

Pressure Ulcers: Prevention, Evaluation, and Management

DANIEL BLUESTEIN, MD, MS, *Eastern Virginia Medical School, Norfolk, Virginia*

ASHKAN JAVAHERI, MD, *Stanford University School of Medicine, Stanford, California, and Veterans Affairs Palo Alto Health Care System, Palo Alto, California*

A pressure ulcer is a localized injury to the skin or underlying tissue, usually over a bony prominence, as a result of unrelieved pressure. Predisposing factors are classified as intrinsic (e.g., limited mobility, poor nutrition, comorbidities, aging skin) or extrinsic (e.g., pressure, friction, shear, moisture). Prevention includes identifying at-risk persons and implementing specific prevention measures, such as following a patient repositioning schedule; keeping the head of the bed at the lowest safe elevation to prevent shear; using pressure-reducing surfaces; and assessing nutrition and providing supplementation, if needed. When an ulcer occurs, documentation of each ulcer (i.e., size, location, eschar and granulation tissue, exudate, odor, sinus tracts, undermining, and infection) and appropriate staging (I through IV) are essential to the wound assessment. Treatment involves management of local and distant infections, removal of necrotic tissue, maintenance of a moist environment for wound healing, and possibly surgery. Debridement is indicated when necrotic tissue is present. Urgent sharp debridement should be performed if advancing cellulitis or sepsis occurs. Mechanical, enzymatic, and autolytic debridement methods are nonurgent treatments. Wound cleansing, preferably with normal saline and appropriate dressings, is a mainstay of treatment for clean ulcers and after debridement. Bacterial load can be managed with cleansing. Topical antibiotics should be considered if there is no improvement in healing after 14 days. Systemic antibiotics are used in patients with advancing cellulitis, osteomyelitis, or systemic infection. (*Am Fam Physician*. 2008;78(10):1186-1194, 1195-1196. Copyright © 2008 American Academy of Family Physicians.)

► Patient information:

A handout on preventing bedsores, written by the authors of this article, is provided on page 1195.



The online version of this article includes supplemental content at <http://www.aafp.org/afp>.

Pressure ulcers, also called decubitus ulcers, bedsores, or pressure sores, range in severity from reddening of the skin to severe, deep craters with exposed muscle or bone. Pressure ulcers significantly threaten the well-being of patients with limited mobility. Although 70 percent of ulcers occur in persons older than 65 years,¹ younger patients with neurologic impairment or severe illness are also susceptible. Prevalence rates range from 4.7 to 32.1 percent in hospital settings² and from 8.5 to 22 percent in nursing homes.³

Etiology

Pressure ulcers are caused by unrelieved pressure, applied with great force over a short period (or with less force over a longer period), that disrupts blood supply to the capillary network, impeding blood flow and depriving tissues of oxygen and nutrients. This external pressure must be greater than arterial capillary pressure to lead to inflow impairment and resultant local ischemia and tissue damage. The most common sites for pressure

ulcers are the sacrum, heels, ischial tuberosities, greater trochanters, and lateral malleoli.

Prevention

RISK ASSESSMENT

Risk assessment begins by identifying risk factors and inspecting the skin. Risk factors for pressure ulcers are classified as intrinsic or extrinsic (*Table 1*).⁴ Caregivers should be educated about risk assessment and prevention and should inspect patients often to prevent pressure ulcers or identify them at early stages. Risk assessment scales may further heighten awareness, but have limited predictive ability and no proven effect on pressure ulcer prevention.⁵ The Braden Scale (*Online Figure A*) is the most commonly used tool for predicting pressure ulcer risk⁶ (<http://www.bradenscale.com/bradenscale.htm>).

INTERVENTIONS

Preventive measures should be used in at-risk patients. Pressure reduction to preserve microcirculation is a mainstay of preventive therapy. There is no evidence to determine

SORT: KEY RECOMMENDATIONS FOR PRACTICE

<i>Clinical recommendation</i>	<i>Evidence rating</i>	<i>References</i>
Compared with standard hospital mattresses, pressure-reducing devices decrease the incidence of pressure ulcers.	A	10, 14
There is no evidence to support the routine use of nutritional supplementation (vitamin C, zinc) and a high-protein diet to promote the healing of pressure ulcers.	C	19
Heel ulcers with stable, dry eschar do not need debridement if there is no edema, erythema, fluctuance, or drainage.	C	8, 16
Ulcer wounds should not be cleaned with skin cleansers or antiseptic agents (e.g., povidone-iodine [Betadine], hydrogen peroxide, acetic acid) because they destroy granulation tissue.	B	8, 27, 28

A = consistent, good-quality patient-oriented evidence; B = inconsistent or limited-quality patient-oriented evidence; C = consensus, disease-oriented evidence, usual practice, expert opinion, or case series. For information about the SORT evidence rating system, go to <http://www.aafp.org/afpsort.xml>.

an optimal patient repositioning schedule, and schedules may need to be determined empirically.⁷ According to recommendations from the Agency for Health Care Policy and Research, patients who are bedridden should be repositioned every two hours.⁸ To minimize shear, the head of the bed should not be elevated more than 30 degrees and should be maintained at the lowest degree of elevation needed to prevent other medical complications, such as aspiration and worsening congestive heart failure symptoms.⁷ Some patients can reduce pressure by repositioning themselves using manual aids, such as a trapeze bar.

