# **Pressure Sores**

The complex etiology of pressure sores requires a multidisciplinary approach to treatment. The surgeon cannot simply schedule the patient for surgery and expect a healed wound that will not reoccur. Nursing home caregivers and primary care physicians should regularly evaluate their elderly and wheelchair-bound patients for the first signs of tissue necrosis. Internists can manage metabolic and physiologic diseases such as providing tight glucose control in diabetics. Dietitians can determine energy requirements and suggest nutritional supplements to meet the patient's caloric goals. Social workers should investigate the patient's home for environmental hazards and help eliminate financial barriers to therapeutic intervention. The plastic surgeon examines the patient for severity of the wound and determines the best surgical course of action, if indicated. The plastic surgeon can coordinate and mobilize the many team members. Case managers schedule appointments, coordinate transportation for routine checkups, and help obtain needed equipment. If a surgical intervention is pursued, specially trained nurses can assist with postsurgical flap checks, dressing changes, and other nursing functions; they are an extension of the plastic surgeon throughout the acute setting. Finally, physical and occupational therapists evaluate patient mobility and recommend custom equipment to help with healing and prevention of future pressure sore formation. These therapists can assist with range of motion exercises to help treat and prevent flexion contractures as well as activities of daily living. With a combined multidisciplinary team, patients receive the best prognosis for long-term pressure sore treatment.

# **Terminology**

The immobilized patient is at risk for developing breakdown of his or her skin and soft tissue. This breakdown is the necrosis of skin, subcutaneous tissue, and muscle, collectively referred to as a pressure sore by the medical community. The term pressure sore has several anecdotal synonyms rooted in history, including bedsore and decubitus ulcer. The term decubitus comes from the Latin word *decumbere* meaning

"to lie down." Each of these words attempts to identify the underlying pathophysiology of wounds due to physical stress. However, these terms fail to address the existence of pressure sores not due to lying in a bed such as in wheelchair-bound patients and in nondependent locations. Currently, the term pressure sore best describes these complex wounds because they are multifactorial in nature and can occur anywhere on the body.

# **Etiology**

Pressure sores are complex wounds that result from 1 or more contributing factors. Stress, time, spasticity, infection, edema, denervation, moisture, and poor nutrition are considered fundamental issues that result in or contribute to pressure sore development. More than 60% of pressure sores develop within the hospital wards.<sup>2</sup> One probable source is the increasing geriatric population requiring hospitalization in whom one third to one half experience a functional decline.<sup>3</sup> Debilitation and development of a pressure sore type of injury poses a legal liability risk to healthcare practitioners and hospital risk managers.<sup>4,5</sup> This nosocomial condition requires vigilant preventative measures.

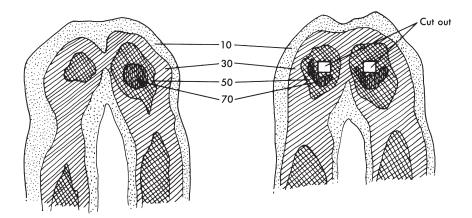
#### Stress

Position and contact with the environment is a critical component to the pressure sore injury. The gravitational pull on the human body results in contact with the bed, wheelchair, or other surface. This contact is enhanced at surfaces of the body that overly bony prominences<sup>6</sup> (Fig 1). The soft tissues become compressed between hard external objects and the underlying bone, leading to localized ischemia and eventual necrosis. This force exerted on tissues is considered the sine qua non of pressure sore formation.

The famous Robert Pershing Wadlow provides a lesson in the pathology of how position and contact with objects can lead to detrimental effects. Mr. Wadlow holds the *Guinness Book of World Records* title for the world's tallest man at 8 feet 11 inches (272 cm) and weighing approximately 200 kg. He developed a blister on his ankle that became infected after being poorly fitted for leg braces. One week later he died from sepsis at the age of 22.

#### Time

Prolonged contact between the human body and hard objects results in ischemic necrosis and pressure sore formation of the intervening tissues. Kosiak and colleagues, demonstrated the inverse parabolic relationship



**FIG 1.** Seated pressure distribution. (With permission from Phillips LG, Robson MC. Pressure ulcerations. In: Jurkiewicz MJ, Krizek TJ, Mathes SJ, Ariyan S, editors. Plastic Surgery: Principles and Practice. Vol. 2. St Louis: CV Mosby; 1990; pp 1223-51.)

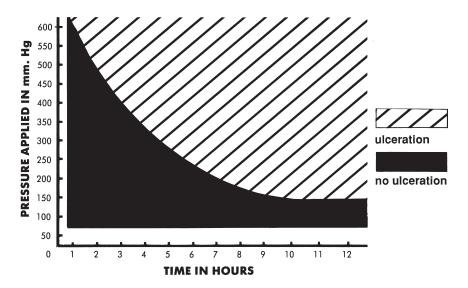


FIG 2. Time versus pressure curve for tissue breakdown in a canine model.

between time and pressure in pressure sore formation using a canine model<sup>8</sup> (Fig 2). A high level of pressure induces tissue ulceration within a short time interval, and conversely, low levels of pressure require a prolonged timeframe to cause tissue damage. The 1 caveat in this study is the use of dogs, a loose-skinned animal with less subcutaneous tissue

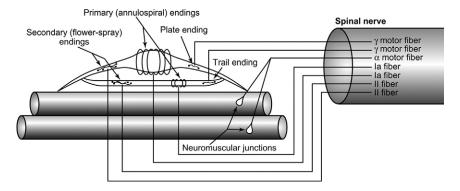


FIG 3. Afferent and efferent innervation of the muscle spindle and extrafusal skeletal motor fibers.

than in humans. A porcine model, whose subcutaneous tissue more closely models humans, was used by Dinsdale to establish irreversible tissue damage after 2 hours of constant pressure of 70 mm Hg.<sup>9</sup> However, if the pressure was relieved for at least 5 minutes, tissue damage was minimized even at elevated levels of stress. Husain used a rat pressure sore model to conclude that sustained low pressure induces more tissue damage than brief periods of high pressure.<sup>10</sup> These basic science investigations form the core body of knowledge describing how the element of time and its relationship to soft tissue necrosis is critical to the formation of pressure sores.

Prolonged contact with objects is associated with patient immobility for several reasons, including broken bones, paralysis, psychiatric diagnoses, and loss of consciousness. Paralysis may be due to trauma, neoplasm, infection, iatrogenic injury, and radiation. Suspicion of an infectious etiology requires careful delineation between a bacterial source such as *Campylobacter jejuni* and viral subtypes that include the West Nile virus and the rare poliomyelitis. Psychiatric diseases such as catatonic schizophrenia should be treated promptly. All of these patients are at risk for the development of pressure sores as the time of immobility continues.

# Spasticity

Spasticity of muscles contributes to pressure sore development by prolonging immobility and impeding surgical intervention. In a healthy person, the normal upper motor neuron pathway inhibits the contraction of alpha motor neurons and the intrafusal fiber gamma motor neurons <sup>18</sup> (Fig 3). Interruption of the upper motor neuron pathway may result in

#### TABLE 1. Ashworth scale for classifying spasticity

- 0 = No increase in tone
- 1 = Slight increase in tone giving a "catch" when the limb was moved in flexion or extension
- 2 = More marked increase in tone but limb easily flexed
- 3 = Considerable increase in tone—passive movement difficult
- 4 = Limb rigid in flexion or extension

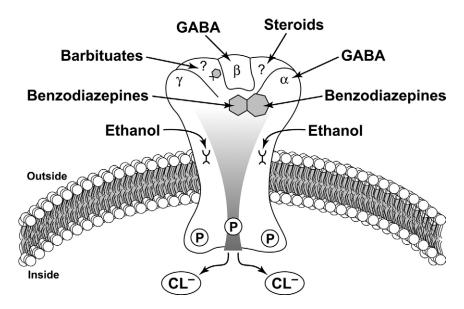
(Adapted from Ashworth B. Preliminary trial of carisoprodol in multiple sclerosis. Practitioner 1964;192:540-2.)

hypertonicity of muscles, clonus, and sometimes involuntary movements. The bladder may also be spastic, which has a decreased carrying capacity and undergoes spontaneous voiding, which contributes to skin maceration. Causes of spasticity include spinal cord trauma, stroke, multiple sclerosis, and cerebral palsy. The degree of spasticity may change over time and is best categorized by the Ashworth scale<sup>19</sup> (Table 1).

