Pressure Ulcer Staging Revisited: Superficial Skin Changes & Deep Pressure Ulcer Framework

R. Gary Sibbald, BSc, MD, MEd, FRCP(C (Med Derm), MACP, FAAD, MAPWCA • Professor • Public Health and Medicine • University of Toronto • Toronto, Ontario, Canada • Director of Toronto Regional Wound Clinics • Director of the International Interprofessional Wound Care Course and Masters of Science Community Health (Prevention and Wound Care) • Dalla Lana School of Public Health • University of Toronto • President of the World Union of Wound Healing Societies • Clinical Editor • Advances in Skin & Wound Care • Ambler, Pennsylvania
Diane L. Krasner, PhD, RN, CWCN, CWS, MAPWCA, FAAN • Clinical Nurse Specialist/Wound Ostomy Continence Nurse • Rest Haven-York • York, Pennsylvania • Wound and Skin Care Consultant
Kevin Y. Woo, PhD, RN, ACNP, FAPWCA • Assistant Professor • Faculty of Health Sciences, School of Nursing • Queen’s University • Kingston, Ontario, Canada • Wound Care Consultant/Advanced Practice Nurse • West Park Health Centre • Toronto, Ontario, Canada

This article was written on behalf of the Members of the Shifting the Original Paradigm Expert Panel (SOPE Panel). See Table 1.

PURPOSE:
To enhance the learner’s competence with knowledge about superficial skin changes and deep pressure ulcer (PrU) framework concepts as compared with the currently used PrU staging and grading classification system.

TARGET AUDIENCE:
This continuing education activity is intended for physicians and nurses with an interest in skin and wound care.

OBJECTIVES:
After participating in this educational activity, the participant should be better able to:
1. Examine the physiologic changes and causes of superficial skin changes and deep PrUs.
2. Demonstrate advantages of using the superficial skin changes and deep PrU framework for documentation.
ABSTRACT

Deficiencies in the current pressure ulcer classification system create the impetus for the current discourse on the clinical, legal, and economic implications of staging and considering shifting the paradigm in pressure ulcer description and assessment.

KEYWORDS: pressure ulcer staging, pressure ulcer grading, superficial skin breakdown, shifting the original paradigm, pressure ulcer description and assessment

INTRODUCTION

Pressure ulcers (PrUs) are a significant problem across the continuum of healthcare settings. According to the results of 9 international PrU prevalence surveys from 1989 to 2005, including a total of 447,930 patients, PrU prevalence rates ranged from 9.2% in 1989 to 10% in 2004. The highest prevalence was estimated at 27.3% in long-term acute care. The majority of PrUs were classified as Stages I and II. In Germany, Kottner et al documented that 10.2% of patients (n = 40,247) in 22 hospitals developed PrUs between 2001 and 2007. Another survey of 37,307 hospitalized patients in France indicated a PrU prevalence rate of 8.9%. Overall, similar prevalence rates were reported around the world. Of concern is the fact that the prevalence and incidence of PrUs have remained essentially the same in the past 30 years; they are highest among older adult patients and patients with spinal cord injuries.

The burden of PrUs as a chronic disease is far-reaching and onerous. The average cost associated with the treatment of Stage IV PrUs and related complications in the United States was $129,248 for a single episode of hospitalization. Some of the most common complications are infections, including cellulitis, osteomyelitis, and systemic sepsis. In a recent economic analysis of annual expenditures for PrU care in Dutch hospitals, the calculated costs were staggering (ranges between €206.3 million and €238.1 million). From a patient’s point of view, the toll of living with PrUs can be devastating. Gorecki et al reviewed 31 studies that investigated quality-of-life issues in patients with PrUs. Emerging themes concerning PrUs include physical restrictions, social isolation, loss of independence, emotional problems, and financial encumbrance. Wound-related pain was expressed as a common and grievous experience even at rest. To prevent and execute prompt interventions for the management of PrUs, a valid and reliable nomenclature is required to communicate accurate assessment and monitor potential progression.

