Grade 1 Pressure Ulcers: We Only See What We Know

This month’s continuing medical education activity (page 571) by Sibbald, Krasner, Woo, and the Original Paradigm Expert Panel (SOPE Panel) develops a set of new lenses and frames by which we can see the reasons to reconceptualize the venerable grade 1 pressure ulcer (PrU). The long-held clinical beliefs about grade 1 PrUs have been the source of review, criticism, and discourse for more than a generation. For many of us who have been taught that PrUs follow a certain trajectory, which can be visualized and furthermore be graded, the article by Sibbald et al gives us a powerful reason to think about how we formulate our clinical beliefs.

Sensitivity and Specificity
Our ability to make an accurate diagnosis for a grade 1 PrU is currently elusive given the lack of technology available to measure sensitivity and specificity or the combined sensitivity or specificity of the target tissues. If the “nonblanching erythema” is considered our visual inspection test (VIT) for an impending PrU, then we must apply rigor to the value of the test. In general, the more sensitive a test is for a disease, the higher its false-positive rate, thus lowering its specificity. Typically, a highly sensitive test is ideal for a screening examination (in our case, the VIT), whereas highly specific tests are best in a confirmatory role. The mnemonics SnOut (Sensitive tests rule Out the condition when they are negative) and SpIn (Specific tests rule In the condition when they are positive) provide some guidelines on how to interpret sensitivity and specificity for an individual patient.1,2

Applying this concept to our ability to accurately diagnose blanching erythema utilizing our VIT is best described as neither diagnostically sensitive nor specific. Of all the ordinal grades describing PrUs, the term grade 1 is more nominal. In addition to the lack of specificity in confirmatory testing issues related to describing PrUs is our language. There are more questions than answers—not only about the meaning of a grade 1 PrU, but also about the words we use to communicate among ourselves about wound care. I refer to this as the taxonomy of discourse.

Taxonomy of Discourse
In the scientific sense, taxonomy is a method of classification. It is the agreed-upon nomenclature we use to categorize, catalog, arrange, and organize a given biologic or clinical model. In some instances, there is international consensus about the nomenclature of specific problems. An example of this is the World Health Organization’s International Classification of Diseases. Taxonomy (classification) gives us the ability to engage in discourse (communication) in a common language. With this common language, advances can occur. This is an important concept for wound care practitioners to debate and incorporate. For example, being able to use appropriate descriptors for the types of wounds we evaluate and treat will help us communicate more effectively among ourselves and with policy makers, researchers, and patients. It will also facilitate communication with wound care practitioners throughout the world, solidifying the “one world” concept of our increasingly global economy.3

Taxonomy of Discourse in Action
We do not have to look far to examine the changing language of wound care; the metamorphosis of our journal’s name is a perfect example. From Decubitus, the journal focused on PrUs; by 1994 the readership’s interests had reached far beyond PrUs, and the name of the journal was changed to Advances in Wound Care. And in 2000, we changed the name again, to Advances in Skin & Wound Care. The inclusion of skin—the largest organ in the body—in the title more appropriately reflects that this total organ system is at risk for wounds. Wound care professionals from other countries, who use a slightly different taxonomy of discourse, provide us the ability to “cross-walk” our wound care languages, exemplified by the current manuscript by the SOPE panel that is advancing our discourse.

A scientific axiom that exemplifies how we crystallize our clinical acumen is attributed to “Indeed, we can only see what we know—that what we have been taught to see.”

“We only see what we know.” —Johann Wolfgang von Goethe (1749–1832).4

Richard “Sal” Salcido, MD

References
4. Sonnenberg A. We only see what we already know—a modified Bayes’ formula to explain inherent limitations of diagnostic tests. Med Hypotheses 2004;63(4):759-63.
Pressure Ulcer Staging Revisited: Superficial Skin Changes & Deep Pressure Ulcer Framework

1. In compliance with the Shifting the Original Paradigm Expert (SOPE) Panel proposal, the clinician now documents a wound's:
   a. superficial moisture.
   b. shear risk.
   c. skin blanching.
   d. staging.

2. Mr. A. had a perpendicular force applied to his ankle that distorted and compressed his underlying soft tissue. The clinician describes the force that caused the PrU as:
   a. shear.
   b. friction.
   c. pressure.
   d. deep tissue damage.

3. An obese patient who is unable to reposition herself in bed tends to slide while her skin remains stationary on the bed surface. Thus, her muscular layer and bony structure move in opposite directions. The clinician is particularly concerned about development of a sacral ulcer due to:
   a. shear.
   b. friction.
   c. pressure.
   d. moisture.

ENROLLMENT FORM: Advances in Skin & Wound Care, December 2011
Pressure Ulcer Staging Revisited: Superficial Skin Changes & Deep Pressure Ulcer Framework®

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Demonstrate advantages of using the superficial skin changes and PrU framework for documentation.
How many of your patients are likely to be impacted by what you learned from this activity?
"20%"  "20%-40%"  "40%-60%"  "60%-80%"  ">80%"

Do you expect that this activity will help you improve your skill or judgment within the next 6 months? (1 = definitely will not change, 5 = definitely will change)
□  □  □  □  □

How will you apply what you learned from this activity (mark all that apply):
□ In diagnosing patients □ In making treatment decisions □
□ In monitoring patients □ As a foundation to learn more □
□ In educating students and colleagues □ In educating patients and their caregivers □
□ As part of a quality or performance improvement project □ To confirm current practice □
□ For maintenance of board certification □ For maintenance of licensure □

How committed are you to applying this activity to your practice in the ways you indicated above? (1 = minimally, 5 = completely)
□  □  □  □  □

Did you perceive any bias for or against any commercial products or devices?
   Yes □ No □
   If yes, please explain: ________________________________

How long did it take you to complete this activity? ______ hours ______ minutes

What are your biggest clinical challenges related to skin and wound care?

[ ] Yes! I am interested in receiving future CME programs from Lippincott CME Institute! (Please place a check mark in the box.)

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4. Ms C. wants to prevent her bedridden father from developing a PrU on his buttocks. The clinician explains that the degree of internal stress and potential for tissue injury is determined by a. the skin’s moisture content. b. his history of previous PrUs on other body parts. c. the synergistic effect of pressure, shear, and/or friction. d. repositioning, skin perfusion, and presence of existing dermatitis.

5. In reviewing the literature, the clinician learns that low pressure over a protracted duration a. protects skin integrity. b. is less harmful than high pressure for a short duration. c. will result in a PrU. d. is just as detrimental as excessive mechanical loads.

6. The evidence suggests that most Stage III and Stage IV PrUs a. started at Stage I PrU and progress to Stage IV PrU. b. can be explained by the outside-in theory of PrU progression. c. originate in a deep tissue compartment and progress outward to dermis. d. begin to deteriorate at the epidermis then quickly moves inward to tissue and muscle.

7. The clinician observes that Mrs F. has a maroon bruise under intact skin. This is best documented as a. Stage I PrU with a maroon bruise. b. suspected deep tissue (sDTI) injury. c. a contact dermatitis due to moisture. d. a bruise most likely from trauma and not related to pressure.

8. The clinician informs the student that for clarity, the SOPE Panel proposes that PrUs be documented using all of the following except a. size, base, and exudate findings. b. partial- and full-thickness depth. c. the customary numerical classification system. d. identification of location and margins.

9. Mr G.’s Stage III PrU has been healing quickly. The nurse documents the PrU today as Stage I. The clinician asks the nurse about the staging change and explains that during the PrU healing process a. physiological back-staging is an accurate description.

b. as a Stage III or IV PrU heals, it replicates the tissues that were previously present. c. a full-thickness PrU heals to a partial-thickness PrU, then total resolution occurs. d. full-thickness ulcers are replaced by granulation tissue and scar tissue.

10. Ms H. asks the clinician about how to prevent Stage I PrUs from progressing to Stage IV PrUs. The clinician explains that interventions to prevent further tissue damage a. are highly successful if prevention begins at initial diagnosis. b. may not prevent Stage IV PrUs due to inaccuracies with initial PrU staging. c. are dependent on the skill of the healthcare providers performing interventions. d. are beneficial in patients motivated to participate in their own care.

11. The patient’s PrU is documented as a Stage II. The clinician examines the patient and determines that advancing necrosis is present in addition to tunneling and signs of infection. The clinician decides that this PrU is a. staged appropriately. b. healing well with the present treatment. c. not appropriately staged because other parameters were overlooked. d. unstageable.

12. The student asks the clinician about the accuracy of the staging system of PrUs. The clinician explains that the numerical classification system of PrUs a. has questionable validity and reliability. b. has high reliability and validity for Stage I wounds only. c. was proven accurate by the Dutch National Prevalence survey. d. has been proven valid through multiple inter-rater reliability studies.

13. Ms K. is a dark-skinned African-American woman in long-term care. She was diagnosed with a Stage I PrU yesterday due to non-blanchable erythema. Today, the clinician questions that finding because a. skin changes are more likely due to Candida dermatitis than PrUs. b. skin changes are more easily found on dark pigmented skin than lighter skin. c. research has demonstrated a lower prevalence of PrUs among dark-skinned long-term-care residents. d. dark pigmentation makes erythema and blanching more difficult to assess.

14. Mr L. is receiving end-of-life care. Although there are no PrUs present at this time, the clinician explains to the family that a recent consensus conference determined that PrUs a. can be avoided by administering oxygen. b. are largely preventable but not always avoidable. c. will not occur unless the patient is receiving poor skin care. d. are more likely to occur in patients with a family history of PrUs.

15. In a patient with limited mobility, the clinician is concerned about shear stress occurring primarily between a. muscle and bone. b. bone and cartilage. c. muscle and subcutaneous tissue. d. subcutaneous tissue and dermis/epidermis.

16. The clinician assesses Mrs M.’s skin and finds full-thickness skin changes. Using new SOPE Panel terminology and conceptual frameworks, this would be documented as a. a Stage III PrU. b. a sDTI. c. an outside-in damage. d. deep PrU.

17. The clinician documents superficial skin changes. For reimbursement purposes, the facility’s billing department asks the clinician to provide a classification stage for it. The clinician classifies this as a/an a. unstageable PrU. b. Stage I PrU. c. Stage II PrU. d. Stage III PrU.

18. Mr P. has superficial skin changes present on his buttocks. He wants to know how these changes occurred. The clinician explains that these skin changes are primarily due to a. shear and tension. b. moisture and friction. c. immobility. d. pressure and compression.

19. Following SOPE Panel recommendations, the clinician documents “partial-thickness skin loss that appears as a shallow crater without slough.” This is preferable to staging it as a/an a. Stage II PrU. b. Stage III PrU. c. unstageable PrU. d. sDTI.
Pressure Ulcer Staging Revisited: Superficial Skin Changes & Deep Pressure Ulcer Framework

R. Gary Sibbald, BSc, MD, MEd, FRCP (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.

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To earn CME credit, you must read the CME article and complete the quiz and evaluation on the enclosed answer form, answering at least 14 of the 19 questions correctly. This continuing educational activity will expire for physicians on December 31, 2012.

