



Microclimate: Rediscovering an Old Concept in the Aetiology of Pressure Ulcers

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Introduction

The ‘state of the art’ in our understanding of the aetiology of pressure ulcers has been described in both editions of the International Pressure Ulcer guidelines [1, 2] in part produced by the European Pressure Ulcer Advisory Panel (EPUAP). However one key difference between these texts separated only by 5 years, is the inclusion of discussion upon the role of microclimate changes in the recent guidelines [2] that were not present in the earlier report of pressure ulcer aetiology [1].

The concept of microclimate is typically considered to reflect the combination of temperature and humidity or moisture acting at the skin surface at the body-support surface interface [3] and has emerged in the past five years as a new area for exploration when considering pressure ulcer development. However understanding that changes in skin temperature or humidity might influence pressure ulcer development are not new ideas, but rather reflect the rediscovery of views on pressure ulcer aetiology held thirty years ago. In the foreword to the proceedings of the first pressure ulcer conference to be held in the United Kingdom, Roaf [4] commented that ‘we know how to avoid bed sores and tissue necrosis—maintain the circulation, avoid long continued pressure, abrasions, extremes of heat and cold, maintain a favourable microclimate, avoid irritating fluids and infection. The problem is the logistics of this programme.’ So from the beginning of pressure ulcer activity in the UK microclimate and microclimate changes were seen as being a key part of successful pressure ulcer prevention. However by the mid 1980’s there was a considerable increase in the availability of specialist pressure-redistributing beds, mattresses and (to a lesser extent) seat cushions [5] and focus shifted from microclimate management to the quantification of the pressures applied to the skin by various support surfaces (for

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example [6–10]). It has only been in the past five years that microclimate management has re-emerged perhaps partly due to new support surface cover materials and also growing clinical interest in understanding pressure ulcer development [3, 11].

Why Should Skin Temperature and Humidity Influence Pressure Ulcer Development?

Endotherms can maintain body temperature at a minimal metabolic rate across a range of ambient temperatures, the thermal neutral zone (TNZ) [12]. As ambient temperatures approach the lower and upper boundaries of the TNZ metabolic rate must increase to maintain a constant core temperature. Metabolic rate will also rise as core temperature increases with a 1 °C rise in core temperature causing a 10–13% increases in oxygen consumption [13]. So at extremes of ambient temperature and where core temperature rises the demand for oxygen is increased and this increased demand may not be met where skin and soft tissues are loaded so reducing local blood and oxygen supply.

Extremes of moisture or dryness at the skin surface can also be anticipated to produce changes in the skin. High levels of skin wetness (be this from perspiration, incontinence, wound exudate) can reduce dermal collagen cross-linkage so weakening the stratum corneum [14] similar effects can be seen where relative humidity is high with a 25 fold decrease in stratum corneum strength at 100% relative humidity compared with its strength in a 50% relative humidity environment [15]. Excess moisture also changes the skin's coefficient of friction [16] making superficial damage through abrasion more likely. Dry skin also presents clinical challenges [3] with reduced lipid levels in dry skin along with less water content and weakened junctions between the epidermis and the dermis.

Interactions between skin temperature and humidity can also be observed that may induce deleterious changes in skin and soft tissue. For example reduced relative humidity may lead to increased sweat evaporation so reducing skin temperature while delamination of the stratum corneum increases with increasing temperature and humidity [17].

From human physiology through to tissue sample studies there are a range of potential modes of action through which changes in the local microenvironment of loaded skin and soft tissues may accelerate or prompt tissue damage leading to early forms of pressure injury. Gefen [18] modeled the likely interactions between microclimate, pressure and the development of superficial pressure damage. In this model five associations were proposed—that superficial pressure damage was more likely to occur where;

- As skin temperature increased
- As ambient temperature increased
- As relative humidity increased
- As pressure upon the skin increased, and
- As the permeability of bed sheet/clothing decreased.

