Pressure Injuries (Pressure Ulcers) and Wound Care

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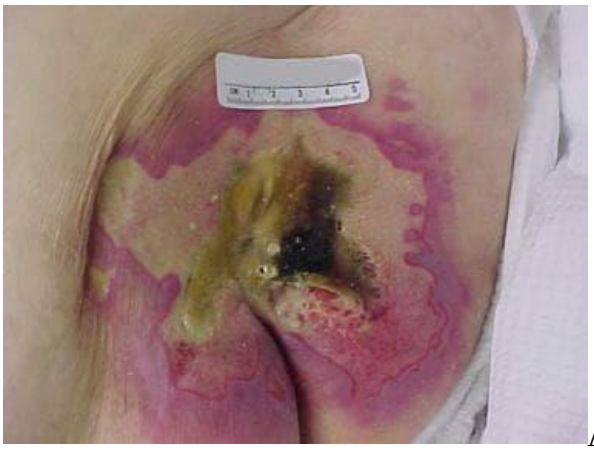
Overview

Practice Essentials

Although the terms decubitus ulcer, pressure sore, and pressure ulcer have often been used interchangeably, the National Pressure Injury Advisory Panel (NPIAP; formerly the National Pressure Ulcer Advisory Panel [NPUAP]) currently considers pressure injury the best term to use, given that open ulceration does not always occur.[1] According to the NPIAP, a pressure injury is localized damage to the skin and underlying soft tissue, usually over a bony prominence or related to a medical or other device. It can present as intact skin or an open ulcer and may be painful. It occurs as a result of intense or prolonged pressure or pressure in combination with shear.

See the image below.


Advanced sacral pressure ulcer shows effects of pressure, shearing, and moisture.

Signs and symptoms

The following important information should be obtained from the history:

- Overall physical and mental health, including life expectancy
- Previous hospitalizations, operations, or ulcerations
- Diet and recent weight changes
- Bowel habits and continence status
- Presence of spasticity or flexion contractures
- Medications and allergies to medications
- Tobacco, alcohol, and recreational drug use
- Place of residence and the support surface used in bed or while sitting
- level of independence, mobility, and ability to comprehend and cooperate with care
- Underlying social and financial support structure

- Presence of specific cultural, religious, or ethnic issues
- Presence of advanced directives, power of attorney, or specific preferences regarding care
- Information related to the current ulceration - Pain, foul odour or discharge, natural history of the present ulcer, and associated medical cause of the ulcer

A thorough physical examination is necessary to evaluate the patient's overall state of health, comorbidities, nutritional status, and mental status. After the general physical examination, attention should be turned to the wound.

For the purposes of workup and treatment, it is helpful to stage the pressure injury according to the system promulgated by the NPUAP,[2] as follows:

- Stage 1 pressure injury - Nonblanchable erythema of intact skin
- Stage 2 pressure injury - Partial-thickness skin loss with exposed dermis
- Stage 3 pressure injury - Full-thickness skin loss
- Stage 4 pressure injury - Full-thickness skin and tissue loss
- Unstageable pressure injury - Obscured full-thickness skin and tissue loss
- Deep pressure injury - Persistent nonblanchable deep red, maroon or purple discoloration

Complications of ulceration include the following:

- Malignant transformation
- Autonomic dysreflexia
- Osteomyelitis
- Pyarthrosis
- Sepsis
- Urethral fistula
- Amyloidosis
- Anemia

Diagnosis

Laboratory studies that may be helpful include the following:

- Complete blood count (CBC) with differential
- Erythrocyte sedimentation rate (ESR)
- Albumin and prealbumin
- Transferrin
- Serum protein

When indicated by the specific clinical situation, the following should be obtained:

- Urinalysis and culture in the presence of urinary incontinence
- Stool examination for faecal WBCs and *Clostridium difficile* toxin when pseudomembranous colitis may be the cause of faecal incontinence
- Blood cultures if bacteraemia or sepsis is suggested

Additional studies that may be considered include the following:

- Plain radiography

- Bone scan
- Magnetic resonance imaging
- Tissue or bone biopsy

Management

General principles of wound assessment and treatment are as follows:

- Wound care may be broadly divided into nonoperative and operative methods
- For stage 1 and 2 pressure injuries, wound care is usually conservative (i.e., nonoperative)
- For stage 3 and 4 lesions, surgical intervention (e.g., flap reconstruction) may be required, though some of these lesions must be treated conservatively because of coexisting medical problems [3]
- Approximately 70%-90% of pressure injuries are superficial and heal by second intention

Successful medical management of pressure injuries relies on the following key principles:

- Reduction of pressure
- Adequate debridement of necrotic and devitalized tissue
- Control of infection
- Meticulous wound care

If surgical reconstruction of a pressure injury is indicated, medical status must be optimized before reconstruction is attempted. General measures for optimizing medical status include the following:

- Control of spasticity
- Nutritional support as appropriate
- Cessation of smoking
- Adequate pain control
- Maintenance of adequate blood volume
- Correction of anaemia
- Maintenance of the cleanliness of the wound and surrounding intact skin
- Management of urinary or faecal incontinence as appropriate
- Management of bacterial contamination or infection

Additional nonsurgical treatment measures include the following:

- Pressure reduction - Repositioning and use of support surfaces
- Wound management - Debridement, cleansing agents, dressings, and antimicrobials
- Newer approaches still being studied - Growth factors (e.g., becaplermin), negative-pressure wound therapy, and electrotherapy

Surgical interventions that may be warranted include the following:

- Surgical debridement
- Diversion of the urinary or faecal stream
- Release of flexion contractures
- Wound closure
- Amputation

Options available for surgical management of pressure injuries are as follows:

- Direct closure (rarely usable for pressure injuries being considered for surgical treatment)
- Skin grafts
- Skin flaps
- Myocutaneous (musculocutaneous) flaps
- Free flaps

The choice of reconstruction approach depends on the location of the pressure injury (eg, ischial, sacral, or trochanteric).

