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Skin Cooling Surfaces: Estimating the Importance of Limiting Skin Temperature

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Therapeutic Surfaces and Skin Temperature

Clinical studies have shown “good evidence of efficacy” of specialty beds, such as when comparing low-air-loss surfaces to standard foam mattresses, for the treatment of pressure ulcers.[1] However, the proportion of this benefit that can be attributed to skin cooling and the proportion due to moisture removal is not known.

When a patient is placed on a support surface, the skin at the interface becomes insulated and body heat is trapped. Over time, the skin temperature on the low back will rise from approximately 90°F to 92°F (32°C to 33°C), its normal non-insulated temperature in a comfortable environment, to approximately 96°F to 99°F (approximately 35° C to 37°C).[2] Utilizing 90-minute supine trials involving a small four-subject study of women ages 62 to 73 years,[2] the effects on sacral temperature of four support surfaces (standard foam — the control — and low-air-loss, gel, and air-fluidized) were compared (see Figure 1). Air-fluidized and low-air-loss (LAL) surfaces were shown to be effective at limiting skin-warming. A gel surface, like foam, has a low ability to transport heat (ie, low thermal conductivity). Because its heat absorption capacity (volumetric specific heat) is much greater, it takes much longer to heat up and warm the skin, delaying the point at which the skin temperature approaches core temperature; hence, it does not typically provide steady cooling.

The Physiological Effects of Skin Warming

Temperature and moisture. The need to maintain cooler skin temperatures has been argued on several points. First, clinicians generally recognize the importance of limiting both skin warming and moisture accumulation to effectively prevent skin breakdown.[3,4] Second, when a patch of skin is warmed beyond approximately 33°C (depending on core temperature), local perspiration in the region increases markedly.[5] Third, the accompanying moisture softens the skin (maceration) and this makes it more susceptible to breakdown.[6-8] Finally, the build-up of moisture increases friction between the skin and the surface materials, resulting in increased shear stresses in the tissue.[9]

The development of LAL surfaces has been a logical response to address these points. As the skin warms, heat from the body is conducted downward through the mattress ticking and carried away by a moving air stream released from the underlying cushions. Some higher quality LAL surfaces allow for airflow through the ticking as well, which promotes evaporation from (and further cools) the skin. The flow of air that reaches the skin is often limited. (It should be emphasized that the moisture-removal rates of even the top LAL surfaces have been shown to be insufficient for the management of incontinence.[10,11]) Therefore, the desired effect of LAL is to

reduce skin temperatures to limit local sweat production and, to a varied extent, to aid with the evaporation of any perspiration that might accumulate and absorb additional heat.[10,11]

Temperature and tissue metabolic rate. The effect of skin temperature on tissue metabolic rate has received modest attention from clinicians, researchers, and manufacturers of therapeutic surfaces. Because elevated skin temperature is associated with increased metabolic demand of 6% to 13% per degree Celsius,[12,13] it is reasonable to conclude that tissue susceptibility to injury is increased, particularly when both nutrient supply and metabolite removal are reduced by loading.[4,14] This has been demonstrated experimentally.[15,16]

Kokate et al[16] demonstrated this important effect conclusively. In this study, a series of temperature-controlled 51-mm diameter discs were used to apply a constant pressure of 100 mm Hg to the backs of 16 young swine for a 5-hour period. Disc temperatures were 25°C, 35°C, 40°C, and 45°C in 64 sites per temperature (264 sites). After 7 days, the animals were sacrificed and histological samples indicated that the severity of the tissue injury at each site was closely related to the imposed temperature. The results, classified by observers blinded to the treatment, indicated that at 100 mm Hg, 25°C, no superficial or deep tissue damage occurred. At 100 mm Hg, 35°C, significant deep tissue damage and necrosis occurred but no superficial damage was evident. At 100 mm Hg, 40°C, significant deep tissue damage and superficial damage to the skin was noted. At 100 mm Hg, 45°C, significant deep tissue damage and superficial damage occurred and was much more severe than what was observed at 40°C.

Both the severity and the depth to which the injury extended were significantly correlated with temperature. The authors also indicated that results were uniform and reproducible at each temperature. They concluded,

“At a given pressure, … lower temperatures exert a significant protective influence with respect to the development of pressure ulcers.” The authors also made the important point that although therapeutic surface manufacturers have devoted tremendous resources to the reduction of interface pressure, virtually all of these surfaces, with the exception of top-performing LAL and air-fluidized surfaces, generate skin temperatures that probably contribute significantly to tissue breakdown.

From a theoretical standpoint, this study appears to generalize a well-established model of tissue breakdown often referred to as the “Pressure versus Time Curve.” The inverse relationship between the intensity and duration of pressure required to cause tissue breakdown has been demonstrated and quantified experimentally using humans,[17] dogs,[18] and swine.[19] The combinations of pressure and time observed by Reswick and Rogers[17] (see Figure 2) suggest that a pressure of 40 mm Hg would have an approximately 50% chance of causing a pressure ulcer if imposed for 8 hours. If imposed for longer than 8 hours, breakdown would be probable and less than 8 hours, improbable. Similarly, at 80 mm Hg, a 4-hour exposure would likely cause breakdown.

