Decubitus Ulcers

Ronald W. Perdue, M.D., and Jim L. Wilson, M.D.

Abstract: Decubitus ulcers are frequently encountered in the elderly and in bedridden or wheelchair-bound patients. Successful treatment is often difficult and expensive. Pressure, time, and friction are the major factors involved in the development of skin ulcers with such risk factors as age, female sex, and nutritional status predisposing to their development. Complications can be life threatening. A number of treatment modalities have been used over the years, but the mainstays of treatment include attention to preventive measures, relief of pressure over bony prominences, and debridement, following the basics of wound care. (J Am Bd Fam Pract 1989; 2:43-8.)

Decubitus ulcers have been the bane of physicians and nurses alike for centuries. They occur in approximately 3 percent of all patients admitted to acute care facilities.[1] In 1966, it was estimated that medical cost to a patient with decubitus ulcers would be approximately $5,000 per year.[2] More recent estimates indicate that this figure may now reach as high as $30,000 per patient per year.[3] In addition, each year in the United States, there are approximately 17,000 cases of litigation involving decubitus ulcers.[3] Because of the increasing numbers of patients who are at risk for developing decubiti, it is important for primary care physicians to understand the risks, pathophysiology, and treatment of decubitus ulcers.

Development of Decubitus Ulcers

Decubitus ulcers are the result of tissue necrosis produced by ischemia that is secondary to pressure exerted over bony prominences. Immobility from any cause is the primary offender in their development, maintenance, and recurrence. For this reason, the infirm elderly and patients with spinal cord injuries are at high risk.

Multiple layers of soft tissue cover the bony prominences of the body and vary in thickness and type depending on anatomic location and nutrition. Skin is the most superficial layer and is composed of an avascular epidermis and a vascular dermis. It serves as a protective barrier, a temperature regulator in water and fat excretion, and as a major sense organ. The skin is cushioned on loose connective tissue composed of fat cells having little intercellular substance and vascularity. This layer is compressible, provides a measure of padding and shock absorption, and permits skin movement over the deeper structures. This layer of subcutaneous fat is extremely vulnerable to shear forces from lateral pressure, which may compromise its blood supply by angular stretching of the vessels and lead to extensive tissue damage and undermining of the ulcer edges. The subcutaneous fat lies on relatively avascular deep fascia comprised of almost pure collagen that is fairly resistant to mechanical forces and vascular compromise. Beneath the fascia lie muscles, periosteum, and bone, which also may be involved in more advanced ulcers.[4]

Three major factors contribute to the development of skin ulcers—pressure, time, and friction. Pressure in the range of 40–100 mmHg is sufficient to produce ischemia and tissue necrosis; however, the duration of pressure appears to be more important than the actual pressure exerted.

In animal studies, cellular ischemia was secondary to microvascular obstruction.[5] Two hours of compression at 60 mmHg led to a reactive hyperemia with recirculation in all microvessels after decompression. During the next 2 hours, platelet and red cell aggregation and white cell adhesion to vascular walls led to a 60 percent decrease in the number of patent microvessels. After 4 hours of compression, recirculation was slow; only 30 percent of the microvessels reperfused, and the tissue became edematous during the time of recirculation. Necrosis often occurred without obvious initial morphological changes in tissue cells, but vascular changes always correlated with the occurrence of necrosis. Pressure to 70 mmHg applied constantly for 2 hours produced irrevocable changes; however, when pressure was alternated with release every 5 minutes, few if any changes were noted.[6]

From the Department of Family Practice, University of South Alabama, Mobile. Address reprint requests to Jim L. Wilson, M.D., Professor and Residency Director, 1504 Springhill Avenue, Mobile, AL 36604.

J Am Board Fam Pract: first published as 10.3122/jabfm.2.1.43 on 1 January 1989. Downloaded from http://www.jabfm.org/ on 7 November 2026 by guest. Protected by copyright.

