Chapter 52: Pressure Ulcers

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This chapter addresses the following Geriatric Fellowship Curriculum Milestones: #29, #42, #63, #64, #65

LEARNING OBJECTIVES

Learning Objectives

- Identify the four pathophysiologic factors of pressure ulcer development.
- Describe the six pressure ulcer classifications according to the National Pressure Ulcer Advisory Panel's (NPUAP's) guidelines.
- Outline the process for pressure ulcer risk screening and risk assessment.
- Describe the essential strategies for a successful pressure ulcer prevention program.
- Describe the standard of care for full-thickness pressure ulcers.

Key Clinical Points

1. **Pressure ulcers are caused by mechanical force compressing tissues between the bony skeleton and external surfaces occluding capillaries and lymphatics with resultant ischemia and buildup of metabolic cellular waste products, release of oxygen free radicals from reperfusion injury, and cellular apoptosis from cell deformation.**
2. **Prevention includes screening for risk followed by risk assessment using standardized risk assessment tools to determine individual-specific risk and implementing targeted prevention interventions based on identified risk factors.**
3. **Scheduled repositioning programs, use of reactive and active support surfaces, assessment and management of nutrition, and use of prophylactic dressings are key prevention strategies.**
4. **Adequate, timely, and complete debridement of necrotic tissue, identification and treatment of infection and management of biofilm development, and providing a moist wound environment are the key tenets of appropriate pressure ulcer care.**
5. **Medical record documentation must include pressure ulcer risk status, prevention strategies, pressure ulcer assessment (size, stage, location, and description of wound bed minimally), treatment plan and evaluation of treatment success.**
6. **Partial-thickness pressure ulcers (stage 2) should heal within 60 days maximum; full-thickness pressure ulcers (stage 3/4/unstageable) should demonstrate improvement in overall ulcer status every 2 to 4 weeks.**

DEFINITION

Pressure ulcers are areas of local tissue trauma, usually developing where soft tissues are compressed between bony prominences and any external surface for prolonged time periods. A pressure ulcer is a sign of local tissue necrosis. Pressure ulcers are most commonly found over bony prominences subjected to external pressure. The most common locations are sacrum, ischial tuberosities, trochanters, and heels with sacral and heel sites most frequent. Pressure exerts the greatest force at the bony tissue interface; therefore, there may be significant muscle and subcutaneous fat tissue destruction underneath intact skin. Other terms for pressure ulcers include pressure injury, bedsore or decubitus ulcer, both of which imply development only in those confined to bed. Since the major causative factor is pressure, and because pressure ulcers occur in positions other than just lying down, pressure ulcer is the preferred term.

EPIDEMIOLOGY

Pressure ulcers occur in all health care settings. Among hospitalized older patients, the prevalence of pressure ulcers in acute care units has declined by 1% to 2% over the last decade with a median estimate of 6.3%. Most (75%) present with stage 1 or stage 2 ulcers. The incidence during hospitalization ranges between 2.8% over 4 days and 9% over 5 days. Critical care settings present increased risk with pressure ulcer prevalence rates reported at 13% and incidence rates reported between 12% and 17%. There are some evidence of improvement in pressure ulcer incidence. The National Database of Nursing Quality Indicators (NDNQI) is the national project established by the American Nurses Association in 1998 to monitor quality indicators in hospitalized patients that are nursing sensitive. In 2000, the NDNQI added hospital-acquired pressure ulcers (HAPUs) as one of the patient safety and quality-of-care indicators to monitor in participant hospitals. Hospital sites in 2011 included 1721 hospitals in 50 states. Participating institutions collect data by medical record abstraction and direct skin assessments and enter it into a secure website. Comparing HAPU data from 2010 (first and second quarter) to 2004 (quarter 2 and 3) and 2006 (quarter 4) to 2007 (quarter 1) shows a decrease in stages 1 to 4 and unstageable pressure ulcers with a rate of 3.8% in 2010 compared to 6.4% in both 2004 and 2006 to 2007.

Pressure ulcers generally occur within the first 2 weeks of hospitalization (the first 5 days in critical care units), and of those patients with an ulcer, more than half develop them after admission. A new area of concern for pressure ulcer development is related to use of medical devices. Medical devices have been associated with 34% of the pressure ulcers that develop in acute care settings. Medical device pressure ulcer prevalence in acute care is estimated from 8.3% to 9.7% with incidence reported as 5.4%. Epidemiologic data now also separate the suspected deep tissue injury (DTI) pressure ulcer classification with acute care incidence rates of DTI estimated to be 0.5% to 9%.

Pressure ulcers represent a significant health concern for those in nursing homes, rehabilitation systems, and for special populations. The incidence of new lesions varies widely by clinical situation: the highest rates are found among orthopedic populations (9%–19% incidence) and quadriplegics (33%–60% incidence). In nursing homes, prevalence estimates vary from 8.6% to 32.2%. Reported prevalence rates are higher among nursing home residents transferred to the hospital. Prevalence in high-risk residents has been estimated to range from 9.6% to 16.8%. Incidence is similarly diverse with estimates ranging from 3.4% to 4.7% for stage 2 to 4 ulcers and higher incidence when stage 1 ulcers are included (range 5.2%–13.6%). Incidence among terminally ill nursing home residents is reported much higher, 37.5%. Approximately 10% of persons admitted to nursing homes have a pressure ulcer.

There is some evidence of health disparities related to pressure ulcer development. African Americans demonstrate a higher incidence of pressure ulcers and more severe pressure ulcers compared to Caucasians in nursing homes with incidence rates reported as 0.56 per person-year compared to 0.35 per person-year for Caucasians. Incidence of stage 2 to 4 pressure ulcers are nearly two times higher among African Americans than Caucasians. Further, pressure ulcer–associated mortality is higher among blacks than whites. Hispanic and non-Hispanic blacks have a higher prevalence of pressure ulcers than non-Hispanic whites (7.6%, 9.7%, and 12.1%, respectively).

Rehabilitation facilities present special concerns related to pressure ulcer development, because patients in these facilities have conditions that limit mobility, such as spinal cord injury (SCI), traumatic brain injury, cerebral vascular accident, burns, multiple trauma, or a chronic neurologic disorder. Prevalence rates range from 12% to 25%. Individuals with SCI are at higher risk for pressure ulcer development, with incidence rates reported at 20% for those undergoing spinal surgery, increasing to 30% to 40% over 1-year time. Prevalence of pressure ulcers in persons with SCI is also high with reports of 33% to 40% during acute rehabilitation and for those living in the community, with recurrence rates up to 40% after an ulcer heals. In the community, the prevalence of pressure ulcers is 6% to 9% in home health care settings, and 1.6% in outpatient clinic settings. In the home health setting, 20% of the pressure ulcers develop in the first week after admission, and the incidence increases 10% each week through week 4. Of those who develop a pressure ulcer, 50% develop the ulcer within 24 days after admission to home health services.

MORBIDITY ASSOCIATED WITH PRESSURE ULCERS

Pressure ulcers can lead to pain and disfigurement. Of those persons with pressure ulcers who are able to report pain, across nursing homes, home health, and hospital settings, 87% report pain with dressing changes, 84% report pain at rest, and 42% report pain at rest and during dressing changes. Further, 18% of those persons reporting dressing change wound pain report pain at the highest level (eg, “excruciating”). Yet, only 6% of those persons reporting pressure ulcer pain receive any medication for pain. There is some evidence that a higher proportion of persons with stage 3 or 4 ulcers report ulcer pain compared to those persons with stage 2 ulcers and they report more severe pain than those with stage 2 pressure ulcers.

Septicemia is the most severe complication from pressure ulcers. The incidence of bacteremia from pressure ulcers is approximately 1.7 per 10,000 hospital discharges. When the pressure ulcer is the source of bacteremia, overall mortality is 48%. Further, septicemia is reported in 40% of pressure ulcer–associated deaths. Clinicians should be aware that transient bacteremia occurs after pressure ulcer debridement in as many as 50% of patients. Other infectious complications of pressure ulcers include wound infection, cellulitis, and osteomyelitis. Infected pressure ulcers are one of the most common infections found in skilled nursing facilities, and are reported in 6% of residents. Of note, pressure ulcers are typically colonized with greater than or equal to 10^5 organisms/mL of normal skin flora. Although greater than or equal to 10^5 organisms/mL of normal skin flora can cause local infection in intact skin and impair wound healing in flaps and skin grafts, chronic wounds such as pressure ulcers may bear microbial growth at this level for prolonged periods without noticeable clinical manifestations of infection and with evidence of healing. Among patients with nonhealing or worsening pressure ulcers, 26% of ulcers have underlying bone pathology consistent with osteomyelitis, 88% are colonized with *Pseudomonas aeruginosa* species, and 34% with *Providencia* species. The presence of either *P aeruginosa* or *Providencia* species should not be considered typical colonization. Infected pressure ulcers also can serve as reservoirs for infections with antibiotic-resistant bacteria. Methicillin-resistant *Staphylococcus aureus* (MRSA) colonization in infected pressure ulcers has been reported as high as 59%. Bacterial biofilms are known to cause chronic inflammation that contributes to the molecular pathology that is present in nonhealing full-thickness pressure ulcers. Approximately 60% of chronic wounds contain biofilms, which may explain the state of chronic inflammation and impaired healing that occurs in many pressure ulcers. The presence of bacterial colonization in pressure ulcers has been suggested to elevate proinflammatory cytokines such as interleukin 1 and tumor necrosis factor which in turn, increases the level of matrix metalloproteinases (MMPs), decreases the level of inhibitors in tissue against the MMPs, and decreases the production of growth factors and fibroblast activity.