Prevention, if achievable, is optimal. Prevention of pressure injuries has two main components:

- Identification of patients at risk
- Interventions designed to reduce the risk

Background

The terms decubitus ulcer (from Latin decumbere, “to lie down”), pressure sore, and pressure ulcer have often been used interchangeably in the medical community. However, as the name suggests, decubitus ulcer occurs at sites overlying bony structures that are prominent when a person is recumbent. Hence, it is not an accurate term for ulcers occurring in other positions, such as prolonged sitting (e.g., ischial tuberosity ulcer). Because the common denominator of all such ulcerations is pressure, pressure ulcer came to be considered the best term to use.

The National Pressure Ulcer Advisory Panel (NPUAP) was an independent non-profit organization formed in 1987 and dedicated to the prevention, management, treatment, and research of pressure ulcers. In April 2016, the NPUAP announced that it was changing its preferred terminology from pressure ulcer to pressure injury, on the grounds that the latter term better described this injury process in both intact and ulcerated skin.[1] In November 2019, the NPUAP changed its name to the National Pressure Injury Advisory Panel (NPIAP).

Currently, the NPIAP defines a pressure injury as localized damage to the skin and underlying soft tissue, usually over a bony prominence or related to a medical or other device.[1] Such injury can present either as intact skin or an open ulcer and may be painful. It results from intense or prolonged pressure or pressure combined with shear. The NPIAP also notes that the tolerance of soft tissue for pressure and shear may be affected by microclimate, nutrition, perfusion, comorbid conditions, and the condition of the soft tissue.

Pressure is exerted on the skin, soft tissue, muscle, and bone by the weight of an individual against a surface beneath. These pressures often exceed capillary filling pressure (~32 mm Hg). In patients with normal sensitivity, mobility, and mental faculty, pressure injuries do not occur. Feedback, conscious and unconscious, from the areas of compression leads them to change their body position, and these changes shift the pressure before any irreversible tissue damage develops. (See Pathophysiology and Etiology.)

Those who cannot avoid long-term uninterrupted pressure over bony prominences (e.g., persons who are elderly, have neurologic impairment, or are undergoing acute hospitalization[4]) are at increased risk for pressure injuries. They cannot protect themselves from the pressure unless they consciously

change position or are helped to do so. Even a highly conscientious patient with an extensive support group and unlimited financial resources may develop such injuries as a result of a brief lapse in avoidance of the ill effects of pressure.[5, 6]

Addressing the overall management of pressure injuries is now a prominent national healthcare issue. Despite current interest and advances in medicine, surgery, nursing care, and self-care education, pressure injuries remain a major cause of morbidity and mortality, and patients with pressure injuries are important users of medical resources.[7, 8, 9, 10, 11, 12, 13, 14]

Many factors are involved in the management of pressure injuries. Nursing plays a pivotal role in this challenging and complex process, using a multifaceted approach that includes skin care, pressure relief, and nutritional support. Prevention is the key to managing pressure injuries, and it begins with a complete medical and nursing history, a risk assessment, and skin examination when the patient is admitted.[9, 10, 15] (See Treatment.)

Factors that subject the tissue at risk to potential skin breakdown should receive particular attention. Patients should be kept clean and dry and should be repositioned frequently. For patients at risk, adequate pressure relief must be provided, along with adequate nutritional support.

For patients who develop pressure injuries, these preventive measures must be used in conjunction with the techniques of general wound care. Nonoperative wound care may involve simple topical therapy, as for pressure injuries with unbroken skin or superficial lesions with nondraining, noninfected granulation tissue. For draining necrotic or infected lesions, treatment also may include absorption agents, calcium alginate dressings, wound coverings, debridement, and antimicrobial therapy.

Other therapeutic modalities, such as whirlpool, physical therapy, and specialty beds, may also be added to the treatment regimen.

Research in the area of pressure injuries—specifically, in the characterization, prevention, and treatment of these lesions—is important for preventing secondary complications in persons with disabilities. As the standards of acute, posttraumatic, and rehabilitation care improve, the population of persons with lifelong functional impairments continues to grow. Consequently, the prevention of secondary complications has become an increasingly prominent concern.

To date, clinical studies of pressure injuries have been difficult to assess because they have often been qualitatively based on random observation and uncontrolled studies. To arrive at more reliable conclusions, more fundamental approaches to these injuries must be considered. Questions that might be asked include the following:

- What are the basic histologic, pathologic, and biochemical markers in an evolving pressure injury?
- Is it ethical to take a biopsy specimen of a human pressure injury for purposes of research?
- What are the multiple variables in the formation of pressure injuries in the human environment?

A monograph prepared by the Research Committee of the NPUAP (now the NPIAP) suggested the following research priorities[7] :

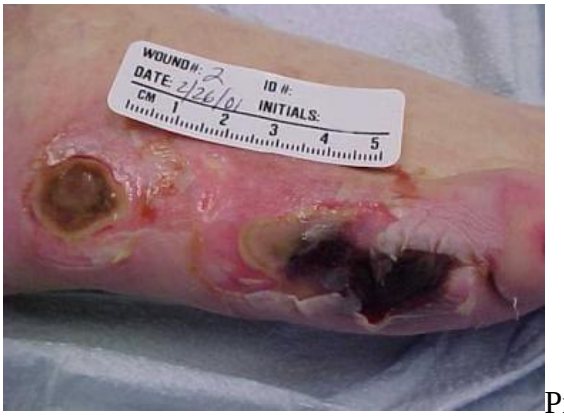
- Outcome-focused research

- Intervention and product efficacy studies
- Basic research related to staging of ulcers
- Refinement of risk assessment methods
- Risk-based, multi-interventional trials

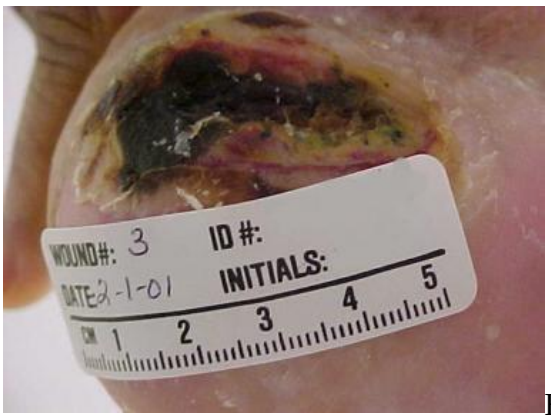
Additional issues requiring investigation included cost issues, ethical decision making, guideline dissemination, public policy, and national outcome evaluations. Methodologic issues, such as research design, study population, and control group use, also were considered to warrant further investigation.

