

Issues and Dilemmas in the Prevention and Treatment of Pressure Ulcers: A Review

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Considerable dogma and rhetoric, rather than evidence-based results, have accompanied recommendations for the prevention and treatment of pressure ulcers. Therapy for pressure ulcers is generally empiric, based on anecdotal experience, or borrowed from the treatment of patients with acute wounds. The treatment of pressure ulcers is problematic because of multiple comorbidities of patients, the chronic duration of pressure ulcers, and often by the physician's relative unfamiliarity with treatment options. Issues and dilemmas in the prevention of pressure ulcers center around risk assessment, means of pressure relief, and nutritional support. Similar issues in the treatment of pressure ulcers include implementing pressure relief, nutritional support, local wound care, the best method of debridement, diagnosing infection, the use of topical growth factors, and surgical treatment. The accumulating data for the prevention and management of pressure ulcers permits an outline of clinical strategies.

PRESSURE ulcers are rare, affecting only about 0.5% of the total population. The distribution is clustered into two groups, peaking once in younger, mostly neurologically impaired persons and again in older persons. The cluster in the geriatric population accounts for about 70% of all pressure ulcers.

A guideline panel made approximately 85 specific recommendations on the basis of a careful literature review in 1993 (1). Only four level-A recommendations and ten level-B recommendations could be made. Few controlled trials existed to evaluate specific treatment modalities. Whereas the general principles of pressure ulcer management seem to be well established, controversy exists over specific areas of prevention and treatment. Many of the studies are compromised by their study design, the inability to control for confounding factors, or funding sources. Fortunately, newer data is becoming available to increase our understanding of management principles.

ISSUES AND DILEMMAS IN PREVENTION

Risk Assessment

Considerable effort has been directed toward risk assessment. In theory, persons who are at high risk for developing pressure ulcers can be identified, and increased effort can be directed to preventing ulcers in these persons. The classical scale is the Norton Score, developed in 1962 and still widely used (2). Patients are classified using five risk factors graded from one to four. Scores range from 5 to 20, with higher scores indicating lower risk. In the initial study, 48% of patients who scored less than 12 developed pressure ulcers, compared with only 5% of those who scored above 18. The generally accepted at-risk score is 14 or less, and patients with scores below 12 are at particularly high risk (3). The Norton score has been expanded into the Waterlow Scale in the United Kingdom (4). A commonly used risk assessment instrument in the United States is the

Braden Scale. This instrument assesses six items: sensory perception, moisture exposure, physical activity, mobility, nutrition, and friction/shear force. Each item is ranked from one (least favorable) to three or four (most favorable) for a maximal total score of 23. A score of 16 or less indicates a high risk (5). A comparison of the instruments is shown in Table 1.

Both the Norton Score and the Braden Scale have good sensitivity (73–92% and 83–100%, respectively) and specificity (61–94% and 64–77%, respectively), but both have poor positive predictive value (around 37% when the pressure ulcer incidence is 20%) (6). In populations with an incidence of pressure ulcers less than 20%, such as nursing homes, the same sensitivity and specificity would produce a positive predictive value of 2%. The Norton and Braden scales show a .73 Kappa statistic agreement among at-risk patients, with the Norton Score tending to classify patients as at risk when the Braden scale classified them as not at risk (7). The net effect of poor positive predictive value means that many patients who will not develop pressure ulcers will receive expensive and unnecessary treatment (8).

In clinical practice, risk assessment has been problematic. First, risk assessment is not universally applied (9). Less than half of the high-risk elderly persons admitted to acute care with a hip fracture had any sort of risk assessment performed (10). It is possible that resistance to implementing risk assessment models is due to recognition by clinicians that the instruments are inadequate. Second, no risk assessment study has demonstrated that persons identified as at-risk, or who have a plan of care based on risk assessment, are less likely to develop a pressure ulcer (10–14). Out of 36 hospitalized, severely ill patients assessed as at-risk, 4.4% (95% confidence interval 1.9–6.9) developed sores, whereas none of the 239 patients who were assessed as not being at-risk developed a sore. The occurrence of pressure ulcers in identified patients who received appropriate intervention suggests that there may be a "floor effect" to the prevention of pressure ulcers in severely ill patients (15). Overall, risk

Table 1. Comparison of Risk Assessment Instruments

Variable	Norton	Braden	Waterlow
Mobility	X	X	X
Moisture exposure	X	X	X
Physical activity	X	X	
General condition	X		X
Nutrition		X	
Appetite			X
Friction/shear force		X	
Sensory perception		X	
Mental status	X		
Skin type			X
Medication			X
Weight			X
Age			X
Gender			X
Other (e.g., disease)			X

Note: X = Scale contains the item.

assessment appears to be underutilized, inaccurate, and unrelated to pressure ulcer development.

In settings with a high incidence of pressure ulcers, the frequency of pressure ulcers has been reduced by careful attention. Usually these interventions involve education, heightened awareness, and organization of wound teams to deal with the problem incidence. Over time, reductions of 25% to 30% have been reported (16,17). The reduction may be transient, unstable with time, due to changes in personnel, or due to random variation (18). However, no trial has reported an elimination of pressure ulcers over time. Single, experimental interventions of pressure relief devices in carefully controlled studies have reduced the incidence of pressure ulcers but have not eliminated the problem. Para-

doxically, the level of nursing staffing has not been associated with the development of pressure ulcers (19).

