REVIEW

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Prevention and treatment of pressure ulcers: What works? What doesn't?

ABSTRACT

Although no gold standard for preventing or treating pressure ulcers has been established, data from clinical trials indicate specific efforts are worthwhile. Preventive strategies include recognizing risk, decreasing the effects of pressure, assessing nutritional status, avoiding excessive bed rest, and preserving the integrity of the skin. Treatment principles include assessing the severity of the wound; reducing pressure, friction, and shear forces; optimizing wound care; removing necrotic debris; managing bacterial contamination; and correcting nutritional deficits.

KEY POINTS

Pressure ulcers are often blamed on poor nursing care in long-term care facilities, but the incidence is actually higher in acute care hospitals.

The most important reversible host factor contributing to wound healing is nutritional status. Several studies suggest that dietary protein, in particular, is important in healing pressure ulcers.

Surgical closure of pressure ulcers results in more rapid resolution of the wound. The chief problems are frequent recurrence and inability of frail patients to tolerate the procedure.

Any therapy that dehydrates the wound, such as dry gauze, heat lamps, air exposure, or liquid antacids, is detrimental to chronic wound healing

The most common complications are increased mortality, osteomyelitis, and sepsis.

ALTHOUGH PRESSURE ULCERS continue to confound the best efforts of doctors, nurses, and medical engineers, the incidence can be reduced and healing can be speeded. This article reviews prevention and treatment, and discusses evidence that the effort is worthwhile.

HOW PRESSURE ULCERS OCCUR

Pressure ulcers are the visible evidence of pathologic changes in the blood supply to dermal tissues. The chief cause is pressure, or force per unit area, applied to susceptible tissues.

Pressure is concentrated wherever weight-bearing points come in contact with surfaces. These weight-bearing points usually occur over bony prominences. Tissues over bony prominences ("hard sites") may differ in resistance to hypoxia or pressure compared with "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 areas over the sacrum, coccyx, ischial tuberosities, and greater trochanters account for most pressure ulcer sites.[2] The sacrum is the most frequent site (36% of ulcers), followed by the heel (30%); other body areas each account for about 6%.[3,4]

WHO GETS PRESSURE ULCERS?

Two groups of patients—those with spinal cord injuries and the elderly—account for most pressure ulcers. Fifty percent of all admissions

to specialized cord-injury hospitals and 8% of all deaths in these facilities are due to pressure ulcers.[5] About 70% of all pressure ulcers occur in persons older than 70 years.[6]

Comorbid conditions, especially those resulting in immobility or reduced tissue perfusion, greatly increase the risk of developing pressure ulcers.

HIGHER INCIDENCE IN HOSPITALS, NOT NURSING HOMES

Pressure ulcers occur across the spectrum of health care settings. The highest incidence (the number of persons with *new* ulcers divided by the number of persons at risk) is actually in the hospital, while the highest prevalence (the number of persons with an ulcer divided by the number of persons at risk) is in long-term care facilities.

From 57% to 60% of ulcers occur in the hospital.[7–9] The incidence in hospitalized patients ranges from 3% to 30%,[10,11] with most estimates centering around 9% to 13%. Pressure ulcers occur early in hospitalized patients, usually within the first 2 weeks.[9] The incidence differs by ward, with orthopedic patients and intensive care patients at greatest risk. Up to 66% of orthopedic patients develop pressure ulcers of varying severity.[12]

In long-term care facilities the prevalence ranges between 2.4%[13] and 23%.[14,15] Fewer than 20% of pressure ulcers occur outside of institutions.[6] In home care patients, the prevalence ranges between 9% and 20%.[16,17]

PRESSURE ULCERS DO NOT ALWAYS INDICATE POOR CARE

Pressure ulcers are increasingly being used as indicators of poor-quality care.[18,19] Failure of nursing care is blamed for most pressure ulcers in both hospitals and nursing homes.

However, the ischemic injury responsible for pressure ulcers in hospitalized patients likely occurs very early, in the emergency room or on the operating table.[20] Furthermore, most pressure ulcers occur within the first 2 weeks of hospitalization, rather than uniformly throughout hospitalization. Seventy-five percent of pressure ulcers in orthopedic patients develop within the first 2 weeks of hospitalization, with 34% occurring within the first week.[21]

This nonrandom timing seems to indicate that pressure ulcer development is more related to initial injury than to length of time in a ward nursing setting.

Can ulcers be prevented?

