

ORIGINAL INVESTIGATION

Biophysical Effects of Water and Synthetic Urine on Skin

Harvey N. Mayrovitz, PhD, and Nancy Sims, RN, LMT, MLDT

Pressure ulcers commonly occur on areas subjected to pressure and exposed to increased wetness from perspiration and urine.¹⁻² However, the role of wetness compared with the wetting substance's composition in the skin breakdown process is unclear. It has been stated that skin integrity is compromised by dampness, somewhat independently of the source of the wetness.³ This view is consistent with findings reported for the role of urinary ammonia in infant diaper dermatitis.⁴ Experimental animal work and some pediatric findings suggest interactions between urine and fecal enzymes that promote skin damage.^{1,5} Because interactions between urine and fecal enzymes depend on breakdown of urinary urea, such an effect would seem to depend on the presence of urine. Some of the main extrinsic factors involved in diaper dermatitis include skin wetness, urine and its components, feces and its components, and skin pH, and projections for their role in adult perineal skin injury have been summarized in the literature.² No doubt, there are interactions among these constituents and others, such as skin temperature,⁶ that are yet unknown factors. It is known, however, that wetting of skin with water or urine increases skin permeability to noxious substances⁷⁻⁸ and this altered skin barrier function likely plays some role in the breakdown process.

How wetness may affect mechanical properties of skin and underlying tissue has not previously been addressed. A pos-

ABSTRACT

OBJECTIVE: Pressure ulcers often occur at sites subjected to pressure and wetness. Although skin wetness is a risk factor for pressure ulcers, the mechanisms and effects of wetness versus urine constituents on skin breakdown is unclear. The hypothesis that wetness reduces skin hardness and, thereby, increases vulnerability of underlying blood vessels to pressure-induced flow reductions was tested in this study.

DESIGN: Pads saturated with water and with a water solution mixed with the main chemical constituents of urine (synthetic urine; s-urine) were applied to forearm skin of 10 healthy subjects for 5.5 hours. Skin hardness, blood flow change caused by 60 mm Hg of pressure, erythema, and temperature were compared among dry, water, and s-urine test sites.

PARTICIPANTS: 10 healthy women.

SETTING: Research Center, Nova Southeastern University, Health Professions Division, Fort Lauderdale, FL.

MAIN RESULTS: S-urine and water caused significant reductions in initial hardness and caused greater initial perfusion decreases during pressure load when compared with dry sites. Skin temperature and erythema were lower at wet sites when compared with dry sites.

CONCLUSIONS: The findings of this study are consistent with the concept that sustained skin wetness increases vulnerability to pressure-induced blood flow reduction. The effect appears to be mainly dependent on wetness, but urine constituents may exacerbate the effect. In addition, wetness-related skin cooling may play a role. In the healthy subjects studied, the blood flow decrease was not sustained due to perfusion recovery under pressure. Skin wetness would likely have more sustained effects in patients with compromised recovery mechanisms. Measures to diminish skin exposure to wetness in these patients, whatever the wetness source, are an important consideration in a multifaceted strategy to reduce the risk of pressure ulcers.

ADV SKIN WOUND CARE 2001;14:302-8

sible mechanical connection between skin wetness and pressure effects is suggested from an almost 90-year-old observation that pressure required to compress nailfold capillaries is reduced when skin is moist rather than dry.⁹ This phenomenon may

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Support for this study was provided by a Nova Southeastern University, Health Professions Division Research Grant.

Submitted March 6, 2000; accepted in revised form May 26, 2000.

be attributed to a reduction in the moist skin's stiffness and less elastic resistance to oppose a pressure load. A natural extension of this observation is that this mechanical process may render blood perfusion of moist tissue more vulnerable to pressure-induced blood flow deficits. Whether this effect would depend on skin wetness alone or the wetting agent's constituents is unknown. The primary purpose of this study was to investigate the relative effects of water versus a water solution containing the main chemical constituents of urine on skin tissue hardness, erythema, temperature, and blood perfusion changes caused by a standardized local pressure load.

