Heel-Skin Microvascular Blood Perfusion Responses to Sustained Pressure Loading and Unloading

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ABSTRACT

Objective: Sustained heel pressure during surgery and during acute and longterm care residence can cause heel blood flow deprivation sufficient to cause pressure ulcers. Because little is known about the amount and distribution of heel blood perfusion changes under these conditions, the aim of this study was to characterize the main features of these changes.

Methods: Heel blood perfusion by laser Doppler imaging (LDI, 40×40 mm scans) was measured in 11 vascularly normal persons before (10 minutes), during heel loading (40 minutes) and after off-loading (20 minutes). Loading was done with subjects supine and one heel on a transparent plate through which LDI data were obtained during loading. Analyses were on progressively increasing areas around the central compression site using 10×10 , 20×20 , 30×30 , and 40×40 mm assay areas at each of multiple time points during the 70-minute test.

Results: (1) Heel perfusion is rapidly and significantly reduced on loading (P < 0.01) with the greatest reduction within the central heel area; (2) perfusion remains uniformly depressed throughout the loading interval; (3) off-loading is associated with a rapid onset, specially heterogeneous hyperemia which exceeds baseline (P < 0.01) for 10 minutes.

Conclusions: The present seminal findings may serve as a guide to develop sorely needed microvascular tests to help classify heel breakdown risk on a patient-by-patient basis.

KEY WORDS: heel pressure, blood flow deprivation, perfusion depression.

INTRODUCTION

Sustained pressure on the heel that occurs during open heart surgery and other surgical procedures may result in sufficient local heel blood flow deprivation to place the heel at-risk for subsequent skin breakdown and development of heel pressure ulcers. In addition, pressure ulcer development secondary to continuous pressure over bony prominences is a hazard in all patients, and particularly in patients who are elderly, infirm, vasularly compromised or who have other contributory risk factors. Thus, pressure ulcers are a general, frequent, and costly problem in acute, long-term, and home-care populations. Pro-

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longed localized pressure over bony prominences (e.g., ischium, greater trochanter, heel), combined with the impacts of frictional and shear forces on local blood perfusion lay the foundation for tissue ischemia and potentially subsequent necrosis. Localized heat and humidity are additional factors which increase breakdown risk, in part, due to the associated metabolic effects on blood flow demand which may already be compromised. The heel under pressure is prone to tissue breakdown because of its small surface area of contact, high local pressures, and limited basal blood flow. Heel-breakdown incidence has been reported to be as high as 29.5% (1) and other studies have indicated that the heel is among the most common locations of pressure ulcer occurrence (2–3). When such ulcers develop in patients who have diabetes or peripheral arterial disease the possibility of developing non-healing ulcers and subsequent amputation is a very real possibility. Though local, mechanically-induced blood flow ø

deficits play a central role in the heel-breakdown process, little is known about the details of the blood perfusion changes which accompany heel loading and subsequent off-loading. The purpose of the present study was to investigate and provide seminal information regarding the main features of local heel blood perfusion dynamics in vascularly normal individuals subjected to sustained (40 minutes) heel loading and subsequent off-loading. This was done by measuring heel-skin blood perfusion by laser-Doppler imaging before, during, and after heel loading in 11 volunteer subjects.

METHODS

Study Population

Eleven healthy women gave Institutional Review Board-approved informed consent and served as volunteer subjects. Women were chosen for this initial study because about 70% of long-term care residents are women. Participants ranged in age from 31–60 years (mean \pm sem, 42 \pm 7 years) and were of different ethnic backgrounds (five Caucasian, five Hispanic, and one African-American). Three subjects were current cigarette smokers.

Preparatory Sequence

Tests were done in a temperature controlled laboratory maintained at 22-23°C. On arrival, subjects took a supine position on an examination table with a foam overlay mattress. Subjects were positioned with both heels extending over the table edge so as to not be influenced by external pressure. The solidstate head of a laser-Doppler imaging (LDI) system (LISCA Development AB, Linkoping, Sweden), was placed under the right (experimental) heel at a distance of 19 cm and set to scan an area of 40×40 mm at a high resolution. This setting corresponds to a scan of 40×40 pixels (1600 individual perfusion data points within the scanned area). Initially, the right heel was placed briefly on a transparent plastic plate (3 mm thick) which was later used as the pressure loading surface. This surface was supported across a specially constructed stand, at a distance of 19 cm from the LDI head. The center of the heel pressure area in contact with the plastic plate, was determined visually and marked with a 2×2 mm felt square. The plastic plate was removed and the subject remained in a supine position and the feet were covered with a black sheet which served as a backdrop for the LDI scans. Overhead room lights were turned off to minimize potential ambient light effects on perfusion measurements. The LDI head

was aligned with the beam centered at the 2×2 mm felt target site on the heel.

