Accurate physical and psychosocial assessment is imperative to determine an appropriate plan of care for the patient with a pressure ulcer.1 Chronic wounds occur in 2.8 million people in the US and cost billions of dollars to treat.2 However, Patel and Granick3 examined 50 medical school curricula and found that medical students receive an average of only 4 hours of instruction on wound-related topics (including anatomy and physiology of wounds and wound healing) during their entire medical school training. Nurses receive similar minimal training. Vogelpohl and Dougherty4 reviewed 10 nursing textbooks and reported that on average only 200 lines of text and 10 tables were presented on wounds, some of which were inaccurate. Many nurses learn human anatomy using only textbook descriptions and pictures and lack a thorough knowledge of the appearance of human tissues in either a cadaver or surgical specimen. The lack of wound and pressure ulcer training also has an impact on practice. Subsequent inaccurate assessments of ulcer size, stage, and visible tissues may affect payment and processes of care needed to support healing can be developed in error. At times, these inaccuracies can lead to fines and litigation.

In addition, the nomenclature used to describe pressure ulcers and the tissues within the ulcer bed has not advanced in pace with the language used in other healthcare disciplines.
Although the pressure ulcer staging system has been refined significantly through the years (see Table 1) ongoing concern remains regarding healthcare professionals’ knowledge of anatomy and physiology necessary to accurately assess a patient’s pressure ulcer.3-5

In May 2008, a multidisciplinary panel of wound experts was assembled to provide anatomically accurate and practical terms associated with pressure ulcer assessment, healing, and nonhealing in order to help clinicians identify and describe tissue types and pressure ulcer stages.

Methods
A panel of American experts in pressure ulcers and wound healing was convened by the Hill-Rom Company, Inc. The group reviewed the current literature on wound healing and wound tissue terms. Photographs that clearly depicted the type of visible tissue in healing and nonhealing pressure ulcers were solicited before the meeting. The group examined the submitted photographs of wounds and discussed the anatomical tissues or characteristics of other visible tissue in each photograph. Descriptions of the tissues were drafted, defined, and revised using a consensus method. The final terminology and paper were circulated to all panelists. The National Pressure Ulcer Advisory Panel® (NPUAP) pressure ulcer staging system was used as the major focus because this staging system is used to secure payment. However, the definitions developed are not specific to pressure ulcer wounds because certain tissues (eg, eschar, slough, and granulation tissue) can be seen in many wound types.

Pressure Ulcer Location
Pressure ulcers can occur in any anatomical site but are most often found over bony prominences.6 These include the occiput (back of the head or skull), scapula (shoulder blade — ie, the bone forming the back of the shoulder), sacrum (triangular bone at the base of the spine), coccyx (end of the spine, tail bone), ischium (lowest of the three bones that make up each half of the pelvic sitting bone; the ulcer is present in the gluteal fold), trochanter (either of two rough knobs on the upper femur [thigh bone] where the muscles between the thigh and pelvis are attached), posterior superior iliac spine (PSIS, the bony spine under the dimples of the skin in the upper buttock region; a line passing between the right and left dimples lies at the level of the sacroiliac joint), calcaneus (posterior aspect of the heel, heel bone), lateral malleolus (ankle bone protuberance on the outside of the leg), and medial malleolus (ankle bone protuberance on the inside of the leg) (see Figure 1a,b). In shallow ulcers? Anatomical, histological, and physiological characteristics, as well as appearance upon palpation of normal tissues, are described for each tissue type (see Figure 1a,b).