METHODS

Subjects

Ten female volunteers (28.8 ± 2.8 years) selected from the School of Pharmacy participated in the study after signing a consent form approved by the Institutional Review Board. To participate in the study, volunteers had to be at least 21 years of age and in generally good health. Subjects were not eligible for the study if they (1) had a history of arm vascular abnormalities or disease, (2) were taking any medication or substance affecting blood vessels, (3) were pregnant or possibly pregnant, (4) had known allergies to any substance, (5) had an open skin area on their arm, or (6) had diabetes. No attempt was made to test these subjects on a constant day of their menstrual cycle.

Procedures

Subjects wore clothing with short sleeves to the laboratory. Four 1.5-inch diameter pads were placed on their volar forearms and covered with water-impermeable transparent dressings. Two of the pads were saturated with 6 mL of fluid: 1 was saturated with plain water and the other with synthetic urine (s-urine). S-urine formulations have been used for many applications with several variants of its

constituents.¹⁰⁻¹³ The formulation used in the present study was derived from these and included constituents in grams per liter of sterile water. The s-urine formulation used in this study included 25 g urea; 9 g sodium chloride; 2.5 g disodium hydrogen orthophosphate, anhydrous; 3 g ammonium chloride; 2 g creatinine; and 3 g sodium sulfite, hydrated. The solution had a pH of 7.8.

One wet pad and 1 dry control pad were placed on each arm. The proximal edges of the wet pads were placed approximately 4 cm distal to the antecubital space. The dry control pads were placed distal to the wet pads such that their proximal edges were about 2.5 cm from the distal edges of the wet pads. Subjects were then excused to attend classes. They returned about 5 hours later for testing. Overall, the pads were left in place for 325 ± 15 minutes.

Blood perfusion measurements

Pads were removed in random order and biophysical measurements were taken at each test site. After pad removal, a standard right angle laser Doppler probe (Vasamedics, Minneapolis, MN) was taped to the center of the test site. Principles of operation have previously been described.¹⁴⁻¹⁵ The probe transmits a low-intensity laser light signal into the

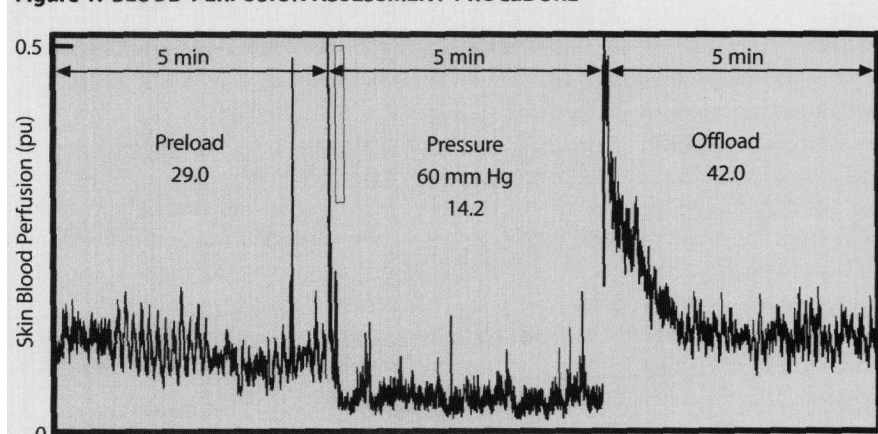
skin to a depth of about 1 mm and the reflected light is used to measure local blood perfusion through the laser Doppler. The Doppler-shifted signal contains information about the speed and density of moving red blood cells in a tissue region to a depth of 1 mm to 2 mm. Speed and density information is processed to yield a parameter perfusion that is proportional to blood flow.