Prolonged hospitalization, slow recovery from comorbid conditions, and increased death rates are consistently observed in older individuals who develop pressure ulcers in both hospitals and nursing homes. Hospital-acquired pressure ulcer development is associated with higher in-hospital mortality (11.2%), mortality 30 days after discharge (15.3%), and longer hospital stays (11.6 ± 10.1 days for those with HAPU vs 4.9 ± 5.2 days for those without).

In addition, failure of an ulcer to heal or improve has been associated with a higher rate of death in nursing home residents. Nursing home residents whose pressure ulcer healed within 6 months show a lower mortality rate (11% vs 64%) than residents with ulcers that did not heal within 6 months. It is unclear how pressure ulcers lead to death. The link between pressure ulcers and mortality may be related to an unidentified causal pathway, to pressure ulcers as a marker for coexisting morbidity in frail, sick patients, or to the association between fatal sepsis and pressure ulcers as cause of death. Whatever the link, pressure ulcers are reported as a cause of death among 114,000 persons per year (age-adjusted mortality rate of 3.8 per 100,000 population).

Pressure ulcers are a quality issue for all areas of health care. Pressure ulcer incidence and severity are used as markers of quality care by regulators in long-term care facilities, home care agencies, and acute care hospitals. The Joint Commission estimates that 2.5 million patients in acute care hospitals are treated for pressure ulcers each year and this number is likely to increase as the population ages. Unlike facility-specific conditions (such as surgical site infection or ventilator-associated pneumonia), pressure ulcers present across all care settings and patients, especially among geriatric populations.

Pressure ulcers are costly. Annual costs were estimated at $10.5 to $17.8 billion for 2010. The cost for managing a single full-thickness pressure ulcer is as much as $70,000. One study reports the average hospital-associated costs for managing one full-thickness stage 4 ulcer and related complications for one hospital admission at $129,248 and costs for managing a community-acquired stage 4 ulcer over an average of four hospital admissions at $124,327. The Centers for Medicare and Medicaid Services (CMS) reports the cost of treating a pressure ulcer in acute care (as a secondary diagnosis) is $43,180.00 per hospital stay. Contributing cost factors include increased length of stay due to pressure ulcer complications such as pain, infection, high-tech support surfaces, and decreased functional ability. The Agency for Healthcare Research and Quality (AHRQ) reported that pressure ulcer–

related hospitalizations ranged from 13 to 14 days and cost $16,755 to $20,430 compared to the average stay of 5 days and costs approximately $10,000. Health care utilization and costs of caring for persons with SCI who experience the complication of a severe (stage 3/4) pressure ulcer are high (in excess of $100,000 annually). As a result, pressure ulcers remain on the national agenda for improving quality. This emphasis on pressure ulcers across the spectrum of health care settings highlights the importance of the condition for clinicians. Pressure ulcers have also received attention in the courtroom. Organizations have been prosecuted for negligence related to pressure ulcer care and development, and, in a landmark case, one health care facility operator was found guilty of manslaughter for a resident's death related to improper care for her pressure ulcers.

PATHOPHYSIOLOGY

Pressure ulcers are the result of mechanical injury to the skin and underlying tissues. The primary external factors involved in pressure ulcer development include pressure (stress), shear, and friction. More recently deformation (strain), heat, re-perfusion injury, and impaired lymphatic function have been considered as additional primary forces involved in pressure damage. Four hypotheses for the pathophysiology behind pressure ulcer development include the following:

1. Ischemia caused by capillary occlusion
2. Impairment in lymphatic flow with increase in metabolic waste products
3. Reperfusion injury (damage that occurs because of the inflammatory response that occurs when blood flow resumes to the ischemic tissues)
4. Deformation of tissue cells

Pressure (stress) and deformation (strain) are involved in all four pressure ulcer development hypotheses. Pressure is the perpendicular force or load exerted on a specific area, causing ischemia and hypoxia of the tissues. The gravitational pull on the skeleton causes loading and deformation of the soft tissue between the bony prominence and external support surface. High-pressure areas in the supine position are the occiput, sacrum, and heels. In the sitting position, the ischial tuberosities exert the highest pressure, and the trochanters are affected in the side-lying position. The mechanical physical forces of shear which is force applied against a surface as it moves or slides in an opposite but parallel direction stretching tissues and displacing blood vessels laterally and deformation which stretches and pulls cells are also key factors in pressure ulcer development. Friction, which is the resistance to motion or rubbing of one object or surface against another in a parallel direction, and moisture which macerates tissues and increases the coefficient of friction between surfaces are major forces in superficial damage over bony prominences, abrading the epidermal surface, and increasing the risk for infection. As the amount of soft tissue available for compression decreases, the pressure gradient increases, thus, most pressure ulcers occur over bony prominences where there is less tissue for compression and the pressure gradient within the vascular network is altered.

The changes in the vascular network allow an increase in the interstitial fluid pressure, which exceeds the venous flow. This results in an additional increase in the pressure and impedes arteriolar circulation. The capillary vessels collapse and thrombosis occurs. Increased capillary arteriole pressure leads to fluid loss through the capillaries, tissue edema, and subsequent autolysis. Lymphatic flow is decreased, allowing further tissue edema, and contributing to the tissue necrosis.

Pressure, over time, occludes blood and lymphatic circulation, causing deficient tissue nutrition and accumulation of waste products, as a result of ischemia. If pressure is relieved before a critical time period is reached, a normal compensatory mechanism, reactive hyperemia, restores tissue nutrition and compensates for compromised circulation. If pressure is not relieved, the blood vessels collapse and thrombose. The tissues are deprived of oxygen, nutrients, and waste removal. In the absence of oxygen, cells use anaerobic pathways for metabolism and produce toxic by-products. The toxic by-products lead to tissue acidosis, increased cell membrane permeability, edema, and eventual cell death.

Deformation of tissues is a key factor in the damage seen in DTI and full-thickness pressure ulcers and may be the force that initiates cell death. Tissues are deformed due to the bone's compression of the soft tissue against the external surface. How the tissues are deformed depends on the tissues (ie, size and shape of the different tissue layers), the mechanical properties of the involved tissues (eg, stiffness, strength), and the magnitude and distribution of the external mechanical force applied to the tissues. The tissues' ability to withstand deformation can change with time due to aging, lifestyle changes, injury, or disease. The sustained mechanical deformation damages cells directly and also obstructs blood flow. In fact it is likely that tissue deformation is the major underlying factor in muscle damage. Several investigators have examined the reaction of cells to deformation. Observing single muscle cells has demonstrated that deformations exceeding 80% have consistently ruptured cell membranes, causing immediate and

irreversible damage. Observations of an entire muscle have demonstrated similar findings; 2 hours of sustained deformation at strains higher than 50% inflicted irreversible damage to muscle tissue. As with hypoxia and ischemia, muscle tissue is more sensitive to deformation forces and irreversible damage may be present at the muscle layer without such damage occurring in the skin or subcutaneous layers. The cell death and local tissue necrosis change the geometry and characteristics of the tissues, which further increases the deformation force exacerbating the pressure injury. Heat accumulation or increased skin temperature intensifies the effects of ischemia and hypoxia on tissues. Increased skin temperature causes an increase in metabolic rate, which increases the need for oxygen in the tissues.

When prolonged pressure is finally relieved, the damage does not end. As the vascular network is relieved of pressure, the tissues are reperfused and reoxygenated. The sudden entry of oxygen into previously ischemic tissues releases oxygen-free radicals known as *superoxide anions*, *hydroxyl radicals*, and *hydrogen peroxide*, all of which induce new endothelial damage and decrease microvascular integrity causing postischemic or reperfusion injury.

These cellular changes result in inflammation and edema *locally* at the site of injury. The inflammatory changes exist in the tissues before any damage is fully visible on the skin surface. This is the *nonvisible spectrum of pressure-induced tissue damage*, the preclinical stage of disease in the physiology cascade leading to frank ulceration. These inflammatory changes with tissue edema can occur from 3 to 10 days before visible skin breakdown.

PRESENTATION AND DETECTION

Detection of Pressure-Induced Tissue Damage

Detection of prestage 1, or preclinical, pressure-induced tissue damage is important because early intervention may prevent decline to more severe pressure damage. There are several nonvisual methods of detecting pressure damage currently being explored for use in clinical practice: ultrasound, thermography, spectroscopy, and surface electrical capacitance. Of these, ultrasound, thermography, and surface electrical capacitance show promise for use clinically.

High-resolution ultrasound is a noninvasive method of visualizing skin and soft tissues that provides echogenic images of skin and deeper tissues. Over 50% of ultrasound images obtained from nursing home residents indicated abnormal findings with tissue edema present compared to ultrasound images obtained from healthy volunteers. Nearly 80% of those with abnormal ultrasound images did not have documentation of erythema suggesting that ultrasound technology can detect tissue damage before clinical signs occur. Advantages of ultrasound technology for detecting early pressure ulcer development include (1) reproducibility, (2) noninvasiveness, and (3) objective and quantitative data on tissue damage below skin surface. However, ultrasound equipment is large and expensive, requires skilled technicians to obtain useful images, and requires trained providers to interpret images.