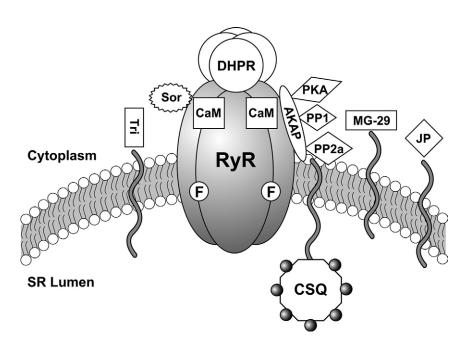
Specific treatments for spasticity should be related to the severity of illness. One fundamental treatment option is the use of physical therapists to provide range-of-motion and stretching exercises for patients. This is particularly beneficial in adults with multiple sclerosis and those who have suffered a stroke.<sup>20,21</sup> Pharmacological treatment may use medications including GABA<sub>A</sub> and GABA<sub>B</sub> agonists (eg, diazepam and baclofen, respectively) and sarcoplasmic reticulum calcium channel blockers (eg. dantrolene), which bind to specific sites on receptors<sup>22,23</sup> (Figs 4 and 5). Children with cerebral palsy can demonstrate improvement in both upper and lower extremity spasticity using intrathecal baclofen infusion pump therapy.<sup>24,25</sup> If physical and pharmacological therapies fail to improve the spastic condition, patients should be evaluated by a physiatrist. Failure to benefit from conservative therapy may require more complex neurosurgical interventions, including phenol injections, neurectomy, myelotomy, dorsal rhizotomy, and cordectomy. 26-28 However, highly invasive remedies should be reserved for 18 months to 2 years after injury to allow for maximal recovery from a spinal cord procedure.

#### Infection

Skin is the first and most important barrier to infection by microorganisms. Normally, the skin is in a symbiotic relationship with certain microbes to prevent pathological infestation. Normal inhabitants of skin are predominantly of the genera *Staphylococcus* (especially *S. epidermidis*) and *Micrococcus*. These organisms prevent infection by competitively binding to skin, using essential nutrients, and excreting unfavorable



**FIG 4.** The  $GABA_A$  receptor.



**FIG 5.** The ryanodine receptor is responsible for mediating intracellular calcium mobilization in muscle cells. Dantrolene prevents calcium release by the ryanodine receptor in the sarcoplasmic reticulum.

compounds.<sup>29</sup> An example of the latter is *Propionibacterium acnes*, which secretes oleic acid, an inhibitor of fungi and Gram-negative bacteria.

By maintaining proper hygiene, the opportunity for pathologic bacteria to cause infection is reduced. Similarly, nosocomial sources of infection are combated by frequent hand washing and the use of alcohol-based skin cleansers. Soiling of the skin—such as from urine and feces—can introduce microorganisms to the skin surface. This is especially of concern in the paraplegic, wheelchair-bound patient with a developing pressure sore. The use of Foley catheters or intermittent catheterization is a must-use prevention technique for patients with neurogenic bladders. The presence of infectious microbes, which flourish in the media-like cellular breakdown products due to tissue necrosis, at a minimum prevents healing of the damaged tissues. Soft tissue infections can spread superficially along tissue planes and deeper into subcutaneous structures, requiring advanced surgical debridement, which complicates reconstructive efforts.

Several studies have examined colonization of traumatized tissue. Surgical sites can become infected from distant, endogenous sources of bacteria, and a pressure sore can become a nidus for infection.<sup>32</sup> After attaching metallic spheres to rabbit gluteal muscles, Groth injected a bacterial solution into their veins.<sup>33</sup> The bacteria localized to sites of compression and resulted in necrosis at lower pressures when compared with control animals not receiving the bacterial injection. Similarly, injection of bacteria and then applying pressure serves as a homing device for microbes.<sup>10</sup> In fact, experimental surgical wounds created at sites of pressure that are infected with a known bacterial load have bacterial growth that is 10<sup>2</sup> higher than controls.<sup>34</sup> Thus, compressed skin has less resistance to bacterial invasion.<sup>35</sup>

### Edema, Denervation, and Moisture

The fluid environment of pressure sores contributes to both infection and necrosis. Fluid accumulates either as edema in the interstitial spaces or as moisture surrounding the outside of the wound area. Compression of soft tissues causes edema by occluding venous outflow and raising the interstitial pressure in the affected tissue. Edema can be secondary to patient disease states. Malnutrition and cirrhosis of the liver causing hypoalbuminemia, a low cardiac ejection fraction, and renal diseases can cause edema. The resulting edema accumulates in the dependent regions of the body, which is a factor of positioning in the bed or chair.

The investigation into how edema affects tissue perfusion has been critical to our understanding why tissue necrosis occurs. Landis used a

microinjection technique to discover the blood pressure of individual capillaries. <sup>36</sup> An external pressure of greater than 12 mm Hg will result in occlusion at the venous end of the capillary. End capillary venous pressure then rises and causes plasma extravasation and ensuing edema. Further increasing the pressure to 32 mm Hg occludes blood flow into the capillary, preventing tissue perfusion with oxygen and nutrients and removal of toxic metabolic waste products. With the passage of time, ischemia and necrosis result, and this promotes pressure sore formation as discussed previously.

A contributing factor to edema is denervation of blood vessels. The inability of the capillary's afferent supply to regulate its diameter defaults to a state of dilation.<sup>37</sup> Such denervation edema has been documented in the ipsilateral one half of the tongue after reconstructive surgery.<sup>38</sup> Documentation of denervation edema is best accomplished using magnetic resonance imaging.<sup>39</sup>

Several studies on denervation edema demonstrate the serious nature of this problem. Acutely denervated rat cremaster muscles experience a more severe ischemia-reperfusion injury after 3 hours of ischemia. Alison and colleagues studied the effects of denervation on island pedicle flaps in sheep buttocks that receive an intradermal inoculation with 10<sup>7</sup> *Staphylococcus aureus*. The prolonged denervation group demonstrated a 25-fold increase in bacterial counts and a significant increase in tissue edema. A small study comparing 6 patients with chronic sympathetic denervation to 5 healthy controls found that nondenervated limbs decreased blood flow by 50% when venous pressure was elevated to more than 30 mm Hg. At 40 mm Hg, nondenervated limbs have one third less the capillary filtration of chronically denervated limbs. These studies demonstrate the importance of intact innervation to regulate blood flow and prevent edema in tissues.

Moisture is the external accumulation of fluid on skin. The source of moisture can be from perspiration, incontinence, and wound exudate. These liquids cause maceration—the wrinkling of skin—which contributes to the setup for infection after prolonged exposure. Thus, maceration is a contributing factor to the prevention of wound healing. Moisture on pressure sores should be prevented by (1) frequent dry dressing changes, (2) the use of urinary catheters for incontinent patients, and (3) bowel regimens that include examination for and cleansing of stool.

#### **Nutrition**

Nutritional intervention for pressure sore patients must be part of the plastic surgical evaluation. Poor nutrition is a risk factor for the formation

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Males: 66 + 13.8 (kg) + 5 (cm height) - 6.8 (age)
Females: 655 + 9.6 (kg) + 1.8 (cm height) - 4.7 (age)
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of pressure sores, their exacerbation, and prevent healing from surgical procedures. Certain basic metabolic components are recognized as essential for survival and the repairing of surgical wounds. The patient should be evaluated carefully by a nutritional expert to determine the caloric needs and whether these demands are being met. The causes of malnutrition are manifold: poor intake (eg, alcoholism), chronic nausea and vomiting (eg, pancreatitis), hypermetabolic states (eg, burns), socioeconomic forces (eg, natural disasters), and internal organ diseases (eg, nephrotic syndrome). Supplementing nutritional intake is best performed by encouraging an appropriate oral diet. However, duodenal feeding tubes or parenteral feeds should be used to prevent a catabolic state.

Proper caloric intake is necessary for the human body to function. Energy requirements are a product of the body's basal energy expenditure. This can be calculated using the Harris Benedict formula (Table 2). Although debatable, a patient's daily caloric need is the basal energy expenditure multiplied by a stress factor: 1.3 for mild stress, 1.4 for moderate stress, and 1.5 for severe stress. In comparison, burn patients are variably hypermetabolic and should be examined clinically at the time of hospitalization. <sup>44</sup> Protein requirement also ranges from 1 to 2 g/kg of body weight per day. <sup>45</sup> The end goal is to meet nutritional requirements for normal wound healing without overfeeding.

