



Wound
Ostomy and
Continence
Nurses
Society

WOCN Society Response to NPUAP White Papers: Deep Tissue Injury, Stage I Pressure Ulcers, and Stage II Pressure Ulcers

9th National NPUAP Conference, February 25–26, 2005

The WOCN Society would like to thank the National Pressure Ulcer Advisory Panel (NPUAP) for the opportunity to participate in a dialogue about deep tissue injury and Stage I and II pressure ulcers. The WOCN Society leadership asked several Wound, Ostomy, Continence Education Program Directors to write and post responses to the NPUAP White Papers on behalf of the WOCN Society. WOCN's official response and NPUAP's questions, which were posted online, are repeated here in the hope that they will contribute to a thought-provoking discussion.

Deep Tissue Injury

What clinical indicators would you use to define a Stage I pressure ulcer?

The general consensus of staff nurses and expert WOC nurses who were consulted was that both are guided by the existing, accepted descriptions of Stage I and II ulcers. These definitions are posted on many units for easy reference. Many of the nurses we consulted felt that their abilities to identify Stage I and II pressure ulcers improved with experience (and with more experience with dealing with patients with dark complexions).

Good lighting and correct positioning are critical. Staff nurses are generally not as aware as WOC nurses are about "bruising under intact skin," although they had seen it. In general, WOC nurses were upstaging these ulcers to at least Stage II or III because they recognized the tearing of fine blood vessels. Generally, they had not considered the possibility of full thickness necrosis like Stage IV. All were interested to learn more.

How can deep tissue injury (DTI) be best identified in patients with darkly pigmented skin?

Education and experience are key. WOC nurses were much more aware than staff nurses of pressure ulceration in darkly pigmented skin. But staff nurses who cared for diverse patients seemed to be more knowledgeable when they had continuing education from WOC staff or from nursing education.

How long does it take for DTI to develop?

There is no specific formulaic answer to this question based on staff and WOC nurse input. Heels and sacrum were mentioned as most often affected. WOC nurses mentioned tissue tolerance. In some clients, DTI can develop rapidly when patients are affected by multifactorial deleterious events and characteristics. Others may never develop DTI despite expectation to the contrary. The consensus was that this depends on patient condition and how it changes.

One condition that everybody agreed upon is "terminal status." Patients who are dying will have the tendency to develop new skin breakdown (i.e., skin failure) as other organs fail. All concurred that existing pressure ulcers of

lesser staging (I and II) almost always worsen over time to at least Stage III among dying patients. Staff nurses who reported this worked in long-term acute care settings and are used to seeing patients slowly descend in a downward spiral in viability; skin integrity alterations accompany this descent.

What level of pressure/stress is required to develop a DTI?

This depends on the patient and situation. If a patient is sick enough and challenged by multiple factors, the lesser type Stage I and II ulcers can (and usually will) worsen to DTI despite interventions. One person said, "even a sandbed won't help some people who are deteriorating."

How do co-morbidities (especially rapid weight loss and loss of sensation) affect the time to develop DTI?

Co-morbidities in general hasten the deterioration to DTI. It can happen despite interventions if a patient is sick enough. Many staff and WOC nurses mentioned the heel as a prime risk site. However, no known research exists about "time to DTI."

Does DTI that never reaches the skin really represent a pressure ulcer?

Yes, it does.

How would a revised staging system impact documentation, regulation, and reimbursement?

A revised staging system would affect all aspects of documentation, regulation, and reimbursement. Most WOC nurses and staff nurses who were consulted felt that the current system does not need to be discarded but that all participants need better education. Despite current education, incorrect staging occurs. Overhauling the entire system would be counterproductive because the current system is not working perfectly yet.

How would the addition of DTI to the nomenclature of pressure ulcers affect liability? How would it affect healthcare quality measures?

Rather than revising the entire system, DTI should be the object of intense education. Just as staff and WOC nurses have become more expert in staging, awareness of the need for recognizing special forms of severity can be enacted. Bruising under intact skin could be categorized as unstageable with special documentation of the likelihood

of deeper tissue injury. Reimbursement should be reformed to recognize that nonstageable is not a low-care need but a marker for more intense care and reimbursement. Liability may actually decrease because the true nature of tissue damage depth would be documented. On the other hand, liability may not be decreased at all. Quality indicators could include special attention to the DTI phenomenon. Documentation changes would have to be based on education of all involved. Changes implemented in patient care that result from a change in documentation would be required to make a substantive impact on patient safety and quality care.