However, the current PrU classification systems (staging, grading, and categories) are often misinterpreted or misused in several settings, including clinical practice, regulatory and reimbursement arenas, and legal and economic systems. An improved delineation of the primary etiologic factors will improve treatment and subsequent patient outcomes. Despite the common use of the term pressure ulcers, superficial skin breakdown is often associated with moisture and/or friction rather than pressure (see Case Study 1). In addition, most superficial ulcers do not progress to deeper Stages III and IV lesions, so that the concept of staging or progression may need to be re-examined.

The Shifting the Original Paradigm Expert Panel (SOPE Panel) was established to explore current approaches to PrU staging and causation with the intent to improve clinical care and patient outcomes. Building on the foundations of earlier examinations of the scientific literature, expert opinion and classifications of the National Pressure Ulcer Advisory Panel (NPUAP), European Pressure Ulcer Advisory Panel (EPUAP), Wound Ostomy Continence Nurses Society, and other organizations, the panel deliberated on PrU causation and current classification systems. The 14-member SOPE Panel (Table 1) includes 2 co-chairpersons, a medical writer, the panel facilitator, and the 10 expert panel participants. The international interprofessional panel critically analyzed present PrU classification systems and examined emerging evidence on the pathophysiological mechanisms (an initial meeting was held in Baltimore, Maryland, in March 2010).

This panel asserts that the current PrU classification systems (staging, grading, categories), in their present iterations, create problems from clinical, regulatory, legal, and economic perspectives. The advisory panel is proposing a conceptual framework to...
describe superficial moisture and friction changes and deep tissue damage (often pressure and shear related). By reading this article, clinicians will be able to interpret the proposed framework for describing these skin changes and their classifications.

The rationale for the new superficial skin changes and deep PrU framework is developed from previous literature and the expert consensus of the panel. The following 10 statements have been formulated to clarify the conceptual framework. The PrU classification refers to systems with stages, grades, or categories as outlined by the NPUAP, EPUAP, Wound, Ostomy and Continence Nurses Society, and other international organizations.

STATEMENT 1
Pressure ulcers are clinical signs indicating tissue damage. This damage results from prolonged and excessive tissue deformation (compression, shear, and tension), including possible ischemic distortion of the vasculature.

Congruent with the current consensus endorsed by the NPUAP, PrUs are defined as localized tissue injury involving an intricate interplay of multiple external forces, such as pressure, shear, and friction. Briefly, pressure is defined as the perpendicular force that is applied to the skin, distorting and compressing underlying soft tissues, especially over bony prominences. Shear or shear stress is produced by displacement or deformation of tissue (usually in a diagonal direction) altering the original alignment of tissue as one layer of tissue slides over the deeper structure in opposite directions (bony skeleton moving in an opposite direction to the surface skin). In contrast, friction describes the resistance to movement created between 2 surfaces, such as the superficial layers of skin and the adjoining support surface. Any pressure injury that is accompanied by other forces (shear and friction) will result in an enhanced tissue injury.

Together, the synergistic effect of pressure, shear, and/or friction will determine the degree of internal stress and potential for tissue injury. The mechanisms responsible for actual tissue damage are linked to excessive deformation of cells, disruption of cytoskeletal architecture, obstruction of the lymphatic drainage, reduced blood flow, and ischemia. Following a localized ischemic injury, a cascade of physiological events occurs characterized by anaerobic metabolism, production of toxic metabolites, acidosis, increased cell membrane permeability, cellular edema, cell death, and finally tissue necrosis. Paradoxically, reperfusion of ischemic tissues introduces an influx of inflammatory mediators and free radicals that can initially promote further tissue damage rather than restore normal function. Although injury is often incurred by excessive mechanical loads, studies have indicated that exposure of low pressure over a protracted duration may have the same detrimental effect. Not all local injuries will result in PrUs. The final outcome will depend on the host and the extent of the insult and the local intrinsic factors that modulate tissue tolerance to injury. Key intrinsic factors are discussed in the rest of this document.