Evaluation of the patient's current nutritional status often includes clinical laboratory tests such as albumin or prealbumin. Fifty percent of serum protein synthesized by the liver is albumin, the most abundant plasma protein. Yet, serum albumin levels are not valuable indicators of nutritional status in the marasmic and critically ill patient. An exclusion is made for individuals with kwashiorkor whereby the high carbohydrate diet results in a state of low serum albumin and tissue edema. Studies also fail to link prealbumin as a predictor of patient nutritional status and prognosis in critically ill patients. Clinical judgment should be applied in patients receiving nutritional therapy; the patient should be gaining strength and the wound site should be inspected regularly for signs of healing.

Many nutrients, vitamins, and minerals contribute to the wound healing process. One well-demonstrated example is the synthesis of collagen by fibroblasts (Fig 6). Amino acids, particularly lysine and proline, must be

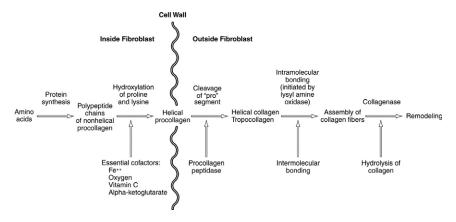
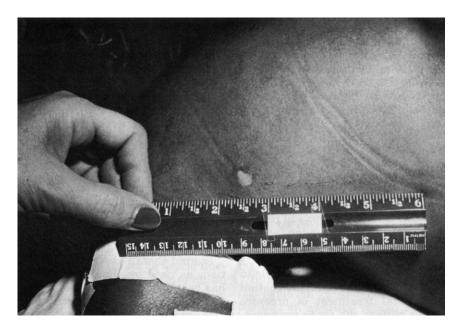


FIG 6. The pathway for collagen synthesis.

available to synthesize the collagen protein. Additionally, oxygen, iron, vitamin C, and alpha-ketoglutarate are necessary cofactors for the hydroxylation of lysine and proline to allow intermolecular bonding of the collagen polypeptides into the triple helix procollagen. Failure to ingest adequate amounts of vitamin C results in decreased strength of healing scars, a prolonged healing process, and even dehiscence of previously healed scars.<sup>49</sup>

Other supplements should be provided to the pressure sore patient for optimal wound healing. Calcium is used for the conversion of procollagen to fibrillar collagen. Iron, folate, and vitamin B<sub>12</sub> supplementation helps to optimize erythropoiesis in the anemic patient. Adequate levels of vitamin K are essential for blood clotting. Additionally, patients taking corticosteroids should receive vitamin A supplementation to counteract the lysosomal stabilization that retards the wound healing process. <sup>50</sup>

In addition to nutritional deficiencies, metabolic disturbances such as diabetes should be monitored carefully and treated to prevent delaying the normal healing process. Diabetes mellitus is a chronic disorder of hyperglycemia requiring daily monitoring for euglycemia and quarterly hemoglobin A<sub>1C</sub> levels. Several problems are associated with the hyperglycemic state. In a diabetic rat model, Komesu and colleagues demonstrated a prolongation in the inflammatory response phase of wound healing. Hahey and colleagues established that diabetic mice fail to generate proper interleukin-6 levels after 7 days of healing. Histological analysis of these wounds found decreased neovascularization and less organization of granulation tissue in the wounds of diabetic mice.



**FIG 7.** Bed rail-induced pressure sore. (With permission from Colen SR. Pressure sores. In: McCarthy JG, editor. Plastic Surgery: The Trunk and Lower Extremity. Philadelphia: WB Saunders; 1990; Chapter 77, pp 3797-838.)

Robson and Heggers<sup>56</sup> performed in vitro and in vivo studies on infection and varying glycemic levels. They demonstrated an increased risk of Gram-positive septicemia in patients with elevated levels of serum glucose of at least 130 mg/100 mL.<sup>56</sup> Conversely, Gram-negative sepsis occurred in patients with blood glucose levels of less than 110 mg/100 mL. It is not surprising that a diabetic patient with an ulcer is at greater risk of infection and complications above and beyond nondiabetic patients, despite tight glucose control and meticulous wound care.<sup>53</sup> Thus, the metabolic status of diabetic patients affects their ability to resist bacterial infection, prevent skin breakdown, and promote wound healing.

### Location of Pressure Sores

The vast majority of pressure sores result from contact between the patient and the posterior or lateral surface of the pelvis. The surgeon must be cognizant that other locations of the body can undergo stress necrosis from prolonged contact with objects (Figs 7 and 8). This includes arms leaning over bed rails, nasogastric and feeding tubes taped upward against the nasal ala, and the forehead or mental area in prone patients.



FIG 8. Penile pressure sore from a Foley catheter and severe penile edema.

As noted previously, the immobilized person is most susceptible to pressure sore formation. From the Yeoman and Hardy study of 240 paraplegics we know that more than 50% of pressure sores form at the ischium and sacrum<sup>57</sup> (Table 3). The study also documented the heel, external malleoli, tibial crest, and costal margin as nonpelvic sites susceptible to pressure sore formation. Such information was virtually duplicated by Dansereau and Conway in their study of 2000 paraplegics with pressure sores, adding only the high incidence of trochanteric pressure sores<sup>58</sup> (Table 4). In a study from Denmark by Petersen and Bittmann, almost two thirds of pressure sores occurred while patients are in the hospital.<sup>2</sup> Fifty-three percent of patients were bedridden, one third were wheelchair bound, and only 10% were ambulatory patients. Again, sacral, ischial, and trochanteric pressure sores were the most common sites (Fig 9). Preventive measures require early ambulation, frequent turning of their patients, and the use of adequate support surfaces to disperse the patient's weight evenly and minimize external compression of soft tissues.

TABLE 3. Pressure sore locations from the Yeoman and Hardy study

Site	Number	Percentage
Ischium	68	28
Sacrum	64	27
Heel	44	18
Trochanter	27	12
External malleoli	20	8
Tibial crest	10	4
Anterosuperior spine	5	2
Costal margin	2	1
Total	240	100

(With permission from Colen SR. Pressure sores. In McCarthy JG, editor. Plastic Surgery: The Trunk and Lower Extremity. Philadelphia: WB Saunders; 1991; Chapter 77, pp 3797-838.)

TABLE 4. Pressure sore locations from the Dansereau et al study

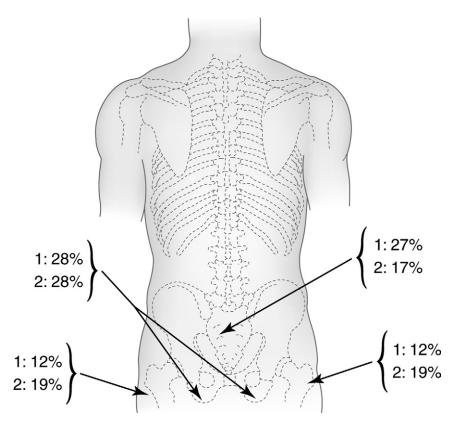
Ischial tuberosity	28%
Trochanter	19%
Sacrum	17%
Heel	9%
Malleolus	5%
Pretibial	5%
Patella	4%
Foot	3%
Anterosuperior spine	2.5%
Elbow	1.5%
Miscellaneous	6%
	100%

(With permission from Colen SR. Pressure sores. In McCarthy JG, editor. Plastic Surgery: The Trunk and Lower Extremity. Philadelphia: WB Saunders; 1990; Chapter 77, pp 3797-838.)

### Cone of Necrosis

The configuration of the pressure sore is that of a 3-dimensional cone<sup>61</sup> (Fig 10). Similar to the proverbial iceberg, the majority of injury due to pressure necrosis is deeper and larger below the surface (Fig 11). Although pressure causes ischemia of the skin, the subcutaneous muscle has a higher metabolic activity and thus is more sensitive to prolonged anoxia. Further, objects trap the underlying muscle between the skin and the deeper bony prominence. Thus, the human endoskeleton acts as a pressure point for the external object.

