Medical Management of Pressure Sores

Aubrey L. Knight, MD
Roanoke, Virginia

The pressure sore is a common clinical problem that is frequently seen by family physicians. Prevention is the best treatment, but once the condition occurs, adherence to several principles makes treatment easily standardized. These principles include improvement in the general health of the patient, relief of pressure, cleanliness, disinfection, and stimulation of granulation tissue. Any treatment regimen that is to be considered for use should achieve these goals. This article reviews the treatment options available and gives recommendations for the medical management of pressure sores based on these principles and the available data.

Pressure sores are frequently encountered as a clinical problem, partly because of the increasing population in long-term care facilities and rehabilitation units. Pressure sores are defined as changes in the skin and underlying tissue resulting from pressure, shear, or friction. If not attended, these forces will cause ulceration. These sores are commonly called decubitus ulcers, but in this instance this term is a misnomer, as decubitus is derived from the latin meaning "lying down," which is not a prerequisite for the development of these changes.[1]

The best treatment for pressure sores is prevention, but even under the best conditions, prevention is not always possible. A multitude of treatments has been asserted to be effective in the management of pressure sores, yet treatment remains one of the more difficult clinical tasks. To clarify the factors associated with pressure sores and thereby relate possible modes of therapy to these factors, a discussion of the epidemiology, etiology, classification, and prevention will precede a review of the treatment options for pressure sores.

EPIDEMIOLOGY

The incidence of pressure sores is greatest among patients with spinal cord injuries,[2] cerebrovascular disease, and long hospital confinements[3,4] as well as in the elderly population,[5,6] particularly those in long-term care facilities. Allman et al[7] found that 5 percent of hospital patients had pressure sores, and 12 percent were at risk because they had been confined to a bed for at least one week. Hypoalbuminemia, fecal incontinence, and fractures were the most important factors associated with the presence of pressure sores in that group.

Other factors that may contribute to the likelihood of developing pressure sores include volume depletion, both increased and decreased body weight, anemia, renal failure, diabetes, malignancy, sedation, major surgery, vitamin deficiencies, and many metabolic disorders.[1–9] Edema compromises the blood supply to the overlying skin and increases the distance between the skin and blood supply, thus reducing the efficacy of oxygen delivery to the skin. Finally, the aging skin itself, its accompanying reduced epidermal thickness and elasticity, increases the risk for ulceration.

Patient neglect, a problem that may be overlooked by physicians, needs to be considered as a contributing factor, particularly when there are repeated pressure sores.

In the study by Allman and his colleagues, the patients with pressure sores and those at risk for the development of pressure sores showed a high utilization of medical resources and had a mean length of hospitalization of 5.0 and 3.5 times greater, respectively, than that noted for all patients hospitalized.[7] A 1980 survey revealed that medical costs resulting from pressure sores can increase from $2,000 to $10,000 per patient, with a national annual cost of $3 billion to $5 billion.[10] In one study the in-hospital mortality rate of patients with pressure sores was found to be five times as great as among all patients hospitalized.[7]

ETIOLOGY

Pressure sores generally develop in focal areas over bony prominences. Ninety percent of the lesions are on the

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From the Department of Family Medicine, University of Maryland School of Medicine, Baltimore, Maryland. Requests for reprints should be addressed to Dr. Aubrey L. Knight, Department of Family Practice Education, Roanoke Memorial Hospitals, Belleview at Jefferson Streets, Roanoke, VA 25033.

lower part of the body; the sacrum, greater trochanter, ischial tuberosity, malleoli, and heels are the most common sites.[11] Pressure sores may form at any body site overlying a bony prominence, however, including elbows, occiput, scapulae, knees, and spinous processes. Patients are exposed at these areas to the four primary mechanisms in the development of pressure sores: pressure, shearing forces, friction, and moisture.[1-11] Pressure over a bony prominence will cause trauma to the tissues. The first step in ulceration is reactive hyperemia, which will persist for about 24 hours without ulceration. If pressure is not relieved at this time, ulceration ensues. The amount of time to ulceration may be less if any of the risk factors mentioned above are present.

