The Relative Contributions of Interface Pressure, Shear Stress, and Temperature on Tissue Ischemia: a Cross-sectional Pilot Study

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Abstract

Tissue ischemia is thought to play a major role in the development of pressure ulcers. Pressure, shear, and temperature are acknowledged contributors, but the relative magnitude of each factor is largely unknown. A cross-sectional pilot study was conducted on the sacrums of four healthy volunteers to estimate the relative contributions of each variable by systematically varying and assessing the resulting level of ischemia in the skin tissue. Using a repeated measures design, 21 combinations of temperature (28˚ C, 32˚ C, and 36˚ C); pressure (0 kPa, 8.0 kPa, and 13.3 kPa), corresponding to 0 mm Hg, 60 mm Hg, and 100 mm Hg; and shear stress (0 kPa, 6.7 kPa, and 14.0 kPa), corresponding to 0 mm Hg, 50 mm Hg, and 100 mm Hg (practical testing values), were tested twice, for a total of 168 trials. Using laser Doppler flowmetry, the magnitude of post-load reactive hyperemia was used as an index of ischemia. Fixed Effects and Ranks linear regression models were developed to predict three different indices of reactive hyperemic magnitude with pressure, shear stress, and temperature as the variables. Pressure and temperature were always highly significant predictors of the extent of reactive hyperemia ($P<0.0001$ for Perfusion Area, peak minus baseline blood flow, and Normalized Peak blood flow), and the contributions of shear stress were insignificant ($P=0.5351$ for Perfusion Area, $P=0.6403$ for Peak minus Baseline blood flow, and $P=0.8941$ for Normalized Peak blood flow). Depending upon the model, comparison of coefficients suggested that an increase of 1.0˚ C contributes as much to reactive hyperemia in the skin as 12 mm Hg to 15 mm Hg of interface pressure (coefficient ratios of temperature/pressure are 14.33 for Perfusion Area, 11.77 for Peak minus Baseline, and 12.97 for Normalized Peak, respectively). The findings also indicate that post-load metabolic repayment varied with temperature only at higher pressures, suggesting protective vasodilation was able to keep pace with mild compression. If confirmed in subsequent studies, the results suggest that managing both skin pressure and temperature may reduce the risk of ischemia.

Keywords: pressure ulcer, in vivo, shear, temperature, risk factors, hyperemia


Potential Conflicts of Interest: Mr. C. Lachenbruch is an employee of Hill-Rom, Batesville, IN.

Literature Review

Pressure ulcers are a healthcare and economic concern worldwide. Although recent laboratory studies have led researchers to conclude that pressure ulcer formation has many causes and origins, the consensus of early laboratory studies support the hypothesis that ischemia is one of the major causes of pressure ulcers. When blood vessels on the arterial side are compressed by pressure on the skin, perfusion can be occluded and distribution of nutrients to the tissue is reduced. At the same time, compression of veins and venules can hinder the removal of metabolic wastes, and they accumulate. Sustained ischemia eventually results in tissue necrosis, and finally, in ulceration. Although various studies have identified that shear stress and elevated skin temperature influence tissue ischemia in tissue under loading, few have attempted to examine the relative importance of their contributions. The answer to this question has importance for both therapeutic intervention and product design.

Effect of shear on ulcer formation. Theoretically, sustained ischemia can be caused by any mechanical stress...
that sufficiently deforms the tissue and disrupts the vascular and lymphatic networks; therefore, both pressure and shear are thought to work together to cause ischemia. It has been hypothesized that because capillary loops in the skin are oriented vertically, they would be more resistant to direct, vertically oriented pressure but could be readily kinked and disrupted by shear.