Anatomy

Pressure injuries are typically described in terms of location and depth of involvement. The hip and buttock regions account for up to 70% of all pressure injuries, with ischial tuberosity, trochanteric, and sacral locations being most common.[16] The lower extremities account for an additional 15-25% of all pressure injuries, with malleolar, heel, patellar, and pretibial locations being most common (see the images below).



Pressure ulcers of lateral aspect of right foot.



Heel pressure ulcer.

The remaining small percentage of pressure injuries may occur in any location that experiences long periods of uninterrupted pressure.[16] The nose, chin, forehead, occiput, chest, back, and elbow are among the more common of the infrequent sites for pressure injuries. No surface of the body can be considered immune to the effects of pressure.

Pressure injuries can involve different levels of tissue. Muscle has been proved to be most susceptible to pressure. However, Daniel and Faibisoff found that muscle rarely was interposed between bone and skin in normal weightbearing positions in cadaver and clinical dissections.[17]

Pathophysiology

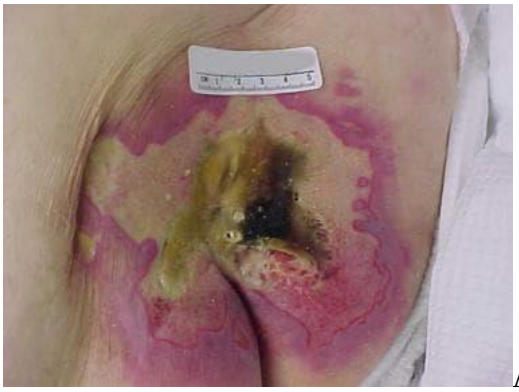
In 1873, Sir James Paget described the production of pressure ulcers remarkably well, and his description is still quite accurate today.[18] Many factors contribute to the development of pressure injuries, but pressure leading to ischemia and necrosis is the final common pathway.

In this view, pressure injuries result from constant pressure sufficient to impair local blood flow to soft tissue for an extended period. This external pressure must be greater than the arterial capillary pressure (32 mm Hg) to impair inflow and greater than the venous capillary closing pressure (8-12 mm Hg) to impede the return of flow for an extended time.

Tissues are capable withstanding enormous pressures for brief periods, but prolonged exposure to pressures just slightly above capillary filling pressure initiates a downward spiral toward tissue necrosis and ulceration.[19, 20] The inciting event is compression of the tissues against an external object such as a mattress, wheelchair pad, bed rail, or other surface.

Lindan et al documented ranges of pressure applied to various anatomic points in certain positions.[21] The points of highest pressure with the patient supine included the sacrum, heel, and occiput (40-60 mm Hg). With the patient prone, the chest and knees absorbed the highest pressure (50 mm Hg). When the patient is sitting, the ischial tuberosities were under the most pressure (100 mm Hg). Obviously, these pressures are greater than the end capillary pressure, which is why these are the areas where pressure injuries are most common.

Shear forces and friction aggravate the effects of pressure and are important components of the mechanism of injury (see the image below).[22] Maceration may occur in a patient who has incontinence, predisposing the skin to injury. Pressure, shear forces, and friction cause microcirculatory occlusion and consequent ischemia, which leads to inflammation and tissue anoxia. Tissue anoxia leads to cell death, necrosis, and ulceration.


Advanced sacral pressure ulcer shows effects of pressure, shearing, and moisture.

Of the various tissues at risk for death due to pressure, muscle tissue is damaged first, before skin and subcutaneous tissue, probably because of its increased need for oxygen and higher metabolic requirements.[23, 24] Irreversible changes may occur during as little as 2 hours of uninterrupted pressure. Skin can withstand ischemia from direct pressure for up to 12 hours. By the time ulceration is present through the skin level, significant damage of underlying muscle may already have occurred, making the overall shape of the ulcer an inverted cone.

Reperfusion has been suggested as a cause of additional damage to the ulcerated area, inducing an ulcer to enlarge or become more chronic—as, for example, when a paraplegic or quadriplegic patient is turned from one side to the other in an attempt to combat prolonged pressure on a given side. The exact mechanism of ischemia-reperfusion injury is yet to be fully understood. Continued production of inflammatory mediators and reactive oxygen species during ischemia-reperfusion may contribute to the chronicity of pressure ulcers.

Etiology

Impaired mobility is probably the most common reason why patients are exposed to the prolonged uninterrupted pressure that causes pressure injuries. This situation may be present in patients who are neurologically impaired, heavily sedated or anesthetized, restrained, demented, or recovering from a traumatic injury. These patients cannot alter their position far enough or often enough to relieve the pressure. Prolonged immobility may lead to muscle and soft tissue atrophy, decreasing the bulk over which bony prominences are supported.

Contractures and spasticity often contribute to ulcer formation by repeatedly exposing tissues to trauma through flexion of a joint. Contractures rigidly hold a joint in flexion, whereas spasticity subjects tissues to repeated friction and shear forces. Skin breakdown and pressure injuries may frequently be found under and between toes and on the palm of the hand.

Inability to perceive pain, whether from neurologic impairment or from medication, contributes to pressure injuries by removing one of the most important stimuli for repositioning and pressure relief. Conversely, pain from surgical incisions, fracture sites, or other sources may make the patient unwilling or unable to change position.

The quality of the skin also influences whether pressure leads to ulceration. Paralysis, insensibility, and aging lead to atrophy of the skin with thinning of this protective barrier. A decrease in epidermal turnover, a flattening of the dermal-epidermal junction, and a loss of vascularity occur with advanced age.