Perfusion was measured and recorded continuously at each site in sequential order for 15 minutes, divided into 3 5-minute intervals. The first interval was during the preload baseline period, the second was during tissue loading, and the third was after load removal (Figure 1). For convenience, blood perfusion is expressed as the interval's flow integral calculated by the product of the perfusion multiplied by time. The data were processed off-line. Average laser Doppler perfusion were obtained in perfusion units (pu) by dividing the flow integral by 300 seconds.

Tissue loading

The tissue was loaded for 5 minutes with a weight of 258 grams placed on the laser Doppler probe (diameter = 2.0 cm). The weight was a solid brass rod that had its contact cross-section machined to fit directly on the probe. The rod lay flat

Figure 1. BLOOD PERFUSION ASSESSMENT PROCEDURE



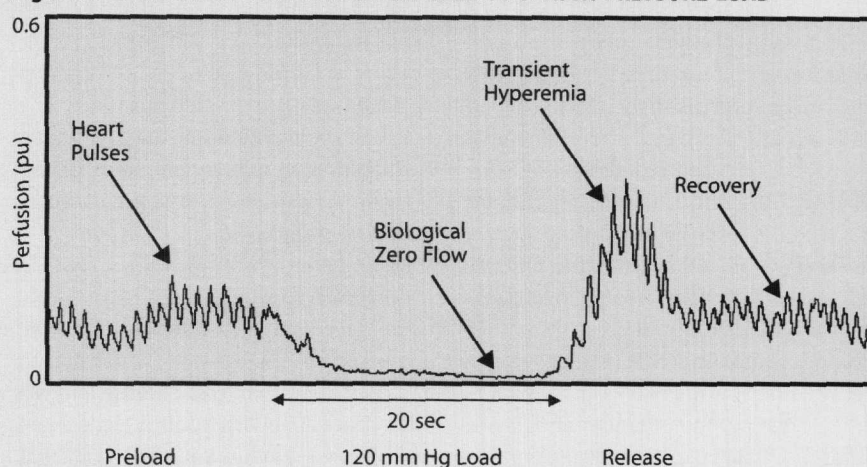
against the portion of the probe in contact with the skin and was near self-supporting during the 5-minute loading interval. The experimenter manually minimized any slight movements away from the probe's vertical 90° position through light finger pressure. The skin surface pressure level was confirmed through measurements of the interface pressure using a pressure measurement system. Based on previous work using a similar loading procedure,¹⁶⁻¹⁷ it was reasoned that if substantially larger pressure were used, differential effects due to wetness may be masked. Typically, when loads in excess of arterial pressure are used, flow is reduced to a biological zero value (Figure 2). The pressure load used in this study was standardized to 60 mm Hg.

Tissue hardness

Tissue hardness was measured at each test site prior to pad placement and after removal with a handheld durometer (Model 1600-OO; Rex Gauge Company, Buffalo Grove, IL). The device weighs about 6 ounces and records hardness in relation to the deformation resistance encountered by a small, 3/32-inch, spherical, spring-loaded indenter that is in contact with the skin and coupled to a calibrated analog meter with a range from 0 to 100 points. The higher the reading, the harder the material being tested. Durometers are calibrated according to engineering standards (American Society for Testing Materials D2240) and the type used in this study (Type OO) is optimal for human skin work. A similar durometer device has been used to measure skin hardness of patients with scleroderma.¹⁹ For this device, units of measure are calibrated in durometer points and are dimensionless.