Because such low pressures can cause decubitus ulcers, the pressures generated by routine activities, e.g., lying or sitting, seem staggering. The pressure over the ischial tuberosities when sitting in an unpadded chair approaches 300 mmHg. This is more than four times the pressure needed to cause irrevocable microvascular changes in the intervening tissues in just 2 hours. This does not occur normally because of frequent weight shifts that redistribute blood flow in the affected area. However, in those who are immobile, comatose, or hypoesthetic, the mechanisms necessary to trigger frequent weight changes are lost. Padding with a 2-inch foam pad will reduce pressures over the ischial tuberosities to 150 mmHg. This pressure is maintained even when up to 6 inches of padding is used. Areas that are especially prone to decubitus formation in the supine or prone positions include the sacrum, greater femoral trochanters, ear lobes, occiput, spine, scapulae, heels, malleoli, fibular head, knees, anterior and posterior iliac crests, elbows, and chest wall.[5]

Other factors that may decrease the amount of pressure and the time necessary for ulcer formation include shearing forces and friction. Shearing forces disrupt the blood supply to the fat cells of the subcutaneous tissue and allow for separation along fascial planes. This occurs most frequently in the region of the sacrum and is at least partially related to elevation of the head of the bed above 30 degrees in bedridden patients. Sacral ulcers may be extended to the scapula or communicate with trochanteric or ischial ulcers. Friction over bony prominences because of wrinkled bedclothes or sliding the patient across surfaces may initiate blisters or abrasions and may lead to pressure sore formation.[5]

Classification

Many classifications of decubitus ulcers have been used. A modification of Shea's classification[4] and other similar modifications[7] are clinically applicable. In this schema, a grade I pressure area is an acute inflammatory response involving the epidermis. It is an irregular, ill-defined area of soft tissue erythema accompanied by induration and heat that persists for more than 24 hours. The epidermis remains intact, and changes are reversible.

A grade II pressure is characterized by blistering or breaking of the epidermis surrounded by erythema and induration. This stage is also potentially reversible. A grade III ulcer is an inflammatory fibroblastic response extending through the dermis to the junction of the subcutaneous fat. Clinically, it is an irregular, shallow ulcer with subcutaneous fat at its base and is surrounded by erythema, induration, and heat.

A grade IV ulcer extends through the full thickness of skin into the deep fascia and muscle. The base often consists of draining, necrotic, foul-smelling tissue, and undermining of the surface tissues may be excessive. A grade V ulcer penetrates the underlying bone, causing osteomyelitis. It has no anatomic limit and is surrounded by erythema and induration. Clinically, it presents as an extensive ulcer with exposed bone, joint, muscle, or fascia at its base.

Risk Factors

In a study performed at a 300-bed community teaching hospital in New England, women patients developed pressure sores more frequently than men,[8] 25 percent compared with 12.8 percent of men. There was also a linear relation between skin integrity and age; 96.9 percent of patients less than 35 years of age had intact skin, while only 18.8 percent of patients more than 84 years of age were uneffected. Other identifying risk factors from greatest to least frequency were: altered nutritional status, impaired activity, impaired mobility, incontinence, and altered mental status. In another study of 634 hospitalized adult patients, 17 percent were at risk or had pressure sores. The greatest risk factors in bedridden patients were hypoalbuminemia, fecal incontinence, and fractures.[9]

Several other factors also predispose to ulcer formation. These include sensory loss, motor paralysis, malnutrition, spasticity and joint contractures, anemia, edema, infection, diabetes, dehydration, and oversedation.[10,11] These predisposing factors will also delay wound closure once a decubitus ulcer is established.

Basics of Wound Care

A grade I pressure sore will heal within 24 hours by definition and requires only relief of pressure over the affected area. Decubiti of grade II through grade III heal by secondary intention. A clean, noninfected wound will heal by the process of epithelialization from the wound edges followed by contraction and finally scar tissue formation.

J Am Board Fam Pract: first published as 10.3122/jabfm.2.1.43 on 1 January 1989. Downloaded from http://www.jabfm.org/ on 7 November 2026 by guest. Protected by copyright.

