Relative Contributions of Interface Pressure, Shear Stress, and Temperature on Ischemic-induced, Skin-reactive Hyperemia in Healthy Volunteers: A Repeated Measures Laboratory Study

ORIGINAL RESEARCH

Issue Number:
Volume 61 - Issue 2 - February 2015 ISSN 1943-2720

Index:
Ostomy Wound Manage. 2015;61(2):16–25.

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Keywords:
nontherapeutic human experimentation, pressure, Shear, temperature ischemia, reactive hyperemia

Abstract

Although the primary risk factors for pressure ulcer development — pressure, shear, skin temperature, moisture, and friction — have been identified for decades, the relative contribution of each to this risk remains unclear. To confirm the results of and expand upon earlier research into the relative contributions of interface pressures, shear stress, and skin temperature among 4 healthy volunteers, a study involving 6 additional healthy 40- to 75-year-old volunteers was conducted and results of the 2 studies were pooled.

All 3 variables (interface pressures, shear stress, and skin temperature) were systematically and randomly varied. In the prone position, volunteers each underwent 18 test conditions representing different combinations of temperature (28˚ C, 32˚ C, 36˚ C), pressure (8.0 and 13.3 kPa), and shear (0, 6.7, and 14.0 kPa) using a computer-controlled indenter applied to the sacrum for 20 minutes exerting weights of 100 g and 200 g to induce 0.98 N and 1.96 N of shear force, respectively. Each condition was tested twice, resulting in a total of 360 trials. Magnitude of postload reactive hyperemia as an index of ischemia was measured by laser Doppler flowmetry. Fixed

effects regression models were used to predict 3 different indices of reactive hyperemic magnitude. Friedman tests were performed to compare the reactive hyperemia among 3 different skin temperatures or shear stresses under the same amount of localized pressure. In all regression models, pressure and temperature were highly significant predictors of the extent of reactive hyperemia (P <0.0001 and P <0.0001, respectively); the contributions of shear stress were not statistically significant (P = 0.149). With higher temperature, reactive hyperemia increased significantly, especially at greater localized pressure and shear stress, and the difference was more profound between 32˚ C and 36˚ C than between 28˚ C and 32˚ C. These results confirm that, in laboratory settings, temperature is an important factor in tissue ischemia. Additional studies examining the relative importance of pressure, shear, and temperature and potential effects of lowering temperature on tissue ischemia in healthy volunteers and patients at risk for pressure ulcer development are warranted. Because deformation at weight-bearing areas often results in blood flow occlusion, actively lowering the temperature may reduce the severity of ischemia and lower pressure ulcer risk. In this study, shear did not appear to contribute to ischemia in the dermal tissues when assessed using laser Doppler; further work is needed to examine its effect on deeper layers, particularly with regard to nonischemic mechanisms.

Potential Conflicts of Interest: Dr. C. Lachenbruch is employed by Hill-Rom, Batesville, IN.

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Introduction

Pressure ulcers are multifactorial medical conditions that occur in people with disabilities, hospitalized patients, and the elderly.[1-3] Although various studies[4-10] have identified interface pressure, shear stress, and elevated skin temperature as contributors to tissue ischemia, very few have attempted to examine the relative magnitudes of their contributions. The answer to this question has importance for both therapeutic intervention and product design.

The present study is a follow-up to a previously published repeated measures laboratory study that aimed to estimate the relative contribution of pressure, shear stress, and skin temperature by systematically varying all 3 factors over realistic ranges and assessing resulting level of skin tissue ischemia.[11] The initial pilot study[12] presented a model to evaluate the extent to which each variable contributes to ischemia by measuring the magnitude of the postload reactive hyperemia associated with each set of pressure, shear, and temperature loading conditions at the sacrum using laser Doppler flowmetry (LDF). The degree of reactive hyperemia (ie, the transient increase in blood flow to a region that occurs following a brief load period) has been shown in a previous repeated measures laboratory study[13] of healthy participants to be proportional to the degree of ischemia for a given participant. The primary conclusions of the pilot study were: 1)

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pressure and temperature were highly significant predictors of postload reactive hyperemia in the layers of tissue interrogated by laser Doppler and shear stress was not, 2) a 1.0˚ C increase or decrease in temperature has as much effect on reactive hyperemia as an 8–15 mm Hg increase or decrease in interface pressure, and 3) this effect was more pronounced at higher temperatures. The purpose of the current study was to determine the degree to which interface pressure, shear stress, and skin temperature contribute to ischemia in the upper 1–3 mm of the tissue in healthy volunteers between 40 and 75 years of age. The present study was motivated primarily by the need to increase the sample size and to verify and incorporate the findings of the pilot work.