Nola and Vistnes performed histological analysis of skin and muscle over bony prominences subjected to pressure in animals. <sup>62</sup> Bony prominences that developed superficial ulceration demonstrated breakdown of the dermis and necrosis of muscle fibers. Histology samples taken from sites of intact,

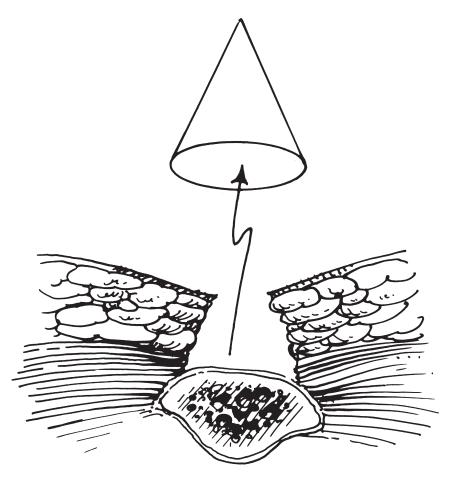


**FIG 9.** Location and percentages of pelvic pressure sores. 1 = Yeoman and Hardy study<sup>59</sup>; 2 = Dansereau study.

nonulcerated skin showed significant muscle necrosis underneath. Similarly, Daniel and Faibisoff found almost total muscle atrophy in myocutaneous flaps used to cover pressure sores. These studies suggest that plastic surgeons should avoid transposing myocutaneous flaps to cover pressure-bearing bony prominences. However, muscle can be used to obliterate an anatomical dead space from excision of bone and scar tissue, and innervated (ie, sensory) flaps can be used to prevent recurrence. 64

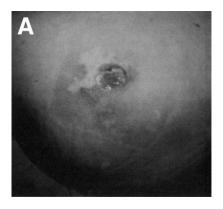
# Stages of Pressure Sores

There are 4 generally accepted stages of pressure sores (Table 5). Stage I is exemplified by continued erythema of skin that cannot be blanched with a gloved finger. The skin is still intact and no ulceration is yet evident. Progression to stage II involves damage to the skin via ulceration, blistering,



**FIG 10.** The cone of necrosis. (With permission from Phillips LG, Robson MC. Pressure ulcerations. In: Jurkiewicz MJ, Krizek TJ, Mathes SJ, Ariyan S, editors. Plastic Surgery: Principles and Practice. Vol. 2. St Louis: CV Mosby; 1990; pp 1223-51.)

or abrasion. Often these 2 stages are indicators of deeper necrosis. They also may occur independently as sheer injuries, caused by sliding against sheets or support surfaces. Stage III lesions represent full-thickness destruction of the skin. Although the ulcer may not visibly extend into muscle, the pressure itself usually causes necrosis of the underlying muscle as discussed previously. Stage IV lesions include involvement of muscle, tendons, joints, nerves, and even bone. Stage III and IV lesions require surgical intervention because these defects are often large and infected and require sharp debridement.



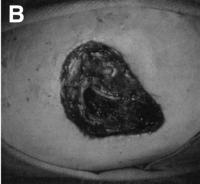


FIG 11. External appearance versus the large base of a trochanteric pressure sore. (With permission from Phillips LG, Robson MC. Pressure ulcerations. In: Jurkiewicz MJ, Krizek TJ, Mathes SJ, Ariyan S, editors. Plastic Surgery: Principles and Practice. Vol. 2. St Louis: CV Mosby; 1990; pp 1223-51.)

TABLE 5. Consensus classification/stages of pressure sores

Pressure sore staging system					
Stage I	Nonblanchable erythema of intact skin (heralding lesion of skin ulceration).				
Stage II	Partial-thickness skin loss involving epidermis or dermis. The ulcer is superficial and presents clinically as an abrasion, blister, or shallow crater.				
Stage III	Full-thickness skin loss involving damage to or necrosis of subcutaneous tissue that may extend down to, but not through, underlying fascia. The sore presents clinically as a deep crater with or without undermining of adjacent tissue.				
Stage IV	Full-thickness skin loss with extensive destruction, tissue necrosis, or damage in muscle, bone, or supporting structures (for example, tendon or joint capsule).				

Adapted from U.S. Department of Health and Human Services (HHS): Pressure Ulcers in Adults: Prediction and Prevention. Clinical Practice Guideline No. 3, Publication 97-0047. HHS, Rockville, MD, 1992.

# **Prevention Strategies**

Since the vast majority of pressure sores occur in the hospital, caregivers must have a regimen for inspecting the skin of immobilized patients. In addition, the presence of spasticity in a limb, proximity of bed rails, nasogastric tubes, and other solid objects should encourage frequent assessment for skin breakdown. The formation of a pressure sore depends on the severity of pressure, shape of the object creating the pressure, location on the body, and the length of time. Thus, patient repositioning and the use of padding or special beds (see below) is a crucial prophylactic mechanism for tissue breakdown.

TABLE 6. The Braden scale<sup>65</sup>

BRADEN scale					
Sensory perception	1 Completely limited	2 Very limited	3 Slightly limited	4 No impairment	
Moisture	1 Constantly moist	2 Very moist	3 Occasionally moist	4 Rarely moist	
Activity	1 Bedfast	2 Chair fast	3 Walks occasionally	4 Walks frequently	
Mobility	1 Completely mobile	2 Very limited	3 Slightly impaired	4 No limitations	
Nutrition	1 Very poor	2 Probably inadequate	3 Adequate	4 Excellent	
Friction & shear	1 Problem	2 Potential problem	3 No apparent problem		
A score of 18 or	Total Score				
Completed by: In	itials:	Date:	Time:		

Nurses are the eyes and ears of the away physician, and they must be attentive to the risk factors for pressure sore formation. A valuable tool for patient care is the Braden scale, an inventory survey used by nurses to assess a patient's risk for pressure sore formation<sup>65</sup> (Table 6). There are 6 categories: sensory perception, moisture, activity, mobility, nutrition, and friction and shear. The first 5 categories are ranked 1 to 4, with a score of 1 assigned to a malady condition and a score of 4 for problem-free categories. Friction and shear are ranked only 1 to 3, with 3 meaning no problem. A sum of 18 or less is the threshold for an increased risk for pressure sore formation, and the nurse should inform the treating physicians. These patients require preventive care to avoid nosocomial pressure sore development.

The primary cause of pressure sore progression through the various stages is continued pressure. This cycle must be interrupted at the earliest point of recognition to allow adequate tissue perfusion and healing of the wound. One particular strategy is to disperse the pressure evenly across the body surface as it contacts the patient. Nurses must eliminate wrinkles and folds in the bedding and remove foreign objects from skin contact. The patient should be aligned in the median of the bed, away from the railing.

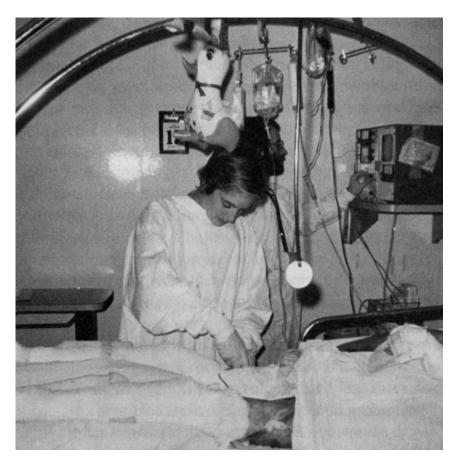
Spinal cord injury patients are at an increased risk for pressure sore development in the immediate post-injury time period. Linares and colleagues retrospectively interviewed acute spinal injury patients and found a direct correlation between pressure sore formation and failure to turn the patient within 2 hours after injury.<sup>68</sup> In a follow-up prospective study, this same group of researchers concluded that time on the spine board correlated directly with acute pressure sore development. In

addition, there is an inverse relationship between systolic blood pressure and early pressure sore formation.<sup>69</sup> Thus, regular turning of immobilized persons, especially spinal cord injury patients, should begin within 2 hours of injury, even during assessment and transport.

