CE 1.0
ANCC CONTACT HOUR

Damage control:

Differentiating incontinence-associated dermatitis from pressure injury

By Kathleen Francis, DNP, FNP-BC, CWOCN

SKIN INJURIES such as incontinence-associated dermatitis (IAD) and pressure injuries are increasingly monitored and benchmarked as a quality indicator for hospitals and nursing facilities.[1-4] Pressure injuries are documented by Medicare- and Medicaid-certified skilled nursing facilities in the Minimum Data Set, and pressure injury incidence rates are publicly reported as a quality measure in the Nursing Home Compare website.[5]

Many clinicians have difficulty distinguishing superficial skin injuries from early-stage pressure injuries because the differences can be very subtle. Mistaking IAD for an early-stage pressure injury can result in ineffective treatment and possible deterioration to full-thickness injury.[6,7] This is particularly true in patients with dark skin tones because visual cues associated with these types of skin injuries may not be easy to detect.[8-12] Likewise, failure to detect blanchable and nonblanchable erythema in a patient with dark skin tones may increase the patient's risk of developing a full-thickness skin injury.[13]

This article discusses how to differentiate, classify, and document IAD and pressure injuries with an emphasis on assessing patients with dark skin.

Correct assessment is critical

Accurate classification of skin injuries and identification of their etiology is essential to ensure that the correct treatment plan is initiated to halt the injury and support healing. Besides impacting patient care, inaccurate identification of skin injuries can negatively affect benchmarking data and payments to the facility.[2,14] Many clinicians have difficulty

VOYAGERIX / SHUTTERSTOCK

making the correct identification because these injuries can have very subtle differences.[15-19]

Pressure injuries have traditionally been classified using a staging system based on depth of tissue injury. These have been revised over the years to incorporate the current understanding of the science of pressure injury pathology and etiology (see *NPUAP pressure injury stages*).[20,21] Full discussion and description of all pressure injury stages is beyond the scope of this article, but additional information can be accessed from the National Pressure Ulcer Advisory Panel (NPUAP) website at www.npuap.org/resources/educational-and-clinical-resources/npuap-pressure-injury-stages/.

The NPUAP staging system should be used only for injuries caused by pressure.[20,22] Other skin injuries can be classified as either full thickness or partial thickness, depending on the depth of tissue damage in the wound. This discussion focuses on Stage 1 and 2 pressure injuries and deep tissue pressure injuries (DTPI) as defined by the NPUAP because these are the pressure injuries most likely to be confused with IAD.

The difficulty in differentiating Stage 1, Stage 2, or suspected DTPI from IAD has been documented in the literature as problematic, particularly in patients with dark skin.[6,7,13,15,23] Improving the nurse's ability to correctly identify skin injuries can lead to better patient outcomes.

Skin deep

The skin is the most visible and one of the largest organs in the human body.[24-26] Its functions include protection against physical injury, sunlight, and microorganisms; prevention of fluid loss from the internal environment; regulation of body temperature; continual reception of sensations from the environment, such as temperature and pain; and Vitamin D synthesis through the action of sunlight on the skin.[27]

The skin consists of two main layers: the epidermis, a superficial and thinner epithelial tissue layer, and the dermis, which is a layer of deeper and thicker connective tissue. The basement membrane zone is an interface between the dermis and epidermis. A layer of subcutaneous tissue called the hypodermis stores fat and contains large blood vessels.[27]

Skin thickness varies depending on its location on the body; for example, skin on the soles of the feet is thicker than the skin covering an eyelid. The amount of adipose tissue in the subcutaneous layer can also vary. The pinnae of the ears, the bridge of the nose, and the malleoli, for example, have little to no adipose tissue. As a result, skin injury in these areas can easily become full thickness even though they appear as shallow injuries.[22]

The skin is capable of regeneration and, under normal conditions, is strong enough to withstand minor insults without epithelial erosion.[28,29] Partial-thickness injuries can regenerate via epithelial migration, leaving little to no evidence of prior injury.[26,30] However, skin that's overhydrated or overly dry can be at risk for mechanical injury, such as friction, shear, and pressure. (See *Differentiating friction and shear injuries*.) Overhydrated skin loses its barrier function, making it susceptible to erythema and denudation from enzymes in urine and stool.[25,28,31]