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.
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. 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

| R. Gary Sibbald, BSc, MD, MEd, FRCPC (Med Derm), MACP, FAAD, MAPWCA, Co-chairperson |
| Diane L. Krasner, PhD, RN, CWCN, CWS, MAPWCA, FAAN, Co-chairperson |
| Kevin Woo, PhD, RN, ACNP, FAPWCA, Medical writer |
| Cynthia Sylvia, MSc, MA, RN, CWCN, Panel facilitator |
| Michael S. Brogan, DPT, PhD, CWS, FACCWS |
| Karen J. Farid, BSN, MA, CWN, DNP(c) |
| Caroline E. Fife, MD |
| Amit Gefen, PhD |
| Karen Lou Kennedy-Evans, RN, CS, FNP |
| Jan Kottner, PhD, RN |
| Steven R. Kravitz, DPM, FAPWCA, FACFAS |
| Thomas Serena, MD, FACS |
| Thomas P. Stewart, PhD |
| David R. Thomas, MD, FACP, AGSF, GSAF |
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 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 I 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,
Moisture-associated skin weight loss and urinary incontinence. 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

### CASE STUDY 1

This 85-year-old man presented with failure to thrive and coped independently with:
- weight loss
- diarrhea with fecal incontinence
- urinary incontinence.

The patient was admitted to the hospital and classified with a Stage II pressure ulcer due to the erosion on the upper left buttock. A hydrocolloid dressing was initially used to facilitate healing of the ulcer with a marked worsening of the local skin condition. A dermatology consult was ordered.

The failure to thrive was due to undiagnosed diabetes and congestive heart failure. The patient was in an incontinent brief with frequent incontinence of stool and/or urine, resulting in excessive moisture. Thus, it was difficult for the nurses to change the incontinent brief fast enough to keep up with the constant moisture.

The primary diagnosis of the skin disorder is a variant of contact irritant dermatitis called moisture-associated dermatitis. The area of skin erosion is not over a bony prominence, and there are irregular margins to the red areas of dermatitis and not the smooth, well-demarked edges that would be confined to an area of pressure in a Stage/level II pressure ulcer. In addition to moisture, the diaper rubbing against the skin when the patient moves will lead to friction between the 2 surfaces, aggravating the local skin injury.

There is no pressure component to this skin rash, and this is not a pressure ulcer.

Treatment consisted of establishing a bowel routine to facilitate evacuation of stool 3 times per week once Clostridium difficile colitis was ruled out. Clinicians also recommended stopping systemic antibiotics because there
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 re-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 implicate 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 shall and deep necrosis, and other related phenomena.

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**STATEMENT 6**

Some patients may develop PrUs despite clinical practices that are based on current evidence or standards of care.

Much debate had been centered on the preventability of PrUs. To date, there is no evidence that PrUs can be completely prevented. In a recent consensus conference that involved 24 stakeholder organizations from various disciplines, it was unanimously agreed that PrUs are largely preventable but not always avoidable. To maintain hemodynamic stability and local temperature change. Development of PrUs may also be unavoidable when prevention strategies are deemed futile or even precarious. For instance, repositioning may precipitate vascular collapse or exacerbate shortness of breath.

**STATEMENT 7**

The Superficial Skin Changes & Deep Pressure Ulcer Framework proposes that superficial skin changes occur from the outside in, and deep PrUs from the inside out. Current PrU classifications confuse this reality and need to be modified.

**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 Minimum Data Set data consisted of 29,040 observations on 13,457 residents at 108 skilled nursing facilities validated that incontinent residents were at risk for developing PrUs (odds ratio, 1.4; 95% confidence interval, 1.1-1.6). Excessive moisture leads to overhydration or maceration of the skin, weakening connective tissues that is most vulnerable to pressure-related skin damage. The incidence of heel PrUs is approximately 19% to 32%. The heel has a pointed shape with 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.

**CASE STUDY 2**

A 60-year-old man with diabetes presented with a cold painful foot and an absent pedal pulse.

In the interventional radiology suite, he had an angiogram of the left leg with dilation of his anterior tibial artery. There was a return of the dorsalis pedis pulse and a warm foot. The patient was on the stretcher in the interventional suite hallway for 4 hours. When he returned to the medical floor, dusky erythema was noted in the heel region that subsequently became necrotic over a couple of days and gradually ulcerated over the next week despite the use of a heel-protective device to keep the heel off the bed when recumbent.

An examination of the foot revealed a black eschar 7 × 4 cm with a blister on the lateral surface that was drained to reveal purulent contents. A diagnosis of gangrene with proximal cellulitis and purulent blister was made. The patient was treated with systemic clavulanic acid + amoxicillin combination. Topically, the wound was painted with a povidone-iodine preparation (maintenance wound) until the deep infection was controlled. Subsequently, conservative surgical debridement was facilitated by the use of a hydrogel dressing. (See “Special Preparations in Wound Bed Preparation 2011: An Update” in the September 2011 issue of Advances in Skin & Wound Care.) The wound healed over a 6-week period.

This is a description of a deep tissue injury due to a combination of sustained pressure with shear due to the frequent repositioning of the leg during the procedure. The injury started at the muscular layer and subsequently ulcerated on the skin surface. The vascular compromise, infection, and potential reperfusion injury after revascularization all contributed to the skin breakdown.
The interruption of normal barrier function increases skin permeability to irritants and increases 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 suspected pressure damage 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, boggi- ness, or induration.

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<tr>
<td>Superficial skin changes (outside in)</td>
<td>aGrade/Stage II (see note below regarding Grade/Stage I)</td>
<td>Primarily due to moisture and friction Partial thickness Examples include skin tears, incontinence-associated dermatitis, contact dermatitis, friction-associated blisters</td>
</tr>
<tr>
<td>Deep PrUs (inside out)</td>
<td>aGrade/Stage III</td>
<td>Primarily due to tissue deformation (compression, shear, and tension) Full thickness Not all suspected deep tissue injuries evolve into PrUs</td>
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<td>aGrade/Stage IV</td>
<td>aSuspected deep tissue injury</td>
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<td>aUnstageable</td>
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aPressure ulcer staging (NPUAP system) or grades (EPUAP system). Suspected deep tissue injury and unstageable categories in the NPUAP system are included in grade 4 ulcers of the EPUAP system.

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).

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...
Stage 1 PrUs are not true ulcers and may be due to
Not all suspected deep tissue injury breaks down into Stages
kin Wound Care 2010;23:225-36.
Skin tears should not be included in PrU prevalence and
content and patient examination. J Wound
Superficial skin damage (often classified as PrUs) is frequently
L, Van Hecke A, Vanderwee K. Pressure ulcers: development
& state: evidence of racial disparity? Med
Friction and Microclimate in Context. A
Reverse staging to monitor pressure ulcer
So 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.
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Controversies in Pressure Ulcer Classification Systems

In December 2011, a panel of experts rocked the pressure ulcer world by attacking some of the underpinnings of the current pressure ulcer classification systems (Staging, Grading, Categories). They said that some of the language creates problems from clinical, regulatory, legal and economic perspectives. The advisory panel is proposing the new Superficial Changes & Deep Pressure Ulcer Theory©. Here is one piece of what they asserted:

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

The current numerical pressure ulcer classification systems are intended to describe the anatomic depth of tissue damage. Stage 1 is characterized by non-blanchable erythema of intact skin that may be coupled with alterations in skin temperature and tissue consistency. Stage 2 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 3 pressure ulcers and to deeper supporting structures such as muscle, fascia, joint capsule and bone that are classified as stage 4 pressure ulcers. Evolution of pressure ulcers does not necessarily follow a predictable linear pattern from superficial to deep; from Stage 1 ulcers to Stage 2, then to Stage 3 and finally Stage 4 ulcers.

Accumulating evidence suggests that a number of pressure ulcers (most Stage 3 and 4 ulcers) may initially originate in the deep tissue compartment and progress outward to the dermis and epidermis (inside out theory). Deep tissue injury may not be visible to naked eyes 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. Deep tissue injury has the appearance of a purple or maroon bruise under intact skin that resembles and is often mistaken for a stage 1 pressure ulcer. Donnelly documented that 10% of pressure ulcers were initially diagnosed as stage 1 by visual inspection and evolved to stage 3/4 within days. It is possible that a proportion of the stage 1 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 dermatological diagnoses including; moisture associated dermatitis, fungal or yeast intertrigo or other dermatological 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.

Modified with permission from Dr. Diane Krasner, one of the authors of the Shifting the Original Paradigm article published in Advances in Skin and Wound Care December 2011.
Bed Entrapment: Killed by the Mattress

Selecting the correct mattress for pressure relief should take into account these factors in MATTRESS:

- Microclimate and moisture
- Activity levels
- Tissue tolerance
- Total body weight
- Repositioning needs
- Edema
- Shear and friction
- Symptom management

When considering support surfaces in bed, healthcare providers must consider the risk of entrapment. Health Canada and the FDA have released documents defining the seven zones of entrapment and guidance measurements:

1. Within the bed rail
2. Under the rail
3. Between the rail and the mattress
4. Under the rail at rail ends
5. Between split bed rails
6. Between end of rail and side edge of head or foot board
7. Between head or foot board and mattress end.

Prescription of a therapeutic support surface, whether an overlay or mattress replacement, may impact several of these zones (e.g. zone 2, 3, and 7). A standard measuring device is available to check to see if the new support surface increases the risk of entrapment by allowing spaces greater than those outlined in the guideline. The risk of entrapment may also be greater with support surfaces with large air bladders (these are usually found on low air loss, alternating, or rotating surfaces). These surfaces tend to collapse the further the individual moves to the edge of the surface, even when a perimeter border is present within the mattress.

When an entrapment risk has been identified, bed rails should only be used with extreme caution, and be based on the needs of the individual patient. Some patients find the half bed rail at the head section helpful for repositioning. Another approach for people at high risk is to use an adjustable bed with a very low deck height and a floor mat. This approach allows the bed to be raised during care, to a comfortable height for care providers, but allows the bed to be low enough to help prevent injury if the person falls out of bed. Foam wedges and other devices are also available to help reduce the risk of entrapment.

The standard of care will focus on correct selection of the mattress based on identification of risk, and monitoring the patient.

Modified with permission from Kestral Woundsource Devices White Paper, coauthored by Dr. Diane Krasner, November 2011
Liability: Wrong Wound Care Treatment

Many wound care clinicians remember the “good old days” when wound dressing product selection simply involved choosing between a handful of products that were essentially variations on the same theme. There was gauze, impregnated gauze and filled gauze pads. In the earlier 20th century, clinicians added antimicrobial solutions, creams and ointments (like Dakin's solution developed during World War I and silver sulfadiazine developed in the 1960's) and the wound care formulary was limited and simplistic.

Fast forward to the 21st century and wound care clinicians are confronted with a totally different situation: hundreds of products, scientific rationale for moist interactive dressings and an emerging evidence-base for product selection.

Current wound care expertise encompasses numerous dressing-related skills including:

- Treating the cause of the wound and addressing patient centered concerns to set the stage for local wound care
- Properly assessing the wound and identifying the dressing requirements
- Selecting dressings based on their form and function for an individual wound's needs
- Meeting setting-specific requirements for dressing change frequency and maintenance
- Addressing formulary or healthcare system availability as well as reimbursement requirements

Wound care product selection today must be as sophisticated and as evidence-based as possible. This Kestrel White Paper presents a conceptual framework for the wound dressing product selection process that is based on three principles:

- Holistic Perspectives
- Interprofessional Considerations
- Patient-Centered Concerns

Selecting appropriate wound dressing products and supportive care to maximize healing and patient outcomes is a complex process. Dressing and local wound care options based on science and best practices must be filtered by clinical experience and must be consistent with patient preferences, care-giver requirements and setting/access issues. Additionally, effective dressing selection and local wound care planning involve the perspectives of the entire interprofessional team.

