Prevention and Treatment of Pressure Ulcers

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Pressure ulcers are complex chronic wounds for which no gold standard for prevention or treatment has yet been established. Several attempts at developing guidelines has been undertaken by different organizations. Pressure ulcers are devastating comorbidities for patients and difficult to prevent or manage. Whether or not pressure ulcers are preventable remains controversial. The strategy for prevention includes recognizing the risk, decreasing the effects of pressure, assessing nutritional status, avoiding excessive bed rest and prolonged sitting, and preserving the integrity of the skin. The principles of treatment of pressure ulcers include assessing severity, reducing pressure, friction and shear forces, optimizing local wound care, removing necrotic debris, managing bacterial contamination, and correcting nutritional deficits. **(J Am Med Dir Assoc 2006; 7: 46–59)**

Pressure ulcers are the visible evidence of pathological changes in the blood supply to dermal tissues. The chief cause is pressure, or force per unit area, applied to susceptible tissues. Muscle tissue, subcutaneous fat, and dermal tissue are differentially affected in that order. Comorbid conditions, especially those resulting in immobility, such as paralysis, or reducing tissue perfusion, such as hypoxia, greatly increase the risk of developing pressure ulcers.

Pressure is concentrated wherever weight-bearing points come in contact with surfaces. These weight-bearing points usually occur over a bony prominence. Tissues over a bony prominence ("hard sites") may differ in resistance to hypoxia or pressure compared to "soft sites" away from bone.[1] This may explain the frequency of pressure ulcer development in these sites. About 95% of pressure ulcers occur in the lower part of the body. The sacral and coccygeal areas, ischial tuberosities, and greater trochanteric areas account for the majority of pressure ulcer sites.[2] The sacrum is the most frequent site (36% of ulcers). The heel is the next most common site (30%), with other body areas each accounting for about 6% of pressure ulcers.[3] About 70% of all pressure ulcers occur in persons over the age of 65 years.[4]

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DOI: 10.1016/j.jamda.2005.10.004

PRINCIPLES OF PRESSURE ULCER PREVENTION

Pressure ulcers are increasingly described as an indicator of quality of care.[5,6] However, whether or not pressure ulcers are preventable remains controversial.[7] In surveys of prevention practices among hospitalized Medicare beneficiaries, there was no link between documentation of a quality indicator and the incidence of pressure ulcers. In a multicenter retrospective cohort study of 2425 patients aged 65 years and older discharged from acute care hospitals, 6 processes of care for prevention of pressure ulcers were evaluated, including use of daily skin assessment, use of a pressure-reducing device, documentation of being at risk, repositioning for a minimum of 2 hours, nutritional consultation initiated for patients with nutritional risk factors, and staging of pressure ulcer. In fact, older adults who had documentation of being at risk and/or who received a pressure-reducing device and/or were turned every 2 hours had a higher incidence of pressure ulcer development.[8]

Compliance with quality indicators in 16 nursing facilities in California was assessed after dividing the homes into those with the highest or the lowest quartile incidence of pressure ulcers. The homes in the lowest quartile had a 2.7% to 5.5% incidence of pressure ulcers while the high-quartile nursing facilities had a 16.6% to 29.8% incidence of pressure ulcers. Sixteen process of care quality indicators were assessed, including 10 specific to pressure ulcers. No differences in the pressure ulcer quality indicators derived from the Minimum Data Set was observed between nursing facilities with low and high pressure ulcer incidence. Moreover, there was no difference in direct clinical observation of processes of care between nursing facilities with low and high pressure ulcer incidence.[9]

In 20 facilities with Medicare-certified beds, 12 quality indicators were derived from expert opinion and data available in the Minimum Data Set. An intensive intervention included education, direct facility assistance, and multiple site visits by study personnel. The result of the intervention was positive; that is, the facilities showed improvement on 8 out of 12 quality indicators. The trial suggests that a nursing facility can improve documentation of care in relationship to pressure ulcers. However, there was no improvement in 4 of the 12 quality indicators. Most importantly, there was no improvement in the incidence of pressure ulcers despite the relatively intense intervention. No difference was observed in the proportion of low-risk residents who developed a pressure ulcer during their stay, or in the proportion of high-risk residents who developed a pressure ulcer during their stay.[10] These data suggest that development of a pressure ulcer may not be as tightly linked to quality of care processes as has been suggested.

Systematic efforts at education, heightened awareness of pressure ulcer prevention, and specific interventions by interdisciplinary wound teams suggest that a high incidence of pressure ulcers can be reduced. Over time, reductions in incidence of pressure ulcers of 25% to 30% have been reported.[11,12] The reduction may be transient, unstable over time, vary with changes in personnel, or occur due to random variation.[13] However, no trial has reported an elimination of pressure ulcers over time, and a "floor effect" for pressure ulcer incidence has been noted, despite aggressive measures for prevention.[14] These data confirm a growing body of evidence that severs the hypothesized link between pressure ulcer incidence and quality of care indicators. The data suggest that pressure ulcers can be but not always are measures of quality of care.

Pressure ulcers often occur in terminally ill patients where the goals of care may not include prevention or cure of a pressure ulcer.[15] In orthopedic patients or intensive care patients, the necessity for immobilization may preclude turning or the use of pressure-relieving devices. For most patients the opportunity to prevent pressure ulcers occurs early in the course of the illness. The strategy for prevention includes recognizing the risk, decreasing the effects of pressure, assessing nutritional status, avoiding excessive bed rest and prolonged sitting, and preserving the integrity of the skin. However, the effectiveness of some prevention measures has not been demonstrated.