Unique characteristics of darker skin tones include higher lipid content and the presence of a greater number of melanosomes in the stratum corneum compared with levels of lipid and melanosomes in light skin. These differences can mask erythema and may cause inflammation to appear as violet-black or black.[26] Because darker skin has more melanin than light skin, assessment of blanching can also be muted, complicating identification of early skin injury.[10,26]

Incontinence-associated dermatitis

Moisture-associated skin damage (MASD) is defined as damage to the skin secondary to persistent exposure to moisture in the affected area.[32,33] IAD, a form of MASD, has been defined as a skin injury or inflammation that presents with erythema and edema, with or without blisters, and denudation that occurs as a result of the skin's loss of moisture barrier from exposure to urine and stool.[32,33]

Differentiating friction and shear injuries

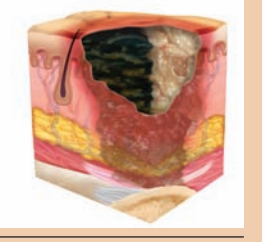
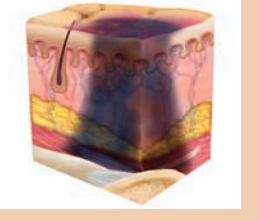
Friction is defined as the resistance to motion in a parallel direction relative to the common boundary of two surfaces causing superficial skin injury when the skin rubs against another surface.[2] Such injuries can easily occur when a patient is dragged during repositioning instead of being lifted.[33,37] This type of injury is shallow and limited to the epidermis; it doesn't result in the ischemic skin changes associated with pressure injury.

Friction is no longer considered an etiologic factor for pressure injury, and wounds caused by friction shouldn't be classified as pressure injuries.[22,33,38]

Shear is defined as a mechanical force that's parallel and opposite. It's also affected by the presence of pressure and is considered an etiologic factor in pressure injury development.[21,22,33,37] Deep shear force can create skin damage by the interaction of parallel movement and pressure while the body is in motion.

Shear injuries commonly occur when a patient slides down in bed. The muscle and deep underlying vessels can be stretched and compressed between the bone and support surface as the patient moves downward and the skin is held in place.[21,31] This can cause deep tissue injury resulting from ischemia.[31,33]

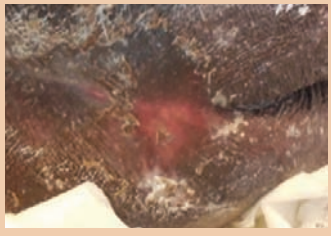
NPUAP pressure injury staging[39]

Pressure injury stage	NPUAP definition	
Stage 1	Intact skin with a localized area of nonblanchable erythema, which may appear differently in darkly pigmented skin. Presence of blanchable erythema or changes in sensation, temperature, or firmness may precede visual changes. Color changes do not include purple or maroon discoloration; these may indicate deep tissue pressure injury.	Stage 1 pressure injury – lightly pigmented  Stage 1 pressure injury – darkly pigmented 
Stage 2	Partial-thickness loss of skin with exposed dermis. The wound bed is viable, pink or red, moist, and may also present as an intact or ruptured serum-filled blister. Adipose (fat) is not visible and deeper tissues are not visible. Granulation tissue, slough, and eschar are not present. These injuries commonly result from adverse microclimate and shear in the skin over the pelvis and shear in the heel. This stage should not be used to describe moisture associated skin damage (MASD) including incontinence associated dermatitis (IAD), intertriginous dermatitis (ITD), medical adhesive related skin injury (MARSI), or traumatic wounds (skin tears, burns, abrasions).	
Stage 3	Full-thickness loss of skin, in which adipose (fat) is visible in the ulcer and granulation tissue and epibole (rolled wound edges) are often present. Slough and/or eschar may be visible. The depth of tissue damage varies by anatomical location; areas of significant adiposity can develop deep wounds. Undermining and tunneling may occur. Fascia, muscle, tendon, ligament, cartilage, and/or bone are not exposed. If slough or eschar obscures the extent of tissue loss, this is an Unstageable Pressure Injury.	
Stage 4	Full-thickness skin and tissue loss with exposed or directly palpable fascia, muscle, tendon, ligament, cartilage, or bone in the ulcer. Slough and/or eschar may be visible. Epibole (rolled edges), undermining, and/or tunneling often occur. Depth varies by anatomical location. If slough or eschar obscures the extent of tissue loss, this is an Unstageable Pressure Injury.	
Unstageable	Full-thickness skin and tissue loss in which the extent of tissue damage within the ulcer cannot be confirmed because it is obscured by slough or eschar. If slough or eschar is removed, a Stage 3 or Stage 4 pressure injury will be revealed. Stable eschar (that is, dry, adherent, intact without erythema or fluctuance) on the heel or ischemic limb should not be softened or removed.	
Deep tissue pressure injury	Intact or nonintact skin with localized area of persistent nonblanchable deep red, maroon, purple discoloration, or epidermal separation revealing a dark wound bed or blood-filled blister. Pain and temperature change often precede skin color changes. Discoloration may appear differently in darkly pigmented skin. This injury results from intense and/or prolonged pressure and shear forces at the bone-muscle interface. The wound may evolve rapidly to reveal the actual extent of tissue injury, or may resolve without tissue loss. If necrotic tissue, subcutaneous tissue, granulation tissue, fascia, muscle, or other underlying structures are visible, this indicates a full thickness pressure injury (Unstageable, Stage 3, or Stage 4). Do not use DTPI to describe vascular, traumatic, neuropathic, or dermatologic conditions.	

