Pressure Ulcers: Clinical Features and Management

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Pressure ulcers are a common and frustrating problem affecting geriatric patients. Changing demographics in the United States have resulted in an increasingly older elderly population. The frailty, comorbidity, and impaired mobility and cognition characteristic of this population predispose them to the development of such ulcers,[1] ultimately resulting in a significant financial burden to society. In recent years, pressure ulcers have even become a basis for litigation,[2] a development made even more unfortunate by the fact that pressure ulcers are preventable. Given these considerations, it is imperative that health providers be knowledgeable about the condition and its consequences.

EPIDEMIOLOGY

The prevalence of advanced pressure ulcers is high in older persons, peaking in those between age 70 and 80 years. These ulcers occur in hospitals, nursing homes, and community settings, with a prevalence ranging from 1.2% to 11.2%. The highest incidence of new ulcers occurs in hospitals, but the highest prevalence of ulcers occurs in long-term care facilities. Whereas the general incidence of pressure ulcers in hospitals varies, ranging from 1.2% to nearly 3%, it may be as high as 50% in acute care geriatric units and is associated with a high mortality rate.[3,4] Moreover, the morbidity associated with the development of pressure ulcers increases the length of stay and resultant expense of hospitalization, hardly affordable consequences in today's era of health care economics. Specifically, the duration of hospitalization increases up to 5-fold when patients develop pressure ulcers, with the mean hospital cost nearly doubling.[3]

DEFINITION

A pressure ulcer is any lesion caused by unrelieved pressure, usually over a bony prominence, that results in damage to underlying tissue.[5] The terms *pressure ulcer* and *pressure sore* are preferred to the synonymous terms *decubitus ulcers* and *bed sores* because the former terms underscore the importance of unrelieved pressure as a primary risk factor in the pathogenesis of the lesions. In addition, these ulcers can occur in patients who are neither bed-bound nor in decubitus positions, so "bed sores" and "decubitus ulcers" are much less accurate terms. For the purposes of this review, the term *pressure ulcers* will be used.

STAGING

The National Pressure Ulcer Advisory Panel recommends a 4-stage classification of pressure ulcers, based on the extent of tissue involvement (**Table 1**). This classification system is endorsed by the Agency for Health Care Policy and Research.

Stage I ulcers consist of intact skin with nonblanchable erythema resulting from extravasation of blood from ischemic, leaky blood vessels. If erythema is blanchable, the process most likely involves congested vessels and so vanishes shortly after pressure relief. Stage I ulcers are cone-shaped, with the apex toward the skin, suggesting minimal involvement at the surface but providing no indication of the potentially more extensive involvement beneath the surface.[6] These ulcers should not be taken lightly, because the tissues beneath them might have more significant necrosis that can easily progress to more advanced stages of ulceration. In this regard, it is important to remember that muscle is much more susceptible to ischemia than is fat or dermis, because of higher metabolic activity and relatively lesser blood supply.

Stage II ulcers are associated with discontinuity of the skin (eg, an abrasion, a blister, or a shallow crater) and involve the epidermis and/or dermis. Stage III

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ulcers extend deeper into the subcutaneous fascia, and stage IV ulcers are associated with involvement of the muscle, bone, or supporting structures (eg, tendons, ligaments, joint capsules).

There are limitations to this staging system. For example, an eschar-covered ulcer cannot be staged unless the eschar is débrided. In dark-skinned persons, nonblanchable erythema may not be readily visible; instead, increased warmth, induration, or edema over bony prominences may be the only apparent sign. Ulcers beneath orthopaedic devices (eg, casts) may be missed, so vigilance is necessary.