Knowing the performance parameters of dressing categories/individual products and matching these attributes to an individual’s wound can optimize the healing process. But dressings are only one piece of the puzzle. Dressings alone will not promote wound healing, unless the underlying cause(s) for the wound are also addressed (e.g. treatment of the wound cause, blood supply, nutrition, patient centered concerns, local wound care etc.). As the wound changes, the plan of care must change and dressing products may have to be changed. Appropriate dressing product selection:

- Optimizes the local wound healing environment
- Reduces local pain and suffering
- Improves activities of daily living and quality of life
Inappropriate dressing selection can:

- Cause the wound status to deteriorate (e.g. wound margin maceration, increased risk of superficial critical colonization or deep infection, skin stripping).
- Increase local pressure or pain especially at dressing change (dressing removal and cleansing).
- Increase costs with the need for frequent dressing changes or the selection of an inappropriate advanced or active dressing.

National and international wound care guidelines and best practice documents mean that there is no longer a local standard of care. No matter where you practice, you will be held to national/international standards of wound care practice. Some experts have argued that the selection of the wrong dressing is just as problematic as the administration of the wrong drug and the clinician would be just as liable in a court of law. If dressings can be shown to delay the healing process (e.g. wet-to-dry gauze dressings in a wound that requires moist wound healing, pain from inappropriate adhesives, failure to treat critical colonization that can lead to deep infection), their use might be deemed negligent by a jury in a court case.

Modified with permission from Dr. Diane Krasner, coauthor of Wound Dressing Product Selection, 2010
Skin Changes at End of Life

Dr. Alois Alzheimer was on call in 1901 when a 51-year-old woman, Frau Auguste D., was admitted to his asylum for the insane in Frankfort. Dr. Alzheimer followed this patient, studied her symptoms and presented her case to his colleagues as what came to be known as Alzheimer's Disease. When Frau Auguste D. died on April 8, 1906, her medical record listed the cause of death as “septicemia due to decubitus.” Alzheimer noted, “at the end, she was confined to bed in a fetal position, was incontinent and in spite of all the care and attention given to her, she suffered from decubitus.” So, here we have the first identified patient with Alzheimer's Disease having developed immobility and two pressure ulcers with end stage Alzheimer's. In our modern times, end stage Alzheimer's Disease has become an all-too-frequent scenario with multiple complications including SCALE (Skin Changes at Life's End).

Also known as Kennedy Terminal Ulcers, these are a specific subgroup of pressure ulcers that some individuals develop as they are dying. They are usually shaped like a pear, butterfly, or horseshoe, and are located predominantly on the coccyx or sacrum (but have been reported in other anatomical areas). The ulcers are a variety of colors including red, yellow or black, are sudden in onset, typically deteriorate rapidly, and usually indicate that death is imminent.

Physiologic changes that occur as a result of the dying process (days to weeks) may affect the skin and soft tissues. These changes may manifest as observable (objective) changes in skin color, turgor, or integrity, or as subjective symptoms such as localized pain. Here is the medical legal issue: clinicians assert that these changes can be unavoidable and may occur with the application of appropriate interventions that meet or exceed the standard of care.

When the dying process compromises the homeostatic mechanisms of the body, a number of vital organs may become compromised. The body may react by shunting blood away from the skin to these vital organs, resulting in decreased skin and soft tissue perfusion and a reduction of the normal cutaneous metabolic processes. Minor insults can lead to major complications such as skin hemorrhage, gangrene, infection, skin tears and pressure ulcers that may be markers of SCALE.

Skin changes at life’s end are a reflection of compromised skin (reduced soft tissue perfusion, decreased tolerance to external insults, and impaired removal of metabolic wastes). When a patient experiences SCALE, tolerance to external insults (such as pressure) decreases to such an extent that it may become clinically and logistically impossible to prevent skin breakdown and the possible invasion of the skin by microorganisms. Compromised immune response may also play an important role, especially with advanced cancer patients and with the administration of corticosteroids and other immunosuppressant agents.

Skin changes may develop at life’s end despite optimal care, as it may be impossible to protect the skin from environmental insults in its compromised state. These changes are often related to other cofactors including aging, co-existing diseases and drug adverse events. SCALE, by definition occurs at life’s end, but skin compromise may not be limited to end of life situations; it may also occur with acute or chronic illnesses, and in the context of multiple organ failure that is not limited to the end of life.

Modified with permission for Dr. Diane Krasner, a coauthor of Skin Changes at Life’s End, SCALE Final Consensus Statement, October 1, 2009
The Sore in Pressure Sore

Bill is a 70 year-old man who developed paraplegia. During his prolonged hospitalization, a stage IV pressure sore formed. One year later, it is still present and it dominates his life at home. Pressure sores may have a huge impact on the quality of a patient’s life. There is a financial impact of prolonged treatment – dressing changes, supplies, debridements, flap surgeries. There is a medical impact of complications and risk of death from sepsis. There is a personal impact of physical restrictions, social isolation, loss of independence, and emotional problems. There is dealing with odors and limitations on the length of time one can sit. But pain is one of the biggest factors that affects the quality of the patient’s life.

A variety of pain scales are used to measure that which is subjective. The universally accepted measurement techniques are the utilization of visual analog scales (10-cm line with no pain at one end and worst possible pain at the other end, and the patient places an “x” at the appropriate point), Faces Pain Scale (various levels of happy and sad faces), or the numerical rating scale. The numerical rating scale asks if the patient has any pain on a 0- to 10-point scale with the anchors that 0 is no pain, 5 is the pain associated with a bee sting, and 10 would be the amount of pain experienced by slamming the car door on your thumb. Even in patients who cannot respond verbally, such as those with dementia, pain still needs to be assessed. There are pain scales for these patients that rely on nonverbal clues such as facial grimaces and pupil dilatation. (Assessment of pain for people with dementia can be found at www.hartfordign.org.)

Pain levels should be recorded before dressing change, during dressing change, and after dressing reapplication. Krasner has defined wound associated pain at dressing change (intermittent and recurrent) versus incident pain from debridement or the persistent pain between dressing changes. Woo carried the Krasner concept further and demonstrated that anxiety and other patient-related factors could intensify the pain experience.

The Wound Associated Pain Model of Woo and Sibbald defines pain from the cause of the wound as often being persistent or present between dressing changes and distinguishes this pain from the pain associated with local wound care components (dressing change, debridement, infection, lack of moisture balance). All of these factors can be modified by patient-centered concerns, including previous pain experience, anxiety, depression, mobility and awareness or lack of comfort with the setting, and the procedure or treatment plan. Pain is an under-recognized and undertreated component of chronic wound care that has been demonstrated to be more important to patients than healthcare professionals. Causes of pain at dressing change include the dressing material adhering to wound base, skin stripping from strong adhesives, and aggressive trauma from cleansing technique (eg, scrubbing with gauze).

Many patients also express chronic persistent pain between dressing changes even at rest. A systematized approach should examine other systemic and disease factors that may play a role in precipitating and sustaining persistent wound-related pain. Common systemic factors are bacterial damage from superficial critical colonization or deep and surrounding compartment infections, deep structural damage (eg, acute Charcot foot in patients with diabetes), abnormal inflammatory conditions (eg, vasculitis, pyoderma gangrenosum), or periwound contact irritant skin damage from enzyme-rich wound exudate.
Bill has a Morphine pump in his abdomen to deal with his pain. He takes supplemental Morphine by mouth. There are times he sleeps all day and is awake all night. The impact of chronic unrelenting pain can be devastating, eroding the individual’s quality of life and constituting a significant amount of stress. Increased levels of stress have been demonstrated to lower pain threshold and decrease tolerance. The result is a vicious cycle of pain, stress/anxiety, anticipation of pain, and worsening of pain. Increased stress also activates the hypothalamus-pituitary-adrenal axis, producing hormones that modulate the immune system compromising normal wound healing. Medications including nonnarcotic for moderate pain and narcotic analgesics for moderate to severe pain are required to treat severe pain as outlined below. A consult from a pain and symptom management team may be considered. Comprehensive management should also include careful selection of atraumatic dressing, prevention of local trauma, treatment of infection, patient empowerment, stress reduction, and patient education.

Modified by Pat Iyer with permission from Dr. Diane Krasner, coauthor of Special Considerations in Wound Bed Preparation 2011, an Update, Advances in Skin and Wound Care, September 2011
The Wound that Does Not Heal

Chronic, nonhealing wounds are disabling and constitute a significant burden on patients’ activities of daily living (ADLS) and the healthcare system. Of persons with diabetes, 2% to 3% develop a foot ulcer annually, whereas the lifetime risk of a person with diabetes developing a foot ulcer is as high as 25%. It is estimated that venous leg ulcers affect 1% of the adult population and 3.6% of people older than 65 years. As our society continues to age, the problem of pressure ulcers is growing. Each of these common types of chronic wounds will require accurate and concise diagnosis and appropriate treatment.

For patient wounds that do not have the ability to heal, the approach is different. These individuals with the inability to heal (nonhealable wound) may be due to inadequate blood supply and/or the inability to treat the cause or wound-exacerbating factors that cannot be corrected. There may be systemic disease, nutritional impairments or medications that delay or inhibit healing. When a healable wound does not progress at the expected rate, a chronic and stalled wound results. These wounds are more prevalent in older adults and are attributed to the aged skin and comorbidities, such as neuropathy, coexisting arterial compromise, edema, unrelieved pressure, inadequate protein intake, coexisting malignancy, and some medications. Persistent inflammation may be the cause of a stalled wound and in some cases may not be correctable. The presence of multiple illnesses in some older adult patients implies that healing is not a realistic end point.

The second category, a maintenance wound, is when the patient refuses the treatment of the cause (eg, will not wear compression) or a health system error or barrier (no plantar pressure redistribution is provided in the form of footwear or the patient cannot afford the device). These may change, and periodic re-evaluation may be indicated.

Chronic wounds are often recalcitrant to healing, and they may not follow the expected pathway that estimates a wound should be 30% smaller (surface area) at week 4 to heal in 12 weeks.

In the medical legal world there may be an assumption that most if not all wounds can be healed with proper care. In the medical world, what percentage of wounds are considered nonhealable? In a study of 173 wounds, 70% were considered healable, 25% were considered maintenance, and 5% were considered nonhealable including skin changes at life’s end.

Modified with permission from Dr. Diane Krasner, coauthor of Special Considerations in Wound Bed Preparation 2011, an Update, Advances in Skin and Wound Care, September 2011
Abstract

An expert panel was established to formulate a consensus statement on Skin Changes At Life’s End (SCALE). The panel consists of 18 internationally recognized key opinion leaders including clinicians, caregivers, medical researchers, legal experts, academicians, a medical writer and leaders of professional organizations. The inaugural forum was held on April 4-6, 2008 in Chicago, IL, and was made possible by an unrestricted educational grant from Gaymar Industries, Inc. The panel discussed the nature of SCALE, including the proposed concepts of the Kennedy Terminal Ulcer (KTU) and skin failure along with other end of life skin changes. The final consensus document and statements were edited and reviewed by the panel after the meeting. The document and statements were initially externally reviewed by 49 international distinguished reviewers. A modified Delphi process was used to determine the final statements and 51 international distinguished reviewers reached consensus on the final statements.

The skin is the body’s largest organ and like any other organ is subject to a loss of integrity. It has an increased risk for injury due to both internal and external insults. The panel concluded that: our current comprehension of skin changes that can occur at life’s end is limited; that SCALE process is insidious and difficult to prospectively determine; additional research and expert consensus is necessary; and contrary to popular myth, not all pressure ulcers are avoidable.