In addition, the skin becomes more susceptible to minor traumatic forces, such as the friction and shear forces typically exerted during the moving of a patient. Trauma that causes deep ithelialization or skin tears removes the barrier to bacterial contamination and leads to transdermal water loss, creating maceration and causing the skin to adhere to clothing and bedding.

Incontinence or the presence of a fistula contributes to ulceration in several ways. These conditions cause the skin to be continually moist, thus leading to maceration. In addition, frequent soiling has the effect of regularly introducing bacteria into an open wound.

Bacterial contamination, though not truly an etiologic factor, must be considered in the treatment of pressure injuries, in that it can delay or prevent wound healing. These lesions are warm, moist reservoirs for bacterial overgrowth, where antibiotic resistance may develop. A pressure injury may progress from simple contamination (as in any open wound) to gross infection (indicating bacterial tissue invasion). This may lead to uncommon but life-threatening complications (e.g., bacteremia, sepsis, myonecrosis, gangrene, or necrotizing fasciitis).

Malnutrition, hypoproteinaemia, and anaemia reflect the overall status of the patient and can contribute to tissue vulnerability to trauma as well as cause delayed wound healing. Poor nutritional

status certainly contributes to the chronicity often seen in these lesions and inhibits the ability of the immune system to prevent infections. Anaemia indicates poor oxygen-carrying capacity of the blood. Vascular disease and hypovolemia also may impair blood flow to the region of ulceration.

In patients with normal sensitivity, mobility, and mental faculty, pressure injuries are unlikely. Conscious or unconscious feedback from the areas of compression leads them to change position, thereby shifting the pressure from one area to another long before any irreversible ischemic damage occurs. In individuals who cannot avoid long periods of uninterrupted pressure, the risk of necrosis and ulceration is increased. These individuals cannot protect themselves from the pressure unless they consciously change position or are helped to do so.

Epidemiology

United States statistics

Pressure injuries are common among patients hospitalized in acute- and chronic-care facilities. It has been estimated that about 1 million pressure injuries occur in the United States; however, definitive information on the epidemiology and natural history of this condition is still limited. Unfortunately, studies to date have been encumbered by methodologic issues and variability in describing the lesions.[7, 25]

Reported incidences of pressure injuries in hospitalized patients range from 2.7% to 29%, and reported prevalence in hospitalized patients range from 3.5% to 69%.[26, 27, 28, 29, 30] Patients in critical care units have an increased risk of pressure injuries, as evidenced by a 33% incidence and a 41% prevalence.[31, 32]

The fifth National Pressure Ulcer Prevalence Survey, conducted in 1999 among patients in acute care hospitals, showed an overall prevalence of 14.8%, with 7.1% of ulcers having occurred during that hospital visit.[33] Of the various hospital settings, intensive care units (ICUs) had the highest prevalence, at 21.5%. The largest single age group of patients with pressure injuries consisted of patients aged 71-80 years (29%).

Elderly patients admitted to acute care hospitals for nonelective orthopaedic procedures are at even greater risk for pressure injuries than other hospitalized patients are, with a 66% incidence.[34, 35] In a study of 658 patients aged 65 years or older who underwent surgery for hip fracture, Baumgarten et al found that 36.1% developed an acquired pressure injury within 32 days after hospital admission.[36]

In nursing homes, the prevalence of pressure injuries is 2.6-24%.[25, 37] ; the incidence is 25% in residents admitted from an acute care hospital.[37] Patients with pre-existing pressure injuries show a 26% incidence of additional pressure injury formation over a 6-month period. The incidence in chronic care hospitals is reported to be 10.8%,[38] whereas 33% of those admitted to a chronic care hospital have pressure injuries.[39] Long-term follow-up demonstrates that most ulcers heal within 1 year.[25, 40]

Among patients with neurologic impairments, pressure injuries occur with an incidence of 7-8% annually,[41] with a lifetime risk estimated to be 25-85%.[42] Moreover, pressure injuries are listed as the direct cause of death in 7-8% of all individuals with paraplegia; these individuals also have the

highest recurrence rate (80%).[43] In persons with spinal cord injury (SCI) and associated comorbidity, the incidence of pressure injuries is in the range of 25-66%.[44, 45, 46, 47]

A study of the prevalence of pressure injuries in community residents with SCI demonstrated that those with higher-level SCI lesions carry a greater risk of developing pressure injuries than those with lower-level lesions do.[44] Of 100 patients with pressure injuries, 33 had injuries that were classified as stage 2 or greater. Black patients had more severe injuries than other racial groups did.

Some authors speculate that detecting erythema can be more difficult with skin that has darker pigmentation.[48] Because prolonged nonblanching erythema is typically an early warning sign of pressure injury risk and development, difficulty in detecting erythema can result in failure to recognize grade I pressure injuries.

International statistics

In a study from Germany that reviewed the prevalence of pressure injuries in more than 18,000 patients residing in long-term care facilities, the prevalence was found to have decreased from 12.5% in 2002 to 5% in 2008.[49] This decrease is thought to be due to more effective management strategies and better prevention.

Age-related demographics

The prevalence of pressure injuries appears to have a bimodal age distribution. A small peak occurs during the third decade of life, reflecting ulceration in those with traumatic neurologic injury. Immobility and lack of sensation make these patients susceptible to developing pressure injuries. Treatment of these lesions in this patient population represents a financial challenge, with one hospital reporting an average cost of $78,000 for each admission of a patient with a pressure injury.

As patients move from the age category of 40-58 years to the age category of 75 years or older, a larger increase in the incidence of pressure injuries occurs.[50] Two thirds of pressure injuries occur in patients older than 70 years.[38] As elderly individuals become the fastest-growing segment of the population, with an estimated 1.5 million people living in extended-care facilities, the problem of pressure injuries will have an even more profound influence on the American economy.[51]

Sex-related demographics

Most younger individuals suffering from pressure injuries are males. The higher incidence in males reflects the greater number of men suffering traumatic SCIs. In the older population, most patients with pressure injuries are women, as a consequence of their survival advantage over men.

