Chapter 5
General Principles of Pressure Ulcer Management

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Abstract Despite considerable research in management of pressure ulcers over the last decades, substantial issues remain unresolved. Pressure reduction is thought to be critical in healing of pressure ulcer. With the possible exception of air-fluidized beds, one type of pressure-reducing device has not been shown to be superior to another. Nutritional support is a cornerstone of clinical care and should be optimized in all persons, including persons with pressure ulcers, consistent with medical goals and patient wishes. Revisions in the staging system for pressure ulcers have resulted in more precision for clinical description and may help guide choice of therapy. However, new categories of staging may be more difficult for non-wound care specialists. Diagnosing clinical infection in pressure ulcers remains problematic and rests on careful clinical observation. The Pressure Ulcer Scale for Healing tool adequately assesses pressure ulcer status and has proved sensitive to change over time.

Keywords Pressure ulcer • Chronic wound • Pressure reduction • Pressure ulcer nutrition • Wound assessment • Pressure ulcer staging • PUSH tool • Wound infection

Pressure ulcers can be considered rare, affecting only about 0.5 % of the total population. In acute healthcare settings, the prevalence and incidence of pressure ulcers have changed little over the past 2 decades [1]. In a state-wide survey in acute hospital settings, the incidence of pressure ulcers varied from 7.0 to 8.3 per 100,000 population, but did not change from 1987 to 2000 [2]. In a voluntary convenience sample among acute hospital patients, the pressure ulcer incidence remained stable from 1999 to 2004 (8 % versus 7 %, respectively) [3]. In a hospital setting, the point

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prevalence of pressure ulcers was 24 % in 2002 and 23 % in 2006, despite shorter lengths of stay in 2006. When Grade 1 pressure ulcers were excluded, the prevalence rates increased from 8 % in 2002 to 12 % in 2006. This increase in pressure ulcers occurred despite an increase in interventions including turning and repositioning and the use of a pressure-reducing device (25 % in 2002 versus 41 % in 2006) [4]. The distribution of pressure ulcers clusters in two groups, peaking first in younger, mostly neurologically impaired persons, and again in older persons. The cluster in the geriatric population accounts for about 70 % of all pressure ulcers [5].

Acute wounds proceed to healing through a well-researched sequential progression. Pressure ulcers, like other chronic wounds (diabetic ulcers, venous stasis ulcer, and arterial ulcers), fail to proceed through an orderly and timely process to produce anatomical or functional integrity.

Normally, fibroblasts and epithelial cells grow rapidly in skin tissue cultures, covering 80 % of in vitro surfaces within the first 3 days. In contrast, biopsy specimens from pressure ulcers usually do not grow until much later, covering only 70 % of surfaces by 14 days [6]. The result is slow clinical healing.

In a home care setting, about 75 % of stage 2 pressure ulcers healed in 8 weeks, but only 17 % of stage 3 or 4 pressure ulcers healed in that time [7]. In a nursing home setting, 23 % of stage 2 pressure ulcers remained unhealed at 1 year, and 48 % of stage 4 pressure ulcers are unhealed at 1 year. At 2 years, 8 % of stage 2 pressure ulcers, 29 % of stage 3 pressure ulcers, and 38 % of stage 4 pressure ulcers remained unhealed [8]. In 19,981 long-term care residents with pressure ulcers, 45 % of stage 2 ulcers healed and 31 % of stage 4 ulcers healed over 6-month follow-up period [9]. The median days to healing for stage 3 or stage 4 pressure ulcers in a nursing home setting was 140 days [10]. The considerable length of time to healing increases the morbidity and cost of treating pressure ulcers and is often frustrating to the patient and caregivers.

Pressure ulcers occur in persons with multiple comorbidities. The chief among these is immobility. In hospital settings, bedfast immobility increased the risk of developing a pressure ulcers by 23-fold, outweighing all other variables [11].