Decrease Pressure, Friction, and Shear

In those patients at risk, the first preventive action is to reduce the effect of pressure, friction, and shear forces. The most commonly recommended method for reducing pressure is frequent turning and positioning. A 2-hour turning schedule for spinal-injury patients was deducted empirically in 1946.[16] The exact interval for optimal turning in prevention is unknown. The interval may be shortened or lengthened by host factors. In healthy older volunteers, intervals of 1 hour to 1.5 hours rather than the traditional 2-hour schedule were required to prevent skin erythema on a standard mattress.[17] Turning the patient to relieve pressure may be difficult to achieve despite best nursing efforts and is very costly. Despite commonsense approaches to turning, positioning, and improving passive activity, no published data support the view that pressure ulcers can be completely prevented by passive positioning.[18,19]

Because of the limitations and cost of turning schedules, a number of pressure-reducing devices have been developed for prevention of pressure injury. In a randomized controlled trial in 838 high-risk nursing home residents, a turning schedule of every 4 hours in combination with the use of a pressure-reducing viscoelastic mattress reduced the number of pressure ulcers significantly compared to a turning schedule of every 6 hours on a viscoelastic mattress, or a turning schedule of every 2 or every 4 hours on a non–pressure-reducing mattress.[20]

The theoretical goal is to reduce tissue pressure below capillary closing pressure of 32 mm Hg. Devices can be defined as pressure relieving (consistently reducing interface pressure below 32 mm Hg) or pressure reducing (less than standard support surfaces, but not below 32 mm Hg). The majority of devices are pressure reducing. Pressure-reducing devices can be further classified as static or dynamic. Static surfaces are stationary and attempt to distribute local pressure over a larger body surface. Examples include foam mattresses and devices filled with water, gel, or air. Dynamic devices use a power source to produce air currents and promote uniform pressure distribution over body surfaces. Examples include alternating pressure pads, air suspension devices, and air-fluidized surfaces.

When compared to a standard hospital mattress, a variety of pressure-reducing devices lower the incidence of pressure ulcers by about 60%.[21] The capability of devices to reduce pressure differs depending on body site. Sacral pressure reduction can be achieved in healthy volunteers by several devices. Three dynamic air support systems lower pressure at the trochanter compared to a conventional mattress. However, no device reduced pressure over the trochanter to physiological levels.[22,23] Few currently marketed devices, including air-fluidized beds, will consistently reduce heel pressure below minimal capillary pressure.[24] It is important to note that although some dynamic air mattresses and flotation systems can reduce pressure to near physiological levels, all benefit is lost if the head of the bed is elevated to 30 degrees, such as for tube feedings.[25]

The differences among devices remain confusing (Table 1). No statistically significant difference has been found between alternating pressure, constant low pressure, foam overlays, silicone overlays, or air- or water-filled devices.[21] Therefore, a pressure-reducing device should be selected on the basis of cost and ease of use. The cost of pressure-reducing devices varies considerably, with air-fluidized and low-air-loss systems the most expensive and static support overlays the least expensive. Dynamic devices are often noisy and disturbing to patients. Mechanical difficulties are frequent with all types of devices. The data also demonstrate that pressure ulcers develop in some patients in spite of the use of pressure-reducing devices. Overall, the data suggest that patients likely to develop a pressure ulcer should be treated with a pressure-

Table 1. *Medicare Classification of Pressure-Reducing Devices*

Classification	Medicare Code	Description
Group 1 devices		
Powered pressure-reducing mattress overlay systems	E0180	Pressure pad, alternating with pump or low air loss
Pressure pad	E0181	Pressure pad, heavy duty, alternating with pump, provides either sequential inflation or deflation of air cells, with cell height of 2.5 inches or greater
Dry pressure mattress	E0184	Density that provides adequate pressure reduction with a foam height of 5 inches or greater
Gel mattress overlay	E0185	Height of 2 inches or greater
Air mattress overlay, pump	E0186	Height of 5 inches or greater
Water mattress overlay	E0187	Height of 5 inches or greater
Sheepskin	E0188	Lambswool sheepskin pad, any size
Foam mattress	E0189	Base thickness of 2 inches or greater and peak height of 3 inches or greater if convoluted overlay (eg, egg crate), or an overall height of at least 3 inches if nonconvoluted overlay; foam of such density and other qualities that it provides adequate pressure reduction
Powered pressure-reducing bed	E0193	Powered air flotation bed (low air loss therapy) with at least 3 independent sections in which the air pressure is custom adjusted by restrictive manifolding that provides constant force equalization
Gel mattress	E0196	Height of 5 inches or greater
Air mattress overlay	E0197	Interconnected air cells having a cell height of 3 inches or greater that are inflated with an air pump
Water mattress overlay	E0198	Water pressure pad for mattress, standard mattress length and width filled height of 3 inches or greater
Nonpowered pressure-reducing mattress overlays	E0199	Dry pressure pad for mattress, standard mattress length and width, at least 3 inches
Group 2 devices		
Powered pressure-reducing mattress	E0277	Alternating pressure, low air loss, or powered flotation without low air loss, with an air pump or blower that provides either sequential inflation and deflation of the air cells or low interface pressure throughout the mattress; inflated cell height is 5 inches or greater
Nonpowered, advanced pressure-reducing overlay	E0371	Height and design of individual cells provides significantly more pressure reduction than a Group 1 overlay, total height of 3 inches or greater
Powered pressure-reducing mattress overlay	E0372	Standard mattress length and width, low air loss, powered flotation without low air loss, or alternating pressure, with an air pump or blower that provides either sequential inflation and deflation of the air cells, with inflated cell height 3.5 inches or greater
Nonpowered, advanced pressure-reducing mattress	E0373	Height and design of individual cells provides significantly more pressure reduction than a Group I overlay, with a total height of 5 inches or greater
Group 3 devices		
Air-fluidized bed	E0194	Device employing a circulation of filtered air through silicone-coated ceramic beads, creating the characteristics of fluid
Kinetic bed	EO194	Device that rotates the bed rather than the person

reducing device, although no device appears to be superior to another.

Assess Nutritional Status

Nutritional status has been thought to influence the incidence, progression, and severity of pressure sores.[26] Experimental studies in animal models suggest a biologically plausible relationship between undernutrition and development of pressure ulcers. When pressure was applied for 4 hours to the skin of both well-nourished animals and malnourished animals, pressure ulcers occurred equally in both groups. However, the degree of ischemic skin destruction was more severe in the malnourished animals. Epithelialization of the pressure lesions occurred in normal animals at 3 days post-injury, while necrosis of the epidermis was still present in the malnourished animals.[27] These data suggest that while pressure damage may occur independently of nutritional status, malnourished animals may have impaired healing after a pressure injury.