STATEMENT 2
Current numerical PrU classification systems (staging, grading, or categories) are problematic and misleading because they imply that PrUs progress through defined stages (from I to IV).

The current numerical PrU classification systems are intended to describe the anatomic depth of tissue damage. Stage I is characterized by nonblanchable erythema of intact skin that may be coupled with alterations in skin temperature and tissue consistency. Stage II is a superficial lesion involving the erosion of epidermis with epidermal base or an ulcer with loss of epidermis and a dermal base. Full-thickness tissue damage may extend to subcutaneous tissue as in Stage III PrUs and to deeper supporting structures, such as muscle, fascia, joint capsule, and bone that are classified as Stage IV PrUs. The evolution of PrUs does not necessarily follow a predictable linear pattern from superficial to deep, from Stage I changes to Stage II erosions or ulcers, then to deeper Stage III ulcers, and finally Stage IV ulcers.

Accumulating evidence suggests that a number of PrUs (most Stages III and IV ulcers) may initially originate in the deep tissue compartment and progress outward to the dermis and epidermis (inside-out theory). To expiate on this argument, deep tissue injury may not be visible to the naked eye but may take hours to days before any clinical signs are evident. Once observed, deep tissue injury can deteriorate rapidly into deep craters despite stringent and optimal treatment that meets the standard of care. Suspected deep tissue injury has the appearance of a purple or maroon bruise under intact skin that resembles and is often mistaken for a Stage I PrU. Donnelly documented that 10% of PrUs were initially diagnosed as Stage I PrUs. By visual inspection and evolved to Stage III or IV within days. It is possible that a proportion of the Stage I ulcers in this study were misclassified and that they were really deep tissue injuries given how quickly these ulcers evolved over time. Other skin lesions with color change may reflect different dermatologic diagnoses, including moisture-associated dermatitis, fungal or yeast intertrigo, or other dermatologic conditions.

By eliminating the current numerical classification system and documenting the partial-thickness and full-thickness depth along with the appropriate physical findings (location, size, base, exudate, and margins), healthcare providers may prevent misleading communication.
STATEMENT 3
Eliminating numerical PrU classification systems (staging, grading, or categories) in their present iterations may offer benefit from clinical, scientific, epidemiological, regulatory, legal, and economic perspectives.

Misuse and misinterpretation of the current PrU classification systems are common. First, there is a tendency to use the current numerical classification systems to capture progression of healing as deep PrUs become shallower. The practice of reverse or back-staging is physiologically inaccurate. As healing occurs, full-thickness ulcers are replaced by granulation tissue, not the missing muscle, subcutaneous fat, or dermis. To complicate matters further, there is much confusion surrounding the description of wounds that recur in a previously ulcerated area that is replaced with scar tissue. Never reverse stage an ulcer, so that a Stage IV ulcer becomes Stage III or II as it heals.

Second, the assumption that intervention can reliably prevent Stage I PrUs from progressing to Stage IV ulcers is faulty and dangerous. What is presumed to be a Stage I PrU may be a deep tissue injury, and the skin lesion often will continue to evolve despite optimal care. Sato et al followed the natural evolution of Stage I PrUs as evidenced by nonblanchable erythema among 30 long-term-care patients. Of the ulcers that resolved, 90% (n = 18) exhibited a hypoechoic region indicating tissue damage between the epidermis and dermis by ultrasonography corroborating the fact that the initial diagnosis was actually superficial skin damage. Of patients whose ulcers continued to deteriorate, 54.5% (n = 6) displayed ultrasonography evidence of damage that was located in the deep tissues. Deep tissue injury that was present below the erythematous areas was not identified and was misclassified as superficial skin changes. The current classification systems are inadequate and misleading in the description of skin changes with coexisting deep structural damage.

Third, the extent of tissue damage cannot be determined if the wound bed is obscured by slough and necrotic tissues. Although these wounds are regarded as unstageable by definition, unstageable PrUs that are covered by necrotic tissue represent full-thickness damage. The high number of unstageable wounds creates a reimbursement problem in care settings where payment is related to the stage of the PrU. The EPUAP has chosen to include what the NPUAP designates as unstageable as a Stage IV PrU.