Relieving Pressure

Pressure ulcers are thought to be due to unrelieved pressure. Relief of unrelenting pressure should eliminate the problem. Despite commonsense approaches to turning, positioning, and improving passive activity, no published data supports the view that pressure ulcers can be prevented by passive positioning (20,21).

Bioengineering advances have produced devices that can reduce tissue interface pressures over body surfaces. Table 2 summarizes clinical trials of the effect of pressure-reducing devices compared with a standard hospital mattress. The definition of a "standard" mattress varies around the world, making comparisons more difficult. The data indicate that several pressure-reducing devices are superior to a standard mattress, with a relative risk reduction of about 70%. When the absolute risk reduction can be calculated, the number needed to treat is as low as 3 (12). When compared with a "standard mattress," the net effect of these strategies has been demonstrated to be effective in reducing the incidence of pressure ulcers.

When various devices, such as an alternating air mattress or a low-air-loss bed, are compared among themselves rather than with a standard mattress, little difference between devices is apparent (Table 3). In high-risk patients in acute or long-term care settings, the incidence of pressure ulcers has been surprisingly high, despite allocation to any studied device.

Overall, the data suggest that patients likely to develop a pressure ulcer should be treated with a pressure-reducing device, although no device appears to be superior to an-

Table 2. Comparison of a Standard Mattress to Pressure-Reducing Devices in the Prevention of Pressure Ulcers

Population	Intervention	Results	Relative Risk (95% CI)
Prospective, randomized trial in acute care patients with femoral neck fracture (101)	DeCube mattress vs standard mattress	Incidence 25% on experimental mattress vs 64% control	0.35 (0.14-0.85)
Prospective, randomized trial in surgical and oncology patients (102)	Softfoam mattress vs standard mattress	Incidence 7% on treatment device vs 34% control	0.20 (0.09-0.45)
Prospective, non-random trial in long-term care (103)	Clinifloat foam mattress vs standard mattress	Incidence 2.0% on treatment vs 2.4% control	No difference between groups
Meta-analysis (12)	Standard mattress vs foam support	Four trials	Average risk reduction of 71% (57%-81%)
Prospective, randomized trial in orthopedic patients (104)	Bead bed vs standard mattress		0.32 (0.14-0.76)
Prospective, randomized controlled trial in an intensive care unit (105)	Air-filled support vs standard mattress	Incidence 0% on air mattress vs 37% on standard mattress	0.06 (0-0.99)
Prospective, randomized trial in acute care (106)	Alternating-pressure mattress vs water mattress vs standard mattress	Incidence 4% alternating vs 5% water vs 13% on standard mattress	0.35 (0.15-0.79) water mattress compared with standard
Meta-analysis (12)	Standard mattress vs constant low-pressure support	Three trials	Average risk reduction of 70%
Prospective, randomized trials in intensive care units (107,108)	Standard mattress vs kinetic turning beds	Two trials	No difference between groups
Prospective, randomized controlled trial in intensive care unit (109)	LAL vs standard mattress	Incidence 12.2% on LAL vs 51% on standard mattress	0.24 (0.11-0.53)
Quasi-experimental design in an intensive care unit (110)	Low-air-loss bed vs standard mattress	Incidence 19% on LAL vs 15% on standard mattress	No difference between groups

Notes: RR = relative risk; CI = confidence interval; LAL = low-air-loss; Constant Low Pressure support = static air-filled, water-filled, gel-filled, bead-filled, or silicone-filled supports, sheepskin, or foam supports. Meta-analysis results include only the studies meeting the author's inclusion criteria. Some trials included in the meta-analysis are listed separately. See reference for specifics.

Table 3. Comparison Among Pressure-Reducing Devices in the Prevention of Pressure Ulcers

Population	Intervention	Results	Relative Risk (95% CI)
Prospective, nonrandom, controlled-trial intensive care patients (111)	Static foam vs two dynamic replacement mattresses	Incidence 8% in all groups	No difference between groups
Prospective, randomized trial in high-risk intensive care patients (112)	Air-filled vs gel-filled pad	Incidence 32% on gel pad vs 32% on air pad	No difference between groups
Prospective, controlled trial in intensive care unit (113)	Alternating pressure mattress vs various other overlays	Incidence 13% alternating pressure vs 34% all other devices	0.38 (0.22-0.66)
Prospective, controlled trial in intensive care unit (114)	Alternating air mattress vs static air mattress vs water mattress	Incidence 20% alternating vs 5% static vs 10% water	No difference between groups
Prospective, randomized trial in intensive care unit (115)	Two groups of risk-based pressure-reducing systems	Incidence 24% vs 30% in both groups	No difference between groups
Prospective, randomized trial in acute care (116)	3" convoluted foam mattress vs 4" solid foam mattress	Incidence 47% vs 31%	Higher rate on 3" foam
Prospective, randomized trial in orthopedic patients (117)	Airwave vs Large Cell Ripple alternating air mattress	Incidence 16% vs 34% ($p > .05$)	No difference between groups
Prospective, randomized trial in long-term care (118)	Maxifloat overlay vs 4" foam mattress		0.42 (0.18-0.96)
Prospective, randomized trial in long-term care (119)	Two pressure-reducing devices	Incidence 32% on both devices	No difference between groups
Prospective, randomized trial in long-term care (neurological impairment) (120)	Alternating air mattress vs silicone overlay	Incidence 25% in both groups	No difference between groups
Prospective, randomized trial in long-term care (neurological impairment) (121)	Alternating air mattress vs silicone overlay	Incidence >50% in both groups	No difference between groups
Prospective, randomized trial in acute and long-term care (122)	LAL bed vs all other surfaces	Incidence 19% on experimental device vs 7% on all other surfaces	Experimental device was worse
Meta-analysis (12)	Silicone overlay, water, foam, static-air mattress	Six trials	No difference between groups
Prospective, randomized controlled trial in operating theater (123)	Operating table overlay vs standard table		0.53 (0.33-0.85)
Prospective, randomized controlled trial in operating theater (124)	Alternating pressure support vs gel pad and standard mattress		0.21 (0.06-0.7)