Table 2. *Epidemiological Association of Nutritional Markers With Development of a Pressure Ulcer*

First Author	Year	Setting	Associated With Presence of PU	Not Associated With Presence of PU
Allman[32]	1987	AC	Albumin	Weight, hemoglobin, TLC, nutritional assessment
Gorse[33]	1987	AC	Albumin	Nutritional assessment score
Inman[34]	1993	AC, ICU	Albumin (measured at 3 days)	Serum protein, hemoglobin, weight
Allman[35]	1986	AC	BMI, TLC	Albumin, TSF, arm circumference, weight loss, hemoglobin, nitrogen balance
Hargrink[36]	1998	AC, orthopedic		Nocturnal enteral feeding
Anthony[37]	2000	AC	Albumin <32 g/L	
Moolten[38]	1972	LTC	Albumin <35 g/L	
Pinchcofsky-Devin[39]	1986	LTC	Severe malnutrition	Mild-to-moderate malnutrition or normal nutrition
Berlowitz[40]	1989	LTC	Impaired nutritional intake	Albumin, serum protein, hemoglobin, TLC, BMI/weight
Bennett[41]	1989	LTC		Weight, BMI, weight gain
Brandeis[42]	1990	LTC	Dependency in feeding	BMI/weight, TSF
Trumbore[43]	1990	LTC	Albumin, cholesterol	
Breslow[44]	1991	LTC	Albumin, hemoglobin	Serum protein, cholesterol, zinc, copper, transferrin, body weight, BMI, TLC
Bergstrom[45]	1992	LTC	Dietary protein intake 93% of RDA vs 119%, dietary iron	Serum protein, cholesterol, zinc, copper, transferrin, weight, BMI, TLC
Ferrell[46]	1993	LTC		Albumin, serum protein, BMI, hematocrit,
Guralnik[47]	1988	Community		Albumin, BMI, impaired nutrition, hemoglobin

PU, Pressure ulcer; AC, acute care; LTC, long-term care; BMI, body mass index; TLC, total lymphocyte count; TSF, triceps skinfold thickness; ICU, intensive care unit; RDA, Recommend Daily Allowance.

Observational studies have suggested a relationship between pressure ulcers and undernutrition. At hospital admission, patients who are undernourished are twice as likely to develop pressure ulcers as non-undernourished patients.[28] In a long-term care setting, 59% of residents were diagnosed as undernourished on admission. Among these residents, 7.3% were classified as severely undernourished. Pressure ulcers occurred in 65% of these severely undernourished residents. No pressure ulcer developed in the mildly to moderately undernourished or well-nourished groups.[29]

The estimated percent intake of dietary protein predicted development of pressure ulcers in another long-term care setting. Patients with pressure ulcers ingested 93% of the recommended daily intake of protein compared with an intake of 119% of the recommended protein in the non–pressure ulcer group. Only dietary intake of protein was important in this study. The total dietary intake of calories or the calculated intake of vitamins A and C, iron, and zinc did not predict ulcer development.[30]

Impaired nutritional intake, defined as a persistently poor appetite, meals held due to gastrointestinal disease, or a prescribed diet less than 1100 kcal or 50 g protein per day, predicted pressure ulcer development in another long-term care setting.[31] However, no other nutritional variable, including albumin, serum protein, hemoglobin, total lymphocyte count, body mass index, or body weight, was univariately significant.

The association of pressure ulcers with traditional markers of nutritional status has been found in some, but not all of the studies (Table 2).[32–47] Furthermore, there does not appear to be a consistent relationship between any of the nutritional parameters and pressure ulcers. The association of nutritional markers and pressure ulcers is confounded by lack of adjustment for comorbidity or severity of illness.[48] Decreases in serum albumin may reflect the presence of inflammatory cytokine production or comorbidity rather than nutritional status. Physiological stress (such as surgical operations), cortisol excess, and hypermetabolic states reduce serum albumin even in the presence of adequate protein intake. The discordance of the associations reported in these observations suggests that serum albumin may act as an acute phase reactant.[49]

These observational studies suggest an association of undernutrtion and pressure ulcers. It is easy to hypothesize that severely undernourished persons with loss of body fat would be at higher risk of developing pressure ulcers. However, the epidemiological association of markers for undernutrition and

Table 3. *Nutritional Interventions in the Prevention of Pressure Ulcers*

First Author	Year	Setting	Intervention	Risk Reduction	95% CI
Delmi[51]	1990	Acute femur fracture	Standard diet vs Standard diet plus nutritional supplement	0.79	0.14 to 4.39
Hartgrink[36]	1998	Acute hip fracture	Standard diet vs Standard diet and overnight nasogastric tube feeding	0.92	0.64 to 1.32
Houwing[52]	2003	Acute hip fracture	Standard diet plus water placebo vs Standard diet plus 1 daily supplement (includes arginine)	59% vs 55%	NS
Bourdel- Marchasson[53]	2000	Critical illness, age >65 years	Standard diet vs Standard diet plus 2 oral supplements	0.83	0.70 to 0.99

95% CI, 95% confidence interval.

pressure ulcers is often not adjusted for comorbidy or other risk factors and may merely indicate that sicker patients are more likely to develop pressure ulcers. Despite this association, a causal relationship of poor nutritional status to pressure ulcers has not been established.[50] Both undernutrition and pressure ulcers frequently coexist in the same persons.