Epidermis. The epidermis is the outermost layer of the skin. It forms a waterproof barrier around the body surface through its keratinized epithelial cells. The epidermis is avascular (lacking blood vessels) and is nourished by diffusion from the dermis. The four principal types of cells that make up the epidermis are keratinocytes, melanocytes, Langerhans cells, and Merkel cells. Keratinocytes are stratified, squamous epithelial cells on the outermost surface of the skin. They prevent invasion of the body by outside substances and prevent losses from the body to the outside environment. Turnover time for keratinocytes is about 30 days, with maturing cells replacing them from lower levels of the skin. Melanocytes provide protection from ultraviolet damage through the production of the pigment melanin. The relative activity of melanocytes and type of melanin produced influence skin tone. Langerhans cells provide immune protection. Merkel cells function remains a topic of debate, but they are thought to have a role in sensory or neuroendocrine activity.7,8

Although the base of the epidermis is living tissue, the outermost layer of epidermis consists of 25 to 30 layers of dead cells and little fiber. The layers in between are increasingly keratinized in that they are tough and insoluble the closer they become to the surface. The epidermis’ color is based on the density of melanocytes and regional blood flow. The bottom of the epidermis is the basal lamina, upon which new epidermal cells migrate when a wound heals.

Over most of the body, the epidermis is very thin, approximately 0.07 to 0.12 mm (the thickness of plastic wrap), similar to the thickness of peeling, sunburned skin. Epidermal thickness varies from very thin (eyelids) to thicker areas on the palms of the hands and the soles of the feet, where it can be up to 1.2 mm thick. The epidermis’ translucent quality can be appreciated when examining serum-filled blisters; because they often have a roof of thin epidermis, these blisters quickly
<table>
<thead>
<tr>
<th>Pressure ulcer definition</th>
<th>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 also are associated with pressure ulcers; the significance of these factors is yet to be elucidated</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pressure ulcer stages</td>
<td></td>
</tr>
<tr>
<td>Stage I</td>
<td>Intact skin with nonblanchable 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</td>
</tr>
<tr>
<td>Further description: The area may be painful, firm, soft, warmer or cooler as compared to adjacent tissue. Stage I may be difficult to detect in individuals with dark skin tones. May indicate “at risk” persons (a heralding sign of risk)</td>
<td></td>
</tr>
<tr>
<td>Stage II</td>
<td>Partial-thickness loss of dermis presenting as a shallow open ulcer with a red pink wound bed, without slough. Also may present as an intact or open/ruptured serum-filled blister</td>
</tr>
<tr>
<td>Further description: Presents as a shiny or dry shallow ulcer without slough or bruising.* This stage should not be used to describe skin tears, tape burns, perineal dermatitis, maceration, or excoriation. Bruising indicates suspected deep tissue injury</td>
<td></td>
</tr>
<tr>
<td>Stage III</td>
<td>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</td>
</tr>
<tr>
<td>Further description: The depth of a Stage III pressure ulcer varies by anatomical location. The bridge of the nose, ear, occiput, and malleolus do not have subcutaneous tissue and Stage III ulcers can be shallow. In contrast, areas of significant adiposity can develop extremely deep Stage III pressure ulcers. Bone/tendon is not visible or directly palpable</td>
<td></td>
</tr>
<tr>
<td>Stage IV</td>
<td>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</td>
</tr>
<tr>
<td>Further description: The depth of a Stage IV pressure ulcer varies by anatomical location. The bridge of the nose, ear, occiput, and malleolus do not have subcutaneous tissue and these ulcers can be shallow. Stage IV ulcers can extend into muscle and/or supporting structures (eg, fascia, tendon, or joint capsule) making osteomyelitis possible. Exposed bone/tendon is visible or directly palpable</td>
<td></td>
</tr>
<tr>
<td>Unstageable</td>
<td>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</td>
</tr>
<tr>
<td>Further description: Until enough slough and/or eschar is removed to expose the base of the wound, the true depth, and therefore stage, cannot be determined. Stable (dry, adherent, intact without erythema or fluctuance) eschar on the heels serves as “the body’s natural (biological) cover” and should not be removed</td>
<td></td>
</tr>
<tr>
<td>Suspected deep tissue injury</td>
<td>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. Deep tissue injury may be difficult to detect in individuals with dark skin tones. Evolution may include a thin blister over a dark wound bed. The wound may further evolve and become covered by thin eschar. Evolution may be rapid exposing additional layers of tissue even with optimal treatment</td>
</tr>
</tbody>
</table>