Several laboratory studies have confirmed the contribution of shear to tissue breakdown. Dinsdale conducted an animal study in which a motorized device was configured to apply repetitive loads in the form of cyclic pressure and frictional forces. He found that when pressure was combined with frictional rubbing, a lower pressure was sufficient to cause ulceration than when pressure alone was applied. From the associated chemical analysis, he concluded the tissue damage was primarily mechanical and not caused by an ischemic mechanism. Bennett et al also studied the combined influence of pressure and shear on blood flow. Measuring pulsatile arteriolar blood flow on the palms of the hands of four healthy volunteers in a laboratory study, they concluded that when applied shear stress was sufficiently high (greater than approximately 10.0 kPa), the pressure necessary to produce occlusion was half that required when little or no shear stress was present. Using a self-developed parallel-plate capacitance shear sensor, Goossens et al measured the combined effects of pressure and shear stresses in the laboratory on skin oxygen tension at the sacrum on four young, healthy subjects. They found the mean pressure required to reduce the skin oxygen tension to 1.3 kPa was 11.6 kPa when no shear stress was applied and only 8.7 kPa when applied in combination with shear stress of 3.1 kPa. In another laboratory study, Zhang et al measured perfusion on the back of the thighs of five healthy volunteers using laser Doppler over a wide range of pressure and shear stresses. They also modeled the resulting stress fields in the tissue in an effort to determine the effect on perfusion. Model results suggested flow would be reduced in proportion to the resultant local mechanical stress — i.e., to be proportional to \( \text{pressure}^2 + \text{shear} \). This generally was supported by their lab measurements on human subjects. It should be noted that because their measurements of blood flow were made using laser Doppler, they reflect perfusion in the top 2 mm to 2.5 mm layers of tissue. Linder-Ganz and Gefen used finite element modeling of muscle tissue validated by animal studies using magnetic resonance imaging to evaluate the functioning of capillaries in loaded muscle tissue. Model results indicated that over a range of pressures of 12 kPa to 120 kPa with no shear, the percentage of open capillary cross-sectional area decreased by up to 71%, and the open capillary cross-sectional area decreased more rapidly when shear strains were added.

Effect of increased skin temperature. While shear historically has been accepted as one of the principle causative factors for pressure ulcers of ischemic origin, fewer studies have focused on the importance of increased skin temperature. Because tissue metabolism generally increases by 6% to 13% per degree Celsius, it is believed that cooler tissue better withstands ischemia when loaded and therefore lengthens the time period before tissue necrosis occurs. This effect has been demonstrated in two similar animal studies. Kokate et al applied constant pressures of 13.3 kPa for 5 hours to 16 swine in the laboratory. The pressure was applied by four temperature-controlled indenters on each animal set to maintain 25˚C, 30˚C, 35˚C, and 40˚C. After 7 days, the animals were sacrificed, and histological samples indicated that the severity of tissue injury at each site was closely related to the imposed temperature. The results, classified by observers blinded to the treatment, indicated that at 13.3 kPa, 25˚C, no superficial or deep tissue damage occurred. At 13.3 kPa, 35˚C, significant deep tissue damage and necrosis occurred, but no superficial damage was evident. At 13.3 kPa, 40˚C, significant deep tissue damage and superficial damage to the skin was noted, and at 13.3 kPa, 45˚C, the significant deep tissue damage and superficial damage observed was much more severe than what was observed at 40˚C. In summary, at identical pressure-time loads, with increasing temperature, the damage became much more severe and included deep and superficial tissue layers.

Iaizzo et al, using the same experimental model, Kokate used in the original study he co-authored, extended the result to finer increments of pressure, temperature, and time to develop mathematical formulae for the breakdown thresholds of epidermis, dermis, fat, and muscle tissue, respectively. In this study, 70 different experiments were conducted with temperatures ranging from 25˚C to 45˚C and applied for periods ranging from 5 minutes to 4 hours.
from 25˚C to 45˚C, pressures from 10 mm Hg to 150 mm Hg (1.3 kPa to 20.0 kPa), and intervals from 1 hour to 10 hours. This appears to be the only published model in which the relative contributions of pressure and temperature have been quantified. More recently, Tzen et al21 developed a noninvasive model that demonstrated the effect of mild skin cooling on the effect of ischemia during a specified pressure load. They imposed localized pressures of 8 kPa for 30 minutes with or without local skin cooling to 25.0˚C on sacral skin of 10 young healthy adults (18 to 40 years old). They then measured the magnitude of the resulting reactive hyperemia inflow as an index of ischemia using the laser Doppler flowmetry (LDF) system. Time series results showed that normalized peak skin blood flow (SBF) post-load was significantly lower with cooling (P = 0.019), which indicated a reduced effect of tissue ischemia.