Race-related demographics

A study by Howard and Taylor found the incidence of pressure injuries in nursing home residents in the south-eastern United States to be higher in black patients than in white ones.[52] The authors examined data from 113,869 nursing home residents, none of whom had pressure injuries at nursing home admission. They determined that 4.7% of black residents developed postadmission ulcerations, compared with 3.4% of white residents.

In addition, the racial differences in pressure injury incidence displayed a sex predilection based on patient characteristics.[52] The variation in incidence between black and white males was noted in residents who were dependent in mobility, whereas the difference in incidence between black and white females was noted in residents who were bedfast and living in nursing homes with fewer than 200 beds.

Prognosis

Pressure injuries are listed as the direct cause of death in 7-8% of all patients with paraplegia.[53, 41] As many as one third of hospitalized patients with pressure injuries die during their hospitalization. More than half of those who develop a pressure injury in the hospital will die within the next 12 months. As a rule, these patients die of their primary disease process rather than of pressure ulceration, but the pressure injury may be a contributing factor in some instances.

Each year, approximately 60,000 people die of complications of pressure injuries.[54] Individuals with pressure ulcers have a 4.5-times greater risk of death than persons with the same risk factors but without pressure injuries.[8] A secondary complication, wound-related bacteremia, can increase the risk of mortality to 55%.[54, 55, 56, 57]

The most common causes of fatality for patients with chronic pressure injuries are renal failure and amyloidosis. In general, mortality is higher for patients who develop a new pressure injury and in whom the injury fails to heal.

Infection is the most common major complication of pressure injuries. The offending pathologic organisms can be either anaerobic or aerobic. Aerobic pathogens commonly are present in all pressure injuries,[58] whereas anaerobes tend to be present more often in larger wounds (65% in grade 3 and above).[59]

The organisms most commonly isolated from pressure ulcers are as follows:

- *Proteus mirabilis*
- Group D streptococci
- *Escherichia coli*
- *Staphylococcus*
- *Pseudomonas*
- *Corynebacterium*

Patients with bacteraemia are more likely to have Bacteroides species in their pressure injuries.[59] These wounds need not be cultured routinely unless systemic signs of infection are present (eg, malodorous drainage, leucocytosis, fever, hypotension, increased heart rate, changes in mental status).

Clinical alertness is vital because the signs commonly associated with impeding or fulminating infection are frequently absent in elderly or immunocompromised patients. In geriatric patients with pressure injuries, bacteraemia is reported to occur at a rate of 3.5 per 10,000 hospital discharges.[8]

In view of the high mortality in this population (nearly 50%),[56] it is important that antibiotic treatment of wound infection or secondary bacteraemia provide the appropriate spectrum of coverage specific to the offending organisms. Because indiscriminate use of antibiotics leads to resistant

organisms and because the specific drugs of choice and antimicrobial agents change rapidly, management of these complex problems may be facilitated by consulting an infectious disease specialist.

Sepsis also can occur secondary to osteomyelitis, which has been reported to occur in 26% of nonhealing ulcers.[8] A prospective study demonstrated that osteomyelitis was associated with nonhealing grade 4 pressure injuries in 86% of the study population.[60, 61] This study utilized three-phase technetium methyl diphosphate radionuclide flow to detect early osteomyelitis.

Various tests can be used to diagnose osteomyelitis in patients with pressure injuries. Plain radiographs have a sensitivity of 78% and a specificity of 50%, but radiographic findings often are not present in the early stages of infection. Bone scans are more sensitive, but their specificity is low (50%). Bone biopsy has the highest specificity (96%) and sensitivity (73%).[60, 61]

A combination of diagnostic tests (e.g., white blood cell [WBC] count, erythrocyte sedimentation rate [ESR], and plain radiography) provides a sensitivity of 89% and a specificity of 88%. If all three test results are positive, the positive predictive value of this combination is 69%. If all three test results are negative, the negative predictive value is 96%.[60, 61]

Osteomyelitis should be considered whenever an ulcer does not heal, especially if the ulcer is over a bony prominence. Clinicians also should rule out other conditions associated with nonhealing ulcers, such as heterotopic calcification or ossification. Most findings indicate that antibiotic treatment for osteomyelitis should last 6-8 weeks. Surgery is needed for some cases of chronic osteomyelitis.[45]

Systemic amyloidosis can result from chronic suppurative pressure injuries. Additional complications of pressure injuries include spreading cellulitis, a sinus tract abscess, septic arthritis, squamous cell carcinoma in the ulcer, a periurethral fistula, and heterotopic ossification. Because some of the secondary complications of pressure injuries can preclude wound healing, they should be aggressively prevented and treated.[62] Complications may include infection, pain, depression, and even death.

Patient Education

Patients and their support system must realize that it is their responsibility to avoid recurrent and new ulceration and that this is a lifelong process.[63] Education on the proper avoidance of pressure should begin in the hospital and continue into the home.

For patient education resources, see the Skin, Hair, and Nails Center and Diabetes Center, as well as Wound Care and Diabetic Foot Care.

Presentation

History

The clinical presentation of pressure injuries (pressure ulcers) can be deceiving to the inexperienced observer. Soft tissue, muscle, and skin resist pressure to differing degrees. Generally, muscle is the least resistant and will become necrotic before skin breaks down. Also, pressure is not equally distributed from the bony surface to the overlying skin; it is greatest at the bony prominence,

decreasing gradually toward the periphery. A small area of skin breakdown may represent only the tip of the iceberg, with a large cavity and extensive undermining of skin edges beneath.