Whether pressure ulcers are preventable remains controversial. When aggressive measures for preventing pressure ulcers have been applied, a "floor effect" for incidence has been noted: the incidence can be reduced to a certain level, but no lower.[22] Further evidence of a floor effect comes from randomized, controlled trials of preventive interventions, which have not shown a reduction in incidence to zero.

Systematic efforts at education, heightened awareness, and specific interventions by interdisciplinary wound teams suggest that a high incidence of pressure ulcers can be reduced. Over time, reductions of 25% to 30% have been reported.[23,24] However, the reduction may be transient or unstable over time, may vary with changes in personnel, or may be due to random variation.[25] No trial has reported elimination of pressure ulcers over time.

Some pressure ulcers seem inevitable, even with the best care

Overall, the data suggest that pressure ulcers can be but are not always signs of poor-quality care. For example, they often occur in terminally ill patients, for whom the goals of care may not include prevention of pressure ulcers. In orthopedic patients or intensive care patients, the necessity for immobilization may preclude turning or the use of pressure-relieving devices.

PREVENTION: STRATEGIES TO DECREASE PRESSURE, FRICTION, AND SHEAR

The opportunity to prevent pressure ulcers, for most patients, is 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 preserving the integrity of the skin.

In patients at risk, the first preventive action is to reduce the effect of pressure, friction, and shear forces. The theoretic goal is to

reduce tissue pressure below the capillary closing pressure of 32 mm Hg.

Turning every 2 hours may not be enough

The most expedient method for reducing pressure is to turn and position the patient frequently. A 2-hour turning schedule for spinal-injury patients was deduced empirically in 1946.[26] However, turning the patient to relieve pressure may be difficult to achieve despite the best nursing efforts, and is very costly. In a study demonstrating the effectiveness of turning,[27] higher hospital staff ratios may have resulted in demonstrating effectiveness.

The optimal interval for turning is unknown and may be shortened or lengthened by host factors. In healthy older volunteers, intervals of 1 to 1 1/2 hours rather than the traditional 2 hours were required to prevent skin erythema on a standard mattress.[28] Furthermore, even though turning, positioning, and increasing passive activity seems like a common-sense approach, no published data support the view that it can actually prevent pressure ulcers.[27,29]

The best time to intervene is early

Are preventive devices effective?

Because of the limitations and cost of turning the patient frequently, a number of devices have been developed for preventing pressure injury. Devices can be classified as pressure-relieving (consistently reducing interface pressure to less than 32 mm Hg) or pressure-reducing (pressure less than standard support surfaces, but not below 32 mm Hg). Most devices are pressure-reducing.

Pressure-reducing devices can be further classified as static or dynamic. Static surfaces are stationary and designed to distribute local pressure over a larger body surface. Examples include foam mattresses and devices filled with water, gel, or air. Sheepskin, although still used, is not effective. Dynamic devices use an electric air pump that either inflates and deflates air cells in the mattress in an alternating pattern or that forces air up through a layer of fine ceramic spheres, causing them to act like a fluid and promoting uniform pressure distribution over body surfaces. The number of pressure-reducing devices available is staggering and confusing.

In some prospective, randomized trials, use of pressure-reducing devices led to a lower incidence and severity of pressure ulcers than with a standard hospital mattress among orthopedic patients,[30,31] surgical and oncology patients,[32] intensive care patients,[33,34] and acute hospital patients.[35] Not all trials, however, showed a difference in preventing pressure ulcers in acute care[36–38] or in long-term care.[39] Pressure ulcers develop in some patients in spite of the use of pressure-reducing devices. In a sample of elderly patients in a community hospital, 25% of those with limited mobility developed a new pressure ulcer—and 96% of the patients who developed an ulcer did so while on various pressure-reducing devices.[40]

Pressure-relieving devices differ in effectiveness depending on body site. In studies in normal volunteers,[41,42] several devices reduced pressure on the sacrum, and three dynamic air support systems lowered pressure at the trochanter compared with a conventional mattress. However, no device reduced pressure over the trochanter to physiologic levels. Few currently marketed devices, including air-fluidized beds, consistently reduce heel pressure below minimal capillary pressure.[43]

In addition, although some dynamic air mattresses and flotation systems can reduce pressure to near-physiologic levels, all benefit is lost if the head of the bed is elevated to 30 degrees, such as for tube feedings.[44]

Several trials compared different devices—dynamic air mattresses, water flotation systems, and static support overlays—in terms of the incidence and severity of pressure ulcers that occurred with their use.[41,45–47] In these studies, no device was more effective than any other in preventing pressure ulcers.

