Pressure Sores

Classification and Management

J. DARRELL SHEA, M.D.*

Of all the problems facing the physician responsible for the care of severely injured or disabled patients, one of the most frustrating is the prevention and management of skin breakdown known as pressure sores. In the past they have been called decubitus ulcers, bed sores, and ischemic ulcers. The problem is no less disconcerting today than it was four hundred years ago when Fabricus[10] suggested that a "pneuma" resulting from nerve severance, plus loss of blood supply, caused skin ulceration. Charcot,[7] in 1879, also believed that nerve injury released a neurotrophic factor that led to tissue necrosis. Nerve injury was also implicated by Munro,[16] who felt that the autonomic reflex arc controlling skin circulation was interrupted. In guinea pigs with sectioned spinal cords, Brown-Sequard[4] treated ulcers by keeping the area clean and free of pressure, thus associating pressure and moisture as contributing factors. No specific neurotrophic factor has been satisfactorily demonstrated. However, several other conditions have been suggested as being primary causes including a deficient blood supply, absent sensation and reflexes with incontinence, muscle wasting and atony, bacterial infection, anemia, and malnutrition.

Recommendations for treatment have been no less confusing than theories of etiology. Almost any concoction imaginable has been placed in these ulcers with the intention of promoting wound healing. A partial list includes poultices of carrots and turnips, bread and charcoal, Dakin's solution, antibiotics, enzymes, vitamins, cod liver oil, dried blood plasma, gold, aluminum and silver, chlorophyll, sugar and brine baths.[1-3, 11-13, 17, 19] Several physical and mechanical modalities have also been devised for preventing and treating skin breakdown, including electric lamps, ultraviolet light, hyperbaric oxygen, rubber rings, and donuts, water beds, sawdust beds, and a variety of pressure beds and paddings.[2, 3, 11, 12, 19] Since 1945, the literature has defined with increasing boldness the indications, techniques, and principles of surgical closure of pressure sores.[2, 6, 8, 13, 18] A critical review of this vast literature is difficult because a clear definition of the lesion or lesions being treated is lacking. Proper evaluation of a therapeutic technique necessitates a clear understanding of the disease process under treatment. As with all disease processes, pressure sores do not appear *de novo* but develop in an orderly pathophysiologic manner which is influenced by a variety of local and systemic factors. The mechanism of etiology as outlined in this report, considers these various elements and suggests an orderly evolution and classification which serves as a guide for both treatment and prognosis.

MECHANISM (FIG. 1)

Bony prominences of the body are covered by multiple layers of soft tissue which vary in thickness and type depending on anatomic location and nutrition. Figure 1

Received February 21, 1975.

* 115 West Columbia, Orlando, Florida 32806.

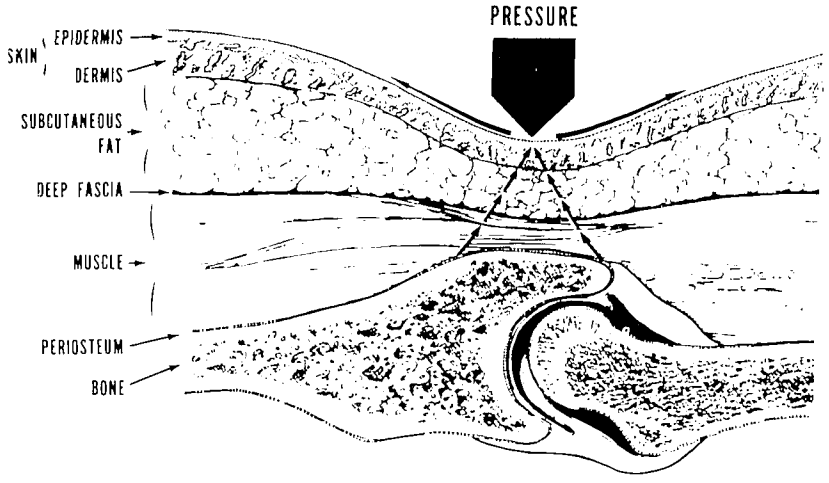

FIG. 1. Mechanism. Pressure over a bony prominence compresses all intervening soft tissue with a resulting wide three dimensional pressure gradient causing varying degrees of ischemia and damage.

diagrammatically portrays these layers, each of which has distinguishing histologic, physiologic, and physical characteristics. The most superficial layer, skin, is a dense tissue differentiated into two layers, an avascular epidermis with numerous rete pegs protruding into the deeper fibro-elastic dermis containing several organs and appendages, including blood vessels, hair follicles, sweat glands, and various sensory nerve endings. Skin serves many functions, including protection, body temperature regulation, water and fat excretion, as well as being a major sense organ. The loose connective tissue deep to the skin is composed primarily of fat cells with little intercellular substance and vascularity. It is compressible, providing a measure of shock absorption and padding, as well as permitting movement of the skin on the deeper structures. However, this subcutaneous fat, or panniculus adiposus, lacks significant tensile strength and is thus vulnerable to mechanical forces, such as sheer or lateral pressure which may compromise its blood supply by angular stretching of the vessels. The subcutaneous fat is based on a dense, firm, relatively avascular deep fascia which envelops the body, giving contour and shape. The almost pure collagen content of this layer is relatively resistant to mechanical forces and less susceptible to vascular compromise. Deep to the fascia lie muscles with individual myofascial envelopes and distinct fascial planes. Muscle, profusely endowed with nerves and blood vessels, tolerates compression poorly but is actively resistant to tension. The amount of muscle overlying any bony prominence is variable, usually limited to tapering insertions, and origins. Periosteum, the deepest soft tissue layer overlying bone, blends with the capsular structures of adjacent joints.