Specific areas requiring research and consensus include: 1) the identification of critical etiological and pathophysiological factors involved in SCALE, 2) clinical and diagnostic criteria for describing conditions identified with SCALE, and 3) recommendations for evidence-informed pathways of care.

The statements from this consensus document are designed to facilitate the implementation of knowledge-transfer-into-practice techniques for quality patient outcomes. This implementation process should include interprofessional teams (clinicians, lay people and policy makers) concerned with the care of individuals at life’s end to adequately address the medical, social, legal, and financial ramifications of SCALE.

The statements from this consensus document are designed to facilitate the implementation of knowledge-transfer-into-practice techniques for quality patient outcomes. This implementation process should include interprofessional teams (clinicians, lay people and policy makers) concerned with the care of individuals at life’s end to adequately address the medical, social, legal, and financial ramifications of SCALE.

The content of this document is based on the results of a two-day round table discussion held on April 4-6, 2008 in Chicago, IL, and was made possible by an unrestricted educational grant from Gaymar Industries, Inc. Additional input was received from an international panel of 49 and 51 distinguished reviewers using a modified Delphi Method process. The information contained herein does not necessarily represent the opinions of all panel members, distinguished reviewers, or Gaymar Industries, Inc.

Disclaimer: The content of this document is intended for general information purposes and is not intended to be a substitute for medical or legal advice. Do not rely on information in this article in place of medical or legal advice.

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SCALE Expert Panel Members

Co-Chairpersons
R. Gary Sibbald, BSc, MD, FRCPC (Med, Derm), MACP, FAAD, MEd, FAPWCA University of Toronto, Toronto, Canada, gary.sibbald@utoronto.ca

Diane L. Krasner, PhD, RN, CWCN, CWS, BCLNC, FAAN, Wound & Skin Care Consultant, York, PA, USA, dlkrasner@aol.com  
**Corresponding Author:** 212 East Market Street, York, PA 17403 USA

Medical Writer
James Lutz, MS, CCRA, Lutz Consulting, LLC, Medical Writing Services, Buellton, CA, USA, jlutzmail@aol.com

Panel Facilitator
Cynthia Sylvia, MSc, MA, RN, CWOCN, Gaymar Industries, Inc., Orchard Park, NY, USA, csylvia@gaymar.com

Additional Panel Members
Oscar Alvarez, PhD, CCT, FAPWCA, Center for Curative and Palliative Wound Care, Calvary Hospital, Bronx, NY, USA, oalvarez@calvaryhospital.org

Elizabeth A. Ayello, PhD, RN, ACNS-BC, ETN, FAPWCA, FAAN, Excelsior College School of Nursing, USA, elizabeth@ayello.com

Sharon Baranoski, MSN, RN, CWOCN, APN, DAPWCA, FAAN, Wound Care Dynamics, Inc., Shorewood, IL, USA, nrsebear@aol.com

William J. Ennis, DO, MBA, FACOS, University of Illinois, Palos Heights, IL, USA, w.ennis@comcast.net

Nancy Ann Faller, RN, MSN, PhD, ETN, CS, Carlisle, PA, USA, nafaller@aol.com

Jane Hall, Medical Malpractice Defense Attorney, Huie, Fernambucq & Stewart, LLP, Birmingham, AL, USA, jgh@hfsllp.com

Rick E. Hall, BA, RN, CWCN, Helping Hands Wound Care, Wichita, KS, USA, mnurse66@yahoo.com

Karen Lou Kennedy-Evans, RN, CS, FNP, KL Kennedy, LLC, Tucson, AZ, USA, ktulcer@aol.com

Diane Langemo, PhD, RN, FAAN, Langemo & Assoc, Grand Forks, ND, USA, dianelangemo@aol.com

Joy Schank, RN, MSN, ANP, CWOCN, Schank Companies, Himrod, NY, USA, joyschank@yahoo.com

Thomas P. Stewart, PhD, Gaymar Industries, Inc., Orchard Park, NY & S.U.N.Y. at Buffalo, USA, tstewart@gaymar.com

Nancy A. Stotts, RN, CNS, EdD, FAAN, University of California, San Francisco, San Francisco, CA, USA, nancy.stotts@nursing.ucsf.edu

David R. Thomas, MD, FACP, AGSF, GSAF, CMD, St. Louis University, St. Louis, MO, USA, thomasdr@slu.edu

Dot Weir, RN, CWON, CWS, Osceola Regional Medical Center, Kissimmee, FL, USA, dorothy.weir@hcahealthcare.com
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Background for Skin Changes At Life’s End (SCALE)

Organ dysfunction is a familiar concept in the health sciences, and can occur at any time but most often occurs at life’s end, during an acute critical illness or with severe trauma. Body organs particularly the heart and kidneys undergo progressive limitation of function as a normal process related to aging and the end of life. End of life is defined as a phase of life when a person is living with an illness that will often worsen and eventually cause death. This time period is not limited to the short period of time when the person is moribund. It is well accepted that during the end stages of life, any of a number of vital body systems (e.g. the renal, hepatic, cardiac, pulmonary, or nervous systems) can be compromised to varying degrees and will eventually totally cease functioning. The process of organ compromise can have devastating effects, resulting in injury or interference with functioning of other organ systems that may contribute to further deterioration and eventual death.

We propose that the skin, the largest organ of the body, is no different, and also can become dysfunctional with varying degrees of resultant compromise. The skin is essentially a window into the health of the body, and if read correctly, can provide a great deal of insight into what is happening inside the body. Skin compromise, including changes related to decreased cutaneous perfusion and localized hypoxia (blood supply and local tissue factors) can occur at the tissue, cellular, or molecular level. The end result is a reduced availability of oxygen and the body’s ability to utilize vital nutrients and other factors required to sustain normal skin function. When this compromised state occurs, the manifestations are termed, Skin Changes At Life’s End (SCALE). It should be noted that the acronym SCALE is a mnemonic used to describe a group of clinical phenomena, and should not be confused with a risk assessment tool. The term applies to all individuals across the continuum of care settings.

Skin organ compromise at life’s end is not a new concept in the literature. The first clinical description in modern medical literature appeared in 1989 with the Kennedy Terminal Ulcer (KTU). Kennedy described the KTU as a specific subgroup of pressure ulcers that some individuals develop as they are dying. They are usually shaped like a pear, butterfly, or horseshoe, and are located predominantly on the coccyx or sacrum (but have been reported in other anatomical areas). The ulcers are a variety of colors including red, yellow or black, are sudden in onset, typically deteriorate rapidly, and usually indicate that death is imminent. This initial report was based on retrospective chart reviews of individuals with pressure ulcers. It sparked further inquiry into how long these individuals within the facility lived after occurrence of a pressure ulcer. Just over half (55.7%) died within six weeks of discovery of their pressure ulcer(s). The observations were further supported by Hanson and colleagues (1991), who reported that 62.5% of pressure ulcers in hospice patients occurred in the 2 weeks prior to death. Further evidence for the existence of the KTU is mostly observational in nature, but is consistent with the premise that skin function can become compromised at life’s end.

It is noteworthy that while Kennedy independently described the KTU in 1989, a similar condition was actually first described much earlier in the French medical literature by Jean-Martin Charcot (1825-1893). In a medical textbook written in 1877, Charcot described a specific type of ulcer that is butterfly in shape and occurring over the sacrum. Patients that developed these ulcers usually died shortly thereafter, hence he termed the ulcer Decubitus Ominosus. However, Charcot attributed the ulcers to being neuropathic rather than pressure in origin. Charcot’s writings of Decubitus Ominosus...
were all but forgotten in the medical literature until recently with renewed interest in skin organ compromise. The fact that two experts in the field of chronic wounds independently reported the same clinical phenomenon, with very similar descriptions, 112 years apart, lends credence to the possible existence of terminal pressure ulcers as a result of end-of-life skin organ compromise.

Also of historical interest is the original work of Dr. Alois Alzheimer in Germany. He was on call in 1901 when a 51 year old woman, Frau Auguste D, was admitted to his asylum for the insane in Frankfort. Dr. Alzheimer followed this patient, studied her symptoms and presented her case to his colleagues as what came to be known as Alzheimer's Disease. When Frau Auguste D. died on April 8, 1906, her medical record listed the cause of death as “septicemia due to decubitus.” Alzheimer noted, “at the end, she was confined to bed in a fetal position, was incontinent and in spite of all the care and attention given to her, she suffered from decubitus.” So, here we have the first identified patient with Alzheimer’s Disease having developed immobility and two pressure ulcers with end stage Alzheimer’s. In our modern times, end stage Alzheimer’s Disease has become an all-too-frequent scenario with multiple complications including SCALE.

In 2003, Langemo proposed a working definition of skin failure; that it is a result of hypoperfusion, creating an extreme inflammatory reaction concomitant with severe dysfunction or failure of multiple organ systems. Three years later, Langemo and Brown (2006) conducted a comprehensive review of the literature on the concept of skin failure that focused largely on pressure ulcer development. They presented a discussion of changes in the skin that can occur with aging, the development of pressure ulcers, multiorgan failure, and “skin failure” (both acute and chronic as well as end of life). In the early 1990’s two publications by Parish & Witkowski had presented logical arguments about the mechanism of pressure ulcer occurrence at the end of life, suggesting that they may not be preventable in those individuals with multiple organ failure. Although the term skin failure has been introduced, it is not currently a widely accepted term in the dermatological or the wound literature.

Despite the limited scientific literature, there is consensus from the narrative literature that some pressure ulcers may be unavoidable including those that are manifestations of SCALE. We propose that at the end of life, failure of the homeostatic mechanisms that support the skin can occur, resulting in a diminished reserve to handle insults such as minimal pressure. Therefore, contrary to popular myth, not all pressure ulcers are avoidable.

Many members of the SCALE Panel acknowledge the need for systematic study of the phenomenon.

**Goals and Objectives of the SCALE Panel**

The overall goal of the SCALE Expert Panel was to initiate stakeholder discussion of skin changes at the end of life, a phenomenon that we have termed SCALE. An objective was to examine the concept of unavoidable pressure ulcers that can occur as a result of SCALE. While reaching consensus on the various aspects of this topic is an important outcome, this endeavor will require a more rigorous scientific investigative approach that was beyond the scope of this ground breaking meeting. The purpose of this initial meeting was to generate a series of statements that will serve as a platform for future consensus discussions. The objective of this document is to present these panel statements, disseminate them for public discussion, and to further the development of the body of scientific knowledge on this important topic.

**Methodology**

A modified three phase Delphi Method approach was used to reach consensus on the 10 statements reported in this document. The Delphi Method relies on expert panel input to reach consensus on a topic of interest. Our approach consisted of three separate phases of consensus building involving an international group of 69 noted experts in the field.
of wound care.

**Phase 1:** A panel of 18 experts in the field of wound care with expertise in wound and skin care convened in a round table format on April 4-6, 2008 in Chicago, IL, USA. Audio proceedings and written notes from this round table discussion were used to generate a Preliminary Consensus Document (PCD). This PCD was returned to the original panel for review and was modified as necessary to reach panel consensus.

**Phase 2:** The PCD was presented and distributed at numerous international conferences seeking public comment from September 2008 through June 2009. The document was published, and also available for public download from the web site of the panel sponsor (Gaymar Industries, Inc.). The PCD was further reviewed by a selected international panel of 49 Distinguished Reviewers with noted expertise in wound care and palliative medicine.