Results of trials of nutritional intervention in prevention of pressure ulcers has been disappointing (Table 3).[36,51–53] An observational study of hospitalized, critically ill patients given nutritional supplements suggests no effect on development of a pressure ulcer. Oral supplements were given to 33% of one group compared with 87% of another group. There was no difference in pressure ulcer incidence (26% vs 20%), pressure ulcer prevalence at discharge (15% vs 10%), mortality (16% vs 14%), length of stay (17.3 days vs 17.4 days), or nosocomial infections (26% vs 19%).[54]

In a prospective trial of supplemental nutritional products given to severely ill older patients, the intervention produced a small decrease in the frequency of pressure ulcers. The cumulative incidence of pressure ulcers was 41% in the nutritional intervention group versus 47% in the control group.[55] However, the subjects were assigned by wards and were not similar at baseline. The nutritional intervention group had a lower risk for developing pressure ulcers and was more independent.

The effect of overnight supplemental enteral feeding in patients with a fracture of the hip and a high pressure-sore risk score was evaluated in a randomized clinical trial. Of the 62 patients randomized for enteral feeding, only 25 tolerated their tube for more than 1 week, and only 16 tolerated their tube for 2 weeks. Comparison of the actually tube-fed group (n = 25 at 1 week, n = 16 at 2 weeks) and the control group showed 2 to 3 times higher protein and energy intake ($P < .0001$), and a significantly higher total serum protein and serum albumin after 1 and 2 weeks in the actually tube-fed group (all P values $< .001$). However, the development of pressure ulcers and severity of the ulcers were not significantly influenced in the actually tube-fed group. It is possible that the lack of effect on supplemental enteral feeding was because of poor tolerance of the feedings. No difference was found for the total serum protein or serum albumin after 1 and 2 weeks.[36]

Provide Good Skin Care

Several studies indicate that incontinence produces a 5-fold risk of pressure ulcer development,[56] but fecal incontinence was not separated from urinary incontinence. When urinary incontinence is looked at separately, there has been no independent association between pressure ulcers and urinary incontinence. Fecal incontinence is much more important.[57–59]

An interventional trial explored the provision of exercise and incontinence care every 2 hours for 32 weeks by a research staff compared with usual nursing home staff care. The intervention subjects improved in urinary and fecal incontinence, physical activity, and skin wetness compared with the control group. However, despite these improvements, no difference in the incidence rate of pressure ulcers occurred between groups.[60]

These data imply that skin health quality indicators may not be improved or pressure ulcers prevented even in the presence of adequate staffing resources. A Foley catheter placed in an elderly patient solely for incontinence is probably a greater risk than that of diapering and incontinence itself.

PRINCIPLES OF PRESSURE ULCER TREATMENT

Pressure ulcers are extremely difficult to heal. Once developed, this type of chronic wound is very resistant to any known medical therapy. Pressure ulcers fail to proceed through the normal sequence to produce anatomical or functional integrity described in healing acute wounds.[61] Fibroblasts and epithelial cells from normal skin grow rapidly in skin tissue cultures, covering 80% of in vitro surfaces within the first 3 days. In contrast, biopsy specimens from pressure ulcers usually do not grow until much later, covering only 70% of surfaces by 14 days.[62] There is no hemorrhage in chronic wounds and thus more difficulty bringing wound factors into contact with tissue. Platelet release and fibrinolytic activity are diminished. Finally, there are complex polymicrobial colonizations that are poorly understood.[63]

As few as 13% of pressure ulcers heal by 2 weeks in acute hospital settings.[64] In long-term care settings, the rate of healing depends on initial stage of the pressure ulcer. Healing rates for Stage III pressure ulcers (see Assess the Pressure

Ulcer for definition) may be as high as 59% at 6 months, but other patients require a treatment duration of up to 1 year. Only one third of Stage IV pressure ulcers heal after 6 months of therapy but one half of patients admitted with pressure ulcers have died during this time period.[65] Thus, prevention offers the best opportunity for management.

The principles of treatment of pressure ulcers include assessing severity; reducing pressure, friction, and shear forces; optimizing local wound care; removing necrotic debris; managing bacterial contamination; and correcting nutritional deficits.

Assess the Pressure Ulcer

Several differing scales have been proposed for assessing the severity of pressure ulcers. The most common staging, recommended by a National Pressure Ulcer Advisory Panel Task Force and Omnibus Reconciliation Act nursing home guidelines, derives from a modification of the Shea Scale.[66] Under this schematic, pressure ulcers are divided into 4 clinical stages.

The first response of the epidermis to pressure is hyperemia. Blanchable erythema occurs when capillary refilling occurs after gentle pressure is applied to the area. Nonblanchable erythema exists when pressure of a finger in the reddened area does not produce a blanching or capillary refilling. A **Stage I** pressure ulcer is defined by nonblanchable erythema of intact skin. Nonblanchable erythema is believed to indicate extravasation of blood from the capillaries. A Stage I pressure ulcer always understates the underlying damage since the epidermis is the last tissue to show ischemic injury. Diagnosing Stage I pressure ulcers in darkly pigmented skin is problematic, and may present as a persistent red, blue, or purple hue.[67]

Stage II ulcers extend through the epidermis or dermis. The ulcer is superficial and presents clinically as an abrasion, blister, or shallow crater. **Stage III** pressure ulcers are full-thickness skin loss involving damage or necroses of subcutaneous tissue that may extend down to, but not through, underlying fascia. The ulcer presents clinically as a deep crater with or without undermining of adjacent tissue. **Stage IV** pressure ulcers are full-thickness wounds with extensive destruction, tissue necrosis, or damage to muscle, bone, or supporting structures. Undermining and sinus tracts are frequently associated with Stage IV pressure ulcers. The sacrum, with 38% of all ulcers, remains the most common site for pressure ulcer occurrence. Stage I pressure ulcers are the most frequent ulcers (47%), followed by stage 2 ulcers (33%). The average number of ulcers per patient was 1.7.[68]

This staging system for pressure ulcers has several limitations. The primary difficulty lies in the inability to distinguish progression between stages. Pressure ulcers do not progress absolutely through Stage I to Stage IV, but may appear to develop from "the inside out" as a result of the initial injury. Healing from Stage IV does not progress through Stage III to Stage I, but rather heals by contraction and scar tissue formation. Since pressure ulcers heal by contraction and scar formation, "reverse staging" is inaccurate in assessing healing. Thus, improvement or deterioration between clinical stages cannot be determined. Clinical staging is inaccurate unless all eschar is removed, since the staging system only reflects depth of the ulcer. No single measure of wound characteristics has been useful in measuring healing.[69] The Pressure Ulcer Status for Healing (PUSH) tool was developed and validated by the National Pressure Ulcer Advisory Panel to measure healing of pressure ulcers. The tool measures 3 components, size, exudate amount, and tissue type, to arrive at a numerical score for ulcer status. In clinical development and validation studies, the PUSH tool adequately assesses ulcer status and is sensitive to change over time.[70,71] The PUSH tool is shown in Figure 1.