Pressure on any bony prominence from sitting or lying is transmitted from the surface to the underlying dense bone, compressing all intervening tissue to varying degrees. The elasticity of these tissues results in the force being distributed in a hammock or sling effect with the greatest pressure over the bone diminishing progressively to the periphery. A pressure gradient also exists from the surface to the bone as the force is dissipated in a cone-shaped manner by each succeeding layer. Sitting pressures on skin overlying the ischial tuberosity in a normal individual have been experimentally measured and have been found to exceed 300 mm Hg.[5] The mean blood pressure of normal skin capillaries has been reported to vary between 12 to 70 mm Hg.[14, 15] Therefore, it

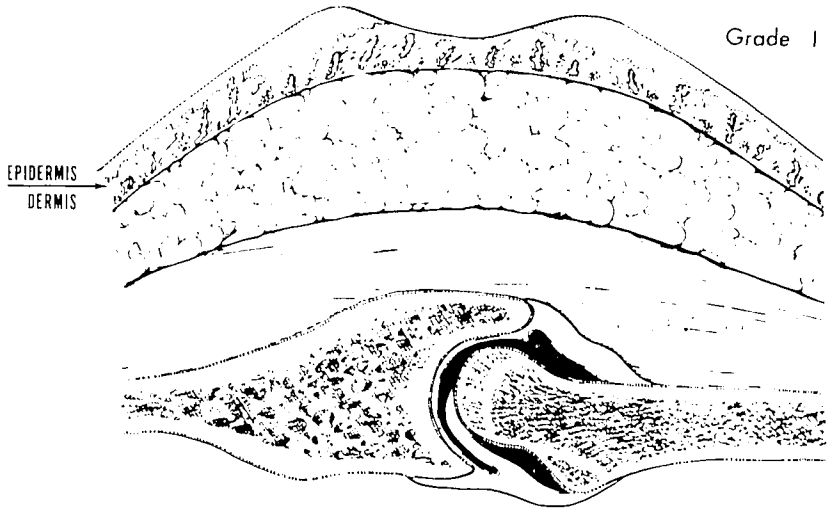

FIG. 2. Grade I Pressure Sore. An acute inflammatory reaction involving all soft tissue layers with a moist irregular partial thickness ulceration limited to the epidermis exposing the underlying dermis.

is readily evident that the weight-bearing pressure upon any bony prominence is sufficient enough to obstruct and compromise the blood flow to a large area of soft tissue. The ingress and egress of blood is impeded, causing a local ischemia in all involved soft tissue. However, the individual tissue cells continue to metabolize normally, producing toxic metabolic by-products which accumulate locally, increasing the rate of cell death normally occurring in any cell population. The surviving and aging cells are severely compromised and thus are more readily susceptible to damage by mechanical forces, including pressure. Obviously, the magnitude of cellular death depends on the normal metabolic activity and vascularity of each tissue type. Compounding the problem is the local mechanical irritation of the skin from abrasion by shifting on coarse surfaces and the macerating effect of urine, stool, and body discharges with their attendant bacterial population.

Relief of pressure allows perfusion of all tissues with removal of toxic materials and the restoration of nutrition to compromised cells. In the normally innervated individual, pain secondary to nerve ending stimulation by anoxia and local chemical irritation encourages early position change with minimal cell damage. The paralyzed, anesthetic, or unconscious patient, unable to recognize ischemia, fails to move resulting in injury to all tissues. Relief of prolonged pressure results in leakage of cells from ruptured and damaged vessels, accumulation of edema fluid, augmentation in size and number of local small vessels and the migration of acute inflammatory cells, all clinically manifest as an acute inflammatory reaction.

GRADE I PRESSURE SORE

(FIGS. 2 AND 3)

An acute inflammatory response involving all soft tissue layers is characteristic of the earliest stage of injury of Grade I pressure sore. The dilatation of blood vessels and accumulating edema fluid secondary to the ischemia causes a thickening and distortion of all tissue layers between the surface and the underlying bone. However, the most apparent superficial clinical presentation of a Grade I pressure sore is an irregular, ill-defined area of soft tissue swelling and induration with associated heat and erythema overlying a bony prominence. A normally innervated patient will complain of pain in the area. The extreme of Grade I involvement is a moist superficial irregular ulceration limited to the epidermis ex-

FIG. 3. Grade I Pressure Sore. Photograph of sacral area demonstrating Grade I sores which present clinically as an indurated area of swelling, heat, and erythema with a superficial breakdown limited to the epidermis.

posing the underlying dermis and resembling an abrasion.