Pressure-reducing devices can reduce pressure or relieve pressure (i.e., lower tissue pressure to less than the capillary closing pressure of 32 mm Hg) and are classified

as static (stationary) or dynamic.⁹ Static devices include foam, water, gel, and air mattresses or mattress overlays. Dynamic devices, such as alternating pressure devices and low-air-loss and air-fluidized surfaces, use a power source to redistribute localized pressure. Dynamic devices are generally noisy and more expensive than static devices. Pressure-reducing surfaces lower ulcer incidence by 60 percent compared with standard hospital mattresses, although there is no clear difference among pressure-reducing devices.^{10,11} The benefit of dynamic versus static surfaces is unclear. Dynamic surfaces should be considered if a patient cannot reposition him- or herself independently or if the patient has a poorly healing ulcer.⁷ If there is less than 1 inch of material between the

Table 1. Risk Factors for Pressure Ulcers

Intrinsic	Intrinsic (continued)	Intrinsic (continued)
Limited mobility	Comorbidities	Aging skin
Spinal cord injury	Diabetes mellitus	Loss of elasticity
Cerebrovascular accident	Depression or psychosis	Decreased cutaneous blood flow
Progressive neurologic disorders (Parkinson disease, Alzheimer disease, multiple sclerosis)	Vasculitis or other collagen vascular disorders	Changes in dermal pH
Pain	Peripheral vascular disease	Flattening of rete ridges
Fractures	Decreased pain sensation	Loss of subcutaneous fat
Postsurgical procedures	Immunodeficiency or use of corticosteroid therapy	Decreased dermal-epidermal blood flow
Coma or sedation	Congestive heart failure	Extrinsic
Arthropathies	Malignancies	Pressure from any hard surface (e.g., bed, wheelchair, stretcher)
Poor nutrition	End-stage renal disease	Friction from patient's inability to move well in bed
Anorexia	Chronic obstructive pulmonary disease	Shear from involuntary muscle movements
Dehydration	Dementia	Moisture
Poor dentition		Bowel or bladder incontinence
Dietary restriction		Excessive perspiration
Weak sense of smell or taste		Wound drainage
Poverty or lack of access to food		

Adapted with permission from Garcia AD, Thomas DR. Assessment and management of chronic pressure ulcers in the elderly. Med Clin North Am. 2006;90(5):928-929.

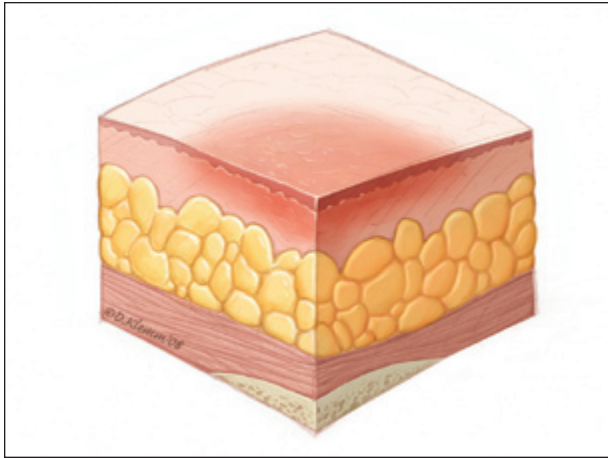


Figure 1. Stage I pressure ulcer. Intact skin with non-blanching redness.

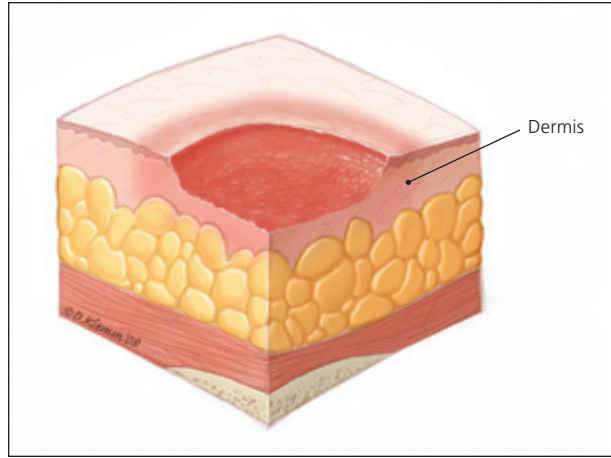


Figure 2. Stage II pressure ulcer. Shallow, open ulcer with red-pink wound bed.

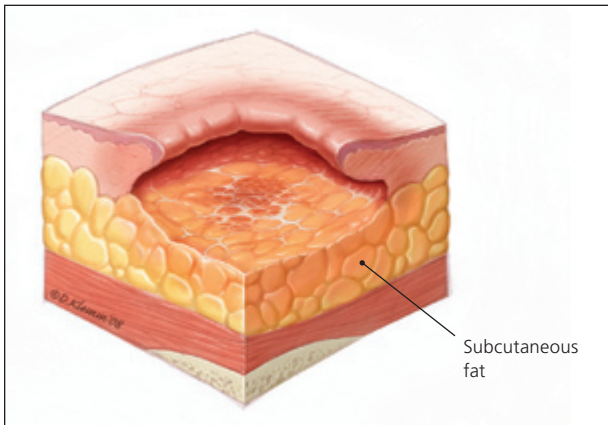


Figure 3. Stage III pressure ulcer. Full-thickness tissue loss with visible subcutaneous fat.