Thermography, the measurement of skin surface temperature and with some equipment, temperatures of tissues below the skin surface may provide a method for detecting nonvisual pressure damage. Both increased and decreased skin temperature (compared to adjacent normal tissue) is associated with stage 1 pressure ulcers in rehabilitation patients with pressure-induced erythema. Skin temperature variability also differentiates between nursing home residents at high and low risk for pressure ulcer development and between those residents who do and do not develop pressure ulcers. In a study of nursing home residents using a handheld thermistor to measure skin surface temperature, temperatures for sacral and buttocks sites with erythema or stage 1 pressure ulcers were significantly higher than for normal skin. Skin surface temperature was responsive to visual changes in the skin, with higher temperatures associated with any change in the skin, deterioration or improvement. Further, a decrease in skin surface temperature by 4°F predicted stage 1 pressure ulcers that were visible on the skin the following week. One disadvantage to using temperature for detecting nonvisual pressure damage is determination of the normal or baseline temperature for the individual and the specific anatomic site (all studies looked at determining a baseline skin temperature for an individual and specific anatomic site).

Measurement of the water content of the skin and underlying tissue can be accomplished using surface electrical capacitance devices. These devices detect and measure water or edema as the initial inflammatory response of injured tissues *below* the stratum corneum. Using dielectric parameters, high-frequency, low-power electromagnetic waves of 300 MHz or less are transmitted via a device that is manually placed on the skin. In the skin, the induced electrical field interacts with water molecules closest to the probe and displays subepidermal moisture (SEM) values. At the cellular level, SEM indirectly measures the action potential of sodium and potassium across cell membranes. When cells are injured such as occurs with early pressure-induced tissue damage, cellular permeability increases and the action potential across the cell membrane is decreased allowing quick/high electrical charges to pass through the tissues (eg, SEM increases). Devices to measure SEM are small, portable, handheld dermal phase meters, which require

light skin touch with readings available within 3 to 8 seconds. In nursing home residents, critical care patients, veterans with SCI, and persons with dark skin tones SEM has detected inflammatory changes in the tissues, identifying prestage 1, stage 1, and DTI pressure ulcers on the sacrum and heels. In nursing home residents SEM was higher (eg, increased edema and inflammation) when residents exhibited no visible skin damage at the time but a stage 1 or more severe pressure ulcer was visible on the skin 1 week later. SEM identified 26% and 30% of the stage 1 pressure ulcers the following week. For populations at high risk or as a method of screening and monitoring specific populations SEM use holds great promise and appears the most clinically practical of the measures being examined for early detection.

Presentation

The first clinical sign of pressure ulcer formation, blanchable erythema, presents as discoloration of a patch or flat, nonraised area of the skin larger than 1 cm. This discoloration presents as redness or erythema that varies in intensity from pink to bright red in light-skinned patients. In dark-skinned patients, the discoloration appears as deeper normal ethnic pigmentation; a purple or blue-gray hue to the skin. Other characteristics include slight edema and increased temperature of the area. The beginning clinical indicators of pressure ulceration all relate to the signs of inflammation in the tissues. This beginning stage of damage is transient if the pressure is relieved. If pressure is not relieved, the damage can progress. One method of monitoring existing erythema for progression of tissue damage is to test capillary refill every 8 hours. When capillary refill is not present, it indicates an increase in tissue damage.

Nonblanchable erythema involves more severe damage to underlying tissues and is commonly the first stage of pressure ulceration (stage 1 pressure ulcer). The color of the skin is more intense. It varies from dark red to purple or cyanotic in both light- and dark-skinned patients. Dark-skinned patients exhibit deepening of normal skin color, a purple or gray hue to the skin, and changes in skin texture, with induration and an orange-peel appearance. Skin temperature is cool compared with healthy tissues, and the area may feel indurated. This stage of tissue destruction is also reversible, although tissues may take 1 to 3 weeks to return to normal.

The result of further deterioration in the tissues is evidenced as the epidermis is disrupted with subepidermal blisters, crusts, or scaling present (stage 2 pressure ulcer). If properly treated, the situation may resolve in 2 to 4 weeks. The early pressure ulcer is superficial, with indistinct margins and a red, shiny base, and reflects continued tissue insult and progressive injury. It is usually surrounded by erythema. If not dealt with aggressively, the lesion may progress to a chronic, deep ulcer. Superficial ulcers also may begin at the skin surface as the result of friction and moisture on the skin, the effects of both are increased with pressure. While superficial ulcers may progress to deeper ulcers, many deep ulcers do not originate at the skin surface; they begin at the bony prominence and soft tissue interface, and spread to involve the skin structures.

The chronic, deep, full-thickness ulcer usually has a dusky red wound base and does not bleed easily. It is surrounded by blanchable or nonblanchable erythema or deepening of normal skin tone in persons with dark skin tones, induration, and warmth. Undermining, or pocketing, and tunneling may be present with a large necrotic cavity. Eschar formation may be a result of larger vessel damage below skin surface from shearing forces. Eschar is the formation of an acellular dehydrated compressed area of necrosis, usually surrounded by an outer rind of blanchable erythema. Eschar formation indicates a full-thickness loss of skin.

ASSESSMENT

Assessment involves screening for risk of developing pressure ulcers, assessment of the severity of the tissue damage (staging), and evaluation of ulcer healing over the course of treatment.

Risk Screening and Risk Assessment

Pressure ulcer development is related to multiple factors, with immobility or severely restricted mobility being the most important risk factor for all populations and a necessary condition for the development of pressure ulcers. A study of geriatric patients, who were monitored for movements using devices on the bed, showed that individuals with more than 50 movements a night did not develop pressure ulcers compared to 90% of individuals with 20 or fewer spontaneous body movements at night who developed a pressure ulcer.

Incontinence, malnutrition, impaired mental status, and altered sensation or response to pain and discomfort are all risk factors with strong relationships to pressure ulcer development in prospective studies. Fecal incontinence has a more powerful relationship to pressure ulcer development than urinary incontinence. Certain risk factors can be easily identified and used to quickly screen individuals in order to place them

immediately on a general prevention program. Screening is particularly useful for acute care hospitals and specific populations. Screening factors range from individual factors such as age to factors that are more related to procedures or medical conditions such as emergent admission to the hospital or prolonged surgical time. Some factors that might be used as a screen for pressure ulcer risk include the following:

- Demographic characteristics: African-American race, and advanced age (over 75 years).
- Individuals of all ages who have limited mobility or for whom movement is not possible without staff or caregiver assistance including persons who are unable to move due to sedation.
- Persons admitted to the hospital from a nursing home. These individuals are already frail as they require nursing home care and are acutely ill.
- Persons admitted to the critical care unit from the emergency department.
- Persons over 65 years admitted to the acute care hospital who are scheduled for a surgical procedure anticipated to be 4 hours or longer.
- Patients who experience intraoperative hypotensive episodes.
- Older patients admitted to the hospital from the community who were found down for a prolonged time.
- Patients undergoing special therapy, for example, oncology patients admitted for bone marrow transplant or with graft-versus-host disease.
- Patients with hospital transport times more than 1 hour.
- Patients readmitted to a nursing home from the hospital.
- All persons who have had a previous pressure ulcer and especially persons with SCI or dysfunction with prior pressure ulcers.
- Patients admitted with any diagnosis affecting skin integrity (besides obvious open wounds this includes gangrene, nutritional deficiencies, diabetes, and anemia).
- Patients admitted with system failure including paralysis, senility, respiratory failure, acute renal failure, cerebral vascular accident, and congestive heart failure.
- Patients admitted with infection: sepsis, osteomyelitis, pneumonia, bacterial infections, and urinary tract infections.
- Intensive care unit patients with sepsis, surgery times more than or equal to 8 hours, or long-term vasopressor therapy (consider at high risk).

For practitioners to intervene cost-effectively, a method of screening for risk of developing pressure ulcers is necessary followed by more specific risk assessment to develop individualized prevention strategies. Use of a risk assessment tool is a mechanism to identify those persons at risk of developing pressure ulcers in multiple health care settings and for developing care plans that reflect specific risk. Risk assessment is recommended in all clinical practice guidelines for pressure ulcers. The purpose in identifying patients at risk for pressure ulcer development is to allow for appropriate use of resources for prevention.

The use of a risk assessment tool allows targeting of interventions to specific risk factors for individual patients. There is, however, limited evidence to support a direct link between use of risk assessment tools and decreased incidence of pressure ulcers. Multifaceted prevention interventions that have included use of risk assessment tools have shown decreased pressure ulcer incidence levels from 13% to 23% preintervention to 2% to 5% postintervention. The outcome, however, cannot be solely attributed to use of risk assessment tools. Use of risk assessment tools is linked to increased documentation of prevention interventions and is better than use of clinical judgment alone, particularly for those at moderate risk. While there is no study that provides definitive evidence linking risk assessment directly with pressure ulcer prevention, there is a relationship between conducting risk assessment and initiating preventive interventions leading to decreased pressure ulcer incidence.