Notes: CI = confidence interval; LAL = low-air-loss. Meta-analysis results include only the studies meeting the author's inclusion criteria. Some trials included in the meta-analysis are listed separately. See reference for specifics.

other. Choice is likely to be on the basis of cost, local availability, and comfort.

Nutrition in Preventing Pressure Ulcers

Nutritional status has been thought to influence the incidence, progression, and severity of pressure sores. Pressure ulcers have been associated epidemiologically with markers for poor nutritional status. The data, however, are contradictory (Table 4). Albumin has been associated with the development of pressure ulcers in most, but not all, studies. The association of undernutrition is problematic, because there is no accepted gold standard for the diagnosis of undernutrition and because the markers for nutritional status may reflect underlying disease rather than undernutrition in older ill persons. In studies where albumin is associated with pressure ulcers, the assessment of overall nutritional status is not associated with the presence of a pressure ulcer. Decreases in serum albumin may reflect the presence of inflammatory cytokine production rather than nutritional status. Although poor nutrition is part of total patient care and should be addressed in each patient, no nutritional intervention has shown effectiveness in the prevention of pressure ulcers in published studies (22,23).

An observational study of hospitalized, critically ill patients given nutritional supplements suggests no effect on pressure-ulcer incidence. Oral supplements were given to 32.6% of one group compared with 86.9% of another group.

There was no difference in pressure-ulcer incidence (26.4% vs 20.2%), pressure ulcer prevalence at discharge (14.7% vs 10.3%), mortality (15.6% vs 14.2%), length of stay (17.3 days vs 17.4 days), or nosocomial infections (26.4% vs 19.0%) (24).

The effect of higher caloric and protein intakes in nutritionally supplemented, critically ill older patients did not affect the development of a pressure ulcer. 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. Despite a higher caloric intake in the intervention group (Day 2: 1081 kcal vs 957 kcal; $p = .006$) and higher protein intake (45.9 g protein vs 38.3 g protein in the control group; $p < .001$), the cumulative incidence of pressure ulcers was 41% in the nutritional intervention group versus 47% in the control group (25).

The effect of overnight supplemental enteral feeding in patients with a fracture of the hip and a high pressure-sore risk score has been 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. No difference was found for the development of a pressure ulcer, total serum protein, serum albumin, or the severity of pressure sores after 1 and 2 weeks. Comparison of the tube-fed group ($n = 25$ at 1 week; $n = 16$ at 2 weeks) and the control group showed two to

Table 4. Epidemiological Association of Nutritional Markers With Development of a Pressure Ulcer

First Author	Setting	Associated With the Presence of Pressure Ulcer	Not Associated With the Presence of Pressure Ulcer
Allman (27)	AC	Albumin	Weight, hemoglobin, TLC, nutritional assessment
Gorse (45)	AC	Albumin	Nutritional assessment score
Inman (109)	AC, ICU	Albumin (measured at 3 d)	Serum protein, hemoglobin, weight
Allman (27)	AC	BMI, TLC	Albumin, TSF, arm circumference, weight loss, hemoglobin, nitrogen balance
Hargink (26)	AC, orthopedic		Nocturnal enteral feeding
Anthony (125)	AC	Albumin, <32 g/dl	
Moolten (126)	LTC	24/28 had albumin, <3.5 g/dl	
Pinchcofsky-Devin (127)	LTC	Severe malnutrition by biochemical markers	Mild-to-moderate malnutrition or normal nutrition
Berlowitz (128)	LTC	Impaired nutritional intake	Albumin, serum protein, hemoglobin, TLC, BMI/weight
Bennett (28)	LTC		Weight, BMI, weight gain
Brandeis (129)	LTC	Dependency in feeding	BMI/weight, TSF
Trumbore (130)	LTC	Albumin, cholesterol	
Breslow (131)	LTC	Albumin, hemoglobin	Serum protein, cholesterol, zinc, copper, transferrin, body weight, BMI, TLC
Bergstrom (97)	LTC	Dietary protein intake 93% of RDA, dietary iron	Serum protein, cholesterol, zinc, copper, transferrin, weight, BMI, TLC
Ferrell (29)	LTC		Albumin, serum protein, BMI, hematocrit
Bourdel-Marchasson (24)	LTC		Oral nutritional supplement (26% vs 20% incidence)
Gurañnik (132)	Community		Albumin, BMI, impaired nutrition, hemoglobin

Note: 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.

three 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 tube-fed group (all $p < .001$). However, the development and severity of pressure ulcers were not significantly influenced in the tube-fed group. It is possible that the lack of effect on supplemental enteral feeding was due to poor tolerance of the feedings (26).