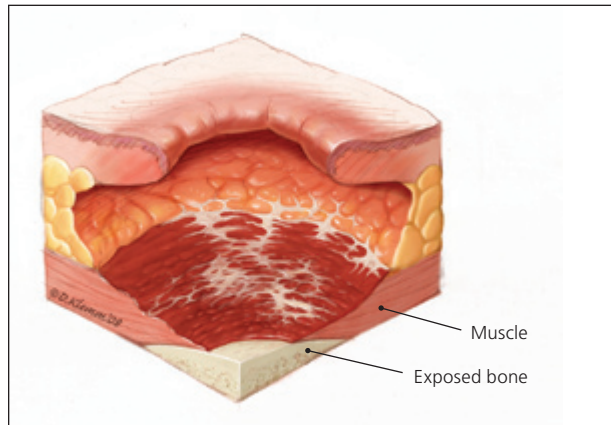


Figure 4. Stage IV pressure ulcer. Full-thickness tissue loss with exposed muscle and bone.

bed and pressure ulcer when feeling beneath the static surface, the device may not be effective and an alternative should be considered.⁷ Other pressure-reducing devices include chair cushions and pillows, foam wedges, and materials that are placed between the knees or used to relieve heel pressure. Ring cushions can cause pressure points and should not be used.

Other preventive interventions include nutritional and skin care assessments. Although poor nutrition is associated with pressure ulcers, a causal relationship has not been established.¹² One large trial has shown that oral nutritional supplementation reduces risk, but several other trials have not.¹³ A Cochrane review concluded that there is insufficient evidence on the relationship between nutrition and pressure ulcer prevention.¹⁴ A more recent meta-analysis concluded that dietitian consultation and the use of skin moisturizers are reasonable preventive measures.¹¹ However, the role of bactericidal and growth factor preparations is unclear. Continence care programs have not proved successful.¹⁵ Despite proper risk assessment and preventive interventions, some pressure ulcers are unavoidable.

Assessment

Assessment of an established pressure ulcer involves a complete medical evaluation of the patient. A comprehensive history includes the onset and duration of ulcers, previous wound care, risk factors, and a list of health problems and medications. Other factors such as psychological health, behavioral and cognitive status, social and financial resources, and access to caregivers are critical in the initial assessment and may influence treatment plans. The presence of a pressure ulcer may indicate that the patient does not have access to adequate services or support. The patient may need more intensive support services, or caregivers may need more training, respite, or assistance with lifting and turning the patient. Patients with communication or sensory disorders are particularly vulnerable to pressure ulcers because they may not feel discomfort or may express discomfort in atypical ways.

The physician should note the number, location, and size (length, width, and depth) of ulcers and assess for the presence of exudate, odor, sinus tracts, necrosis or eschar formation, tunneling, undermining, infection, healing (granulation and epithelialization), and wound

Table 2. NPUAP Staging System for Pressure Ulcers

Stage	Description
Suspected deep-tissue injury	Purple or maroon localized area of discolored, intact skin or blood-filled blister caused by damage to underlying soft tissue from pressure or shear; the discoloration may be preceded by tissue that is painful, firm, mushy, boggy, or warmer or cooler compared with adjacent tissue
I	Intact skin with nonblanchable redness of a localized area, usually over a bony prominence; dark pigmented skin may not have visible blanching, and the affected area may differ from the surrounding area; the affected tissue may be painful, firm, soft, or warmer or cooler compared with adjacent tissue
II	Partial-thickness loss of dermis appearing as a shallow, open ulcer with a red-pink wound bed, without slough; may also appear as an intact or open/ruptured serum-filled blister; this stage should not be used to describe skin tears, tape burns, perineal dermatitis, macerations, or excoriations
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*
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 includes undermining and tunneling*
Unstageable	Full-thickness tissue loss with the base of the ulcer covered by slough (yellow, tan, gray, green, or brown) or eschar (tan, brown, or black) in the wound bed

NPUAP = National Pressure Ulcer Advisory Panel.

*—The depth of a stage III or IV pressure ulcer varies by anatomic location. Because the bridge of the nose, ear, occiput, and malleolus do not have subcutaneous tissue, ulcers on these areas can be shallow. In contrast, areas of significant adiposity can develop extremely deep stage III or IV ulcers. In stage IV ulcers, exposed bone or tendon is visible or directly palpable.

Adapted with permission from the National Pressure Ulcer Advisory Panel. Updated staging system. <http://www.npuap.org/pr2.htm>. Accessed December 17, 2007.

margins. Most importantly, the physician should determine the stage of each ulcer (*Figures 1 through 4*).

Table 2 presents the National Pressure Ulcer Advisory Panel's staging system for pressure ulcers.¹⁶ In a person with dark skin pigmentation, a stage I ulcer may appear as a persistent red, blue, or purple discoloration. The stage of an ulcer cannot be determined until enough slough or eschar is removed to expose the base of the wound. Ulcers do not progress through stages in formation or healing. The Pressure Ulcer Scale for Healing tool (*Figure 5*) can be used to monitor healing progress.¹⁷

Nutritional Evaluation

Despite the consensus that adequate nutrition is important in wound healing, documentation of its effect on ulcer healing is limited; recommendations are based on observational evidence and expert opinion. Nutritional screening is part of the general evaluation of patients with pressure ulcers. *Table 3* presents markers for identifying protein-calorie malnutrition.¹⁸ In patients who are malnourished, dietary consultation is recommended and a swallowing evaluation should be considered. Intervention should include encouraging adequate dietary intake using the patient's favorite foods, mealtime assistance, and snacks throughout the day. High-calorie foods and supplements should be used to prevent malnutrition. If oral dietary intake is inadequate or impractical, enteral or parenteral feeding should be considered, if compatible with the patient's wishes, to achieve positive nitrogen balance (approximately 30 to 35 calories per kg per

day and 1.25 to 1.5 g of protein per kg per day). Protein, vitamin C, and zinc supplements should be considered if intake is insufficient and deficiency is present, although data supporting their effectiveness in accelerating healing have been inconsistent.¹⁹