Instrumentation

Because of the need for, and usefulness of, measuring heel blood perfusion at multiple sites surrounding the central pressure area, and the need to make measurements during heel loading, laser-Doppler imaging (1,11,19,20,21) was chosen for its noncontact and large skin-area sampling features. Thus, whereas standard laser-Doppler requires placement of probes on skin (14,15) LDI is non-contact and provides perfusion data over a larger area. The system used emits a low power laser beam (635 nm) that penetrates the tissue to a variable depth ranging from a few hundred to about 1000 micrometers to provide spatial perfusion data (13). In the presence of moving blood cells, the laser light is Dopplerbroadened, partially backscattered, and the reflected light collected by a photodetector within the solidstate laser head. Signals are processed according to an instrumental algorithm that results in an output proportional to tissue blood perfusion. Based on relative perfusion in each of the sampled areas of the scanned region expressed in arbitrary units (a.u.), a color-coded image is generated in which spatial regions having perfusions within specified limits are similarly colored. In general, deep red corresponds to the highest perfusion and deep blue the lowest. In between colors from lowest to highest perfusion are light blue, green, and yellow. Further details on LDI use and applications may be found in the literature (6, 12).

Protocol

Subjects were requested to remain as still as possible throughout the 70-minute experimental procedure. With both feet protruding over the edge of the exam table, and thus off-loaded, the experiment began with a 10-minute baseline interval during which four LDI scans were taken of the right heel. At the end of the baseline interval, the right heel was loaded by placing it in contact with the supported clear plastic plate. After re-aligning the scan head with the beam center on the previously marked target, LDI scans were taken at 2, 4, 6, 8, 10, 15, 20, 30, and 40 minutes after initial loading. After 40 minutes of heel loading, the plastic plate was removed and the right heel was again off-loaded and LDI scans were taken 30 seconds, 2, 4, 6, 8, 10, 15, and 20 minutes after off-loading. At the end of each experiment, LDI scans were taken of a background calibration white card with and without the plastic plate interposed.

Analyses

The LDI-generated images were analyzed by computing the average perfusion among areas of 10×10 mm, 20×20 mm, 30×30 mm, and 40×40 mm around the center of the pressure area. This procedure assigned a single numerical value for each average perfusion in the designated areas in arbitrary units. The calibration "zero-flow" background value (0.25 a.u.) was subtracted from all data. The analysis allowed for an investigation into the distribution of blood flow around the center of the pressure area and the possible variations in flow both spatially and temporally. The central "grey" area due to skin covering of the felt-marker is not included in the perfusion analysis.

RESULTS

Composite Response to Compression and Off-Loading

A typical colorized perfusion image sequence for one subject is shown in Fig. 1. In this composite figure,

contours of the same color correspond to heel regions having similar relative blood perfusion levels. Red contours represent the greatest perfusion and deep blue the lowest; intervening colors of yellow and green are intermediate perfusion levels in ascending order. This type of representation is useful to observe rapidly the spatial variations in perfusion bands within different heel regions. The overall quantitative temporal sequence for the entire group is shown in Fig. 2. Each data point is the mean blood perfusion (a.u.) measured in the 10×10 mm area centered around the central heel-compression site. Data points are connected by a spline curve which represents the best smoothed fit to the composite data with error bars showing ± 2 sem at each time point. The hyperemic perfusion following off-loading is significantly (P < 0.01) larger than baseline and remains so for 10 minutes.

Response to Compression

As compared with heel average-baseline perfusion levels, compression loading was associated with a perfusion reduction in all subjects. In Fig. 3, LDI perfusion data for the central area are separated in terms of three key intervals; pre-load baseline, 40minute heel-loading, and the 20-minute recovery interval after off-loading. Perfusion during loading was significantly less than the pre-loaded baseline (P < 0.01). During loading, there appears to be a slight

Figure 1. Sequential LDI scans. Example perfusion response to loading and off-loading illustrated as color-coded sequential images. In order of increasing perfusion values, colors are blue, green, yellow, and red. A rapid hyperemia

is noted at off-loading and the non-uniformity of intra-zone perfusion is seen.





Figure 2. Overall perfusion response. Data points are means which are connected by a spline curve representing the "best fit" to the data; error-bars are ± -2 *SEM*. Data is for the 10 × 10 mm area around the center of pressure; loading begins at time = 0 and off-loading at time = 40 minutes. (*) denotes significantly greater than baseline.

progressive increase; this trend is not significant and end perfusion is not significantly different from mean perfusion during the first 10 minutes of loading.

Analysis of baseline perfusions for each of the four assayed areas showed no significant differences in heel perfusion values among the respective assayed areas with mean values as shown in Fig. 4. During heel-loading, the central 10×10 mm area which samples perfusions nearest to the center of applied pressure, shows the largest perfusion decrement and the largest area (40×40 mm) shows the smallest perfusion decrement.