Relieve Pressure, Friction, and Shear

Two hospital studies demonstrated enhanced healing of pressure ulcers with air-fluidized beds, and 1 home-based study showed no significant difference. In a nursing home, study a low-air-loss bed was more effective in treating pressure ulcers compared to a foam overlay. A total of 6 randomized, controlled trials suggest that air-fluidized beds and low-air-loss beds improved healing rates of pressure ulcers.[72] However, no trial provides definitive evidence that one type of device is superior to a comparable device. Pressure reduction is also important when the patient is sitting in a chair or wheelchair. Empirical turning and positioning of patients is thought to be important although may not always be possible.

Optimize Local Wound Therapy

For centuries, wounds have been dressed to protect the wound from a harmful external environment. The traditional acute wound dressing was an "absorptive cover," which produced a dry wound surface. The principal function of a chronic wound dressing is to provide a moist healing environment.

Moist wound healing allows experimentally induced wounds to resurface up to 40% faster than air-exposed wounds.[73] Wound fluid is thought to contain a variety of growth factors such as interleukin-1, epidermal growth factor, and platelet-derived growth factor-beta, which may enhance healing.[74] A moist environment may maintain a normal electrical voltage gradient across the wound necessary for epithelial migration.[75] Wound fluid under occlusive dressings may increase bacterial overgrowth, stimulating epidermal migration.[76] Wound exudate in chronic ulcers has been found to be an excellent medium for fibroblast stimulation.[77] Removal of this medium by aggressive scrubbing or drying has been shown to be detrimental.

The concept of a moist wound environment led to development of "occlusive dressings." The term "occlusive" describes the inability of a dressing to transmit moisture vapor from the wound to the external atmosphere. The degree to which dressings dry the wound can be measured by the moisture vapor transmission rate (MVTR). An MVTR of less than 35 g of water vapor per square meter per hour is required to maintain a moist wound environment. Woven gauze has an MVTR of 68 $g/m^2/hr$ and impregnated gauze has an MVTR of 57 $g/m^2/hr$. In comparison, hydrocolloid dressings have an MVTR of 8 $g/m^2/hr$.[78]

Occlusive dressings can be divided into broad categories of

Patient Initials:_____ Study ID#:

Study Day #: _____ Date:

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

	0 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	
Length × 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** >24.0 cm^2	**Sub-score**
Exudate amount	**0** None	**1** Light	**2** Moderate	**3** Heavy			**Sub-score**
Tissue type	**0** Closed	**1** Epithelial tissue	**2** Granulation tissue	**3** Slough	**4** Necrotic tissue		**Sub-score**
							Total Score

Length × Width: Measure the greatest length (head to toe) and the greatest width (side to side) using a centimeter ruler. Multiply these 2 measurements (length times 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).

Fig. 1. *Pressure Ulcer Scale for Healing (PUSH Tool) Version 3.0. Reprinted with permission from NPUAP. Copyright 2003, NPUAP.*

Table 4. *Comparison of Occlusive Wound Dressings*

	Moist Saline Gauze	Polymer Films	Polymer Foams	Hydro-gels	Hydro-colloids	Alginates, Granules	Biomembranes
Pain relief	+	+	+	+	+	±	+
Maceration of surrounding skin	±	±	–	–	–	–	–
O_2 permeable	+	+	+	+	–	+	+
H_2O permeable	+	+	+	+	–	+	+
Absorbent	+	–	+	+	±	+	–
Damage to epithelial cells	±	+	–	–	–	–	–
Transparent	–	+	–	–	–	–	–
Resistant to bacteria	–	–	–	–	+	–	+
Ease of application	+	–	+	+	+	+	–

+, positive; –, negative; ±, equivocal.
Sources: Adapted from Helfman et al[79] and Witkowski and Parish.[80]

polymer films, polymer foams, hydrogels, hydrocolloids, alginates, and biomembranes. Each has several advantages and disadvantages. The available agents differ in their properties of permeability to water vapor and wound protection. Understanding these differences is the key to planning for wound management in a particular patient.

Comparative qualities among available agents are shown in Table 4. Most of the occlusive dressings offer pain relief. Only absorbing granules fail to reduce pain. Polymer films are impermeable to liquid but permeable to gas and moisture vapor. Because of low permeability to water vapor, these dressings are not dehydrating to the wound. Nonpermeable polymers such as polyvinylidine and polyethylene can be macerating to normal skin. Polymer films are not absorptive and may leak, particularly when the wound is highly exudative. Most films have an adhesive backing that may remove epithelial cells when the dressing is changed. Polymer films do not eliminate dead space and do not absorb exudate.

Hydrocolloid dressings are complex layered dressings. They are impermeable to moisture vapor and gases and are highly adherent to the skin. Their adhesiveness to surrounding skin is higher than some surgical tapes, but they are nonadherent to wound tissue and do not interfere with epithelization of the wound. The adhesive barrier of a hydrocolloid dressing can be overcome in highly exudative wounds. Excessive exudate may be overcome with an absorptive dressing such as calcium alginate.