Grades IV and V will in most cases require surgical intervention beyond simple debridement.

As with any wound, infection is present and will slow the healing process if bacteria exceed 10^5 organisms per gram of tissue.[5] Sepsis associated with decubiti was studied at the Harbor General Hospital in Torrance, California,[12] where investigators found that the predominant microorganisms isolated from blood of patients whose decubitus ulcers were the only source of infection were *Bacteroides fragilis, Peptococcus, Peptostreptococcus, Proteus mirabilis,* and *Staphylococcus aureus.* Decubitus ulcers yielded *Proteus mirabilis,* Group D streptococci, *Escherichia coli,* staphylococci, and *Pseudomonas* species as the predominant organisms cultured. Appropriate antimicrobial therapy was thought to include gentamicin or kanamycin in combination with clindamycin or chloramphenicol. Antibiotics were administered for suspected sepsis; however, one must remember that the wound itself is relatively avascular and *systemically administered antibiotics cannot be expected to work at the wound site.*

Another basic need for adequate wound healing is proper nutrition, which is best met by supplying sufficient amounts of protein and carbohydrate. Phagocytosis, vascular proliferation, and collagen synthesis are all energy-requiring processes. Protein depletion, capable of causing a 20 percent reduction in body weight, will interfere with wound repair. Reliable indictors of protein status are decreased serum albumin, a decrease in midarm muscle circumference, decreased total iron-binding capacity, decreased total lymphocyte count, the presence of cutaneous anergy, a thinning of the triceps skin fold, and a negative nitrogen balance. In debilitated patients, the required protein intake to maintain a positive nitrogen balance may exceed four times the recommended daily allowances of protein for healthy adults. Vitamins A and C are required in proper amounts on a daily basis for wound repair. Vitamin E should not be given because of its anti-inflammatory effects mimicking steroids, which inhibit wound healing.

Other factors affect wound healing; for instance, local wound eschar formation can act as a temporary biological dressing as long as there is no bacterial growth beneath it, but eschar impedes epithelialization, and debridement is the mainstay of therapy. Hyperbaric oxygen has been advocated in the past on the basis that it accelerates epidermal regeneration.[5]

Complications

Pressure ulcers can produce life-threatening complications such as sepsis, osteomyelitis, pyarthrosis, and fistulae that can extend into the bowel or bladder. Heterotopic calcification and systemic amyloidosis may also result from decubitus ulcers.[7]

Treatment

Over the centuries, literally hundreds, if not thousands, of treatments have been proposed for the care of decubitus ulcers. In the fifteenth century, one such remedy called for March barley and half a bushel of toads to be boiled together. This was then fed to a hen with newly hatched chicks. The hen was then fed to the patient. In the days of Hippocrates, patients were treated first with a warm water wash followed by a sponging with vinegar, excision, and covering with a poultice of verdigris (copper acetate), flower copper (copper oxide), molybdaina (lead oxide), alum, myrrh, frankincense, gall nuts, vine flowers, and wool grease. More recently, aloe vera, gold leaf, insulin, sugar, vitamins, hyperbaric oxygen, and iodine have been used as topical agents, and many of these remedies are still used in spite of the lack of objective data to support their efficacy.

Currently, treatment protocols call for relieving pressure over bony prominences; debridement, following the basics of wound care; and prevention of recurrences. This represents a general outline of pressure sore management with specific suggestions varying from institution to institution. A well-detailed protocol that emphasizes a systematic, standardized approach has recently been published.[14] Surgery beyond debridement may be required in the form of skin grafts, removal of bursae and bony prominences, myocutaneous flaps, and wound closure for grades IV and V ulcers or for multiple ulcers and their complications.[5,11,15]

Treatment Categories

Physical

These measures are designed to relieve pressure and remove necrotic tissue from previously debrided ulcers. Such measures include turning bedfast patients every 2 hours and teaching wheelchair-bound patients to do wheelchair pushups every 10 to 15 minutes.[10] Whirlpool baths and wet-to-dry dressings (discussed below) are also

J Am Board Fam Pract: first published as 10.3122/jabfm.2.1.43 on 1 January 1989. Downloaded from http://www.jabfm.org/ on 7 November 2026 by guest. Protected by copyright.

included in this category and are useful for removing necrotic tissue.