Pressure ulcers do not necessarily progress from stage I to stage IV, nor do they heal in the reverse order. For example, an initial stage IV ulcer that is almost healed is called a healing stage IV ulcer, not a stage I or II ulcer.[7]

Table 1. Staging of Pressure Ulcers*

Stage	Description
I	Nonblanchable erythema of intact skin (in darker skinned persons, discoloration, warmth, edema, induration, or hardness of the skin can also be indicators of a pressure ulcer)
II	Partial-thickness skin loss involving the epidermis, dermis, or both, with the superficial ulcer presenting clinically as an abrasion, blister, or shallow crater
III	Full-thickness skin loss involving damage to or necrosis of subcutaneous tissue that may extend down to, but not through, underlying fascia, with the ulcer presenting clinically as a deep crater (with or without undermining of adjacent tissue)
IV	Full-thickness skin loss with extensive destruction, tissue necrosis, or damage to muscle, bone, or supporting structures (eg, tendon, joint capsule); also potentially associated with undermining and sinus tracts

Adapted from Clinical practice guideline number 15: treatment of pressure ulcers. Rockville (MD): US Department of Health and Human Services, Agency for Health Care Policy and Research; 1994. AHCPR publication 95-0652.

*This staging is consistent with the stages proposed by the National Pressure Ulcer Advisory Panel Consensus Development Conference.

LOCATION

Although pressure ulcers can develop at any site, they occur more frequently over bony prominences, as previously suggested. Patient position and degree of immobility can influence the site of involvement. The most common locations are the sacrum, coccyx, and heels (when persons are in a supine position); the hips and ankles (when persons are lying on their sides); and the buttocks (when persons are seated). Less commonly involved are the earlobes, occiput, chin, elbow, scapula, and knee. The lower half of the body accounts for 95% of pressure ulcers.[6,8]

ASSESSMENT

Comprehensive assessment of an ulcer includes several steps. History taking should determine the duration, prior treatment, and progression of the ulcer. The location and characteristics of each ulcer need to be clearly documented. Ulcer size is typically assessed by either a linear measurement of length and width using paper tapes or a 3-dimensional measurement of length, width, and depth using a wound gauge (eg, a Kundin gauge). Wound depth can also be gauged by using a gloved finger and then measuring the depth of finger insertion; this method is preferable to using cotton swabs or applicators, which can induce trauma.[9]

The color of the ulcer bed may be red (indicating a relatively healthy condition), yellow (consisting of fibrin slough), or black (indicating the presence of necrotic tissue or eschar). It should be determined if the ulcer base contains necrotic, granulation, or epithelial tissue. All necrotic tissue, which is usually moist, yellow, or gray, should be removed. In contrast, granulation tissue is beefy red and bumpy, and epithelial tissue is pale or dark pink, usually occurring at the ulcer borders. The ulcer margins and surrounding skin should be inspected for warmth, induration, and erythema—all signs of infection. The presence of tunneling and of sinus tracts should be documented, and the color, odor, quantity, and nature of the wound exudate should be determined.[9]

Ulcer healing can also be gauged by using the assessment steps described above. All ulcers require periodic reevaluation (at least weekly) by a qualified evaluator. In addition, assessment for pain should be performed on a regular basis (*see* "Treatment" section).

RISK FACTORS

Risk Assessment Scales

Risk factors involved in the pathogenesis of ulcers may be intrinsic, extrinsic, or both (**Table 2**). Risk assessment can be performed using either the Braden or the Norton scale. The Braden scale evaluates 6 factors—level of sensory perception, skin moisture, level of activity, mobility, nutrition, and friction/shear—with a least favorable score of 1 and a more favorable score of 3 or 4. The Norton scale assesses 5 factors—physical condition, mental status, level of activity, mobility, and

Table 2. Risk Factors for Pressure Ulcers

Intrinsic
Aging
Chronic disease (eg, diabetes mellitus, peripheral vascular diseases)
Impaired mobility and limited activity (in cases of contractures, Parkinson's disease, advanced dementia)
Incontinence (fecal more than urinary)
Malnutrition
Sensory impairment (because of neuropathy, cerebrovascular accident)
Extrinsic
Pressure
Friction
Shearing
Moisture

incontinence—with each factor scored from 1 (ie, very bad) to 4 (ie, good). Scores at or below 16 (Braden scale) or 12 (Norton scale) denote a high risk for pressure ulcers.[6]

Intrinsic Factors

Intrinsic factors relate to patient status. Several changes occur in the aging skin, including slower epidermal turnover and decreased vascularity, subcutaneous adiposity, and collagen and elastin content.[10–12] Consequently, the skin becomes susceptible to injury, infection, and delayed wound healing.[1,3,10,11] The inflammatory response is also blunted.