Pressure ulcer risk assessment should be performed on admission to the health care setting and at periodic intervals thereafter. In acute care hospitals, risk assessment should be repeated every 48 hours. Those persons admitted to intensive or critical care units should have risk assessment conducted on admission and if determined at risk, daily thereafter. In home health settings, risk assessment should be conducted weekly for the first 4 weeks with every other week reassessments thereafter depending on patient condition and frequency of home visits. Nursing home residents should

be reassessed for pressure ulcer risk status weekly for the first 4 weeks following admission followed by quarterly assessments.

The most commonly used risk assessment tools are the Norton Scale and the Braden Scale for predicting pressure sore risk. The Norton Scale is the oldest risk assessment instrument. Developed in 1961, it consists of five subscales: physical condition, mental state, activity, mobility, and incontinence. Each parameter is rated on a scale of 1 to 4, with the sum of the ratings for all five parameters yielding a total score, ranging from 5 to 20. Lower scores indicate increased risk, with a score of or below 16 indicating "onset of risk" and scores 12 and below indicating high risk for pressure ulcer formation. A systematic review and meta-analysis of risk assessment tools found sensitivity of 0.75, specificity of 0.68, area under the receiver operating curve 0.74, and relative risk 3.69 (confidence interval [CI] 2.64–5.16) for the Norton tool using a cutoff score of 14 for indicating risk status. The Braden Scale was developed in 1987 and is composed of six subscales: sensory perception, moisture, activity, mobility, nutrition, and friction and shear. All subscales are rated from 1 to 4, except for friction and shear, which is rated from 1 to 3. The subscales may be summed for a total score, with a range from 6 to 23. Lower scores indicate lower function and higher risk for developing a pressure ulcer. The cutoff score for hospitalized adults is considered to be 16, with scores of 16 and below indicating at-risk status. In older patients, some have found cutoff scores of 17 or 18 to be better predictors of risk status. Levels of risk are based on the predictive value of a positive test. Scores of 15 to 16 indicate mild risk, with a 50% to 60% chance of developing a stage 1 pressure ulcer; scores of 12 to 14 indicate moderate risk, with a 65% to 90% chance of developing a stage 1 or 2 lesion; and scores below 12 indicate high risk, with a 90% to 100% chance of developing a stage 2 or deeper pressure ulcer. A systematic review and meta-analysis of risk assessment tools found sensitivity of 0.74, specificity of 0.68, area under the receiver operating curve 0.77, and relative risk 4.26 (CI 3.27–5.55) for the Braden Scale using a cutoff score of 18 for indicating risk status.

Specific prevention strategies should be targeted to risk factors identified in individual patients. In those persons in whom prevention is not successful, the continued monitoring of risk status may prevent further tissue trauma at the wound site and development of additional wound sites.

Assessment of Pressure Ulcer Stage

Pressure ulcers are commonly classified using grading or staging systems based on the observable depth of tissue destruction. The stage is determined on initial assessment by noting the deepest layer of tissue involved. The ulcer is not restaged unless deeper layers of tissue become exposed. The initial method of classifying pressure ulcers was a pathology-based classification system intended to simplify communication for health-care professionals, provide a mechanism for identification of pressure ulcers, and suggest a broad guide for determining whether operative care was needed. Each grade of pressure ulceration was defined by the anatomic limit of soft tissue damage that could be observed. The numeric classification system suggested an orderly evolution of pressure ulceration, however, pressure ulcers do not heal or deteriorate in a linear fashion.

The most commonly used staging system is the NPUAP's system describing six classifications of pressure ulcers. The NPUAP staging system was updated in 2014 in conjunction with the European Pressure Ulcer Advisory Panel (EPUAP) and again in 2016. **Table 52-1** presents the definitions for pressure ulcer stages.

TABLE 52-1

NATIONAL PRESSURE ULCER ADVISORY PANEL, 2016 PRESSURE ULCER STAGING CLASSIFICATIONS

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

Assessment of Pressure Ulcer Healing

Routine pressure ulcer assessment is the base for maintaining and evaluating the therapeutic plan of care. Initial assessment and follow-along assessments at regular intervals to monitor progress are necessary to determine the effectiveness of the treatment plan. Assessment of ulcer status should be performed weekly and whenever a significant change is noted in the wound. Assessment should not be confused with monitoring the ulcer at each dressing change. Monitoring can be performed by less skilled caregivers; however, assessment should be performed on a routine basis by trained clinicians. At a minimum, the ulcer should be assessed for location, depth and stage, size, and wound bed description such as necrotic tissue, exudate, wound edges for undermining and tunneling, and presence or absence of granulation and epithelialization.

There are two research-based pressure ulcer assessment tools for evaluating wound status and healing, the Bates-Jensen Wound Assessment Tool (BWAT) (**Figure 52-1**) and the NPUAP's Pressure Ulcer Scale for Healing tool (PUSH) (**Figure 52-2**). Clinical practice guidelines, expert panels, and

federal nursing home guidelines recommend standardized assessment of pressure ulcers, and many groups recommend use of a standardized tool for pressure ulcer assessment. One prospective study showed improved pressure ulcer healing outcomes (36% of stage 3 and 4 ulcers during 12 weeks with an average healing time of 62 days) when the BWAT was used for standardized pressure ulcer assessment and interventions were tied to the assessment.

FIGURE 52-1.

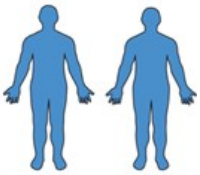
Bates-Jensen Wound Assessment Tool.

BATES-JENSEN WOUND ASSESSMENT TOOL NAME

Complete the rating sheet to assess wound status. Evaluate each item by picking the response that best describes the wound and entering the score in the item score column for the appropriate date. If the wound has healed/resolved, score items 1, 2, 3, & 4 as = 0.

Location: Anatomic site. Circle, identify right **(R)** or left **(L)** and use **"X"** to mark site on body diagrams:

___ Sacrum & coccyx ___ Lateral ankle
___ Trochanter ___ Medial ankle
___ Ischial tuberosity ___ Heel
Other Site



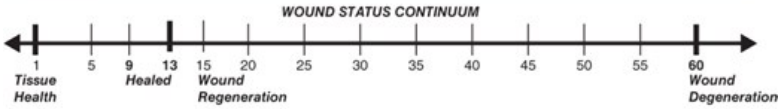
Shape: Overall wound pattern; assess by observing perimeter and depth. Circle and date appropriate description:

___ Irregular ___ Linear or elongated
___ Round/oval ___ Bowl/boat
___ Square/rectangle ___ Butterfly
Other Shape

Item	Assessment	Date Score	Date Score	Date Score
1. Size*	*0 = Healed, resolved wound 1 = Length x width < 4 sq cm 2 = Length x width 4–< 16 sq cm 3 =Length x width 16.1–< 36 sq cm 4 = Length x width 36.1–< 80 sq cm 5 = Length x width > 80 sq cm			
2. Depth*	*0 = Healed, resolved wound 1 = Nonblanchable erythema on intact skin 2 = Partial thickness skin loss involving epidermis &/or dermis 3 = Full thickness skin loss involving damage or necrosis of subcutaneous tissue; may extend down to but not through underlying fascia; &/or mixed partial & full thickness &/or tissue layers obscured by granulation tissue 4 = Obscured by necrosis 5 = Full thickness skin loss with extensive destruction, tissue necrosis or damage to muscle, bone or supporting structures			
3. Edges*	*0 = Healed, resolved wound 1 = Indistinct, diffuse, none clearly visible 2 = Distinct, outline clearly visible, attached, even with wound base 3 = Well-defined, not attached to wound base 4 = Well-defined, not attached to base, rolled under, thickened 5 = Well-defined, fibrotic, scarred, or hyperkeratotic			
4. Under-mining*	*0 = Healed, resolved wound 1 = None present 2 = Undermining < 2 cm in any area 3 = Undermining 2–4 cm involving < 50% wound margins 4 = Undermining 2–4 cm involving > 50% wound margins 5 = Undermining > 4 cm or tunneling in any area			
5. Necrotic Tissue Type	1 = None visible 2 = White/grey nonviable tissue &/or nonadherent yellow slough 3 = Loosely adherent yellow slough 4 = Adherent, soft, black eschar 5 = Firmly adherent, hard, black eschar			
6. Necrotic Tissue Amount	1 = None visible 2 = < 25% of wound bed covered 3 = 25% to 50% of wound covered 4 = > 50% and < 75% of wound covered 5 = 75% to 100% of wound covered			
7. Exudate Type	1 = None 2 = Bloody 3 = Serosanguineous: thin, watery, pale red/pink 4 = Serous: thin, watery, clear 5 = Purulent: thin or thick, opaque, tan/yellow, with or without odor			

Source: J.B. Halter, J.G. Ouslander, S. Studenski, K.P. High, S. Asthana, M.A. Supiano, C. Ritchie, W.R. Hazzard, N.F. Woolard: Hazzard's Geriatric Medicine and Gerontology, Seventh Edition, www.accessmedicine.com