The only devices that consistently relieve pressure on the trochanter, ischium, and sacrum are low-air-loss and air-fluidized beds. Air-fluidized beds have been shown to reduce the development of pressure ulcers in hospitalized intensive care unit patients. When 98 patients were randomized to an air-fluidized bed or a conventional mattress, fewer patients developed pressure ulcers on air-fluidized beds.[40]

How to select a preventive device

A preventive device should be selected on the basis of cost, which varies considerably, and ease of use. Air-fluidized and low-air-loss systems are the most expensive and static support overlays are the least expensive.

Dynamic devices are often noisy and disturbing to patients. Mechanical difficulties are frequent. In one nursing home, 110 air-filled mattresses were required to treat 76 patients, owing to frequent equipment failure. Despite experimental evidence of effective pressure reduction, there was no difference in pressure ulcer incidence compared with the control support surface.[48]

Some static devices are heavy and difficult for nurses to use effectively. The ability of the patient to move and reposition also guides the selection of a device. If the patient cannot shift positions independently, a dynamic device may be superior to a static device. Low-air-loss beds allow the head of the bed to be raised (eg, for tube feeding), whereas air-fluidized beds require a wedged pillow to elevate the upper body.

STUDIES LINK MALNUTRITION WITH RISK

Pressure ulcers and malnutrition frequently coexist in frail patients, but a causal relationship has not been established.

In a prospective study,[49] malnutrition (measured by an index of biochemical and anthropometric variables) was present in 29% of high-risk patients at hospital admission. At 4 weeks, 17% of malnourished patients had developed a pressure ulcer, compared with 9% of well-nourished patients. Patients malnourished at hospital admission were twice as likely to develop pressure ulcers as nonmalnourished patients (relative risk 2.1, 95% confidence interval 1.1 to 4.2).

In a nursing home study,[50] 59% of residents were found to be malnourished and 7.3% were severely malnourished. Pressure ulcers occurred in 65% of the most severely malnourished patients. No pressure ulcers developed in the mild-to-moderately malnourished or well-nourished groups. Although pressure ulcers occurred more frequently in malnourished patients in these studies, not all malnourished patients developed ulcers.

Bergstrom and Braden[51] reported that low dietary protein intake predicted the development of pressure ulcers. Patients with pressure ulcers took in 93% of the recommended daily intake of protein, compared with an intake of 119% in patients without pressure ulcers. Only dietary protein intake 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.

Berlowitz and Wilking[52] reported that impaired nutritional intake (defined as a persistently poor appetite, meals held due to gastrointestinal disease, or a prescribed diet of less than 1,100 calories or 50 g protein per day) predicted pressure ulcer development in a nursing home. However, no other single nutritional variable reached univariate significance.

In an observational study of home care patients,[53] patients with pressure ulcers took in a mean of 185 kcal/day less than patients without ulcers, but the difference was not statistically significant (95% confidence interval –413 to 43). Similarly, they took in a mean of 6.73 fewer grams of protein (95% confidence interval –16.20 to 2.74).

Nutritional supplementation proves disappointing as prevention

Despite an epidemiologic association between malnutrition and pressure ulcers, trials of nutritional intervention in preventing pressure ulcers have been disappointing. An observational study of hospitalized, critically ill patients[54] suggested that nutritional supplements had no effect on preventing pressure ulcers. Oral supplements were given to 32.6% of one group compared with 86.9% of another group. There was no statistically significant 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%).

Bourdel-Marchasson et al,[55] in a prospective trial in critically ill older patients, found that nutritional supplementation did not affect development of pressure ulcers. Despite a higher caloric intake in the intervention

Select a pressure-reducing device on the basis of cost and ease of use

group on the second day (1,081 kcal vs 957 kcal, $P = .006$) and higher protein intake (45.9 g vs 38.3 g, $P < .001$), the cumulative incidence of pressure ulcers was 41% in the nutritional intervention group vs 47% in the control group. A limitation of the study was that 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.

Hartgrink et al,[56] in a randomized trial, evaluated the effect of overnight supplemental enteral feeding in patients with a fracture of the hip and at high risk of developing pressure ulcers. Of the 62 patients randomized for enteral feeding, only 25 tolerated the feeding tube for more than 1 week, and only 16 tolerated it for 2 weeks. Compared with a control group, those who were actually receiving tube feedings had two to three times higher protein and energy intake ($P < .0001$), and significantly higher total serum protein and serum albumin levels after 1 and 2 weeks (all P values $< .001$). However, the tube feedings did not significantly reduce the incidence of pressure ulcers. It is possible that the lack of effect on supplemental enteral feeding was due to poor tolerance of the feedings. No difference was found in the total serum protein or serum albumin levels after 1 and 2 weeks.