Impaired or restricted mobility is an intrinsic risk factor, as evidenced by the increased risk for pressure ulcers in patients with cerebrovascular disease, Parkinson's disease, advanced dementia, contractures, and (most commonly) orthopaedic injuries.[1,6,12,13] Other diseases that restrict mobility, such as diabetic neuropathy and spinal cord injuries, likewise increase the risk for pressure ulcers.[1] Not surprisingly, spontaneous nocturnal movements during sleep can prevent ulcer development; data indicate that elderly persons with more than 50 nocturnal movements did not develop ulcers, whereas 90% of those with fewer than 20 developed at least a single ulcer.[13]

Other relevant intrinsic factors include incontinence and nutritional status. Although moisture is a risk, urinary incontinence, per se, has not been clearly shown to promote the development of pressure ulcers. However, fecal incontinence appears to increase risk, perhaps by causing skin contamination with bile salts, bacterial toxins, and moisture.[1,6,14] Likewise, malnutrition has also been associated with the development of pressure ulcers.[12,13]

Extrinsic Factors

Pressure, friction, shearing, and moisture are well-recognized extrinsic factors in the pathogenesis of pressure ulcers. Unrelieved pressure appears to be the most necessary requirement for ulcer development. Normal arteriole, capillary, and venule pressures are 32, 20 and 12 mm Hg, respectively.[6] Pressure generated under the ischial tuberosities while a person is seated can reach 300 mm Hg, and sacral pressure can range from 100 to 150 mm Hg while a person lies on a standard hospital mattress.[6,8,12] Excess pressure (ie, above 32 mm Hg) results in occlusion of capillary flow, causing ischemic injury and extravasation of fluid, cells, and protein. Muscle can be damaged by pressures exceeding 60 mm Hg for more than an hour,[8,12,14,15] although the skin is more resistant. These facts provide a rationale for the frequent repositioning of patients and the 2-hour turning schedules in standard protocols (data from animal studies also support these measures).[6,13]

Friction results from the rubbing of a body part against another body part or support surface, causing damage to the stratum corneum. This damage presents clinically as a skin tear or abrasion and occurs commonly when patients are pulled across surfaces during transfers.[1,14] Shearing, on the other hand, results when subcutaneous tissue and skin slide on each other in opposing directions, resulting in angulation of the arterioles and in ischemia. Shearing commonly occurs when the head of the bed is elevated more than 30 degrees, causing the patient to slide down.[13,14,16] Finally, moisture, when excessive, results in maceration and weakening of the skin; causative factors include excessive perspiration, fecal or urinary incontinence, and heavy wound exudate.[1,6]

COMPLICATIONS

Infection is a common complication of pressure ulcers and may be local, in the form of cellulitis and osteomyelitis, or distant, resulting from bacteremia. Although all stage II, III, and IV ulcers invariably are colonized with bacteria, this fact by itself does not indicate infection. The presence of fever, leukocytosis, delirium, and malodorous drainage does, however, suggest infection. Tissue cultures of poorly healing pressure ulcers can indicate the presence of gram-negative bacilli (eg, *Pseudomonas aeruginosa*, *Providencia* species) and anaerobic bacteria (*Bacteroides* and *Clostridium* species—thus

Table 3. A Simplified Approach to the Management of Pressure Ulcers in Elderly Patients

Prevention of Extrinsic Risk Factors	Prevention of Intrinsic Risk Factors	Treatment of Pressure Ulcers
Reposition patient (every 2 h when in bed, every 15 min when seated)	Provide appropriate skin care	Manage risk factors (as outlined in previous 2 columns)
Consider providing patients a trapeze device, if appropriate	Use only lukewarm water and mild soap	Assess pain
Avoid shearing (eg, keep the maximum elevation of the head of the bed < 30 degrees)	Use moisturizers for dry skin	Provide local wound care
Minimize friction	Manage incontinence	Débridement (surgical, mechanical, enzymatic, autolytic)
Consider using heel protectors and seat cushions (eg, foam cushions, pillows)	Use absorbent underpads	Cleansing (using normal saline and an irrigation pressure of 4–15 psi)
Choose appropriate support surfaces (static versus dynamic)	Be attentive to soiling	Dressings (gauze, foam, hydrocolloid, polyurethane, alginate, hydrogel)
	Consider using moisture barriers	Determine whether antibiotic therapy (local versus systemic) is indicated to treat infection
	Institute urinary catheterization, when appropriate	Manage any complications
	Optimize nutritional status	Determine whether surgical closure of the wound or adjuvant therapy is indicated
	Encourage early ambulation and rehabilitation	
	Manage any coexisting chronic disease(s)	

making elderly patients vulnerable to tetanus infection).[17,18] Moreover, even healing ulcers appear to contain isolates of staphylococci and enterococci.[18]