The risk of developing a pressure ulcer in a hospital setting is highest in patients with a hip fracture and among patients in the intensive care unit. Fifteen percent of patients undergoing hip surgery developed a pressure ulcer during their acute hospital stay. By 32 days after initial hospital admission, the cumulative incidence was 36 % [12].

In the intensive care setting (ICU), pressure ulcer incidence ranged from 4 to 12 % [13]. Subjects who developed a pressure ulcer had a higher severity of illness, including sepsis, hemodynamic instability, mechanical ventilation, use of vasopressors, sedation, use of parenteral nutrition, and requiring insulin therapy for diabetes. Risk factors included higher body temperature, tachycardia or bradycardia, hyperkalemia, acidosis, elevated creatinine, elevated glucose, and a higher C-reactive protein. However, subjects who developed a pressure ulcer did not differ in age, body mass index, or treatment days in the hospital before ICU treatment [14].

Patients undergoing surgery frequently acquire intraoperative pressure ulcers, with a range from as low as 12 % to as high as 66 % [15, 16]. Reduced hemoglobin,

high creatinine, altered level of consciousness, frequency of repositioning, and number of vasoactive infusions are significantly associated with pressure ulcer development at different time points in the first 3 days after a surgical procedure [17, 18]. Cardiovascular surgery is associated with a particularly high incidence of pressure ulcers, ranging from 5 % [19] to 30 %, though to be due to longer intraoperative duration and hemodynamic instability [20, 21]. Finally, pressure ulcers also occur frequently at the end of life.

General Principles of Pressure Ulcer Management

General treatment of pressure ulcers can be divided into broad categories: improving the general condition of the patient, pressure reduction and repositioning, general nutritional support, assessing the wound, and measuring progress toward healing.

Managing Patient Comorbidities

Comorbid conditions, especially those resulting in immobility or paralysis, or reduced tissue perfusion, such as hypoxia due to respiratory or cardiac disease, greatly increase the risk of developing pressure ulcers. In theory, persons who are at high risk for developing pressure ulcers can be identified and increased effort can be directed to preventing ulcers in these persons.

Considerable effort has been directed toward risk assessment. The classical risk assessment scale is the Norton Score. Patients are classified using five risk factors graded from one to four. Scores range from 5 to 20, with higher scores indicating lower risk. In the initial study, 48 % of patients who scored less than 12 developed pressure ulcers, compared with only 5 % of those who scored above 18. The generally accepted at-risk score is 14 or less and patients with scores below 12 are at particularly high risk.

The Braden Scale is commonly used for risk assessment instrument in the USA. This instrument assesses six items, including sensory perception, moisture exposure, physical activity, mobility, nutrition, and friction/shear force. Each item is ranked from one (least favorable) to three or four (most favorable) for a maximum total score of 23. The choice of a cutpoint affects the prediction score. Generally accepted scores of 18–16 or less indicate a high risk.

Risk assessment logically directs attention to the highest risk patients in hospital settings. However, a systematic review found no decrease in pressure ulcer incidence that could be attributed to the use of an assessment scale, despite an increase in intensity of interventions [22].

Careful management of patient comorbidities is a goal of medical therapy, but whether modification of extrinsic risk factors can improve healing of pressure ulcers has not been demonstrated.

Reduce Pressure, Friction, and Shear

The most frequently recommended intervention for prevention and treatment of pressure ulcers is frequent repositioning by physically turning the patient. The exact frequency of turning and positioning has not been studied and recommendations vary by country. In the USA, an arbitrary 2 h is recommended. Whether this is effective is controversial. In prevention trials, patients who were turned and positioned every 2 h failed to show a decrease in the incidence of pressure ulcers [23, 24]. No randomized, controlled trials have been done evaluating turning and repositioning for the treatment of pressure ulcers.