Mechanical

Mechanical means of assisting in pressure relief include gel flotation pads, sheepskin pads, and air-fluidized beds.[15] Gel flotation pads have been used over bony prominences to protect against the loss of intervening tissues. Sheepskins have been used as padding for bedfast and wheelchair-bound patients, but these are hot and uncomfortable and they must be changed frequently when the patient is incontinent to avoid maceration predisposing to decubiti.

There are various types of beds and mattresses that attempt to equalize the distribution of pressure over the entire surface area of body contact. It has been calculated that if the weight of a 68 kg man could be distributed evenly, the skin pressure would be less than 18 mmHg.[5] The air-fluidized bed comes closest to this calculated figure. These beds consist of a large rectangular box filled with soda lime glass beads suspended by warm air blown into the box under pressure.[16] When fluidized, the pressure against the skin surface is 15–30 mmHg. With this device, tissue maceration in the incontinent patient is avoided because the patient's excretions are quickly filtered away from the body. In addition, body excretions cause clumping of the glass beads, which then fall to the bottom of the box and are captured by an easily removable and cleanable filtering mechanism. The disadvantages of this bed are the cost and the difficulty involved in performing procedures on the patient while the bed is fluidized.[16]

Topical

A variety of topical treatments have been recommended, many of which are controversial and based on anecdotal information. Among these are aloe vera, which proponents claim accelerates healing, enhances granulation, works as an antiseptic, provides pain relief, and regenerates nerve fiber; gold leaf, which is believed to have an electrostatic or electrochemical influence on reepithelialization or may produce healing by acting as an inert occlusive membrane; insulin, which is proposed to enhance amino acid transport and to allow for better protein synthesis and the eventual deposition of collagen; various forms of carbohydrates, including sugar and glycerine, brown sugar and mineral oil, honey, and topical hyperalimentation fluid. These are proposed to work by acting as a hydrostatic agent forming a hypertonic solution that increases the flow of serum and nutrients to the wound site and serving as a host for bacteria in the wound tissue.[13] Antacids, which cause drying of the ulcers have also been used,[17] but this may actually slow healing, because reepithelialization occurs fastest in a moist environment once the eschar is removed and bacterial growth is minimized.

The least complicated topical agent used is saline wet-to-dry dressings. However, even this has been found to be detrimental when continued after necrotic tissue is removed and reepithelialization begins. Unfortunately, during the dry phase of dressing changes, new epithelial cells are stripped from the wound slowing spontaneous closure.[5] For this reason, moisture-retaining occlusive dressings have been used once the ulcer is free of bacterial infection. A moist environment not only allows enhanced epithelialization but may enhance the body's natural defenses against infection by improving leukocytic activity.[6]

The enzymatic spray, Granulex™, has been available since the early 1970s. Each 0.82 mL contains 0.1 mg trypsin, 72.5 mg balsam peru, and 650 mg castor oil. Trypsin is used as a physiological debrider of eschar and necrotic tissue. Balsam peru seems to stimulate blood flow and may act as a bactericidal agent. Castor oil improves epithelialization by reducing epithelial dessication and cornification. Yucel and Basmajian found that the rate of healing with enzymatic spray treatment was 2.5 times faster in treated patients than the control patients.[18]

Because systemic antibodies are often ineffective because of poor circulation around an ulcer, topical antibiotics have been advocated. Among these are silver sulfadiazine and povidone-iodine. Kucan and colleagues found that within 3 weeks' time, silver sulfadiazine reduced microbial counts in decubitus ulcers below 10^5 per gram of tissue in 100 percent of the patients treated.[19] These investigators also attempted to compare silver sulfadiazine with povidone-iodine and saline. Unfortunately, results are difficult to evaluate because the frequency of dressing changes varied for each of the three treatments studied. As mentioned previously, frequent dressing changes strip new epithelial cells from the wound during the dry phase of wet-to-dry dressings, and this may affect the overall healing process.