Shearing forces are caused by the sliding of adjacent surfaces, producing a relative displacement and resulting in compression of capillary flow. Subcutaneous fat is particularly vulnerable to the effects of shearing forces. Elevation of the head of the bed by more than 30 degrees causes the body to slide down, producing a shear in the sacral and coccygeal region.

Friction is the force created when two surfaces move across each other, such as would occur with maneuvering a patient on the bed. The impact of friction damages the outer protective skin layer, especially vulnerable in the elderly, thus accelerating the onset of ulceration.

Moisture, most frequently resulting from soiling by urine or feces and by perspiration, will also increase the risk of pressure ulceration. A high correlation exists between urinary and fecal incontinence and ulceration.[3] In paraplegic patients, the moisture of excessive sweating in the area of denervation may macerate the skin.

CLASSIFICATION

The most widely used classification scheme for pressure sores was first described by Shea in 1975.[12] This scheme divides pressure sores into four grades, depending on the tissue involvement.

A grade I pressure sore is an acute inflammatory response in the layers of the skin. The clinical presentation of a grade I pressure sore is a well-defined area of soft-tissue swelling, erythema, and heat, usually over a bony prominence. The epidermis remains intact; thus recognition of a grade I sore is essential because it is a reversible lesion treated by relief of pressure and attention to correction of other reversible risk factors.

A grade II pressure sore is an extension of the inflammatory response leading to a fibroblastic response. Clinically, a grade II sore presents as a break or blistering of the epidermis, with surrounding erythema or induration. The grade II pressure sore is also a potentially reversible lesion; thus treatment is directed toward preventing further insult.

A grade III pressure sore is an inflammatory response that progresses through the dermis and into the subcutaneous layers. From this point rapid spread is likely to occur. The clinical presentation of a grade III pressure sore is that of the classic "decubitus ulcer." One finds an irregular full-thickness ulcer extending into the subcutaneous fat surrounded by erythema, induration, and heat. There is often a draining, foul-smelling, necrotic base. Typically, these ulcers do not heal with relief of pressure alone. The risk of sepsis is high[13]; consequently, attention to bacterial flora is important, and the use of antibiotic therapy is often necessary.

A grade IV pressure sore penetrates the deep fascia, thus eliminating the last barrier to extensive spread. This penetration leads to involvement of muscle or bone. Clinically, a grade IV sore resembles a grade III sore except that bone, joint, or muscle can be identified. Grade IV pressure sores can lead to osteomyelitis, septic joints, and so on, thus creating an extremely toxic condition that may prove fatal.

PREVENTION

To prevent pressure sores, the most effective measure is frequent positional changes. Exton-Smith and Sherwin[14] found that elderly patients who changed position fewer than 20 times per night developed pressure sores. This study highlights the importance of turning debilitated patients, either through nursing care or the use of various devices.

Repositioning patients at least every two hours, including those in wheelchairs, is essential in the prevention of ulcers. When repositioned, the patient should be lifted, not dragged, from a bed or wheelchair to avoid friction and the subsequent damage to the epidermis. At the time of positioning, the patient's skin should be examined for areas of redness suggesting early pressure changes. Routine skin care should include keeping the skin clean and free of stool, urine, and moisture.

Special pads and beds as well as mechanical devices are available and are intended to alter pressure over bony prominences. Devices used for specific body sites, such as gel pads,[15] foam cushions, wheelchair cushions, and sheepskin pads,[16] are practical for use in prevention for specific areas. No single device has yet been developed that is effective in the prevention of all pressure sores. Special mattresses, such as the water mattress,[17-19] the alternating pressure mattress,[20,21] the air-fluidized bed,[22] and the Clinitron bed,[23] however, aid in the prevention and treatment of pressure sores. These beds tend to relieve

pressure by using air or buoyancy to keep the patient's weight evenly distributed.[24] Such devices, however, should not be relied upon as a substitute for basic nursing care.