Methods and Procedures

Participants. Ten healthy adult volunteers (5 men, 5 women, ages 40–75 years; 4 from pilot study, 6 for current research) were recruited. They did not have hypertension, diabetes, or any cardiopulmonary or vascular diseases (see Table 1).

Table 1. Participant characteristics

Study phase	Age	Gender	Ethnicity	Weight (lb)	Height (inches)	Body mass index (kg/m²)
1	55	Female	Caucasian	116	62	21.2
1	45	Female	Asian	132	64	22.7
1	51	Male	Caucasian	185	73	24.4
1	57	Male	Caucasian	215	73	28.4
2	54	Female	Caucasian	160	66	25.8
2	66	Female	Caucasian	160	66	25.8
2	66	Female	Caucasian	140	64	24.0
2	67	Male	Caucasian	160	73.5	23.4
2	67	Male	Caucasian	185	68	28.1
2	73	Male	Caucasian	166	67	26.0

Table 2. Trial conditions: shear stress at each combination of temperature and pressure

		Pressure		
		P = 0 kPa	P = 8.0 kPa	P = 13.3 kPa
Temperature	T = 28˚ C	S = 0 kPa	S = 0, 6.7, 14.0 kPa	S = 0, 6.7, 14.0 kPa
	T = 32˚ C	S = 0 kPa	S = 0, 6.7, 14.0 kPa	S = 0, 6.7, 14.0 kPa
	T = 36˚ C	S = 0 kPa	S = 0, 6.7, 14.0 kPa	S = 0, 6.7, 14.0 kPa

Table 3. Fixed effects linear regression

	Coefficient	SD	*P* value	Statistically significant
Peak minus baseline (overall Rsq = 0.285)				
Pressure	0.058	0.009	<0.0001	Yes
Temperature	0.810	0.752	<0.0001	Yes
Shear stress	0.005	0.003	0.149	No
Constant	-25.7	2.52	<0.0001	Yes
Normalized peak (overall Rsq = 0.036)				
Pressure	1.390	0.463	0.003	Yes
Temperature	11.288	3.770	0.003	Yes
Shear stress	-0.119	0.186	0.525	No
Constant	-402.9	126.5	0.002	Yes
Perfusion area (overall Rsq = 0.169)				
Pressure	15.720	2.410	<0.0001	Yes
Temperature	179.990	19.300	<0.0001	Yes
Shear stress	0.962	0.975	0.323	No
Constant	-6177	658	<0.0001	Yes

Design. This study used a repeated measures design. Volunteers each underwent 18 test conditions representing different combinations of temperature (28˚ C, 32˚ C, 36˚ C), pressure (8.0 and 13.3 kPa), and shear (0, 6.7, and 14.0 kPa) applied to the sacrum; each condition was tested twice. A total of 360 trials was conducted. The 18 combinations are listed in Table 2.

Instrumentation. A computer-controlled indenter was used to control the pressure and temperature applied to the skin. A weight was attached to the base of the indenter via a string over a pulley system to create shear force. Weights of 100 g and 200 g were used to induce 0.98 N and 1.96 N of shear force, respectively. A LDF probe located at the center of the indenter measured skin blood flow (SBF). Participants were in the prone position while the tests were performed on the sacrum. Loads were applied for 20 minutes. A figure depicting the experimental set-up and details of the instrumentation and test setting were published previously.[11,14]

Procedure. All procedures took place at the Tissue Integrity Management Laboratory at the University of Pittsburgh, Pittsburgh, PA. All participants signed a consent form approved by the Institutional Review Board at the University of Pittsburgh. Room temperature was maintained at 20˚ C ± 1˚ C. The order of test conditions was randomized for each participant. Each participant initially completed 1 trial of each of the 18 conditions in randomized order, and then completed the second trial of the 18 conditions in a newly determined randomized order. The test trials alternated between the 2 sides of the sacrum, and the location for the initial test was randomized at the beginning of each test day.