A number of physical devices have been developed to reduce pressure, friction, and shear forces. The aim is to reduce surface interface pressure to below capillary closing pressure of 32 mm/Hg. These devices are classified into three groups, chiefly for reimbursement purposes. Group 1 devices include support surfaces designed as a replacement for a standard hospital or home mattress or as an overlay placed on top of a standard mattress. Products in this category include replacement mattresses, pressure pads, and mattress overlays (foam, air, water, or gel). Group 2 support surfaces are similar replacement mattresses or overlay products. Products in this category include powered air flotation beds, powered pressure-reducing air mattresses, and non-powered advanced pressure-reducing mattresses. Group 3 support surfaces are complete bed systems which use the circulation of filtered air through silicone beads, commonly known as air-fluidized beds.

While there is clear evidence that pressure reduction leads to a decrease in pressure ulcer incidence, only about 22 controlled trials have examined the effect of pressure reduction on the healing of pressure ulcers.

Five studies which compared air-fluidized beds with other surfaces all reported better healing in terms of reduction in pressure ulcer size or stage with the use of air-fluidized beds.

In four trials comparing different brands and types of alternating pressure beds, there was no evidence of differences in healing or reduction in ulcer size among the various beds. The evidence in three trials comparing alternating pressure beds with other types of beds was also inconclusive. No evidence of differences in outcomes with low-air-loss beds compared with foam surfaces was observed in three studies, or in a single study that with low-air-loss beds compared with low-air-loss overlays. A meta-analysis directly comparing different devices for improved healing has not shown a difference among devices [25].

The conceptual model of pressure ulcers suggests that relieving tissue interface pressure should aid in healing. However, no study has demonstrated that one type of device is superior to another, with the possible exception of air-fluidized beds. The choice of a pressure-reducing device should be based on patient comfort, ease of use, durability, and cost. Reimbursement for air-fluidized beds is limited by third-party payors.

Development of ischial ulcers due to confinement in a wheelchair is common. Two small trials have evaluated alternating pressure cushions used in wheelchairs with other types of cushions. One study randomized 44 community-dwelling

wheelchair users with spinal cord injuries who had stage 2 or 3 pressure ulcers to either an alternating pressure cushion or a standard foam cushion. At 30-day follow-up, better rates of healing measured as reduction in wound area, days to 30 % wound closure, and probability of wound closure within 30 days were observed in the group using the alternating pressure cushion [26]. The second study of 25 hospital or nursing home residents compared an alternating pressure cushion to a dry floatation wheelchair cushion. There was no difference in pressure ulcer rate of healing between groups [27].

Address Nutritional Status

Caloric requirements in persons with pressure ulcers suggest that 30 kcal/kg/day is a reasonable target. This clinical estimate is derived from the premise that persons under stress may require higher energy intake. Most clinical observations using nutrition prediction formulas, adjusted for stress, confirm this estimate.

An optimum dietary protein intake in patients with pressure ulcers is unknown, but may be higher than current adult recommendations of 0.8 g/kg/day. Half of the chronically ill elderly persons are unable to maintain nitrogen balance at this level [28]. Increasing protein intake beyond 1.5 g/kg/day may not increase protein synthesis and may cause dehydration [29]. A reasonable protein requirement is therefore between 1.2 and 1.5 g/kg/day. Specific amino acids such as arginine and branched-chain amino acids have not demonstrated an effect on pressure ulcer healing [30].

Nutritional interventions for the healing of pressure ulcers rest on the theory that undernourished patients do not ingest sufficient energy, proteins, vitamins, or minerals to provide for adequate wound healing. However, the results of nutritional interventions in pressure ulcer treatment have been uniformly disappointing [30]. Reaching a target of 30 kcal/kg per day, or increasing protein intake to 1.5 g/kg/day, did not seem to produce any significant effect on wound healing in a nutritional intervention trial [31].