Recognition of the Grade I sore is essential because it is a reversible lesion treated by relief of pressure, local cleansing of the skin to control bacterial contamination, and general supportive measures, including diet, correction of anemia and dehydration. A rapid resolution of the reactive process will occur within 5 to 10 days. These same measures apply to the prevention of pressure sores. Contrary to general opinion, relief of pressure to bony prominences is not complicated. There is no normal physiologic situation where an individual maintains an absolutely immobile ischemic position in excess of two hours. Why, therefore, must the disabled be permitted to lie unmoved for extended periods? Position can be changed every two to three hours with minimal physical and professional effort if one does not attempt to turn the patient a full 180 degrees each time but merely rolls him 20 to 30 degrees to a slightly tilted position supported by pillows. Primary reliance on mechanical devices for control of pressure

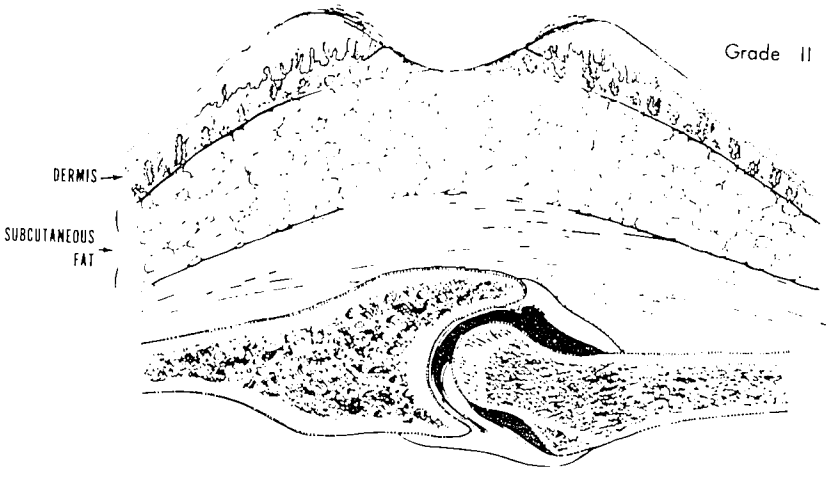

FIG. 4. Grade II Pressure Sore. Involves all soft tissue also, presenting with a full thickness skin ulcer extending to the underlying subcutaneous fat. Note increased reaction in the subcutaneous fat and muscle.

FIG. 5. Grade II Pressure Sore. Histologic Section. This section demonstrates a shallow full thickness skin ulcer with thickening of the epidermis and rete pegs overlying an area of chronic inflammation and fibrosis of the panniculus adiposus distorting normal histologic patterns. Interstitial edema, dilated and thrombosed blood vessels are evident.

is cautioned against because these tend to create a false sense of security which may lead to neglect."

GRADE II PRESSURE SORE

(FIGS. 4 TO 6)

Continuation of the intense local pressure or frequent, repeated prolonged insults will intensify and extend the acute inflammatory response leading to a fibroblastic response in all layers, tending to obscure the normally well defined tissue layers. On histologic cross section a mixed acute and chronic inflammatory process, involving the deeper as well as the superficial layers, is seen. Bacterial growth, maceration by perspiration, urine, and mechanical abrasion cause a superficial ulceration of Grade I to extend wider and deeper penetrating through the full thickness of the dermis to the junction with the subcutaneous fat. The Grade II pressure sore presents clinically as a shallow full thickness skin ulcer whose edges are more distinct with early fibrosis and pigmentation changes blending into a broad indistinct area of heat, erythema, and induration. Although clinically a deeper and broader ulceration with a much greater inflammatory and fibrotic involvement, the Grade II pressure sore is also a reversible lesion. Treatment, as with the Grade I pressure sore, is directed toward preventing further insult and breakdown by relieving pressure and permitting the normally well vascularized skin to heal through local wound cleansing.

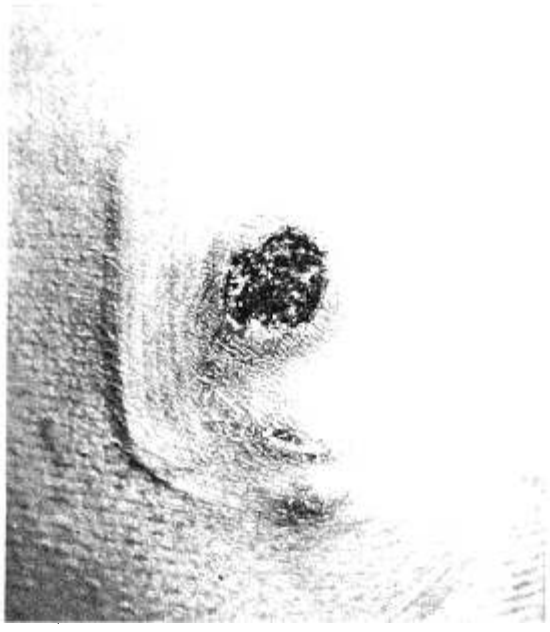

FIG. 6. Grade II Pressure Sore. Photograph of sacral area demonstrating a Grade II pressure sore with a relatively distinct shallow ulcer exposing subcutaneous fat in its base and surrounded by a broad indistinct area of heat, erythema, and induration.