The NPUAP guidelines suggest that MASD can increase the risk of pressure injury development and this correlation has been well documented.[2,17,33]

Skin lesions resulting from IAD may be located on the buttocks, perineum, groins, vulva, scrotum, and perirectal area, as well as on the posterior and medial thighs.[30-33] The location of injury depends on the presence of urinary, fecal, or dual incontinence and can be most severe when the patient is having frequent episodes of incontinence and exposure to liquid fecal effluent.[18,28,31] Prolonged exposure to chemical irritants in urine and stool impairs the skin's protective acid mantle, thereby disrupting its "brick and mortar" barrier function.[18,28]

Types of skin injuries: Differential assessment

Differential characteristics	Friction	Incontinence dermatitis	Pressure/shear
	 Friction injury	 Incontinence dermatitis on sacrum and buttock	 Pressure injury on sacrum and coccyx
Location	Fleshy, soft tissue areas that are exposed to repetitive rubbing	• Perineal areas: groin, buttocks, vulva, perirectal areas, scrotum, and upper thighs	• Over bony prominences or medical device. • Atypical: over fleshy areas exposed to prolonged pressure.
Depth	Usually partial thicknesss (superficial)	• Usually partial thickness (superficial)	• Usually full thickness (stage 3 or 4), DTPI, or unstagable.
Wound bed	Wound bed is usually pink/red with no evidence of necrosis.	• Pink wound bed, may be blistering, or lifting of the epithelial layer/denudation. • In patients with dark skin tones, look for areas of hyperpigmentation.	• Well-defined lesions. • Tunneling and undermining may be present. • Tissue necrosis may be evident (purple discoloration, blood-filled blister, slough or eschar). • In patients with dark skin tones, palpate for warmth and induration or bogginess, as this may be an early sign of pressure injury.
Wound margins	• Edges may be well defined or irregular. • Skin flap may or may not be attached.	• Diffuse margins; satellite lesions are associated with fungal component.	• Well-defined, distinct borders.
Patient history	Restless, frequent sliding in chair; may be diaphoretic; may be malnourished; may have edema; may be on steroids, which can affect skin turgor and elasticity.	• Prolonged, repeated exposure to urine and/or stool. • Patient may complain of burning sensation in area of injury.	• Prolonged immobility or exposure to shear. • Use of medical device in area of injury. • Patient may have periods of hypotension and require vasopressor administration. • Patient may report pain at the site of injury.

Source: Adapted with permission from Doughty DB, McNichol LL. General concepts related to skin and soft tissue injury caused by mechanical factors. In: Doughty D, McNichol L, eds. *Wound Ostomy Continence Nurse Society Core Curriculum: Wound Management.* 1st ed. Philadelphia, PA: Wolters Kluwer; 2016:273-279.

FRICTION INJURY IMAGE COURTESY OF WOUND OSTOMY AND CONTINENCE NURSING SOCIETY. USED WITH PERMISSION. OTHER PHOTOS COURTESY OF AUTHOR.

IAD typically presents as moist, erythematous skin irritation and inflammation with superficial epithelial edema and diffuse, irregular borders.[18,28,31] IAD can also present with erosion of the epithelial layer or denudation. In addition, weeping and blistering or vesicle formation can occur.