Nutritional supplementation may provide benefit in terms of weight gain. However, the effects of nutritional supplementation are not dramatic, and it is not clear whether nutritional supplementation is beneficial to all patients or only to those with evidence of nutritional deficiencies [32]. The chapter on nutritional intervention in pressure ulcers describes the evidence in greater detail.

Nutritional interventions for treatment of pressure ulcer fall broadly into three categories. These include mixed nutritional supplementation consisting of hypercaloric formulas and vitamins with or without protein supplementation, protein or amino acid supplementation with or without additional calories and vitamin supplementation, and specific nutrient supplementation with vitamins or minerals such as ascorbic acid (vitamin C) or zinc.

The deficiency of several vitamins has significant effects on wound healing. However, supplementation of vitamins to accelerate wound healing is controversial. High doses of vitamin C have not been shown to accelerate wound healing [33]. In a 12-week study of 88 patients who received either 10 or 500 mg of ascorbic acid

twice daily, the healing rates and the healing velocity of their pressure ulcers were not different in the higher dosed group [34].

Zinc supplementation has not been shown to accelerate healing except in zinc-deficient patients [35]. High serum zinc levels interfere with healing and supplementation above 150 mg/day may interfere with copper metabolism [36].

Failure to ingest adequate energy or protein is common in the population who develop pressure ulcers. The causes include general debility, comorbid conditions, or the anorexia/cachexia syndrome. Addressing nutritional intake is important in all persons. There does not appear to be a specific regimen that improves healing of pressure ulcers.

In persons who are unable or unwilling to meet protein-energy requirements orally, enteral tube feeding is often recommended. In a study of enteral tube feedings in long-term care, 49 patients were followed for 3 months [37]. Patients received 1.6 times basal energy expenditure daily, 1.4 g of protein per kilogram per day, and 85 % or more of their total recommended daily allowance. At the end of 3 months, there was no difference in number or healing of pressure ulcers.

In an observational study of nursing home residents referred to the hospital for a percutaneous endoscopic gastroscopy (PEG), persons who did not have a pressure ulcer at the time of PEG insertion ($n = 1,124$) were 2.3 times more likely to develop a new pressure ulcer (95 % CI, 2.0–2.7). In those subjects who had a pressure ulcer at the time of PEG insertion ($n = 452$), the ulcer was 30 % less likely to heal (odds ratio 0.70, 95 % CI, 0.6–0.9) [38]. There are several possibilities for this unexpected observation, but the data suggest that incidence or healing of pressure ulcers is independent of enteral tube feeding and that the effectiveness of enteral feeding in pressure ulcers is not established. Whether this is due to poor effect of feeding or adverse selection of sicker patients is not clear.

Assessing the Ulcer

Several differing scales have been proposed for assessing the severity of pressure ulcers by clinical staging. The most common staging system, recommended by the National Pressure Ulcer Task Force and nursing home guidelines, derives from a modification of the Shea Scale [39]. Under this schematic, pressure ulcers are divided into four clinical stages. The staging system for pressure ulcers relies solely on a description of the depth of the wound. The area of the wound and other wound characteristics are not considered in the clinical staging system.

The staging system classifies pressure ulcers by the visible layers of damaged tissue from the surface toward the bone. This often leads to the false assumption that there is an orderly progression of an ulcer from stage 1 to stage 4. However, it is clear that pressure ulcers do not always progress in the top-to-bottom manner. Current research clearly demonstrates that a bottom-to-top pathogenesis is commonplace. This evolutionary process in the understanding of tissue injury has led to the expansion of the classification system into six stages in the USA (see Table 5.1).