GRADE III PRESSURE SORE

(FIGS. 7 TO 9)

Failure to appreciate the early stages of soft tissue breakdown and to institute proper wound care as outlined will permit progression of a reversible Grade II sore through the dermis into the subcutaneous fat where extensive and rapid undermining occurs. Previous wound contamination matures rapidly to frank infection compounding the problem of fat necrosis from small vessel thrombosis, ischemia, and chronic inflammation. Reaction in the skin is intensified as the epidermis thickens and rolls over the edge toward the ulcer base, creating a distinct ulcer margin with varying degrees of pigmentation. An intense reactive fibrosis, inflammation and retraction in both dermis and the subcutaneous fat distorts any previous tissue distinctions. The deep fascia, being relatively avascular and physically resistant, limits the depth of penetration of the ischemic necrotic process encouraging peripheral spread and undermining. Muscle, although not directly involved in the ulcer, is distorted by swelling and inflammation with some loss of fibrillar detail. Spasticity secondary to inflammation and infection causes muscle contractures and joint deformity compromising patient care. Intermuscular fascial planes separated by edema fluid become more apparent. Bone response to the intense local inflammation includes subperiosteal new bone and local osteoporosis. Contiguous joints develop capsular swelling and synovial effusion further contributing to deformity.

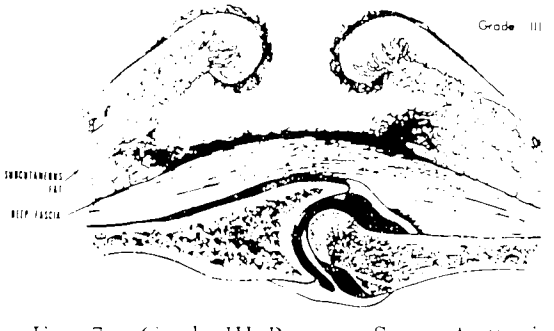

FIG. 7. Grade III Pressure Sore. A "typical decubitus" with a necrotic, foul smelling infected ulcer limited by the deep fascia but extensively involving the fat with undermining of the skin. Note muscle, periosteum, and joint involvement.

Clinically, the Grade III pressure sore is the classical "decubitus ulcer" with an irregular full thickness skin defect extending into the subcutaneous fat exposing a draining, foul smelling, infected, necrotic base which has undermined the skin for a variable distance. The skin edge is rolled with an altered dark and light pigmentation which sharply outlines the ulcer. The adjacent joints are flexed with a limited range of motion complicating nursing care and positioning. Invariably more than one grade of sore is present. Fever, dehydration, anemia, and leukocytosis are compounded by profound loss of fluid and protein from these