From the National Pressure Ulcer Advisory Panel (NPUAP)\(^6\)
Dermis. Located directly beneath the epidermis, the dermis is highly vascular and provides oxygen and nourishment to the epidermal layers above and to the living, deeper epidermal layers. The dermis is the thickest portion of the skin, averaging 1 mm thick (the thickness of a credit card) over most of the body; thickness varies from 0.3 mm on the eyelid to 3.0 mm on the back. The dermis contains collagen, elastin, and proteoglycan molecules that give the skin tone (suppleness) and strength to provide protection against stress and strain. The dermis consists of two major layers: the upper, papillary layer contains thin collagen fibers arranged in a proteoglycan-rich matrix and the lower, reticular layer is thicker and consists of large collagen fibers arranged in a basket-weave pattern parallel to the skin surface. The dermis is tightly connected to the epidermis by a basement membrane and encases many nerve endings sensitive to touch and heat.

The dermis contains hair follicles; sweat, sebaceous, and apocrine glands; and blood vessels. The blood vessels nourish dermis cells as well as the stratum basale (basal cell layer) of the epidermis. Dermis exposed in a wound appears wet (glistening or moist) and pink or red. If the epidermis and part of the dermis are missing, the pressure ulcer is described as Stage II.6,7

Superficial fascia. Superficial fascia, the tissue network that surrounds subcutaneous fat, is located directly beneath the dermis. In healthy skin, superficial fascia looks like a thin, glistening spider web.

Subcutaneous fat. In contrast to visceral fat found in the peritoneal cavity, subcutaneous fat is found just beneath the skin. Subcutaneous fat is stored energy and aids in thermal regulation. Subcutaneous fat cells contain a single droplet of oil. Fat cells are enmeshed in the superficial fascia. Subcutaneous fat is pale yellow, waxy, globular and oily, and glistens. When it dries, fat congeals (crystallizes) and turns to tan or yellow-brown, a common presentation in open wounds. When fat is visible in any portion of a pressure ulcer, the ulcer is assessed as Stage III or unstageable if fat covers the major portion of the wound.6,7

Muscle. Skeletal muscle contracts to move skeletal bones. In an ulcer bed, healthy muscle is shiny and red with visible striations, bulky, and will spring back when touched. Healthy muscle is highly vascular (many blood vessels) and deep red in color. In contrast, avascular or ischemic muscle is dull red, cyanotic or pale, and mushy. Visible muscle may be confused with granulation tissue. The primary difference between the two is that muscle is smooth and healthy granulation tissue is bumpy.

If muscle is exposed, the wound will not heal until it becomes covered by granulation tissue or is surgically covered with a flap. Re-epithelialization will not occur over muscle because no basal lamina is present for epithelial cells to migrate across until granulation tissue has formed. Muscle that remains exposed to the environment will eventually die. Necrotic muscle liquefies and produces dark brown, odorous drainage. If muscle is the deepest tissue visible in a pressure ulcer, the ulcer is full-thickness and classified as a Stage IV pressure ulcer.6

Deep fascia. Deep fascia is the dense fibrous connective tissue that surrounds the muscles, bones, nerves, and blood vessels. A high density of collagen and elastin fibers provides its strength. Although essentially avascular, deep fascia is richly innervated with pain receptors (nociceptors). Fascia appears as a glistening surface on the top of muscle. Stage IV pressure ulcers can feature visible deep fascia.6,7

Tendon. Tendon is a tough band of fibrous connective tissue that connects muscle to bone. Shiny white with fibrous striations, tendon springs back when touched. When a joint is moved, an intact tendon will move; this is one way to dif-
ferentiate the tendon from other white tissue or slough. Once the tendon is exposed to the environment, it dries, and the sheath often will not survive. If tendon is visible in a pressure ulcer, the ulcer is Stage IV.6-8