Renee Anderson, MSN RN CWOCN, is University of Washington Wound Management Education Program Director. She can be reached at mandranderson3@comcast.net.

Paula Erwin-Toth, MSN ET RN CWOCN CNS, is Rupert B. Turnbull School of ET Nursing Wound, Ostomy, Continence Education Program Director. She can be reached at ERWINP@ccl.org.

Janice Beitz, PhD RN CS CNOR CWOCN, is a Lasalle University School of Nursing Wound, Ostomy, Continence Education Program Director. She can be reached at beitz@lasalle.edu.

Stage I Pressure Ulcers

Does the current nomenclature “Stage I” pressure ulcer adequately describe the phenomenon? Is it a superficial lesion or any pressure-induced skin damage with intact skin?

“Stage I” does not adequately describe the pressure ulcer phenomenon or explain whether this is a true pressure ulcer if the skin is intact. Use of the term “ulcer” to describe pressure damage at this stage is problematic and inconsistent with the definition of an ulcer.

Identification of Stage I relies on clinical examination and visual identification of erythema, which varies in intensity and duration and may be blanchable versus non-blanchable, persistent or transient. Differences due to darkly pigmented skin’s resilience and color must be noted. Although tactile examination of the skin has been recommended, the reliability of this is undetermined. Reliance on color and tactile assessment is unreliable and not proven to adequately reflect injury to epidermis, dermis, or underlying tissue.

At the heart of the dilemma of staging ulcers is the unanswered question of whether pressure ulcers occur in a top-down or bottom-up fashion and if pressure injuries can damage only the epidermis with intact skin. Extrinsic factors (e.g., friction, shear, moisture) may impact tissue tolerance, but the damage starts at the bone/muscle interface.

If only the epidermis is involved, is it a pressure ulcer or injury due to friction, maceration, or other dermatological conditions?

The issue of the progression of ulcers (i.e., top-down or bottom-up theories of pressure injury) is unresolved and inadequately explained by research. Limited investigations and current literature provide some support for the bottom- (i.e., bone) up theory, indicating that the deeper tissues are involved in pressure injury.

The emerging opinion is that true pressure ulcers are from injury to soft tissue and muscle that occur from pressure and is most pronounced over bony prominences.

Should any clinical indicators currently in the Stage I definition be deleted or any other clinical indicators included?

The clinical indicators are subjective in nature and vary highly among patients and clinicians. Additional parameters recently included in the definition of Stage I are more consistent with deep tissue damage. For example, the indicators “boggy” and “firm” suggest deeper soft tissue injury and are somewhat subjective, vary from patient to patient, and lack validation. Itching has been included in the definition and also needs further validation.

Should a nonhealing Stage I be reclassified as DTI?

A “healing Stage I” may not reflect true pressure damage and “nonhealing” is consistent with deep tissue damage.

What is the appropriate time frame for reclassifying a Stage I pressure ulcer to DTI?

Research is lacking and variable in nature to support a specific time frame for the progression of tissue damage due to pressure. Progression of damage would depend on the amount of pressure, duration, location, “host,” and tissue tolerance and is affected by numerous intrinsic and extrinsic factors.

What is the purpose of a staging system?

The greatest benefit of a pressure ulcer classification system is to facilitate *early detection of pressure injury so that preventive and/or therapeutic interventions can be implemented. It encourages clinicians to look for and at the damaged area or ulcer.*

Staging serves a role in classifying the extent of pressure damage for research, prevalence and incidence studies, and provides a basis for inclusion/exclusion criteria in planned studies.

Staging has limited value in directing therapy, which is guided by other, multiple factors that can be identified and assessed including size, shape, depth, presence of undermining/tunnels, tissue type, exudate, wound edges, presence of necrosis, signs of infection, as well as patient, caretaker, and socioeconomic and environmental factors that contribute to pressure injury.

Staging should not be used as the sole parameter for treatment, evaluation of therapeutic effectiveness, or reimbursement.

Comments

Clinicians and caretakers have difficulty with correctly identifying underlying tissue damage in the presence of intact epidermis based on visual and tactile assessment alone.