Fourth, the level of tissue involvement reflects only 1 dimension of PrU severity; other descriptors/parameters (eg, signs of infection, advancing necrosis, increasing in size, tunnelling) are equally important and can negatively impact patient outcomes and quality of life.

STATEMENT 4
Because there are problems with both the validity and reliability of current classification systems, clinical practice guidelines and protocols based on these classification systems should not be considered the legal standard of care.

Although adopted by several practice guidelines, validity and reliability of the current classification systems of PrUs remain contentious. A Dutch National Prevalence survey identified 226 patients with Stage I PrUs, but half of the potential PrUs (49.7%) could no longer be detected upon reassessment on the same day. Results of this study raised concerns about the accuracy of PrU assessment based on current classification systems. Gunningberg and Ehrenberg compared the prevalence of PrUs based on individual chart documentation versus physical examination by 2 nurses. One hundred nine PrUs were identified (33.3%) upon examination, but only 59 (14.3%) of the ulcers were documented in patients’ records. The most conspicuous discrepancy was noticed in the assessment of Stage 1 PrUs; only 33% (27/80) of Stage I PrUs were documented. Although documentation practices may be below current standards, part of the discrepancy may be due to the lack of accurate criteria and methods for the assessment of Stage I PrUs. It may be preferable to omit Stage I ulcers from prevalence and incidence studies.

Reliability of the current classification system is deficient. In 1 study, 473 nurses were asked to classify 56 photographs of skin lesions as normal skin, blanchable erythema, PrUs (4 grades, EPUAP classification), or incontinence lesions. The multirater κ with good agreement should be 0.70, but was only 0.37 (P < .001). The same pictures were presented to another 86 nurses for classification on 2 separate occasions with an interval of 1 month. The intrarater agreement was poor, with the calculated κ = 0.38. Stausberg et al expected that accuracy can be refined by PrU classification system experience and knowledge. They recruited 5 nursing experts and 2 physicians to classify 100 wound images. The percentage of expert clinician agreement was only 63.5% for grading buttock and hip region PrUs.

Many variables may affect the accuracy of PrU classification. Early detection of visual skin changes including erythema and blanching response is particularly challenging for individuals with darkly pigmented skin. The difficulty distinguishing pigment irregularities from early PrU skin changes in darkly pigmented individuals may have explained the higher prevalence of PrUs among black long-term-care residents (18.2%) compared with the white residents (13.8%) in 619 long-term-care facilities (n = 59, 740).

Nonblanchable erythema is a nonspecific clinical sign. Differentiation between PrUs and moisture-associated skin damage or incontinence-associated dermatitis (IAD) and a variety of dermatologic conditions, such as contact irritant/allergic dermatitis,
candidiasis, and cutaneous fungal infections, has created another diagnostic challenge. Fungal-related skin lesions are often accompanied with scale and crust (defined in the SCALE [Skin Changes at Life’s End] document). Moisture-associated skin damage/IAD connotes the diffuse erythematous irritation and inflammation precipitated by prolonged exposure of the skin to perspiration, urine, or stool (Case Study 1). Denudement and erosion of the skin are common and attributable to several factors: weakening of intercellular bonds in the epidermal layer, damage from chemical irritants and digestive enzymes present in excrement, or physical trauma created during incontinence care. Despite the distinguishing and recognizable features between a PrU and moisture-associated skin damage, Beeckman et al reported that merely 44.5% of photographs were correctly classified as PrUs versus IAD by a cohort of nurses (n = 1217). Inappropriate classification of skin lesions may artificially inflate the prevalence of Stage I PrUs, exposing the responsible facility to unnecessary scrutiny by legal and regulatory bodies. It is best to omit Stage I ulcers from prevalence and incidence studies (report Stages II-IV, unstageable).

STATEMENT 5
Current PrU classification systems (staging, grading, or categories) have financial implications (penalties and/or reimbursement) and may place persons with PrUs and healthcare professionals in jeopardy.