Because of a process called creep, in which skin and underlying tissue deform slowly with time during loading,^{18,19} the initial skin hardness reading is usually the highest value. Some reduction in skin

Figure 2. EXAMPLE OF PERFUSION RESPONSE TO A HIGH PRESSURE LOAD



If a high pressure is used to load the tissue, the skin blood perfusion is invariably reduced to a very low level, referred to as the biological zero. In the example shown, the blood flow pulsations in synchrony with the heart beat are clearly seen before the 120 mm Hg load is applied. During the pressure loading interval, these pulses are rapidly lost. When the pressure is released, there is usually a transient hyperemia, a return of the pulses, and a normalization of pulsatile and mean blood perfusion during recovery.

hardness is seen with time. Previous work has shown that most or all of the change in skin hardness is complete in less than 30 seconds in a healthy arm.¹⁸ This was confirmed in preliminary tests of hands conducted by the investigators. In the present study, the hardness measurement was taken for 60 seconds and the initial and final hardness measurements were separately analyzed.

Erythema and temperature

Erythema was measured at each test site before pad placement and after removal using an erythema meter (Dia-Stron, LTD, Manchester, United Kingdom). This device illuminates the skin with a tungsten-halogen white light via a fiberoptic cable and probe and then collects the diffusely reflected light from the skin. The amount of energy reflected at 546 nm, the peak absorption wavelength for hemoglobin, is inversely proportional to the amount of hemoglobin. A second reflected wavelength of 671 nm is used as a reference signal to compensate for skin tone, probe alignment, and other factors.

From the reflected energy at these 2 wavelengths, an erythema index is determined that is linearly related to the amount of hemoglobin concentration or degree of redness. Six erythema measurements were made in rapid succession for each test site. These were accomplished by placing the probe in gentle contact with the skin, recording the erythema index, removing the probe, and repeating the sequence 5 more times. Each measurement was made at a different spot within the test site. It took about 2 minutes to acquire the 6 measurements. The average of these 6 measurements was used to represent the erythema index of each site.

Skin temperature was measured in the central region of each site after pad removal with a fast-response thermocouple. Temperature measurement was taken with a standard surface temperature probe with a 1-cm diameter contact area.

Statistical analysis

Statistical comparisons were based on the nonparametric Wilcoxon signed rank

test for paired differences with a significance level of $P < .05$. Data are presented as mean \pm standard error of the mean.

RESULTS

Skin exposure to s-urine and water caused reductions in initial tissue hardness of -4.0 ± 0.8 durometer points for s-urine sites and -3.5 ± 0.9 durometer points for water sites ($P < .01$; Figure 3). A significant reduction was maintained after 1 minute in skin exposed to s-urine (-2.6 ± 0.6 durometer points, $P < .01$).

Significant reductions in skin erythema were seen in sites exposed to s-urine, -11.1 ± 5.6 Erythema Units (EU), and water, -18.3 ± 7.0 EU ($P < .05$; Figure 4). Differences between sites exposed to s-urine and water were not significant. After exposure, there was a tendency—although not significant—for erythema levels to increase at each of the corresponding dry control sites. Erythema changes were $+9.8 \pm 5.3$ EU at the control site on the s-urine arm and $+6.2 \pm 3.6$ EU at the control site on the water arm.

Skin temperature comparisons were measured approximately 5 minutes after pad removal. Sites exposed to s-urine and water had reduced temperatures when compared with their corresponding dry control sites. The temperature differential was $-1.2^\circ\text{C} \pm 0.3^\circ\text{C}$ for s-urine sites and $-1.5^\circ\text{C} \pm 0.3^\circ\text{C}$ for water sites ($P < .01$).

Prior to loading, there were no significant differences between wet and dry site average perfusions on arms exposed to s-urine or water. For the arms exposed to s-urine, the 5-minute perfusion integral for the wet sites was 30.4 ± 6.2 (pu \times min) compared with 39.2 ± 11.4 (pu \times min) for the dry control sites. On the arms exposed to water, the 5-minute perfusion integral was 18.8 ± 3.8 (pu \times min) for the wet sites and 22.6 ± 2.0 (pu \times min) for the dry control sites.