Hospital staffing can be a challenge, especially when the wards have high numbers of immobilized patients or those with impaired mobility. To help alleviate this nursing burden, several bed technologies exist to assist in patient turning, also known as kinetic therapy. These include the Foster and Stryker frames, the CircOlectric bed, and the Keane RotoRest bed. The Foster and Stryker frames alternate the patient from a prone to a supine position via hand turning of the frames. The CircOlectric bed is an automatic turning device allowing for vertical elevation of the head, but is rather large, expensive, and time-consuming to monitor (Fig 12). The Keane RotoRest bed is the least burdensome of the group (Fig 13). Nurses place patients on this 4-inch thick mattress with numerous side attachments and forward braces. The bed then automatically and gently rocks the patient from side to side. The benefit of kinetic therapy is to shift the pressure points and to enhance lung perfusion, which minimizes atelectasis and nosocomial pneumonia. <sup>70,71</sup>

Regular turning of patients should be supplemented with minimization of point pressure. Three types of bed technology are available to address this issue: the air mattress, waterbeds, and air-fluidized beds. Air mattresses are categorized by 2 types: the static air mattress and the alternating pressure mattress. The static air mattress is lightweight and inexpensive but not favored by nurses who must wrestle with the device to move patients. The alternating pressure mattresses utilize internal air cells and an electric air pump to inflate and deflate the air pockets. These beds require correct patient positioning, monitoring of the cells for leakage, and an electrical source. Leakage in the tubing or mattress cells can burn up the electric pump, which creates noise pollution to those in proximity of the pump's normal operation. The efficacy of alternating pressure mattresses is questionable with few well-controlled studies to support the device.<sup>72</sup> Water mattresses are heavy, bulky, and cannot be fashioned into a seated position. Leakage of their water volumes can endanger the patient and flood the room.

All of the previously mentioned mattresses have polyvinyl outer coverings. Patients often complain of the additional heat retention created by the material. Also, the occlusive nature to the mattress shell prevents moisture wicking and promotes skin maceration, infection, and pressure sore formation. To combat the numerous tribulations associated with mattresses, the air-fluidized bed uses both warmed air and optical glass



**FIG 12.** A CircOlectric bed. (With permission from Phillips LG, Robson MC. Pressure ulcerations. In: Jurkiewicz MJ, Krizek TJ, Mathes SJ, Ariyan S, editors. Plastic Surgery: Principles and Practice. Vol. 2. St Louis: CV Mosby; 1990; pp 1223-51.)

spheres to literally float patients. The best known of these is the Clinitron bed (Fig 14). The maximal pressure exerted on the patient skin surface is 10 mm Hg, which effectively eliminates the pressure element of tissue necrosis. Additionally, the polyester sheet containing the spheres allows for absorption and drying of the patient contact surface.

The sparse popularity and limitation of the air-fluidized bed use comes from its disadvantages. Dehydration may occur particularly in the extensively burned patient who cannot effectively limit evaporative water losses. The bulky largesse of a bed weighs approximately 1 metric ton. Moving the air-fluidized bed requires extreme caution because the device can gain momentum and injure the patient or transporting team. Bolsters,

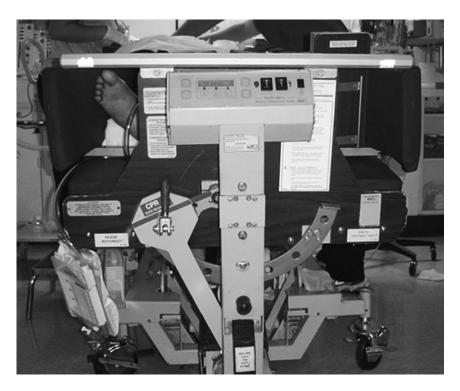


FIG 13. A Keane RotoRest bed.

dressings, or pillows effectively negate the bed's ability to evenly distribute pressure throughout the patient's contact surface area. Elderly patients may develop disorientation in these beds.

#### Clinical Evaluation

All patients admitted to the hospital should be evaluated continually for possible pressure sore formation. This begins with a clinical index of suspicion based on admitting diagnosis, current state of mobility, type of treatment being rendered to the patient, and the iatrogenic effect on the patient's life. Those patients with a history of a previous pressure sore(s) are of particular concern. Patients with diabetes, paraplegia, and quadriplegia may have some component of insensate body parts over bony prominences. The inability to recognize numbness or pain due to pressure necrosis increases a person's susceptibility to ulceration and pressure sore formation. Further, physically incapable or deconditioned persons cannot move despite their desire to readjust and relieve the pressure. All these



**FIG 14.** A Clinitron bed. (With permission from Phillips LG, Robson MC. Pressure ulcerations. In: Jurkiewicz MJ, Krizek TJ, Mathes SJ, Ariyan S, editors. Plastic Surgery: Principles and Practice. Vol. 2. St Louis: CV Mosby; 1990; pp 1223-51.)

patients need a caregiver to be mindful of the condition and help move the patient every 2 hours or sooner.

The initial discovery of skin breakdown can come from the patient or caregiver. Drainage from the wound soils the patient's clothes or linens. Patient discomfort during the recovery from their medical illness may precede external evidence of pressure sore development in the subcutaneous tissues. The usual method of pressure sore discovery comes from a thorough physical examination despite the absence of signs or symptoms.

Inspection of the wound itself demonstrates the classic hallmark signs: rubor (erythema), tumor (edema), calor (warmth), and dolor (pain) in the

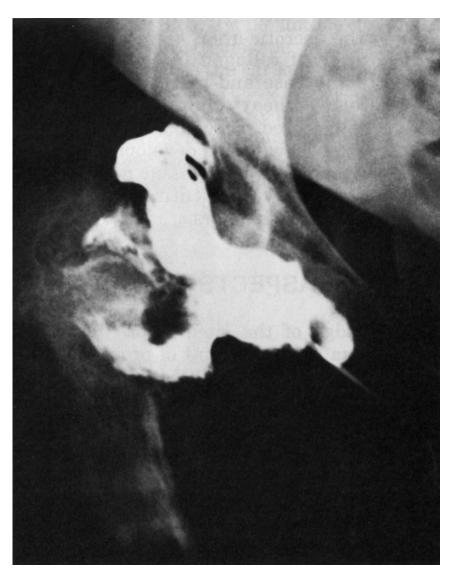
sensate patient. Gentle pushing on the wound with a gloved hand may reveal underlying fluctuance or loculated fluid from liquefaction necrosis of subcutaneous tissues. This may be covered by an eschar composed of necrotic skin that has desiccated. Such wounds slough their eschar cap and reveal a deep, cone-shaped ulcer cavity.<sup>74</sup> If the wound is suspected to connect with surrounding structures, a sinogram may be desired (Fig 15). Pressure sores can involve surrounding bone, joint spaces, and sometimes rectal or urethral fistulas with infection and exacerbation of injury.

Stage IV pressure sores have abundant necrotic tissue and often surrounding cellulitis. Blood cultures are in order if the patient has signs and symptoms of sepsis: fever, elevated white blood cell count with a "left" shift, rigors, sweating, or change in mental status. Contributing factors must be addressed. This includes biopsies of the pressure sore itself to be sent for quantitative and qualitative wound cultures. The wound probably has a bacterial count greater than 10<sup>5</sup> bacteria/g of tissue, thus negating the effect of systemic antibiotics. Further, the poor oxygen tension of the wound is a function of distant blood vessels, which contributes to poor circulation of antibiotics. Once the wound is surgically debrided, targeted antibiotic therapy and local wound care promote wound healing.

Urine incontinence is defined as involuntary loss of urine and must be addressed to prevent maceration and soiling of the wound. Urine culture with request for bacterial sensitivities should be sent to the laboratory, and a Foley catheter can prevent further urine contact with the wound. A raison de'être must be investigated because some patients may have treatable conditions such as a urinary tract infection or bladder spasticity. Urology consultation can provide a diagnosis and treatment regimen for patients.

Stool incontinence warrants frequent dressing or clothing changes. This is particularly difficult for patients suffering from diarrhea. Diarrhea that accompanies recent administration of antibiotics mandates sending stool cultures for possible *Clostridium difficile* infection despite the absence of the cytotoxin. If no pathological or bacterial source of the diarrhea is identified, the use of codeine or Lomotil can assist the bowel to create more formed stools. Spinal cord injury patients with formed stools may benefit from routine use of suppositories to induce scheduled bowel movements. Patients with recurrent pressure sores complicated by fecal contamination should be referred to a general surgeon for a possible diverting colostomy, although this practice should be used with caution.

Preoperative labs can identify other sources of problems that delay wound healing. The presence of anemia and hypoalbuminemia should



**FIG 15.** A sinogram demonstrating joint capsule involvement. (With permission from Colen SR. Pressure sores. In McCarthy JG, editor. Plastic Surgery: The Trunk and Lower Extremity. Philadelphia: WB Saunders; 1990; Chapter 77, pp 3797-838.)

trigger suspicion of a poor nutritional intake or other chronic disease state. Hemoglobin  $A_{\rm IC}$  and random blood glucose levels are necessary in the diabetic patient. All such issues should be addressed via consultation with the appropriate specialist.