Table 1. Test conditions: shear stress (S) at each combination of temperature (T) and pressure (P)

<table>
<thead>
<tr>
<th>Skin temperature</th>
<th>P = 0 kPa</th>
<th>P = 8.0 kPa</th>
<th>P = 13.3 kPa</th>
</tr>
</thead>
<tbody>
<tr>
<td>T = 28˚C</td>
<td>S = 0 kPa</td>
<td>S = 0, 6.7, and 14.0 kPa</td>
<td>S = 0, 6.7, and 14.0 kPa</td>
</tr>
<tr>
<td>T = 32˚C</td>
<td>S = 0 kPa</td>
<td>S = 0, 6.7, and 14.0 kPa</td>
<td>S = 0, 6.7, and 14.0 kPa</td>
</tr>
<tr>
<td>T = 36˚C</td>
<td>S = 0 kPa</td>
<td>S = 0, 6.7, and 14.0 kPa</td>
<td>S = 0, 6.7, and 14.0 kPa</td>
</tr>
</tbody>
</table>
Selection of the three shear forces was based on the practical aspects of the existing experimental set-up. Because shear stress does not exist without localized pressure, the 6.7 kPa and 14.0 kPa shear conditions were not applied at 0 kPa of pressure. Each condition (combination of pressure, skin temperature, and shear stress) was tested twice, and a total of 42 tests were conducted on each volunteer.

Procedure. A reactive hyperemic response was induced to quantify tissue ischemia noninvasively using LDF measurement of SBF. Each one of the 42 tests took 30 minutes to complete and was composed of three time periods: 5 minutes for collection of baseline SBF (no pressure, shear stress, or temperature control), followed by 15 minutes of measurement with one of the combinations of pressure, shear, stress and skin temperature listed in Table 1, and then 10 minutes for collection of the reactive hyperemic response (no pressure, shear stress, or temperature control). Figure 2 demonstrates the three time periods described above.

All procedures were performed in the Tissue Integrity Management Laboratory at the University of Pittsburgh. The room temperature was maintained at 20 ± 1˚C. The order of test conditions was randomized for each participant. To minimize the carryover effect of a previous trial, the tests alternated between the left and right sides of the sacral skin, and the location for the initial test was randomized at the beginning of each test day. Two to eight tests were performed on the volunteer each visit depending on his/her schedule, and each volunteer completed the 42 tests within 1 month.

Instrumentation. The instruments used in this study include a customized indenter to control the pressure, temperature, and shear stress applied to the skin, and a LDF system to measure the SBF. The test system (see Figure 3) was used in a previous study by Tzen et al, in which various measures of reactive hyperemia were employed to assess the extent of ischemia resulting from localized pressure with or without temperature control. The system was modified to allow the application of shear stress and allows simultaneous control of pressure, temperature, and shear stress. A customized computer-controlled indenter contains two closed-loop controllers to maintain the amount of pressure and temperature applied on the skin. To create the shear stress, a weight was attached to the base of indenter via a pulley system. Two different weights were used to create two different amounts of shear stress. The 100-g weight imposed a 0.98 N shear force over the 6.45 cm² area of the indenter for a shear stress of 6.7 kPa, and a 200-g weight was used to impose a shear force of 1.96 N over the same area for a shear stress of 14.0 kPa.

A LDF probe was located at the center of the circular indenter. The LDF system was used to collect the SBF
and measure the reactive hyperemic response in the upper 1 mm to 2 mm of the tissue — ie, the epidermis and upper dermis. This response has been used in various studies to measure vascular response and endothelial function; the intensity of reactive hyperemia following stimulus indicates the degree of soft tissue distress.

Outcome measures. To quantitify tissue ischemia non-invasively, reactive hyperemic response was used as the main outcome measure of this study. Raw SBF data were collected at 20 Hz with the LDF. The SBF data were first downsampled to 0.5 Hz, and a Chebyshev I low-pass filter (with the cut-off frequency at 0.15 Hz) was used to filter the downsampled SBF. Four indices were generated from the SBF, including baseline SBF, peak SBF, perfusion area, and normalized peak SBF. Figure 2 demonstrates the selection of these indices from the SBF data.