In the initial evaluation of a patient with pressure injury, the following important information should be obtained from the history:

- Overall physical and mental health, including life expectancy
- Previous hospitalizations, operations, or ulcerations
- Diet and recent weight changes
- Bowel habits and continence status
- Presence of spasticity or flexion contractures
- Medications and allergies to medications
- Tobacco, alcohol, and recreational drug use
- Place of residence and the support surface used in bed or while sitting
- level of independence, mobility, and ability to comprehend and cooperate with care
- Underlying social and financial support structure
- Presence of specific cultural, religious, or ethnic issues
- Presence of advanced directives, power of attorney, or specific preferences regarding care

Information related to the current pressure injury should also be obtained, particularly with regard to the following:

- Pain - Although pain may be present at the injury site, it is more commonly absent because the patient either is paraplegic is in critical condition and unable to acknowledge pain
- Foul odour or discharge - This could be a sign of a more serious infection at the injury site
- Natural history of the present pressure injury - This would include the length of time the injury has been present, the circumstances under which the ulcer developed, and any local treatments currently or previously employed
- Associated medical cause for the injury (e.g., paraplegia, quadriplegia, spina bifida, immobilization in hospital, or multiple sclerosis)

A complete review of systems, including the presence of fevers, night sweats, rigors, weight loss, weakness, or loss of appetite, should be carried out.

Physical Examination

A thorough physical examination is necessary to evaluate the patient's overall state of health, comorbidities, nutritional status, and mental status. The patient's level of comprehension and extent of cooperation dictate the intensity of nursing care that will be required. The presence of contractures or spasticity is important to note and may help identify additional areas at risk for pressure ulceration.

After the general physical examination, attention should be turned to the pressure injury. Adequate examination of the wound may necessitate the administration of intravenous (IV) or oral pain medications to ensure patient comfort. Chronic pain may be present among these patients and may be exacerbated by examination ulcer.

Many classification schemes have been developed to define the severity of pressure ulcers.[64] For a considerable period, the most widely accepted approach was that of Shea, which was modified and

subsequently refined by the National Pressure Ulcer Advisory Panel (NPUAP).[65] In April 2016, the NPUAP (now known as the National Pressure Injury Advisory Panel [NPIAP] since November 2019) announced an updated version of its staging system, along with a change in preferred terminology from pressure ulcer to pressure injury.[1, 2]

The NPIAP system consists of four main stages of pressure injury but is not intended to imply that all pressure injuries follow a standard progression from stage 1 to stage 4 or that healing pressure injuries follow a standard regression from stage 4 to stage 1 to a healed wound. Rather, the system is designed to describe the degree of tissue damage observed at a specific time of examination and is meant to facilitate communication among the various disciplines involved in the study and care of patients with these lesions.

The categories specified in the current NPIAP staging system are as follows[2] :

- Stage 1 pressure injury - Intact skin with a localized area of nonblanchable erythema, which may appear differently in darkly pigmented skin; presence of blanchable erythema or changes in sensation, temperature, or firmness may precede visual changes; colour changes do not include purple or maroon discoloration, which may indicate deep tissue pressure injury
- Stage 2 pressure injury - Partial-thickness skin loss with exposed dermis; the wound bed is viable, pink or red, moist, and may also present as an intact or ruptured serum-filled blister; adipose (fat) and deeper tissues are not visible, and granulation tissue, slough and eschar are not present; these injuries commonly result from adverse microclimate and shear in the skin over the pelvis and shear in the heel
- Stage 3 pressure injury - Full-thickness skin loss, in which adipose (fat) is visible in the ulcer and granulation tissue and epibole (rolled wound edges) are often present; slough or eschar may be visible; the depth of tissue damage varies by anatomic location; areas of significant adiposity can develop deep wounds; undermining and tunnelling may occur; fascia, muscle, tendon, ligament, cartilage, and bone are not exposed
- Stage 4 pressure injury - Full-thickness skin and tissue loss with exposed or directly palpable fascia, muscle, tendon, ligament, cartilage or bone in the ulcer; slough or eschar may be visible; epibole (rolled edges), undermining, and tunnelling often occur; depth varies by anatomic location
- Unstageable pressure injury - Full-thickness skin and tissue loss in which the extent of tissue damage within the ulcer cannot be confirmed because it is obscured by slough or eschar; if slough or eschar is removed, a stage 3 or 4 pressure injury will be revealed
- Deep tissue pressure injury - Intact or nonintact skin with localized area of persistent nonblanchable deep red, maroon, purple discoloration or epidermal separation revealing a dark wound bed or blood-filled blister; pain and temperature change often precede skin colour changes; discoloration may appear differently in darkly pigmented skin; the injury results from intense and/or prolonged pressure and shear forces at the bone-muscle interface

Such staging is only a small part of the initial assessment. The injury location, the size of the skin opening (if present), and the presence of any surrounding maceration or induration must be accurately recorded. The presence of multiple pressure injuries prompts a search for interconnecting tracts with overlying skin bridging that may not be readily apparent. The presence or absence of foul odours, wound drainage, and soiling from urinary or faecal incontinence provides information about bacterial contamination and the need for debridement or diversionary procedures.

Complications

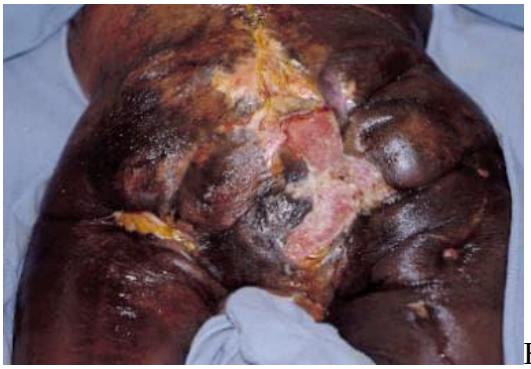
Complications fall into one of two categories: complications of chronic pressure injury (see below) and complications of reconstruction (see Treatment).