Table 5.1 Clinical staging of pressure ulcers

	NPUAP	EPUAP
Stage/category I	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	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. The area may be painful, firm, soft, warmer, or cooler as compared to adjacent tissue. Category I may be difficult to detect in individuals with dark skin tones. May indicate "at-risk" persons
Stage/category II	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	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 or sero-sanginous-filled blister. Presents as a shiny or dry shallow ulcer without slough or bruising. This category should not be used to describe skin tears, tape burns, incontinence-associated dermatitis, maceration, or excoriation
Stage/category III	Full-thickness tissue loss. Subcutaneous fat may be visible but bone, tendon, or muscle is not exposed. Slough may be present but does not obscure the depth of tissue loss. May include undermining and tunneling	Full-thickness tissue loss. Subcutaneous fat may be visible but bone, tendon, or muscle is not exposed. Slough may be present but does not obscure the depth of tissue loss. May include undermining and tunneling. The depth of a Category/Stage III pressure ulcer varies by anatomical location. The bridge of the nose, ear, occiput, and malleolus do not have (adipose) subcutaneous tissue and Category/Stage III ulcers can be shallow. In contrast, areas of significant adiposity can develop extremely deep Category/Stage III pressure ulcers. Bone/tendon is not visible or directly palpable
Stage/category IV	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	Full thickness tissue loss with exposed bone, tendon, or muscle. Slough or eschar may be present. Often includes undermining and tunneling. The depth of a Category/Stage IV pressure ulcer varies by anatomical location. The bridge of the nose, ear, occiput, and malleolus do not have (adipose) subcutaneous tissue and these ulcers can be shallow. Category/Stage IV ulcers can extend into muscle and/or supporting structures (e.g., fascia, tendon, or joint capsule) making osteomyelitis or osteitis likely to occur. Exposed bone/muscle is visible or directly palpable

(continued)

Table 5.1 (continued)

	NPUAP	EPUAP
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	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
Unstageable	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	Full-thickness tissue loss in which actual depth of the ulcer is completely obscured by slough (yellow, tan, gray, green, or brown) and/or eschar (tan, brown, or black) in the wound bed. Until enough slough and/or eschar are removed to expose the base of the wound, the true depth cannot be determined, but it will be either a Category/Stage III or IV. 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

A comparison of the National Pressure Ulcer Advisory Panel (NPUAP) and the European Pressure Ulcer Advisory Panel (EPUAP) clinical staging systems. In the U SA, convention is to use the term "stage" while in Europe the term "category" is preferred. Adapted from European Pressure Ulcer Advisory Panel and National Pressure Ulcer Advisory Panel (2009) Prevention and treatment of pressure ulcers: quick reference guide. Washington DC: National Pressure Ulcer Advisory Panel

This staging system for pressure ulcers has several limitations. The primary difficulty lies in the inability to distinguish progression between stages. Pressure ulcers do not progress absolutely through stage 1 to stage 4, but may appear to develop from "the inside out" as a result of the initial injury. Surface changes in pressure ulcers are often labeled as a stage 1 when in fact there is a deep tissue injury.

Muscle tissue is more highly susceptible to tissue damage than either fat or skin. In many cases, the changes visible at the surface of the tissue are minor compared to the damage occurring at the deeper layers of muscle. This differential tissue susceptibility suggests that a number of factors are involved in the development of pressure ulcers, including the type of pressure load and biochemical changes in the tissue due to reperfusion injury or tissue susceptibility [1].

Healing of a stage 4 pressure ulcer does not progress through stage 3 to stage 1, but rather healing develops by contraction and scar tissue formation. Thus, "reverse

staging," which describes a healing wound as a proceeding to a lower stage, is inaccurate and the staging system cannot be used to describe healing. Since clinical staging reflects only the depth of the wound, ulcers in which the depth cannot be determined because of eschar are defined as "unstageable."

Clinical staging of pressure ulcers has become more complex and accuracy between stages is subject to observer skills. Deep tissue injury accounted for 9 % of all staged ulcers in 2009, a higher number than either stage 3 or stage 4 pressure ulcers [40].