While the potential modes of action where microclimate changes may impact on tissue viability appear both reasonable and valid, is there clinical evidence associating both microclimate changes and management with the occurrence of current pressure ulcers and the prediction of future pressure injuries?

Skin Temperature and Pressure Ulcers

From the late 1970s regional variations in skin surface temperature were assessed both to identify areas of potential pressure injury and to assess the likely healing of established full-thickness pressure ulcers (for example [19–21]). Newman and Davis [20] reported the development of pressure ulcers (category II and above) at the sacrum of ninety-one elderly hospital in-patients admitted with no visible sacral pressure damage. Of the 91 patients, 19% (n = 17) had unusual thermal patterns at the sacrum where 11 showed a warm area surrounded by a thermal gradient of less than 1 °C/cm while six thermal anomalies were associated with creasing of the skin at the sacrum. Five of the 11 patients with diffuse warm areas developed pressure ulcers (severity unreported) while a further patient with a crease in the sacral skin also developed a pressure ulcer within ten days of admission to hospital. These six pressure ulcers were reported to have developed either exactly where the thermal anomaly was located (n = 4) or adjacent to the anomaly (n = 2). No other subject in this early study developed pressure ulceration. In common with many early reports of pressure ulcer studies no information was provided upon the pressure ulcer preventive care allocated to the study participants. Norton scores [22] were reported by Newman and Davis with the majority 53/91 reported to have a Norton score higher than 14 upon admission suggesting a ‘low risk’ patient population for pressure ulcer development. The sensitivity and specificity of the use of thermography and the Norton score to predict risk of pressure ulcer development in this study were similar (thermography sensitivity 100%, specificity 39.3%; Norton scale sensitivity 83%, specificity 36.1%) however the thermal images required the elderly patients to lie with their sacrum and buttocks exposed for around 30 min potentially creating a poor experience of the first part of their stay in hospital! This early work by Newman and Davis suggested that the temperature of intact skin could form the basis for pressure ulcer prediction especially where the thermal anomaly suggested damage deep within the soft tissues (diffuse warm spot at the skin surface) however the technique appeared no better than standard pressure ulcer risk assessment and was likely to lead to a loss of dignity for the patient being assessed for thermal anomalies.

Sprigle and colleagues [23] reported upon skin temperature changes at anatomical sites prone to pressure ulceration in 65 predominantly non-ambulatory in- and out- patients within an acute rehabilitation hospital. All sixty-five participants had persistent erythema at the bony landmark and the skin temperature at the area of erythema was compared with the temperature at adjacent areas where no erythema was visible. Skin temperature was considered to be similar across the two measurement sites if the difference was below 1 °F. Across the 65 participants eighty skin

sites with erythema and adjacent control locations were measured with the skin temperature similar in 12 cases, cooler in 18 and warmer in 50. This indicates that skin temperature changes are likely to be found between areas of erythema and the surrounding skin but that erythematous areas may be warmer or cooler than the surrounding skin and that areas of early pressure damage may remain at a similar temperature to surrounding non-damaged skin. These results are ambiguous in terms of clarifying the value of using skin temperature to discriminate between areas of erythema and apparently 'normal' skin.

Clark [24] followed a cohort of 52 elderly people newly admitted to hospital with no visible pressure damage at the sacrum for 14 days during their hospital stay. Skin temperature at the interface between the sacrum and bed mattress was recorded upon admission and after 14 days six patients (11.5%) developed superficial pressure damage at the sacrum. Where pressure ulceration occurred the temperature between the sacral skin and mattress upon admission was 34.53 °C (Standard deviation, SD, 0.58), where no pressure ulcers developed sacral skin temperature while lying in bed was similar (35.02 °C SD 0.18). This study was flawed given the lack of control over the selection of the support surface used in bed—11/52 were allocated active support surfaces (alternating mattresses) the other 41 rested on reactive surfaces (foam mattresses) and skin surface temperature stability while lying on alternating surfaces has been reported in a volunteer study [25] where sacral skin temperature remained constant while resting on an alternating mattress but increased on average by 1.3 °C while the subjects rested on a foam mattress.