Management

The management of pressure ulcers is interdisciplinary, including primary care physicians, dermatologists, infectious disease consultants, social workers, psychologists, dietitians, podiatrists, home and wound-care nurses, rehabilitation professionals, and surgeons. The basic components of pressure ulcer management are reducing or relieving pressure on the skin, debriding necrotic tissue, cleansing the wound, managing bacterial load and colonization, and selecting a wound dressing. *Figure 6* is a brief overview of these key components.¹⁸

The pressure-reducing devices used in preventive care also apply to treatment. Static devices are useful in a patient who can change positions independently. A low-air-loss or air-fluidized bed may be necessary for patients with multiple large ulcers or a nonhealing ulcer, after flap surgeries, or when static devices are not effective. No one device is preferred.

Pain assessment should be completed, especially during repositioning, dressing changes, and debridement. Patients at the highest risk of pressure ulcers may not have full sensation or may require alternate pain assessment tools to aid in communication. The goal is to eliminate pain by covering the wound, adjusting pressure-reducing

PUSH Tool

Patient name: _____ Patient identification number: _____

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 subscore for each of these ulcer characteristics. Add the subscores 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.

Length × width*	0	1	2	3	4	5
	0 cm ²	Less than 0.3 cm ²	0.3 to 0.6 cm ²	0.7 to 1.0 cm ²	1.1 to 2.0 cm ²	2.1 to 3.0 cm ²
		6	7	8	9	10
		3.1 to 4.0 cm ²	4.1 to 8.0 cm ²	8.1 to 12.0 cm ²	12.1 to 24.0 cm ²	More than 24.0 cm ²
						Subscore _____
Exudate amount†	0	1	2	3		
	None	Light	Moderate	Heavy		
						Subscore _____
Tissue type‡	0	1	2	3	4	
	Closed (wound completely covered with epithelium [new skin])	Epithelial (superficial ulcers; new pink or shiny tissue that grows in from the edges or as islands on the ulcer surface)	Granulation (pink or beefy-red tissue with a shiny, moist, granular appearance)	Slough (yellow or white tissue that adheres to the ulcer bed in strings or thick clumps or that is mucinous)	Necrotic (eschar; black, brown, or tan tissue that adheres firmly to the wound bed or ulcer edges; may be firmer or softer than surrounding skin)	
						Subscore _____
						Total score _____

*—Measure the greatest length (top to bottom) and the greatest width (side to side) using a centimeter ruler. Multiply these two measures (length × width) to obtain an estimate of surface area in cm². Do not simply guess; always use the same method each time the ulcer is measured.

†—Estimate the amount of exudate (drainage) present after removal of the dressing and before applying a topical agent to the ulcer.

‡—Type of tissue that is present in the wound bed. Score as a 4 if there is any necrotic tissue present, as a 3 if there is any amount of slough present and necrotic tissue is absent, as a 2 if the wound is clean and granulation tissue is present, as a 1 if it is a superficial wound that is reepithelializing, and as a 0 if the wound is closed.

Figure 5. Pressure Ulcer Scale for Healing (PUSH) tool for monitoring the healing of pressure ulcers.

Adapted with permission from Stotts NA, Rodeheaver G, Thomas DR, et al. An instrument to measure healing in pressure ulcers: development and validation of the Pressure Ulcer Scale for Healing (PUSH). *J Gerontol A Biol Sci Med Sci.* 2001;56(12):M795.

surfaces, repositioning the patient, and providing topical or systemic analgesia. Small randomized controlled trials show that topical opioid (diamorphine gel; not available in the United States) and nonopioid (lidocaine/prilocaine [EMLA]) preparations reduce pain during dressing changes and debridement.^{20,21}

Necrotic tissue promotes bacterial growth and impairs wound healing, and it should be debrided until eschar is removed and granulation tissue is present. Debridement, however, is not recommended for heel ulcers that have stable, dry eschar without edema, erythema, fluctuance, or drainage.^{8,16} Debridement methods include sharp, mechanical, enzymatic, and autolytic. Sharp debridement using a sterile scalpel or scissors may be performed at bedside, although more extensive debridement should be performed in the operating room. Sharp debridement

is needed if infection occurs or to remove thick and extensive eschar. Healing after sharp debridement requires adequate vascularization; thus, vascular assessment for lower extremity ulcers is recommended.²² Anticoagulation is a relative contraindication for sharp debridement.

Mechanical debridement includes wet-to-dry dressings, hydrotherapy, wound irrigation, and whirlpool bath debridement.²³ Wet-to-dry dressings adhere to devitalized tissue, which is removed with dressing changes (dry dressings should not be moistened before removal). However, viable tissue may also be removed and the process may be painful.²⁴ Hydrotherapy via whirlpool bath debridement or irrigation may loosen debris. Enzymatic debridement is useful in the long-term care of patients who cannot tolerate sharp debridement; however, it takes longer to be effective and should not be used when infection is present.^{25,26}

Table 3. Markers for Identifying Protein-Calorie Malnutrition in Patients with Pressure Ulcers

- Unintentional weight loss of 5 percent or more in the previous 30 days or of 10 percent or more in the previous 180 days
- Weight less than 80 percent of ideal
- Serum albumin level less than 3.5 g per dL (35 g per L)*
- Prealbumin level less than 15 mg per dL (150 mg per L)*
- Transferrin level less than 200 mg per dL (2 g per L)
- Total lymphocyte count less than 1,500 per mm³ (1.50 × 10⁹ per L)

*—Dehydration can falsely elevate serum albumin and prealbumin. Albumin and prealbumin are negative acute phase reactant and may decrease with inflammation. Physiologic stress, cortisol excess, and hypermetabolic states also reduce serum albumin.