In the presence of intact epidermis, and based on the current staging system, clinicians make *educated guesses* about tissue injury. Further research is needed to establish the validity and reliability of clinical indicators and to investigate the use of noninvasive technology to accurately determine the depth and extent of pressure injury.

Without clinically consistent, measurable parameters or use of additional noninvasive technology, it is not possible for clinicians in routine practice to accurately detect the depth of pressure damage underneath intact epidermis by visual and tactile examination alone. Therefore, if the epidermis is intact, the injury would be classified as “unstageable” to accommodate the indeterminate nature of superficial versus DTI under intact skin.

Recommendations

- Classify as unstageable any pressure-related changes of intact skin over a bony prominence.
- Replace current Stage I definition with the following definition:

Unstageable, persistent, pressure-induced alteration of intact skin over a bony prominence as compared to the adjacent or opposite area on the body including erythema, edema, pain, or tissue changes. In lightly pigmented skin, the area may appear as a defined area of persistent redness, whereas in darker skin tones, the ulcer may appear with persistent red, blue, or purple hues.

Note: Interventions must be instituted (i.e., “unstageable” does not mean “untreatable”). Because any alteration warrants intervention, this definition, along with other risk assessment criteria, can be used to institute preventive or therapeutic interventions for any individual with this manifestation.

Phyllis Bonham, PhD(c) MSN RN CWOCN, is Director of Medical University of South Carolina, Wound Care Specialty Course. She can be reached at bonhamp@musc.edu.

Janet Ramundo, MSN RN FNP CWOCN, is Emory University WOC Nursing Education Center Special Projects Chair. She can be reached at jramund@emory.edu.

Stage II Pressure Ulcers

Should the definition of Stage II pressure ulcers encompass all partial thickness wounds over bony prominences, including those that may have resulted from maceration or denudation injuries?

No. These lesions are not caused by pressure and should not be included in the pressure-ulcer staging system. Inclusion of nonpressure wounds in the pressure ulcer staging system contributes to confusion among clinicians, and fails to focus attention on the relevant etiologic factors. (Labeling these wounds as Stage II pressure ulcers implies that the wounds are caused by pressure when in fact they are caused by some combination of maceration and superficial friction and shear.)

We recommend use of the term “skin breakdown related to...” as an umbrella term (as opposed to the current use of “pressure ulcer” as an umbrella term to include all wounds caused by some combination of external mechanical factors).

Should skin tears be included in the definition of Stage II pressure ulcers or be monitored separately for quality-of-care surveillance?

No. Skin tears should not be included in the definition of Stage II pressure ulcers, for the reasons outlined previously. We agree with the concept of a separate monitoring system for quality-of-care surveillance related to these wounds (and would suggest use of the nationally recognized classification system already established for these wounds).

When there is clinical suspicion of a DTI should the wound be characterized as “unstageable” until the full extent of tissue damage is ascertained?

We would recommend one of the two following approaches to the definition of Stage II ulcers.

- Delete this stage from the pressure-ulcer staging system because true Stage II wounds are partial-thickness injuries caused by friction, maceration, and superficial shear forces and are not pressure-related injuries. (This would eliminate or reduce much of the confusion experienced by clinicians who wonder which partial-thickness wounds to include in prevalence and incidence studies. This would also strengthen the integrity of the staging system because the current inclusion of Stage II lesions perpetuates the perception that pressure ulcers begin at the epidermis and progress downward, when in fact the limited data we have strongly suggests a “bottom-up” progression.)

Or

- Change the definition to “unstageable pressure-induced injury located over a bony prominence and characterized by epidermal sloughing and changes in skin temperature, tissue consistency, and/or sensation.”

Should shallow pressure ulcers with slough be characterized as Stage III pressure ulcers only?

No. If the wound base is obscured by slough, accurate determination of tissue involvement and wound stage must be delayed until the wound base can be visualized. Shallow wounds in which the base is obscured by slough should be classified as unstageable.

Comments

There is a need for clear and consistent differentiation between Stage II and Stage III wounds. For example, it would be helpful for clinicians to develop consensus statements addressing partial-thickness versus full-thickness wound healing. The WOCN Society would advocate for clear statements that indicate that wounds extending beyond deep dermis and involving loss of epidermal appendages are full-thickness wounds that heal by formation of new connective tissue. It must be clear that these are not Stage II wounds.