ISSUES AND DILEMMAS IN TREATMENT

Pressure Relief

Pressure-relieving devices have a therapeutic role in treating pressure ulcers. Air-fluidized therapy, because of

its extremely low tissue interface pressures, has been thought to be the best available therapy for pressure ulcers. In the very short clinical trials summarized in Table 5, air-fluidized therapy has been associated with improved rates of closure of pressure ulcers in hospital settings but not in longer-duration home trials.

When patients with pressure ulcers in an acute hospital setting were randomized to air-fluidized therapy or a vinyl alternating air mattress, patients treated on air-fluidized beds had a decrease in ulcer size over a mean of 15 days. However, there was no difference in the number of ulcers showing a size reduction of at least 50%. The cost was estimated at an additional \$80 per day (27).

Table 5. Comparison Among Pressure-Reducing Devices in the Treatment of Pressure Ulcers

Population	Intervention	Results	Risk Reduction (95% CI)
Prospective, controlled trial in acute care (133)	AF bed vs standard mattress	Change in surface area	Healing better on AF bed
Prospective, randomized trial in acute care (27)	AF bed vs alternating-air mattress	Improvement on AF bed	OR 5.6 (1.4-21.7)
Prospective, randomized controlled trial in home care (134)	AF bed vs variety of alternates	86% improved on AF bed vs 69% on alternates	No difference in rate of healing
Prospective, randomized trial in acute care (135)	LAL bed vs Geomatt mattress		No difference in healing
Prospective, randomized trial in acute care (136)	AP mattress vs convoluted foam	More improvement on AP mattress	No statistical analysis
Prospective, randomized trial in acute care (137)	LAL bed vs replacement mattress	80% improved on LAL vs 70% improved on mattress	No difference between groups
Prospective, randomized controlled trial in acute care (138)	LAL bed vs LAL overlay	Median change in surface area	No difference between groups
Prospective, randomized controlled trial in acute care (139)	AP vs AP	Complete healing	No difference between groups
Prospective, randomized trial in myocutaneous flap surgery (140)	AF bed vs dry flotation	33% breakdown on AF bed vs 17% on flotation	No difference between groups
Prospective, randomized trial in long-term care (141)	LAL bed vs Geomatt foam overlay	Reduction in size by 190% in LAL vs 64% on overlay	LAL better ($p = .04$)

Note: CI = confidence interval; AF = air-fluidized; OR = odds ratio; LAL = low-air-loss; AP = alternating-pressure.

In 95 nursing home patients with severe pressure ulcers treated on air-fluidized beds, 14% of pressure ulcers healed in a mean of 79 days. Forty-four percent of patients had greater than 50% reduction in surface area of the index ulcer. Very few patients had a reduction in ulcer surface area after 1 month of treatment on the specialized bed. The median length of time to healing was 119 days, and the median length of time to improvement was 127 days. The additional cost for the bed was \$50 to \$100 per day (28). In another trial in nursing home patients, a low-air-loss bed produced substantial improvement in ulcer size (9.0 vs 2.5 mm² per day) compared with a 10-cm convoluted foam mattress (29).

When various devices, such as low-air-loss beds or overlay support surfaces, are compared among themselves, little difference between devices is seen (Table 5). Overall, the data suggest that pressure-reducing devices have an impact on healing of pressure ulcers. The choice among devices is not clear, and the use of these devices is very expensive.

Nutrition in the Treatment of Pressure Ulcers

One of the most important potentially reversible host factors contributing to wound healing is nutritional status. Several studies suggest that dietary intake, especially of protein, is important in healing pressure ulcers (Table 6). Greater healing of pressure ulcers has been reported with a higher protein intake irrespective of positive nitrogen balance (30). Breslow (31) evaluated 48 patients with Stage II through Stage IV pressure ulcers in a dietary intervention trial. Undernutrition was defined as a serum albumin below 35 g/l or body weight more than 10% below the midpoint of the age-specific weight range. The results suggested that patients fed a 24% protein diet healed their pressure ulcers at a greater rate than those fed a standard 14% protein diet. However, changes in body weight or in biochemical parameters of nutritional status did not occur between groups. The study was limited by a small sample size (only 28 patients completed the study), nonrandom assignment to treatment groups, confounding effects of air-fluidized beds, and the use of two different feeding routes (31).

Chernoff and colleagues randomized 12 enterally fed patients to formulas containing 17% versus 25% of calories as protein (30). The group that received 1.8 g/kg of protein had a 73% improvement in pressure ulcer surface area compared with a 42% improvement in pressure ulcer surface area in the group receiving 1.2 g/kg of protein, despite the fact that the group that received the higher protein level began the study with larger surface area pressure ulcers (22.6 cm² vs 9.1 cm²).

An 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/d. Half of the chronically ill elderly persons are unable to maintain nitrogen balance at this level (32). Increasing protein intake beyond 1.5 g/kg/d may not increase protein synthesis and may cause dehydration (33). A reasonable protein requirement is therefore between 1.2 and 1.5 g/kg/d.