**Ligament.** A ligament is a short band of connective tissue holding bones together; therefore, ligaments are seen in or near joints. Ligaments appear ribbon-like, striated, and pearly white. The striations feel like tape with embedded threads. Ligaments are broader, flatter, and more loosely woven than tendons. Pressure ulcers with visible ligaments are classified Stage IV.5,7

**Nerve.** A peripheral nerve is long, white, tubular, rubbery, and usually thin (2 to 3 mm in diameter), with a few branch points. Nerves lie within the plane of the wound; rarely will nerve be visible in a pressure ulcer. Touching will briefly inactivate and potentially damage the nerve and should be avoided. A pressure ulcer with visible nerve is Stage IV.7,8

**Bursa.** Bursa (plural, bursae) is a small fluid-filled sac of white fibrous tissue lined with synovial membrane filled with synovial fluid. Bursa provides a cushion between bones, tendons, and/or muscles around a joint. If bursae open, they leak a sticky, mucous-like fluid. Pressure ulcers with visible bursa tissue are classified Stage IV.6,7

**Bone.** Bone is the skeletal frame of the body. Healthy bone is creamy white, hard, and covered with periosteum (sheath of connective tissue that surrounds the bone) that is shiny and slick to the touch. If the periosteum is present, the bone is smooth. If the periosteum is missing, the bone feels somewhat rough. Unhealthy bone is flaky or mealy and will have a gray cast or appear brown or black. Rough or mushy bone suggests the presence of osteomyelitis. Bone that is exposed or allowed to dry out may die. If bone is visible in a pressure ulcer, the ulcer is Stage IV.6-8

**Tissues in Open Wounds**

The panel agreed that during the course of healing many tissues exist in a wound bed. The panel also agreed that proper identification of wound bed tissues allows a pressure ulcer to be correctly staged. During the course of pressure ulcer healing, many forms of tissue debris and tissue types may appear; some but not all may signify healing (see Figure 2). The clinician assessing the wound must correctly identify the tissue types observed and provide appropriate intervention (see Table 2).

**Epithelium.** Epithelialization is regeneration of epidermis across a wound surface. The process of epithelialization begins when basal keratinocytes from the wound edges’ dermal appendages migrate across the wound site and proliferate at its edges, stopping when they meet in the middle. The epithelial cells secrete a scaffolding (basal lamina) as they advance from the wound edges. New tissue appears flat, thin, and lighter in color than usual pigmentation (often light pink).8,9

When a wound is re-epithelialized, initially the epidermal layer is only a few cell layers thick and the new tissue appears translucent. This new epithelium is vulnerable to injury from pressure, shearing, or friction. Maturation of the new cells with a keratinized layer takes 2 to 3 weeks — ie, until the transepidermal water loss barrier returns.

**Granulation tissue.** The consensus group agreed that formation of granulation tissue is an intermediate step in healing full-thickness wounds. Further, because granulation tissue is fragile, it can be easily injured. Healthy granulation tissue is red, shiny and granular. Granulation tissue without adequate blood flow becomes pale.

Granulation tissue is comprised of new capillary tufts, matrix, fibroblasts, and collagen. It provides the early scaffolding to promote healing from the edges of a full-thickness pressure ulcer. Granulation tissue is an intermediate step in the healing process. Granulation tissue does not mature into epithelium. As a wound heals, a layer of epidermis will cover the granulation tissue.

Healthy granulation tissue appears beefy red, bumpy, moist, and shiny. Because granulation tissue is soft and fragile, it can be damaged by dry or adherent dressings, tight wound packing, and/or high intensity wound irrigation, as well as pressure. When too much pressure is applied, granulation tissue darkens. Ischemia (lack of blood flow) can cause granulation tissue to appear paler in color. Infection also can destroy granulation tissue. Injured granulation tissue often leads to a new onset of pain as commonly seen with dressing changes.10

---

**Figure 2. Pressure ulcer healing.**

©2010 Hill-Rom Services Inc. ALL RIGHTS RESERVED
### Table 2. Wound Tissue and Debris