Baseline SBF was calculated by taking an average of the SBF during the first 5 minutes of the experiment. To select the other two indices of reactive hyperemia objectively, a bi-exponential equation (Equation 1) was used to fit the curve of the reactive hyperemic response (SBF collected during the last 10 minutes of the trial). The solid line in Figure 2 demonstrates the curve fit of the reactive hyperemic response. A1, A2, B, r1, and r2 are constants generated by the curve fit from each test. Each test generates one set of constants that is different from the other tests, and these five constants were used to generate the peak SBF and perfusion area in Equation 2 and Equation 3, respectively.

\[y(t) = A_1 e^{-r_1 t} + A_2 e^{-r_2 t} + B\]  
(Equation 1)

\[y_{\text{peak}} = A_1 e^{-\frac{t_1}{r_1}} + A_2 e^{-\frac{t_2}{r_2}} + B\]  
(Equation 2)

\[\text{perf} = \left(\frac{1}{\text{area}}\int_0^t y dt\right)\times 100\%\]  
(Equation 3)

\[y_{\text{normalized-peak}} = \sqrt{\frac{y_{\text{peak}} - y}{y}} \times 100\%\]  
(Equation 4)

Statistical analyses. Three different statistical analyses were performed to test these specific aims of this study. Incremental contributions of the variables. Regression analyses were used to test the incremental contributions of each factor (pressure, shear stress, and temperature) to the reactive hyperemic response. To ensure regression model strength, both Fixed Effects and Rank Regression (non-normality as checked by plotting the residuals after fitting the model) models were computed. Because multiple observations were made on each volunteer (two replications at each temperature, pressure, and shear condition) and the observations were not independent, a panel data method used the volunteer as the panel indicator. Equations were developed using pressure only, pressure and temperature only, and all three variables (pressure, temperature, and shear stress). The STATA software package (version 12, Stata Corp, College Station, TX) was used.

Table 2. Fixed Effects linear regression

<table>
<thead>
<tr>
<th>Independent variables</th>
<th>Coefficient</th>
<th>Coefficient</th>
<th>Coefficient</th>
<th>Constant</th>
<th>Overall R²</th>
<th>LR (P value)a</th>
<th>Incremental variable significance at 0.05*</th>
</tr>
</thead>
<tbody>
<tr>
<td>P</td>
<td>21.372</td>
<td>-117.04</td>
<td>0.1169</td>
<td>Na</td>
<td>Na</td>
<td>Na</td>
<td>Na</td>
</tr>
<tr>
<td>P, T</td>
<td>21.372</td>
<td>306.191</td>
<td>0.3445</td>
<td>&lt; 0.00001</td>
<td>SIG</td>
<td>Na</td>
<td>Na</td>
</tr>
<tr>
<td>P, T, S</td>
<td>20.294</td>
<td>306.191</td>
<td>2.395</td>
<td>-9948.50</td>
<td>0.3466</td>
<td>0.4515</td>
<td>Na</td>
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</tbody>
</table>

<table>
<thead>
<tr>
<th>Dependent variable: Perfusion Area</th>
</tr>
</thead>
<tbody>
<tr>
<td>Independent variables</td>
</tr>
<tr>
<td>-----------------------------------</td>
</tr>
<tr>
<td>P</td>
</tr>
<tr>
<td>P, T</td>
</tr>
<tr>
<td>P, T, S</td>
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</tbody>
</table>

<table>
<thead>
<tr>
<th>Dependent variable: Peak minus Baseline</th>
</tr>
</thead>
<tbody>
<tr>
<td>Independent variables</td>
</tr>
<tr>
<td>----------------------------------------</td>
</tr>
<tr>
<td>P</td>
</tr>
<tr>
<td>P, T</td>
</tr>
<tr>
<td>P, T, S</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Dependent variable: Normalized Peak (peak/baseline)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Independent variables</td>
</tr>
<tr>
<td>---------------------------------------------------</td>
</tr>
<tr>
<td>P</td>
</tr>
<tr>
<td>P, T</td>
</tr>
<tr>
<td>P, T, S</td>
</tr>
</tbody>
</table>

* LR= Likelihood ratio test; P-value for significance of contribution of added variable; Na = not applicable, SIG = significant, Ns = not significant independent variables: P = pressure; T = temperature; S = shear.
to determine the between-estimator for Fixed Effects and Rank Regression models. These were compared for significant contribution by the added variable using a likelihood ratio test. Coefficient ratios of all predictors in the regression model were calculated based on the equation produced. The coefficient ratios reflect the relative contribution of each independent variable to the magnitude of the dependent variable.

Within-subject comparison of variables. The second analysis was a within-subject comparison of reactive hyperemic response to pressure, shear stress, and skin temperature. Because the reactive hyperemia indices were not normally distributed, Friedman's test was used to compare the reactive hyperemic response (average of both trials of each test combination) among different skin temperatures, applied pressures, and shear stresses individually (eg, comparing reactive hyperemia among three different temperatures at one pressure and one shear stress).