The appearance of satellite lesions or vesicles is likely due to secondary cutaneous candidiasis and is often associated with more severe cases of IAD.[18,27,31,32] In patients with darker skin tones, the erythema may be subtle, appearing as a discoloration in comparison with surrounding skin.[34]

Patients with IAD may report a burning sensation with pain and pruritus. If exudate is present, it's often serous and weepy.[33,34] (See *IAD: Light skin tone and dark skin tone*.)

IAD: Light skin tone and dark skin tone

Light skin tone
Note the erythema, diffuse irregular margins, and formation of vesicles (small fluid-filled blisters <0.5cm) that are often seen with this type of skin injury

Dark skin tone
Sacrum of patient with IAD. Note diffuse margins of skin injury, denudation (epithelial erosion), and discoloration.

PHOTOS COURTESY OF THE AUTHOR.

Preventing IAD

Nursing care for patients at risk for IAD includes implementation of a structured skin care regimen, which is especially important for patients who are incontinent. Most regimens involve gentle cleansing with a soft cloth and a no-rinse cleansing formula that contains surfactants to loosen irritants, and application of a barrier ointments.[31,32] See www.Nursing2018.com for supplemental online content, *Sample skin care bundle, based on Braden Risk Tool*.

Moisture barrier ointments serve to protect the skin by providing a barrier to effluent. Many barrier formulations are available; they may be petroleum-based, dimethicone-based, or zinc oxide ointments.[18,28,32-34]

Use of incontinence pads and briefs should also be included in an incontinence management program. Use materials that are highly absorbent and frequently change garments as needed to keep the skin dry and prevent fungal dermatitis.[18,28,32,33]

Differentiating pressure injuries

The NPUAP defines pressure injury as *"localized damage to the skin and underlying soft tissue usually over a bony prominence or related to a medical or other device. This injury can present as intact skin or an open ulcer and may be painful. The injury occurs as a result of intense and/or prolonged pressure or pressure in combination with shear. The tolerance of soft tissue for pressure and shear may be affected by microclimate, nutrition, perfusion, comorbid conditions, and condition of the soft tissue."*[22]

Risk factors for pressure injury can be intrinsic and extrinsic.[16,21] Intrinsic factors, which come from within, include age, comorbidities, nutritional status, hypotension, elevated body temperature, immobility, and signs of stress, such as wrinkling and decreased skin turgor and elasticity.[16,35] Extrinsic risk factors include environmental forces that act to cause injury, such as shear, moisture, and pressure.[16,21,35]

Careful and thorough skin inspection is warranted when assessing for Stage 1 pressure injury or DTPI, particularly in patients with dark skin tones.[10,22] For tips and guidelines, see *Types of skin injuries: Differential assessment*. Updated NPUAP risk assessment tips and prevention interventions can be found on the NPUAP website at www.npuap.org/wp-content/uploads/2016/04/Pressure-Injury-Prevention-Points-2016.pdf.

Assessment strategies

To differentiate skin injuries accurately, first obtain a comprehensive health history to identify any risk factors for pressure injury and/or IAD. Also ask the patient or caregiver about the patient's bathing and toileting routine.

When assessing the patient's skin, always ensure adequate lighting so you can detect subtle changes in skin color. Appropriate lighting with natural lighting, a halogen lamp, or a flashlight is especially important for inspecting dark skin tones. Avoid fluorescent lighting, which may cast a bluish tint on dark skin.[10,36]

Inspect the skin for slight changes in color surrounding the assessment area, and make sure you examine the area from multiple angles. Sullivan reports that hyperpigmentation may

be a sign of early skin injury in patients with dark skin.[11]

Palpate the area for changes in temperature and texture.[10,11,36] Localized heat, edema, or any change in tissue consistency, such as induration or hardness, may be a sign of pressure injury.[10,22]

Ask the patient to describe pain or discomfort. Pressure injuries may be painful; a burning sensation suggests IAD.[10,33] When skin injury is identified, the nurse should document the injury type, characteristics, and measurements. Some facilities also document skin injuries with photographs and rely on these documentation strategies for ongoing assessment for improvement.

Working toward better outcomes

Pressure injury and IAD are common in all healthcare settings. Nurses must accurately identify and classify skin injuries to ensure high-quality patient care. This requires knowledge of normal and abnormal variations in skin tones, especially in dark skin. Armed with clinical knowledge and assessment skills, nurses are prepared to identify early signs and symptoms of skin injury and to implement interventions that will improve patient outcomes across the continuum of care. ■

Localized heat, edema, or a change in tissue consistency, such as induration or hardness, may indicate pressure injury.