<table>
<thead>
<tr>
<th>Type</th>
<th>Color</th>
<th>Characteristics</th>
<th>Composition</th>
<th>Significance</th>
</tr>
</thead>
<tbody>
<tr>
<td>Slough</td>
<td>Most commonly yellow, tan,</td>
<td>Soft, moist, stringy; may be gel-like, mucinoid (mucus-like); may be loosely or</td>
<td>Mixture of serum proteins (fibrin, albumin, immunoglobulin) and collagen; frequently harbors bacteria and biofilms.</td>
<td>If slough is in the wound, assume it is inflamed and colonized with bacteria. If signs of infection are noted, the wound should be debrided. An ulcer with unstable eschar also may be unstageable because the base of wound cannot be seen.</td>
</tr>
<tr>
<td></td>
<td>gray, greenish, or white;</td>
<td>firmly adherent. As slough ages or dries, it becomes more gelatinous; thicker</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>may be shiny</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Stable eschar</td>
<td>Brown or black in color</td>
<td>Usually dry, hard, leathery</td>
<td>Desiccated skin, fat, tendon and/or muscle. Further necrosis of deeper tissue. As tissue dies, it changes in color and consistency, and adheres to wound bed. As necrotic tissue increases in severity, the color finally progresses to brown or black; dry and desiccated ischemia may cause necrosis of underlying tissue</td>
<td>An ulcer with eschar may also be unstageable because the base of wound cannot be seen. Until enough eschar is removed to expose the base of the wound, the true depth cannot be determined. Stable eschar on distal arterial wounds serves as the body’s natural (biological) cover and should not be removed.</td>
</tr>
<tr>
<td>Unstable eschar</td>
<td>Brown or black in color; commonly accompanied by inflammation; can be shiny</td>
<td>Boggy (spongy), slimy</td>
<td>Same as stable eschar – desiccated muscle, tendon, and fat. Further necrosis of deeper tissue.</td>
<td>Bacteria are present on viable tissue underneath the unstable eschar. If signs of infection are noted, the wound should be debrided. An ulcer with unstable eschar also may be unstageable because the base of wound cannot be seen.</td>
</tr>
<tr>
<td>Granulation tissue</td>
<td>Beefy red or deep pink in color; shiny, bumpy surface</td>
<td>Supple, moist</td>
<td>New capillaries, matrix, fibroblasts, collagen</td>
<td>Granulation tissue fills in the wound with early scar tissue. Staging is possible because the wound bed is visible.</td>
</tr>
</tbody>
</table>

Continued on next page
Granulation tissue may stop forming, and wound healing may stall when blood flow is impaired, invasive infection is present, or nutrients are deficient.11

**Slough.** The panel agreed that slough (rhymes with rough) is an inflammatory byproduct and not a physical tissue. It is a coagulum of serum and matrix proteins produced by inflamed wounds. Slough is usually described as a type of necrotic tissue.12 However, slough is a mixture of serum proteins (fibrin, albumin, immunoglobulin) and denatured matrix proteins (collagen). These extracellular fluids form during inflammation and leak into interstitial spaces due to capillary dilation. Therefore, slough is an indicator of wound inflammation. Healing will not begin until the slough is removed and the cause of the inflammation controlled.9

Slough may look like an attached stringy mass and may be loosely or firmly adherent. As slough ages, it becomes thicker. Slough can be many colors, depending on its bacterial composition. White slough indicates bacterial colonization is scant, yellow or green indicates greater count of bacterial composition, and brown also may include hemoglobin.

Slough is seen in Stage III or Stage IV pressure ulcers. Slough in the wound indicates the pressure ulcer is full-thickness. Stage II pressure ulcers do not generate enough of an inflammatory response to produce slough. When dark yellow, slough can be confused with normal anatomical tissue such as cutaneous ligaments, fascia, tendons, ligaments, joint capsules, and bursae. Identification of the actual tissue present is important. Viable normal tissue should not be debrided; slough should be removed. Debridement is indicated for any wound, acute or chronic, when slough or necrotic tissue is present. Once the chronic wound bed is clean and viable tissue is present, debridement is not necessary.13 Although slough is an indication of full-thickness injury, pressure ulcers are unstageable when slough obscures the wound bed.6 Figure 3 shows a Stage IV pressure ulcer of the sacrum. The presence of bone in the ulcer makes this ulcer a Stage IV, even though slough is visible; because the slough does not prevent or obscure the assessment of ulcer depth, the ulcer can be staged.