The deficiency of several vitamins has significant effects on wound healing. However, supplementation of vitamins to accelerate wound healing is controversial. High doses of Vitamin C have not been shown to accelerate wound healing (34). In a 12-week study of 88 patients who received either 10 mg or 500 mg of ascorbic acid twice daily, the healing rates and the healing velocity of their pressure ulcers was not different in the higher-dosed group (35). Zinc supplementation has not been shown to accelerate healing except in zinc deficient patients (36). High serum zinc levels interfere with healing, and supplementation above 150 mg/d may interfere with copper metabolism (22).

Despite evidence that higher protein intake seems to be important in healing pressure ulcers, the use of enteral feeding has been disappointing. In a study of enteral tube feedings in long-term care, 49 patients were followed for 3 months (37). Patients received 1.6 times basal energy expenditure daily, 1.4 g of protein per kilogram per day, and 85% or more of their total recommended daily allowance. At the end of 3 months, there was no difference in the number or healing of pressure ulcers. In a study of survival among residents in long-term care with severe cognitive impairment, 135 residents were followed for 24 months (38). The reasons for the placement of a feeding tube included the presence of a pressure ulcer. Having a feeding tube was not associated with increased survival; in fact, the risk was slightly increased (odds ratio 1.09). The authors conclude that the effectiveness of enteral feeding in pressure ulcers is not established.

Topical Dressings and Local Wound Care

Local wound treatment is directed to providing an optimum wound environment and improving host factors. The most commonly used dressing for pressure ulcers at hospital discharge in the United States is dry gauze (39). The use of dry gauze persists despite clear data suggesting that it results in delayed healing (Table 7). Compared with wet-to-dry gauze dressings, moist dressings are clearly superior. Moist wound healing allows experimentally induced wounds to resurface up to 40% faster than air-exposed wounds (40-42).

Table 6. Nutritional Interventions in the Treatment of Pressure Ulcers

First Author	Setting	Intervention	Outcome
Breslow (31)	Long-term care	24% protein vs 14% protein enteral feeding	Greater healing with higher protein
Chernoff (30)	Long-term care	25% protein vs 17% protein enteral feeding	Greater healing with higher protein
ter Riet (35)	Long-term care	Vitamin C 50 mg vs 1000 mg	No difference in healing
Norris (142)	Long-term care	Zinc sulfate 200 mg TID	No difference in healing
Henderson (37)	Long-term care	Enteral feeding	No difference in prevalence at 3 mo
Mitchell (38)	Long-term care	Enteral feeding	No difference in prevalence after 2 y

Table 7. Moist Dressings Compared With Wet-to-Dry Saline Dressings in the Treatment of Pressure Ulcers

Treatment	Measure	Results OR (95% CI)
Film dressing vs wet-to-dry saline gauze (47)	Complete healing	42.65 (2.23-815.5)
Iodosorb vs wet-to-dry saline gauze (143)	Complete healing	5.94 (0.88-40.1)
Hydrocolloid vs wet-to-dry Dakin's solution (45)	Complete healing	2.46 (1.18-5.12)

Note: OR = odds ratio; CI = confidence interval.

Occlusive dressings have been developed to produce a moist wound environment. The term "occlusive" describes the lessened ability of a dressing to transmit moisture vapor from a wound to the external atmosphere. The degree to which dressings dry the wound can be measured by the moisture vapor transmission rate (MVTR). A 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/m²/h, and impregnated gauze has an MVTR of 57 g/m²/h. In comparison, hydrocolloid dressings have an MVTR of 8 g/m²/h (43). Any therapy that dehydrates the wound, such as dry gauze, heat lamps, air exposure, or liquid antacids, is detrimental to the healing of chronic wounds (44-47).

Occlusive dressings can be divided into broad categories of polymer films, polymer foams, hydrogels, hydrocolloids, alginates, and biomembranes. Each has several advantages and disadvantages. Understanding these differences is the key to planning for wound management in a particular patient. Comparative qualities among available agents are shown in Table 8 (48,49). Most of the occlusive dressings offer pain relief. Only absorbing granules or polymers fail to reduce pain. Polymer films are impermeable to liquid but permeable to both gas and moisture vapor. Because of low permeability to water vapor, these dressings are not dehydrating to the wound. Nonpermeable polymers, such as polyethylene 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 exudates.

Hydrogels are hydrophilic 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.

Hydrocolloid dressings are complex dressings that 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 damage epithelial tissue in the wound. The adhesive barrier is frequently overcome in highly exudative wounds. Hydrocolloid dressings are not recommended over tendons or on wounds with eschar formation. Several of these dressings include a foam padding layer that may reduce pressure to the wound.

Alginates are complex polysaccharide dressings that are highly absorbent. 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.

Only the hydrocolloid and biomembranes offer bacterial resistance. The agents 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 3 weeks.

Saline-soaked gauze that is not allowed to dry is an effective wound dressing. When moist saline gauze has been compared with occlusive-type dressings, the healing of pressure ulcers has been similar with both dressings (50-52). There is little observed difference when various dressings have been compared with moist saline gauze (Table 9). The advantage of occlusive-type dressings has been demonstrated in their cost effectiveness compared with traditional moist gauze dressings. Although the individual dressings are more expensive, cost savings occurs primarily due to a decrease in nursing time for dressing changes (52).