REFERENCES

1. Chen HL, Cai DY, Shen WQ, Liu P. Bibliometric analysis of pressure ulcer research: 1990-2009. *J Wound Ostomy Continence Nurs.* 2010;37(6):627-632.

2. National Pressure Ulcer Advisory Panel, European Pressure Ulcer Advisory Panel, & Pan Pacific Pressure Ulcer Injury Alliance. *Prevention and Treatment of Pressure Ulcers: Quick Reference Guide.* 2014. www.npuap.org/wp-content/uploads/2014/08/Updated-10-16-14-Quick-Reference-Guide-DIGITAL-NPUAP-EPUAP-PPPIA-16Oct2014.pdf.

3. Lyder CH, Wang Y, Metersky M, et al. Hospital-acquired pressure ulcers: results from the national Medicare patient safety monitoring system study. *J Am Geriatr Soc.* 2012;60(9):1603-1608.

4. Thomas A. Assessment of nursing knowledge and wound documentation following a pressure ulcer educational program in a long-term care facility: a capstone project. *Wound Pract Res.* 2012;20(3):142-158.

5. Levine JM, Zulkowski KM. Secondary analysis of office of inspector general's pressure ulcer data: incidence, avoidability, and level of harm. *Adv Skin Wound Care.* 2015;28(9):420-428.

6. Clarke-O'Neill S, Farbrot A, Lagerstedt Eidrup ML, Cottenden A, Fader M. Is it feasible to use incontinence-associated dermatitis assessment tools in routine clinical practice in the long-term care setting? *J Wound Ostomy Continence Nurs.* 2015;42(4):379-388.

7. Lilly D, Estocado N, Spencer-Smith JB, Englebright J. Validation of the NE1 wound assessment tool to improve staging of pressure ulcers on admission by registered nurses. *J Nurs Meas.* 2014;22(3):438-450.

8. Ayello EA, Sibbald RG, Quiambao PCH, Razor B. Introducing a moisture-associated skin assessment photo guide for brown pigmented skin. *World Counc Enterostomal Ther J.* 2014;34(2):18-25.

9. Sibbald GR, Krasner DL, Woo KY. Pressure ulcer staging revisited: superficial skin changes & Deep Pressure Ulcer Framework©. *Adv Skin Wound Care.* 2011;24(12):571-580.

10. Steven M, Struble L, Larson JL. Recognizing pressure injury in the darkly pigmented skin type. *Medsurg Nurs.* 2015;24(5):342-348.

11. Sullivan R. A 5-year retrospective study of descriptors associated with identification of stage I and suspected deep tissue pressure ulcers in persons with darkly pigmented skin. *Wounds.* 2014;26(12):351-359.

12. Bliss DZ, Hurlow J, Cefalu J, Mahlum L, Borchert K, Savik K. Refinement of an instrument for assessing incontinence-associated dermatitis and its severity for use with darker-toned skin. *J Wound Ostomy Continence Nurs.* 2014;41(4):365-370.

13. Bates-Jensen BM, McCreath HE, Pongquan V, Apeles NC. Subepidermal moisture differentiates erythema and stage I pressure ulcers in nursing home residents. *Wound Repair Regen.* 2008;16(2):189-197.

14. Francis K. Incontinence-associated dermatitis: management update. *Am Nurse Today.* 2018;13(1):25-27.

15. Bruce TA, Shever LL, Tschannen D, Gombert J. Reliability of pressure ulcer staging: a review of literature and 1 institution's strategy. *Crit Care Nurs Q.* 2012;35(1):85-101.

16. White-Chu EF, Kiely DK. Factors associated with physician and nurse practitioner pressure ulcer staging practices in rehabilitative and long-term care. *J Am Med Dir Assoc.* 2012;13(8):748-751.

17. Beeckman D, Van Lancker A, Van Hecke A, Verhaeghe S. A systematic review and meta-analysis of incontinence-associated dermatitis, incontinence, and moisture as risk factors for pressure ulcer development. *Res Nurs Health.* 2014;37(3):204-218.

18. Voegeli D. Incontinence-associated dermatitis: new insights into an old problem. *Br J Nurs.* 2016;25(5):256-262.

19. Paulin R, Dowling-Castronovo A. Incontinence-associated dermatitis — it is not a pressure injury. *Urol Nurs.* 2017;37(6):304-309.