Prevention is the best management opportunity

PROVIDE GOOD SKIN CARE

Moisture macerates and injures skin. Sources of moisture include sweat, wound drainage, urine, and feces. Several studies indicated that incontinence increases the risk of pressure ulcer development fivefold,[57] but the studies did not distinguish between fecal and urinary incontinence. When urinary incontinence was looked at separately, it had no independent association with pressure ulcers. Fecal incontinence is much more important.[58–60] Although maceration from urinary incontinence may be a risk factor, use of a Foley catheter in an elderly patient solely for incontinence probably confers a greater risk than that of diapering and incontinence itself. Food crumbs, IV tubing, and other debris in the bed can greatly increase local skin pressure points.

PRINCIPLES OF PRESSURE ULCER HEALING

Pressure ulcers are extremely difficult to heal. Once they develop, this type of chronic wound is very resistant to any known medical therapy. Estimates of complete healing for pressure ulcers are as low as 10%.[61] As few as 13% of pressure ulcers heal by 2 weeks in acute hospital settings.[62] In long-term care, the rate of healing depends on the initial stage of the pressure ulcer. Healing rates for stage 3 pressure ulcers (see below 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 4 pressure ulcers heal after 6 months of therapy, but one half of patients admitted with pressure ulcers die during this time period.[63] Thus, prevention offers the best opportunity for management.

Chronic vs acute wounds

Chronic wounds—of which pressure ulcers are the dominant type—differ from acute wounds in several ways. Unlike acute wounds, pressure ulcers do not proceed through an orderly and timely process of healing to produce anatomical or functional integrity.[64] 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.[65] There is no hemorrhage in chronic wounds and less contact between wound and tissue. Platelet release and fibrinolytic activity are diminished. Finally, there are complex polymicrobial colonizations that are poorly understood.[66]

TREATMENT RULE
ASSESS THE PRESSURE ULCER

Several scales have been proposed for assessing the severity of pressure ulcers. The most commonly used scale, recommended by the National Pressure Ulcer Task Force and the Omnibus Budget Reconciliation Act of 1987 (OBRA) nursing home guidelines, is a modification of the Shea scale.[67] This scale classi-

fies pressure ulcers into four clinical stages.

Stage 1 is defined by nonblanchable erythema of intact skin. 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 blanching or capillary refilling. Nonblanchable erythema is believed to indicate extravasation of blood from the capillaries. A stage 1 pressure ulcer always understates the underlying damage since the epidermis is the last tissue to show ischemic injury. Diagnosing stage 1 pressure ulcers in darkly pigmented skin is problematic.[68]

Stage 2 ulcers extend through the epidermis or dermis. The ulcer is superficial and presents clinically as an abrasion, blister, or shallow crater.

Stage 3 pressure ulcers extend through the full thickness of the skin, with 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 4 pressure ulcers are full-thickness wounds with extensive destruction, tissue necrosis, or damage to muscle, bone, or supporting structures. Frequently, adjacent tissue is undermined and sinus tracts are present.

Of the four stages, stage 1 ulcers are most common, accounting for 47% of pressure ulcers. Stage 2 ulcers are a close second at 33%. Stage 3 and 4 ulcers make up the 20% difference.[3]

This staging system has several limitations. The primary difficulty is that one cannot use it to measure progression or healing. Stage 4 ulcers do not always start as stage 1 ulcers and progress through stages 2 and 3, but may appear to develop from "the inside out" as a result of the initial injury. Moreover, pressure ulcers do not heal in reverse order of stages, but rather heal by contraction and scar tissue formation. Therefore, "reverse staging" is inaccurate in assessing healing. Clinical staging is inaccurate unless all eschar is removed, since the staging system only reflects the depth of the ulcer.

Other staging systems that include descriptions of exudate, necrotic material, or eschar have been suggested to monitor the severity of pressure ulcers, but they have not been demonstrated to be better than the Shea scale.[63]

No single measure of wound characteristics has been useful in measuring healing.[69] Several indexes of healing have been proposed, but most lack validation. The Pressure Ulcer Status for Healing (PUSH) tool, developed and validated by the National Pressure Ulcer Advisory Panel, measures three components—size, exudate amount, and tissue type—to arrive at a numerical score. In clinical development and validation studies, the PUSH tool adequately assessed ulcer status and is sensitive to change over time.[70,71]

TREATMENT RULE
RELIEVE PRESSURE, FRICTION, SHEAR

Pressure-relieving devices have a role in treating pressure ulcers. This therapy was successful in studies in hospitals and in nursing homes, but is very expensive. Clinical trials suggest that devices that reduce or relieve pressure are superior to standard mattresses, but there is no clear advantage of one device over another. Pressure relief or reduction is also important when the patient is sitting in a chair or wheelchair.