A meta-analysis of 5 clinical trials comparing a hydrocolloid dressing with a dry dressing demonstrated that treatment with a hydrocolloid dressing resulted in a statistically significant improvement in the rate of pressure ulcer healing (odds ratio 2.6).[81] Topical application of collagen showed no significant differences in healing compared to a hydrocolloid. Collagen was more expensive and offered no major benefits to patients otherwise eligible for hydrocolloid treatment.[82]

Hydrogels are 3-layer hydrophillic polymers that are insoluble in water but absorb aqueous solutions. They are poor bacterial barriers and are nonadherent to the wound. Because of their high specific heat, these dressings are cooling to the skin, aiding in pain control and reducing inflammation. Most of these dressings require a secondary dressing to secure them to the wound.

Alginates are complex polysaccharide dressings that are highly absorbent in exudative wounds. This high absorbency is particularly suited to exudative wounds. Alginates are nonadherent to the wound, but if the wound is allowed to dry, damage to the epithelial tissue may occur with removal. Alginates can be used under a number of dressings to control exudate, including hydrocolloids.

Hydrocolloid dressings and biomembranes do not allow bacteria on the surface of the dressing to penetrate to the wound. Biomembranes are tissue-derived dressings designed to cover the wound and provide potential wound-healing factors. The biomembranes are very expensive and not readily available.

The dressings differ in the ease of application. This difference is important in pressure ulcers in unusual locations, or when considering for home care. Dressings should be left in place until wound fluid is leaking from the sides, a period of days to 2 weeks.

Saline-soaked gauze that is not allowed to dry is an effective wound dressing. When moist saline gauze has been compared to occlusive-type dressings, healing of pressure ulcers has been similar with both dressings.[83–85] The use of hydrocolloid dressings has been shown to be more cost effective than traditional dressings, primarily because of a decrease in nursing time for dressing changes.[86]

Acute wound healing proceeds in a carefully regulated fashion that is reproducible from wound to wound. A number of growth factors have been demonstrated to mediate the healing process, including transforming growth factor alpha and beta, epidermal growth factor, platelet-derived growth factor, fibroblast growth factor, interleukin 1 and 2, and tumor necrosis factor alpha. Accelerating healing in chronic wounds by using these acute wound factors is attractive. In pressure ulcers, platelet-derived growth factor failed to produce complete healing,[65] although improved time-to-closure of wounds has been shown with platelet-derived growth factor BB and basic fibroblast growth factor (Table 5).[89,93] The development of wound-healing factors is still in infancy but shows great promise.

Vacuum-assisted closure has been used in both acute and chronic wounds. Only 2 randomized, controlled trials in pres-

Table 5. *Growth Factors and Pressure Ulcers*

Growth Factor	N	Healing Rate Active	Healing Rate Placebo	Measure
rhPDGF[87]	124 in 4 groups	23%	0%	Complete closure
rhPDGF [88]	41 in 3 groups	No difference	No difference	Time to 50% closure or ulcer volume
rbFGF[89]	50 in 8 groups	Greater closure ($P < .05$)		>70% closure
rIL 1[90]	26 in 4 groups	No difference	No difference	Closure
rhGM-CSF and rbFGF[91]	61 in 4 groups	No difference	No difference	>85% closure
rNGF[92]	36 in 2 groups (heel only)	44%	5%	Closure

rhPDGF, Recombinant human platelet-derived growth factor; rbFGF, recombinant basic fibroblast growth factor; rIL 1, recombinant human interleukin-1; rhGM-CSF, granulocyte-macrophage colony-stimulating factor; rNGF, topical nerve growth factor.

sure ulcers have been reported. A total of 22 patients with 35 pressure ulcers were randomized to the vacuum-assisted closure device or a system of wound gel products for 6 weeks. Two patients in the vacuum-assisted closure group and 2 patients in the wound gel group healed completely. There was no difference in reduction in ulcer volume between groups.[94] Vacuum-assisted closure was compared to gauze moistened with Ringer's solution in a small trial of pressure ulcer treatment. Time to reach 50% of the initial wound volume was 27 days in the vacuum-assisted group and 28 days in the wet gauze–treated group.[95]

Remove Necrotic Debris

Necrotic debris increases the possibility of bacterial infection and delays wound healing in animal models.[96] This delay in healing results from slow removal of debris required by phagocytosis. Although widely recommended, it remains unclear whether wound debridement is a beneficial process that results in a greater frequency of complete wound healing.[97] There are no studies that compared debridement with no debridement as the control in wound healing. The use of debridement can result in a shorter time to a clean wound bed in anticipation of surgical therapy.

Options for debridement include sharp surgical debridement, mechanical debridement with dry gauze dressings, autolytic debridement with occlusive dressings, or application of exogenous enzymes. Surgical sharp debridement produces the most rapid removal of necrotic debris and is indicated in the presence of infection. Surgical or mechanical debridement can damage healthy tissue or fail to completely clean the wound. Mechanical debridement can be easily accomplished by letting saline gauze dry before removal, but may produce pain with removal. Re-moistening of gauze dressings in an attempt to reduce pain can defeat the debridement effect.

Thin portions of eschar can be removed by occlusion under a semipermeable dressing. Enzymatic debridement can dissolve necrotic debris but possible harm to healthy tissue is debated. Penetration of enzymatic agents is limited in eschar and requires either softening by autolysis or cross-hatching by sharp incision prior to application. Both autolytic and enzymatic debridement require periods of several days to several weeks to achieve results.

Enzymes available in the United States for topical debridement include collagenase, papain/urea, and a papain/urea/chlorophyll combination. A trial in 21 patients with pressure ulcers found a greater reduction in necrotic tissue using papain/urea (95.4%) compared with collagenase (35.8%) at 4 weeks, but the rate of complete healing was not different between groups.[98]

Five trials have not shown that enzymatic agents increased the rate of complete healing in chronic wounds compared to control treatment.[97] One trial showed an increase in wound size with both collagenase and the control treatment, but the increase was significantly less in the enzyme-treated group. Only 1 trial out of 4 that compared a hydrogel with a control treatment found a statistically significant difference between treatments. The single favorable trial suggested a small benefit from treatment with a hydrogel compared with a hydrocolloid dressing. In a single trial comparing different hypdrogels, no statistically significant difference was seen between the 2 hydrogels.

