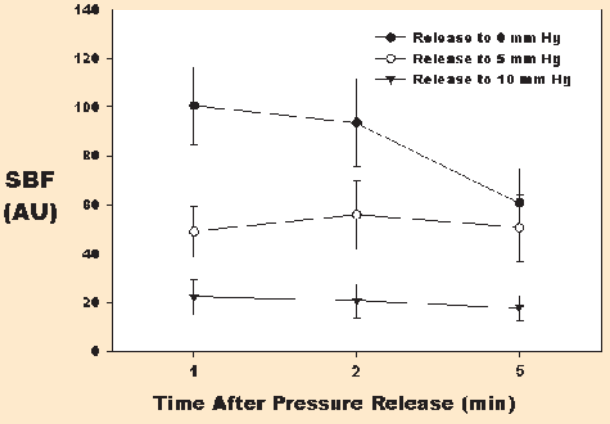


Figure 5.
PRESSURE-RELEASE HYPEREMIA

This figure shows the average SBF during the first minute of hyperemia, the first 2 minutes, and during the full 5 minutes of pressure relief to 0, 5, and 10 mm Hg. Release to 0 mm Hg produced the largest early hyperemia (through 2 minutes); however, there was no significant difference between release to 0 mm Hg or to 5 mm Hg during the full 5-minute interval.



hyperemia potential is present. The impact of depressed vascular responsiveness and/or diminished hyperemic reserve on the qualitative and quantitative aspects of the present findings is unknown. However, it is suspected that when such conditions exist, as in some patients with diabetes mellitus or peripheral vascular disease, the error margins for relief pressure would be significantly reduced. Characterizing these patient groups represents an important major investigative challenge. ●

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CORRECTION

In the article "Physical Assessment of the Diabetic Foot" in the March/April 2003 issue of *Advances in Skin & Wound Care,* the byline for Dr James McGuire was incorrect. The correct byline is James McGuire, DPM, PT, FAPWCA.