Wounds need to be kept moist

In a trial in a hospital,[62] pressure ulcers in patients who were randomized to an air-fluidized bed decreased in surface area by a mean of 1.2 cm^2 over 15 days, while ulcers in patients randomized to an alternating air mattress increased by a mean of 0.5 cm^2 ($P = .01$). However, there was no difference in the number of ulcers that shrunk by at least 50%. The cost was estimated at an additional $80 per day.[62]

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. The index ulcer shrank by more than 50% in surface area in 44% of patients. Very few patients had a reduction in ulcer surface area after 1 month of treatment on the specialized bed. The median time to healing was 119 days and time to improvement was 127 days. The additional cost for the bed was $50 to $100 per day.[72]

Low-air-loss beds produced substantial improvement in ulcer size (9.0 vs 2.5 mm^2) compared with a 10-cm convoluted foam mattress in nursing home patients.[73]

TREATMENT RULE
OPTIMIZE WOUND THERAPY

For centuries, wounds have been dressed to protect them from a harmful external environment. The traditional acute wound dressing was an absorptive cover that dried the wound surface. Even simple dressings promote hemostasis, limit edema, reduce pain, and facilitate gas exchange between tissues. However, removal of these dry dressings leads to subsequent secondary trauma.

Occlusive dressings may be better

The modern era in wound healing began in 1958, when Odland[74] observed that a blister healed faster if left unbroken. In 1962, Winter[75] demonstrated in domestic pigs that wounds occluded by a polyethylene film more than doubled their epithelialization rate.

Do not change occlusive dressings until they leak

Several hypotheses have been put forth to explain this effect. Wound fluid is thought to contain a variety of growth factors that may enhance healing, such as interleukin-1, epidermal growth factor, and platelet-derived growth factor-beta.[76] A moist environment may maintain a normal electrical voltage gradient across the wound, which is necessary for epithelial migration.[77] Under an occlusive dressing, wound fluid may sustain increased bacterial overgrowth, stimulating epidermal migration.[78] Wound exudate in chronic ulcers has been found to be an excellent medium for fibroblast stimulation.[79] Removal of this medium by aggressive scrubbing or drying has been shown to be detrimental. Thus, in both acute and chronic wounds, the principal function of a wound dressing is to provide a moist healing environment.

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). 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 a MVTR of 68, and impregnated gauze has a MVTR of 57. In comparison, hydrocolloid dressings have a MVTR of 8.[80] Moist wound healing allows experimentally induced wounds to resurface up to 40% faster than air-exposed wounds.[81] Any therapy that dehydrates the wound such as dry gauze, heat lamps, air exposure, or liquid antacids is detrimental to chronic wound healing.[82–85]

Pain relief. A primary goal of wound dressing should be to relieve pain. Except in neurologically impaired patients, chronic ulcers may be painful. Unfortunately, persons who develop pressure ulcers often are unable to report pain. A decrease in wound pain with occlusive dressings has been noted in donor sites and venous stasis ulcers,[86,87] but studies in pressure ulcers have been limited. In acute wounds, occlusion has been shown to reduce wound pain,[88,89] enhance autolytic debridement,[90,91] and prevent bacterial contamination.

Types of occlusive dressings

Occlusive dressings can be divided into the broad categories of polymer films, polymer foams, hydrogels, hydrocolloids, alginates, biomembranes, and absorbing granules. Each has advantages and disadvantages.[92,93] The available dressings 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.

Polymer films are impermeable to liquid but permeable to gas and moisture vapor. Because they have low permeability to water vapor, these dressings do not dehydrate the wound. Impermeable polymers such as polyvinylidine and polyethylene can macerate 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 absorb exudate and do not eliminate deadspace (space not occupied by viable wound tissues), the presence of which increases the possibility of infection.

Hydrocolloid dressings are complex dressings similar to ostomy barrier products. 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 do not adhere to wound tissue and do not damage epithelization of the wound. The adhesive barrier is frequently overcome in highly exudative wounds. Hydrocolloid dressings cannot be used over tendons or on wounds with eschar formation. Several of these dressings include a foam padding layer that may reduce pressure to the wound.