J Am Board Fam Pract: first published as 10.3122/jabfm.2.1.43 on 1 January 1989. Downloaded from http://www.jabfm.org/ on 7 November 2026 by guest. Protected by copyright.

Povidone-iodine is usually applied as a 10 percent polyvinylpyrrolidone solution that contains 90 percent water, 8.5 percent polyvinylpyrrolidone, 1 percent available iodine, and iodide. This mixture (Betadine™) is effective against a wide spectrum of microorganisms including the gram-negative bacteria *Proteus mirabilis, Escherischia coli,* and *Pseudomonas;* the gram-positive bacteria *Staphylococcus aureus* and streptococci; fungi; certain viruses; protozoa; and *Mycobacterium tuberculosis.*[20]

Serious systemic iodide toxicity consisting of hepatotoxicity and nephropathy has been found in humans and in experimental animals. Instances of substantial iodine absorption through wounds treated with povidone-iodine have been reported.[21,22] Hyperchloremic acidosis, cardiovascular instability, and oliguric renal failure have occurred concurrently with evidence of systemic iodide accumulation.[21,22] Bradycardia, hypotension, metabolic acidosis, and central nervous system dysfunction have been observed in patients with increased serum iodide concentrations.[21]

Nutrition/Pharmacology

Protein, carbohydrate, and vitamin requirements have been discussed above. Anabolic hormones such as norethandrolone have been used because of the possible side effects of nitrogen retention and weight gain.[12]

Surgery

Debridement has been discussed under basic wound care. Other surgical interventions may be required in extreme decubitus ulcers. Among these are skin grafts, bursa removal (especially of the ischial tuberosities and trochanteric bursae), myocutaneous flaps, and wound closure.[5]

Role of the Nursing Profession

A review of this topic would be incomplete without mention of the role of nurses and nurse specialists in the evaluation and management of decubitus ulcers. Recent studies have shown the effectiveness of enterostomal therapy nurses on decreasing the frequency of hospital-acquired decubitus ulcers.[23] Most of the day-to-day care in the prevention and treatment of decubitus ulcers is carried out by nurses. The importance of a collaborative, cooperative approach by physicians and nurses in dealing with this problem cannot be overemphasized. The nursing literature is an excellent source for many of the recent original research articles dealing with decubitus ulcers. In addition, an excellent in-depth review has appeared in a recent issue of *The Nursing Clinics of North America.*[24]

Conclusion

Treatment of the patient with a decubitus ulcer consists basically of relief of pressure over bony prominences, debridement of the wound as necessary, attention to systemic problems that may slow healing, local wound care, topical antibiotics as needed, and surgery as indicated. Relief of pressure may be accompanied by frequent turning of the bedfast patients and wheelchair push-ups in patients who are confined to wheelchairs, as well as by a variety of beds and mattresses of which the air-fluidized (Clinitron™) bed appears to be the best. Debridement of eschar and removal of necrotic tissue by whirlpool and enzymatic spray (such as Granulex™) are the mainstays of pressure ulcer care. Correction of systemic illness such as sepsis, diabetes, severe anemia, and malnutrition is necessary for good wound healing. Local wound care consists of the use of wet-to-dry dressings until necrotic debris is removed and re-epithelialization begins, at which time an occlusive dressing should be used as long as the wound site is infection free. It is toward this end that topical antibiotics such as silver sulfadiazine or povidone-iodine are used. Surgery is indicated when there is severe tissue loss or multiple, large wounds need to be closed. Surgical intervention may also be necessary to reduce bony prominences or to remove excessive bursae that may themselves increase local pressure or may act as structures through which ulcers may extend into joints to destroy them.