Demarcated or rolled edges in an established Stage II wound actually suggests a healing Stage III pressure ulcer. In our clinical experience, clearly demarcated edges are not unique to any stage of an open ulcer. In contrast, we see rolled nonproliferative edges as indicative of full-thickness wounds (the presence of rolled edges indicates that

the wound is not a Stage II). In addition, we would want to make it clear that rolled nonproliferative edges are frequently associated with nonhealing full-thickness wounds.

Dorothy Doughty, MN RN CWOCN FAAN, is Emory University WOC Nursing Education Center Director. She can be reached at ddought@emory.edu.

Bonnie Sue Rolstad, BA RN CWOCN, is WebWOC Nursing Education Program Director. She can be reached at partners@webwocnurse.com.

Suggested Readings

Agency for Health Care Policy and Research, U.S., Department of Health and Human Services. (1992). *Pressure ulcers in adults: - Prediction and prevention* (AHCPR Publication No. 92-0047). Rockville, MD.

Allman, R., Goode, P., Patrick, M., Burst, N., & Bartolucci, A. (1995). Pressure ulcer risk factors among hospitalized patients with limited activity limitation. *Journal of the American Medical Association, 273*, 865-870.

Allman, R. (1997). Pressure ulcer prevalence, incidence, risk factors, and impact. *Clinics in Geriatric Medicine, 13*(3), 421-436.

Ankrom, M., Bennett, R., Sprigle, S., Langermo, D., Black, J., Berlowicz, D., Lyder, C., and the National Pressure Ulcer Advisory Panel. (2005). Pressure-related deep tissue injury under intact skin and the current pressure ulcer staging systems. *Advances in Skin & Wound Care, 18* (in press).

Arao, H., Obata, M., Shimada, T., & Hagiwara, S. (1998). Morphological characteristics of the dermal papillae in the development of pressure sores. *Journal of Tissue Viability, 8* (3), 17-23.

Ayello, E., Baranoski, S., Lyder, C., & Cuddigan, J. (2003). Pressure ulcers. In S. Baranoski and E. Ayello (Eds.), *Wound Care Essentials Practice Principles* (pp. 240-270). New York: Lippincott, Williams & Wilkins.

Barczak, C., Barnett, R., Childs, E., & Bosley, L. (1997). Fourth national pressure ulcer prevalence survey. *Advances in Wound Care, 10* (4), 18-26.

Beckrich, K. & Aronovitch, S. (1999). Hospital-acquired pressure ulcers: A comparison of costs in medical vs. surgical patients. *Nursing Economics, 17*(5), 263-271.

Bergstrom, N. (1992). A research agenda for pressure ulcer prevention. *Decubitus, 5* (5), 22-30.

Bouten, C., Oomens, C., Baaijens, F., & Bader, D. (2003). The etiology of pressure ulcers: Skin deep or muscle bound? *Archives of Physical Medicine and Rehabilitation, 84*, 616-619.

Calianno, C. (2000). Assessing and preventing pressure ulcers. *Advances in Skin & Wound Care, 17* (5), 244-246.

Colburn, L. (1990). Preventing pressure. *Nursing, 90*, December, 60-63.

Gosnell, J., Johannsen, J., & Ayres, M. (1992). Pressure ulcer incidence and severity in a community hospital. *Decubitus, 5* (5), 56-8, 60, 62.

Gunningberg, L. & Ehrenberg, A. (2004). Accuracy and quality in the nursing documentation of pressure ulcers. *Journal of Wound, Ostomy Continence Nursing 31* (6), 328-335.

Iaizzo, P. (2004). Temperature modulation of pressure ulcer formation: Using a swine model. *Wounds, 16* (11), 336-343.

Kosiak, M. (1959). Etiology and pathology of ischemic ulcers. *Archives of Physical Medicine and Rehabilitation, 40*, 62-68.

Kramer, J. & Kearney, M. (2000). Patient, wound, and treatment characteristics associated with healing in pressure ulcers. *Advances in Skin & Wound Care, 13*, 17-24.