Hydrocolloid dressings have a theoretical disadvantage in that they are impermeable to oxygen. This could be a problem in wounds contaminated by anaerobes, but this effect has not been demonstrated clinically.

The use of hydrocolloid dressings has been shown to be more cost-effective than traditional dressings, primarily because less nursing time is required for dressing changes.[94,95]

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

Alginates are complex polysaccharide dressings that are highly absorbent, making them particularly suited for exudative wounds. Alginates do not adhere to the wound, but if the wound is allowed to dry, damage to the epithelial tissue may occur with removal.

Biomembranes are very expensive and not readily available.

Comparing the occlusive dressings. Most types relieve pain—only absorbing granules do not. The dressings differ in the ease of application. This difference is important in pressure ulcers in unusual locations, or when considering for home care. Only the hydrocolloid and biomembranes offer bacterial resistance.

Occlusive dressings should be left in place until wound fluid is leaking from the sides, a period of several days to 3 weeks.

Saline-soaked gauze that is not allowed to dry is an effective wound dressing. When moist saline gauze was compared with occlusive-type dressings, healing of pressure ulcers was similar with both dressings.[96–98]

Can topical agents improve healing?

Acute wound healing proceeds in a regulated fashion that is reproducible from wound to wound.

Growth factors hold promise as topical treatments

Growth factors given topically have been demonstrated to mediate the healing process, including transforming growth factors alpha and beta, epidermal growth factor, platelet-derived growth factor, fibroblast growth factor, interleukin-1, interleukin-2, and tumor necrosis factor alpha. The concept of accelerating healing in chronic wounds by using these acute wound factors is attractive. However, in trials in pressure ulcers, platelet-derived growth factor failed to produce complete healing,[99] although it did shorten the time to closure of wounds, as did basic fibroblast growth factor.[100,101] The development of wound healing factors is still in its infancy but shows great promise.

Topical agents that promote healing. Several types of topical wound treatments can promote more rapid epidermal resurfacing, as shown in controlled trials (TABLE 1).[102] 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.

The best method of debridement is controversial

Topical agents that delay healing. Conversely, a number of agents have been show to delay healing in controlled trials (TABLE 2). Some agents have concentration-dependent effects, a problem that confounds physicians and researchers alike. Hydrogen peroxide in a 20% solution is an enhancer, while a 3% solution delays healing. The common characteristics of agents that delay healing stem from either wound-drying effects or cytotoxic effects on epidermal cells. Certain antiseptic agents are cytotoxic to human fibroblasts, including povidone-iodine (Betadine), chlorhexidine gluconate (Hibiclens), hexachlorophene (pHisoHex), benzalkonium chloride, and trypsin in balsam of Peru and castor oil (Granulex, others).[95,103–105] In human pressure ulcers, Dakin's solution 0.05% was clearly inferior to a hydrocolloid dressing.[91]

TREATMENT RULE
REMOVE NECROTIC DEBRIS

Necrotic debris increases the possibility of bacterial infection and delays wound healing.[106] This delay is due to the slow removal of debris by phagocytosis.

The preferred method of debriding the wound remains controversial. Options include mechanical debridement with gauze dressings, sharp surgical debridement, autolytic debridement with occlusive dressings, and application of exogenous enzymes.

Surgical sharp debridement produces the most rapid removal of necrotic debris and is indicated in the presence of infection. However, 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. Remoistening of gauze dressings in an attempt to reduce pain can defeat the debridement effect.

Autolytic and enzymatic debridement both require several days to several weeks to achieve results. Thin portions of eschar can be removed by occlusion under a semipermeable dressing. Penetration of enzymatic agents is limited in eschar and requires either softening by autolysis or crosshatching by sharp incision prior to application. Enzymatic debridement can dissolve necrotic debris, but possible harm to healthy tissue is debated. Enzymes available in the United States for topical debridement include collagenase, papain, and a papain-chlorophyll combination. In clinical trials, collagenase reduced necrosis, pus, and odor compared with inactivated control ointment,[107] and produced debridement in 82% of pressure ulcers at 4 weeks compared with petrolatum.[108] Papain produced measurable debridement in 4 days compared with a control vehicle ointment.[109] 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 the groups.[110] Trypsin in balsam of Peru and castor oil was not better than mechanical gauze debridement.[111]

TREATMENT RULE
MANAGE BACTERIAL CONTAMINATION

Bacteria destroy tissue; however, chronic wounds do not appear to follow the usual rules.