Trials of other debridment agents have shown mixed results. Three trials of detranomer polysaccharide found a statistically significant difference compared to control, while 2 trials found the control treatment more effective. A hydrogel significantly reduced necrotic wound area compared with dextranomer polysaccharide paste in one trial, but not in another. Dextranomer polysaccharide was not better than an enzymatic agent in 2 trials. There are no randomized, controlled trials using the papain/chlorophyll combination.

Manage Bacterial Contamination

Biological vectors destroy tissue. Eleck[99] demonstrated that normal skin flora in numbers greater than 10^5 organisms/mL produces local disease in intact skin. In damaged skin, fewer organisms are required to produce infection. Skin grafts and flaps will not heal when greater than 10^5 organisms of certain species of bacteria are present.[100] However, chronic wounds do not appear to follow these rules. Greater than 10^5 numbers of organisms may persist for months or years in chronic wounds without apparent clinical effect. Quantitative microbiology alone is a poor predictor of clinical infection in chronic wounds.[101]

Colonization with bacteria is common and unavoidable. All pressure ulcers become colonized, usually with skin organisms, followed in 48 hours by gram-negative bacteria. The

Table 6. *Nutritional Interventions in the Treatment of Pressure Ulcers*

First Author	Setting	Intervention	Outcome
Breslow[115]	Long-term care	24% protein vs 14% protein enteral feeding	-4.2 cm^2 vs -2.1 cm^2 decrease in surface area
Chernoff[112]	Long-term care	1.8 g/kg protein vs 1.2 g/kg protein enteral feeding	RR 0.11 (95% CI 0.01 to 1.70)
Henderson[116]	Long-term care	1.6 times basal energy expenditure,1.4 g of protein per kilogram per day	65% PU at onset; 61% prevalence at 3 months
Langkamp-Henken[117]	Long-term care	Arginine 0 gm vs arginine 17 gm	No difference in healing
ter Riet[118]	Long-term care	Vitamin C 10 mg vs 1000 mg	RR 0.81 (95% CI 0.50 to 1.30)
Taylor[119]	Acute surgical patients	Vitamin C large dose vs none	84% vs 43% (control) reduction surface area at 30 days
Norris[120]	Acute hip fracture	Zinc	No difference

RR, Relative risk (95% confidence intervals [CI]); PU, pressure ulcer.

presence of microorganisms alone (colonization) does not indicate an infection in pressure ulcers. The diagnosis of infection in pressure ulcers must be based on clinical signs—erythema, edema, odor, fever, or purulent exudate. Foul odor is a particularly important clinical sign, usually signifying anaerobic organisms.[102] Often it is difficult to determine the presence of an infection in a pressure ulcer.

A trial of empiric topical antibiotics is indicated in pressure ulcers failing to progress toward healing. The species of bacteria may make a difference. In worsening pressure ulcers, *Pseudomonas aeruginosa* and *Providencia* species were found in 88% and 34% of ulcers compared to 0% of stationary wounds and 7% of rapidly healing ulcers. *Peptococci, Bacteroides* species, or *Clostridia* were found in over half of worsening or stationary ulcers, but were absent in healing pressure ulcers. Staphylococci and enterococci were frequently isolated from rapidly healing ulcers.[103,104] Based on these findings, *P aeruginosa* and *Providenica* species should not be regarded as simple colonization.

When there is evidence of clinical infection, topical or systemic antimicrobials or antibiotics are required. Systemic antibiotics are indicated when the clinical condition suggests spread of the infection to the blood stream or bone. Several antimicrobial or antibiotic agents reduce bacteria in acute wounds without damaging the wound, including silver sulfadiazine 1% cream, combination antibiotic ointments, and propylene glycol.[105] Topical gentamicin and silver sulfadiazine have been shown to improve clinical appearance of infected wounds and may improve healing.[106,107] Iodine and thimerosal have been noted to increase pain and delay healing.[108] Infections with anaerobes may respond to topical metronidazole.[109]

Healing of pressure ulcers is enhanced under occlusive dressings even though this increases both the absolute number and variety of species of organisms. Despite an increase in numbers of bacteria, occlusive dressings very rarely cause a clinical infection. Hutchinson and McGuckin reviewed 36 studies comparing infection rates under occlusive dressings to gauze or impregnated gauze. Infection rates were 2.6% for occlusive dressings and 7.1% for non-occlusive gauze.[110]

The primary source of bacterial infections in pressure ulcers appears to be the result of supra-infection due to contamination. Therefore, protection of the wound from secondary contamination is an important goal of treatment. Evidence suggests that occlusive dressings protect against clinical infection although the wound may be colonized with bacteria. Lilly found that extracts of wound fluid under hydrocolloid dressings were capable of inhibiting growth of *P aeurginosa* and *S aureus* in vitro.[111]

Correct Nutritional Deficits

Nutritional status should be addressed in all patients, whether or not a pressure ulcer is present. However, very few specific interventions have been demonstrated to be effective in increasing healing of pressure ulcers.

Several studies suggest that dietary intake, especially of protein, is important in healing pressure ulcers. Greater healing of pressure ulcers has been reported with higher protein intake irrespective of positive nitrogen balance.[112] Optimum dietary protein intake in patients with pressure ulcers is unknown, but may be much higher than current adult recommendations of 0.8 g/kg/day. Half of chronically ill elderly persons are unable to maintain nitrogen balance at this level.[113] Increasing protein intake beyond 1.5 g/kg/day may not increase protein synthesis and may cause dehydration.[114] A reasonable protein requirement is therefore between 1.0 and 1.5 g/kg/day.

Several trials have attempted to increase healing or healing rate by the use of nutritional supplements (Table 6). In these trials, increased amounts of protein; enteral feeding; and the use of vitamin C, zinc, or arginine have not influenced the healing rate of pressure ulcers.