References

1. Kenedi RM, Cowden JM, Scales JT, eds. Bedsore biomechanics. Baltimore: University Park Press, 1976.
2. Schell VC, Wolcott LE. Etiology, prevention and management of decubitus ulcers. Mo Med 1966; 63:109-12.
3. Kynes PM. A new perspective on pressure sore prevention. J Enterostomal Ther 1986; 13:42-3.
4. Shea JD. Pressure sores: classification and management. Clin Orthop 1975; 112:89-100.
5. Constantian MV, ed. Pressure ulcers: principles

J Am Board Fam Pract: first published as 10.3122/jabfm.2.1.43 on 1 January 1989. Downloaded from http://www.jabfm.org/ on 7 November 2026 by guest. Protected by copyright.

and techniques of management. Boston: Little, Brown & Company, 1980.
6. Coodley E, Lincer F, Parham A, Uesu C. Management of decubitus ulcers. Compr Ther 1983; 9: 61-6.
7. DeLisa JA, Mikulie MA. Pressure ulcers. What to do if preventive management fails. Postgrad Med 1985; 77:209-20.
8. Pajk M, Craven GA, Cameron-Barry J, Shipps T, Bennum NW. Investigating the problem of pressure sores. J Geron Nur 1986; 12:11-6.
9. Allman RM, Laprade CA, Noel LB, et al. Pressure sores among hospitalized patients. Ann Intern Med 1986; 105:337-42.
10. Reddy MP. Decubitus ulcers: principles of prevention and management. Geriatrics 1983; 38:55-61.
11. Ham RJ. Primary care geriatrics: a case based learning program. Boston: John Wright PSG, Inc., 1983.
12. Galpin JE, Chow AW, Bayer AS, Guze LB. Sepsis associated with decubitus ulcers. Am J Med 1976; 61:346-350.
13. Barnes SH. Myths and misconceptions of pressure sore management. J Enterostomal Ther 1985; 12:29-31.
14. Melcher RE, Longe RL, Gelbart AO. Pressure sores in the elderly. A systematic approach to management. Postgrad Med 1988; 83:299-308.
15. Kerr JC, Stinson SM, Shannon ML. Pressure sores: distinguishing fact from fiction. The Canadian Nurse 1981; 77:23-8.
16. Lucke K, Jarlsberg C. How is the air-fluidized bed best used? Am J Nurs 1985; 85:1338-40.
17. Becker L, Goodemote C. Treating pressure sores with or without antacid. Am J Nurs 1984; 84: 351-2.
18. Yucel VE, Basmajian JV. Decubitus ulcers: healing effect of an enzymatic spray. Arch Phys Med Rehabil 1974; 55:517-9.
19. Kucan JO, Robson MC, Heggers JP, Ko F. Comparison of silver sulfadiazine, povidone-iodine and physiologic saline in the treatment of chronic pressure ulcers. J Am Geriatr Soc 1981; 29: 232-5.
20. Zamora JL. Chemical and microbiologic characteristics and toxicity of povidone-iodine solutions. Am J Surg 1986; 151:400-6.
21. Aronoff GR, Friedman SJ, Doedens DJ, Lavelle KJ. Case report: increased serum iodide concentration from absorption iodine through wounds treated topically with povidone-iodine. Am J Med Sci 1980; 279:173-6.
22. Dela Cruz F, Brown DH, Leikin JB, Franklin C, Hryhorczuk DO. Iodine absorption after topical administration. West J Med 1987; 146:43-5.
23. Kynes PM, Neese DT. The effects of ET nursing assessment on the incidence of hospital-acquired pressure ulcers. J Enterostomal Ther 1987; 14: 148-51.
24. Adams Mondoux LC, ed. Pressure ulcers. In: The Nursing Clinics of North America 1987; 22:357-492. Philadelphia: W.B. Saunders Co.

MOVING?

Please notify us 6 weeks in advance if possible. Use the Address Change Form printed on the reverse side of your subscription label carrier.

J Am Board Fam Pract: first published as 10.3122/jabfm.2.1.43 on 1 January 1989. Downloaded from http://www.jabfm.org/ on 7 November 2026 by guest. Protected by copyright.