**Eschar.** The group agreed that eschar (pronounced S-car) is comprised of dead tissue and becomes dark in color due to the presence or absence of hemoglobin in the tissues. Eschar is necrotic tissue — in particular, desiccated granulation tissue, skin, fat or tendon, or muscle. Eschar is black or brown, colored by hemoglobin in the wound tissues. **Stable eschar** — intact and hard black or brown eschar on the heels surrounded by tissues that are not indurated (hardened), fluctuant (fluid moving under the tissue), crepitant (tissue feels crunchy upon palpation), painful, or draining — is dry, hard, and leathery. Stable eschar may serve as a barrier to prevent bacterial
is a similar situation of stable eschar, sometimes called dry gangrene, and should be left dry. Softening the eschar allows bacteria to advance into the living tissues, leading to wet gangrene.

Wound Edges

Rolled wound edge (also called epiboly or epibole). As full-thickness pressure ulcers heal, the epithelium attempts to advance. However, without a basal laminal layer of tissue, the leading edge of the epithelium cannot migrate and begins to roll onto itself. The edge of the wound is also raised and paler or pinker than surrounding tissue. Generally, this type of a pressure ulcer will not continue to heal without intervention of some kind; healing has stalled. This presentation also has been called a “closed” wound edge.7,8

Flat wound edge. A flat wound edge can be seen when the ulcer is epithelializing. Irregular tissue at the wound border appears as a nonshiny, pale tissue edge. In Stage II pressure ulcers (partial-thickness), epithelial cells migrate from the ulcer edges and up through the hair follicles, so small islands of epithelialization will be seen within the ulcer bed.6,14

Undermining. The panel agreed that undermining is most often due to shear forces in the wound. Undermining is a gap in the tissue edge that creates a lip or overhang of tissue. A wound with undermining has a smaller surface area than at the wound base. Usually the wound has to heal the undermined area first before it can close. Sacral pressure ulcers in patients who can sit up often develop undermining in the upper half of the ulcer, commonly described as from 3 to 9 o’clock.

Ulcer Location

Tissue and wound descriptions always should include the precise location of a pressure ulcer. Simply noting “pressure ulcer present” does not provide enough information. Figure 3 provides correct anatomical terms to be used when describing the location of a pressure ulcer. Neutral skin positions — ie, when skin is not stretched or pulled — should be maintained when identifying ulcer location; otherwise, the actual location of the ulcer is distorted. For example, when the patient is positioned on his side and the buttocks lifted to inspect the ulcer, the ulcer can appear to be over the middle of the buttocks when, in fact, with the tissues in a neutral position, the ulcer is actually over the ischia. This issue becomes increasing complex through future assessments when the patient may be positioned differently.7

Pressure Ulcer Healing

The 1994 Wound Healing Society’s15 (WHS) proposed definitions of wound healing are supported by the consensus panel. According to WHS definitions, an ideally healed wound is one that has returned to normal anatomic structure,
function, and appearance. In humans, this degree of skin wound healing occurs only in epidermal tissue and superficial dermis or mucous membrane. When deeper dermis or beyond is injured, normal appearance cannot return; scar tissue replaces the missing tissue. A *minimally healed wound* demonstrates restoration of anatomic continuity without a sustained functional result. Hence, the wound may recur because the scar has less tensile strength and will never regain all the features of uninjured skin. Between these two extremes of healing, an *acceptably healed wound* is characterized by restoration of sustained function and anatomic continuity. The minimally healed wound is best described as a *closed wound*, recognizing it is more prone to breakdown than intact skin nearby.\(^{13,14}\)

The panel also agreed that persons with closed full-thickness pressure ulcers or any wound on a pressure bearing area remain at risk of future pressure ulcers due to the reduced tensile strength of scar tissue. The panel made no recommendations on how to label the stage a new ulcer on a prior closed ulcer’s scar.