A slight advantage for hydrocolloid dressings compared with hydrogels has been shown in several clinical trials (Table 10). There appears to be little difference demonstrated in clinical trials of foam or polyurethane dressings compared with hydrocolloid dressings. Absorption dressings are more effective than a collagen sponge or povidone-iodine, and calcium alginate has been shown superior to absorption paste (Table 10). The optimum dressing depends on the clinical circumstances of the wound.

Table 8. Comparison of Occlusive Wound Dressings

Characteristic	Moist Saline Gauze	Polymer Films	Polymer Foams	Hydrogels	Hydrocolloids	Alginates, Granules	Biomembranes
Pain relief	+	+	+	+	+	±	+
Maceration of surrounding skin	±	±	-	-	-	-	-
O ₂ permeable	+	+	+	+	-	+	+
H ₂ O permeable	+	+	+	+	-	+	+
Absorbent	+	-	+	+	±	+	-
Damage to epithelial cells	±	+	-	-	-	-	-
Transparent	-	+	-	-	-	-	-
Resistant to bacteria	-	-	-	-	+	-	+
Ease of application	+	-	+	+	+	+	-

Sources: Adapted from Helfman and colleagues (49) and Witkowski and Parish (50).

Table 9. Occlusive Dressing Compared With Moist Saline Dressing in the Treatment of Pressure Ulcers

Treatment	Measure	Results OR (95% CI)
Hydrocolloid (Comfeel) vs moist saline (50)	Complete healing	4.09 (1.18-14.14)
Hydrocolloid (Duoderm) vs moist saline (51)	Complete healing	14.27 (1.76-15.56)
Hydrocolloid vs moist saline (53)	Complete healing	2.03 (0.52-7.52)
Hydrocolloid vs moist saline (144)	Complete healing	1.39 (0.55-3.49)
Hydrocolloid (Duoderm) vs moist saline (145)	34% decrease in surface area vs 9% increase in control	$p = .23$
Hydrocolloid vs moist saline (146)	Complete healing	81% of HCD vs 78% of saline group ($p > .05$)
Polyurethane (Opsite) vs moist saline (147)	% reduction in area	22.3 (-27.43-72.09)
Foam dressing (Epi-Lock) vs moist saline (148)	Complete healing	2.62 (0.58-11.89)
Hydrogel (Carrasyn) vs moist saline (149)	Complete healing	0.93 (0.28-3.09)
Hydrogel vs moist saline (150)	Relative volume	64% vs 26% of initial size

Note: OR = odds ratio; CI = confidence interval.

Certain antiseptic agents are cytotoxic to human fibroblasts, including povidone iodine, chlorhexidine gluconate, hexachlorophene, benzalkonium chloride, and trypsin/balsam peru/castor oil (53-56). In animal models, povidone-iodine 1%, acetic acid 3%, and sodium hypochlorite 0.5% adversely affected wound healing (57). In human pressure ulcers, Dakin's solution 0.05% was clearly inferior to a hydrocolloid dressing (45) (Table 7). Other types of local wound treatments can interfere with healing (Table 11). Several types of topical wound treatments can promote more rapid epidermal resurfacing. The results of controlled trials for several agents are shown in Table 12 (58). The range of acceleration in healing varies from 18% to 36%. Note that most of these agents, or their vehicles, are occlusive. Whether the benefit is independent of the occlusive vehicle is not known.

Debridement

Necrotic debris increases the possibility of bacterial infection and delays wound healing (59). The preferred method of debriding the pressure ulcers remains controversial. Options include mechanical debridement with dry gauze dressings, autolytic debridement with occlusive dressings, application of exogenous enzymes, or sharp surgical debridement. Sharp surgical debridement produces the most rapid removal of necrotic debris and is indicated in the presence of infection. Mechanical debridement can be easily accomplished by letting the saline gauze dressing dry before removal. Remoistening of gauze dressings in an attempt to

reduce pain can defeat the debridement effect. Both surgical and mechanical debridement can damage healthy tissue or fail to completely clean the wound. Debridement with a dry gauze should be stopped as soon as a clean wound bed is obtained because dry dressings have been associated with delayed healing.

Thin portions of eschar can be removed by occlusion under a semipermeable dressing. Both autolytic and enzymatic debridement requires periods of several days to several weeks to achieve results. Enzymatic debridement can dissolve necrotic debris, but whether it harms 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.

Three enzyme preparations are currently marketed in the United States for debridement: collagenase, papain/urea, and a papain/urea-chlorophyll combination. Collagenase reduced necrosis, pus, and odor compared with inactivated control ointment (60) and produced debridement in 82% of pressure ulcers at 4 weeks compared with petrolatum (61). Papain produced measurable debridement in 4 days compared with the control vehicle ointment (62). 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 (63). The issue of when to debride, and by what method, remains controversial. Whether debridement improves the rate of healing remains undefined.