Pressure ulcers are often adopted by organizations as a benchmark to indicate quality of care. The Centers for Medicare & Medicaid Services stipulates federal guidelines for the prevention and treatment of PrUs. If they fail to comply, healthcare organizations are subjected to financial penalties in the higher range of US $3050 to $10,000 for each day that immediate jeopardy exists (ie, development of avoidable Stage IV PrU increases the potential for serious complications). The impetus of regulatory changes is fueled by the common misconception that all tissue injury invariably begins with superficial damage (Stages I and II) and progresses to deep ulcers (Stages III and IV) because of negligence and poor quality of care. The propensity to connect PrU occurrence with substandard care has increased the frequency of litigation against responsible clinicians and organizations. In the United Kingdom, the National Health System receives about 10,000 new claims for clinical negligence annually, and this number is rising. Voss et al reviewed legal databases in the United States and reported that the number of long-term-care PrU-related legal cases per year increased from an average of 7 (1984 to August 31, 1999) to 18 (September 1, 1999, to April 12, 2002). The mean recovery cost for all residents inflicted with PrUs increased substantially from $3,359,259 in 1999 to $13,554,168 in 2002 (a 403% increase in...
was no evidence of a suspected pneumonia on chest X-ray, and sputum sample was negative for pathogenic bacteria. A condom catheter was used to control urinary incontinence. A skin swab from the buttock for bacteria grew normal bacterial flora but identified the presence of a yeast—*Candida albicans*. The yeast could be suspected with the presence of satellite papules and pustules around the edge of the incontinence-associated dermatitis.

For primary treatment of the skin, 1% hydrocortisone powder in clotrimazole (antiyeast, antifungal) cream was applied to the red skin. The hydrocortisone will treat the inflammation of the contact irritant dermatitis, and the clotrimazole will treat the coexisting yeast. By mixing the powder into the antifungal cream, this does not dilute the active components, as would be the case of mixing equal parts of 2 creams. To provide a barrier over the combination cream, zinc oxide ointment can be applied with a tongue depressor. The skin eruption cleared completely in 5 days.

When the red rash disappears, the zinc oxide barrier can be applied as a single layer.

The authors acknowledge that other related etiologies may not be elucidated by this theory (ischemic necrosis, device-related lesions, and multiple causative etiologies).

Development of PrUs is a dynamic and complex process that involves the combined effect of pressure, shear, and friction. Amid the confusion surrounding the pathophysiology of PrUs, increasing evidence lends support to distinct mechanisms that are responsible for pressure-related damage in superficial versus deep tissue compartments. It has been proposed that shear stress (and therefore the potential for injury) is more pronounced between the muscle and bones compared with any other layers of tissue. Shear stress is created as the more malleable muscular layer slides against the relatively stiff bony structure, and they move in opposite directions. Gefen reviewed existing literature pertaining to the effects of sitting on tissue loads and concluded that mechanical loads are consistently and considerably higher in areas overlying bony prominences, such as the ischial tuberosities, than overall interface pressures in the gluteal region. Shabshin et al evaluated the impact of various seating surfaces on internal soft-tissue deformations under the ischial tuberosities, using weight-bearing magnetic resonance imaging. They reached the conclusion that tissue deformations were maximal in muscle.

These studies implicated that deep ulcers evolve from inside out, starting at the deep tissue layer that is most vulnerable to pressure injury (Case Study 2). Early work by Witkowski and Parish demonstrated that the necrosis of subcutaneous fat tissue was evident in biopsies of intact skin with nonblanchable erythema (n = 14). Of interest, histological examination of the epidermis was normal. Several animal model experiments confirmed that pathological changes were initially observed in the muscle that seemed to be more sensitive to pressure-induced damage compared with the skin. Muscle has a high metabolic activity and is therefore inherently susceptible to pressure loading and related ischemia. Pressure changes have a lower threshold of injury when combined with shear. Mini-mizing shear (eg, positioning the head of bed at <30-degree angle) is important to prevent deep pressure-associated injury.