Nonhealing ulcers also are associated with osteomyelitis in up to a quarter of cases.[3] The triad of leukocytosis, elevated sedimentation rate, and abnormalities on plain radiographs appears to correlate with osteomyelitis in two thirds of cases.[13,19] Computed tomography scanning may be useful in the diagnosis of osteomyelitis (specificity 90%, sensitivity 10%), but bone biopsy appears to be the single best diagnostic test (specificity 96%, sensitivity 73%).[6,18] Technetium Tc 99m and gallium 67 bone scans are sensitive for diagnosing osteomyelitis but are associated with a significant number of false-positive findings. Other complications of nonhealing pressure ulcers include secondary amyloidosis, fistula, abscess, malignant change (rare), and maggot infestation.[7]

Bacteremia associated with pressure ulcers has a greater than 50% mortality.[3,13,20] Because swab cultures of pressure ulcers can be misleading, blood cultures, deep tissue biopsy, and needle aspirations should instead be performed if infection is suspected.[8,12] Organisms from tissue culture typically include anaerobes, gram-negative bacilli, and gram-positive cocci, all potentially leading to bacteremia.[12,13] Hence, broad-spectrum antibiotic coverage is required. Finally, bacteremia may result in infective endocarditis, meningitis, or mycotic aneurysms.

MANAGEMENT

Prevention

In 1994, the Agency for Health Care Policy and Research released clinical practice guidelines on the prevention and treatment of pressure ulcers,[7] and the general principles remain valid today. Although management includes prevention and treatment, prevention is clearly the most effective strategy in care of pressure ulcers (**Table 3**). Patients identified as being at high risk by the Braden or Norton Scales should be cared for accordingly.

Minimizing pressure, friction, and shear. The importance of frequent position changes, preferably at least every 2 hours when the patient is in bed, has been discussed earlier. Turning schedules, including shifts to the supine and both oblique positions, can be posted at the bedside to serve as reminders. The 30-degree left and right oblique positions are particularly useful to prevent ulcer development at the most common sites in the back, hips, and ankles.[12,21] Keeping the head of the bed elevated less than 30 degrees (except after meals) will minimize shear. In addition, pillows may be placed between a patient's knees and ankles to minimize pressure and friction or under the calves to elevate the heels. Any benefit gained from heel protectors appears to be from their ability to reduce pressure.[12]

Caution should be exercised to avoid dragging the patient across support surfaces. For example, during

Table 4. Support Surfaces

Static support surfaces*
Chair cushions
Heel protectors
Mattresses or overlays (foam, gel, water, air)
Dynamic support surfaces†
Air-fluidized beds
Alternating air-pressure mattresses
Low–air loss beds

*No moving parts.

†Moving parts run by energy.

transfers, bed linen can be used to lift patients to minimize friction. While seated, patients should be repositioned frequently (at least every 15 minutes) to relieve pressure on the buttocks. Pillows or foam cushions on wheelchairs can help decrease pressure[12,13]; however, the use of doughnut-type cushions has now become obsolete because, contrary to earlier beliefs, they worsen ischemia by decreasing blood flow to the center of the cushion area.[13,19,21] Patients who can reposition themselves should be provided a trapeze device.

Skin care and moisture prevention. The skin of patients at high risk for pressure ulcers must be inspected regularly. Once again, excessive moisture from incontinence or perspiration can cause macerated skin. After a patient has soiled him/herself, affected areas should be cleaned with mild soap, and absorbent underpads or briefs should be used for protection, along with topical moisture barriers. For dry skin, mild soaps and lukewarm water, as well as moisturizing cream, should be used. Massaging the bony prominence may be harmful and is best avoided.[12,13]

Pressure-reducing devices. Although frequent repositioning relieves pressure, support surfaces can decrease pressure over bony prominences. The goal of such devices is to reduce the pressure on the skin to 30 to 35 mm Hg (or lower), if possible.[1] Standard hospital beds are inadequate in this regard. Many devices are available, although none is a substitute for good nursing care.