Because full-thickness pressure ulcers heal only by scar and contraction, the wound only can be said to have closed. Wound collagen never achieves the normal structure of dermal collagen and therefore the scar remains vulnerable to breakdown.\(^8\) Patients with closed pressure ulcers remain vulnerable to tissue loss on top of the scar as long as they live. It also is recognized that burn patients, patients who need a prosthetic limb, and surgical wound patients should be followed to ensure continued healing. Little of this type of follow-up occurs with pressure ulcers, despite recidivism rates.

The panel agreed that the long-term healing of pressure ulcers should be studied and provides the basis for discussions between wound care professionals, researchers, and payors. Development of a set of interventions and protocols for continued treatment of pressure ulcers could reduce recurrence and subsequent healthcare expenditures. Wound healing professionals and payors are beginning to recognize this issue and some work and discussions have begun.\(^{16}\)

The Centers for Medicare and Medicaid Services (CMS) already accept that on the OASIS data set, a Stage III or Stage IV pressure ulcer defined as closed is still considered a pressure ulcer.\(^{17}\) The panel agrees and would not encourage the use of the word healed with regard to pressure ulcers that are full-thickness, Stage III, and Stage IV. These wounds may close, meaning the integrity of the skin is repaired, but they are not healed in that the original tissue was not replaced.

Likewise, the panel supported the idea that patients whose pressure ulcers have been closed with a fasciocutaneous or myocutaneous flap also should be followed to ensure the flap survives. Even though new vascularized tissue has been placed in the pressure ulcer bed, the flap must inset and survive in the new area, leaving the flap at risk for weeks following surgery.

Flaps cannot tolerate extremes of pressure and shear; patients with flaps need to be advanced slowly onto surfaces that place pressure on the flap. This pressure strain encourages alignment of fibroblasts to improve wound tensile strength. Further study on new interventions to ensure flap viability beyond the initial surgical recovery is needed. Studies also are needed on flap healing and tissue remodeling. The panel suggests that, at a minimum, blood flow data should be collected and transepidermal water loss and the quality of scar tissue (deformability and elasticity) be measured.

### New Pressure Ulcer Concepts

The panel discussed areas where pressure ulcer information is emerging, lacking, and/or requires additional study.

**Biofilms and pressure ulcers.** Biofilms, a recent discovery in wound healing, are structured communities of microorganisms existing in a self-developed polymeric matrix. James et al\(^{16}\) cultured acute and chronic wounds (n = 93 subjects), including pressure ulcers, and examined the wound tissue using molecular analysis. Microscopic analysis of chronic wound specimens revealed the presence of densely aggregated colonies of bacteria often surrounded by an extracellular matrix. These morphological observations are characteristics of biofilms, and provide evidence that biofilms may be present in chronic wounds. Similar biofilms were not observed in acute wound specimens. **In vivo** studies have shown delayed wound healing when biofilm is present.\(^{19,20}\) It is hypothesized that the biofilm adheres to the open wound bed and may stimulate inflammation.\(^{18,21}\) Because of their protective covering, biofilms often resist antibiotics or white blood cells. The panel agreed that biofilms can form in Stage III or Stage IV pressure ulcers and supported the idea that the only way to control biofilm is to remove it; methods of removal are yet to be defined.\(^{21}\)

**Pressure ulcers on mucous membrane.** The panel described experiences of seeing pressure ulcers on mucous membrane from medical devices secured at the orifice. The mucous membrane lines all body passages that “communicate” with the air (eg, the respiratory, gastrointestinal [GI], and genitourinary [GU] tracts) and contains cells analogous to skin layers and associated mucus-secreting glands. Mucous membranes keep the surface moist; they work like sebaceous (sweat) glands in that they have tubes that connect to the surface and secrete a substance — in this case, mucus. The composition of mucous membranes changes the staging and treatment of pressure ulcers. Ulcers on mucous membranes are often gray or white in appearance.\(^{22}\) According to a position statement developed by NPUAP, the panel agreed that pressure ulcers on mucous membrane should not be staged but rather described as mucosal pressure ulcers with location and size documented.\(^{22}\)