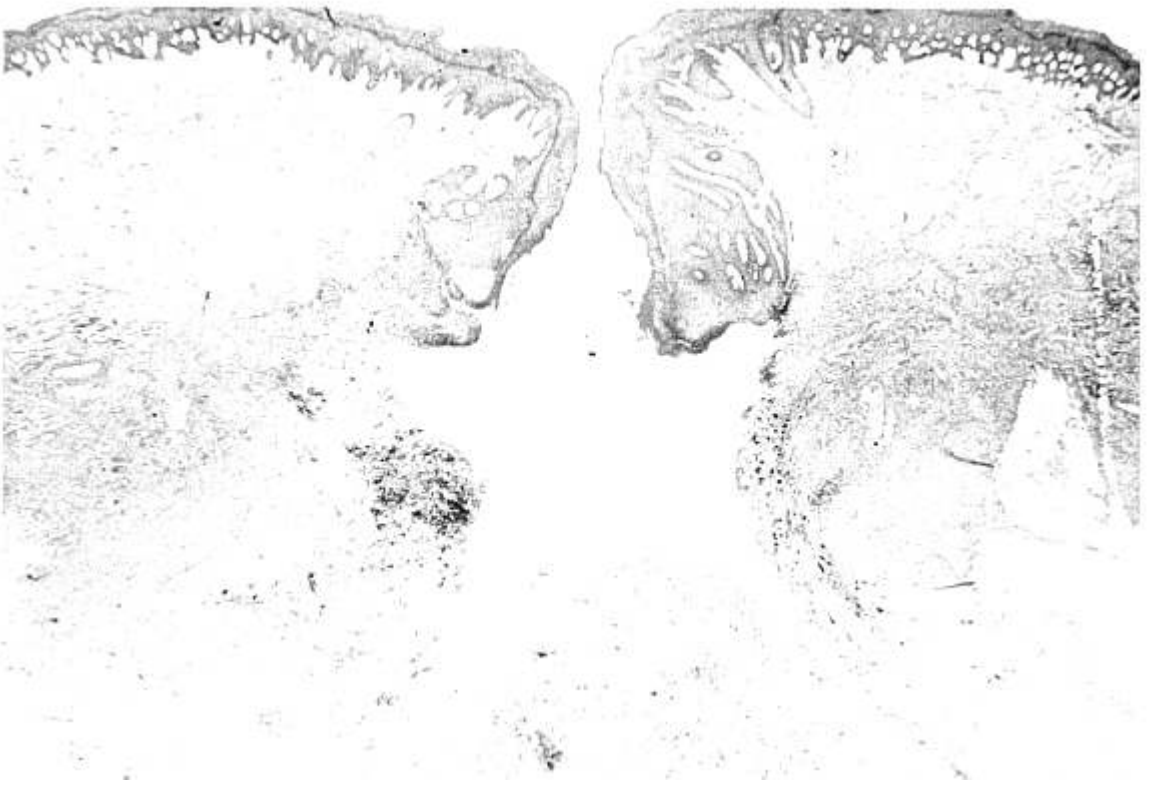

FIG. 8. Grade III Pressure Sore. Histologic Section. The ulcer margins are thickened and rolled with extensive epidermal reaction. Chronic inflammation, reactive fibrosis and tissue necrosis in the base have extended peripherally, obliterating the subcutaneous fat.

open draining wounds, further compromising patient recovery.

Treatment. In contrast to the localized Grades I and II, the Grade III sore is a life-threatening lesion, affecting the entire body and requiring a broader comprehensive therapeutic approach. Local wound healing is seriously compromised by anemia, dehydration, protein depletion, and infection. Blood transfusion treats anemia and helps to correct protein depletion. Intravenous fluids containing appropriate antibiotics and vitamins treat dehydration, electrolyte imbalance, and infection. A balanced, high protein diet is essential. Local wound care is based on well established surgical principles of incision and drainage of abscess cavities, adequate debridement of necrotic tissue and closure of all open clean wounds. The Grade III pressure sore differs very little from wounds in general, but for some unknown reason an aura of mystique has arisen about the care of this very obvious ulcer. No other wound in the body has been subjected to such a variety of unusual treatments ranging from vegetable poultices to sugar and charcoal or baked in hot lights, soaked in brine, or pressed upon by an array of mechanical contrivances.

Initial care involves recognition of the lesion and collection of bacterial cultures. Obvious dead necrotic tissue in the ulcer base may be easily, sharply debrided at the bedside with a disposable suture removal set or scalpel. Larger loculations or abscess cavities must be drained. Regular debridement of the ulcer bed is accomplished by *wet to dry dressings* consisting of sterile

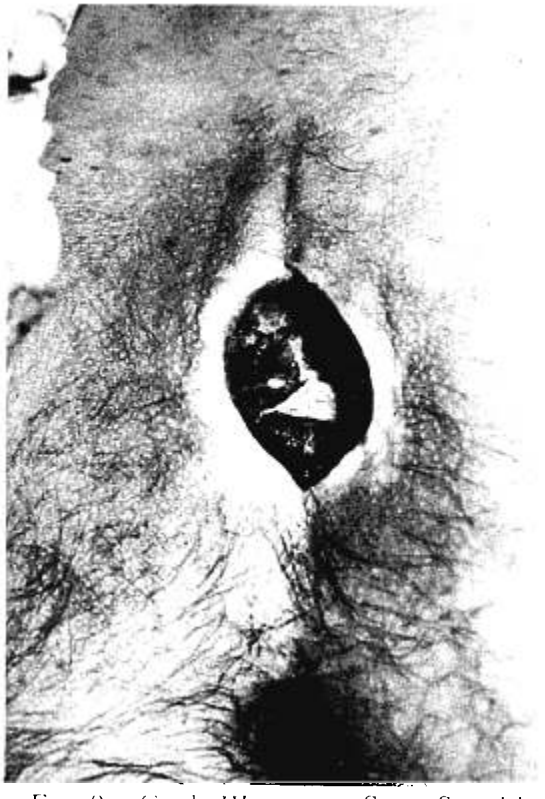

FIG. 9. Grade III pressure Sore. Sacral lesion with distinct edges of altered pigmentation exposing a necrotic base of deep fascia with extensive undermining of the skin.

coarse mesh gauze, preferably in one long continuous piece soaked in sterile saline and packed into the ulcer being careful to penetrate into all recesses and undermined areas.

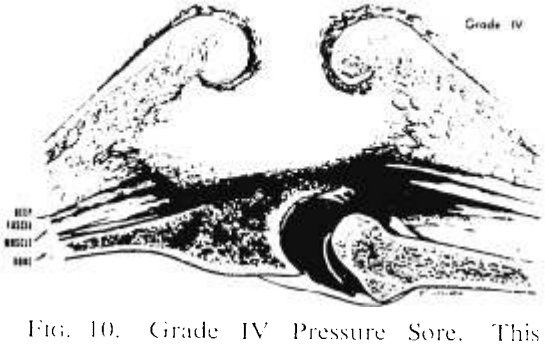

FIG. 10. Grade IV Pressure Sore. This sore has penetrated the deep fascia causing extensive soft tissue spread with osteomyelitis and septic, dislocated joints. These are frequently fatal.