TREATMENT

There is general agreement that once a pressure sore develops, treatment involves adherence to five basic principles:[1,4,25,26]

1. Improvement in the patient's general health, thereby eliminating, if possible, those factors that contributed to the problem
2. Restoration of blood supply to the sore by relief of pressure
3. Keeping the wound clean
4. Disinfection
5. Stimulation of granulation tissue

Attention to the general health of the patient includes correction of those conditions contributing to the development of pressure sores such as incontinence, anemia, hypoalbuminemia, edema, and so on. Just as such specific devices as padding and special beds are aids in the prevention of pressure sores, they also may be helpful, along with strict attention to positioning, in returning blood flow to the area of breakdown.

Many of the agents that are considered helpful in the treatment of pressure sores are based on the third through fifth principles listed above. Specific remedies should achieve the goals of one or more of the above principles prior to becoming a standard of treatment. The many agents that have been used in attempts to treat pressure sores fall into the following categories: topical agents, dressings, physical agents, and systemic agents.

Topical Agents

Many topical agents have been reported to be successful in the treatment of pressure sores, and herein lies the confusion. Despite many enthusiastic claims of successful treatments,[8,27–47] there are few well-controlled studies.

Most topical agents have a limited effectiveness, and some may actually be harmful.[48] For example, both neomycin and vitamin E may produce an allergic dermatitis, and neomycin can cause ototoxicity and nephrotoxity.[48] Liquid antacids are dehydrating and therefore are not usually indicated.[48]

Topical antibiotics have been used and studied but have not been proven to be superior to careful cleansing and wet-to-dry dressings. In addition, topical antibiotics may sensitize the tissue, promote the appearance of resistant organisms, and cause systemic toxicity.[4,49] Many of the studies of topical antibiotics offer no supporting data or have no controls. Such topical antibiotics as neomycin,[26] gentamicin,[27] and silver sulfadiazine[29] have been studied.

Metronidazole has been recommended as an adjunct to the treatment of pressure sores based on a hastening of healing by control of anaerobes.[30–32] It has been used both topically and administered orally. The results are encouraging, but further study is needed to prove the efficacy and to elicit the preferred route of administration of this medication.

Sugar, honey, and other monosaccharides have been applied to open pressure sores.[33,34] The exact mechanism of sugar treatment is unknown, but several theories exist. It has been suggested that bacteria will digest the sugar rather than the living tissue. Another theory is that sugar provides nutrients to the tissue itself, as a lack of nutrients contributes to the cellular necrosis.[24]

Pace[35] used several preparations containing benzoyl peroxide in a protocol for the treatment of pressure sores. The theory behind the use of benzoyl peroxide is that it is an inexpensive method of supplying oxygen to the tissue. Results were impressive, but once again the study was uncontrolled.

Heavy metal ions such as gold leaf have been reported to heal pressure sores. Wolf et al[36] exhibited the effectiveness of gold leaf, but only when control of infection and anemia had been achieved. Other heavy metal ions that have been reported to be effective against pressure sores include bismuth,[37] zinc,[38,39] and titanium.[37] The therapeutic rationale behind these compounds is that they cause epithelialization of cells and a mild foreign body response that promotes healing.

Freeman and Joyner[40] treated 24 patients with decubitus ulcers using an absorbable gelatin sponge. All ulcers healed, but there was concomitant use of antibiotics and nursing care, with no control group utilized.

Another category of topical agents comprises those agents that act to debride the tissue enzymatically. Because necrotic tissue prevents healing and creates favorable conditions for bacterial contamination, dead tissue must be removed to promote healing. Removal of necrotic tissue can be accomplished by either surgical or enzymatic debridement.