Data collection and processing. Data were collected in 2 phases, each approximately 3 weeks in length. Results from the first 4 participants (Phase I) were collected approximately 6 months earlier than the data on the final 6 participants in Phase II. Mean values of all indices did not differ significantly between participants in Phase I and Phase II.

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The raw SBF data were collected at 20 Hz via LDF. It was first downsampled to 0.5 Hz, and a tenth-order Chebyshev I low-pass filter (cutoff frequency at 0.15 Hz) was used to filter the downsampled SBF. Three different indices of reactive hyperemia were selected as the main outcome measure of this study: peak minus baseline SBF (baseline is an average of SBF during the preload period), normalized peak perfusion ([peak-baseline]/baseline), and perfusion area (perfusion integrated over time within the reactive hyperemic spike). To select these parameters objectively, a bi-exponential equation was used to fit the curve of the reactive hyperemic response. The 3 main outcomes measures were calculated from each trial. Average values were calculated for each patient, then regression equations were calculated to fit the curve and determine each parameter as published previously.[11]

Statistical analysis. Fixed effects linear regression analyses were used to determine the contributions of interface pressure, shear, and skin temperature. Because multiple observations were made of each participant (2 replications at each temperature, pressure, and shear condition), the observations were not independent. This required a panel data method using the participant as the panel indicator. The STATA software package (StataCorp LP, College Station, TX) was used to determine the between-estimator for fixed effects models with only pressure, pressure and temperature, and pressure, temperature, and shear in the equation.

Friedman tests were performed on the averaged reactive hyperemic parameters from both trials for each test condition to compare the reactive hyperemic response among different skin temperatures or shear force under the same amount of localized pressure on the skin. The SPSS software package (SPSS for Mac, Version 13, IBM Corp, Armonck, NY) was used for both tests.

A limited validation of the 3 fixed effects regression models was conducted using the raw data set collected previously by Tzen et al.[15] This data set includes the interface pressure and skin temperature data as independent variables and the same 3 dependent variables (measures of the magnitude of reactive hyperemia) used in the present study. The data also were collected using the same instrumentation. The validation process consisted of comparing the predicted magnitudes of reactive hyperemia of each of the 3 indices using the models obtained in the present study to data obtained in the earlier study. Predicted response and actual response were compared using simple linear regression. The normality of the residuals then was evaluated using the Ryan-Joiner test.

Results

Incremental contributions of each variable to regressions. Table 3 presents the statistical variables associated with the fixed effects regressions to predict the 3 postload reactive hyperemia indices. For all 3 dependent variables, temperature and pressure were significant contributors to the prediction of the magnitude of reactive hyperemia. The contribution of shear stress was not a significant contributor in any of the 3 cases ($P = 0.149$). The P value predictors of peak minus baseline perfusion that were significant were pressure ($P < 0.0001$) and temperature ($P < 0.0001$). Similarly, as predictors of normalized peak perfusion, shear

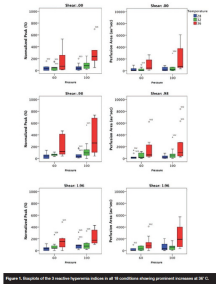
was not significant ($P = 0.525$). Pressure ($P = 0.0003$) and temperature ($P = 0.0003$) were statistically significant. With perfusion area as the dependent variable, the findings were pressure ($P < 0.0001$), temperature ($P < 0.0001$), and shear ($P = 0.323$, not significant).

Coefficient ratios of fixed effects regressions. The ratios of the coefficients in each of the fixed effects regressions reflect the relative contribution of each independent variable to the magnitude of the dependent variable (see Table 4). Because only pressure and temperature are significant contributors for the reactive hyperemia indices, only coefficient ratios of pressure and temperature were calculated. One degree Celsius of temperature contributes as much to peak minus baseline SBF as 14.0 mm Hg interface pressure, as much as 8.0 mm Hg pressure (1.1 kPa) to the normalized peak SBF index, and as much as 11.4 mm Hg pressure (1.5 kPa) to the perfusion area index.