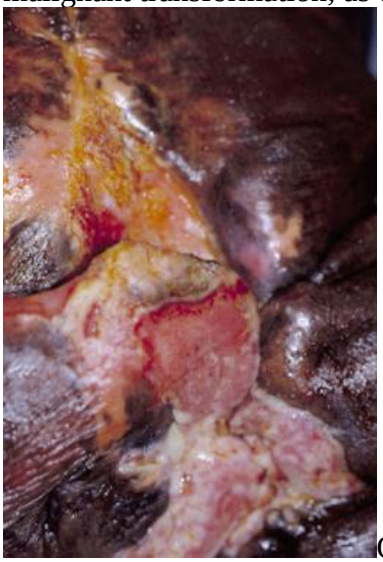
Complications of chronic injury include the following:

- Malignant transformation
- Autonomic dysreflexia
- Osteomyelitis
- Pyarthrosis
- Sepsis
- Urethral fistula
- Amyloidosis
- Anemia

Malignant transformation

The most serious complication of chronic ulceration is malignant transformation or degeneration (see the images below), also referred to as Marjolin ulceration. Although Marjolin initially described malignant transformation of a chronic scar from a burn wound, the term Marjolin ulcer has been commonly applied to the malignant transformation of any chronic wound, including pressure injuries, osteomyelitis, venous stasis ulcers, urethral fistulas, anal fistulas, and other traumatic wounds.[66]


Heaps of verrucous white tissue around the ulcer suggest malignant transformation, as observed with Marjolin ulcers.


Close-up view of area with heaps of verrucous white tissue around the ulcer, the presence of which suggests malignant transformation (as observed with Marjolin ulcers).

Histologically, this malignant transformation is a well-differentiated squamous cell carcinoma[67] ; however, its behaviour is very aggressive in pressure injuries, considerably more so than in burns or osteomyelitis.[68, 66] There is a high likelihood of nodal metastasis at the time of diagnosis. Any long-standing, nonhealing wound should alert the examiner to the need for biopsy.

Marjolin ulcers arising from burns or osteomyelitis have been treated with wide local excision, amputation, and lymph node dissection. Because pressure injury carcinoma is substantially more aggressive, more radical treatment (eg, hemicorporectomy and regional node dissection) is required if a cure is to be effected.[68, 66]

The actual rate for malignant transformation of a pressure injury is not known but can be assumed to be low, in that only 18 cases have been described in the literature to date. Although apparently rare, pressure ulcer carcinoma is highly lethal: 12 of the 18 known patients died within 2 years.

Autonomic dysreflexia

Autonomic dysreflexia is a disordered autonomic response to specific stimuli. It includes sweating and flushing proximal to the injury, nasal congestion, headache, intermittent hypertension, piloerection, and brady tachycardia. Patients with midthoracic spinal cord lesions are most prone to this response. When autonomic dysreflexia is suggested, the patient is first positioned with the head up and monitored for changes in heart rate and blood pressure. Then, the precipitating stimulus must be removed.

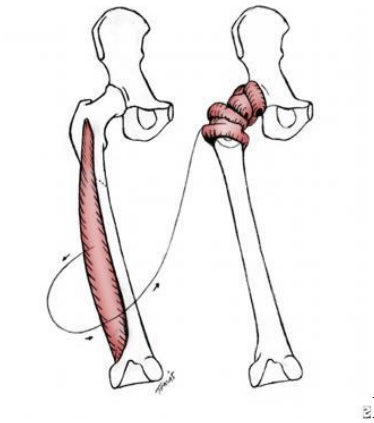
The most common precipitating cause of autonomic dysreflexia is bladder distention, which is treated by inserting a Foley catheter or irrigating an already placed Foley catheter to remove blockage. Rectal examination to evaluate for faecal impaction should be considered. Nifedipine, hydralazine, or topical nitro-glycerine may be administered to stabilize blood pressure. If autonomic dysreflexia does not respond to these measures, spinal anaesthesia may be required.

Osteomyelitis

Foremost in the treatment of osteomyelitis is the removal of all nonviable bone, down to bone that bleeds bright red.[69] In the reconstruction of reconstructing pressure injuries associated with osteomyelitis, it is important to use bone that is in the base flaps and has a muscle component. The muscle is placed over this bone after appropriate bone debridement. The flap reconstruction can be performed at the same time as the bone debridement. A 6-week course of IV antibiotics is then administered.

Pyarthrosis

Pyarthrosis of the hip joint can occur with communication of ischial or trochanteric ulcers. Often, the femoral head contains osteomyelitis, which necessitates its removal. The Girdlestone arthroplasty procedure has been employed in this situation (i.e., hip pyarthrosis), including removal of the femoral head and reconstruction of this space with the vastus lateralis muscle flap (see the image below).[70]

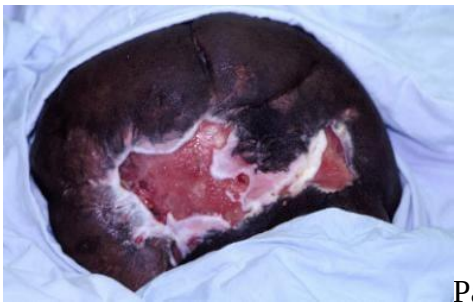
 Illustrated is Girdlestone arthroplasty for femoral head osteomyelitis pyarthrosis of hip joint. Femoral head is removed, and hip joint space is reconstructed with vastus lateralis muscle flap.

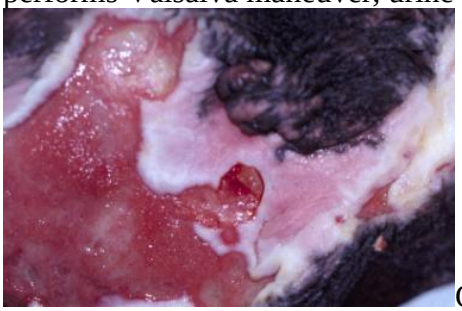
Sepsis

Pressure injuries do not cause sepsis. In patients who present with sepsis and pressure injuries, the sepsis is usually caused by a urinary tract infection. These wounds are almost always open to drain and therefore do not constitute debridement emergencies. Only on rare occasions are these injuries entirely occluded by a thick leathery eschar that prevents open drainage. In these cases, debridement is required to facilitate drainage and prevent systemic infection. In general, patients do not die of pressure injuries, but they can die with them.

Urethral fistula

Pressure injuries can also erode into the urethra (see the images below). Treatment of this complication (ie, urethral fistula) involves urinary diversion. Pressure injury reconstruction can be considered once the fistula has healed.

 Patient has urethral fistula within his pressure ulcer. When he performs Valsalva maneuver, urine leaks through this opening.