### Research Needed

The panel supported the need for research in the areas of
wound healing and pressure ulcers. Specific needs include:

- Which methods should be used for tissue identification training?
- Are the definitions/descriptions provided here a basis for building on reliability/validity data regarding wound assessment?
- Is the revised NPUAP-EPUAP staging system for pressure ulcers valid and reliable?
- Are the Wound Healing Society definitions of wound healing still valid?
- What are the best practices for flap recovery and healing?
- What are the implications for practice concerning flap recovery and healing?

Nurses in all settings must be able to recognize pressure ulcer deterioration and notify the medical staff of the need to modify the treatment plan. Since the CMS rule was implemented in October 2008, all patients must be examined for the presence of pressure ulcers upon admission to acute care; therefore, all nurses, house officers, and hospitalists need to be able to correctly stage pressure ulcers. In long-term care, nurses examine the ulcers to determine healing (or not) and whether changes are necessary in the care plan; likewise, nurses in home care examine the ulcer, assess healing, and teach the family and patient to continue ulcer monitoring. Goals for wound healing in hospice may differ from other settings; the priority for nurses in this setting is to identify palliative care (ie, pain and odor management) rather than healing.

The Need for Good Communication/Common Language

Pressure ulcers are a complex and chronic healthcare problem, followed for weeks, months, or years by many providers in many settings. The panel agreed that having a common and specific language facilitates accurate communication in order to ensure a safe and effective treatment plan. Pressure ulcers rarely can be managed to complete closure with only one form of topical treatment; it is not uncommon for a single ulcer to have many types of dressings as the wound deteriorates or improves. The ulcer may also need serial maintenance debridement and more than one support surface for a bed or wheelchair over the trajectory of ulcer healing. Care decisions to make the needed changes must come from a clear and accurate assessment of the patient and ulcer and effective communication on the patient’s condition.

For example, if a pressure ulcer develops during an acute care stay, the chart may describe a “pressure ulcer on the buttocks.” From this description, ulcer location, size, and what treatment might be most appropriate are not clear. When this hypothetical patient moves into long-term care, the ulcer is deemed a Stage III pressure ulcer on the sacrum and its size is recorded along with the presence of drainage, odor, and tissue type. During the patient’s stay in long-term care, the ulcer shows signs of wound healing and it is back-staged to Stage II in the medical record in order to be in compliance with their documentation standards. Once again, the patient moves, this time to home care. Believing the ulcer is a Stage II, a care process is established with a goal of healing. The full-thickness pressure ulcer cannot heal in the conventional manner; it closes over time but leaves a scar. This scarred area will remain at risk for the rest of this patient’s life. Miscommunication involving this “Stage II” pressure ulcer did not reveal the need for seating cushions and the like to prevent recurrence. Thus, identifying tissues within a pressure ulcer is an essential first step in staging the pressure ulcer and labeling the wound as healing or not healing.

The descriptors included in these recommendations are offered as a mechanism to help clinicians identify anatomical markers and/or structures within the wound and subsequently accurately identify tissue types and stage pressure ulcers. The panels’ intention was to describe pressure ulcers only and the information should not be extrapolated to other types of wounds or ulcers; also, it is not intended to provide treatment recommendations.

Conclusion

A panel of wound care experts met to discuss and provide an overview of tissue types and other considerations relevant to assessing and subsequently managing pressure ulcers, as well as the appropriate terminology for healing versus closure. Because pressure ulcers are managed across a variety of healthcare settings by caregivers with all levels of training, agreement on tissue terminology, assessment, and fluid/consistent documentation is an important first step toward optimal care. Continued validation of the definitions and descriptions contained herein will enhance knowledge and inform practice.

References