Item	Assessment	Date Score	Date Score	Date Score
8. Exudate Amount	1 = None, dry wound 2 = Scant, wound moist but no observable exudate 3 = Small 4 = Moderate 5 = Large			
9. Skin Color Surrounding Wound	1 = Pink or normal for ethnic group 2 = Bright red &/or blanches to touch 3 = White or grey pallor or hypopigmented 4 = Dark red or purple &/or nonblanchable 5 = Black or hyperpigmented			
10. Peripheral Tissue Edema	1 = No swelling or edema 2 = Nonpitting edema extends < 4 cm around wound 3 = Nonpitting edema extends > 4 cm around wound 4 = Pitting edema extends < 4 cm around wound 5 = Crepitus and/or pitting edema extends > 4 cm around wound			
11. Peripheral Tissue Induration	1 = None present 2 = Induration, < 2 cm around wound 3 = Induration 2–4 cm extending < 50% around wound 4 = Induration 2–4 cm extending > 50% around wound 5 = Induration > 4 cm in any area around wound			
12. Granulation Tissue	1 = Skin intact or partial thickness wound 2 = Bright, beefy red; 75% to 100% of wound filled &/or tissue overgrowth 3 = Bright, beefy red; < 75% & > 25% of wound filled 4 = Pink, &/or dull, dusky red &/or fills 25% of wound 5 = No granulation tissue present			
13. Epithelialization	1 = 100% wound covered, surface intact 2 = 75% to < 100% wound covered &/or epithelial tissue extends > 0.5 cm into wound bed 3 = 50% to < 75% wound covered &/or epithelial tissue extends to < 0.5 cm into wound bed 4 = 25% to < 50% wound covered 5 = < 25% wound covered			
	TOTAL SCORE			
	SIGNATURE			



Plot the total score on the Wound Status Continuum by putting an "X" on the line and the date beneath the line.
Plot multiple scores with their dates to see-at-a-glance regeneration or degeneration of the wound.

FIGURE 52-2.

National Pressure Ulcer Advisory Panel Pressure Ulcer Scale for Healing tool. (© National Pressure Ulcer Advisory Panel.)

PUSH Tool 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 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	**0** 0 cm^2	**1** < 0.3 cm^2	**2** 0.3–0.6 cm^2	**3** 0.7–1.0 cm^2	**4** 1.1–2.0 cm^2	**5** 2.1–3.0 cm^2	
× Width		**6** 3.1–4.0 cm^2	**7** 4.1–8.0 cm^2	**8** 8.1–12.0 cm^2	**9** 12.1–24.0 cm^2	**10** > 24 cm^2	**Subscore**
Exudate Amount	**0** None	**1** Light	**2** Moderate	**3** Heavy			**Subscore**
Tissue Type	**0** Closed	**1** Epithelial Tissue	**2** Granulation Tissue	**3** Slough	**4** Necrotic Tissue		**Subscore**
							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 × width) to obtain an estimate of surface area in square centimeters (cm^2). 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).

Version 3.0: 9/15/98
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The PUSH tool incorporates surface area measurements, exudate amount, and surface appearance. The clinician measures the size of the wound, using length and width to calculate surface area and chooses the appropriate size category of 10 categories. Exudate is evaluated as none (0), light (1), moderate (2), and heavy (3). Tissue type is rated as closed (0), epithelial tissue (1), granulation tissue (2), slough (3), and necrotic tissue (4). Each of the three items is scored, then the three subscores can be summed for a total score. The PUSH tool offers a quick assessment to predict healing outcomes, but assessment of additional wound characteristics may still be needed in order to develop a treatment plan for the pressure ulcer.

The BWAT, developed in 1990 and revised in 2001, evaluates 13 wound characteristics using a five-point numerical rating scale, and rates them from best (scored as 1) to worst (scored as 5) possible (see **Figure 52-1**). Characteristics include size, depth, edges, undermining or pockets, necrotic tissue type and amount, exudate type and amount, surrounding skin color, peripheral tissue edema and induration, granulation tissue, and epithelialization. Similar to the PUSH tool, once characteristics have been scored, they can be summed for a total score (range 13–65). The total score differentiates between early pressure ulcers (stage 1 and 2) and stage 3 and 4 ulcers with mean total scores of 23 versus 32, respectively. A 1-week improvement in the total score demonstrates a positive predictive value of 65% (sensitivity 61%; specificity 52%) for achieving 50% wound healing within a 6-week time period. The BWAT has been incorporated in a variety of electronic medical records. Both the PUSH tool scores and the BWAT tool scores are highly correlated, and use is based on provider and institutional preference.

In general, pressure ulcer healing is accelerated during the initial 3 months after development with 31% of stage 3 and 23% of stage 4 ulcers healing within that time frame. In both partial- and full-thickness pressure ulcers, reduction in size after 1 to 2 weeks of therapy has been shown to be predictive of healing outcomes. Clinical practice guidelines suggest pressure ulcers should show evidence of improvement within 2 to 4 weeks after initiating appropriate treatment, and if no improvement is evident, the treatment plan should be reevaluated. Improvement rates for stage 3 and 4 ulcers are slower than stage 2 ulcers with 75% of stage 2 wounds healing in 60 days, while only 17% of stage 3 or 4 ulcers heal in the same time period.

MANAGEMENT

Local Treatment

Pressure ulcer management includes debridement of necrotic tissue, management of infection and biofilms, adequate wound cleansing, and application of appropriate topical therapy. Wound debridement is necessary to reduce the necrotic tissue burden, decrease risk for infection, and promote granulation tissue formation. Benefits of debridement also may include removal of senescent fibroblasts and nonmigratory hyperproliferative epithelium, and stimulation of blood-borne growth factor production. Debridement is not indicated for dry eschar presenting on the heel or when the pressure ulcer presents on an ischemic limb.

Five methods of debridement (eg, surgical or sharp, mechanical, autolytic, enzymatic, biosurgical) are available. Choice of debridement method is based on clinician preference and availability rather than specific evidence. Clinical practice guidelines on pressure ulcer treatment recommend wound debridement with surgical or sharp debridement for extensive necrosis or when obtaining a clean wound bed quickly is important and more conservative methods (autolytic and enzymatic) for those in long-term care or home care environments. Adequate wound debridement is essential to wound bed preparation and healing. Initial debridement with additional debridement at intervals is often necessary to maintain a biofilm-free wound bed and support healing.

Sharp debridement involves use of a scalpel, scissors, or other sharp instruments to remove nonviable tissue. It is the most rapid form of debridement, and it is indicated over other methods for removing thick, adherent, and/or large amounts of nonviable tissue and when advancing cellulitis or signs of sepsis are present. Health care professionals who use sharp debridement must demonstrate their competency in sharp wound debridement skills and meet licensing requirements. One multicenter, randomized, controlled trial comparing the effects of topical growth factor versus placebo on healing noted that independent of treatment effects, centers that used sharp debridement more frequently experienced better healing rates than those that used sharp debridement less frequently. Sharp debridement is rapid, but is also considered nonselective as viable tissues may be inadvertently removed along with necrotic tissue.

Mechanical debridement involves the use of wet-to-dry dressings, whirlpool, lavage, or wound irrigation. Wet-to-dry gauze dressings continue to be used for debridement, despite the significant disadvantages of increased time/labor for application/removal of the dressings, removing viable tissue as well as nonviable tissue, and pain. This method of debridement should be used cautiously, as it can traumatize new granulation tissue and epithelial tissue, and adequate analgesia should be administered when this method is employed. It is not recommended in clinical practice guidelines.

Enzymatic debridement involves applying a concentrated, commercially prepared enzyme to the surface of the necrotic tissue, in the expectation that it will aggressively degrade necrosis by digesting devitalized tissue. The main enzyme ointment available in the United States is collagenase. Some of the effects noticed with enzymatic agents have been attributed to autolysis. Enzymatic ointments have yielded consistently positive results for their efficacy in wound debridement. Debridement with enzymatic ointments is faster than with autolysis, and more conservative than sharp debridement.

Autolytic debridement is the process of using the body's own mechanisms to remove nonviable tissue. Maintaining a moist wound environment allows collection of fluid at the wound site, which allows enzymes within the wound fluid to digest necrotic tissue. Autolytic debridement typically involves adequate wound cleansing to wash out the partially degraded nonviable tissue. It is more effective than wet-to-dry gauze dressings, as it selectively removes only the necrotic tissue and therefore protects healthy tissues. Autolytic debridement may be slower to achieve a clean ulcer bed than other methods.

Biosurgery is the fifth method of debridement. Biosurgery is the application of maggots (disinfected fly larvae, *Phaenicia sericata*) to the wound typically at a density of 5 to 8/cm^2. Comparative controlled studies evaluating the use of maggot therapy for pressure ulcer debridement have shown a higher proportion of complete debridement in maggot-treated wounds versus standard debridement therapy (80% vs 48%, respectively). Biosurgery may not be acceptable to all patients, and may not be available in all areas.

Pressure ulcers are the result of ischemia and as such, they are more susceptible to infection. Stage 3, 4, and unstageable pressure ulcers should be evaluated for infection. One difference in dealing with infection in chronic wounds such as pressure ulcers compared to acute wounds is the assessment and treatment of bacterial biofilms. Biofilms are the critical colonization of microorganisms on the wound bed that develop support structures that protect the bacteria. Biofilms cause chronic inflammation and have enhanced resistance to endogenous antibodies and phagocytic cells and exogenous antibiotics and antimicrobial solutions. Approximately 60% of chronic wounds contain bacterial biofilms, and this may be the

underlying pathology preventing wounds like pressure ulcers from healing. Biofilm presence in pressure ulcers should be suspected when any of the following exist:

- Lack of signs of healing for 2 weeks *with* appropriate care
- Friable granulation tissue
- Odor
- Increased pain, heat, exudate, or necrotic tissue
- Change in exudate character
- Pocketing or tunneling in the wound bed

Bacterial burden of the pressure ulcer should be determined by tissue biopsy or quantitative swab technique (do not swab necrotic tissue or exudate, rotate end of swab over 1 cm^2 area for 5 seconds with sufficient force to cause tissue fluid expression). Use of antiseptic solutions for a course of therapy may be beneficial in reducing and/or preventing bacterial biofilms and supporting granulation tissue development and wound healing.