The Hyperemic Response to Off-Loading

Pressure-release with off-loading is associated with a rapid increase in perfusion which significantly exceeds baseline (P < 0.01) for an interval extending for 10 minutes as illustrated in Figs. 2 and 3. The amount of hyperemia is dependent on the specific regions scanned, which as shown in Fig. 4, decreases with increasing assayed heel areas. However, initial

hyperperfusions were significantly greater than corresponding baseline-area perfusion for all areas (P < 0.01).

DISCUSSION

The present study used laser Doppler imaging to characterize heel skin microcirculatory changes accompanying heel loading and off-loading. This was done as a first step in a process targeted to gaining insight into potential blood-perfusion linkages to pressure ulcer development. The salient findings demonstrate the following main points: (1) Heel perfusion, even in healthy individuals, is rapidly and significantly reduced upon loading with the greatest reduction occurring within the central heel loading area; (2) blood perfusion remains depressed throughout the loading duration with only a hint of an adaptive vasodilatory response during sustained loading; (3) off-loading is associated with a rapid onset hyperemia, which appears to be a signature of a heel blood flow functional deficit occurring during the loading interval which likely serves to offset rela-



Figure 3. Perfusion response by key intervals. Loading causes a significant (P < 0.01) reduction in heel blood perfusion as compared with baseline (\blacklozenge). The off-loading interval is associated with a significant (P < 0.01) hyperemia lasting 10 minutes (*).

tive flow deficits during loading. Standard clinical practice recommendations for the prevention of pressure ulcer development are to relieve pressure in patients via turning or other off-loading procedures at about 2-hour intervals. The loading interval here used (40 minutes), even though much less than this recommendation, still caused a significant hyperemic response. Although not specifically evaluated in the present study, it is likely that longer intervals of unrelieved pressure would require greater hyperemic responses to repay the greater blood perfusion debt. Longer durations of loading may also cause cumulative, localized heel injurious effects which may serve to suppress vasodilatory reserve, thereby compounding and exacerbating dependent tissue injury and integrity. This may be an issue even in vascularly healthy individuals as studied here, but is an important aspect in infirm patients and those with an already compromised lower extremity vascular status.

These concepts are consistent with previous results in which standard laser Doppler probes were used to assess skin blood perfusion at sites prone to skin breakdown. Although the presence of the probe undoubtedly changes localized pressures and flows

when under compression and registers perfusion at only one small sample site, certain general features of the perfusion responses are useful for comparative purposes. Varying pressure levels applied to the heel of patients and volunteers with an indenter device showed a progressive decrease in perfusion which, at 50 mmHg, reduced local heel perfusion to zero (2). Similar results were observed when heels were placed on standard hospital mattresses. Interface pressures between the heel and mattress have been reported higher than systolic arterial pressure (8) with associated zero heel-skin blood perfusion in both healthy and ill individuals during loading accompanied by hyperemic responses when off-loaded. A possible suppression in thermal vasodilatory reserve (and by implication reduced hyperemic capacity) at a bony-prominence site also prone to pressure ulcer development (greater trochanter) has previously been demonstrated for individuals older than 60 years of age and for hospital in-patients as compared with age-matched non-patients (9). Thermographic estimation of trochanteric hyperemic responses following loading shows responses that appear to be a function of individual susceptibility to loading forces (5) and by implication, dependent on individual vasodilatory capacity. Other studies of 231



Figure 4. Spatial differences in perfusion. Areas closest to the center of pressure tend to have the greatest perfusion deficits during loading and the greatest hyperperfusion during off-loading; (\blacklozenge) significantly greater and (*) significantly less than the 10 × 10 mm area.

greater trochanter skin perfusion suggest the possibility of an adaptive vasodilatory response during loading in healthy subjects, both young and elderly (17), but with significantly more variability and an absence of vasodilation in infirm individuals (10).

The present findings, together with previous reports, thus suggest that a key microvascular functional parameter which may impact on pressure ulcer development likelihood is adequacy of functional microvascular reserve. Such a parameter, of course, is useful only if the continuous duration of loading to which a patient is exposed is less than a critical (but unknown) time during which irreversible tissue ischemic injury has not occurred. Laser Doppler imaging has shown that perfusion deficits are present under normal heel-lying conditions with heterogeneous perfusion responses within the heel area that are generally greatest within central compression areas. In view of the complications and catastrophic sequelae which can be triggered by heel pressure ulcers, new approaches to help minimize their occurrence are needed. One important aspect is the early detection of those individuals who, by virtue of an impaired microcirculation, are at significantly increased risk of heel-skin breakdown. A potentially useful future research target would be to develop suitable microvascular reserve assessment procedures which could be used to better classify relative heel breakdown risk on a patient-by-patient basis prior to cardiac and other surgeries, and prior to admittance to acute and long-term care facilities.

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