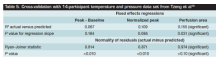
Table 4. Fixed effects linear regression: coefficient ratios

Dependent variable	Temperature (˚ C) / Pressure (mm Hg)
Peak minus baseline skin blood flow	14
Normalized peak skin blood flow	8.1
Perfusion area skin blood flow	11.4

Within-participant comparisons of reactive hyperemia. A prominent trend of increase in reactive hyperemia in all 18 conditions was noted with increased skin temperature (see Figure 1). In all box plots of Figure 1, a smaller difference in the reactive hyperemic response was noted between 28˚ C and 32˚ C than between 32˚ C and 36˚ C. Friedman tests showed the differences in the magnitude of reactive hyperemia were significantly different among the 3 skin temperatures under the same amount of pressure and shear stress, especially at higher pressure in combination with shear stress.



Cross-validation. The fixed effects equations were applied to the data set of Tzen et al,[15] which included pressure, temperature, and all 3 indices of reactive hyperemia; this laboratory study employed a repeated measures design using 10 healthy participants ages 20–40 years. Regressions between the outputs of equations generated in the present study and actual measured indices of reactive hyperemia in Tzen's earlier study resulted in R^2 of 0.155 or less for all 3 indices of reactive hyperemic magnitude. Only the P value for perfusion area was significant ($P = 0.031$); P values for normalized peak and peak minus baseline were 0.085 and 0.184, respectively. Results of the Ryan-Joiner test indicated the residuals (actual minus predicted) were normally distributed for the perfusion area model ($P > 0.10$) but not for the normalized peak ($P < 0.010$) or the peak minus baseline models ($P < 0.010$) (see Table 5).



Discussion

The combined effects of pressure, shear, and temperature loading are important because in a typical clinical situation, they are imposed in combination. These stresses can deform the tissue, distort blood vessels, and interfere with perfusion. Pressure and shear stress require contact between the skin and adjacent bedding or clothing materials. Because these materials tend to be insulative, any time the skin is covered, heat flowing from the body is trapped and the skin warms. When this contact is sustained for several minutes, the skin continues to warm and may approach core temperatures.[16] Therefore, the clinical relevance is that the

skin is most likely to be warmed significantly and subsequently become more susceptible to ischemia when it has been loaded mechanically over a sustained period.

Data from the second phase of the authors' previously published study[11] and current data collected in both phases were incorporated for further investigation of the relative effect of the 3 external factors on tissue ischemia. Both phases employed the same repeated measures design in the laboratory. Participants in this initial phase were 2 healthy men and 2 healthy women ages 40–65 years. Findings from this study were generally consistent with the previously published study. One of the principal results of this study is the observation that the level of ischemia in the skin can be estimated with significantly greater accuracy if both pressure and skin temperature are taken into account rather than by reliance on pressure alone. Both pressure and temperature were highly significant predictors of the magnitude of reactive hyperemia as an index of ischemia.

The relative contributions of 1 mm Hg of pressure and 1˚ C of temperature to ischemia, as quantified by the ratio of the coefficients of the 2 variables in the regressions equations, were relatively consistent. In the fixed effects regressions, 1˚ C contributes 8.0 to 14.1 times as much to ischemia as 1.0 mm Hg depending on the index of reactive hyperemic magnitude, which is similar to the value (1˚ C contributes 7.7 to 14.3 times as much as 1.0 mm Hg pressure) published in the authors' previous study. Findings of this study are also consistent with those of Lachenbruch,[17] who performed secondary analysis of published data and estimated that 1˚ C contributed as much to ischemia as 5 or more mm Hg of pressure. The authors' results also were consistent with those of Iaizzo's repeated measures laboratory study,[18] where histological analysis of affected tissue was performed after imposing a wide range of pressure, shear, and temperature loads to the skin on the back using a swine model. From the tissue results associated with each load condition, Iaizzio was able to quantify the relative effect on tissue breakdown of each variable. His prediction equations indicated that 1˚ C contributes 39.9 times as much as 1.0 mm Hg of pressure for dermis tissue layer. The relative importance of skin temperature based on each prediction equation from different studies varied, and this might be because the relative effect of skin temperature varied at different amounts of localized pressure.