The Superficial Skin Changes & Deep Pressure Ulcer Framework is outlined in Table 2. This construct is a work in progress and warrants further revision as new evidence emerges to explain device-related lesions (such as those induced by catheters and oxygen tubing), hourglass or sandwich-shaped tissue necrosis with relatively healthy tissue sandwiched between shallow and deep necrosis, and other related phenomena.

**STATEMENT 8**
Intrinsic or extrinsic factors can modify the development of superficial skin changes and deep PrUs.
The pathogenesis of PrUs is multifactorial. Intrinsic factors reflect the general condition or health that may predispose individuals to the development of PrUs. Some of these key factors are poor nutritional intake, low body mass index (<18.5 kg/m²), hypoproteinemia, low systolic blood pressure, anemia, contractures and prominent bony prominences, vascular disease, neuropathy, and uncontrolled diabetes. In people with diabetes, hyperglycemia promotes thickening of collagen fibers compromising the tissues’ ability to change shape and handle mechanical loads. Based on mathematical modeling of soft tissue deformations caused by external pressure in sitting positions, Gefen remarked that variability in tissue deformation is influenced by body type and tissue thickness. The decreased tissue thickness is associated with more pronounced tissue deformation, potentially putting the person at risk for skin breakdown. In an experimental study of 14 healthy volunteers (mean age, 30.7 years), the maximum shear force at the coccyx was higher (P < .01) in slender than in obese individuals when the head of bed was raised from a supine position. Proposed extrinsic factors involved in the development of PrUs include skin temperature, moisture, infection, and incontinence. The role of skin surface temperature and humidity in the formation of PrUs warrants further scrutiny. An increase of 1°C (33.8°F) in skin temperature results in approximately a 13% increase in tissue metabolic requirements rendering the skin more vulnerable to mechanical damage.

Tissue composition and geometric shape of the supporting structure play a key role in PrU development. One of the areas that is most vulnerable to pressure-related skin damage is the heels (Case Study 2). The incidence of heel PrUs is approximately 19% to 32%. The heel has a pointed shape with a limited surface area of contact to redistribute pressure, and when this is combined with the low subcutaneous tissue volume, this area is prone to pressure damage. The heel tissue is enveloped within the fibrous septa that allow pressure to build up easily and occlude vascular supply.

**STATEMENT 9**

9A. The current concept of the Stage/Grade II PrU is a misconception because these superficial skin injuries (outside in) are primarily due to moisture and friction.

Superficial skin changes are primarily caused by excess moisture (skin surface may show maceration from increased moisture of the stratum corneum) and friction instead of pressure (Case Study 1). Using a porcine model, Dinsdale analyzed the role of pressure and friction in the production of PrUs. Hemorrhage and leukocyte filtration near the capillaries suggestive of tissue injuries only extended into the dermis. By reducing friction using special garments, 16% of PrUs were circumvented (P = .0286). A retrospective study using Mini-
fibers.56 The interruption of normal barrier function increases skin permeability to irritants and the risk for breakdown. Clinicians should pay more attention to moisture-related skin damage and friction injuries (2 surfaces rubbing against each other) to prevent and treat superficial skin damage.

9B. The Stage/Grade I PrU causation has yet to be fully elucidated and therefore, to improve clinical diagnostic precision, should be excluded from current classification systems until the causation is understood. In clinical practice, clinicians should document intact skin with superficial redness or discoloration as “redness or discoloration of intact skin” until further etiological research is available.

Recognizing the challenges of differentiating Stage I PrUs from deep tissue injury and other dermatologic conditions (fungal and yeast intertrigo, moisture-associated skin changes, contact dermatitis, psoriasis, and other skin conditions), the panel recommends close monitoring and documentation of skin changes over time.

Document the clinical changes. Stage II PrUs can simply be described as a partial-thickness skin loss that appears as an erosion (abrasion), blister, or shallow crater without slough. Stages III and IV PrUs are full-thickness skin loss with evidence of tissue damage or necrosis extending to the visible underlying structures (such as subcutaneous tissue, muscle, bone, tendons, and so on). Intact skin with suspected pressure damage can be documented as localized redness or purple discoloration or bruise-like color coupled with pain, increased warmth, bogginess, or induration.