Cross-validation of the regression models. The third analysis was the cross-validation of the regression model using a previously conducted human volunteer laboratory study measuring reactive hyperemia in young healthy adults.21 A limited two-variable, cross-validation of the Fixed Effects and Rank Regression models was performed using the previously collected pressure and temperature data.21

### Results

Regression models and coefficient ratios. In the Fixed Effects Regression model, temperature and pressure were able to predict the magnitude of all three reactive hyperemia indices significantly better than pressure alone (see Table 2). With Perfusion Area SBF as the dependent variable, the addition of temperature increased $R^2$ from...
ligible contributions of shear stress. The figures for Normalized Peak SBF were 0.2223 for pressure and 0.3482 for pressure and temperature (likelihood ratio P < 0.001). The figures for Normalized Peak SBF were 0.2223 for pressure and 0.3482 for pressure and temperature (likelihood ratio P < 0.001).

Because only pressure and skin temperature were significant predictors in all six regression models, coefficient ratios of pressure and skin temperature for each model were calculated. The Fixed Effects Regression models showed that a 1.0°C increase in temperature has 14.33 times as much effect as a 1 mm Hg increase in interface pressure; 1.0°C also contributes as much as 11.77 mm Hg (1.57 kPa) pressure to Peak minus Baseline SBF, and 12.97 mm Hg (1.73 kPa) pressure to the Normalized Peak SBF index (see Table 4). The Ranks Regression models showed a 1°C of temperature contributes as much to Perfusion Area as 8.74 mm Hg (1.17 kPa) of pressure; for Peak minus Baseline SBF, the values are 8.49 mm Hg (1.13 kPa), and for Normalized Peak SBF, 7.72 mm Hg (1.03 kPa).

Within-subject comparisons of reactive hyperemia caused by three factors. When no shear stress was applied, the Normalized Peak SBF and Perfusion Area SBF were significantly different among the three pressures at 36°C (P = 0.039 and 0.018, respectively). However, at 28°C and 32°C, they were not significantly different among the three pressures (P = 0.145 at 28°C and P = 0.085 at 32°C for Normalized Peak SBF, and P = 0.145 at 28°C and P = 0.085 at 32°C for Perfusion Area) (see Table 5). With respect to the effect of skin temperature on reactive hyperemia at different combinations of pressure and shear stress, Perfusion Area was significantly different among the three temperature for 100 mm Hg of pressure (P = 0.03, 0.039, and 0.039 for 0 mm Hg, 50 mm Hg, and 105 mm Hg of shear stress, respectively). The effect was not significant at 60 mm Hg of pressure (P = 0.472, 0.105, and 0.097 at 0 mm Hg, 52 mm Hg, and 105 mm Hg of shear stress respectively) (see Table 6). No significant differences were noted among the three shear stresses at various combinations of pressure and skin temperature (P > 0.05).

Cross-validation with other study data. Application of the Fixed Effects and Ranks Regression equations to a data set, which included pressure, temperature, and all three indices of reactive hyperemia, showed regressions that were between the outputs of equations generated in the present study (see Table 7). Actual measured indices of reactive hyperemia in that study resulted in P = 0.157 or less in all cases, and the regression slopes were statistically significant at the 0.05 level for Perfusion Area for both the Fixed Effects and Ranks Regressions.

**Discussion**

The purpose of this cross-sectional laboratory study was to determine the degree to which interface pressure,
shear stress, and skin temperature contribute to ischemia. In this study sample of healthy volunteers, it was found that potential ischemia in the skin can be estimated with greater accuracy if both pressure and skin temperature, not shear stresses, are taken into account.

Relative contribution of pressure and temperature to tissue ischemia. The contribution of temperature to the effect of pressure on ischemia was significant across all analyses. The $R^2$ for the Fixed Effects Regressions approximately tripled when temperature was added to the equation compared to using only pressure. The increase in $R^2$ was 60% to 70% for the Ranks Regression equations.

