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Erythema and skin temperature following continuous sitting in spinal cord injured individuals

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Abstract—Pressure sores are a severe and costly problem for many disabled individuals. There is a need for quantitative tools to assess damage produced by external loads on human skin and underlying tissues. Clinically, intensity and size of skin erythema have been used as indicators of tissue damage. Temperature is a quantifiable measure, and various studies have investigated the thermal response to localized pressure. The purpose of this study was to measure the effect of "long-term sitting" on skin temperature and erythema, in a situation that closely approximated what a spinal cord injured individual encounters on a regular basis. The resulting data indicated that: 1) a consistent skin temperature pattern occurred after pressure relief from the seated position; 2) skin temperature of experimentally-induced erythematous areas often remained elevated, even after one hour of pressure relief; and, 3) a qualitative, but not quantitative, correlation exists between erythema size and intensity and skin temperature. Implications of this research include the potential use of temperature to: 1) monitor the effectiveness of various strategies being used to prevent the development of pressure sores; and, 2) predict incipient tissue damage.

Key words: *erythema, hyperemia, pressure sores, skin temperature, spinal cord injury.*

INTRODUCTION

Ulceration of the skin and deeper tissues can occur when external forces are applied over a period of time without relief (3). Numerous etiologies have also been implicated in the formation of such ulcers which are typically referred to as pressure sores, bed sores, decubitus ulcers, and ischemic sores. They are a severe and costly problem for many disabled individuals (12).

Better indicators and improved interventions for pressure sore prevention are required. In this respect, there is a need for quantitative tools to assess the damage produced by external loads on human skin and underlying tissue. A common clinical indicator used in skin assessment of the load-bearing surfaces of wheelchair users (buttocks, greater trochanter, sacrum, etc.) is erythema or skin redness (7). Erythema is described both in terms of the intensity and size of the involved area.

A more quantifiable measure is temperature. Several studies have investigated the thermal response of tissue subjected to localized pressure. Mahanty, *et al.* (17,18), noted a transitory skin temperature response at the pressure application area which increased to a maximum within 2–5 minutes after release of the pressure. The peak temperature rise was noted to be proportional to the magnitude and duration of the applied pressure. Patterson and Fisher (21) studied pressure and temperature patterns in wheelchair users throughout the activities of a normal day. Temperature increased while sitting, but decreased rapidly with pressure relief. Rogers noted that skin temperature elevation persisted at sites subjected to continuous pressures of 60 mmHg for 3 or more hours (23). While these areas did not go on to actual breakdown, the clinical appearance of the traumatized areas suggested impending skin necrosis. Vistnes found that areas subjected to repetitive stress may

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show temperatures 5 degrees Centigrade or more higher than similar unstressed areas and that differences in temperature of as little as 1 degree Centigrade can be clinically significant (24). Thermography has also been studied as an indicator of tissue damage in a variety of other applications (2,6,14,15,19,22).

The study presented here evaluated spinal cord injured subjects seated for 1 to 4 hours without pressure relief. Our purpose was to: 1) examine the effect of long-term sitting on the skin temperature of the affected area; and, 2) correlate the skin temperature recordings to clinically determined degrees of erythema. Furthermore, our intent was to develop a characterization of the tissue response to prolonged loading as a basis for comparing various interventions for pressure sore prevention. Specifically, we have been studying the use of electrical muscle stimulation for pressure sore prevention and are using the trials presented here as controls in a study of the efficacy of this intervention.

Our hypothesis was that surface temperature recordings would be affected by continuous loading, reflecting a certain level of tissue damage, and that they would correlate with clinical measures of erythema.

To the best of our knowledge, this study is the first time a report has been written where these two parameters have been concomitantly measured and analyzed in a situation closely approximating what the spinal cord injured patient encounters on a regular basis.

METHODS

Six Caucasian spinal cord injured individuals were studied. All subjects had a complete or very minimally incomplete motor and sensory paralysis, at or above the T8 level. Onset of paralysis was less than 6-months' duration at the time of participation in the study. None had a history of pressure sores under the ischial tuberosities. Black patients were not studied, because preliminary investigations indicated that the subsequent skin erythema evaluation could not be reliably performed. **Table 1** summarizes relevant clinical data concerning the six patients.

A "Critical Sitting Time" (CST) was established for each subject. The CST was defined as the minimum sitting time at which erythema persists for at least one hour after pressure relief. The erythema, however, could not persist beyond 24 hours of rest. The purpose of the CST was to induce a prolonged erythema, reflective of more than just the phenomenon of reactive hyperemia, but which also did not produce longer-term ischemic tissue damage.