Auscultation of the lungs is a vital component to the preoperative patient evaluation. Detection of wheezing, crackles, or coarse breath sounds merits further analysis. Chest radiographs are warranted in patients with poor diaphragmatic excursion. This includes quadriplegic patients and those with chronic obstructive pulmonary disease. Pulmonary insufficiency can be secondary to a cardiac source, and occasionally, echocardiography is a necessary preoperative measure.

The continued popularity of nicotine and the many products containing nicotine—including cigarettes, cigars, and smokeless "chew"—should be considered in performing flaps. Cigarette smoking decreases subcutaneous tissue oxygen tension lasting up to 50 minutes after smoking due to nicotine's vasoconstrictor effects. Reus and colleagues compared outcomes in elective free-tissue transfers between 51 nonsmokers and 93 smokers who quit perioperatively. There was no difference in anastomotic patency, but smokers experienced delayed wound healing at the recipient site and more often required an additional procedure to achieve final wound closure.

Designing a care plan for the pressure sore requires a full and frank discussion of expected and possible outcomes. The patient needs complete understanding of the large investment being made to treat this potentially lethal condition. The clinician must carefully assess the patient's ability to understand and provide the acute cooperation and the long-term commitment of preventing recurrence. Family care providers must be included both to educate the patient and to provide an outline for the ensuing care, which has the greatest chance of compliance and ultimate success.

### Treatment of Pressure Sores

Therapeutic surgical intervention follows maximization of patient health and education. The goals are similar in all pressure sore cases (Table 7).

The history and physical examination of the pressure sore patient is critical to determine which goals may be achieved. Some debilitated patients cannot tolerate a surgical procedure and should be considered for nonoperative therapeutic intervention. Comorbid conditions include congestive heart failure, noncardiogenic pulmonary diseases, atherosclerosis with carotid or coronary artery disease, and uncooperative patients. Terminally ill patients are treated more supportively and less aggressively. The specific problem should be evaluated by the anesthesiologist, psychologist, or social worker to determine limitations or causes of a suboptimal outcome, including recurrence. All

#### TABLE 7. Goals of treatment

- 1. Total excisional debridement of the necrotic tissue, ulcer base, scar tissue, bursa, undermined skin (if not extensive), and heterotopic calcification
- Excision of infected bone, bony prominence recontouring to eliminate isolated pressure points
- 3. Acquisition of hemostasis after debridement to healthy tissue and placement of low suction drainage(s), if appropriate
- Obliteration of the anatomical dead space using muscle, myocutaneous flaps, and/or deepithelialized skin flaps to provide sufficient bulk to cushion bony prominences
- Coverage of the wound with vascularized flaps placed to create a tension-free repair over the defect
- Placement of the flap so as to position the stress point away from the suture line, a point of diminished blood flow
- 7. Restoration of the patient's activities of daily living before developing the pressure sore

patients require the same basic care to treat local or distant infection, remove necrotic tissue if indicated, and promote healing. Once these goals are attained, treatment is directed to surgical or nonsurgical closure of the wound.

# Nonsurgical Treatment

Myriad information is available on the subject of topical wound therapy. Topical therapy for the treatment of a pressure sore includes a spectrum of enzymes for debridement, elemental pastes, antimicrobials, and solutions. We kucan and colleagues compared the efficacy of silver sulfadiazine to saline and povidone-iodine in preparing pressure ulcers for closure. All wounds treated with silver sulfadiazine (15 patients) reduced the bacterial count to less than 10<sup>5</sup>/g of tissue within 3 weeks. Comparatively, the use of saline was more effective than povidone-iodine (79% versus 64%, respectively).

Topical antimicrobials include compounds such as bacitracin, polymyxin, neomycin, silvadene, and sulfamylon. One problem with these antibiotics is patient sensitization, allergy, and systemic toxicity. The clinician should look for these complications when treating large wounds subjected to frequent application of the compound in question. Although there is no such concept as the typical pressure sore—only typical locations—the microbial flora is presumed to be either *Staphylococcus*, *Proteus*, *Pseudomonas*, or *Bacteroides* species. The astute clinician should send tissue biopsies before starting any broad-spectrum topical antimicrobial. The wound-specific flora is best treated with an antimicrobial causing the highest level of growth inhibition as determined by laboratory testing. Systemic antibiotics are then used as an adjunct to the

topical antimicrobials for treatment of cellulitis, osteomyelitis, or joint-space infections.

Spontaneous wound closure in small to medium size pressure sores enhances as the bacterial counts decline. The finalized product is scar tissue with poorly developed skin covering with a poor vascular supply and requires manual lubrication. The stiff and friable skin can readily be injured by pressure or shearing, and recurrence is higher than for surgical intervention.

Topical application of growth factors should be considered in treating the nonoperative pressure sore. Robson and colleagues demonstrated a substantial reduction in pressure ulcer volume from the application of platelet-derived growth factor, a potent activator of wound healing cells. Conversely, a prospectively randomized, blind, placebo-controlled trial failed to demonstrate improved pressure ulcer healing with interleukin- $1_{\beta}$ . For the more challenging diabetic patients, local injection of basic fibroblast growth factor improves wound breaking strength in a diabetic rat model to a statistically significant level (P < 0.001). Growth factor treatment of pressure sores remains a clinical trial treatment, in part due to its significant cost.

The main goal in treating pressure sores is to alleviate the pressure point. Although infection is 1 of the many inhibitors to wound healing, the pressure and shearing forces must be eliminated or closure will not occur. Therefore, the patient should be shifted in position every 2 hours and 1 of the bed technologies should be employed to maximize pressure distribution. Additionally, there are health care products designed as local pressure distributors. One such product is Allevyn Adhesive, which absorbs moisture and attempts to redistribute the wound pressure forces. Other brand name products include Tielle, Mepilex Border, Tegaderm, CombiDERM, Lyofoam, and Biatain. There are very few randomized, controlled trials with these products. Each manufacturer has specific designs and patterns to fit variously shaped pressure ulcers based on the wound location. This adds to the complexity in choosing a product and deciding about the cost effectiveness.

Another technology-based wound closure device is the subatmospheric vacuum-assisted closure technique. <sup>88</sup> Vacuum Assisted Closure (V.A.C.) Therapy, promoted by Kinetic Concepts, Incorporated, uses controlled, localized negative pressure to promote granulation tissue formation, decrease tissue edema, and remove infectious materials (Fig 16). In a retrospective review of 25 patient charts, Weed and colleagues, did not find the V.A.C. to reduce bacterial colonization but rather increased it. <sup>89</sup> A prospective, randomized trial of 54 patients found vacuum-assisted



FIG 16. Vacuum-assisted closure device applied to a wound.

closure to decrease nonfermentative Gram-negative bacilli (P < 0.05), increase *S. aureus* (P < 0.05), and effect no change in quantitative bacterial load.<sup>90</sup>

The V.A.C. is used for numerous wound types. A thorough literature search identifies numerous case reports describing V.A.C. therapy for open sternal wounds, fascial reapproximation in trauma patients with open abdomens, gynecologic oncology wound failures, securing split-thickness skin grafts, and full-thickness skin graft positioning for degloving injuries. However, toxic shock syndrome has been reported from the use of the V.A.C., and 1 meta-analysis sharply notes the weak evidence for V.A.C. therapy versus conventional methods. In a porcine model, Miller and colleagues failed to establish a histologically based benefit to the V.A.C. therapy versus wet-to-dry dressing changes.

The use of V.A.C. therapy in pressure ulcers is unsubstantiated by the literature. Again, an operative intervention will provide the ideal outcome in larger pressure sores. The surgeon must decide the most practical approach and potential benefit of this emerging technique on a per-patient basis. <sup>101</sup>

# Operative Technique

Pressure sore wound debridement is best performed in the operating room under general endotracheal anesthesia, where lighting and hemostasis are superior. The anesthesiologist can carefully monitor the patient for signs of pain, excess blood loss, hypotension, and shock while the surgeon focuses on treating the wound. Local therapy is directed to obtain complete removal of necrotic debris, decreased bacterial load, and preparation for wound closure and healing. Beyond sharp debridement the surgeon can use pulsating jet lavage on the wound. Animal studies suggest that substantial wound bacterial count reductions can more consistently be achieved using pulsating jet lavage versus bulb syringe. However, 2 studies of high pressure jet lavage performed on ex vivo ovine muscle suggest this therapy may cause damage to healthy tissues and bacterial retention. 103,104

The surgeon should remain cognizant that there may be extensive necrosis within the wound that involves muscle, fat, and skin and be prepared to debride as needed. The patient's condition may have progressed to involve the underlying bone or nearby joint. The surgical end result must be well-vascularized tissues with increased tissue oxygenation and removal of the physical barriers to wound healing by whatever closure technique selected.