STATEMENT 10

The stakeholders should develop an educational, clinical, and research agenda to validate the evidence base for the Superficial Skin Changes & Deep Pressure Ulcer Framework along with other future discoveries and insights.

Suspected deep tissue injury cannot be verified by visual inspection alone; therefore, other approaches are required. Studies using ultrasound, infrared thermometry, and other methods to measure subepidermal moisture level and tissue elasticity have yielded varying degrees of success in differentiating superficial from deep tissue damage.57–59 Even if this injury has occurred, the body’s reparative mechanisms may prevent ulceration in some cases. Further research is required to validate this proposed framework and render modifications as new evidence emerges. The future research agenda must address the identification of a noninvasive real-time assessment approach and analysis of tissue damage at the point of care to provide guidance for appropriate management. Education is integral to narrow the gap between current theory and improved patient outcomes in clinical practice.

CONCLUSIONS

Deficiencies in the current PrU classification system create the impetus for the current discourse on the clinical, legal, and economic implications of staging and considering shifting the paradigm for PrU assessment. Pressure ulcers do not usually progress in sequence from Stage I to Stage IV. The so-called Stage I PrUs are defined as intact skin and not an ulcer (dermal

---

Table 2

<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Superficial skin changes (outside in)</td>
<td>Grade/Stage II (see note below regarding Grade/Stage I)</td>
<td>Primarily due to moisture and friction</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Partial thickness</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Examples include skin tears,</td>
</tr>
<tr>
<td></td>
<td></td>
<td>incontinence-associated dermatitis, contact</td>
</tr>
<tr>
<td></td>
<td></td>
<td>dermatitis, friction-associated blisters</td>
</tr>
<tr>
<td>Deep PrUs (inside out)</td>
<td>Grade/Stage III</td>
<td>Primarily due to tissue deformation (compression, shear, and tension)</td>
</tr>
<tr>
<td></td>
<td>Grade/Stage IV</td>
<td>Full thickness</td>
</tr>
<tr>
<td></td>
<td>Suspected deep tissue injury</td>
<td>Not all suspected deep tissue injuries evolve into PrUs</td>
</tr>
<tr>
<td></td>
<td>Unstageable</td>
<td></td>
</tr>
</tbody>
</table>

Note: The Stage/Grade I PrU causation has yet to be fully elucidated and therefore, to improve clinical diagnostic precision, should be excluded from current classification systems until the causation is understood. Fixed erythema of the skin should be documented as intact skin with superficial redness as “erythema or discoloration of intact skin” until research demonstrates potential causative etiologies and their differentiation. 2010 Shifting the Original Paradigm Expert Panel (SOPE Panel).

The proposed delineation of superficial skin changes and deep PrUs based on distinct mechanisms would allow accurate communication of causative factors and resulting skin conditions. The proposed paradigm would also avoid unfair penalty as deep PrUs do not always begin as a superficial skin damage.

PRACTICE PEARLS

- Stage 1 PrUs are not true ulcers and may be due to multiple etiologies, including moisture-associated dermatitis and suspected deep tissue injury.
- Skin tears should not be included in PrU prevalence and incidence data.
- Superficial skin damage (often classified as PrUs) is frequently related to surface moisture and friction. The source of the injury needs to be identified and treated.
- Most Stages III and IV PrUs are associated with pressure and shear. They develop from the inside out and do not generally evolve from Stage I or II ulcers.
- Not all suspected deep tissue injury breaks down into Stages III and IV PrUs. There is often a lag period between the injury and the subsequent skin breakdown.

REFERENCES

15. Farid KJ. Applying observations from forensic science to understanding the development of pressure ulcers. Ostomy Wound Manage 2007;53(4):26-8, 30, 32.

For more than 70 additional continuing education articles related to Skin and Wound Care topics, go to NursingCenter.com/CE.