Eleck[112] demonstrated that normal skin flora in numbers greater than 10^5 organisms per mL produces local disease in intact skin. In damaged skin, fewer organisms are required to produce infection. Skin grafts and flaps will not heal if more than 10^5 organisms of certain species of bacteria per mL are present.[113]

In chronic wounds, organisms in numbers greater than 10^5 per mL may persist for months or years in chronic wounds without apparent clinical effect. Therefore, quantitative microbiology alone is a poor predictor of clinical infection in chronic wounds.[114]

Colonization with bacteria is common and unavoidable. All chronic wounds become colonized, usually with skin organisms, followed in 48 hours by gram-negative bacteria. The presence of microorganisms alone (colonization) does not indicate an infection in pressure ulcers.

The diagnosis of infection in chronic wounds 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.[115] Often it is difficult to determine the presence of an infection in a chronic pressure ulcer.

Use of antibiotics

An empiric trial of topical antibiotics is indicated in wounds that fail to progress toward healing. The species of bacteria may make a difference. *Pseudomonas aeruginosa* was found in 88% of worsening pressure ulcers and *Providencia* species in 34%, compared with 0% of stationary wounds and 7% of rapidly healing ulcers. *Peptococcus, Bacteroides* species, or *Clostridia* was 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.[116,117] Based on these findings, the presence of *P aeruginosa* and *Providencia* species should not be regarded as simple colonization.

When there is evidence of clinical infection, topical or systemic antimicrobials 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.[118] Topical gentamicin and silver sulfadiazine have been shown to improve the clinical appearance of infected wounds and may improve healing.[119,120] Iodine and thimerosal have been noted to increase pain and delay healing.[121] Infections with anaerobes may respond to topical metronidazole.[122] Systemic antibiotics are indicated when the clinical condition suggests the infection has spread to the blood stream or bone.

Use of occlusive dressings

Healing of chronic wounds is enhanced under occlusive dressings even though they increase both the absolute number and variety of species of organisms. Bacterial infections in chronic wounds appear to be primarily the result of superinfection due to contamination. Therefore, protecting 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[123] found that extracts of wound fluid under hydrocolloid dressings were capable of inhibiting growth of *P aeruginosa* and *Staphylococcus aureus* in vitro. Wounds with extensive undermining create pockets for infection, with an increased likelihood of infection with anaerobic organisms.[124] Obliteration of dead space reduces the possibility of infection.

Despite an increase in numbers of bacteria, occlusive dressings very rarely cause a clinical infection. Hutchinson and McGuckin[124] reviewed 36 studies comparing infection rates under occlusive dressings vs gauze or impregnated gauze. Infection rates were 2.6% for occlusive dressings and 7.1% for nonocclusive gauze.

Do not regard *Pseudomonas aeruginosa* or *Providencia* as colonization

TREATMENT RULE
CORRECT NUTRITIONAL DEFICITS

The most important reversible host factor contributing to wound healing is nutritional status. Several studies suggest that dietary intake, especially of protein, is important in healing pressure ulcers.

Observational protein trials

Greater healing of pressure ulcers has been reported with higher protein intake irrespective of positive nitrogen balance.[125]

Allman et al[62] reported on the nutritional status of 65 patients with pressure ulcers in a trial of air-fluidized bed therapy. The most significant characteristic associated with improvement in pressure ulcers was dietary intake of protein. No other single marker of nutritional status, including serum albumin, total lymphocyte count, or history of weight loss, was related to improvement. However, dietary intake of protein was estimated only at baseline and subsequent dietary intake was not recorded.

Trials of protein supplementation

Breslow et al[126] enrolled 48 patients with stage 2 through 4 pressure ulcers in a dietary intervention trial. Malnutrition was defined as a serum albumin level less than 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 had healing of their pressure ulcers at a greater rate than those fed a 14% protein standard diet. However, there were no differences between the groups in changes in body weight or in biochemical measures of nutritional status. The study was limited by a small sample size (28 patients completed the study), nonrandom assignment to treatment groups, confounding effects of air-fluidized beds, and the use of two different feeding routes.

Chernoff et al[125] randomized 12 enterally fed patients to receive formulas containing either 17% or 25% of calories as protein. The group that received 1.8 g/kg of protein had a 73% decrease in pressure ulcer surface area compared with 42% in the group receiving 1.2 g/kg of protein. The high-protein group began the study with larger surface area pressure ulcers (22.6 cm^2 vs 9.1 cm^2). The serum albumin level did not appear to be a predictor of the development of pressure ulcers or healing rate, although values were not given.