There was considerable variability among the sites in response to local pres-

sure loading (Figure 5). Based on the perfusion integral during the 5-minute load interval with 60 mm Hg, the s-urine sites showed an overall mean perfusion decrease of -5.1 ± 5.9 (pu \times min) and water sites showed a perfusion decrease of -2.8 ± 2.5 (pu \times min) when compared with their preload perfusion. Similar variability was found for each of the dry con-

trol sites during the same interval. For wet and dry sites, 60% of responses to local pressure loading resulted in a perfusion integral increase during the loading interval; perfusion decreased in the remaining 40%. There was no detectable correlation between the amount of perfusion change produced by loading and skin hardness or erythema changes

Figure 3. SKIN HARDNESS CHANGES

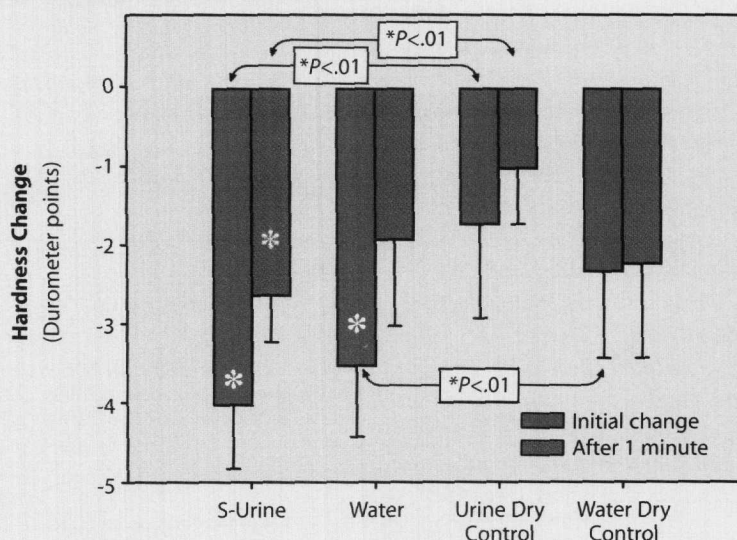
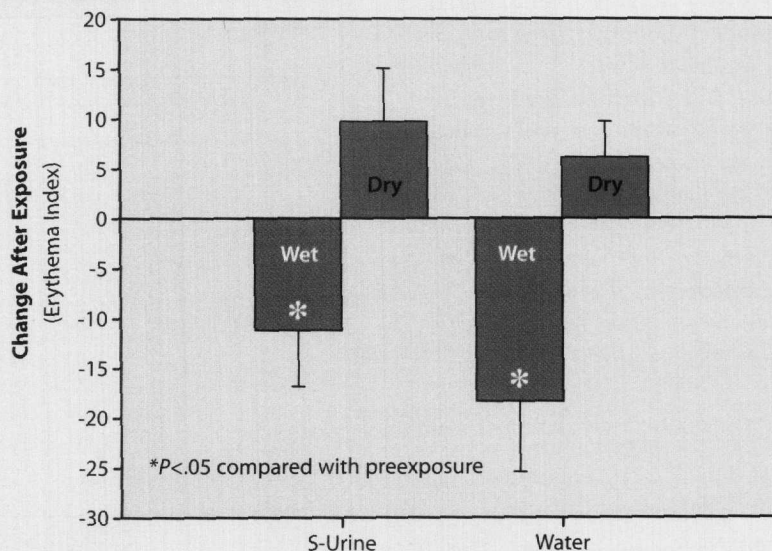


