Lecture as part of a series by Parafricta

Microclimate and Pressure Ulcer Development

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What is the role of skin microclimate in promoting or exacerbating pressure injuries?

When we were first talking about pressure injuries in the UK in the 1970s, microclimate was even then brought up as a risk factor:

"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." – Roaf, 1976

But then the topic disappeared. We became fixated on how to use support surfaces to manage mechanical loading. The focus was on pressure redistribution.

Microclimate as a factor has been rediscovered in the last 10 years.

Theory on How Microclimate Affects Skin

Increased skin temperature – elevated metabolic demand in loaded skin and soft tissue with compromised supply (10C rise 10% increased metabolic demand)

Increased humidity – increased exposure to shear and physical damage to skin (delamination stratum corneum)

Susceptibility to superficial PU increases:

- As skin temperature increases
- As ambient temperature increases
- As relative humidity increases
- As permeability of sheet/clothing decreases
- As pressure on the skin increases

Do microclimate changes predict skin changes in practice?

These are the studies on it:

1. Sae-Sia et al (2005)

- 17 spinal cord injuries or CVA patients
- None had PIs on admission to hospital
- Measured sacral skin temp measure in supine position after 5 min, then
- positioned them laterally and re-recorded skin temp after 15 min
- First measurement 24-96 hours post admission and then again 48-72 hours post first measurement
- Ambient conditions were challenging (room temp over 30 degrees and humidity over 75%)
- Of the 17 patients, 9 developed PIs (47%) mostly category I

Conclusion: Skin temp increased 1.2 degrees C between 1 and 4 days prior to PI development

But the interpretation requires thought: there was more going on than temperature. So can we conclude that the PI incidences were based on temperature - or were other factors at play?

2. Yusuf (2015)

- 86 acute care patients, follow up 15 days
- 20 developed skin changes, mainly at the sacrum
- More females in the skin change group
- Lower braden scores in the skin change group
- Challenging environmental conditions (room temp is 30 degrees C and humidity 75%)
- Measured interface pressure, skin temp and moisture all measured at sacrum
- Measured temperature every 3 days for max of 15 days

Temperature:

Mean difference Sacrum – umbilicus (control location)
No skin changes 0.6 (0.8): skin changes 0.9 (0.6), p=0.071

Moisture:

Difference between sacrum and umbilicus
Capacitance method (Corneometer)
Total difference sacrum moisture

Skin changes (n=20) 6.9 (18.1) AU
No skin changes (n=51) 4.3 (19.0) AU
High environmental humidity (60%) high perspiration in both groups

What's the clinical significance?

Conclusion: skin moisture was slightly elevated in the group with skin changes

High perspiration in BOTH groups (because of high humidity)

Equipment malfunction made them only collect partial data.

There were different sheeting materials on different beds – that complicated the study.

Independent risk factors (mean braden score and the type of sheeting on the beds) make it hard to draw concrete conclusions.

3. Yoshimura (2015)

They looked at PI development during surgery

Evaluated:

- Skin temperature 5cm above navel
- Perspiration – visual assessment plus changes in weight of silica gel positioned at lower side lateral abdomen
- Pressure mapping

- 29 subjects, 7 developed category I PU all resolved
- NS age, BMI, Braden score, albumin, C-reactive protein
- Differences – gender (6/7 PU+ were female), haemoglobin (lower in non PU subjects)
- Length of surgery longer in PU+ group: 7.6 (1.1) v 6.7 (0.9) hours
- Core temperature and change in skin temperature higher at end of surgery in PU+ group: 38.3 0C (0.3) v 37.9 (0.5); 2.7 0C (0.3) v 1.9 (0.8). All patients received forced air patient warming
- Peak pressures higher in PU+ group: 119.1 mmHg (36.8) v 94.5 (23.1)
- Similar perspiration (mg/hr): 7.6 (4.3) v 7.1 (4.1)

Conclusion:

Change in skin temperature independently related to PU development (0.1 0C; OR 1.44, 95% CI 1.09 – 2.33) when adjusted for average peak pressure and length of surgery

4. Forriez (2017)

- Adult ICU patients (mechanical ventilation, continuous sedation, no surgical wound at interface, PU category II, body weight >227kg, diaphoresis, diarrhoea, seeping skin lesion, traumatic injury)
- N=34, mean age 70 years
- Skin humidity, skin impedance
- Skin temperature, infra-red thermometer
- Measurements 4 hour before coverlet use then four hourly up to 20 hours

- Skin temperature: Decreased at scapula 36.9 0C (36.2 – 37.3 25th – 75th percentiles) to 36.50C (36.0 – 37.0)
- No differences at sacrum and buttocks (sacrum 36.8 0C (36.5 – 37.1) to 37.0 0C (36.2 – 37.3)
- Humidity: Significant decreases at scapula, sacrum and buttocks, no change at occiput.
- Small changes in skin humidity - sacrum 13.4% (12.2 – 15.4) to 11.4% (10.7 – 12.2) – statistically significant but clinical relevance?
- Subjective assessment of health professionals. Improvement in temperature? No 13/15 (86.6%)
- Improvement in humidity: No 12/15 (80,.0%)

General Observations

Limited objective data in-vitro and in-vivo

Little characterisation of the normal interactions between skin and fabric so interpretation of available data is challenging

Existing in vivo experimental studies have rarely led to any significant results and solid conclusions (Zhong et al 2006)

Dramatic variation of skin conditions (surface roughness, hydration, adhesion between skin layers, etc.) among individuals as well as among different anatomic sites of the same person

Lack of communications between researchers in the areas of textiles and dermatology

Ultimate Conclusion

Currently, evidence is WEAK that changing microclimate affects PI development.

We look forward to finding the smoking gun – the clear link between microclimate and PI. We need more work on microclimate so we can link the theoretical aspect with the practical.

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