**Phase 3:** Written input received from the panel of Distinguished Reviewers and from the various public presentations was used to generate A Final Consensus Document (FCD). This FCD was then returned to the original 18-member Expert Panel and a 52-member Distinguished Reviewer Panel for voting on each of the 10 statements for consensus. A quorum of 80% that strongly agree or somewhat agree with each statement was used as a pre-determined threshold for having achieved consensus on each of the statements. Fifty two individuals voted on the final consensus process.

In addition to the PCD and FCD documents, an annotated bibliography of literature pertinent to SCALE was generated and is available for download from the web site of the panel sponsor (Gaymar Industries, Inc.).

**Panel Statements**

As a result of the two-day panel discussion and subsequent panel revisions, and with input from 69 noted wound care experts in a modified Delphi Method approach, the following 10 statements are proposed by the SCALE Expert Panel:

**Statement 1**

*Physiologic changes that occur as a result of the dying process (days to weeks) may affect the skin and soft tissues and may manifest as observable (objective) changes in skin color, turgor, or integrity, or as subjective symptoms such as localized pain. These changes can be unavoidable and may occur with the application of appropriate interventions that meet or exceed the standard of care.*

When the dying process compromises the homeostatic mechanisms of the body, a number of vital organs may become compromised. The body may react by shunting blood away from the skin to these vital organs, resulting in decreased skin and soft tissue perfusion and a reduction of the normal cutaneous metabolic processes. Minor insults can lead to major complications such as skin hemorrhage, gangrene, infection, skin tears and pressure ulcers that may be markers of SCALE. See Statement 6 for further discussion.

**Statement 2**

*The plan of care and patient response should be clearly documented and reflected in the entire medical record. Charting by exception is an appropriate method of documentation.*

The record should document the patient’s clinical condition including co-morbidities, pressure ulcer risk factors, significant changes, and clinical interventions that are consistent with the patient’s wishes and recognized guidelines for care. Facility policies and guidelines for record keeping should be followed and facilities should update these policies and guidelines as appropriate. The impact of the interventions should be assessed and revised as appropriate. This documentation may take many forms. Specific approaches to documentation of care should be consistent with professional, legal, and regulatory guidelines, and may involve narrative documentation, the use of flow sheets, or other documentation systems/tools.
If a patient is to be treated as palliative, it should be stated in the medical record, ideally with a reference to a family/caregiver meeting, and that consensus was reached. If specific palliative scales such as the Palliative Performance Scale,\textsuperscript{23} or other palliative tools were utilized,\textsuperscript{24} they should be included in the medical record. Palliative care must be patient-centered, with skin and wound care being only a part of the total plan of care.

It is not reasonable to expect that the medical record will be an all-inclusive account of the individual’s care. Charting by exception is an appropriate method of documentation. This form of documentation should allow the recording of unusual findings and pertinent patient risk factors. Some methods of clinical documentation are antiquated in light of today’s complexity of patient care and rapidly changing interprofessional healthcare environment; many current documentation systems need to be revised and streamlined.

**Statement 3**

*Patient centered concerns should be addressed including pain and activities of daily living.*

A comprehensive, individualized plan of care should not only address the patient’s skin changes and comorbidities, but any patient concerns that impact quality of life including psychological and emotional issues. Research suggests that for wound patients, health-related quality of life is especially impacted by pain, change in body image, odors and mobility issues. It is not uncommon for these factors to have an effect on aspects of daily living, nutrition, mobility, psychological factors, sleep patterns and socialization.\textsuperscript{25, 26} Addressing these patient-centered concerns optimizes activities of daily living and enhance a patient’s dignity.

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**A comprehensive, individualized plan of care should not only address the patient’s skin changes and comorbidities, but any patient concerns that impact quality of life including psychological and emotional issues.**

**Statement 4**

*Skin changes at life’s end are a reflection of compromised skin (reduced soft tissue perfusion, decreased tolerance to external insults, and impaired removal of metabolic wastes).*

When a patient experiences SCALE, tolerance to external insults (such as pressure) decreases to such an extent that it may become clinically and logistically impossible to prevent skin breakdown and the possible invasion of the skin by microorganisms. Compromised immune response may also play an important role, especially with advanced cancer patients and with the administration of corticosteroids and other immunosuppressant agents.

Skin changes may develop at life’s end despite optimal care, as it may be impossible to protect the skin from environmental insults in its compromised state. These changes are often related to other cofactors including aging, co-existing diseases and drug adverse events. SCALE, by definition occurs at life’s end, but skin compromise may not be limited to end of life situations; it may also occur with acute or chronic illnesses, and in the context of multiple organ failure that is not limited to the end of life.\textsuperscript{8} However, these situations are beyond the scope of this panel’s goals and objectives.
**Statement 5**

**Expectations around the patient’s end of life goals and concerns should be communicated among the members of the interprofessional team and the patient’s circle of care. The discussion should include the potential for SCALE including other skin changes, skin breakdown and pressure ulcers.**

It is important that the provider(s) communicate and document goals of care, interventions, and outcomes related to specific interventions (See Statement 2). The patient’s circle of care includes the members of the patient unit including family, significant others, caregivers, and other healthcare professionals that may be external to the current interprofessional team. Communication with the interprofessional team and the patient’s circle of care should be documented. The education plan should include realistic expectations surrounding end of life issues with input from the patient if possible. Communication of what to expect during end of life is important and this should include changes in skin integrity.

Being mindful of local protected health information disclosure regulations (e.g. USA: HIPAA, 1996), the patient’s circle of care needs to be aware that an individual at the end of life may develop skin breakdown, even when care is appropriate. They need to understand that skin function may be compromised to a point where there is diminished reserve to tolerate even minimal pressure or external insult. Educating the patient’s circle of care up front may help reduce the chances of shock and emotional reactions if end of life skin conditions occur.

This education includes information that as one nears end of life, mobility decreases. The individual frequently has a “position of comfort” that the patient may choose to maintain, resulting in a greater potential for skin breakdown. Some patients elect to continue to lie on the pressure ulcer, stating it is the most comfortable position for them. Respecting the coherent patient’s wishes is important.

With the recognition that these skin conditions are sometimes a normal part of the dying process, there is less potential for assigning blame, and a greater understanding that skin organ compromise may be an unavoidable part of the dying process.

**The patient’s circle of care includes the members of the patient unit including family, significant others, caregivers, and other healthcare professionals that may be external to the current interprofessional team.**

Discussions regarding specific trade-offs in skin care should be documented in the medical record. For example, patients may develop pressure ulcers when they cannot be (or do not want to be) turned due to pain or the existence of other medical conditions. Pressure ulcers may also occur in states of critical hypoperfusion due to underlying physical factors such as severe anemia, hypoxia, hypotension, peripheral arterial disease, or severe malnutrition. Care decisions must be made with the total goals of the patient in mind, and may be dependent on the setting of care, trajectory of the illness, and priorities for the patient and family. Comfort may be the overriding and acceptable goal, even though it may be in conflict with best skin care practice. In summary, the patient and family should have a greater understanding that skin organ compromise may be an unavoidable part of the dying process.

**Statement 6**

**Risk factors symptoms and signs associated with SCALE have not been fully elucidated, but may include:**

- **Weakness and progressive limitation of mobility.**
- **Suboptimal nutrition including loss of appetite, weight loss, cachexia and wasting, low serum albumin/pre-albumin, and low hemoglobin as well as dehydration.**
• **Diminished tissue perfusion, impaired skin oxygenation, decreased local skin temperature, mottled discoloration, and skin necrosis.**

• **Loss of skin integrity from any of a number of factors including equipment or devices, incontinence, chemical irritants, chronic exposure to body fluids, skin tears, pressure, shear, friction, and infections.**

• **Impaired immune function.**

Diminished tissue perfusion is the most significant risk factors for SCALE and generally occurs in areas of the body with end arteries, such as the fingers, toes, ears, and nose. These areas may exhibit early signs of vascular compromise and ultimate collapse, such as dusky erythema, mottled discoloration, local cooling, and eventually infarcts and gangrene.

As the body faces a critical illness or disease state, a normal protective function may be to shunt a larger percentage of cardiac output from the skin to more vital internal organs, thus averting immediate death. Chronic shunting of blood to the vital organs may also occur as a result of limited fluid intake over a long period of time. Most of the skin has collateral vascular supply but distal locations such as the fingers, toes, ears and nose have a single vascular route and are more susceptible to a critical decrease in tissue oxygenation due to vasoconstriction. Furthermore, the ability to tolerate pressure is limited in poorly perfused body areas.

Additional literature reviews and clinical research are needed to more thoroughly comprehend and document all of the potential risk factors associated with SCALE and their clinical manifestations.

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**Statement 7**

A total skin assessment should be performed regularly and document all areas of concern consistent with the wishes and condition of the patient. Pay special attention to bony prominences and skin areas with underlying cartilage. Areas of special concern include the sacrum, coccyx, ischial tuberosities, trochanters, scapulae, occiput, heels, digits, nose and ears. Describe the skin or wound abnormality exactly as assessed.

It is important to assess the whole body because there may be signs that relate to skin compromise. Table 1 provides a limited list of dermatologic terms that may be useful when describing areas of concern. Table 2 provides descriptive terms for lesions based on characteristics and size.

**Statement 8**

Consultation with a qualified health care professional is recommended for any skin changes associated with increased pain, signs of infection, skin breakdown (when the goal may be healing), and whenever the patient’s circle of care expresses a significant concern.

There are very definite descriptive terms for skin changes that can be used to facilitate communication between health care professionals (see Statement 7). Until more is known about SCALE, subjective symptoms need to be reported and objective skin changes described. This will allow for identification and characterization of potential end of life skin changes.

An accurate diagnosis can lead to decisions about the area of concern and whether it is related to end of life care and/or other factors. The diagnosis will help determine appropriate treatment and establish realistic outcomes for skin changes. For pressure ulcers, it is important to determine if the ulcer may be (i) healable within an individual’s life expectancy, (ii) maintained, or (iii) non-healable or palliative. For pressure ulcers, it is important to determine if the ulcer may be (i) healable within an individual’s life expectancy, (ii) maintained, or (iii) non-healable or palliative. The treatment plan will depend on an accurate diagnosis, the individual’s life expectancy and wishes, family members’ expectations, institutional policies,
and the availability of an interprofessional team to optimize care. Remember that patient status can change and appropriate reassessments with determination of likely outcomes may be necessary.

It is important to remember that a maintenance or non-healable wound classification does not necessarily equate with withholding treatment. For example, the patient may benefit with improved quality of life from surgical debridement and/or the use of advanced support surfaces.

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Table 1: Useful dermatologic terms for describing areas of concern. Additional terms can be found in the Glossary included at end of this document.