Patients with pressure ulcers may need more protein than currently recommended

The optimum dietary protein intake in patients with pressure ulcers is unknown, but may be much higher than the current adult recommendation of 0.8 g/kg/day. Half of chronically ill elderly persons are unable to maintain nitrogen balance at this level.[127] On the other hand, increasing protein intake beyond 1.5 g/kg/day may not increase protein synthesis and may cause dehydration.[128] A reasonable protein requirement is therefore between 1.0 and 1.5 g/kg/day.

A reasonable protein requirement is 1–1.5 g/kg/day

Vitamin supplementation is controversial

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.[129] Zinc supplementation has not been shown to accelerate healing except in zinc-deficient patients.[130] High serum zinc levels interfere with healing, and supplementation above 150 mg/day may interfere with copper metabolism.[131,132] Zinc deficiency may be common in elderly subjects.

SURGICAL MANAGEMENT

Surgical closure of pressure ulcers results in more rapid resolution of the wound. The chief problems are that the ulcers frequently recur, and many frail patients cannot tolerate the procedure. The efficacy of surgical repair of pressure ulcers is high in the short term. However, its efficacy in the long term has been questioned, even in younger patients.[133]

Nowhere does the difference in pressure ulcers between younger spinal-cord injury patients and elderly patients become so pronounced as in discussing surgical management. In one series,[134] 40 patients selected for surgical closure of pressure ulcers were divided into three groups: traumatic paraplegics (mean age 32 years), nontraumatic paraplegics (mean age 22 years), and nontraumatic nonparaplegics (mean age 73 years). In elderly nontraumatic nonparaplegic patients, 84% of surgically treated pressure ulcers were healed at discharge. Twelve percent of surgically treated patients in this group had another pressure ulcer at discharge. Within 7.7 months, 40% of the surgically treated pressure ulcers in this group recurred and 69% of the patients had a pressure ulcer at a different site.

In the (younger) 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.[134] After 10 years of follow-up in 16 surgically treated patients, only 1 patient remained alive and free of pressure ulcers.[135]

A decision analysis[136] demonstrated that myocutaneous flap procedures for stage 3 pressure ulcers were favorable unless the success rate for surgery was less than 30% or the healing rate with medical therapy was more than 40%. The added cost for the procedure was estimated at $17,000 per treatment episode compared with medical therapy.

In spinal cord injury patients, the rate of surgical complications and recurrence is high. Surgical complications occurred in 40% of patients, and ulcers recurred or new ulcers developed in 79.2% of patients.[137]

COMPLICATIONS OF PRESSURE ULCERS

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

Increased 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.[66] Patients who develop a new pressure ulcer within 6 weeks after hospitalization are three times as likely to die as patients who do not develop a pressure ulcer.[138]

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.[59] Residents in a skilled nursing facility who had pressure ulcers had a 6-month mortality rate of 77.3%, while patients without pressure ulcers had a mortality rate of 18.3%.[69] 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.[139]

It is not clear how pressure ulcers contribute to increased mortality. Although several investigators found a threefold increase in mortality with the development of a new pressure ulcer, the severity of the pressure ulcer did not correlate with increased risk. Patients with stage 2 pressure ulcers have been equally likely to die as patients with stage 4 pressure ulcers.[139] In the absence of complications, it is difficult to imagine how stage 1 or 2 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, in which pressure ulcers were associated with an increased rate of mortality but not with the rate of acute hospitalization.[138]

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,[140] 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 2.4), the admitting physician's estimate of life expectancy of less than 5 years (relative risk 2.1), and the comorbidity damage index score (relative risk 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.

Needle biopsy of bone is the best test for osteomyelitis

Osteomyelitis

Osteomyelitis is a frequent complication of pressure ulcers, reported in 38% of patients with infected pressure ulcers.[141] Diagnosing

contiguous osteomyelitis in pressure ulcers is difficult. Plain radiographs cannot differentiate true osteomyelitis from pressure changes to bone.[142] Radionuclide studies, including technetium-99m and gallium-67 scans, are sensitive but have a false-positive rate of 41%.[141] Computed tomography may be more useful, with a specificity of 90%, although the sensitivity is only 10%.[143] Needle biopsy of bone is the most useful single test, with a sensitivity of 73% and a specificity of 96%.[144]

Sepsis

Bacteremia from pressure ulcers is uncommon, but probably underestimated. The incidence of bacteremia from pressure ulcers is about 1.7 per 10,000 hospital discharges.[145] 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. The overall mortality rate 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.[146] In 51 elderly patients with bacteremia attributed to pressure ulcers, 33% had polymicrobial isolates.[145]

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