No cover dressing is applied, permitting the saline gauze to dry from exposure. A bed cradle is used to keep the blankets off the wound. Three hours later when the drying coarse mesh gauze has become partially adherent to the necrotic ulcer base, it is withdrawn, removing a portion of the dead tissue. This simple, inexpensive technique is repeated every 3 to 4 hours around the clock until the ulcer is completely debrided, presenting a granulating base usually within 10 to 14 days. By this time, the patient is systematically stabilized and ready for surgical closure.

Although many Grade III sores will heal by secondary intention, the resulting scars are thin, avascular, friable, and adherent, and thus subject to easy breakdown, providing minimal protection for the bony prominence. Prolonged hospitalization with intense nursing care is necessary, which is costly, physiologically, psychologically, and fiscally.

Surgical closure, the principles and techniques have been well documented, offers several advantages, including removal of all involved tissue by generous resection, modification and removal of the offending bony prominences, and resurfacing with multiple layers of well vascularized normal soft tissue, including muscle, subcutaneous fat, and skin, which are approximated with minimal scarring. Hospitalization and rehabilitation are considerably shortened, and the incidence of recurrence in trained patients is reduced.

GRADE IV PRESSURE SORE (FIGS. 10 AND 11)

Penetration of the deep fascia by the infectious necrotic process eliminates the last barrier to extensive spread. Undermining now progresses rapidly, developing osteomyelitis. Septic arthritis with subluxation and dislocation occurs in contiguous joints causing further deformity. Body cavities may be penetrated in extreme situations or

bony pelvic support seriously weakened by bone dissolution permitting herniation of abdominal viscera. Ulcers communicate via fascial plane extension to adjacent and distant Grade III or Grade IV sores which are invariably present.

Clinical presentation of a Grade IV sore resembles that of a Grade III except that bone can be identified in the base of the ulceration which is more extensively undermined with profuse drainage and necrosis. In some Grade IV sores about the pelvis, the floor of the ulcer may bulge or extrude in association with increasing intra-abdominal pressure. Adjacent joints become dislocated and deformed. X-rays indicate extensive bone involvement with osteomyelitis and loss of bone substance.

Grade IV pressure sores with profuse multiple ulcer drainage, extensive soft tissue necrosis and osteomyelitis, septic joint dislocation, dehydration, and anemia, create an extremely toxic situation which frequently proves fatal. Heroic and aggressive life-saving measures are necessary, usually in the form of radical surgical procedures. As with the Grade III sores, general support measures are essential: blood transfusion, intravenous fluids with antibiotics, and correction of electrolyte abnormalities. Local wound culture identifies offending bacterial organisms and their sensitivities. Surgical incision and drainage with generous resection of all affected necrotic tissue is essential, including partial or total joint resections where necessary. Fascial planes must be drained and all wounds left open to permit frequent dressings and wound inspection. Secondary closure with design of appropriate flaps is done only when all infection is controlled, all necrotic tissue removed, and the patient generally stabilized. Extensive and multiple ulceration, infection and necrosis, necessitates radical resection of ulcers occasionally to the extent of amputation to provide total thigh or limb flaps for adequate soft tissue coverage. In extreme

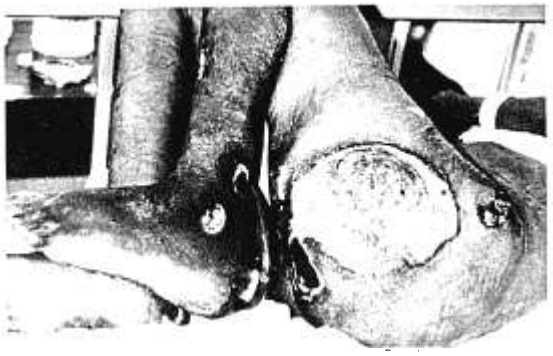

FIG. 11. Grade IV Pressure Sore. This lesion extends deep to the fascia exposing infected greater trochanter and hip joint. Coexisting Grade III (ischium, tibia, iliac crest) and Grade IV sores (hip, lateral malleolus) may communicate subcutaneously with adjacent sores and body cavities. Characteristic fixed flexion deformities associated with advanced ulceration are illustrated.

cases of pelvic involvement, hemicorporectomy or translumbar amputation may be the only alternative to death. A useful alternative in the severely involved patient with multiple ulcerations about the pelvis, deformities, and septic hip joints is unilateral or bilateral resection of the proximal femur and hip in two stages (proximal femorectomy).[20] This permits adequate wide resection of all involved tissue, corrects the

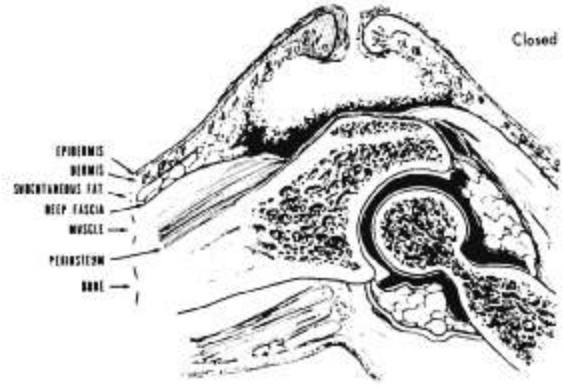

FIG. 12. Closed Pressure Sore. A large bursa-like cavity lined by chronic reactive fibrosis extending to the deep fascia or bone drains through a small sinus. Adjacent and muscle reaction is minimal.