Figure 4. SKIN ERYTHEMA CHANGES



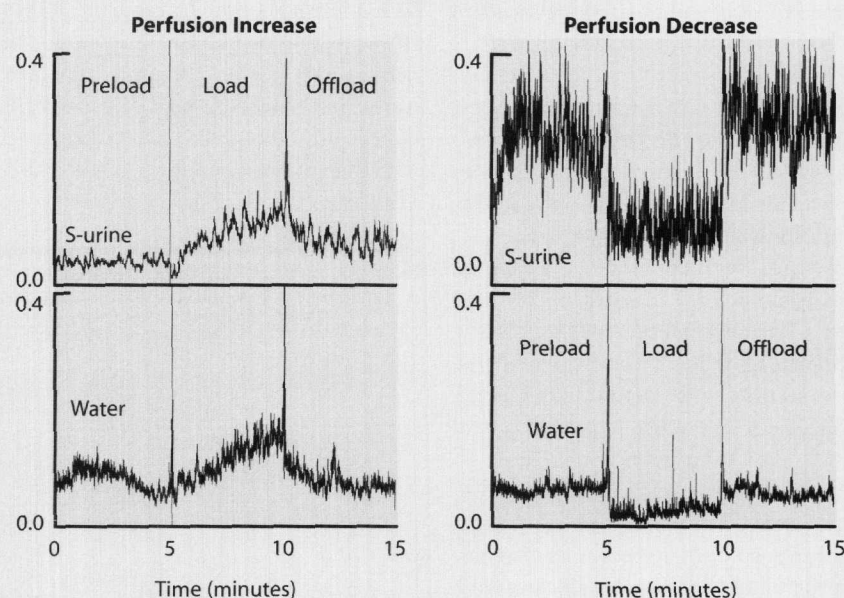
caused by water or urine exposure.

To determine the early effects of pressure loading on blood perfusion that were reasoned to be unaffected by subsequent flow recovery, the minimum perfusion during the first minute of loading was compared with the minimum perfusion during the last minute prior to loading. The comparisons were conducted using absolute perfusion change and the percentage of the preload values. Results showed that absolute and percentage reductions at sites exposed to s-urine were significantly greater than their corresponding dry control sites. The absolute flow reductions were -0.04 ± 0.009 pu for s-urine sites and 0.01 ± 0.007 pu for their corresponding dry control sites ($P < .05$). The percentage reductions were $-59.2\% \pm 5.2\%$ for s-urine sites and $-21.1\% \pm 16.9\%$ for their corresponding dry control sites ($P < .01$). Changes for sites exposed to water tended to be less, but not significantly different, from sites exposed to s-urine (0.03 ± 0.01 pu and $-49.0\% \pm 12.1\%$). Figure 6 summarizes the difference in initial flow responses.

DISCUSSION

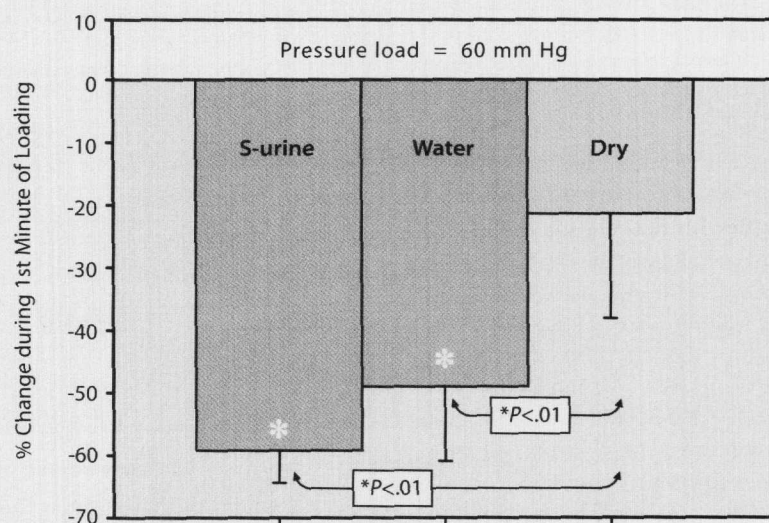
Pressure ulcers are a significant problem for some elderly bedridden individuals and persons recovering from surgical procedures.²⁰ Common sites for these ulcers are areas subjected to combined pressure and increased wetness from urine, perspiration, and other sources. The main focus of this study was to investigate the possible role of skin wetness and the wetting agent's constituents on parameters that affect skin breakdown. Specifically, it was hypothesized that sustained wetness would reduce tissue resistance to pressure loading, rendering underlying blood vessels more vulnerable to pressure-induced blood flow deficits. In fact, skin wetness maintained for approximately 5.5 hours caused significant reductions in initial skin hardness of forearm skin exposed to