- Linder-Ganz, E. & Gefen, A. (2004). Mechanical compression-induced pressure sores in rat hindlimb: Muscle stiffness, histology, and computational models. *Journal of Applied Physiology, 96*, 2034-2049.
- Meehan, M. & Hill, W. (2002). Pressure ulcers in nursing homes: Does negligence litigation exceed available evidence? *Ostomy/Wound Management, 48* (3), 46-54.
- Ochs, D., Meltzer, D., Payne, W., Hill, D., & Robson, M. (1999). Truncal pressure ulcers are neither hypoxic nor ischemic and should respond to exogenous growth factor therapy. *Wounds, 11* (5), 110-116.
- Macklebust, J. (1997). Policy implications of using reverse staging to monitor pressure ulcer status. *Advances in Wound Care, 10* (5), 32-35.
- Pieper, B. & Mattern, J. (1997). Critical care nurses' knowledge of pressure ulcer prevention, staging and description. *Ostomy/Wound Management, 43* (2), 22-31.
- Pieper, B. (2000). Mechanical forces: Pressure, shear, and friction. In R. Bryant (Ed.), *Acute and chronic wounds: Nursing management* (2nd ed., pp. 221-264). St. Louis: CV Mosby Inc.
- Quirino, J., Santos, V., Quednau, Tl, Martins, A., Lima, P., & Almeida, M. (2003). Pain in pressure ulcers. *Wounds, 15* (12), 381-389.
- Russell, L. (2002a). Pressure ulcer classification: The systems and the pitfalls. *British Journal of Nursing, 11*(12) Suppl, S49-50, S52, S54-47.
- Russell, L. (2002b). Pressure ulcer classification: Defining early skin damage. *British Journal of Nursing, 11* (16) Suppl, S33, S36, S40-41.
- Salcido, R., Donofrio, J., Fisher, S., LeGrand, E., Dickey, K., Carney, J., Schosser, R., & Liang, R. (1994). Histopathology of pressure ulcers as result of sequential computer-controlled pressure sessions in a fuzzy rat model. *Advances in Wound Care, 7* (5), 23-4, 26, 28.
- Sanada, H., Nagakawa, T., Yamato, M., Higashidani, K., Tsuru, H., & Sugama, J. (1997). The role of skin blood flow in pressure development during surgery. *Advances in Wound Care, 10* (6), 29-34.
- Shea, J. (1975). Pressure sores: Classification and management. *Clinical Orthopaedics & Related Research, 112*, 89-100.
- Sprigle, S., Linden, M., & Riordan, B. (2003). Analysis of localized erythema using clinical indicators and spectroscopy. *Ostomy/Wound Management, 49* (3), 42-52.
- Verluisen, M. (1985). Pressure sores in elderly patients. The epidemiology related to hip operations. *Journal of Bone & Joint Surgery, 67* (1), 10-13.
- Wall, J. (2003). Preventing pressure ulcers among wheelchair users: preliminary comments on the development of a self-administered risk assessment tool. *Journal of Tissue Viability, 13* (2), 48-60.
- Wendelken, M., Markowitz, L., Patel, M., & Alvarez, O. (2003). Objective non-invasive wound assessment using B-mode ultrasonography. *Wounds, 15* (11), 351-360.
- Witowski, J. & Parish, L. (1982). Histopathology of the decubitus ulcer. *Journal of the American Academy of Dermatology, 6* (6), 1014-1021.
- Wong, V. & Stotts, N. (2003). Physiology and prevention of heel ulcers: The state of science. *Journal of Wound, Ostomy Continence Nursing, 30* (4), 191-198.
- Wound Ostomy and Continence Nurses Society. (2003). *Guideline for prevention and management of pressure ulcers. WOCN Clinical Practice Guideline No 2*. Glenview, IL: C. Ratliff & D. Bryant.
- Zinn, L. (2005a). Stopping pressure ulcers-before they start. Interview with Steve Mogensen and John Hertig. *Nursing Homes Magazine*. Available at http://www.nursinghomesmagazine.com/Past_Issues.htm?ID=3150.
- Zinn, L. (2005b). Battling pressure ulcers: Consistency Means Success. Interview with Courtney H. Lyder, ND. *Nursing Homes Magazine*. Available at http://www.nursinghomesmagazine.com/Past_Issues.htm?ID=2797.



Wound
Ostomy and
Continence
Nurses
Society

**4700 W. Lake Avenue
Glenview, IL 60025-1485
www.wocn.org
888/224-9626**