<table>
<thead>
<tr>
<th>Term</th>
<th>Definition</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Bruise</strong></td>
<td>An injury producing a hematoma or diffuse extravasation of blood without rupture of the skin. Often presents as a reddish, purple, black discoloration of the skin.</td>
</tr>
<tr>
<td><strong>Crust</strong></td>
<td>A hard outer layer or covering; cutaneous crusts are often formed by dried serum, pus or blood on the surface of a ruptured blister or pustule.</td>
</tr>
<tr>
<td><strong>Erosion (denudation)</strong></td>
<td>A loss of surface skin with an epidermal base.</td>
</tr>
<tr>
<td><strong>Eschar</strong></td>
<td>Thick adherent, necrotic tissue that is typically dry and brown, black or gray in color.</td>
</tr>
<tr>
<td><strong>Fissure</strong></td>
<td>A thin linear loss of skin with a dermal or deeper base.</td>
</tr>
<tr>
<td><strong>Hematoma</strong></td>
<td>A collection of blood in the soft tissues.</td>
</tr>
<tr>
<td><strong>Lesion</strong></td>
<td>Any change in the skin that may be a normal or abnormal variant including a wound or injury. It encompasses everything from macular lesions (color changes without elevation or depression of the skin) through total skin breakdown.</td>
</tr>
<tr>
<td><strong>Mottling of skin due to vascular stasis</strong></td>
<td>An area of skin composed of macular lesions of varying shades or colors over the smaller or medium sized blood vessels.</td>
</tr>
<tr>
<td><strong>Scale</strong></td>
<td>Surface keratin that may be thick or thin, resembling a fish scale, cast off (desquamating) from the skin.</td>
</tr>
<tr>
<td><strong>Skin Tear</strong></td>
<td>A traumatic wound occurring principally on the extremities of older adults as a result of friction alone or with shearing and frictional forces, that separate the epidermis from the dermis (partial-thickness wound) or which separate both the epidermis and the dermis from the underlying structures (full-thickness wound).</td>
</tr>
<tr>
<td><strong>Slough</strong></td>
<td>Yellow, green, tan, or white putrefied debris often partly separated from the surface of the wound bed.</td>
</tr>
<tr>
<td><strong>Ulcer</strong></td>
<td>A loss of surface skin with a dermal or deeper base.</td>
</tr>
</tbody>
</table>

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**Statement 9**

The probable skin change etiology and goals of care should be determined. Consider the 5 Ps for determining appropriate intervention strategies:

- **Prevention**
- **Prescription** (may heal with appropriate treatment)
- **Preservation** (maintenance without deterioration)
- **Palliation** (provide comfort and care)
- **Preference** (patient desires)

Prevention is important for well being, enhanced quality of life, potential reimbursement, and to avoid unplanned medical consequences for end of life care. The skin becomes fragile when stressed with...
decreased oxygen availability associated with the end of life. The plan of care needs to address excessive pressure, friction, shear, moisture, suboptimal nutrition, and immobilization.

Prescription refers to the interventions for a treatable lesion. Even with the stress of dying, some lesions are healable after appropriate treatment. Interventions must be aimed at treating the cause and at patient centered concerns (pain, quality of life), before addressing the components of local wound care as consistent with the patient’s goals and wishes.

Preservation refers to situations where the opportunity for wound healing or improvement is limited, so maintenance of the wound in its present clinical state is the desired outcome. A maintenance wound may have the potential to heal, but there may be other overriding medical factors that could direct the interprofessional team to maintain the status quo. For example there may be limited access to care, or the patient may simply refuse treatment.

Palliation refers to those situations in which the goal of treatment is comfort and care, not healing. A palliative or non-healable wound may deteriorate due to a general decline in the health of the patient as part of the dying process, or due to hypoperfusion associated with non-correctable critical ischemia. In some situations, palliative wounds may also benefit from some treatment interventions such as surgical debridement or support surfaces, even when the goal is not to heal the wound.

Preference includes taking into account the preferences of the patient and the patient’s circle of care.

The 5P enabler can be used in combination with the SOAPIE mnemonic to help explain the process of translating this recommendation into practice (Figure 1). Realistic outcomes can be derived from appropriate SOAPIE processes with the 5 skin Ps becoming the guide to the realistic outcomes for each individual.

**S = Subjective skin & wound assessment:** The person at the end of life needs to be assessed by history, including an assessment of the risk for developing a skin change or pressure ulcer (Braden Scale or other valid and reliable risk assessment scale).

**O = Objective observation of skin & wound:** A physical exam should identify and document skin changes that may be associated with the end of life or other etiologies including any existing pressure ulcers.

**A = Assess and document etiology:** An assessment should then be made of the general condition of the patient and a care plan.

**P = Plan of care:** A care plan should be developed that includes a decision on skin care considering the 5P’s as outlined in the Figure 1. This plan of care should also consider input and wishes from the patient and the patient’s circle of care.

**I = Implement appropriate plan of care:** For successful implementation, the plan of care must be matched with the healthcare system resources (availability of equipment and personnel) along with appropriate education and feedback from the patient’s circle of care and as consistent with the patient’s goals and wishes.

**E = Evaluate and educate all stakeholders:** The interprofessional team also needs to facilitate appropriate education, management, and periodic reevaluation of the care plan as the patient’s health status changes.

### Table 2: Dermatological descriptions of lesions based on characteristics and size.

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<th>Lesion Characteristics</th>
<th>Lesion Size</th>
<th>&lt;1 cm</th>
<th>&gt;1 cm</th>
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<tr>
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<td>Macule</td>
<td>Patch</td>
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<td>Elevated</td>
<td>Papule</td>
<td>Plaque</td>
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<td>Blister</td>
<td>Vesicle</td>
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Figure 1: The SOAPIE mnemonic with the 5P enabler.

Statement 10

Patients and concerned individuals should be educated regarding SCALE and the plan of care.

Education needs to be directed not only to the patient but also the patient’s circle of care. Within the confines allowed by local protected health information regulations (e.g. HIPAA, 1996, USA), the patient’s circle of care needs to be included in decision making processes regarding goals of care and the communication of the meaning and method of accomplishing those decisions. Collaboration and communication should be ongoing with designated representatives from the patient’s circle of care and the clinical team connecting at regular intervals. Documentation of decision making, educational efforts, and the patient’s circle of care perspective is recommended. If adherence to the plan of care cannot be achieved, this should be documented in the medical record (including the reasons), and alternative plans proposed if available and feasible.

Education also extends beyond the patient’s circle of care, to other involved healthcare professionals, healthcare administrators, policy makers, and to the payers. Healthcare professionals need to facilitate communication and collaboration across care settings and disciplines; organizations need to prepare staff to identify and manage SCALE. Ongoing discussions with key stakeholders will additionally provide a stimulus for additional evidence based research and education regarding all aspects of SCALE.

Healthcare professionals need to facilitate communication and collaboration across care settings and disciplines; organizations need to prepare staff to identify and manage SCALE.
Recommendations for Future Research

Conduct and disseminate through publications and presentations:

- A thorough review of the literature concerning all aspects of SCALE.

- Research to identify the mechanisms for the proposed decreased hypoperfusion and oxygenation of the skin and soft tissues involved with SCALE and resulting outcomes.

- Research to determine the mechanisms for the proposed, tissue, cellular, and molecular dysfunctions that occur during SCALE.

- Research that helps to clarify and distinguish skin and soft tissue damage associated with SCALE from pressure ulcers and other skin disorders not associated with skin organ compromise or the end of life.

- Research into predictive tools for the onset and measurement of SCALE and the timing of life’s end (possibly adaptive use of the Palliative Performance Scale (http://palliative.info/resource_material/PPSv2.pdf)

- Qualitative research to explore the impact of SCALE on the patient, the patient’s circle of care, and professional caregivers with regard to healthcare-related quality of life.

- Development of a database of patients (with histories) suspected of exhibiting SCALE to analyze them retrospectively for skin and soft tissue changes and risk factors that occurred just prior to death. Isolate the skin changes and risk factors involved and determine how important each individual variable is to the occurrence of SCALE.

- Research cataloging patients who do not exhibit SCALE to identify factors that may help prevent the occurrence of SCALE.

- Develop a registry of Kennedy Terminal Ulcers to better categorize this phenomenon, including location, clinical description, patient and ulcer outcomes, and the presence of other end of life skin changes including lesions in other locations.

- Both prospective and retrospective prevalence research of individuals suspected of exhibiting SCALE, particularly among hospice patients.

- Research on specific medical and physiologic conditions that may contribute to SCALE. These include but may not be limited to malignancy, hypotension and hemodynamic instability, administration of potent vasoconstrictors, peripheral arterial and vascular disease, hypoxia, malnutrition, and severe anemia.

Conclusions

SCALE Panel members are in agreement that there are observable changes in the skin at the end of life. Our current understanding of this complex phenomenon is limited and the panel concludes that additional research is necessary to assess the etiology of SCALE, to clinically describe and diagnose the related skin changes, and to recommend appropriate pathways of care. The panel recommends that clinicians, laypeople, and policy makers need to be better educated in the medical, social, legal and financial ramifications of SCALE.

Health care organizations need to ensure the provision of resources that enable health care professionals to identify and care for SCALE while maintaining the dignity of the patient, family and circle of care to the end of life.
**Glossary of Terms**

**Arterial Ulcer:** An ulcer that occurs almost exclusively in the distal lower extremity due to inadequate perfusion/ischemia.\(^{37}\)

**Avoidable (pressure ulcer):** The resident (individual) developed a pressure ulcer and the facility did not do one or more of the following: evaluate the resident's clinical condition and pressure ulcer risk factors; define and implement interventions that are consistent with resident needs, resident goals, and recognized standards of practice; monitor and evaluate the impact of the interventions; or revise the interventions as appropriate (CMS definition).\(^{38}\)

**Charting by Exception (CBE):** Charting by exception is premised on an assumption that the patient has manifested a normal response to all interventions unless an abnormal response is charted.\(^{39}\) This type of charting is often performed with flow sheets that are based on preestablished guidelines, protocols, and procedures that identify and document the standard patient management and care delivery. Clinicians need to make additional documentation when the patient's condition deviates from the standard or what's expected.\(^{40}\)

**Crust:** A hard outer layer or covering; cutaneous crusts are often formed by dried serum, pus or blood (one or more components may co-exist) on the surface of a ruptured blister or pustule.\(^{29}\)

**Decubitus Ominosus:** Medical term first used by Jean-Martin Charcot in the 19th century to signify a sacral ulcer that presages death.

**Delphi Method:** A systematic, interactive forecasting method which relies on a panel of independent experts. The carefully selected experts answer questionnaires in two or more rounds. After each round, a facilitator provides an anonymous summary of the experts’ forecasts from the previous round as well as the reasons they provided for their judgments. Thus, experts are encouraged to revise their earlier answers in light of the replies of other members of their panel. It is believed that during this process the range of the answers will decrease and the group will converge towards the “correct” answer. Finally, the process is stopped after a pre-defined stop criterion (e.g. number of rounds, achievement of consensus, stability of results) and the mean or median scores of the final rounds determine the results.\(^{19}\)

**Denudation:** See erosion.

**Diabetic Ulcer:** A wound occurring most often in the feet of people with diabetes due most commonly to neuropathy and/or peripheral vascular disease.\(^{41}\)

**End of Life:** End of life is defined as a phase of life when a person is living with an illness that will worsen and eventually cause death. It is not limited to the short period of time when the person is moribund.\(^1\)

**Erosion:** A loss of surface skin with an epidermal base.

**Fissure:** A thin linear loss of skin with a dermal or deeper base.

**Healable (wound):** A wound occurring on an individual whose body can support the phases of wound healing within the individuals expected lifetime.

**Healed (wound):** A wound that has attained closure of the epidermal surface. A recently closed wound may only have 20% tensile strength of skin that has never been wounded and may be susceptible to recurrent ulceration.

**Kennedy Terminal Ulcer:** A pressure ulcer that some individuals develop as they are dying. It is usually shaped like a pear, butterfly, or horseshoe, usually on the coccyx or sacrum (but has been reported on other anatomical areas), has colors of red, yellow or black, is sudden in onset, and usually is associated with imminent death.\(^2, 42-49\)

**Lesion:** Any change in the skin that may be a normal or abnormal variant including a wound
or injury.²⁹ It encompasses everything from macular lesions (color changes without elevation or depression of the skin) through total skin breakdown.