Appropriate patient position in the operating room gives optimal exposure of the wound. The wound is cleaned and repaired in a position of maximal tension, whereby all other physical positions decrease tension on the suture lines. Thus, any size limitations are addressed intraoperatively and the possibility of postoperative dehiscence is minimized.

Progressing to the operating room for sharp debridement is delayed until all potential sources of infection have been addressed. Hence, the possibility of respiratory, urinary, or other site infections cross-contaminating the debrided pressure sore is minimized. The wound itself almost certainly contains a substantial bacterial load and necessitates debridement. Studies have demonstrated that freshly incised skin has less resistance to bacterial incursion, and measures should be taken to minimize seeding of the cleansed wound. Standard antiseptic skin prep and aseptic technique block external sources of infection such as the surgeon and operating room equipment. This is further enhanced by administering perioperative systemic antibiotics to prevent seeding of distant bacteria into the surgically cleansed wound. The wound can be cleansed with buffered Dakin's solution (sodium hypochlorite, 0.025% concentration), which is both bactericidal and nonhazardous to neutro-

phils and fibroblasts, although Silvadene, mafenide, and Betadine are frequently used.

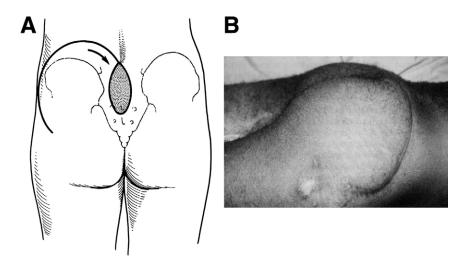
Ensuring complete pressure ulcer excision necessitates the removal of dead tissue to prevent incomplete excision of skin margins. Such an effort to clean these fastidious wounds would be marred by recurrence of infection and wound dehiscence. One technique to ensure completeness of debridement is the use of a dilute 1% solution of methylene blue, which stains the exposed tissues. This can be followed with a hydrogen peroxide lavage to remove excess dye. <sup>108</sup> Excision of all stained tissues visually guides the surgeon's blade. This is supplemented with the presence of fresh bleeding of tissues, although hemostasis must be achieved. Following skin and necrotic tissue excision is bone debridement and contouring performed with rongeurs and rasps.

A debrided wound is now ready for closure. Surgical closure of the cleansed pressure sore is best achieved using local rotation, fasciocutaneous, or musculocutaneous flaps. Skin grafting is an option for superficial ulceration, but the long-term stability is around 30%. Both animal and clinical studies confirm the superiority of musculocutaneous flaps over skin grafting of infected wounds. Paraplegic patients are susceptible to flap loss secondary to postoperative hematoma as a function of sympathetic tone loss and physiologic inability to vasoconstrict blood vessels 71,111

#### Sacrum

The supine, bedridden patient and the acutely injured spinal cord patient typify the victim of skin and tissue breakdown over the sacral prominence. Small sacral wounds can, theoretically, be excised and closed primarily. Such primary repairs place tension on the wound and a suture line directly over a pressure point. As a result, the wound may not heal, dehisce, or reoccur as the patient returns to the supine position postoperatively. Skin grafting is another possible option, but the nature of healed skin grafts is to provide skin coverage and not padding. This type of closure has the best results in ambulatory patients that are sensate and recovering from an acute illness or injury.

Sacral wound repair requires adequate site preparation. When performing the bone ostectomy/contouring, the surgeon should take care not to perforate the anterior sacral cortex over which lies the presacral venous plexus. Various flaps may be designed to cover the debrided sacral wound. The local buttock rotation flap is an excellent primary method for closure of the prepared sacral wound. The design requires an elliptical incision extending from the superior pole of the wound then superiorly



**FIG 17.** Sacral rotation flap. (With permission from Phillips LG, Robson MC. Pressure ulcerations. In: Jurkiewicz MJ, Krizek TJ, Mathes SJ, Ariyan S, editors. Plastic Surgery: Principles and Practice. Vol. 2. St Louis: CV Mosby; 1990; pp 1223-51.)

and laterally toward the posterior iliac crest and then inferiorly (Fig 17). Both the superior and inferior gluteal arteries supply this portion of tissue. Any lateral defect can be closed with a skin graft, and recurrence of the sacral pressure sore allows for readvancement of the gluteal flap.

The transverse lumbosacral flap is an arterial skin flap. This reliable flap is supplied by the lumbar perforating vessels and uninterrupted subdermal vascular plexus to the terminal portion. A large, transverse U-shaped flap is elevated so that the inferior portion can cover the wound defect (Fig 18). The created superior deficit is closed with a skin graft. This flap has less subcutaneous tissue to pad the sacrum than the buttock flap.

With both the local buttock rotation flap and the transverse lumbosacral flap, a deficit is created so that the primary wound can close the sacral wound. Closure of these iatrogenic, secondary wounds is performed with skin grafting. As discussed above, closure of the primary wound with skin grafting is suboptimal at best. However, these secondary wounds are not subjected to the pressure and stress compared with the sacral area and other pressure points and will heal.

Skin rotation flaps receive their blood supplies from deeper musculocutaneous blood vessels that perforate and branch upward. The last 30 years of plastic surgery has been devoted to the incorporation of muscle and skin along a vascular pedicle to ensure tissue viability, increase the arc of rotation, and gain length. Ger is attributed with early use of muscle flaps

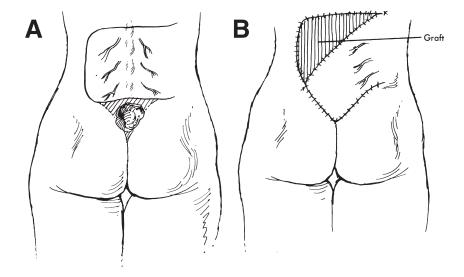


FIG 18. Transverse lumbosacral flap. (With permission from Phillips LG, Robson MC. Pressure ulcerations. In: Jurkiewicz MJ, Krizek TJ, Mathes SJ, Ariyan S, editors. Plastic Surgery: Principles and Practice. Vol. 2. St Louis: CV Mosby; 1990; pp 1223-51.)

to close leg ulcers and later pressure sores. 113,114 Muscle flaps were initially used to cover exposed bone, and then the muscle was covered with a skin graft. 115 Skin-bearing portions of muscle, termed musculocutaneous flaps, were attempted for pressure sore closure and demonstrated the viability of this closure method. 116

Sacral pressure sores can be closed using musculocutanous flaps when primary closure or a local rotation flap is not an option. The majority of these musculocutaneous flaps are based on some portion of the superior or inferior gluteal arteries. The musculocutaneous flap can be fashioned for V to Y closure. Gaining popularity is the use of fasciocutaneous flaps to close various wounds, including flaps based on the gluteal artery for sacral pressure sores. Porman and Maral demonstrated the reliability of the gluteal fasciocutaneous flap with V to Y closure for 15 sacral pressure sore patients without wound dehiscence or flap necrosis at a minimum follow-up time of 6 weeks.

#### Ischium

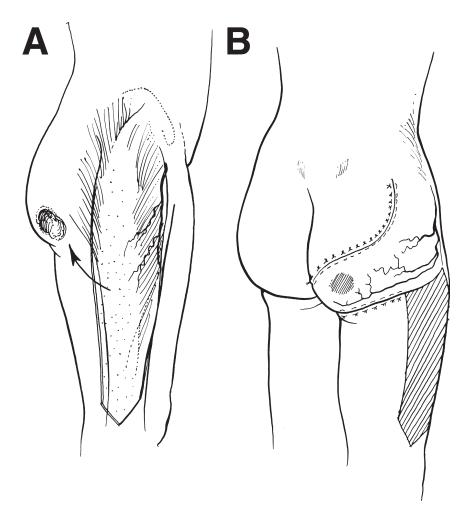
Ischial pressure sores are common to the wheelchair-bound seated patient. These wounds befuddled early attempts at nonoperative care and conservative closure with skin grafts. Conway and Griffith documented 100 ischial pressure sore patient care events whereby nonoperative

treatment resulted in a recurrence rate of 77% and total success rate of merely 18%. <sup>121</sup> Comparatively, split-thickness skin grafting yielded a similarly high recurrence rate and a total success rate of 11%. The major complicating factor for ischial pressure sore patients is the necessity for mobility and eventual return of the patient to the source of tissue breakdown, usually the wheelchair.