Figure 5. VARIABILITY IN BLOOD PERFUSION RESPONSE TO PRESSURE LOADING



These examples illustrate the variability in blood flow adaptation that occurs under pressure loading. Results from 2 subjects in the left panel demonstrated an actual progressive perfusion increase during the loading interval. Results from 2 subjects in the right panel demonstrated a large perfusion decrease that remained lower than during the preload interval, even after the load interval. Responses are shown to the same scale.

Figure 6. INITIAL BLOOD PERFUSION REDUCTION DUE TO PRESSURE LOADING



s-urine and water and in final hardness for skin exposed to s-urine. This finding supports the hypothesis that wet skin provides less support for pressure-related deformation and suggests a possibly greater effect caused by s-urine. Because skin wetness was associated with a slightly lower skin temperature after pad removal when compared with corresponding dry control sites, it is unclear what role the reduced temperature may have played in the reduction of tissue hardness. Patel et al have reported that skin stiffness is less at lower temperatures⁶ and this may have impacted the skin hardness measurements in the present study.

Despite the tissue hardness reduction accompanying wetness, no consistent pattern of changes in blood flow occurred during the 5 minutes of localized pressure loading. Blood perfusion tended to increase during this interval, following an initial flow decrease in about 60% of cases. The physiologic adaptive flow²¹ increase during loading may have masked an initially greater flow deficit within wet tissue. This hypothesis was tested through assessment of blood perfusion during the first minute of loading. Results of this analysis showed that the initial perfusion decrease caused by pressure was significantly greater in wet skin when compared with dry skin. This supports, in part, the hypothesis that reduced tissue hardness renders tissue perfusion more vulnerable to pressure loading, even though the flow reduction was not sustained for the healthy subjects in this study.

The erythema reduction following the 5.5 hours of wetness was somewhat similar, although less dramatic, than the skin blanching-like effects of digits immersed in water for a long duration. Erythema measurements showed a consistent decrease in average skin redness due to exposure to s-urine and water. Visual observations of the wet test sites often

showed a mottled nonuniform pattern of normal skin tone mixed with regions having a relative blanch-like appearance.

Even up to 15 minutes after dressing removal, a consistently lower skin temperature was found for wet sites when compared with dry control sites. The reduced temperature is likely partially related to the greater heat loss of the damp skin. Despite the reduced temperature and erythema of the wet sites, however, the preload blood perfusion of wet and dry sites were not significantly different. A possible direct effect of the lowered skin temperature on blood perfusion regulation in response to the pressure load cannot be excluded as a contributing factor. Further work in this area is needed to more precisely estimate its effect.

CONCLUSIONS

The overall findings of this study are consistent with the concept that sustained skin wetness increases the vulnerability of underlying blood vessels to pressure-induced blood flow reduction. The effect appears to be mainly dependent on wetness, although skin temperature reduction may also play a possible role. Because near equal temperature reductions were seen in sites exposed to s-urine and water, it appears that temperature effects, if present, would only partially account for these findings. The fact that only sites exposed to s-urine demonstrated a final significant reduction in hardness suggests that s-urine constituents may exacerbate the skin's vulnerability to pressure-induced blood flow reduction.

The effect on blood flow of water and s-urine under pressure load was not sustained on average due to the blood flow adaptation and recovery systems of the young healthy subjects in this study. In patients with compromised recovery mechanisms, however, skin wetness would likely have more sustained effects.

Measures to diminish skin exposure to wetness in such patients, whatever the wetness source, are important considerations in a multifaceted strategy to reduce the risk of pressure ulcers. ●

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