FIG. 13. Closed Pressure Sore. Histologic section from patient in Figure 12. Narrow, deep ulcer lined by thickened epidermis opens into a large cavity lined by thick fibrotic reactive tissue. Areas of adipose tissue are noted at edges of the section.

hip flexion deformity, and retains the lower limbs. Coincident fixed knee flexion contractures and/or ulceration can also be managed by resection of the distal femur at the knee permitting knee extension, thus facilitating nursing care, turning in bed, transfers, and maintenance of a body image. These resections usually heal with significant degrees of fibrosis, providing reasonable stability to the limbs for sitting or lying.

CLOSED PRESSURE SORES
(FIGS. 12 TO 14)

A special situation exists in the closed pressure sore which differs uniquely from the above described open lesion but yet is caused by the same pathologic processes. The term *closed pressure sore* was selected to characterize the innocent clinical presentation that conceals a deep potentially, rapidly fatal lesion. In some situations repeated prolonged pressure insults combined with sheer stress causes an ischemic necrosis in the subcutaneous fat without skin ulceration leading to the development of a bursa-like cavity filled with necrotic debris. Reactive fibrosis and thickening of the surrounding soft tissue tends to wall off the process which may extend for a considerable distance peripherally resembling a Grade III sore in extent and depth. An intense periostitis in the adjacent bone causes a prominent accumulation of new bone further intensifying the local pressure. The pigmented, thickened, and fibrotic overlying skin eventually ruptures, creating a small skin defect draining a large base which becomes contaminated by locally resident bacteria.

Clinically, the closed pressure sore is unremarkable in its presentation with a small benign-appearing ulcer measuring a few millimeters in diameter and overlying a bony prominence with minimal drainage requiring at most one small daily dressing. Although the discharge is contaminated, systemic manifestations of infection as seen in Grade III sores are not common. These closed pressure sores commonly develop about the pelvis overlying bony prominences such as the ischial tuberosity and the greater trochanter in a relatively healthy trained wheelchair bound paraplegic. Recognition of and treatment of chronic closed pressure sores by wide excision of the ulcer, development of an appropriate local rotation flap, resection of the pseudo-incapsulated bursal sac and smoothing of the bony prominence are essential. These lesions are deceivingly extensive, occasionally requiring X-rays and sinograms for accurate delineation. Not uncommonly, because of their proximity to the perineum, these chronic draining closed pressure sores become infected, rapidly converting from a relatively benign local draining cyst-like lesion to a rampaging closed

Grade IV type lesion with extensive involvement of bone joints and possibly penetrating into the pelvis but with minimal external evidence of the site or extent of involvement. In spite of a diligent clinical search for the site of an obvious severe life-threatening infection, these lesions are frequently overlooked, and the full extent of involvement is not appreciated until post-mortem examination.

Treatment is an awareness of the lesion and wide surgical excision. Closure may be by simple skin edge approximation but frequently the large resultant surgical defect must be obliterated by a local muscle flap.

DISCUSSION

Fundamentally, pressure sores differ little from other wounds, sharing normal tissue responses to injury. Although no single factor can be completely indicted as the only cause of a full thickness soft tissue breakdown, pressure is considered as a primary initiating cause. The advanced stages of Grade III and IV are the result of additional interactions of multiple complex mechanical, chemical, bacterial, and physiologic factors. Recognition of this dynamic disease spectrum permits a correlation of identifiable stages with appropriate treatment. Treatment can be generally divided into nonoperative and operative methods. Basically, all nonoperative methods share three common objectives of (1) pressure relief, (2) wound debridement and cleansing, and (3) promotion of wound healing. Mechanical measures and devices such as turning, air mattresses, and water beds are directed primarily at the first objective and are generally effective. However, mechanical devices do have certain disadvantages, including mechanical breakdown and failure, technical complexity, which may limit completely efficient use by all personnel and a tendency to create a false sense of security and therefore neglect. In some cases they contribute to joint contracture. Reported success of the various poultices and applications is readily appreciated when one realizes that everything that is put into these ulcers must be cleaned out, thereby accomplishing the second objective of cleaning and debriding the wound. Simple nontoxic, inexpensive physiologic saline and gauze are readily available for use in the easily

TABLE 1. Pressure Sores: Classification—Treatment

Grade	*Anatomic Limit*	*Treatment*
I	Dermis	Local wound care
II	Subcutaneous fat	Local wound care
III	Deep fascia	Surgery
IV	No limit—extensive	Radical surgery
Closed	Deep fascia	Surgery