The relative contributions of pressure and temperature on the effect of ischemia, as quantified by the ratio of the coefficients of the two variables in the regressions equations, were relatively consistent. This study found $1^\circ C$ contributes 7.72 to 14.33 times as much to the effect of ischemia as 1 mm Hg (0.133 kPa), depending on the index of reactive hyperemia and the regression model used. The only directly comparable figure in the literature comes from Lachenbruch, who estimated from secondary analysis of published data that $1^\circ C$ contributed as much to ischemia as 5 mm Hg or more (0.67 kPa) of pressure. Another comparable model was developed based on histological analysis of animals. Iaizzo developed a quantitative model for skin breakdown by systematically subjecting swine to specified ranges of pressure, temperature, and duration. After histological analysis of the tissue, the model estimated the pressure required to cause skin breakdown after a given duration at a specified temperature. His prediction equations indicated that $1^\circ C$ contributes 11.5, 2.5, and 39.9 times as much as 1.0 mm Hg (0.133 kPa) of pressure for muscle tissue, subcutaneous fat, and dermis respectively.

Contribution of shear stress to tissue ischemia. Another finding in the present study was the apparently negligible contribution of shear stress to ischemia in the skin. This is somewhat at odds with current expectations based on several previous preclinical studies and vascular architecture but consistent with others, given the focus on blood flow in the skin. In their review and analysis of vascular architecture, Lin et al hypothesized that because capillary loops in the skin are oriented vertically, they should be more resistant to direct, vertically oriented pressure but could be readily kinked and disrupted by lateral shear forces. Goossens, citing both Lin’s intuitive argument and the tendency for shear to be applied over broader areas than pressure, states, “Shear can be considered even more significant than pressure in the causation of pressure ulcers.” Similarly, Zhang et al concluded that with respect to blood flow, “Shear can be considered at least as important as normal force.”

Quantitatively, Bennett et al estimated that shear was approximately half as effective as pressure at occluding pulsatile arterial flow in the palm of the hand. Zhang et al, taking a modeling approach supported by transcutaneous oxygen measurements, concluded the reduction in tissue oxygen level was proportional to the vector sum of the pressure and shear loading on the tissue, which is equivalent to concluding that pressure and shear are equally important. Goossens, also using transcutaneous oxygen measurements, observed that a full “cut-off” pressure could be achieved at the sacrum of healthy subjects at a mean pressure of 11.6 kPa with no shear stress or 8.7 kPa and 3.1 kPa shear stress. Using finite element models, Linder-Ganz and Gefen concluded that, over a wide range of applied pressures, the percentage of open capillaries in muscle tissue decreased significantly with the addition of 8% shear strain.

The current study’s finding that shear stress made a negligible contribution to sacral ischemia may be a consequence of different measurement of tissue response. In this study, researchers measured the relatively prolonged physiological effect on skin tissue and vascular deformation with reactive hyperemia instead of the direct effect on blood flow or oxygen reduction with the presence of shear force — ie, reactive hyperemia was collected after 15 minutes of loading was removed, different from previous studies where blood flow changes or tissue oxygenation were measured immediately following load application. Reactive hyperemia is mediated by the accumulation of metabolic vasodilator substances, nerve reflexes to maintain blood pressure, and changes in intramural pressure. Because shear force could not be applied at 0 kPa of pressure, comparisons of reactive hyperemia only were made among different shear stresses with pressures of 8.0 kPa or 13.3 kPa. The additional blockade of blood flow or the tissue deformation caused by shear stress may not be profound enough when 8.0 kPa or 13.3 kPa of pressure was applied for a relatively long period.

<table>
<thead>
<tr>
<th>Feature</th>
<th>Baseline SBF</th>
<th>Peak - SBF</th>
<th>Normalized Peak SBF</th>
<th>Perfusion area SBF</th>
</tr>
</thead>
<tbody>
<tr>
<td>Index of reactive hyperemia magnitude</td>
<td>Peak - Baseline SBF</td>
<td>Normalized Peak SBF</td>
<td>Perfusion area SBF</td>
<td></td>
</tr>
<tr>
<td>$R^2$ actual versus predicted</td>
<td>0.075</td>
<td>0.125</td>
<td>0.158</td>
<td></td>
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<tr>
<td>$P$-value for regression slope</td>
<td>0.157</td>
<td>0.064</td>
<td><strong>0.036</strong>a</td>
<td></td>
</tr>
<tr>
<td>Ranks Regressions</td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>$R^2$ actual versus predicted</td>
<td>0.137</td>
<td>0.105</td>
<td>0.149</td>
<td></td>
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<tr>
<td>$P$-value for regression slope</td>
<td>0.052</td>
<td>0.093</td>
<td><strong>0.042</strong>a</td>
<td></td>
</tr>
<tr>
<td>a significant at 0.05 level; SBF = skin blood flow</td>
<td></td>
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of time. These results may indicate that the harmful effect caused by shear stress when added to pressure are not significant when the blood vessels are completely or nearly completely occluded by pressure for a period of time.