The temperature data introduced by Kokate et al[16] suggest a more generalized form of the pressure-time relation (illustrated qualitatively, see Figure 3) such that the breakdown curve for cooler skin would be farther from the origin and warmer skin closer to the origin.

From a practical standpoint, Kokate’s data strongly suggest that limiting skin warming exerts a protective effect and apparently reduces the tendency for tissue to break down when a given pressure is applied to the skin for a specific period.

Mechanism of protective effect. The probable mechanism behind the previous observations was demonstrated in a study by Patel et al.[20] Using hairless fuzzy rats selected partially because their skin anatomy is similar to humans, the researchers looked at the combined effects of interface pressure and temperature on skin blood flow. In the first series of experiments, mean skin perfusion was measured using laser Doppler flowmetry (LDF) at a constant interface pressure of 3.7 mm Hg at temperatures of 28°C, 30°C, 32°C, 34°C, and 36°C. This low pressure was selected to determine the effect on skin perfusion of increasing temperature when the tissue was nearly unloaded. The results show an increase in perfusion with each temperature increment, suggesting that under this low interface pressure condition, the vasodilatory mechanisms were able meet the increased metabolic demand of the warmer skin tissue.

In a second series of experiments from this study, interface pressure was increased in small steps as skin perfusion was measured (see Figure 4). At temperatures of 28°C and 36°C, significantly greater baseline perfusion at the higher skin temperatures is noted, indicating increased metabolic requirement. At low interface pressures, an increase in perfusion at both temperatures occurred as the pressure was increased. The moderate increase in perfusion observed with mild increases in pressure is apparently due to an active compensatory mechanism functioning to dilate the vessels to help maintain flow under load.[21] However, as the pressure was increased, perfusion dropped off markedly at both temperatures. At pressures beyond approximately 20 mm Hg to 25 mm Hg, mean perfusion at 28°C and 36°C was equivalent at all levels of increased pressure. Because the baseline requirements are greater for the warmed skin, it is logical to conclude that the blood flow deficit and consequent ischemia would be much more severe at the higher skin temperature for any pressure greater than approximately 25 mm Hg. The authors' conclusion was that the vasodilatory response to warming could not occur at the higher pressure because the vessels were compressed and mechanically obstructed from doing so.

This conclusion is also supported by a final series of experiments in the same study where skin perfusion was again measured by LDF at skin temperatures of 28°C and 36°C. The LDF probes simultaneously applied interface pressures to the skin of 3.7 mm Hg, 18 mm Hg, and 73 mm Hg, respectively. The results indicate that warming from 28°C to 36°C caused perfusion to significantly increase at 3.7 mm Hg and 18 mm Hg (see Table 1). No increase in skin perfusion occurred with temperature, however, at 73 mm Hg. This result is identical to that shown in Figure 4. Again, these results were consistent with the authors' conclusion that the vasodilatory response to warming occurred at low to moderate interface pressure but could not occur at the higher pressure due to mechanical compression of the vessels.

Discussion: Estimating the Magnitude of the Skin Cooling Protective Effect Using Published Data

Skin perfusion data. To be of value to surface designers and clinicians who may be faced with design trade-offs, the magnitude of the temperature effect and how this compares to more familiar variables such as interface pressure that tend to drive surface design and selection decisions must be addressed. The data selected for review above can be helpful. Few attempts have been made in the literature to quantitatively compare the effects of pressure and shear[22,23] and few, if any, to compare interface pressure and temperature.

Estimating the combined effect of pressure and temperature on the tissue by redrawing and editing Figure 4 (see Figure 5) provides several insights. The far left data point at each temperature represents the baseline (BL) perfusion required to meet the skin's metabolic requirement at 28°C and 36°C, respectively, when the vessels are free to dilate unencumbered by significant pressure load. These BL requirements were approximately 20.2 perfusion units (PU), labeled BL at 36° in the figure, and approximately 16 PU (BL at 28°). At tissue interface pressures of 40 mm Hg (heavy vertical line), the mean levels of skin perfusion were 13.5 PU and 12.8 PU at 28°C and 36°C, respectively. Therefore, the apparent blood flow deficit at 28°C should be the difference between the baseline flow at this temperature and the flow at 40 mm Hg, approximately 16 - 13.5 = 2.5 PU. At 36°C, the estimated deficit would be 7.4 PU, or nearly three times as great as that at the lower temperature at the same interface pressure.

The same two curves in Figure 6 show the interface pressure at which a deficit of 7.4 PU would occur at 28°C is estimated graphically to be approximately 56 mm Hg — ie, equal perfusion deficits of 7.4 PU occur at 40 mm and 36°C and 56 mm Hg and 28°C. The protective effect of cooling the skin from 36°C to 28°C, therefore, is estimated to be equivalent to reducing the interface pressure from 56 mm Hg to 40 mm Hg, a reduction of 29%.