Support surfaces include overlays (placed on top of standard beds), special mattresses, or customized beds. There are 2 types of support surfaces: static, with no moving parts, and dynamic, with moving parts run by energy (Table 4). Sheepskin and 2-inch foam (flat or egg-crate) mattresses are static and inexpensive ($40 to $50), but they are not effective in preventing pressure ulcers.[12,13] However, a 4-inch foam mattress can reduce pressure much more than can a standard hospital bed and is not expensive.[8,14] Mattresses may be composed of gel, foam, air, or water. Air and water mattresses are effective but may leak, so they need constant maintenance.[16] Foam may be used to pad stretchers, wheelchairs, and chairs. Dynamic support surfaces, frequently indicated in the management of advanced nonhealing or multiple ulcers, will be discussed in the "Treatment" section.

Studies do not conclusively favor a single support surface for all situations. Choice should be individualized and based on support area, moisture retention, heat accumulation, pressure or shear factors, and cost. Moreover, support surfaces do not always prevent pressure ulcers; in fact, in one study, the majority of patients developed pressure ulcers while on some form of support device.[6]

Treatment

Once an ulcer has developed, proper documentation is essential, beginning with the assessment of risk factors and a description of the ulcer. Medical status should be optimized, nutrition corrected, pain assessed, positioning schedules enforced, infections treated, and method of débridement and dressings individualized (Table 3).

Nutrition. An adequate daily diet of at least 30 to 35 cal/kg body weight, including 1.25 to 1.5 g/kg of protein, must be provided. If required, enteral support can be administered, as long as the intestines are functional, with parenteral nutrition used as an alternative. Restrictive diets should be minimized.[21,22] Unfortunately, some recent evaluations have shown that improved nutritional status may not prevent development of pressure ulcers.[23]

Although low albumin levels have been associated with ulcers in many studies, hypoalbuminemia, per se, has not been shown to be a predictor of ulcer development; similarly, anemia, low total lymphocyte counts, and low body weight have also not been associated with ulcer development.[22] Nevertheless, low albumin levels, body weight, and total lymphocyte counts all have negative associations with ulcer healing.[22] Dietary supplementation with vitamin C and zinc has been linked to wound healing; however, such supplementation does not have healing benefits unless there is nutritional deficiency.[6,22] The recommendation is to provide these specific nutrients or to use a high-potency multi–vitamin-mineral combination when either the diet is inadequate or deficiency is suspected.[7]

Pain control. Pain is common in patients with pressure ulcers and should be recognized. Assessment should be performed regularly, using a rating scale (eg,

a visual analog scale, the Wong-Baker FACES Pain Rating Scale). It should be determined whether pain interferes with function. In cognitively impaired patients, pain may be difficult to assess.[3] It is essential that analgesia be considered during dressing changes and particularly during débridement. Occlusive dressings and proper positioning can likewise alleviate pain.[19]

Support surfaces used for treatment. Whereas static support surfaces can be used for prevention (and sometimes for treatment) of pressure ulcers in the elderly, dynamic support surfaces with motorized parts are indicated for treatment in specific situations. Such situations include the presence of multiple ulcers at different sites (typically, stage III or IV), failure of ulcers to heal with static supports, and patients' inability to assume optimum positions for healing.[7]

The primary dynamic support surfaces are alternating air-pressure mattresses, low–air loss beds, or air-fluidized beds (Table 4). These types of surfaces are considered when static support surfaces do not meet their intended goals. In alternating air-pressure mattresses, different air compartments are alternatively inflated and deflated by a bedside pump[13]; these mattresses cost less than the more specialized low–air loss or air-fluidized beds but are nevertheless effective.[16] Low–air loss beds contain numerous air cells that are filled to specific pressures, based on patient weight and habitus, with air escaping through porous fabric; the bed can be deflated to facilitate turning and positioning of the patient, and the head and foot of the bed can be raised or lowered, as can the entire bed. Air-fluidized beds contain microspheric, silicon-coated beads that allow greater air loss than occurs with low–air loss beds, providing a characteristic feel of water; the patient "floats" on the system but should lie flat and be monitored for hydration and fever, because the air is warm.[1] Both the low–air loss and air-fluidized beds lower pressures to approximately 10 mm Hg, but the cost of renting such a bed ranges from $50 to $100 per day.[14] In today's era of cost containment, the high cost of specialized beds has to be weighed against their benefits, so their use should be limited to indicated settings.

