ALL PRESSURE ULCERS ARE THE RESULT OF DEEP TISSUE DAMAGE

Dan Berlowitz, MD, MPH
Center for Health Quality, Outcomes and Economic Research
Department of Veterans Affairs, Bedford MA
A strategy designed to limit or solve a problem requires, first and foremost, a clear understanding of the nature and underlying causes of the problem.

Cooney JAGS 1997
Question 1.
Are superficial ulcers not “pressure ulcers”?

Question 2.
Do factors other than pressure significantly contribute to the development of a stage 3/4 ulcer?
Question 1

What is the definition of “pressure ulcer?”

Any lesion caused by unrelieved pressure resulting in damage of underlying tissue.
But traditional teaching is that pressure ulcers are result of four external factors
- pressure
- shear
- friction
- moisture
Consequently many lesions not caused by pressure are labeled “pressure ulcers” particularly if over a bony prominence.

- Maceration from incontinence
- Superficial erosion from friction
- Erythema from shear-induced tearing of blood vessels
Little evidence that pressure-induced damage starts at surface and works down.

– pressures usually higher in deep tissues
– muscle most susceptible to pressure induced injury
Even if pressure does cause superficial injury, should we care?

– Less harm from stage 1 / 2 ulcers
– Most important if marker for more severe damage
Conclusion Question 1:
Most/all superficial ulcers are not really pressure ulcers
Supports contention that all pressure ulcers result from deep tissue damage
Question 2

Friction and moisture as a cause of stage 3 / 4 pressure ulcers

– is there an association in epidemiological studies?
– Is there a plausible physiologic mechanism?
## Rate of Stage 3 / 4 Pressure Ulcer Development by MDS Continence Category

<table>
<thead>
<tr>
<th></th>
<th>Continent</th>
<th>Usually Continent</th>
<th>Occasionally Incontinent</th>
<th>Frequently Incontinent</th>
<th>In-Continent</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Foley as Continent</strong></td>
<td>0.56%</td>
<td>0.22%</td>
<td>0.23%</td>
<td>0.52%</td>
<td>0.76%</td>
</tr>
<tr>
<td><strong>Foley as Incontinent</strong></td>
<td>0.28%</td>
<td>0.22%</td>
<td>0.23%</td>
<td>0.52%</td>
<td>0.91%</td>
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</tbody>
</table>
Patients who are immobile are more likely to be incontinent

- Is incontinence then an independent predictor after adjusting for immobility?
Results of Studies Using Multivariate Analyses

Many have been unable to adequately separate wetness from immobility.

Urinary incontinence often not an independent predictor.
- Allman et al. Study of 286 hospitalized patients (JAMA 1995)
- Brandeis et al. Study of 4232 nursing home residents (JAGS 1994)
- Horn et al. Study of 1524 nursing home residents (JAGS 2004)
Mechanisms for Injury

1. Deep injury occurs from pressure. Superficial erosion makes it more likely to come to surface and form ulcer.

2. Alteration of frictional properties contributes to increased shear. Shear does contribute to deep tissue injury.

3. Moisture alters skin hardness. Pressure than cause greater decrease in blood flow.
Figure 3. SKIN HARDNESS CHANGES

Hardness Change (Durometer points)

S-Urine  Water  Urine Dry Control  Water Dry Control

*P<.01

Initial change  After 1 minute

Mayrovitz & Sims. Advances in Skin & Wound Care 2001;14(6):302
Figure 6. INITIAL BLOOD PERFUSION REDUCTION DUE TO PRESSURE LOADING

Pressure load = 60 mm Hg

% Change during 1st Minute of Loading

S-urine

Water

Dry

*P<.01

*P<.01

Mayrovitz & Sims. Advances in Skin & Wound Care 2001;14(6):302
Limitations of Study

Doppler probe measures only to skin depth of 1-2 mm
– nothing known about deeper tissues
Study Conclusions

Sustained skin wetness increases vulnerability of underlying blood vessels to pressure induced blood flow reduction

Mainly dependent on wetness although skin temperature reductions also play role

But how much of an effect on deep tissues?
Conclusion Question 2

Friction and moisture seem to have little role in the development of stage 3 / 4 pressure ulcers

Pressure has leading role
Are all pressure ulcers the result of deep tissue injury

– that’s what the data suggest!