Table 1.
Sex, Spinal Cord Injury Level, Height, Weight, Age, and Critical Seating Time (CST) for each of the six subjects.

Subject No.	Sex	SCI Level	Weight (lbs.)	Height (in.)	Age (yrs.)	CST (hrs.)
1	Male	T8	147	65	30	1.75
2	Male	C6	149	68	20	2.50
3	Male	C5	158	68	29	2.50
4	Male	C7	122	68	18	2.00
5	Female	C6	98	65	30	2.25
6	Male	T2	165	73	23	4.00

The weight shown is an average of weights taken over the trial period.

Critical seating times for all subjects are displayed in **Table 1**.

The CST was also conceived as an attempt to reduce the effect that other factors may have on the immediate production of erythema after pressure relief. These other factors include (among many others) degree of atrophy, buttock shape, skin composition, and body weight.

The minimum of a 1-hour period of erythema was chosen empirically, based on clinical experience with spinal cord injured patients, as well as on previous studies which defined: 1) redness from hyperemia of the skin as that which disappears within one hour after pressure is removed; and, 2) redness from ischemic tissue damage of the skin as that which requires 36 hours to disappear (13).

Determination of the CST began with a trial seating session of 30 minutes. A vinyl-covered 2-inch layer of high-density foam over plywood was provided as a standardized seating surface. The subject was not allowed to perform any pressure relief maneuvers while sitting in the wheelchair. The sitting duration was increased with each session by half-hour increments until erythema persisted for one hour. The CST was established when this result was repeated in two consecutive trials.

Trial sessions involved assessing the subject immediately after sitting for the CST without pressure relief. With the patient in the side-lying position, each side of the buttocks was visually graded on a predetermined 1–10 redness intensity scale, and the maximum and minimum diameters (a and b) of the erythematous area over the ischial tuberosity were measured bilaterally. The area of erythema was then calculated as $1/4\pi ab$. The intensity scale was based on a color chart using five color samples. Colors were chosen based on clinical experience from prelimi-

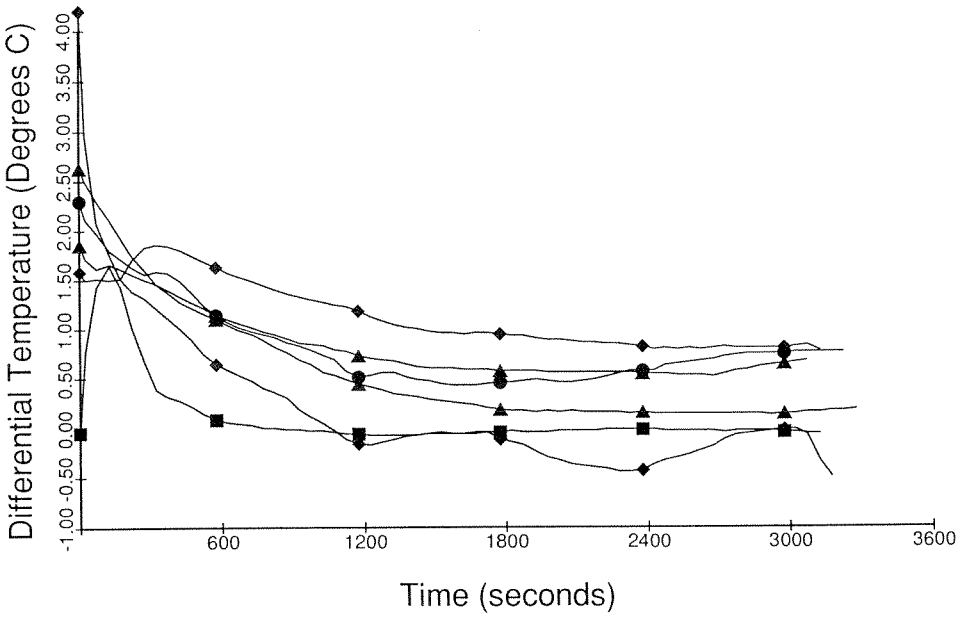

Figure 1.
Plot of differential temperature versus time. (Data from one trial for each of the six subjects are displayed.)

nary investigations. The odd numbers on the scale (i.e., 1, 3, 5, 7, 9) represented erythema intensities falling in between the even-numbered color samples.