The best method of preventing biofilm development is adequate, timely, and complete debridement of necrotic tissue followed by appropriate topical therapy.

Pressure ulcer cleansing at each dressing change is recommended in clinical practice guidelines on pressure ulcer treatment. However, there is evidence that antiseptic solutions such as 5% mafenide acetate (Sulfamylon solution), 10% povidone with 1% free iodine (Betadine), 0.25% sodium hypochlorite ("half strength" Dakin solution), 3% hydrogen peroxide, and 0.25% acetic acid have varying effects on wound healing parameters as well as antimicrobial management in an animal wound model yet, how this affects human wounds is unclear.

Use of antiseptic and antimicrobial solutions for cleansing *clean* pressure ulcers is not indicated based on in vitro studies of the toxicity of topical wound cleansers. Findings from in vitro studies have not been confirmed in human wounds. Use of antiseptic and antimicrobial solutions for cleansing pressure ulcers with *necrotic* debris should be employed thoughtfully with attention to the solution chosen, the characteristics of the microorganisms present in the wound, and duration of use (eg, course of therapy for 2 weeks with evaluation for continuation at that time).

In general, if an ulcer contains necrotic debris or is infected, then antimicrobial activity is more important than cellular toxicity. The chemical and mechanical trauma of wound cleansing should be balanced by the dirtiness of the wound. For wounds with large amounts of debris, more vigorous mechanical force and stronger solutions may be used, while for clean wounds, less force and physiologic solutions such as normal saline should be used.

Topical therapy for pressure ulcers should be provided using moist wound healing dressings. Randomized controlled trials as well as several comparative studies provide compelling support for use of moist wound healing dressings instead of any form of dry gauze dressings (eg, wet-to-dry gauze, dry gauze dressing, or impregnated gauze dressing) for pressure ulcers. Moist wound healing allows wounds to re-epithelialize up to 40% faster than wounds left open to air. Controlled trials suggest that the use of semiocclusive dressings such as transparent films and hydrocolloid dressings improves healing of stage 2 pressure ulcers. These dressings are changed every 3 to 5 days, which allows wound fluid to gather underneath the dressing, facilitating epithelial migration. Moderate evidence exists to specifically support use of hydrocolloid dressings for pressure ulcer care in stage 3/4 pressure ulcers. One multicenter, randomized trial demonstrated faster healing in stage 3/4 pressure ulcers when treated sequentially with calcium alginate dressings followed by hydrocolloid dressings versus nonsequentially with hydrocolloid dressings. However, the relative merits of different categories of moisture retentive dressings versus another remain unclear. **Table 52-2** presents general characteristics of moisture retentive dressing categories.

TABLE 52-2

GENERAL CHARACTERISTICS OF MOISTURE RETENTIVE DRESSING CATEGORIES

DRESSING CATEGORY	DEFINITION	USES	NOTES

Composite Dressings	Combine one dressing group with another to address wound characteristics. For example, gauze/foam and transparent film dressing properties, hydrocolloid, and alginates, etc.	Absorbent (depends on combination of dressings used in the composite) Wicks away excess moisture Nonadherent to wound bed Use depends on the combination of dressings used in the composite	• Some may be difficult to apply • If nonadherent to wound bed then requires secondary dressing to hold in place • Can be confusing to caregivers as combines various dressing category properties
Transparent Film Dressings	Polyurethane and polyethylene membrane film coated with a layer of acrylic hypoallergenic adhesive. Moisture vapor transmission rates (MVTR) vary	Appropriate for partial-thickness wounds Promotes epithelialization Semipermeable Bacterial barrier Autolysis Wound visible Protects against friction Self adhesive	• May reinjure area on removal • Nonabsorbent so can lead to wound edge maceration • Tendency to remove prematurely • Indicated for minimal exudate, does not absorb drainage • Not indicated for moderate-to-heavy exudate
Hydrocolloids Regular or thin wafers, paste, granules	Gelatin, pectin, carboxymethylcellulose in a polyisobutylene adhesive base with a polyurethane or film backing	Absorbs low-to-moderate wound fluid Autolysis Thermal insulation Bacterial barrier Reduces pain Translucent to opaque Easy to apply Controls odor (until dressing removed) Impermeable to semipermeable	• Not indicated for heavy exudate, limited absorbent abilities when used alone • Use with other products increases absorbent abilities • Odor on removal • Regular wafers are opaque, thin wafers allow some wound visualization • Edges may melt down and stick to linens • Some difficult to remove • Possible sensitivity to adhesive backing • Use with close supervision on immunosuppressed and diabetic patients,

			extensive burns, infected lesions
Hydrogels Sheets, wafers Amorphous gels Impregnated gauze	May or may not be supported by a fabric net, high water content, varying amounts of gel-forming material (glycerin, copolymer, water, propylene glycol, humectant)	Absorbs low-to-moderate drainage Autolysis Nonadhesive, may have adhesive borders Semipermeable or impermeable depending on backing Thermal insulation Reduces pain Conformable Carrier for topical medication	• Can dry out • May macerate surrounding tissues • Requires secondary dressing or tape to keep in place • Does not cause reinjury upon removal • Cooling effect can help relieve wound pain • Candidiasis may present from inappropriate usage
Wound Fillers (Exudate Absorbers) Beads, flakes Pastes, powders	Consists of copolymer starch, dextranomer beads or hydrocolloid paste that swell on contact with wound fluid to form gel, dextranomers, polysaccharides, starch, natural polymers, and colloidal particles	Moisture retentive Absorptive (moderate to large) Useful to fill cavities, pockets, undermining Can be used with topical medications	• Nonadhesive • Requires a secondary dressing to hold in place • May have burning sensation on application • May have odor • Some require mixing • Some require wound irrigation for nontraumatic removal
Alginates Ropes, pads, wafers	Calcium-sodium salts of alginic acid (naturally occuring polymer in seaweed)	Absorptive (moderate to large) Useful to fill cavities, pockets, undermining Moisture retentive Can use with topical medications or on infected wounds Reduces pain Thermal insulation	• Nonadherent, requires secondary dressing to hold in place • Hemostatic properties • May require wound irrigation for removal • No reinjury on removal • May dry out, should not be used on wounds with low exudate
Foams Wafers (thick or thin), pillows, composite dressings with thin film covers, available with surfactant impregnated or charcoal layer	Inert material that is hydrophilic and nonadherent, modified polyurethane foam	Absorptive (moderate to large) Autolysis Can be used with	• Nonadhesive unless used with composite dressing • Requires tape or secondary dressing to hold in place

		topical medications and on infected wounds Conformable Thermal insulation	• Nontraumatic removal • Opaque • Waterproof, inert
Hydrofibers Pads, wafers, ropes	Soft nonwoven pad or ribbon dressings made from sodium carboxymethyl-cellulose fibers, similar absorbent material used in hydrocolloid dressings	Absorptive (moderate to large) Autolysis Thermal insulation Reduces pain	• Nonadherent, requires secondary dressing to hold in place • Hemostatic properties • May require wound irrigation for removal • No reinjury on removal

Evidence supporting use of advanced wound therapy in pressure ulcers is unclear. There is some evidence supporting use of negative pressure wound therapy (NPWT) in large stage 3 and 4 nonhealing pressure ulcers with poor granulation tissue or excess exudate. Several clinical practice guidelines and panels have recommended use of NPWT with large stage 3 and 4 pressure ulcers that have failed to improve with standard care with moist wound healing. A case series of 10 patients with stage 4 pressure ulcers treated with NPWT showed greater than 50% average reduction in wound volume and depth (55% and 61%, respectively) over 4 weeks. Some have suggested that NPWT use should result in pressure ulcer improvement within a 2-week time period with further use questionable if improvement is not evident. Use of skin and tissue substitutes has not been examined specific to pressure ulcers but may be of benefit in certain populations with full-thickness ulcers.

Surgery

Surgical treatment of pressure ulcers includes primary closure, a variety of approaches to skin grafts and myocutaneous flaps, and removal of underlying bony prominences. In patients with large infected pressure ulcers, more aggressive procedures such as amputation and hemicorporectomy are sometimes required. Surgical complication rates (including dehiscence, infection, necrosis, and hematoma) for both younger paraplegic patients and nonparaplegic elders are as high as 50%, and pressure ulcer recurrence at the same site has been reported ranging from 30% to 70%. Thus, the long-term outcomes have not been ideal even though 70% to 80% of surgically treated pressure ulcers are healed upon discharge from the hospital. Further, while recurrence of pressure ulcers at the same site is lower for elders (40%) compared to younger paraplegic patients (more than 70%), 30% of elders develop new ulcer sites, and mortality in elders ranges from nearly 50% to 68%.

Thus, the benefits of surgical closure for pressure ulcer are uncertain. In addition to questions about the efficacy of surgical intervention, geriatric patients present with multiple chronic diseases and conditions that may make them less than ideal surgical candidates or affect rehabilitation efforts after surgery.