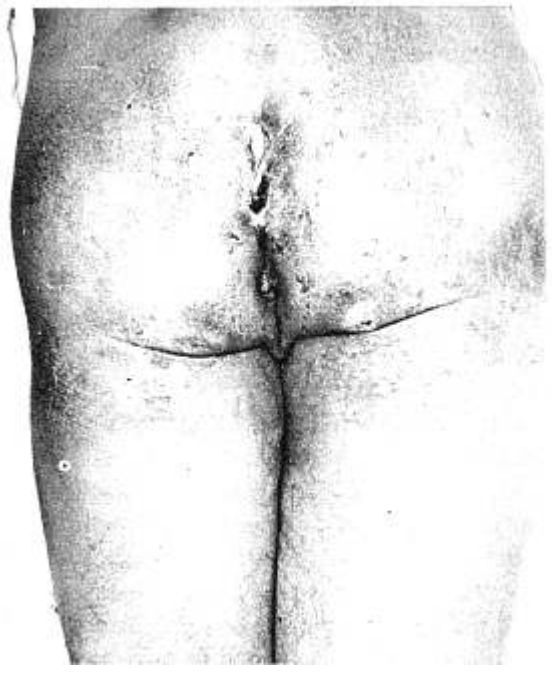

FIG. 14. Closed Pressure Sore. Right ischial area presents a two millimeter ulcer or sinus opening into the large cavity demonstrated in Figure 13. Similar pigmented area overlying left ischial tuberosity has not yet ruptured into underlying fluid filled space. Grade II and Grade III sacral pressure sores are also present.

mastered technique of wet to dry dressings as described here. Promotion of wound healing by systemic support with transfusion, rehydration, antibiotics, and electrolytes is sound basic medicine. Local physical, chemical, or pharmacologic means of improving the blood supply in the already inflamed wound are of questionable value and may in fact retard healing.

Nonoperative methods are sufficient for Grades I and II but are inadequate for more advanced lesions because of their costly unpredictable healing, resulting in thin friable scars (Table 1). Grades III, IV, and closed sores are primarily surgical lesions requiring application of a fourth treatment objective of excision and closure of the ulcer defect with adequate layers of soft tissue.

In addition to its therapeutic implications, an effective classification system provides a means of identification for both clinical and research study. Treatment programs are difficult to evaluate if an adequate description of the lesions being managed is lacking. A therapeutic classification system also simplifies communications between physicians and nurses as well as permits a clearer notation in clinical records for further reference.

SUMMARY

Four grades of pressure can be recognized on the basis of pathophysiology of soft tissue breakdown overlying bony prominences. Management is correlated with the extent of the lesion and ranges from local wound care, turning and systemic support for Grade I and II, to local and radical excision with soft tissue flap closure for the more extensive Grades III and IV.

REFERENCES

1. Astley, G. M.: Bedsores, Am. J. Surg. 50:734, 1940.
2. Bailey, B. N.: Bedsores. London, Edward Arnold Ltd., 1967.
3. Bardsley, C., Fowler, H., Moody, E., Teigen, E., and Sommer, J.: Pressure sores, a regimen for treating and preventing them. Am. J. Nurs. 64:82, 1964.
4. Brown-Sequard, C. E.: Experimental Researches Applied to Physiology and Pathology. New York, H. Baillaire, 1853.
5. Bush, C. A.: Study of pressures on skin under ischial tuberosities and thighs during sitting, Arch. Phys. Med. Rehab. 50: 207, 1969.
6. Campbell, R. M.: The surgical management of pressure sores. Surg. Clin. North Am. 39:509, 1959.
7. Charcot, J. M.: Lectures on the Diseases of the Nervous System. Delivered at La Saltpetriere, Translated by G. Sigerson, Philadelphia: Henry C. Lea, 1879.
8. Conway, H. and Griffith, B. H.: Plastic surgery for closure of decubitus ulcers in patients with paraplegia, Am. J. Surg. 91: 946, 1956.
9. Edberg, E. L., Cerny, K., and Stauffer, E. S.: Prevention and treatment of pressure sores, Phys. Ther. 53:246, 1973.
10. Fabricus, H.: De gangraena et sphacelo tractatus methodicus, Leyden, 1593.
11. Fischer, B. H.: Topical hyperbaric oxygen treatment of pressure sores and skin ulcers, Lancet 2:405, 1969.
12. Guttman, L.: The problem of treatment of pressure sores in spinal paraplegics, Br. J. Plast. Surg. 8:196, 1955.
13. Kahn, S.: A guide to the treatment of decubitus (pressure) ulcers in paraplegia, Surg. Clin. North Am. 40:1657, 1960.
14. Kosiak, M.: Etiology and pathology of ischemic ulcers, Arch. Phys. Med. Rehab. 40:62, 1959.
15. Kosiak, M.: Etiology of decubitus ulcers, Arch. Phys. Med. Rehab. 42:19, 1961.
16. Munro, D.: Care of back following spinal cord injuries, N. Engl. J. Med. 223:391, 1940.
17. Nyquist, R. H.: Brine bath treatments for decubitus ulcers, JAMA 169:927, 1959.
18. Sanchez, S., Eamegdool, S., and Conway, H.: Surgical treatment of decubitus ulcers in paraplegics, Plast. Reconstr. Surg. 43: 25, 1969.
19. Smigel, J. O. and Russell, A.: The do's and dont's of therapy for decubitus lesions with emphasis on use of the electric lamp, J. Am. Geriat. Soc. 10:973, 1962.
20. Wharton, G. and Morgan, T. H.: Personal communication, January 1973.