Adapted with permission from Hess CT. Wound Care. 4th ed. Springhouse, Penn.: Springhouse; 2002.

Wounds should be cleansed initially and with each dressing change. Use of a 35-mL syringe and 19-gauge angiocatheter provides a degree of force that is effective yet safe; use of normal saline is preferred. Wound cleansing with antiseptic agents (e.g., povidone-iodine [Betadine], hydrogen peroxide, acetic acid) should be avoided because they destroy granulation tissue.²⁷

Dressings that maintain a moist wound environment facilitate healing and can be used for autolytic debridement.²⁸ Synthetic dressings (Table 4) reduce caregiver time, cause less discomfort, and potentially provide more consistent moisture.¹⁸ These dressings include transparent films, hydrogels, alginates, foams, and hydrocolloids. Transparent films effectively retain moisture, and may be used alone for partial-thickness ulcers or combined with hydrogels or hydrocolloids for full-thickness wounds. Hydrogels can be used for deep wounds with light exudate. Alginates and foams are highly absorbent and are useful for wounds with moderate to heavy exudate. Hydrocolloids retain moisture and are useful for promoting autolytic debridement. Dressing selection is dictated by clinical judgment and wound characteristics; no moist dressing (including saline-moistened gauze) is superior.²⁹ A wet-to-dry dressing should only be used for debridement and is not a substitute for a wound dressing. Because there are numerous dressing options, physicians should be familiar with one or two products in each category or should obtain recommendations from a wound care consultant.

Management of Pressure Ulcers

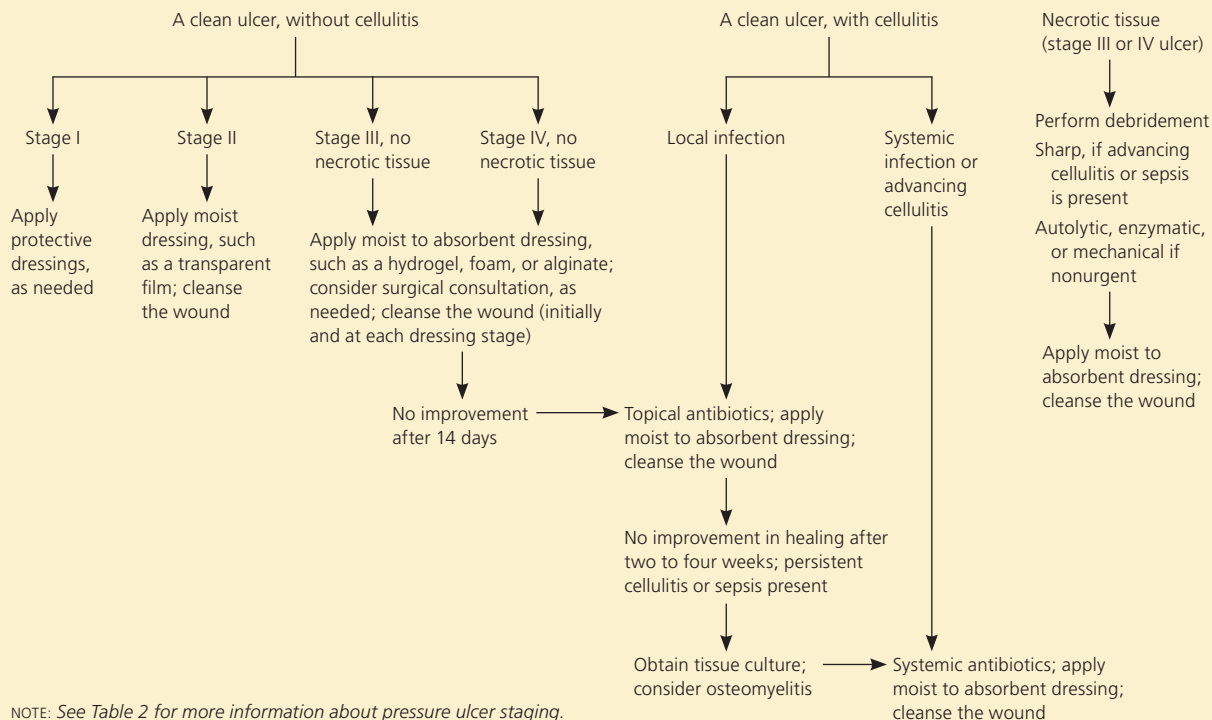


Figure 6. Algorithm for the management of pressure ulcers.

Adapted with permission from Hess CT. Wound Care. 4th ed. Springhouse, Penn.: Springhouse; 2002:54-55.