Immediately thereafter, thermistor probes were placed over the ischial tuberosity at the site of highest intensity erythema with one probe directly on the erythematous area and four additional probes spaced around it radially at a distance of six centimeters. The one-centimeter-in-diameter thermistors were attached to the body using two-sided adhesive tabs. This method was a modification of that described by Mahanty, *et al.* (18). The thermistors were interfaced to a microcomputer system, and the temperatures from all probes were recorded in memory at a rate of one per second for 60 minutes after the seating session. The differential temperature was the key parameter studied and was calculated as a function of time by:

$$T_D(t)=T_1 - T_{2\text{-}5}$$

where T_D is the differential temperature, T_1 is the temperature at the center of the erythematous area, and $T_{2\text{-}5}$ equals the average temperature of the four surrounding probes. It was not possible to control environmental temperature beyond the conditions normally employed on the inpatient unit, where the trials took place. This was not felt to be a limiting factor given that only temperature differentials between nearby locations (as opposed to absolute temperatures) were used throughout the data analysis.

At the end of one hour, the skin was again visually graded on the 1–10 scale and the erythematous area was measured bilaterally. Results reported here include two CST trial sessions for five of the subjects, and one CST trial for the sixth. Regression analyses were performed on the erythema and temperature data to relate changes in erythema size and intensity to changes in the T_D.

RESULTS

Temperature

A consistent T_D pattern, in terms of graphical shape and T_D decline, was obtained both between Trial One and Trial Two and each of the six subjects tested. **Figure 1** shows a representative graph of continuous 1-second T_D determinations versus time for one trial of each of the six subjects. The wavy lines reflect the slight variations of temperature that were present over time. The temperature differential starts at an elevated level and typically declines over time. In nine of the eleven trials, the temperature differential remained above zero degrees Centigrade at the end of one hour, and in eight of the eleven trials the differential temperature was level or rising at the end of the hour. The peak temperature differential varied between 0.4–3.35 degrees Centigrade, and occurred 1.5 to 5 minutes after the thermistors were applied (which took approximately 3 minutes).

Erythema duration, area, and intensity

The size of the erythematous area decreased in all subjects between the first inspection immediately after pressure relief and the second inspection one hour later. The range of the area decrease was from 24 percent to 100 percent. The intensity of the erythema also decreased by 1

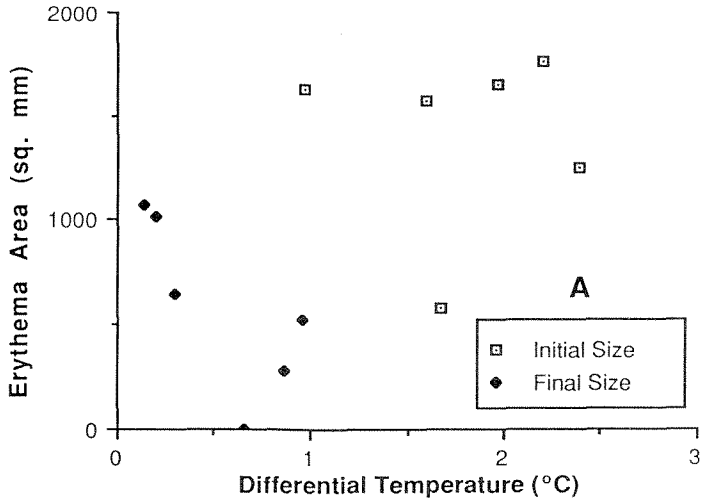

Figure 2.
Erythema area (sq mm) versus differential temperature (degrees C).

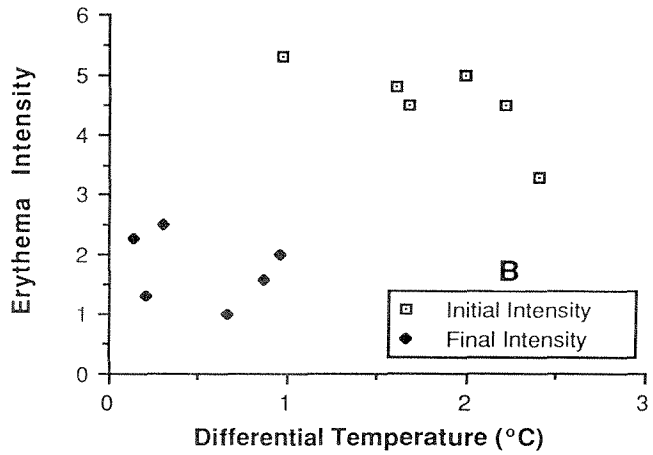

Figure 3.
Erythema intensity (1–10) versus differential temperature (degrees C).

to 4 units on the predetermined 1–10 scale between the two inspections (**Figure 2** and **Figure 3**).