**Maintenance (wound):** An attempt to keep an ulcer from deteriorating by providing good wound care. The wound may not heal due to patient choice or a lack of the health care system to provide optimal resources to promote healing.

**Non-healable (wound):** A wound that often deteriorates and occurs on an individual whose body cannot support the phases of wound healing within the individual’s expected lifetime. There may be inadequate vascular supply to support healing or the cause of the wound cannot be corrected.

**Palliative skin care:** Providing comfort and support for the body’s cutaneous surface (part of the practice of palliative medicine) is not a time-confined but rather a goal-oriented and patient-centered care delivery model.⁵⁰ Palliative wound care is the evolving body of knowledge and skills that take a holistic approach to relieving suffering and improving quality of life for patients (individuals) and families living with chronic wounds, whether the wound is healable, can be maintained or may deteriorate.³²

**Patient circle of care:** This is not a legal term, but rather a social term that includes all of the stakeholders in the patient’s health and well being. The term includes, but is not limited to, the patient, a legal guardian or responsible party, a spouse or significant other, interested friends or family members, caregivers, and any other individual(s) who may have an interest in the patient’s care and well being.

**Pressure Ulcer:** A pressure ulcer is localized injury to the skin and/or underlying tissue usually over a bony prominence, as a result of pressure, or pressure in combination with shear and/or friction. A number of contributing or confounding factors are also associated with pressure ulcers; the significance of these factors is yet to be elucidated.⁵¹

**Scale (skin):** Surface keratin that may be thick or thin, resembling a fish scale, cast off (desquamating) from the skin.²⁹

**SCALE:** The acronym for Skin Changes at Life’s End.

**Skin breakdown:** An interruption in the integrity of the skin surface leading to defect in the epidermal covering with an epidermal, dermal or deeper base.

**Skin compromise:** A state in which skin’s protective function is at risk of breaking down.

**Skin failure:** An acute episode where the skin and subcutaneous tissues die (become necrotic) due to hypoperfusion that occurs concurrent with severe dysfunction or failure of other organ systems.⁸

**Skin tear:** A traumatic wound occurring principally on the extremities of older adults as a result of friction alone or with shearing and frictional forces that separate the epidermis from the dermis (partial-thickness wound) or a deeper split that separates both the epidermis and the dermis from the underlying structures (full-thickness wound).³⁰

**Stakeholders:** An individual, facility, or organization with an interest in Skin Changes at Life’s End (SCALE).

**Stage I Pressure Ulcer:** Intact skin with non-blanchable redness of a localized area usually over a bony prominence. Darkly pigmented skin may not have visible blanching; its color may differ from the surrounding area.⁵¹

**Stage II Pressure Ulcer:** Partial thickness loss of dermis presenting as a shallow open ulcer with a red pink wound bed, without slough. May also present as an intact or open/ruptured serum-filled blister.⁵¹

**Stage III Pressure Ulcer:** Full thickness tissue loss. Subcutaneous fat may be visible but bone, tendon or muscle are not exposed. Slough may be present but does not obscure the depth of tissue loss. May include undermining and tunneling.⁵¹

**Stage IV Pressure Ulcer:** Full thickness tissue loss
with exposed bone, tendon or muscle. Slough or eschar may be present on some parts of the wound bed. Often include undermining and tunneling.\textsuperscript{51}

**Suspected Deep Tissue Injury:** Purple or maroon localized area of discolored intact skin or blood-filled blister due to damage of underlying soft tissue from pressure and/or shear. The area may be preceded by tissue that is painful, firm, mushy, boggy, warmer or cooler as compared to adjacent tissue.\textsuperscript{51}

**Terminal tissue trauma:** Damage to the integumentary system that has occurred at the end of life.

**Ulcer:** A loss of surface skin with a dermal or deeper base.

**Unavoidable (pressure ulcer):** The resident developed a pressure ulcer even though the facility had evaluated the resident’s clinical condition and pressure ulcer risk factors; defined and implemented interventions that are consistent with resident needs, goals, and recognized standards of practice; monitored and evaluated the impact of the interventions; and revised the approaches as appropriate (CMS definition).\textsuperscript{58}

**Unstageable Pressure Ulcer:** Full thickness tissue loss in which the base of the ulcer is covered by slough (yellow, tan, gray, green or brown) and/or eschar (tan, brown or black) in the wound bed.\textsuperscript{51}

**Venous Ulcer:** A ulceration that occurs on the lower limb secondary to underlying venous disease; formerly called stasis ulcers.\textsuperscript{52}

**References**


27. Langemo DK. When the goal is palliative care. Adv Skin Wound Care 2006;19(3):148-54.


40. Smith LM. How to chart by exception. Nursing 2002 (SEPT).


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The SCALE Annotated Bibliography and future updates to the SCALE Consensus Statement will be posted at: www.gaymar.com > Clinical Support and Education > SCALE Consensus Documents.

Corresponding Author:
Dr. Diane L. Krasner
212 East Market Street
York, PA 17403 USA
dlkrasner@aol.com
WOUND DRESSING PRODUCT SELECTION:

A Holistic, Interprofessional Patient-Centered Approach©

A WoundSource White Paper | September 2010

Diane L. Krasner PhD RN CWCN CWS MAPWCA FAAN
R. Gary Sibbald BSc, MD, FRCPC (Med, Derm), MACP, FAAD, MEd, MAPWCA
Kevin Y. Woo BSc MSc PhD RN GNC(C) ACNP FAPWCA

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INTRODUCTION & CONCEPTUAL FRAMEWORK

Many wound care clinicians remember the “good old days” when wound dressing product selection simply involved choosing between a handful of products that were essentially variations on the same theme. There was gauze, impregnated gauze and filled gauze pads. In the earlier 20th century, clinicians added antimicrobial solutions, creams and ointments (like Dakin’s solution developed during World War I and silver sulfadiazine developed in the 1960’s) and the wound care formulary was limited and simplistic.

Fast forward to the 21st century and wound care clinicians are confronted with a totally different situation: hundreds of products, scientific rationale for moist interactive dressings and an emerging evidence-base for product selection.

Current wound care expertise encompasses numerous dressing-related skills including:

- Treating the cause of the wound and addressing patient centered concerns to set the stage for local wound care
- Properly assessing the wound and identifying the dressing requirements
- Selecting dressings based on their form and function for an individual wound’s needs
- Meeting setting-specific requirements for dressing change frequency and maintenance
- Addressing formulary or healthcare system availability as well as reimbursement requirements

Wound care product selection today must be as sophisticated and as evidence-based as possible. This Kestrel White Paper presents a conceptual framework for the wound dressing product selection process that is based on three principles:

- Holistic Perspectives (1)
- Interprofessional Considerations (2)
- Patient-Centered Concerns (3)

This conceptual framework is illustrated in Figure 1 below and is discussed in detail in this paper.
WOUND DRESSING PRODUCT SELECTION FOR THE 21ST CENTURY

For every complex problem, there is a simple solution, and it is wrong.
—H. L. Menken

Selecting appropriate wound dressing products and supportive care to maximize healing and patient outcomes is a complex process. Dressing and local wound care options based on science and best practices must be filtered by clinical experience and must be consistent with patient preferences, caregiver requirements and setting/access issues (4). Additionally, effective dressing selection and local wound care planning involve the perspectives of the entire interprofessional team (2).

Knowing the performance parameters of dressing categories/individual products and matching these attributes to an individual's wound can optimize the healing process (5). But dressings are only one piece of the puzzle. Dressings alone will not promote wound healing, unless the underlying cause(s) for the wound are also addressed (e.g. treatment of the wound cause, blood supply, nutrition, patient centered concerns, local wound care etc.). As the wound changes, the plan of care must change and dressing products may have to be changed. Appropriate dressing product selection:

• Optimizes the local wound healing environment
• Reduces local pain and suffering
• Improves activities of daily living and quality of life

Inappropriate dressing selection can:

• Cause the wound status to deteriorate (e.g. wound margin maceration, increased risk of superficial critical colonization or deep infection, skin stripping).
• Increase local pressure or pain especially at dressing change (dressing removal and cleansing).
• Increase costs with the need for frequent dressing changes or the selection of an inappropriate advanced or active dressing.

National and international wound care guidelines and best practice documents mean that there is no longer a local standard of care. No matter where you practice, you will be held to national/international standards of wound care practice (6). Some experts have argued that the selection of the wrong dressing is just as problematic as the administration of the wrong drug and the clinician would be just as liable in a court of law. If dressings can be shown to delay the healing process (e.g. wet-to-dry gauze dressings in a wound that requires moist wound healing, pain from inappropriate adhesives, failure to treat critical colonization that can lead to deep infection), their use might be deemed negligent by a jury in a court case.
INTERPROFESSIONAL CONSIDERATIONS
- Frequency of dressing changes
- Wound access
- Consistent with other treatments (local topical, regional, systemic)
- Patient not allergic to component
- Not a common sensitizer

PATIENT-CENTERED CONCERNS
- Ease of use
- Pain management
- Odor control
- Compatible with ADLs
- Body image / Psychosocial
- Reimbursement/Costs

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HOLISTIC PERSPECTIVES

Wound dressing product selection must be consistent and congruent with the total plan of care for the person with a wound. Four questions that the clinician should consider are:

1. What type of wound is it? What is the underlying etiology/cause and can you treat or correct the cause? (e.g. pressure, venous, neuropathic, neuroischemic, ischemic, etc)
2. Is it healable, maintenance or non-healable/palliative?(7)
3. Is the wound colonized, critically colonized or infected in the deep or wound margin? (8, 9)
4. Is the plan of care aggressive/active, maintenance or palliative/non-healable?

If the overall plan of care for the person is aggressive/active, the dressing plan should be aggressive/active. So for example, if a person has an exudating, infected diabetic foot ulcer with osteomyelitis that is being treated with hyperbaric oxygen therapy and serial debridements, a dressing such as a silver alginate or a silver foam dressing would be the dressing of choice (10). On the other hand, if a person is dying and on hospice and the goal of care is to palliate an exudating, infected diabetic foot wound with osteomyelitis, then a topical antimicrobial such as cadexamer iodine or povidone iodine, chlorhexidine or its derivatives (PHMB: Polyhexylmethylenebiguanide) might be a congruent choice.

In clinical practice, occasions occur frequently when patients are too sick to choose an aggressive pathway for their wound care. A common scenario is when a patient is in critical condition in an intensive care unit, on a respirator, immobilized, anticoagulated and his life hangs in the balance. The patient develops a sacral pressure ulcer that quickly goes from a partial thickness lesion to a full thickness wound with eschar. A holistic approach to wound care would lead to the maintenance pathway. Aggressive/active care in a critically ill patient would be unreasonable. Debridement of eschar in an anticoagulated patient with little healing potential is not a reasonable or prudent practice. A more reasonable option is to maintain the wound using a dressing that would protect the area and keep the eschar stable until the patient improves (at which point the aggressive/active pathway kicks in) or the patient deteriorates (at which point the palliative/nonhealable pathway is chosen).
WOUND ASSESSMENT AND IDENTIFICATION OF WOUND DRESSING REQUIREMENTS

Wound care requires a holistic approach looking at the ‘whole’ patient not just the ‘hole’ in the patient. The very first step of the assessment should aim to determine the accurate wound diagnosis and the cause of the wound. Despite the importance of dressings, wound healing can only be optimized when the underlying wound cause is corrected. For example, strategies to reduce tissue deformation (pressure, friction and shear) are crucial to promote healing of pressure ulcers. Patients with venous leg ulcers benefit from venous congestion improving compression therapies (bandages for healing or support stockings to prevent recurrence). Footwear or devices should be considered to redistribute pressure away from diabetic or other neurotropic foot ulcers. It is important to remember that wounds are not likely to heal if arterial supply is deficient unless patients undergo bypass or dilation of the affected arteries. Other related factors that may influence wound healing and warrant regular evaluation include nutrition, coexisting medical diseases and certain medications. When healing is not the realistic objective, moisture is contraindicated; instead, conservative debridement without cutting into living tissue, bacterial reduction, and moisture reduction should be considered.