SURGICAL MANAGEMENT

Nowhere does the difference in pressure ulcers among younger spinal cord injury patients and elderly patients be-

come so pronounced as in discussing surgical management. Surgical closure of pressure ulcers results in a more rapid resolution of the wound. The chief problems are the frequent recurrence of ulcers and the inability of the frail patient to tolerate the procedure. The efficacy of surgical repair of pressure ulcers is high in the short term. The efficacy for long-term management has been questioned, even in younger patients.[121]

In a series of 40 patients selected for surgical closure of pressure ulcers, patients were divided into 3 subgroups. In nontraumatic, nonparaplegic elderly patients with a mean age of 73, 84% of surgically treated pressure ulcers were healed at discharge. Twelve percent of surgically treated patients had another pressure ulcer at discharge. Within 7.7 months, 40% of surgically treated pressure ulcers recurred and 69% of the patients had a pressure ulcer at a different site. In patients with traumatic paraplegia, 74% of operated pressure ulcers were healed at discharge and 76% of patients were free of pressure ulcers. Within 10.9 months, 79% of operated ulcers recurred, and 79% of patients had additional pressure ulcers. Only 21% of traumatic paraplegics and 31% of nontraumatic nonparaplegic elderly patients remained healed after muscle-flap coverage for pressure ulcers.[122] After 10 years of follow-up in 16 surgically treated patients, only 1 patient remained alive and free of pressure ulcers.[123]

A decision analysis demonstrated that myocutaneous flap procedures for Stage III pressure ulcers was favorable unless the success rate for surgery was less than 30% or the healing rate with medical therapy was less than 40%. The added cost for the procedure was estimated at $17,000 per treatment episode compared to medical therapy.[124]

In spinal cord injury patients the rate of surgical complications and recurrance is high. Surgical complications occurred in 40% of patients, and ulcer recurrence or development of a new ulcer occurred in 79.2% of patients.[125]

COMPLICATIONS OF PRESSURE ULCERS

The most common complications related to pressure ulcers are increased mortality, osteomyelitis, and sepsis.

Pressure ulcers have been associated with increased mortality rates in both acute and long-term care settings. Death has been reported to occur during acute hospitalization in 67% of patients who develop a pressure ulcer compared to 15% of at-risk patients without pressure ulcers.[85] Patients who develop a new pressure ulcer within 6 weeks after hospitalization are 3 times as likely to die as patients not developing a pressure ulcer.[126] In long-term care settings, development of a pressure ulcer within 3 months among newly admitted patients was associated with a 92% mortality rate, compared to a mortality rate of 4% among residents who did not subsequently develop a pressure ulcer.[127] Residents in a skilled nursing facility who had pressure ulcers experienced a 6-month mortality rate of 77.3%, while patients without pressure ulcers had a mortality rate of 18.3%.[128] Patients whose pressure ulcers healed within 6 months had a significantly lower mortality rate (11% vs 64%) than patients whose pressure ulcers did not heal.[129]

Despite this association with death rates, it is not clear how pressure ulcers contribute to increased mortality. Although several authors have found a 3-fold increase in mortality with the development of a new pressure ulcer, the severity of the pressure ulcer has not correlated with increased risk. Patients with Stage II pressure ulcers have been equally as likely to die as patients with Stage IV pressure ulcers.[130] In the absence of complications, it is difficult to imagine how Stage I or II pressure ulcers contribute to death. Pressure ulcers may not directly cause death, but the association with mortality may be due to their occurrence in otherwise frail, sick patients. Evidence for this is suggested in a prospective study of residents of 51 nursing homes, where pressure ulcers were associated with an increased rate of mortality but not with rate of acute hospitalization.[131]

A correction for the presence and severity of coexisting conditions can eliminate the association of pressure ulcers with death. In a prospective study of high-risk patients in an acute hospital setting, the development of a new pressure ulcer predicted death within 1 year. When the development of a pressure ulcer was entered into a multivariate risk analysis with measures of comorbidity, pressure ulcers were not independently associated with mortality. Independent risk factors for mortality in this study included weight loss reported in the 6 months before admission (relative risk [RR] 2.4), the admitting physician's estimate of life expectancy of less than 5 years (RR 2.1), and the Co-morbidity Damage Index score (RR 1.1). Global measures of disease severity and comorbidity and a history of weight loss are more important predictors of mortality at 1 year than development of a new pressure ulcer.[132]

Osteomyelitis is a frequent complication of pressure ulcers, reported in 38% of patients with infected pressure ulcers.[133] Diagnosis of contiguous osteomyelitis in pressure ulcers is difficult. Plain radiographs are unable to differentiate true osteomyelitis from pressure changes to bone.[134] Radionuclide studies, including technetium-99m and gallium-67, are sensitive but have a false-positive rate of 41%.[133] Computed tomography may be more useful, with a specificity of 90%, although the sensitivity is only 10%.[135] Needle biopsy of bone is the most useful single test, with a sensitivity of 73% and a specificity of 96%.[136]

Bacteremia from pressure ulcers is low, but probably underestimated. The incidence of bacteremia from a pressure ulcer is about 1.7 per 10,000 hospital discharges.[137] Sepsis is a serious consequence of pressure ulcers and a frequent cause of death. In a study of 21 patients with sepsis syndrome attributed to pressure ulcers, 76% had bacteremia that originated from the pressure ulcer. Overall, mortality was 48% and all patients over age 60 died despite empiric antibiotic treatment. In 5 patients, bacteremia persisted despite antibiotic treatment and resolved only after local debridement. However, the subjects in this report were selected because of the presence of sepsis and does not estimate the frequency of sepsis related to pressure ulcers in the general population.[138]

SUMMARY

Pressure ulcers are complex chronic wounds for which no gold standard for prevention or treatment has yet been estab-

lished. The strategy for prevention includes recognizing the risk, decreasing the effects of pressure, assessing nutritional status, avoiding excessive bed rest, and preserving the integrity of the skin. The principles of treatment of pressure ulcers include assessing severity; reducing pressure, friction, and shear forces; optimizing local wound care; removing necrotic debris; managing bacterial contamination; and correcting nutritional deficits.

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