Correlation between erythema and temperature

Despite the fact that after 60 minutes of observation a decrease in the T_D and the area and intensity of the erythema was noted, linear regression analyses performed on the erythema and temperature data showed that no linear relationship existed between: percent change in area and percent change in T_D (r^2=0.007); change in area and change in T_D (r^2=0.016); change in intensity and change in T_D (r^2=0.173); size and T_D (r^2=0.083); or intensity and T_D (r^2=0.150).

DISCUSSION

Establishing a relationship between an indicator of tissue damage and pressure sore formation is difficult in the real world environment. This is due to the fact that the incidence rate of pressure sores and length of time involved makes general population studies difficult to perform. Previous studies have looked indirectly at the relationship between temperature and tissue damage by relating temperature with the magnitude and duration of external load (17,18). The experimental protocol of this study measures temperature under circumstances which approximate the actual situation leading to pressure sore formation in spinal cord injured individuals. Also, the intensity and duration of erythema, and the apparent sequelae of external loading are used as measures of tissue damage, and are compared with temperature. This is in contrast to measuring external load and temperature, and thereafter assuming a relationship between loading conditions and tissue damage.

A notable finding was the length of time T_D remained elevated (it did not return to baseline after one hour in nine of eleven trials). Measurement of the T_D in subjects prior to a CST trial indicated that it is very close to zero degrees Centigrade (after prolonged rest in the side-lying position), as would be expected. Also, in eight of eleven trials, T_D reached a minimum and then leveled off or even increased a small amount over the hour following the CST. The implication is that tissue changes are occurring even beyond an hour as a consequence of the CST trial.

The principal cause of the transient temperature rise occurring after pressure application is believed to be a result of the phenomenon called reactive hyperemia. This phenomenon has been noted for many years and refers to the increased blood flow that occurs at skin and deeper tissue levels upon the release of an obstruction to the circulation (5,16). Holloway (8), studying the effect of local pressure application on skin blood flow, observed a reactive hyperemic effect upon pressure release for pressures of 90 mmHg or larger, applied for 90 seconds. Most of the numerous studies on reactive hyperemia (5,9,16,20), however, involve total occlusion of blood vessels with subsequent release of the occlusion. Occlusion times are far shorter (typically less than 15 minutes), than the length of time our spinal cord injured subjects sat in their wheelchairs without pressure relief. The duration of the hyperemic response in these previous reports are also shorter as compared to the present study.

Theories concerning the mechanisms involved in the

long-term temperature change noted in our experiment include: 1) a prolonged period of reactive hyperemia occurring as a consequence of a local tissue phenomenon occurring in skin and subcutaneous tissues and independent of central nervous system, local reflex, or hormonal control (16); 2) a different type of local hyperemia, not associated with the initial reactive hyperemia (i.e., increased capillary flow in underlying tissues); or, 3) a less-likely unidentified phenomenon not associated with hyperemia at all (i.e., increased tissue metabolism or a mild inflammatory response).

The fact that linear regression analysis did not show a statistically significant linear correlation between skin redness and temperature is not surprising. Kosiak noted that skin ulceration can occur days after initial erythema had disappeared (10,11). Goller, *et al.*, noted that there may be "no correlation in the color of the skin and temperature of the skin," due to the fact that the former reflects capillary flow, the latter arteriolar flow, and the two can be quite distinct (6). This concept, however, is not widely accepted. Another possibility is that the thermistors may be detecting heat or increased blood flow from below the skin surface (e.g., at the muscle level), and this fact may partially explain the lack of a quantitative correlation.

CONCLUSION

In summary, temperature and erythema measurements were performed on spinal cord injured patients after sitting for 1 to 4 hours without pressure relief. The data indicate that: 1) a consistent temperature pattern occurs; 2) most of the erythematous areas induced on the skin did not return to zero degrees Centigrade even after one hour of observation; and, 3) a qualitative, but not quantitative, correlation exists between erythema size and intensity and skin temperature.

Implications of this research include the potential use of temperature recordings to: 1) monitor the effectiveness of various strategies being used to prevent the development of pressure sores (e.g., wheelchair cushions, electrical muscle stimulation); and, 2) predict incipient tissue damage. This second point has been studied on an empirical basis (2), but requires further study. Future work might include the use of multiple probes localizing the exact tissue sites of temperature elevation to help further elucidate the relationship between pressure, erythema, and temperature in the etiology of pressure sores.

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