Table 4. Overview of Different Dressings for Pressure Ulcers

<i>Dressing type</i>	<i>Description</i>	<i>Indication</i>	<i>Advantages</i>
Transparent film	Adhesive, semipermeable, polyurethane membrane that allows water to vaporize and cross the barrier	Management of stage I and II pressure ulcers with light or no exudate May be used with hydrogel or hydrocolloid dressings for full-thickness wounds	Retains moisture Impermeable to bacteria and other contaminants Facilitates autolytic debridement Allows for wound observation Does not require secondary dressing (e.g., tape, wrap)
Hydrogel	Water- or glycerin-based amorphous gels, impregnated gauze, or sheet dressings Amorphous and impregnated gauze fill the dead space tissue and can be used for deep wounds	Management of stages II, III, and IV ulcers; deep wounds; and wounds with necrosis or slough	Soothing, reduces pain Rehydrates wound bed Facilitates autolytic debridement Fills dead tissue space Easy to apply and remove Can be used in infected wounds or to pack deep wounds
Alginate	Derived from brown seaweed; composed of soft, nonwoven fibers shaped into ropes or pads	May be used as primary dressing for stages III and IV ulcers, wounds with moderate to heavy exudate or tunneling, and infected or noninfected wounds	Absorbs up to 20 times its weight Forms a gel within the wound Conforms to the shape of the wound Facilitates autolytic debridement Fills in dead tissue space Easy to apply and remove
Foam	Provides a moist environment and thermal insulation; available as pads, sheets, and pillow dressings	May be used as primary dressing (to provide absorption and insulation) or as secondary dressing (for wounds with packing) for stages II to IV ulcers with variable drainage	Nonadherent, although some have adherent borders Repels contaminants Easy to apply and remove Absorbs light to heavy exudate May be used under compression Recommended for fragile skin
Hydrocolloid	Occlusive or semioclusive dressings composed of materials such as gelatin and pectin; available in various forms (e.g., wafers, pastes, powders)	May be used as primary or secondary dressing for stages II to IV ulcers, wounds with slough and necrosis, or wounds with light to moderate exudate Some may be used for stage I ulcers	Impermeable to bacteria and other contaminants Facilitates autolytic debridement Self-adherent, molds well Allows observation, if transparent May be used under compression products (compression stockings, wraps, Unna boot) May be applied over alginate dressing to control drainage
Moistened gauze	2 × 2- or 4 × 4-inch square of gauze soaked in saline for packing	May be used for stages III and IV ulcers and for deep wounds, especially those with tunneling or undermining	Accessible

Information from reference 18.

Urinary catheters or rectal tubes may be needed to prevent bacterial infection from feces or urine. Pressure ulcers are invariably colonized with bacteria; however, wound cleansing and debridement minimize bacterial load. A trial of topical antibiotics, such as silver sulfadiazine cream (Silvadene), should be used for up to two weeks for clean ulcers that are not healing properly after two to four weeks of optimal wound care. Quantitative bacteria tissue cultures should be performed for nonhealing ulcers after a trial of topical antibiotics or if there are signs of

infection (e.g., increased drainage, odor, surrounding erythema, pain, warmth). A superficial swab specimen may be used; however, a needle aspiration or ulcer biopsy (preferred) is more clinically significant.³⁰ Systemic antibiotics are not recommended unless there is evidence of advancing cellulitis, osteomyelitis, and bacteremia.

Ulcers are difficult to resolve. Although more than 70 percent of stage II ulcers heal after six months of appropriate treatment, only 50 percent of stage III ulcers and 30 percent of stage IV ulcers heal within

<i>Disadvantages</i>	<i>Example (brand names)</i>
Not recommended for infected wounds or wounds with drainage	Bioclusive, Carrafilm, Dermaview, Mefilm,
Requires border of intact skin for adhesion	Opsite, Polyskin, Suresite, 3M Tegaderm, Uniflex
May dislodge in high-friction areas	
Not recommended on fragile skin	
Not recommended for wounds with heavy exudate	Acryderm, Aquaflo, Aquagauze, Carradres, Carraguaze, Carrasmart, Carrasyn, Dermagauze, Dermasyn, Felxigel, SAF-Gel, Solosite, 3M Tegagel, Transigel
Dehydrates easily if not covered	
Difficult to secure (amorphous and impregnated gauze need secondary dressing)	
May cause maceration	
Not recommended with light exudate or dry scarring or for superficial wounds	Algicell, Algisite M, Carboflex, Carraginate, Dermaginate, Kalginate, Kaltostat, Melgisorb, Restore Calcicare, Sorbsan, 3M Tegagen
May dehydrate the wound bed	
Requires secondary dressing	
Not effective for wounds with dry eschar	Allevyn, Biatain, Carrasmart, Curafoam, Dermalevin, Epigard, Hydrocell, Lyofoam, Mepilex, Optifoam, Polyderm, Polymem, SOF-foam, Tielle, Vigifoam
May require a secondary dressing	
Not recommended for wounds with heavy exudate, sinus tracts, or infection	Carrasmart, Combiderm, Comfeel, Dermafilm, Duoderm, Exuderm, Hyperion, MPM Excel, Nuderm, Primacol, RepliCare, Restore, Sorbex, 3M Tegaderm, Ultec
May curl at edges	
May injure fragile skin upon removal	
Contraindicated for wounds with packing	
Must be remoistened often	Fluffed Kerlix, Plain Nugauze
Time-consuming to apply	

this period. Surgical consultation should be obtained for patients with clean stage III or IV ulcers that do not respond to optimal patient care or when quality of life would be improved with rapid wound closure. Surgical approaches include direct closure; skin grafts; and skin, musculocutaneous, and free flaps. However, randomized controlled trials of surgical repair are lacking and recurrence rates are high.

