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

ALL PRESSURE ULCERS ARE THE RESULT OF DEEP TISSUE DAMAGE

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

	Continent	Usually Continent	Occasionally Incontinent	Frequently Incontinent	In-Continent
Foley as Continent	0.56%	0.22%	0.23%	0.52%	0.76%
Foley as Incontinent	0.28%	0.22%	0.23%	0.52%	0.91%

- Patients who are immobile are more likely to be incontinent
  - Is incontinence then an independent predictor after adjusting for immobility?

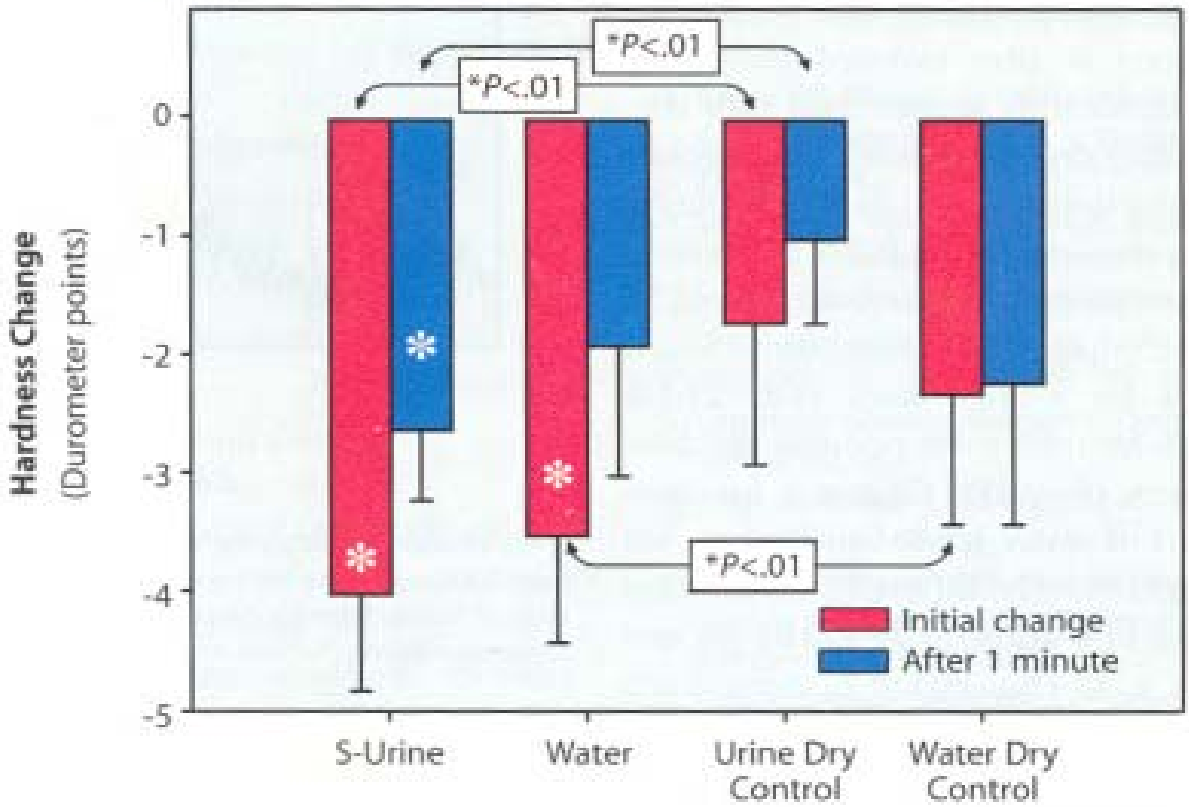
Results of Studies Using Multivariate Analyses