Table 10. Comparisons Between Occlusive Dressings in the Treatment of Pressure Ulcers

Dressing	Measure	Results (95% CI)
Hydrogel (Biofilm) vs hydrocolloid (Duoderm) (151)	Complete healing	0.40 (0.13-1.19)
Hydrogel (Clearsite) vs hydrocolloid (Duoderm) (152)	Mean reduction per week	-4.7 (-20.95-11.5)
Hydrogel vs hydrocolloid (153)		No difference in healing rate
Hydrocolloid (Duoderm) vs foam (Tielle) (154)	Complete healing	1.90 (0.77-4.67)
Hydrocolloid (Duoderm) vs foam (Alleevyn) (155)	Complete healing	1.09 (0.31-3.77)
Polyurethane (Alleevyn) vs hydrocolloid (Duoderm) (156)	Complete healing	0.25 (0.06-1.14)
Polyurethane (Tielle) vs hydrocolloid (Duoderm) (157)	Complete healing	1.32 (0.54-3.21)
Poly-hema vs hydrocolloid (Duoderm) (158)	Complete healing	1.20 (0.34-4.14)
Amino acid copolymer (Inerpan) vs hydrocolloid (Comfeel) (159)	Complete healing	0.56 (0.29-1.08)
Calcium alginate vs dextranomer paste (160)	>75% healing	3.05 (1.06-8.76)
Dextranomer paste vs collagen sponge (46)	Mean time to healing (d)	20 vs 47 d
Absorption dressing vs povidone-iodine (161)	% reduction in length	8.6 (-10.98-28.18)

Table 11. Agents That Delay Epidermal Resurfacing

Dressing	Relative Rate of Healing (%)
Neomycin sulfate	-5
Dakin's solution (1%)	-6
Hibiclens	-7
Hydrogen peroxide (3%)	-8
Povidone iodine solution	-10
Wet to dry gauze	-15
Liquid detergent	-28
Furacin	-30
Triamcinalone acetonide (0.1%)	-34

Source: Alvarez (58). Used with permission.

Growth Factors

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. The factors described include 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 growth factors is attractive. The development of wound healing factors is still in infancy but shows great promise. Several of these factors have been favorable in animal models; however, they have not been as successful in human trials (Table 13).

In pressure ulcers, recombinant platelet-derived growth factors (rhPDGF-BB) failed to improve the rate of complete healing (64), although a 15% difference in the percentage of initial volume of ulcers has been shown with PDGF-BB in another study (65). Another report showed that more subjects had >70% wound closure with basic fibroblast growth factor 100 µg/ml ($p = .05$) (66).

The magnitude of the difference in those trials that have shown an effect is small, considering the high cost of growth factor therapy.

Diagnosing Infection

Colonization of pressure ulcers with bacteria is common and unavoidable. All chronic wounds become colonized, initially with skin organisms followed in 48 hours by gram-negative bacteria. In worsening pressure ulcers, *Pseudomonas aeruginosa* and *Providencia* species were found in 88% and 34% of ulcers, respectively, compared with 0% of sta-

tionary 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 (67,68). On the basis of these findings, *P. aeruginosa* and *Providencia* species should not be regarded as simple colonization. Although normal skin flora in numbers >10⁵ organisms/ml produce local disease in intact skin (69) and skin grafts and flaps show poor healing when >10⁵ organisms of certain species of bacteria are present (70), chronic wounds do not appear to follow these rules. Greater than 10⁵ 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 (71). The presence of microorganisms alone (colonization) does not indicate an infection in pressure ulcers. The diagnosis of infection in chronic wounds must be made on the basis of clinical signs—erythema, edema, odor, fever, or purulent exudate. A foul odor is a particularly important clinical sign, usually signifying anaerobic organisms (72). However, wounds with a reported foul odor are not always infected. When there is evidence of clinical infection, topical or systemic antimicrobial or antibiotics are required. Reduction of colony-forming units (CFUs) has been used as the end point in evaluating antimicrobial efficacy in acute wounds. Several antimicrobial or antibiotic agents reduce CFUs without damaging the wound, including silver sulfadiazine 1% cream, combination antibiotic ointments, and propylene glycol (73). Topical gentamicin and silver sulfadiazine have been shown to improve the clinical appearance of infected wounds and may improve healing (74,75). Iodine and thimerosal have been noted to increase pain and delay healing (76). Infections with anaerobes may respond to topical metronidazole (77). Systemic antibiotics are indicated when the clinical condition suggests spread of the infection to bone or to the blood stream.

The incidence of bacteremia from pressure ulcers is about 1.7 per 10,000 hospital discharges (78). However, sepsis is a serious complication of pressure ulcers and is a frequent cause of death. In a small 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 five patients, bacteremia persisted despite antibiotic treatment and resolved only after local debridement (79). The bacteremia occurring with pressure ulcers is likely to be polymicrobial (78).

Osteomyelitis is a frequent complication of pressure ulcers and diabetic ulcers, reported in 38% of patients who have infected pressure ulcers (80) and in 59 of 96 (61%) of foot infections in diabetic patients (81). Diagnosis of contiguous osteomyelitis in pressure ulcers is difficult. Plain radiographs are unable to differentiate true osteomyelitis from pressure changes to bone (82). Radionuclide studies, including technetium-99m and gallium-67, are sensitive but have a false-positive rate of 41% (80). Computed tomography may be more useful, with a specificity of 90%, although the sensitivity is only 10% (83). A needle biopsy of bone is the

Table 12. Agents That Promote Epidermal Resurfacing

Dressing	Relative Rate of Healing (%)
DuoDerm	+36
Blisterfilm	+33
Benzoyl peroxide (20%)	+33
Bacitracin zinc	+30
Silvadene	+28
Neosporin	+28
Polysporin	+25
I&J first aid cream	+20
Bioclusive	+20
Op-Site	+18

Source: Alvarez (58). Used with permission.