Wound débridement. Wound débridement is a vital part of local wound care. The presence of necrotic tissue or an eschar (ie, black, hard, devitalized tissue) delays growth of granulation tissue. Débridement in such cases should be accomplished in 1 of 4 ways: sharp (surgical), mechanical, enzymatic, or autolytic (**Table 5**). Eschars in the heel are an exception. Heel eschars provide a natural protective cover that should not be débrided unless there is evidence of edema, fluctuation, or drainage; if any of these complications exist, débridement is indicated.[7]

Table 5. Forms of Débridement

Sharp or surgical
Scalpel, scissors, or other sharp instruments are used.
This form of débridement is the most rapid method of removing devitalized tissue.
It is particularly useful in treating infected ulcers.
Analgesia is required.
An operating room and anesthesia are required for cases of extensive débridement.
When appropriate, prophylaxis should be used to prevent infective endocarditis.
Mechanical
This form of débridement can be performed using wet-to-dry dressings, whirlpool therapy, and wound irrigation.
It is nonselective, removing nonvitalized but also vitalized tissue.
It is labor intensive.
Analgesia is required.
Enzymatic
Collagenase, fibrinolysin, and proteinases can be used.
This form of débridement spares viable tissue.
Pain is minimal.
This is a slow form of débridement.
It is less effective with bulky necrotic tissues.
It is not suitable for infected ulcers.
Autolytic
Indigenous enzymes in wound fluids digest necrotic tissue.
This form of débridement uses occlusive dressings.
Pain is minimal, if present at all.
This is the slowest form of débridement.
It is not labor intensive.
It is not suitable for infected ulcers.

Surgical or sharp débridement is the fastest method and can be performed at the bedside with scissors and a scalpel or in the operating room. Such débridement is indicated in the presence of advancing cellulitis and extensive necrosis.[6,8,14] Because bacteremia can occur during surgical débridement, use of antibiotic prophylaxis to prevent infective endocarditis may be necessary. Analgesia should be provided.

Mechanical débridement can be performed with wet-to-dry dressings, whirlpool therapy, or wound irrigation. Wet-to-dry dressings entail the use of saline-moistened gauze placed on the ulcer, which is removed

Table 6. Pressure Ulcers: Evaluation and Control of Infection

Evaluation for infection
Clinically assessing the ulcer
Performing appropriate cultures (eg, blood, tissue) and determining the leukocyte count
Obtaining radiographs, computed tomography scans, and radioisotope scans, if osteomyelitis is suspected
Performing bone biopsy and culture, if osteomyelitis is suspected
Determining whether distal sites are affected
Control of infection
Limited local use of antibiotics for nonhealing infected wounds
Infective endocarditis prophylaxis prior to débridement, when appropriate
For cases involving sepsis, broad-spectrum initial antibiotic coverage (eg, ampicillin, sulbactam, imipenem, ticarcillin/clavulanate, piperacillin, tazobactam, clindamycin [with aminoglycoside or ciprofloxacin]) for aerobic gram-negative rods, gram-positive cocci, and anaerobes
Appropriate revisions to drug regimen once culture results are available
Prolonged treatment when indicated (eg, when there are complications such as osteomyelitis or infective endocarditis)

Data from Goode and Allman[12], Allman[13], and Thomas.[18]

once dry. Necrotic tissue comes off with the gauze, but the technique is not selective and healthy tissue may also be removed. This method is also labor intensive and painful. Whirlpool therapy is expensive but effective. Wound irrigation may be adequately performed with 35-mL syringes and 19-gauge needles or angiocatheters for optimum pressure and removal of devitalized tissue.[12,13,19]

Enzymatic débridement is accomplished by the use of collagenase, papain, streptokinase/streptodornase, trypsin, or similar enzymes that dissolve necrotic debris.