- 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

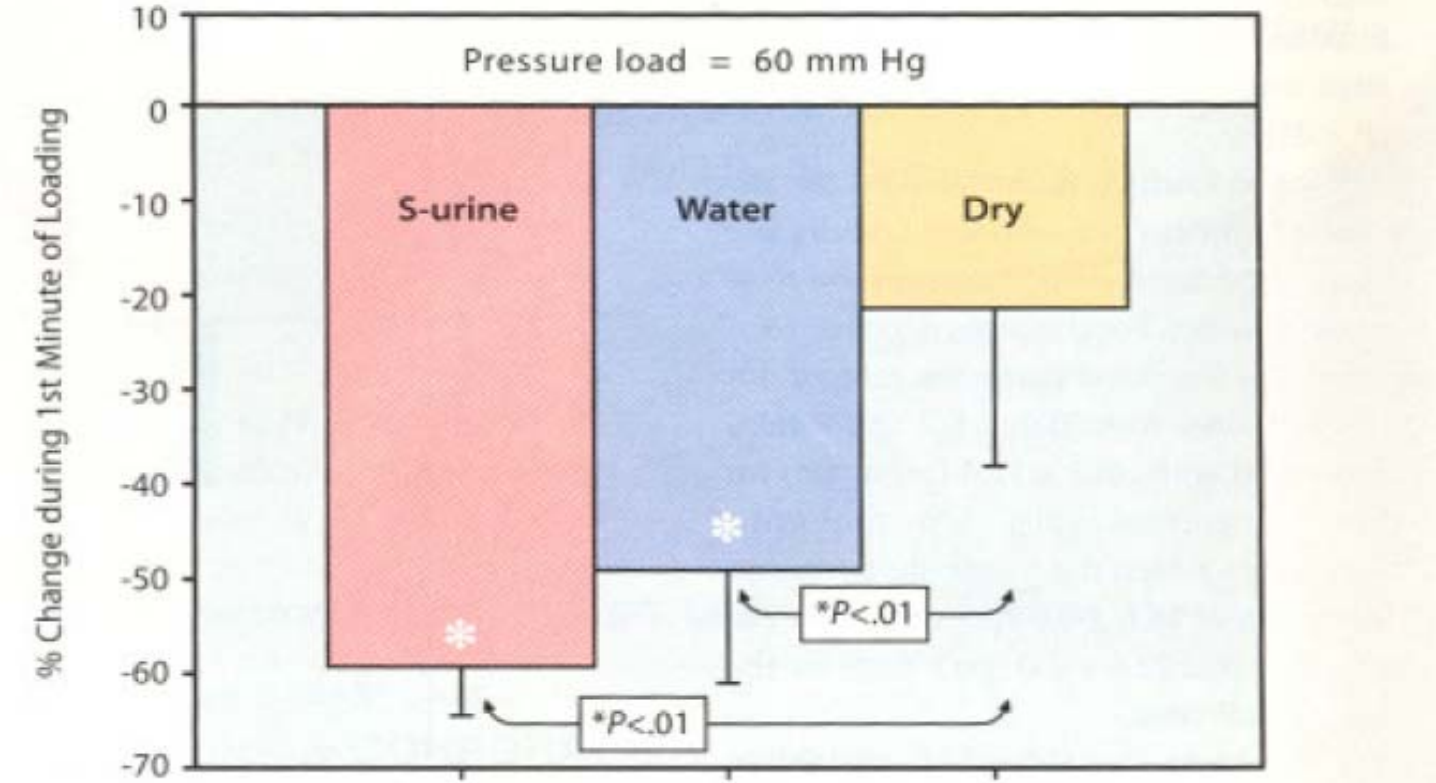
1. Deep injury occurs from pressure. Superficial erosion makes it more likely to come to surface and form ulcer
2. Alteration of frictional properties contributes to increased shear. Shear does contribute to deep tissue injury
3. Moisture alters skin hardness. Pressure than cause greater decrease in blood flow.

Figure 3. SKIN HARDNESS CHANGES



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

Figure 6. INITIAL BLOOD PERFUSION REDUCTION DUE TO PRESSURE LOADING



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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!