Growth factors (e.g., platelet-derived growth factor becaplermin [Regranex])^{31,32} and vacuum-assisted closure for

recalcitrant stage III and IV ulcers are emerging management options.³³ The role of electromagnetic therapy,³⁴ ultrasound,³⁵ and hyperbaric oxygen therapy is unclear.³⁶

Complications

Although noninfectious complications of pressure ulcers occur, systemic infections are the most prevalent. Noninfectious complications include amyloidosis, heterotopic bone formation, perineal-urethral fistula, pseudoaneurysm, Marjolin ulcer, and systemic complications of topical treatment. Infectious complications include bacteremia and sepsis, cellulitis, endocarditis, meningitis, osteomyelitis, septic arthritis, and sinus tracts or abscesses.⁸ Osteomyelitis has been reported in 17 to 32 percent of infected ulcers and may lead to nonhealing ulcers with or without systemic manifestations.³⁷ Plain radiographs and bone scans are often unreliable. Magnetic resonance imaging has a 98 percent sensitivity and 89 percent specificity for osteomyelitis in patients with pressure ulcers³⁸; however, needle biopsy of the bone (via orthopedic consultation) is recommended and can guide antibiotic therapy. Bacteremia may occur with or without osteomyelitis, causing unexplained fever, tachycardia, hypotension, or altered mental status.³⁹ Overall mortality is high with both conditions,⁴⁰ and empirical antibiotics pending culture results should cover methicillin-resistant *Staphylococcus aureus*, anaerobes, enterococci, and gram-negative organisms, such as *Pseudomonas*, *Proteus*, and *Providencia* species.⁴¹

The authors thank Cathy Flynn, BA; Sherry Allen, LPN, CPS; and Corrine Alvey, RN, BSN, CWOCN, for their assistance in the preparation of the manuscript.

The views expressed in this article are those of the authors and do not necessarily represent the views of the Department of Veterans Affairs.

The Authors

DANIEL BLUESTEIN, MD, MS, CMD, AGSF, is a professor in the Department of Family and Community Medicine at Eastern Virginia Medical School, Norfolk, and is director of the department's Geriatrics Division. He received his medical degree from the University of Massachusetts Medical School, Worcester, and completed a family medicine residency at the University of Maryland School of Medicine, Baltimore. Dr. Bluestein holds a certificate of added qualification in geriatrics and is a fellow of the American Geriatrics Society.

ASHKAN JAVAHERI, MD, is a geriatric medicine fellow at Stanford (Calif.) University School of Medicine. He received his medical degree from Shahid Beheshti University of Medical Sciences, Tehran, Iran, and completed a family and community medicine residency at Eastern Virginia Medical School.

Address correspondence to Daniel Bluestein, MD, MS, CMD, AGSF, Dept. of Family and Community Medicine, Eastern Virginia Medical School, 825 Fairfax Ave., Norfolk, VA 23507 (e-mail: bluestda@evms.edu). Reprints are not available from the authors.

Author disclosure: Nothing to disclose.