The first step in wound care is to carefully document the wound characteristics;
- Location
- Size: Longest length and the widest width (at right angle to the longest length or oriented by a head to foot perspective)
- Depth as usually measured by a cotton swab or sterile probe
- Undermining and tunneling: location on the clock and extent as measured by a probe
- Wound Margin: normal, macerated, erythema, edema, warmth or increased temperature
- Wound Base: by percentage
  - Black-brown firm eschar
  - Brown yellow soft slough (harmful)
  - Yellow firm tissue that may serve as a foundation for granulation (healthy)
  - Pink firm healthy granulation tissue or unhealthy red friable tissue
- Exudate:
  - serous, sanuginous, or pustular or combinations
  - Large, moderate, scant, absent
- Epithelial edge
  - Sloped purple of advancing healing epithelial margin
  - Steep slope of stalled chronic wound
- Exposed tissues (tendon, bone) that may not allow granulation on top
- Foreign bodies (e.g. gauze fragments, sutures, hardware)
To prepare a wound bed for healing, devitalized and damaged tissue such as firm eschar or sloughy materials that promote bacteria growth should be removed or debrided. Topical dressings are used to promote autolytic debridement through the activities of phagocytic cells and endogenous enzymes. Another key function of wound dressings is to manage localized wound infection. All chronic wounds are colonized by bacteria. If bacteria were allowed to proliferate crossing a critical threshold, local tissue damage can lead to delayed healing. Many modern dressings contain active antimicrobial ingredients that are released into the wound surface compartment in an exchange with wound fluid. Dressings with silver are one of the most popular choices of topical agents. Alternatively, bacteria can be entrapped and sequestered in the micro-architecture of a dressing where they may be inactivated. For non-healable wounds, topical antiseptics dry the wound surface and provide bacterial reduction.

For wounds that have the potential to heal, moisture balance (not too much or too little) is essential for all phases of wound repair. An ideal dressing should be able to keep the wound bed moist for cellular proliferation and migration but at the same time sequester excess drainage to avoid peri-wound damage.

The major categories of wound dressings are foams, alginates, hydrofibers, hydrogels, and hydrocolloids. These are discussed briefly below.

**FOAM DRESSINGS** are designed to wick up a large volume of exudate. The fluid handling capacity of various foams can be affected by the polyurethane film backing and its ability to transfer moisture vapor out of the dressing but form a barrier to bacterial contamination. Depending on the level of wound exudate, foams have a wear time of one to seven days.

Foams absorb moisture but also give moisture back to a wound if the gradient on the surface becomes dehydrated. This function can lead to periwound maceration but advanced foam dressings have variable pore sizes that will facilitate partial moisture retention and partial moisture exchange with the wound surface. These second generation foams are less likely to macerate the wound margin. Foams have also been combined with antiseptics (silver, PHMB) and other agents to serve as a delivery vehicle for active therapies at the wound surface (third generation of foam development.) Foams that are associated with excessive periwound maceration can be cut to the wound size, fenestrated on the top to wick to a secondary dressing or changed more frequently.

**ALGINATE DRESSINGS** are also capable of handling copious exudate while the gelling effect of these materials will keep the wound base moist. Unlike foams, calcium alginites are bioresorbable (may disappear) and bind fluid to the outside of the fibers rather than the inner pores. Alginites are derived from brown seaweed or kelp. Depending on the species and origin of the calcium alginate (leaf, stem), they may have more gelling (high manuronic acid concentration) or a higher fiber strength (high galuronic acid concentration). These dressings are manufactured in sheets (lateral fluid wicking) or in ropes (vertical fluid wicking). When the alginate is extracted from kelp, it is a sodium hydrogel that can be combined with calcium to form a fibrous structure. When they are applied to the wound, the calcium as part of the alginate is released into the wound and may also trigger the coagulation cascade to facilitate hemostasis. The sodium is exchanged for calcium at the level of the alginate, recreating a sodium alginate hydrogel. In comparison to foams, calcium alginites are less absorptive but they have the ability to act as excellent autolytic debriders. Dressings with alginites are often changed daily or as infrequently as three times a week.
HYDROFIBER DRESSINGS consist of Carboxymethylcellulose and have a water hating (hydrophobic) component (methylcellulose) that gives this dressing its tensile strength and a water loving (hydrophilic) component (Carboxy) that acts as a fluid lock. As the dressing absorbs fluid, the hydrofiber is converted into a gel consistency. Hydrofiber dressings are thin and have moderate absorbency forming a fluid lock. When the hydrofiber is saturated wound fluid strike through will occur. These dressings require a secondary dressing to keep them in place because the addition of an adhesive will interfere with the fluid absorption properties of the dressing.

HYDROGEL DRESSINGS are usually indicated for dry wounds. The major ingredient of hydrogels is water (70 to 90%) that donates moisture into the wound base. The backbone for a hydrogel may be a hydrocolloid, propylene glycol, saline or other substance. This backbone gives them their viscosity or tack to stay on the wound bed. They are excellent autolytic debriders and preserve moisture balance, largely through donating moisture to the wound surface. They are often changed daily to three times a week.

HYDROCOLLOID DRESSINGS consists of a backing (often a film or polyurethane) with carboxymethylcellulose, water absorptive components (such as gelatin and pectin) and an adhesive. Hydrocolloids are designed for wear times of one to seven days and for this reason their absorbency is lower than foams or calcium alginates but similar to hydrogels. When these dressings are used for autolytic debridement, they may need to be changed more frequently and may require the removal of non-viable slough from the surface of the wound to prevent odor or secondary bacterial proliferation under the wound. These dressings often lower the wound surface pH that may contribute to their antimicrobial effect. Some hydrocolloids leave more residue on the wound surface than others and this residue may contribute to wound odor under the hydrocolloid dressing.

FILM DRESSINGS are often used for local protection. The choice of a non-adherent (no adhesive) versus a film with adhesive backing should be determined by the fragility of the surrounding skin. Film materials are semi-occlusive with various degrees of permeability (referred to as the Moisture Vapor Transmission Rate: MVTR) that allow a water molecule to pass through the dressing and evaporate into the ambient environment at a variable rate depending on the moisture vapor transmission rate. They are not designed for fluid accumulation below the film. When fluid develops under the dressing it needs to be evacuated or the dressing changed because the relatively alkaline pH under these dressings with fluid accumulation will promote bacterial proliferation. As an alternative to traditional adhesives (accrylates, hydrocolloids), silicone coatings have been used to reduce local trauma and prevent pain on dressing removal.
INTERPROFESSIONAL CONSIDERATIONS

When different professional groups are involved in a wound patient’s care, there may be interprofessional considerations that will have bearing on the dressing selected. Finding a way to accommodate each discipline’s unique perspectives and needs enhances interprofessional wound care (see Figure 2 below)(2). Here are several common examples:

- In an acute care facility, the Surgical Team wants to examine a dehisced surgical wound on daily rounds, so a dressing that is changed daily and can be lifted off and replaced without compromising the dressing adherence is the best choice (e.g. silicone foam vs. adhesive foam or gauze dressing).
- In any setting, the physicians need to measure the output from a draining tube site (e.g. nephrostomy tube site). Discontinuing absorbent gauze pads (e.g. abdominal dressings – ABDs) and using a cut-to-fit urostomy pouch allows accurate measurement of the drainage and protects the peri-wound skin from maceration and erosion.
- In an outpatient wound center, the hyperbaricist needs to assess the wound daily following hyperbaric treatment. A non-adhesive, daily or between treatment dressing change is needed (e.g. hydrogel, hydrocolloid or other modern moist interactive dressing).
- In a nursing home, the Physical Therapy Department will begin Rehabilitation Therapy on a resident with a diabetic foot ulcer. A dressing that minimizes pressure on the wound bed when the resident ambulates is optimal (e.g. a piece of alginate rope versus a gauze 2x2) with a thin but secure secondary dressing to avoid interfering with the plantar pressure redistribution.

Interprofessional collaboration on dressing selection can prevent complications (such as skin stripping or skin tears) from changing dressings too frequently, having inappropriate adhesive backing or inadequate moisture balance or lacking required anti-microbial properties. Careful coordination reduces costs and dressing-associated labor.

Another occasion when careful dressing coordination is needed is during wound patient/ client transfers from one healthcare setting or service to another including a discharge home. For example, the optimal dressing for acute care may not be available or reimbursed in long-term care or home care. In the United States, if the person has been a resident in long-term care and is moved to hospice care, the Hospice will provide the dressings for the resident while he/she is in the nursing home as part of the per diem Hospice Benefit. This may necessitate a change of dressing depending on the hospice dressing formulary.

Finally, is your interprofessional team up to standards? Are you able to provide holistic, patient-centered care? Ask yourself the following three questions:

- Does your wound team have the resources (human and otherwise) and knowledge to provide advanced patient-centered wound care?
- Do you have the referral sources in place to meet the needs of selected wound patients (especially their psychosocial and social needs) along with rehabilitation support?
- Does your wound team and dressing formulary enable you to address the needs of special populations (such as bariatric, diabetic, frail elderly and palliative) in a timely and appropriate manner?
Individualized wound care plans that address specific patient centered concerns are most likely to succeed and promote the best outcomes for the patient with a wound. Standardized, “canned” wound care plans often fail because they do not promote patient adherence / coherence. The patient may be labeled “non-compliant” when the real problem is that the care plan has not been properly individualized to the person’s specific needs/problems and he/she cannot possibly comply with the routine way. The road to wound care planning success is paved with careful attention to patient-centered concerns, including pain management, odor control, body image & psychosocial concerns and reimbursement/cost issues.

Common examples of patient centered concerns that impact dressing product selection include:

- Premedicating patients who experience dressing change pain prior to dressing changes and allowing adequate time for the premedication to take effect
- Selecting when appropriate non-adherent dressings to reduce pain and trauma at dressing change
- Addressing odor control issues by utilizing absorbent and/or charcoal dressings and adjusting dressing change frequency
- When possible selecting secondary dressing that enable patients to shower, bathe and perform other usual activities of daily living
- Choosing dressings that are easy to apply and that address the needs of the patient and the patient caregivers

Whenever possible, ordering dressings that are reimbursed by the patient’s insurance and that are easily purchased/accessed.
CONCLUSION

When developing wound dressing product formularies and clinical practice guidelines, be sure to follow a formal process that includes a review of relevant existing clinical practice guidelines and regulatory requirements, such as those in the United States from the Centers for Medicare and Medicaid. For guidance on this process readers are referred to: SELECT: Evaluation and Implementation of Clinical Practice Guidelines. A Guidance Document from the American Professional Wound Care Association (11).

Two excellent online wound dressing product resources are available to help build a dressing formulary by generic category: Wound Source (Kestrel Health Information, Inc.) [www.woundsource.com/product-category/dressings](http://www.woundsource.com/product-category/dressings) and World Wide Wounds (U.K.), [www.worldwidewounds.com](http://www.worldwidewounds.com)
REFERENCES