Surgery is quicker and generally more effective than chemical debridement. For the eschar that often covers the ulcer, surgery is the only effective means of removal. The risks of surgical debridement, however, are hemorrhage, pain, possible loss of normal tissue, and bacteremia.[4]

It is recommended that enzymatic debridement be reserved for intervals between surgical debridement to help dissolve thin necrotic layers that are less accessible to excision.[26] The inability of these agents to penetrate eschar

or to remove large amounts of tissue limits their usefulness. In addition, many of the enzymes are inactivated in the presence of soaps and heavy metal ions.[25]

Agents that enzymatically debride tissue do so by a variety of mechanisms. Collagenase[41] is a proteolytic enzyme, whereas streptokinase-streptodornase[42] is an agent that transforms plasminogen into the active proteolytic enzyme, plasmin. Dextranomer[43–45] is said to clean by drawing exudate, bacteria, and other serologic substances into its beads, which are then washed away during dressing changes and wound irrigations.[43] An aerosol foam spray[8,45] is on the market that contains trypsin, an enzyme that acts to debride necrotic tissue, as well as balsam Peru, reported to have a bactericidal effect, and castor oil, which is said to improve epithelialization. Cadexomer iodine[47] is a compound designed to debride ulcers while releasing the antiseptic iodine at the ulcer surface.

When used in the proper clinical context, enzymatic agents have a place in the treatment of pressure sores. Since there are no good studies comparing the different agents, the clinician should be guided by his or her own experience as well as by the convenience and price of the individual products.

Dressings

Whereas the traditional approach to the treatment of pressure sores has been the use of wet-to-dry dressings, more recently several types of occlusive dressings have been reported to be effective in the treatment of pressure sores.[50–53] These dressings include the adhesive hydrocolloid occlusive dressing (HCD), the polyurethane film dressing (PUFD), and the moisture vapor-permeable dressing (MVP).

Alvarez et al[50] compared the effects of HCD, PUFD, and wet-to-dry gauze on superficial wounds in swine. Re-epithelialization was found to be increased under the adhesive oxygen-impermeable HCD and the oxygen-permeable PUFD. Gorse and Messner[51] compared HCD with wet-to-dry dressings in a controlled study and found that 87 percent of sores "improved" with HCD, while 69 percent of sores "improved" with wet-to-dry dressings. PUFD was evaluated in 24 geriatric patients,[52] and a greater degree of healing was reported with PUFD than in the control group.

Whereas the gauze should be changed several times daily, the appeal of the occlusive dressings is that they can usually remain on the pressure sore for several days. This convenience is particularly useful in the outpatient management of pressure sores. Sebern[53] compared traditional gauze dressings with MVP dressings in the home care setting. In grade II pressure sores, there was a 100 percent rate of healing in the MVP-treated group compared with a 52 percent rate of healing in the group that received the more traditional treatment. The healing rates for grade III ulcers were not significantly different in the two dressing groups; however, there was a lower labor and supply cost with the use of the MVP dressing.

The occlusive dressings seem to have a role primarily in the treatment of grade I, grade II, and superficial grade III pressure sores, as the deeper sores of grade III and IV will require more frequent clinical assessment.[51]

Physical Agents

Hydrotherapy, particularly the whirlpool bath, is effective as well in aiding in the disinfection of the wound. This therapy is generally reserved for sores on the lower extremities.

Hyperbaric oxygen was first described to heal pressure ulcers in a case report of an 86-year-old man.[54] The application of oxygen under pressure requires the use of a complicated hyperbaric oxygen chamber. Using hyperbaric oxygen tends to keep the patient off the ulcer for long periods of time, which may have as much to do with the healing as the oxygen itself.

High-frequency, high-intensity sound waves (ultrasound) directed at pressure sores are thought to hasten healing by breaking down cellular obstruction and increasing local circulation[24] as well as by stimulating protein synthesis.[55,56]

Electrotherapy was used on 83 ulcers caused by pressure, venous stasis, and atherosclerosis in one study.[57] Thirty-four lesions healed over a 15-week period, although there was a high dropout rate. In a study in which ulcers were said to have "failed prior conventional therapy,"[58] using the microelectric medical stimulation (MEMS) instrument (a devise that delivers a very low electric energy) resulted in impressive shrinkage in the surface areas of the affected tissue. The project sample, however, consisted of only six patients, and the MEMS was used as an adjunct to preexisting treatment.