Skin tissue oxygenation data. A second method of using existing data to estimate the magnitude of the protective effect of cooler skin temperatures is illustrated by published tissue oxygenation data.[24] As previously mentioned, for a 1-degree Celsius reduction in tissue temperature, the metabolic rate and consequent nutrient demand of tissues generally decrease

by 6% to 13%. The consensus number is 10%.[12,13] As a first estimate, a reduction in nutrient demand of 10% for a 1-degree temperature drop at a given level of blood supply should be associated with an approximate 10% increase in tissue oxygenation. Possibly, this number is somewhat lower due to peripheral vasoconstriction but any reduction in flow is clearly less than the reduction in demand; otherwise, increased resistance to ischemic injury with temperature reduction would not be observed.[15,16]

A rough estimate of the magnitude of this deficit can be determined by looking at a series of sacral interface pressure versus tissue oxygenation ($tcpO_2$) curves published by Bader[24] (see Figure 7). Several data sets of this type can be found in the literature; because they are patient- and site-specific, the calculation to follow is a more illustrative than general result. Note that the three curves — S1 at test temperature, S2 at test temperature, and S3 at test temperature — represent results from three different, apparently bracketing subjects S1, S2, and S3 at an unreported skin/indentor interface temperature (test temperature).

Bader's results for the mid-range subject (S2) are shown in Figure 7. Assuming that at sacral interface pressures greater than 25 mm Hg, a 1-degree reduction in skin temperature has resulted in a 10% drop in metabolic demand and consequently the deficit should be reduced by an equal amount, the reduction in tissue oxygenation would be reduced by approximately 10%. Alternatively, if the supply-demand balance is favorably affected by reducing the tissue metabolic rate by 10%, tissue oxygenation should be boosted by approximately 10%. For example, Bader showed sacral skin tissue oxygenation was reduced by approximately 69% at 55 mm Hg, indicated by the intersection of the heavy vertical line with the oxygenation curve designated "test temperature" (see Figure 8). For a reduction in skin temperature of 1°C, the reduction is estimated to be only 62% at the same interface pressure (heavy vertical line intersection with test temperature — 1 degree). The estimates for 2°C and 3°C reductions are approximately 55% and 50%, respectively.

The 69% reduction in tissue oxygenation that occurs at 55 mm Hg at the standard temperature would be expected to occur at 67 mm Hg if the skin were 3 degrees cooler (horizontal red line intersection with test temperature —3 degrees). In other words, oxygenation is reduced by 69% at standard temperature at 55 mm Hg but only by 49% with three degrees of cooling (vertical red line intersection with test temperature —3 degrees). A 49% reduction in oxygenation occurs at the standard temperature and at 47 mm Hg (blue lines). This suggests that for this subject, a sacral interface pressure of 47 mm Hg at standard foam temperature should generate an ischemic deficit equal to what one would observe at 55 mm Hg if the skin were 3 degrees cooler. The magnitude of this protective effect, though clearly patient-specific and subject to a number of assumptions, is 14% for a 3-degree reduction. This is reasonably consistent with the earlier estimate of 29% for 8 degrees of cooling based on the Patel perfusion study.

Also interesting is the observation that the $tcpO_2$ versus interface pressure curves in Figure 7 are linear beyond approximately 20 mm Hg. This is the more important part of the curve with respect to ischemic damage. The linearity suggests that beyond this approximately 20 mm Hg point, for any given subject, the protective effect of a given temperature reduction could be expressed as a relatively constant percentage reduction in interface pressure. The slopes of the lines suggest a greater protective effect with respect to the increase in tissue oxygenation for a given amount of cooling for the most vulnerable subjects — ie, those with steep reduction in $tcpO_2$ versus pressure.

Design considerations. Multi-subject interface pressure comparisons have been conducted of a dozen or more surfaces as a reference point. In a recent analysis of nearly 1,000 pressure maps from more than a dozen surfaces, one of the parameters of interest was peak sacral pressure (data on file with the author). The most expensive surfaces were nearly 100 times as costly as the least expensive. Yet the difference between the best and worst performers in peak sacral interface pressure was 22.8%. Based on the estimates above, it appears as though a surface capable of achieving 5°C of cooling relative to an equilibrium skin temperature on foam might be able to exert a similar protective effect with respect to tissue ischemia Similarly, different cooling techniques can be combined to allow for design flexibility.

Conclusion

Using simple graphical techniques in conjunction with laser Doppler and tissue oxygenation data from the literature, the magnitude of the tissue “protective effect” that has been shown to be associated with mild skin cooling was estimated. Estimates using laser blood flow results in rat skin suggest that an 8-degree Celsius reduction might be equivalent to that provided by a 29% reduction in interface pressure. Using sacral tissue oxygenation data obtained from humans, the estimate was 14% for 3 degrees. Because of the consistent results, the estimates seem reasonable and could have profound implications for surface design, surface selection, and patient care in hospital and nursing home environments.