Autolytic débridement is performed by placing occlusive dressings (eg, hydrocolloid) over the wound. Enzymes within the wound fluid then self-digest necrotic tissue. This modality is pain-free but slow and contraindicated in cases of infected wounds.[19]

Wound cleansing. Irrigation of wounds is ideally performed with normal saline, using syringes, needles, and angiocatheters, as described above. These steps should result in an irrigation pressure of 8 psi (range: 4 to 15 psi).[7] Normal saline can be prepared inexpensively, even at home. Sponges or gauze should be used gently. Antiseptic agents (eg, Dakin's solution [ie, diluted sodium hypochlorite], acetic acid, povidone iodine, hydrogen peroxide, iodophor, neomycin sulfate, and chlorhexidine gluconate) are not recommended, because they are cytotoxic to healthy tissue.[6,18]

Wound dressing. The principle behind wound dressing of pressure ulcers is to keep the ulcer bed moist and the surrounding skin dry. Wounds may be packed to obliterate dead space, thus preventing abscess formation. Although wet-to-dry dressing may initially help when mechanical débridement is used, once the ulcer is clean, the dressing should be continuously moist, because a moist environment helps the growth of granulation tissue.

Moist occlusive dressings that are conducive to tissue growth include polyurethane and hydrocolloid dressings. Polyurethane dressings are semipermeable to water vapor and oxygen but impermeable to water. Hydrocolloid dressings are nontransparent and impermeable to gas and moisture. Both types of dressings should be changed every 1 to 4 days.[8,24] Use of moist occlusive dressings is not labor intensive but is very effective as treatment of noninfected wounds.

Wound fillers are useful in deep pressure ulcers to absorb exudate and to obliterate dead space. For mild to moderately exudative wounds, hydrogels and moist saline dressings can be used; for wounds with heavy exudate and possible maceration of normal skin around the wound, foams, alginates, and saline-impregnated gauze are recommended.[8,24] Packing should be changed daily.

Control of infection. It is necessary to distinguish between bacterial colonization (contamination not requiring treatment) and infection (requiring treatment) (**Table 6**). If wounds do not heal and signs of infection (eg, erythema, edema, foul odor, purulent exudate, fever) are present, up to 2 weeks of topical administration of antibiotics (eg, silver sulfadiazine, gentamicin) may be tried.[6,7,18]

The occurrence of bacteremia in patients with pressure ulcers is probably underestimated. When there is evidence of cellulitis, bacteremia, or osteomyelitis, a course of systemic antibiotics is indicated, with coverage for anaerobes, gram-negative bacilli, and gram-positive cocci.[12] Needle aspirates or tissue biopsies can provide useful information about the infection, but swab cultures of the ulcer often provide misleading information. In addition, health providers must practice universal precautions when treating elderly patients with pressure ulcers, including frequent hand washing, use of gloves, and use of sterile instruments

for débridement. Isolation precautions, if indicated, must be followed.

Adjunctive therapy. Several therapies that have been used but are, as yet, of unproven benefit in the treatment of pressure ulcers include topical application of gold, phenytoin, aloe vera gel, growth factors, and other agents; use of systemic agents (eg, vasodilators, fibrinolytic agents, growth hormone); hyperbaric oxygen therapy; and infrared, ultraviolet and low-energy laser treatment. Of the proposed adjunctive therapies, only electrotherapy of wounds has shown any benefit.[1,7,12,25]

Surgical options. Surgical treatment of pressure ulcers includes the use of direct closure, skin grafting, and a variety of flaps. Most of these procedures were developed for younger patients, although age is not an absolute contraindication. The benefits and risks of surgery, as well as the patient's life expectancy and comorbidities, should be considered when deciding whether to pursue surgical options. Postoperatively, a pressure-free environment and adequate rehabilitation is essential for success. The recurrence rate of pressure ulcers after surgery is highly variable.[26]

SUMMARY

Pressure ulcers are common in older populations. Whereas it takes only hours for an ulcer to develop, complete healing can take months. Preventive measures are available and begin with risk assessment in every individual. Good nursing care plays a central role in the care of elderly patients with pressure ulcers. However, optimum ulcer management frequently requires a multidisciplinary approach. **HP**

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