Drugs

Pharmacologic interventions for pressure ulcers focus on antibiotics and pain management. Antibiotics may be systemic or local. Clinicians should institute systemic antibiotics for patients exhibiting signs and symptoms of systemic infection such as sepsis or cellulitis with associated fever and an elevated white blood cell count. Systemic antibiotics should be initiated for osteomyelitis or for the prevention of bacterial endocarditis in persons with valvular heart disease and who require debridement of a pressure ulcer. Because of the high mortality of sepsis associated with pressure ulcers despite appropriate antibiotics, broad-spectrum coverage for aerobic gram-negative rods, gram-positive cocci, and anaerobes is indicated pending culture results in patients with suspected bacteremia. Ampicillin-sulbactam, imipenem, meropenem, ticarcillin-clavulanate, piperacillin-tazobactam, and a combination of clindamycin or metronidazole with ciprofloxacin, levofloxacin, or an aminoglycoside are appropriate choices for initial antibiotic therapy. Vancomycin may be required for MRSA.

Use of cadexomer iodine (not the same as povidone iodine) topical dressings has been shown to be effective for pressure ulcers colonized with MRSA.

The most effective strategy for preventing infection and dealing with existing infection is adequate and full debridement of necrotic tissue followed by additional debridement to remove and prevent bacterial biofilm development. In patients with signs and symptoms of systemic infection and in those who are septic, the appropriate debridement method is surgical debridement.

Topical antibiotics are most appropriate for stage 3 or 4 ulcers when there is evidence of local infection such as erythema surrounding a clean nonnecrotic wound, failure to improve with adequate treatment, or friable granulation tissue. A 2-week trial of a broad- spectrum topical antibiotic can be considered for clean pressure ulcers that are not healing after 2 to 4 weeks of optimal management. Use of cadexomer iodine dressing or cleansing with hypochlorous acid solution may also be appropriate in this situation. On the other hand, clinicians should not use povidone-iodine, iodophor, sodium hypochlorite, hydrogen peroxide, or acetic acid as topical therapies on *clean* pressure ulcers. These antiseptic agents have been shown to be toxic to fibroblasts and to impair wound healing in in vitro laboratory studies, and how these solutions affect human wounds is unclear. There is no evidence for using prolonged silver release dressings in routine management of healing pressure ulcers.

There is limited evidence to guide clinicians on appropriate management of pressure ulcer–related pain. The pressure ulcer alone may not require routine pain medication, but medication prior to procedures is essential. Lower levels of pain may be manageable with appropriate wound dressing choice and topical wound analgesia. Nonpharmacologic techniques useful for noncyclic and cyclic wound pain associated with procedures (eg, debridement, dressing changes, repositioning) include use of distraction (eg, talking to the patient while performing the procedure), allowing the patient to call a “time-out” during the procedure, and allowing the patient to control and participate in the procedure.

Pressure ulcer–related pain can be minimized by keeping the pressure ulcer wound bed moist and covered. Use of hydrogels, hydrocolloids, alginates, polymeric membrane foams, and soft silicone dressings allows for less frequent dressing changes, and less trauma and pain on removal as they are nonadherent to the wound bed.

Pharmacologic strategies for wound pain include providing opioids and/or nonsteroidal anti-inflammatory drugs (NSAIDs) 30 minutes prior to the procedure and afterward, and administering topical anesthetics or topical opioids using hydrogels as a transport media. Two options have been successful for use in chronic wound pain, EMLA cream and diamorphine gel. EMLA cream (eutectic mixture of lidocaine 2.5% and prilocaine 2.5%) reduces debridement pain scores in chronic venous ulcers, and may have a vasoactive effect cutaneously. Use of EMLA cream in venous ulcers has been associated with a reduction in pain scores (measured on a 100-mm scale) of 20.6 mm. Low-dose topical morphine (diamorphine) has been used in several small, randomized, placebo-controlled studies to successfully control pressure ulcer–related pain.

Nutrition

Multiple studies have demonstrated a relationship between different markers of malnutrition (eg, serum albumin level, dietary protein intake, inability to feed self, and weight loss) and pressure ulcer formation. Other studies have demonstrated that the severity of the pressure ulcer is associated with the severity of the malnutrition. Malnutrition and/or weight loss has been associated with fourfold higher risk of pressure ulcer development. Although it seems intuitive, it has proven difficult to define a specific causal relationship between malnutrition and pressure ulcer development. Modest evidence exists to support providing oral nutritional supplements to persons at risk for pressure ulcers with relative reduction in pressure ulcer incidence of 25%. Moderately strong evidence exists that use of high-protein nutritional supplements (24%–25% protein) improves pressure ulcer healing. Providing 30 to 35 kcal/kg and 1.25 to 1.5 g/kg of calories and protein daily has been shown to significantly improve pressure ulcer healing. However, provision of nutritional supplementation by tube-feeding to persons with pressure ulcers has not achieved positive results.

No evidence exists for use of supplemental vitamins or minerals (eg, vitamin A, E, C, zinc) in persons with pressure ulcers with no coexisting specific vitamin/mineral deficiency to improve pressure ulcer healing. Moderate evidence exists to support use of high-calorie, high-protein nutritional supplements containing arginine to promote pressure ulcer healing in older adults in general (significant decrease in ulcer size at 8 weeks favoring supplement), older adults who do not have preexisting malnutrition (significant decrease in ulcer size and PUSH score over 8 weeks favoring supplement), and persons with SCI (10 vs 21 weeks to healing favoring supplement). Persons with pressure ulcers or at risk of developing pressure ulcers who also demonstrate malnutrition should have a standard nutritional assessment to identify deficits and nutrition support as indicated. A daily multivitamin and mineral supplement that provides recommended daily allowances of vitamins and minerals is recommended for persons with suspected nutritional deficiencies.

PREVENTION

Pressure ulcer prevention involves scheduled turning and repositioning programs, use of support surfaces to reduce/relieve pressure, nutritional support (discussed earlier), and general skin care. Prevention interventions should be implemented for persons at risk for pressure ulcer development and those with existing pressure ulcers as part of the treatment plan.

Patients at risk for pressure ulcers that are unable to move independently should be placed on scheduled repositioning programs. The recommended time interval for full change of position or turning while in bed is every 2 hours, depending on the individual patient profile and the use of support surfaces. Pressure-reducing support surfaces (eg, foam, air, gel-filled mattress overlays, low-air-loss therapy devices) may reduce the frequency of repositioning required in some patients. One controlled clinical trial evaluating the effects of four different turn intervals in conjunction with standard hospital mattress versus a pressure reduction support surface found differences in pressure ulcer incidence. The four turn intervals evaluated were 2 hours on standard mattress (2 hours), 3 hours on standard mattress (3 hours), 4 hours on pressure-reducing surface (4 hours), 6 hours on pressure-reducing surface (6 hours) and a control group with usual care (no specified turning schedule). Over 4 weeks, 838 geriatric nursing home residents were evaluated. Although there was no difference in the incidence of stage 1 pressure ulcers or erythema, stage 2 pressure ulcer incidence was significantly decreased in the 4-hour group compared to all other groups (3% compared to 20%, no turns; 14.3%, 2 hours; 24.1%, 3 hours; and 15.9%, 6 hours). Others have shown similar findings related to use of a 4-hour repositioning program in conjunction with use of viscoelastic support surfaces.

Repositioning patients to avoid placing pressure on bony prominences, in particular the malleolus and trochanter is important. To avoid placing pressure on the trochanter and outer malleolus, position the patient at a 30-degree side-lying position (eg, 30-degree angle to the support surface) instead of the commonly used 90-degree side-lying position, which increases tissue compression over the trochanter and malleolus. Maintain the head of the bed at the lowest degree of elevation consistent with medical conditions, and limit the amount of time the head of the bed is elevated. This will decrease exposure of the sacral area to shearing and friction forces that may predispose to DTI. Use of footboards and pillows under the lower legs to prevent sliding and to maintain position, is also helpful in reducing shear effects on the skin when in bed.

There are techniques to make turning patients easier and less time consuming. Turning sheets, draw sheets, and pillows are essential for passive movement of patients in bed. Turning sheets are useful in repositioning the patient to a side-lying position, and draw sheets are used for pulling the patient up in bed and help to prevent dragging the patient's skin over the bed surface. Pillows should be used to position patients with minimal tissue compression between the medial knees, the medial malleolus, and the heels. Place pillows between the knees, between the ankles, under the heels as well as behind the back, and under the arms for comfort.

Use of real-time pressure mapping systems has been used to improve frequency of repositioning activities by caregivers. Pressure mapping systems may have a positive impact on pressure ulcer development because the duration and amount of pressure over specific bony prominences are displayed for caregivers allowing for more accurate offloading of bony prominences.

Similar approaches are useful for patients in chairs. Full-body change of position involves standing the patient and resitting them in the chair. Observation of the patient when sitting is also important, because patients who slide out of the chair are at high risk for shear or friction injury. Use of footstools and the foot pedals on wheelchairs and appropriate 90-degree flexion of the hip (may be achieved with pillows, special seat cushions, or orthotic devices) can help prevent chair sliding. Attention to proper alignment and posture is essential. Individuals at risk for pressure ulcer development should avoid uninterrupted sitting in chairs and should be repositioned every hour. The rationale behind the shorter time is extremely high pressure generated on the ischial tuberosities and sacrum in the seated position. When possible, individuals who are able should be taught to shift weight every 15 minutes while seated.