Managing Infection

Colonization of pressure ulcers with bacteria is common and unavoidable. All chronic wounds become colonized, initially with skin organisms, followed in 48 h by gram-negative bacteria. The diagnosis of a wound infection requires two essential criteria, that is, the presence of bacteria in the wound AND evidence that the bacteria is producing tissue damage (usually in the form of an inflammatory response).

The presence of bacteria in a wound can be described in three forms. Wound bacteria can represent contamination (in the wound transiently, not growing), colonization (established in the wound but with no adverse effect), or infection (established in the wound and damaging the tissue and delaying healing) [41].

Greater than 10^5 organisms may persist for months or years in chronic wounds without apparent clinical effect. The presence of microorganisms alone (colonization) does not indicate an infection in pressure ulcers. The diagnosis of infection in chronic wounds must be made on the basis of clinical signs. However, the only two useful signs of clinical infection are advancing cellulitis and increasing pain [42].

A foul odor is often reported as a clinical sign of infection, but this is often misleading if the odor is coming from the wound dressing rather than from the ulcer itself. A foul odor coming from the ulcer usually signifies anaerobic organisms [43].

Noninfected pressure ulcers and venous stasis ulcers routinely grow varying combinations of *Staphylococcus aureus*, coagulase-negative *Staphylococcus* and *Enterococcus* species, gram-negative bacilli such as *Escherichia coli* and *Pseudomonas aeruginosa*, or anaerobic bacteria representing up to 30 % of isolates) [44, 45].

Peptococci, Bacteroides species, or Clostridia are found in over half of worsening or stationary ulcers, but were absent in healing pressure ulcers. Staphylococci and enterococci were frequently isolated from rapidly healing ulcers. In worsening pressure ulcers, *Pseudomonas aeruginosa* and *Providencia* species were found in 88 and 34 % of ulcers, respectively, compared with 0 % of stationary wounds and 7 % of rapidly healing ulcers [46, 47]. On the basis of these findings, *P. aeruginosa* and *Providencia* species should not be regarded as simple colonization.

Occlusive dressings may increase the number of bacteria in a wound (colonization), but very rarely cause a clinical infection. In a systematic review of 36 studies comparing infection rates under occlusive dressings to gauze or impregnated

gauze, infection rates were 2.6 % for occlusive dressings and 7.1 % for non-occlusive gauze [48].

Growth of bacteria from wounds is not synonymous with infection, and treatment based on microbiological results alone is not warranted. It is therefore inappropriate to culture all wounds. Cultures should be taken only from wounds that are clinically suspected to be infected.

No gold standard for infection in chronic wounds exists, making clinical decisions in their management problematic. Clinical criteria of advancing cellulitis, increasing pain not explained by other factors, and delay in progress toward healing seem to indicate a possible wound infection and provide concrete reasons to consider obtaining a culture. The mechanism for obtaining a culture is not certain, but data suggest that sampling by the Levine technique may be the best trade-off. Routine surface swab cultures are likely to be more confusing than helpful [49].

Measuring Progress Toward Healing

A weekly clinical assessment of a pressure ulcer to assess healing is reasonable. Generally recommended measurements include length and width, type of tissue, amount of exudate, and changes in the surrounding skin. No single measure of a wound characteristic has been useful in measuring healing [50].

The Pressure Ulcer Status for Healing (PUSH) tool was developed and validated to measure healing of pressure ulcers. The tool measures three components, including size, exudate amount, and tissue type, to arrive at a numerical score for ulcer status. In clinical development and validation studies, the PUSH tool adequately assesses pressure ulcer status and proved sensitive to change over time [51, 52]. The PUSH tool is shown in Table 5.2.

Summary

Despite considerable research in management of pressure ulcers over the last decades, substantial issues remain unresolved. Careful management of patient comorbidities is a goal of medical therapy, but whether modification of extrinsic risk factors can improve healing of pressure ulcers has not been demonstrated. As with all patients, careful attention to diabetic control, heart failure, and renal insufficiency are goals of therapy.