In addition, the current study also demonstrated that under the same amount of localized pressure and shear stress, differences in reactive hyperemia caused by a 4˚ C increase were more profound between 32˚ C and 36˚ C than between 28˚ C and 32˚ C. Further investigation of this phenomenon is needed to find the target temperature that could help preserve the ischemic tissue and be well-tolerated by patients. Of interest would be studies of the degree to which cooling continues to reduce ischemia and be well-tolerated by the patient and whether this reduced ischemia might have beneficial effects not just for prevention, but also for the resolution of early Stage I pressure ulcers or suspected deep tissue injury.

The contribution of shear stress to sacral ischemia remained relatively negligible in the current observation, findings not consistent with previous studies using other outcome measures. Bennett et al[19] conducted a

repeated measures laboratory study of 4 healthy volunteers in which the relative effects of pressure and shear in occluding pulsatile arterial flow in the palm were measured. The authors estimated that shear had approximately one half the effect of pressure in occluding flow. Goossens et al[20] also measured transcutaneous oxygen levels across a series of pressure and shear conditions at the sacrum on a sample of young (mean age 25.5 years) healthy participants. The authors observed a full “cut-off” pressure could be achieved at the sacrum at a mean pressure of 11.6 kPa with no shear or 8.7 kPa and 3.1 kPa shear stress. Zhang et al[21] also studied the effects of pressure and shear on blood flow using a modeling approach supported by laser Doppler measurements of perfusion. Combined pressure and shear loads were applied to the backs of the thighs of 5 healthy volunteers (mean age 37 years). Finite element models of the tissue were used to estimate internal stresses in the tissue beneath the loading apparatus. The researchers concluded the reduction in blood flow in the affected tissue was proportional to the vector sum of the pressure and shear loading on the tissue. This is equivalent to concluding that pressure and shear are equally important. Linder-Ganz and Gefen[22] used finite element models in conjunction with tissue temperature measurements to evaluate the effects of pressure and shear on perfusion of muscle tissue. In the laboratory, various pressure and shear loads were applied to the gracilis (lateral thigh) muscles of 11 rats for 2 hours using a repeated measures design. The authors 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 inconsistency between the current observation that shear seemed to have little effect on tissue ischemia and previous studies may be due to varying measurement of tissue response employed. Current study measurements involved the relatively prolonged physiological effect of skin tissue and vascular deformation using reactive hyperemia instead of the direct effect on blood flow or oxygen reduction with the presence of shear force. The additional blockage of blood flow or the tissue deformation caused by shear force may not be obvious enough when 8.0 or 13.3 kPa of pressure was induced for a relatively long period of time as in the study presented here. It is also possible shear made a more significant contribution to the ischemic state of the tissue at a depth somewhat beyond the 1- to 3-mm penetration of the laser Doppler. The depth at which shear tends to exert its primary effects is somewhat of an open question. Results from the modeling phase of Zhang et al[21] indicated that compared with pressure, shear contributed to peak stresses much more in the superficial layers of the tissue. An additional conclusion of this modeling work was that the location of the peak stress resulting from shear was immediately ahead of the probe, much like a bow wave ahead of a ship. Perfusion in this leading region was not sampled by the laser Doppler method used in the present study, which sampled blood flow directly beneath the measurement probe. Further studies on the relative contribution of shear to tissue ischemia are needed, as well as further investigation to find the target temperature that could help preserve the ischemic tissue and assist in creating the most efficacious support surface designs.

Limitations

One limitation of this study was that the LDF method used allows assessment of perfusion in only the top 1–3 mm of tissue. It is possible different flow responses took place in the deeper tissues that could not be assessed by this method. Additionally, although the results reported here resulted from 360 trials collected over a wide range of clinically relevant pressure, temperature, and shear conditions, they are based on a sample of 10 healthy adult healthy participants.

Conclusion

Because tissue deformation at weight-bearing areas near bony prominences often results in blood flow occlusion or near occlusion, this and other lab study results suggest actively lowering the skin temperature may reduce the severity of ischemia and lower the risk of developing a pressure ulcer. Additionally, because the level of tissue ischemia can be predicted with much greater accuracy when both pressure and temperature and pressure are known, the results highlight the importance of managing both skin pressure and temperature to limit the occurrence of pressure ulcers.

It should be emphasized that because the vascular response to cooling may differ somewhat in patients with diabetes, frailty, or other conditions, these conclusions should be confirmed in patients who are at risk of pressure ulcers and have these comorbidities.

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