While these clinical studies may have conflicting results on the value of skin surface temperature data as indicators of potential tissue damage, they reinforce the challenges faced when trying to obtain such physiological data in health care settings where control over the ambient environment and allocated pressure ulcer preventive care may not be possible. In the laboratory clearer indications of the impact of modifying skin surface temperature have been seen. Kokate and colleagues [26] loaded 12 metal discs upon the dorsal surface of young pigs. Each disc was loaded to provide a surface pressure of 100 mmHg maintained for five hours, however the discs were presented at different temperatures ranging from 25 °C, 35, 40–45 °C. Where 100 mmHg was applied for five hours at the lowest temperature (25 °C) no skin or muscle damage was observed. As the temperature of the loaded discs increased moderate levels of muscle damage was observed at 35 °C with skin and muscle damage recorded at the higher temperatures. This set of experiments suggests that cooling skin may provide additional protection from pressure damage.

Ten years ago, Lachenbruch [27] summarized these, and other studies of the effect of skin temperature on pressure ulcer development concluding that a 5 °C reduction in skin surface temperature might provide similar benefits in terms of skin integrity as the most expensive patient support surfaces. Whether such a drop in surface temperature would be acceptable to patients is unclear! Recently the effects of cooling on tissue viability have been explored in a rat model [28] where a load of 700 mmHg was applied for 3 h to the trochanteric area of rats with either local warming (+10 °C) or local cooling (−10 °C). Load application with local cooling

reduced the accumulation of cytokine tumor necrosis factor alpha (TNF- α) compared with pressure and local warming, suggesting a protective effect of cooling against inflammation. Under loading with heating or cooling no change in the production of interleukin 1 β was observed. In humans the effect of local cooling or warming has been explored in terms of their effect on the hyperemic response after removal of load from soft tissues [29]. In a group of ten spinal cord injured and ten uninjured controls a 60 mmHg load was applied to the sacrum for 20 min followed by a recovery period of similar duration. Three test protocols were applied pressure without temperature modification, pressure with local cooling (-10°C) and pressure with local heating ($+10^{\circ}\text{C}$). In both the SCI and control groups smaller hyperemic responses were observed where pressure was applied with local cooling compared with either no temperature changes or local heating with this reduced hyperemic response attributed to reduced metabolic and neurogenic activity. Local cooling has been recently associated with changes in cytokine production and reduced hyperemic responses after loading—whether such changes can be translated into interventions that help cool vulnerable skin and soft tissues without reducing the quality of life for patients through reduced skin temperatures is a challenge for the coming years.

Skin Humidity and Pressure Ulcers

High relative humidity at the junction between the skin and support surface has long been related to pressure ulcer development. In 1992, the then US Agency for Health Care Policy and Research issued its pressure ulcer prevention guidelines [30] where it was recommended that relative humidity above 40% should be avoided to help prevent pressure ulcers, the source of this specific threshold is unclear. Clark [24] reported skin-mattress relative humidity at the sacrum of 52 elderly hospital patients admitted with no visible sacral skin damage. Among this cohort six developed superficial sacral pressure ulcers and in this group the mean relative humidity measured at the sacrum upon admission to hospital was 74.1% (SD 11.6). Where no superficial skin damage occurred the mean relative humidity at the sacrum upon admission was considerably lower, 43.0% (SD 3.7) however as noted earlier mattress allocation was not controlled within this study and some subjects were allocated alternating pressure support surfaces with the majority resting upon foam mattresses. Black and colleagues [31] reported a small controlled study comparing pressure ulcer incidence upon two reactive support surfaces—one low air loss bed with ‘microclimate management’ the comparator being a powered mattress. The powered mattress was the support surface used within a single centre cardio-vascular intensive care unit and prior to the study five of the powered mattresses were replaced with the low air loss bed with microclimate management. Eligible subjects were those patients within the intensive care unit (ICU) expected to have a length of stay in ICU for longer than 3 days, did not require a support surface to assist with pulmonary or wound challenges and were not on an end-of-life pathway. Fifty-two subjects were recruited with 31 receiving microclimate management and 21 the