Table 13. Growth Factor Trials in the Treatment of Pressure Ulcers

Growth Factor	n	Measure	Healing Rate Active	Healing Rate Placebo
rhPDGF- β (162)	124 in four groups	Complete closure	23%	0%
rhPDGF- β (163)	41 in three groups	Time-to-50% closure or ulcer volume	No difference	No difference
rbFGF (66)	50 in eight groups	>70% closure	Greater closure	
rIL 1- β (164)	26 in four groups	Complete closure	No difference	No difference
rhGM-CSF and rbFGF (165)	61 in four groups	>85% closure	No difference	No difference

Note: rhPDGF- β = recombinant human platelet-derived growth factor- β ; rbFGF = recombinant basic fibroblast growth factor; rIL 1- β = recombinant human interleukin-1- β ; rhGM-CSF = granulocyte-macrophage colony-stimulating factor.

most useful single test, with a sensitivity of 73% and a specificity of 96% (84).

Despite an increase in the numbers of bacteria, occlusive dressings used to treat chronic wounds very rarely cause a clinical infection. Hutchinson and McGuckin (85) 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 nonocclusive gauze.

Surgical Repair

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 (86). In a series of 40 patients selected for surgical closure of pressure ulcers, patients were divided into three subgroups. In non-traumatic, nonparaplegic elderly patients with a mean age of 73 years, 84% of the surgically treated pressure ulcers were healed at discharge. Twelve percent of the surgically treated patients had another pressure ulcer at discharge. Within 8 months, 40% of the 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 11 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 (87). After 10 years of follow-up in sixteen surgically treated patients, only one patient remained alive and free of pressure ulcers (88).

A decision analysis demonstrated that myocutaneous flap procedures for Stage III pressure ulcers were 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 with medical therapy (89).

GENERAL ISSUES

Rate of Healing

Pressure ulcers, like other chronic wounds, fail to proceed through an orderly and timely process to produce anatomical or functional integrity (90). Normally, fibroblasts and epithelial cells grow rapidly in skin tissue cultures, covering 80% of in vitro surfaces within the first 3 days. In contrast, biopsy specimens from pressure ulcers usually do not grow until much later, covering only 70% of surfaces by 14

days (91). The result is slow healing. About 75% of Stage II pressure ulcers healed in 8 weeks, but only 17% of Stage III/IV pressure ulcers healed in that time (28). Twenty-three percent of Stage II pressure ulcers remain unhealed at 1 year, and 48% of Stage IV pressure ulcers are unhealed at 1 year. At 2 years, 8% of Stage II pressure ulcers, 29% of Stage III pressure ulcers, and 38% of Stage IV pressure ulcers remained unhealed (92). The considerable length of time to healing increases the morbidity and cost of treating pressure ulcers and is often frustrating to the patient and caregivers.

Quality of Care Issues

In long-term care settings, pressure ulcers are used as a quality indicator for caregiving. The occurrence of pressure ulcers, when viewed as a failure of the healthcare system, prevents the comprehensive and constructive attention this topic deserves (93). Whether the factors producing pressure ulcers can be improved by increased vigilance or whether a "floor effect" prevents reduction in the incidence to zero is debated (94).

Pressure Ulcers and Mortality

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 with 15% of at-risk patients without pressure ulcers (95). Patients who develop a new pressure ulcer within 6 weeks after hospitalization are three times as likely to die as patients not developing a pressure ulcer (96). 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 with a mortality rate of 4% among residents who did not subsequently develop a pressure ulcer (97). Residents in a skilled nursing facility who had pressure ulcers experienced a 6-month mortality rate of 77.3%, whereas patients without pressure ulcers had a mortality rate of 18.3% (98). 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 (99).

Despite this association with death rates, it is not clear how pressure ulcers contribute to increased mortality. Although authors have reported a threefold increase in mortality with the development of a new pressure ulcer, the severity of the pressure ulcer has not correlated with an increased risk. Patients with Stage II pressure ulcers have been equally as likely to die as patients with Stage IV pressure ul-

cers (99). This suggests that 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 the rate of acute hospitalization (96).

A correction for the presence and severity of coexistent 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. 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). When adjusted for measures of disease severity, comorbidity, and a history of weight loss, the development of a pressure ulcer was no longer a predictor of mortality at 1 year (100).

SUMMARY

The accumulating data for the prevention and management of pressure ulcers permits an outline of clinical strategies. Risk assessment remains problematic because of its infrequent use in healthcare settings and an apparent floor effect in preventing all pressure ulcers. Pressure-reducing devices are superior to a standard hospital mattress in preventing pressure ulcers. Pressure-reducing devices are effective in improving the healing rate of pressure ulcers. However, it is difficult to distinguish among various devices.

Local wound treatment should aim at maintaining a moist wound environment. Options include moist saline dressings or any number of occlusive dressings. The choice of a particular dressing depends of wound characteristics such as exudate, deadspace, or wound location.

The impact of nutrition on the prevention of pressure ulcers remains controversial. Dietary protein intake seems linked to improved rates of healing, but the results of enteral feeding to achieve this result are disappointing. Debridement by any of several methods may improve time to a clean wound bed, but the effect of debridement on time to healing remains to be demonstrated. The use of topical growth factors in improving healing rates is in its infancy but has not been remarkably effective thus far. Surgical closure in elderly persons has been associated with a high recurrence rate.

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