REFERENCES

- Whittington K, Patrick M, Roberts JL. A national study of pressure ulcer prevalence and incidence in acute care hospitals. *J Wound Ostomy Continence Nurs.* 2000;27(4):209-215.
- Kaltenthaler E, Whitfield MD, Walters SJ, Akehurst RL, Paisley S. UK, USA and Canada: how do their pressure ulcer prevalence and incidence data compare? *J Wound Care.* 2001;10(1):530-535.
- Coleman EA, Martau JM, Lin MK, Kramer AM. Pressure ulcer prevalence in long-term nursing home residents since the implementation of OBRA '87. Omnibus Budget Reconciliation Act. *J Am Geriatr Soc.* 2002;50(4):728-732.
- Garcia AD, Thomas DR. Assessment and management of chronic pressure ulcers in the elderly. *Med Clin North Am.* 2006;90(5):925-944.
- Schoonhoven L, Haalboom JR, Bousema MT, et al., for the prePURSE Study Group. Prospective cohort study of routine use of risk assessment scales for prediction of pressure ulcers. *BMJ.* 2002;325(7368):797.
- Pancorbo-Hidalgo PL, Garcia-Fernandez FP, Lopez-Medina IM, Alvarez-Nieto C. Risk assessment scales for pressure ulcer prevention: a systematic review. *J Adv Nurs.* 2006;54(1):94-110.
- Whitney J, Phillips L, Aslam R, et al. Guidelines for the treatment of pressure ulcers. *Wound Repair Regen.* 2006;14(6):663-679.
- Agency for Health Care Policy and Research. Treatment of pressure ulcers. Rockville, Md.: U.S. Department of Health and Human Services; 1994. AHCPR Publication No. 95-0652. <http://www.ncbi.nlm.nih.gov/books/bv.fcgi?rid=hstat2.chapter.5124>. Accessed December 17, 2007.
- Thomas DR. Prevention and treatment of pressure ulcers. *J Am Med Dir Assoc.* 2006;7(1):46-59.
- Cullum N, McInnes E, Bell-Syer SE, Legood R. Support surfaces for pressure ulcer prevention. *Cochrane Database Syst Rev.* 2004;(3):CD001735.
- Reddy M, Gill SS, Rochon PA. Preventing pressure ulcers: a systematic review. *JAMA.* 2006;296(8):974-984.
- Thomas DR. Improving outcome of pressure ulcers with nutritional interventions: a review of the evidence. *Nutrition.* 2001;17(2):121-125.
- Bourdel-Marchasson I, Barateau M, Rondeau V, et al. A multi-center trial of the effects of oral nutritional supplementation in critically ill older inpatients. GAGE Group. *Nutrition.* 2000;16(1):1-5.
- Langer G, Schloemer G, Knerr A, Kuss O, Behrens J. Nutritional interventions for preventing and treating pressure ulcers. *Cochrane Database Syst Rev.* 2003;(4):CD003216.
- Bates-Jensen BM, Alessi CA, Al-Samarrai NR, Schnelle JF. The effects of an exercise and incontinence intervention on skin health outcomes in nursing home residents. *J Am Geriatr Soc.* 2003;51(3):348-355.
- National Pressure Ulcer Advisory Panel. Updated staging system. <http://www.npuap.org/pr2.htm>. Accessed December 17, 2007.
- Stotts NA, Rodeheaver G, Thomas DR, et al. An instrument to measure healing in pressure ulcers: development and validation of the Pressure Ulcer Scale for Healing (PUSH). *J Gerontol A Biol Sci Med Sci.* 2001;56(12):M795-M799.
- Hess CT. *Wound Care.* 4th ed. Springhouse, Penn.: Springhouse; 2002.
- Royal College of Nursing. The management of pressure ulcers in primary and secondary care. September 2005. <http://www.nice.org.uk/nicemedia/pdf/CG029fullguideline.pdf>. Accessed December 17, 2007.
- Flock P. Pilot study to determine the effectiveness of diamorphine gel to control pressure ulcer pain. *J Pain Symptom Manage.* 2003;25(6):547-554.
- Rosenthal D, Murphy F, Gottschalk R, Baxter M, Lycka B, Nevin K. Using a topical anaesthetic cream to reduce pain during sharp debridement of chronic leg ulcers. *J Wound Care.* 2001;10(1):503-505.
- Registered Nurses' Association of Ontario. Assessment and management of stage I to IV pressure ulcers. 2007. http://guidelines.gov/summary/summary.aspx?ss=15&doc_id=11013&nbr=5793. Accessed July 1, 2008.
- Singhal A, Reis ED, Kerstein MD. Options for nonsurgical debridement of necrotic wounds. *Adv Skin Wound Care.* 2001;14(2):96-100.
- Ovington LG. Hanging wet-to-dry dressings out to dry. *Home Healthc Nurse.* 2001;19(8):477-483.
- Püllen R, Popp R, Volkers P, Füsgen I. Prospective randomized double-blind study of the wound-debriding effects of collagenase and fibrinolysin/deoxyribonuclease in pressure ulcers. *Age Ageing.* 2002;31(2):126-130.
- Bradley M, Cullum N, Nelson EA, Petticrew M, Sheldon T, Torgerson D. Systematic reviews of wound care management: (2). Dressings and topical agents used in the healing of chronic wounds. *Health Technol Assess.* 1999;3(17 pt 2):1-35.
- Rodeheaver GT. Pressure ulcer debridement and cleansing: a review of current literature. *Ostomy Wound Manage.* 1999;45(1A suppl):805-855.
- Kerstein MD, Gemmen E, van Rijswijk L, et al. Cost and cost effectiveness of venous and pressure ulcer protocols of care. *Dis Manage Health Outcomes.* 2001;9(11):651-663.
- Bouza C, Saz Z, Muñoz A, Amate JM. Efficacy of advanced dressings in the treatment of pressure ulcers: a systematic review. *J Wound Care.* 2005;14(5):193-199.
- Rudensky B, Lipschits M, Isaacsohn M, Sonnenblick M. Infected pressure sores: comparison of methods for bacterial identification. *South Med J.* 1992;85(9):901-903.
- Thomas DR. The promise of topical growth factors in healing pressure ulcers. *Ann Intern Med.* 2003;139(8):694-695.
- Robson MC, Phillips LG, Thomason A, Robson LE, Pierce GF. Platelet-derived growth factor BB for the treatment of chronic pressure ulcers. *Lancet.* 1992;339(8784):23-25.
- Argenta LC, Morykwas MJ. Vacuum-assisted closure: a new method for wound control and treatment: clinical experience. *Ann Plast Surg.* 1997;38(6):563-576.
- Olyaei Manesh A, Flemming K, Cullum NA, Ravaghi H. Electromagnetic therapy for treating pressure ulcers. *Cochrane Database Syst Rev.* 2006;(2):CD002930.
- Baba-Akbari Sari A, Flemming K, Cullum NA, Wollina U. Therapeutic ultrasound for pressure ulcers. *Cochrane Database Syst Rev.* 2006;(3):CD001275.
- Kranke P, Bennett M, Roeckl-Wiedmann I, Debus S. Hyperbaric oxygen therapy for chronic wounds. *Cochrane Database Syst Rev.* 2004;(2):CD004123.
- Darouiche RO, Landon GC, Klima M, Musher DM, Markowski J. Osteomyelitis associated with pressure sores. *Arch Intern Med.* 1994;154(7):753-758.
- Huang AB, Schweitzer ME, Hume E, Batte WG. Osteomyelitis of the pelvis/hips in paralyzed patients: accuracy and clinical utility of MRI. *J Comput Assist Tomogr.* 1998;22(3):437-443.
- Bryan CS, Dew CE, Reynolds KL. Bacteremia associated with decubitus ulcers. *Arch Intern Med.* 1983;143(11):2093-2095.
- Wall BM, Mangold T, Huch KM, Corbett C, Cooke CR. Bacteremia in the chronic spinal cord injury population: risk factors for mortality. *J Spinal Cord Med.* 2003;26(3):248-253.
- Livesley NJ, Chow AW. Infected pressure ulcers in elderly individuals. *Clin Infect Dis.* 2002;35(11):1390-1396.