ICU standard bed mattress, the process of allocation to the two regimes was unreported. On average the duration of follow up was 7 days with skin assessments every 3 days. During the study five patients developed a total of eight pressure ulcers—three of these presented as category II wounds under a facemask upon a single patient allocated the microclimate control mattress. The other pressure ulcers presented only among patients upon the standard ICU mattress and these tended to be superficial (two Category I ulcers, two category II and one suspected deep tissue injury). There was also a small number of patients who entered the study with pressure injuries (severity unspecified)—on the standard ICU mattress two patients each with a single ulcer showed deterioration of their wounds during the stay in ICU. One suspected deep tissue injury present upon a patient allocated the microclimate control mattress did not progress to an open wound, the other patients allocated to the microclimate control mattress were reported to show no deterioration of their pressure damage or were lost to follow-up. Black et al. [31] noted that their results may simply reflect the age of the support surfaces with the standard ICU mattresses in use for seven years compared with the new microclimate control mattresses and no data was reported upon the skin microclimate at the sacrum (or other anatomical landmarks) within the two groups of subjects. The effect of support surfaces upon modifying the microclimate at vulnerable body sites is further compromised by the effect of the various under-pads and transfer sheets often placed upon the bed surface to help repositioning and continence management [32]. As yet the impact of humidity, and its control, on pressure ulcer incidence is poorly understood with the influence of other practices (for example transfer sheets) detracting from microclimate management systems ability to moderate microclimate factors.

Discussion

Microclimate and pressure ulcer prevention have been associated for many years although active consideration of microclimate factors were perhaps lost while the clinical and research communities focused upon load management. Re-discovery of the microclimate as a potential factor in pressure ulcer prevention in recent years offers new perspectives on how pressure ulcer prevention could be complemented through management of the microclimate. Increasingly reductions in skin surface temperature are being associated with benefits for pressure ulcer prevention both in modifying the hyperaemic response and reduction proliferation of certain cytokines. However these potentially beneficial changes may be achieved at a cost of reduced patient acceptance due to the local skin cooling such interventions would require. Moisture and humidity management may also offer benefits in terms of reduced superficial pressure ulcer development although as yet the ability of commercially available microclimate management systems to alter the microclimate at vulnerable body locations is poorly reported. Additionally the impact of other care practices (under-pads for example) upon microclimate changes appears to reduce the potential benefit of microclimate management systems.

Microclimate is a growing area of activity in pressure ulcer prevention although several hurdles yet remain. For many years discussion around pressure redistribution focused upon perceived, albeit inaccurate, ‘safe’ thresholds (e.g., 32 mmHg at the skin surface) it would be a weakness of the microclimate debate where similar false thresholds developed for skin temperature and humidity. Zhong and co-workers [33] noted that there is limited *in-vitro* or *in-vivo* data upon the normal interactions between human skin and external fabrics and where available ‘existing *in vivo* experimental studies have rarely led to any significant results and solid conclusions’. Part of this challenge lies in the multiple changes in skin condition between individuals and within individuals at different body sites, these intra- and inter- differences in skin condition will likely lead to microclimate also varying among and within subjects restricting the ability of any single study to clearly demonstrate microclimate changes with skin outcomes. One solution to this challenge [33] may be for stronger dialogue between the textile research field and the pressure ulcer community—perhaps the third edition of this book will feature a joint chapter on developments in microclimate and its effect on pressure ulcer prevention?

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