Repositioning for preventing pressure ulcers at the heel location involves completely offloading the heel using suspension devices or pillows. The goal is to keep the heels free of all pressure or "float" the heels. Elevating the lower leg and calf with pillows or suspension devices spreads the pressure to the lower legs and the heel is no longer subjected to pressure. Heel suspension devices are preferable for long-term use over pillows as it can be difficult for patients to keep their legs on pillows over longer time frames.

Reactive or active support surfaces should be initiated for beds and chairs of persons determined at risk for developing pressure ulcers. Reactive support surfaces are powered or nonpowered surfaces with the ability to only change load distribution properties in response to an applied load. Reactive surfaces are designed to reduce pressure ulcer development by deforming in response to loading with the goal of immersion and envelopment to reduce the deformation caused by pressure over the bony prominence. Active support surfaces are powered surfaces that produce alternating pressure through mechanical means and have the ability to change load distribution properties with or without an applied load. Active surfaces are designed to reduce pressure ulcer development by periodically shifting the areas of support on anatomic locations so that deformation is

not sustained over one area. In general, active support surfaces are recommended for persons at higher risk for pressure ulcer development when frequent repositioning is not possible.

Use of support surfaces instead of standard hospital mattresses results in relative reduction in pressure ulcer incidence of 60%. Additionally, clinicians should advocate for use of support surfaces in the operating room to reduce postoperative pressure ulcer incidence. Use of reactive support surfaces such as mattress overlays (eg, foam, gel, or alternating air pads) or active surfaces such as low-air-loss therapy is appropriate for many patients at risk for pressure ulcers or with stage 1 or 2 pressure ulcers. However, for persons with existing pressure ulcers, use of air-fluidized therapy or low-air-loss therapy may improve healing rates. One retrospective, multisite, comparison study showed faster healing of existing pressure ulcers with air-fluidized therapy compared to both pressure reduction support surfaces and low-air-loss therapy (mean healing rate of 5.2 cm^2/week for the air-fluidized surface group compared to 1.5 cm^2/week for pressure reduction support surface group and 1.8 cm^2/week for low-air-loss therapy group). In addition, the odds of showing improvement in pressure ulcers are more than five times greater when air-fluidized therapy and 4-hour repositioning is implemented compared to alternating air mattresses and 2-hour repositioning in hospitals. Although air-fluidized therapy increased the odds of ulcer improvement, only 12% of hospitalized patients achieved healing of the largest pressure ulcer. In nursing home residents, a randomized, controlled trial suggests that pressure ulcer areas decrease three times faster when low-air-loss beds are used compared with conventional care. Additional controlled trials are needed to define the optimal repositioning schedules for patients on a variety of support surfaces.

Providing topical preparations or fabrics/linens (silk or noncotton blends) to eliminate or reduce the surface tension between the skin and the bed linen or support surface will assist in reducing friction-related injury. Use of appropriate techniques when moving patients so that skin is not dragged across linens will lessen friction-induced skin breakdown. Patients who exhibit voluntary or involuntary repetitive body movements (particularly of the heels or elbows) require stronger interventions. Use of a protective film, such as a transparent film dressing or a skin sealant; a protective dressing, such as a thin hydrocolloid; or protective padding will help to eliminate the surface contact of the area and decrease the friction between the skin and the linens. Even though heel, ankle, and elbow protectors do nothing to reduce or relieve pressure, they can be effective aids against friction. Posthip fracture patients are especially vulnerable to heel ulcers. Elevation of the heel off the bed surface is a useful preventive measure.

Use of prophylactic silicone foam dressings over bony prominences has demonstrated significantly decreased heel (3% vs 12% favoring dressing) and sacral (1% vs 5% favoring dressing) pressure ulcers among critical care patients with the number needed to treat of 10 to prevent any ulcer. Similar findings for preventing heel ulcers among residents in long-term care facilities by prophylactically applying a polyurethane foam hydrocellular dressing have been shown. Use of prophylactic silicone foam, hydrocolloid, foam, or silicone gel dressings around medical devices has also been shown to decrease pressure ulcer development related to medical devices including tracheostomy tubes, nasal intubation tubes, and nasal continuous positive airway pressure (CPAP) devices.

General skin care should include routine skin inspection, incontinence assessment and management, and skin hygiene interventions to maintain skin health. Routine skin inspection should occur daily with particular attention to bony prominences. Reddened areas should not be massaged. Massage can further impair the perfusion to the tissues. The skin should be evaluated for dryness and cracking, and use of moisturizers can be helpful. Attention should also be focused on gentle handling to prevent skin tears. Incontinence assessment and management with scheduled toileting or prompted voiding programs and for those unresponsive to these programs check and change schedules are important. Prompt cleansing after incontinent episodes with warm water and gentle cleansers and use of protective ointments and creams help maintain perineal skin health. **Table 52-3** presents general prevention interventions directed at risk factors for pressure ulcer development.

TABLE 52-3

PRESSURE ULCER PREVENTION INTERVENTIONS

RISK FACTOR	PREVENTION INTERVENTONS
Immobility	Scheduled repositioning programs, use of pressure reduction/relief surfaces in bed and chairs.
Inactivity, limited mobility	Use of trapeze bars for self-movement in bed, encourage ambulation, rehabilitation as appropriate.
Decreased sensory perception	Scheduled repositioning programs, verbal reminders to move/reposition, use of pressure reduction/relief surfaces in bed and chairs.
Malnutrition	Nutritional assessment to determine deficits, nutritional supplementation (high protein), and daily multivitamin if indicated and appropriate to goals.
Excess moisture and incontinence	Scheduled toileting or prompted voiding programs if responsive, routine check and change programs with pads and adult briefs for persons who do not respond to scheduled toileting or prompted voiding, use of skin creams and ointments for protection from moisture.
Friction and shearing	Use of trapeze bars for those with upper body strength, turn sheets and draw sheets for moving in bed, use of cornstarch or lubricants to limit friction between surfaces, thin film or dressings, and pads over bony prominences subject to friction, use of footboards to help prevent sliding while in bed.
Dry skin	Use warm water, gentle cleansers, and limited force for cleansing when soiled and for routine bathing, lubricate and moisturize dry skin, inspect skin daily paying particular attention to bony prominences, avoid massage of reddened areas.

SPECIAL ISSUES

Some patients will benefit most from a palliative care approach. Palliative pressure ulcer care means that the goals are comfort and limiting the extent or impact of the pressure ulcer but without the intent of healing. Palliative care may be indicated for terminally ill patients such as those with end-stage cancer or in the terminal stages of other diseases. Institutionalized older adults with multiple comorbidities or older adults with severe functional decline may also benefit from palliative care. Palliative pressure ulcer care includes adequate debridement of necrotic tissue, identification and treatment of infection, provision of moist wound healing with management of exudate and odor, pain management, and prevention interventions. Prevention should still consist of use of reactive or active support surfaces and attention to scheduled repositioning, although time frames may be adjusted or lengthened to ease the burden on the patient. Providing pain medication 30 to 40 minutes prior to repositioning activity and use of positioning devices may help those with pain on movement.

SUMMARY

Pressure ulcers are chronic wounds and as such, require patience and diligence by clinicians. Some pressure ulcers never heal, and most require long periods of treatment with slow progress. Partial-thickness stage 2 pressure ulcers are more likely to heal than full-thickness stage 3 or 4 pressure ulcers. In fact, stage 2 pressure ulcers are 5.2 times more likely to heal in 6 months than stage 4 pressure ulcers. Among nursing home residents, up to 75% of stage 2 wounds heal within 60 days, while only 17% or fewer full-thickness pressure ulcers heal in the same time period. The best healing rate reported even after 6 months of treatment is 59%. Thus, identification of persons at risk for developing pressure ulcers and aggressive prevention

interventions to actively avoid ulcer development are essential. Prevention includes screening for risk followed by risk assessment using standardized risk assessment tools to determine individual specific risk and implementing targeted prevention interventions based on identified risk factors. Scheduled repositioning programs, use of reactive and active support surfaces, assessment and management of nutrition, and use of prophylactic dressings are key prevention strategies. More research is needed to define optimal turning intervals for various support surfaces, to better elucidate the effect of nutrition interventions on both preventing pressure ulcers and healing pressure ulcers, and fully examine specific use of prophylactic dressings.

For those persons who do develop pressure ulcers, clinicians should provide appropriate treatment during early ulcer stages to capitalize on healing progress in the initial 3 months. Adequate, timely, and complete debridement of necrotic tissue, identification and treatment of infection and management of biofilm development, and providing a moist wound environment are the key tenets of appropriate pressure ulcer care. All preventive and therapeutic interventions, and progress of the ulcer, should be carefully documented in the medical record. Unfortunately, no intervention or combination of interventions has demonstrated the ability to completely eliminate pressure ulcers. Thus, even as we develop more refined and specific screening, detection, and prevention interventions, it is likely we will continue to see pressure ulcers in all health care settings. The information presented in this chapter should provide a foundation for developing a successful approach to both those at risk for pressure ulcer development and those with existing pressure ulcers.

FURTHER READING

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