There is a clear benefit of pressure-reducing devices in prevention of pressure ulcers compared to standard hospital mattresses. It is reasonable to conclude that pressure-reducing devices may improve healing of pressure ulcers, but with the possible exception of air-fluidized beds, one type of device has not been shown to be superior to another.

Table 5.2 PUSH tool version 3.0

Patient name: _____   Patient ID#: _____

Ulcer location: _____   Date: _____

DIRECTIONS: Observe and measure the pressure ulcer. Categorize the ulcer with respect to surface area, exudate, and type of wound tissue. Record a sub-score for each of these ulcer characteristics. Add the sub-scores to obtain the total score. A comparison of total scores measured over time provides an indication of the improvement or deterioration in pressure ulcer healing

	0	1	2	3	4	5	
Length × width	0 cm²	<0.3 cm²	0.3–0.6 cm²	0.7–1.0 cm²	1.1–2.0 cm²	2.1–3.0 cm²	Sub-score
		6	7	8	9	10	
		3.1–4.0 cm²	4.1–8.0 cm²	8.1–12.0 cm²	12.1–24.0 cm²	>24.0 cm²	
Exudate amount	0 None	1 Light	2 Moderate	3 Heavy			Sub-score
Tissue type	0 Closed	1 Epithelial tissue	2 Granulation tissue	3 Slough	4 Necrotic tissue		Sub-score
							Total score

Length × width: Measure the greatest length (head to toe) and the greatest width (side to side) using a centimeter ruler. Multiply these two measurements (length times width) to obtain an estimate of surface area in square centimeters (cm²). Caveat: Do not guess! Always use a centimeter ruler and always use the same method each time the ulcer is measured

Exudate amount: Estimate the amount of exudate (drainage) present after removal of the dressing and before applying any topical agent to the ulcer. Estimate the exudate (drainage) as none, light, moderate, or heavy

Tissue type: This refers to the types of tissue that are present in the wound (ulcer) bed. Score as a "4" if there is any necrotic tissue present. Score as a "3" if there is any amount of slough present and necrotic tissue is absent. Score as a "2" if the wound is clean and contains granulation tissue. A superficial wound that is reepithelializing is scored as a "1." When the wound is closed, score as a "0"

4—*Necrotic tissue (eschar)*: black, brown, or tan tissue that adheres firmly to the wound bed or ulcer edges and may be either firmer or softer than surrounding skin

3—*Slough*: yellow or white tissue that adheres to the ulcer bed in strings or thick clumps, or is mucinous

2—*Granulation tissue*: pink or beefy red tissue with a shiny, moist, granular appearance

1—*Epithelial tissue*: for superficial ulcers, new pink or shiny tissue (skin) that grows in from the edges or as islands on the ulcer surface

0—*Closed/resurfaced*: the wound is completely covered with epithelium (new skin)

PUSH Tool Version 3.0. Adapted from Stotts et al. [52]

Nutritional therapy should be addressed in concert with overall nutritional goals. Clinical estimates for caloric requirements in persons with pressure ulcers suggest that 30 kcal/kg/day and 1.2–1.5 g/kg/day of protein is a reasonable target. Specific nutritional supplements, supertherapeutic doses of vitamin C and zinc, have not been shown to be clearly effective in healing. Percutaneous endoscopic gastrostomy tube feeding has not improved outcome, and paradoxically may increase mortality risk.

Revisions in the staging system for pressure ulcers have resulted in more precision for clinical description and may help guide of choice of therapy. However, new categories of staging may be more difficult for non-wound care specialists.

Diagnosing clinical infection in pressure ulcers remains problematic and rests on careful clinical observation. The decision to treat a pressure ulcer with antibiotics is currently difficult for most clinicians.

Monitoring the progress of a pressure ulcer over time depends on clinical experience. The Pressure Ulcer Scale for Healing tool adequately assesses pressure ulcer status and has proved sensitive to change over time.

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