20. Edsberg LE, Black JM, Goldberg M, McNichol L, Moore L, Sieggreen M. Revised National Pressure Ulcer Advisory Panel pressure injury staging system: revised pressure injury staging system. *J Wound Ostomy Continence Nurs.* 2016;43(6):585-597.

21. Stechmiller JK, Cowan LJ, Oomens CW. Bottom-up (pressure shear) injuries. In: Doughty D, McNichol L, eds. *Core Curriculum: Wound Management.* Philadelphia, PA: Wolters Kluwer; 2016:313-332.

22. National Pressure Ulcer Advisory Panel. Pressure injury prevention points. 2016. www.npuap.org/wp-content/uploads/2016/04/Pressure-Injury-Prevention-Points-2016.pdf.

23. Mahoney M, Rozenboom B, Doughty D, Smith H. Issues related to accurate classification of buttocks wounds. *J Wound Ostomy Continence Nurs.* 2011;38(6):635-642.

24. Casey G. Disorders of the skin. *Nurs N Z.* 2016;22(3):20-24.

25. Mufti A, Ayello EA, Sibbald RG. Anatomy and physiology of the skin. In: Doughty D, McNichol L, eds. *Core Curriculum: Wound Management.* Philadelphia, PA: Wolters Kluwer; 2016:3-23.

26. Wysocki AB. Anatomy and physiology of skin and soft tissue. In: Bryant R, Nix D, eds. *Acute & Chronic Wounds: Current Management Concepts.* 4th ed. St. Louis, MO: Elsevier Mosby; 2012:40-62.

27. Porth CM. *Essentials of Pathophysiology.* 4th ed. Philadelphia, PA: Wolters Kluwer/Lippincott Williams & Wilkins; 2014:1142.

28. Holroyd S. Incontinence-associated dermatitis: identification, prevention and care. *Br J Nurs.* 2015;24(9):S37-S43.

29. McLafferty E, Hendry C, Alistair F. The integumentary system: anatomy, physiology and function of skin. *Nurs Stand.* 2012;27(3):35-42.

30. Beitz JM. Wound healing. In: Doughty D, McNichol L, eds. *Core Curriculum: Wound Management.* Philadelphia, PA: Wolters Kluwer; 2016:24-35.

31. Gray M, Beeckman D, Bliss DZ, et al. Incontinence-associated dermatitis: a comprehensive review and update. *J Wound Ostomy Continence Nurs.* 2012;39(1):61-74.

32. Black JM, Gray M, Bliss DZ, et al. MASD part 2: incontinence-associated dermatitis and intertriginous dermatitis: a consensus. *J Wound Ostomy Continence Nurs.* 2011;38(4):359-370.

33. Gray M, McNichol L, Nix D. Incontinence-associated dermatitis: progress, promises, and ongoing challenges. *J Wound Ostomy Continence Nurs.* 2016;43(2):188-192.

34. Doughty DB, McNichol LL. General concepts related to skin and soft tissue injury caused by mechanical factors. In: Doughty D, McNichol L, eds. *Wound Ostomy Continence Nurse Society Core Curriculum: Wound Management.* 1st ed. Philadelphia, PA: Wolters Kluwer; 2016: 273-279.

35. Gefen A. From incontinence-associated dermatitis to pressure ulcers. *J Wound Care.* 2014;23(7):345.

36. Sommers MS. Color awareness: a must for patient assessment. *Am Nurse Today.* 2011;6(1).

37. Wound Ostomy Continence Nursing Society. *Guideline for Prevention and Management of Pressure Ulcers.* Mt. Laurel, NJ: Wound Ostomy Continence Nursing Society; 2016.

38. Berke CT. Pathology and clinical presentation of friction injuries: case series and literature review. *J Wound Ostomy Continence Nurs.* 2015;42(1):47-61.

39. National Pressure Ulcer Advisory Panel. NPUAP pressure injury stages. 2016. www.npuap.org/resources/educational-and-clinical-resources/npuap-pressure-injury-stages.

Kathleen Francis is NP coordinator of the Wound Ostomy Continence Specialist team at Maimonides Medical Center in Brooklyn, N.Y.

The author and planners have disclosed no potential conflicts of interest, financial or otherwise.

DOI-10.1097/01.NURSE.0000532739.93967.20