The carbon dioxide surgical laser unit has been used in the treatment of pressure sores based on two effects. Not only is the laser unit capable of vaporizing the necrotic tissue in the ulcer, but also it can be used to remove the pressure producing bony prominences.[48]

Systemic Agents

Several systemic agents have been described as being helpful in the management of pressure sores. Systemic antibiotics are generally reserved for the treatment of such complications as cellulitis, abscess, osteomyelitis, and bacteremia.

Zinc sulfate administered orally helps accelerate wound healing in the zinc-deficient patient.[10] It is postulated that the beneficial effects of zinc are related to an improvement

in circulation to an ischemic area as well as to the role that zinc plays in protein synthesis.[59] No convincing data have demonstrated the value of routine use of zinc in the treatment of pressure sores, however. Hallbook and Lanner[60] demonstrated an accelerated rate of healing when patients who had low serum concentrations of zinc were administered oral zinc. There were, however, no changes in healing rates when zinc was administered to patients with a normal serum zinc.

Better evidence exists to justify the routine use of ascorbic acid as an adjunct in the management of pressure sores.[61] Vitamin C is thought to exert its effect on collagen formation.

The practice of administering insulin in small doses as a part of decubitus management programs was once popular and is still considered useful by some.[62] Appetite improvement has been offered as an explanation for the potential benefit.

CONCLUSIONS

This review illustrates the great confusion and complexity that surrounds the treatment of pressure sores. There are literally hundreds of agents that are touted to improve healing, but until adequate trials can prove the efficacy of these agents, conservative therapy remains the preferred method.

For grade I and grade II pressure sores, strict nursing care and attention to cleanliness will frequently be all that is necessary. There seems to be good evidence that occlusive dressings, when used in grades I and II sores, are capable of accomplishing many of the goals in local care of wounds:[63] prevention of contamination, dermal and epidermal repair, and protection of the surrounding skin. As mentioned earlier, however, the use of occlusive dressings in more extensive sores is not currently recommended, as these sores will require more frequent clinical assessments and often need packing.

The more extensive sores require strict adherence to the five basic principles: (1) improvement in the general health of the patient, (2) pressure relief, (3) cleanliness, (4) disinfection, and (5) stimulation of granulation tissue. General medical care should focus on nutritional repletion and correction of anemia, edema, and metabolic abnormalities. Strict nursing care with regard to positioning is important, as the patient must not lie on the ulcer. Pressure-relieving devices should be used to aid in preventing further breakdown in the susceptible patient.

Debridement is often a necessary aid in disinfection as well as in the stimulation of granulation tissue. Initial management concerning debridement should include the use of wet-to-dry dressings soaked in normal saline and removed every three to four hours. Large, deep ulcers are commonly debrided in whirlpool baths. The water of the whirlpool can serve to soften the eschar and wash bacteria from the wound.

Surgical debridement is necessary for the removal of the eschar that often forms within the ulcer crater. Enzymatic debridement should be used at intervals between surgical debridement to dissolve the thin necrotic layers that are less accessible to surgery.

Surgical closure is an alternative to allowing the ulcer to heal by secondary intention. Patients who might benefit from surgical closure of their pressure sores include those whose ulcer is greater than 2 cm in diameter and who can tolerate the surgery.[64]

The use of other topical preparations on the ulcer will frequently be counterproductive, and unless such agents can be shown in well-controlled studies to achieve one or more of the goals of therapy without the potential for serious untoward effects, their use is not recommended.

Despite the frequency of pressure sores, the pathophysiology and management are poorly appreciated by physicians. Adherence to preventive techniques in all patients who are at risk is the most effective method of combating this potentially life-threatening complication.

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