A second explanation for the inconsistency between current results and previous studies is that the applied shear force made a contribution to the ischemic state of the tissue at a depth beyond the 1- to 2-mm measurement depth of the laser Doppler probe used in this study. The depth at which shear tends to negatively affect tissue integrity remains an unanswered question in the literature.6

Kottner et al,27 in a review of pressure ulcer definitions and classifications, discussed the importance of factors other than pressure that contribute to decubitus ulcers. In particular, the authors state, “Comparable to pressure forces, deeper structures like muscle and subcutaneous fat are more sensitive and vulnerable than the skin to shear because the skin contains collagen and elastic fibers that provide tensile strength”. On the other side of the argument, Zhang et al16 used a modeling approach to conclude shear contributes to peak stresses in the superficial layers of the tissue much more than pressure. An additional conclusion of this modeling work was that the location of the peak stresses resulting from the applied shear force was immediately ahead of the probe, much like a bow wave ahead of a ship, and the perfusion in this region was not sampled by the method of this study.

The current result appears to be consistent with an early experimental study by Dinsdale13 performed on swine to investigate the relative importance of friction and pressure to pressure ulcer development. The histological results revealed the addition of friction to pressure loading decreased the magnitude of pressure needed to cause both partial- and full-thickness wounds. The study also included an experiment designed to measure blood flow in the skin under the various loading conditions. This experiment revealed the addition of friction did not increase ischemia (ie, decrease blood flow) in the epidermis compared to pressure without added friction. The current observation that shear stress did not increase ischemia, as assessed through reactive hyperemia, is consistent with Dinsdale’s results.

Whether shear stress is a significant contributor to ischemic stress is an important issue and should be the subject of future studies using alternative study methods.

Contribution of skin temperature to tissue ischemia. Finally, current findings indicate post-load metabolic repayment varied with temperature only at higher pressures, perhaps suggesting that decreasing metabolic rate by lowering temperature is primarily effective when blood vessels are completely or nearly completely occluded by high pressure and the resulting deformation. Higher temperatures at nonoccluding pressures may result in temperature-induced vasodilation to offset the higher metabolic rate. Therefore, when pressure deforms the tissue enough to occlude the blood flow, the body’s natural compensatory mechanism to temperature-induced higher metabolic demand, increased flow via vasodilation, is not sufficient to meet this demand, resulting in tissue ischemia.

Limitations

Characteristics of this study that impact the interpretation of results and comparison with other results in the literature are sample size, measurement technique, and load application methods. Because this is a pilot study that investigated the relative contribution of pressure, shear stress, and skin temperature in a systematic manner, the study was only performed on four
healthy volunteers in order to focus to the greatest extent possible on the relationship between the tissue loading effect on ischemia and the extent of reactive hyperemia. In clinical practice, the extent of reactive hyperemia occurring after ischemia also may be affected by individual patient characteristics (e.g., diabetes and vascular diseases) and should be studied.

Measurement technique using LDF facilitated assessment of perfusion in only the top 1 to 2 mm of tissue. Flow responses in deeper tissues were not assessed, which is a limitation of this study model.

Furthermore, the load application method using a rigid indenter may result in a different condition resulting from a typically compliant support surface. Specifically, the device used in this study applied a unidirectional shear stress parallel to the surface that is simpler than the more complicated shear stresses that may occur in the tissue surrounding an irregular bony prominence.

Conclusion

Results of this small, cross-sectional pilot study suggest that addition of temperature measurement allows for more accurate assessment of reactive hyperemia and subsequent potential risk of tissue damage compared to measurement of pressure alone. A small increase (1°C) in skin temperature contributes 12 to 15 times as much to reactive hyperemia (as an indicator of tissue ischemia). In addition, the effects of higher skin temperature are more pronounced at higher interface pressure. Because tissue deformation at weight-bearing areas near bony prominences often results in blood flow occlusion or near occlusion, actively lowering the temperature may reduce the effects of ischemia as indicated by the reactive hyperemic response and lower the risk of developing a pressure ulcer. The results of this study suggest that managing both interface pressure and skin temperature may reduce the risk of pressure ulcer development caused by tissue ischemia. If confirmed in future studies, microclimate control of the skin could become an important consideration for patients at risk of prolonged ischemia.

References