The ischium is a critical structure to pelvic stability in the seated position, and these pelvic bony prominences are subject to high pressures in the supine patient. The high recurrence rate with ischial pressure sores mandates surgical intervention. Recontouring the underlying bone before flap closure has been extensively investigated. Conway and Griffith reduced their ischial pressure sore recurrence rate from 38% to 3% via ischiectomy. Others have recommended prophylactic bilateral ischiectomy for unilateral disease. The result of bilateral ischiectomy is transferred pressure to the perineum where the patient is at increased risk for developing a urethral fistula, urethral diverticula, or perineal pressure sore, and is a practice which should not be followed.

Flap design depends on the size of the defect and previous closure methods for recurrent ischial pressure sores. Local random skin flaps are best used for small, shallow wounds. Other options include the tensor fascial lata flap, a reliable pedicle with a length advantage, although this flap may not provide adequate bulk for some patients <sup>128</sup> (Fig 19). The gluteal thigh flap is another option that provides bulk, reliability, and—interestingly—sensation to the ischial area. <sup>129</sup> Other described flaps include use of the hamstrings and total thigh flaps for multiple, recurrent pressure sores. <sup>130-132</sup>

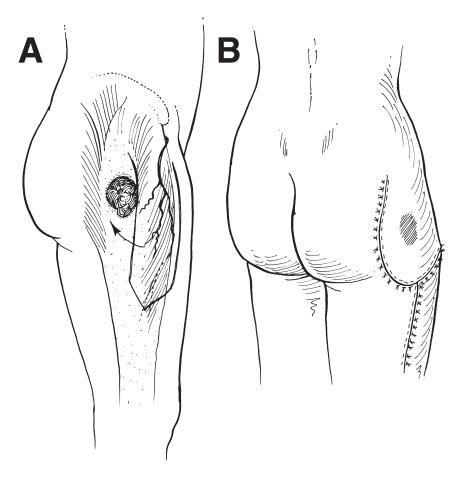
In a rather unique maneuver, Jones accomplished coverage and sensation for a T7 paraplegic patient with recurrent ischial pressure sores using a free medial gastrocnemius flap. Sensation was restored by anastomosis between a proximal intercostal nerve, sural nerve graft, and the tibial motor nerve branch of the gastrocnemius. Hallock has also reported success with a free medial gastrocnemius flap to close a recurrent ischial wound. If muscle is not desired, an inferior gluteal artery perforator flap can be fashioned. Homma and colleagues confirmed excellent ischial pressure sore repair in 10 paraplegic patients using a posteromedial thigh fasciocutaneous flap with no recurrence in 7 of the patients at 77 months postoperatively. The authors attribute their success of this flap to meticulous outlining of the musculocutaneous perforator, centering the flap over the artery, and preservation of the proximal fascia.



**FIG 19.** Tensor fascia lata musculocutaneous flap for ischial pressure sores. (With permission from Phillips LG, Robson MC. Pressure ulcerations. In: Jurkiewicz MJ, Krizek TJ, Mathes SJ, Ariyan S, editors. Plastic Surgery: Principles and Practice. Vol. 2. St Louis: CV Mosby; 1990; pp 1223-51.)

### Trochanter

Skin and tissue breakdown over the greater trochanter—the lateral prominence of the proximal femur—is most associated with prolonged lateral decubitus positioning and large patients in narrow wheelchairs. Conway and Griffith studied 82 patients with 122 trochanteric pressure sores. Conservative, nonsurgical therapy in this cohort worked in approximately 41% of cases, and skin grafting these wounds demonstrated a 33% long-term success rate. These tenacious wounds are like



**FIG 20.** Tensor fascia lata musculocutaneous flap for trochanteric pressure sores. (With permission from Phillips LG, Robson MC. Pressure ulcerations. In: Jurkiewicz MJ, Krizek TJ, Mathes SJ, Ariyan S, editors. Plastic Surgery: Principles and Practice. Vol. 2. St Louis: CV Mosby; 1990; pp 1223-51.)

ischial and sacral pressure sores. They require the typical approach for proper healing: excision of dead and necrotic tissue, debridement down to healthy and viable tissue, and proper flap coverage.

The primary flap used to cover debrided trochanter pressure sore wounds is the tensor fascia lata musculocutaneous flap (Fig 20). The tensor fascia lata is a workhorse flap applicable to both ischial, trochanteric, and sometimes sacral defects. In a small series by Josvay and colleagues, 8 of 9 attempts resulted in closure of trochanteric wounds using the tensor fascia lata flap. All of these wounds were closed primarily with the V to Y advancement technique. If closure of the

harvest site cannot be accomplished primarily, a skin graft is used. Other options include the use of muscle flaps without skin, such as the gluteus and vastus lateralis; coverage is then achieved with a skin graft over the muscle belly.

The main cause of pressure recurrence is the insensate nature of the wound site. The tensor fascia lata flap is the ideal method to close trochanteric pressure sores because of the lumbar innervation that provides sensation in those patients with low spinal cord injuries. Restoring sensation is a powerful preventative measure for pressure sore patients as they can determine themselves when to shift position and avoid tissue breakdown.

Dibbell and colleagues have used the tensor fascia lata flap with its lateral femoral cutaneous nerve for pressure sores in meningomyelocele patients. <sup>138</sup>

# Postoperative Care

Much of the care for the postoperative patient is the same for the closure of pressure sore wounds as other flaps. The suture edges are covered with a nonadherent mesh such as Adaptic. This is covered with absorptive gauze to remove serosanguineous drainage from the skin and prevent maceration. The gauze is changed as necessary to keep the skin clean. Drains are useful monitoring devices for excessive bleeding from underneath the flap; drains also help prevent flap loss from hematoma or seroma formation by controlled, low-pressure evacuation and help the flap adhere to the underlying wound bed.

The operating physician should monitor the patient's postoperative progress at regular intervals. However, the nursing staff is the crucial physician extender to keep an eye on the patient and report problems immediately. The patient should be moved every 2 hours to prevent pressure sore development at other sites, pressure necrosis of the flap, or wound dehiscence. Another option is the use of an air-fluidized bed, which extends the time between turning patients. Whatever the methodology, the wound should avoid excess moisture and fecal contamination. The majority of focus will be on the flap tissue, but other classic postoperative issues should not be ignored. Pain is controlled with appropriate analgesic medications such as morphine, fentanyl, or nonsteroidal anti-inflammatory drugs. For patients with neuropathic and refractory pain, Neurontin should be considered. 139 The patient may have nausea, ileus, and poor ventilatory effort from the general anesthetic. Feeding should begin as tolerated by the patient to enhance tissue healing. Scheduled nebulizer therapy and deep breathing exercises are useful for atelectasis. Muscle spasms not adequately treated preoperatively require immediate pharmacological therapy because this condition can compromise the flap and certainly challenges patient comfort. Valium and baclofen are reliable options to manage postoperative muscle spasms.<sup>140</sup>

The last goal of surgical intervention is to restore the patient's level of independence before the pressure sore developed. This requires at least 2 months of postoperative attention by the surgeon and a lifetime of care by the patient. After 1 to 2 weeks, the drains can be carefully removed if the serous fluid is clear and less than 30 mL per day. Beginning at 2 weeks, the wound edges are sufficiently healed to support the patient's sitting or lying on the flap for up to 15 minutes 3 times per day, gradually increasing by small increments in time. The exception to this is for ischial pressure sore repairs, which require a brief period of pressure relief every 10 minutes, even after healing is complete.

Several unresolved problems may prevent achievement of a healed, uncomplicated wound. Pain can be related to distress and depressive symptoms. <sup>141</sup> The patients must be compliant and motivated to heal their injury despite their altered body image. This requires good nutrition, rehabilitation, and strengthening of their deconditioned status, and continence maintenance if necessary. Comatose, spinal cord injured, and elderly patients are the most challenging to heal and require the highest level of nursing care. Each of these patients may not be able to sense, determine, or comply with the need to shift their body. Should recurrence of the wound develop, the patient, similar to other victims of chronic disease states, needs encouragement, support, and commitment from their caregivers.

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