 Close-up view in patient who has urethral fistula within his pressure ulcer. When he performs Valsalva maneuver, urine leaks through this opening.

Workup

Laboratory Studies

A complete blood count (CBC) with differential may show an elevated white blood cell (WBC) count indicative of inflammation or invasive infection. The erythrocyte sedimentation rate (ESR) should be determined. An ESR higher than 120 mm/hr and a WBC count greater than 15,000/µL suggest osteomyelitis.

Nutritional parameters should be evaluated to assess adequate nutritional stores needed for adequate wound healing. Useful tests include the following:

- Albumin level - This should be optimized to at least 3.5 g/mL before flap reconstruction
- Prealbumin level
- Transferrin level
- Serum protein level

When indicated by the specific clinical situation, the following laboratory studies should be obtained:

- Urinalysis and culture in the presence of urinary incontinence
- Stool examination for faecal WBCs and *Clostridium difficile* toxin when pseudomembranous colitis may be the cause of faecal incontinence
- Blood cultures if bacteraemia or sepsis is suggested

These patients often have anaemia of chronic disease, suggested by a low mean corpuscular volume, and can be considered for a transfusion in order to achieve a preoperative haemoglobin level higher than 12 g/dL.

Imaging Studies

A diagnosis of underlying osteomyelitis can be evaluated first with plain films. Osteomyelitis may also be suggested by positive bone scan findings. A negative bone scan finding generally excludes osteomyelitis; however, patients with an open wound, such as a pressure injury, can often have a falsely positive bone scan. A positive bone scan finding can be evaluated further by means of magnetic resonance imaging (MRI) or bone biopsy (see below).

Biopsy

A tissue biopsy should be performed for wounds that do not demonstrate clinical improvement despite adequate care and for wounds in which tissue invasion by bacteria is suggested. This allows quantification and identification of bacterial species and their antibiotic susceptibilities. Biopsy also enables the clinician to distinguish between simple contamination and tissue invasion, an important distinction that is not revealed by the common practice of swabbing the wound surface for culture.

Tissue biopsy of chronic wounds is indicated to rule out the presence of an underlying malignancy (ie, Marjolin ulceration). Whenever a chronic pressure injury has been stable for months or years but has recently deteriorated, a biopsy should be performed. Scar carcinoma is uncommon and typically occurs in wounds that have been open for many years.

Bone biopsy is the criterion standard for the diagnosis of osteomyelitis within a pressure injury.[71] It should be considered in patients with an elevated ESR, an elevated WBC count, and or abnormal pelvic films suggestive of osteomyelitis, as well as in cases of stage 4 pressure injury with exposed bone. If osteomyelitis is confirmed, treatment with a prolonged course of antibiotic therapy may be indicated.

Histologic Findings

A concept that has been developed with regard to wound healing has to do with the presence of bacterial biofilms within a wound. Biofilms are structured communities of bacteria that may exist on a wound surface. A microscopic analysis of chronic wound specimens revealed the presence of densely aggregated bacterial colonies, often within their own extracellular matrix; however, these microscopic findings were not seen in acute wounds.[72]

Multiple in vivo studies have shown that wound healing is delayed when these biofilms are present in the wound.[73, 74] The biofilms seem to protect the underlying bacteria and provide resistance to antibiotic treatment and the body's own immune system. Discussions among expert panels on wound healing have concluded that the most effective means of managing biofilms within wounds is to remove the biofilm; however, optimal methods of removal have yet to be clearly defined.[75]

Treatment

Approach Considerations

Once a pressure injury (pressure ulcer) has developed, immediate treatment is required.[10] Commonly used treatments over the years have included innovative mattresses, ointments, creams, solutions, dressings, ultrasonography, ultraviolet heat lamps, sugar, and surgery. In choosing a treatment strategy, consideration should be given to the stage of the wound and the purpose of the treatment (e.g., protection, moisture, or removal of necrotic tissue). An algorithm for assessment and treatment is available.[57, 76, 77]

General principles of wound assessment and treatment are as follows:

- Wound care may be broadly divided into nonoperative and operative methods
- For stage 1 and 2 pressure injuries, wound care is usually conservative (ie, nonoperative)
- For stage 3 and 4 lesions, surgical intervention (eg, flap reconstruction) may be required, though some of these lesions must be treated conservatively because of coexisting medical problems [3]
- Approximately 70-90% of pressure injuries are superficial and heal by second intention

With thorough and comprehensive medical management, many pressure injuries may heal completely without the need for surgical intervention. Successful medical management of pressure ulcers relies on the following key principles:

- Reduction of pressure
- Adequate debridement of necrotic and devitalized tissue
- Control of infection
- Meticulous wound care

If surgical reconstruction of a pressure injury is indicated, it cannot be emphasized too strongly that medical management must be optimized before reconstruction is attempted; otherwise, reconstruction is doomed to failure. That is, spasticity must be controlled, nutritional status must be optimized, and the wound must be clean and free of infection. If there is significant faecal soiling into the injury, diverting colostomy should be considered before reconstruction. If there is a urethral fistula, it should be diverted and healed before reconstruction.

Wound reconstruction can be considered once the bacterial load has been sufficiently minimized to reduce the risk of infectious complications. Furthermore, the patient's social situation and nutritional status must be optimized (albumin level >3.5 g/mL) to reduce the risk of an adverse outcome.

Because the complication rate after pressure injury reconstruction can be extremely high, patients who are poor surgical candidates in general should not undergo this procedure. Those who do not have a proper support network and a pressure-release bed at home also are not good candidates for pressure injury reconstruction, because of the risk of recurrence or other complications. Patients who do not comply with nonoperative measures used to promote healing by secondary intention are poor reconstruction candidates as well.

Treatment options of unproven efficacy that are currently being studied include hyperbaric oxygen therapy, electrotherapy, growth factors, and negative-pressure wound therapy (NPWT). Initial studies of electrotherapy seem promising, and topical application of the recombinant human growth factor becaplermin has been approved for use in patients with diabetic neuropathic ulcers of the lower extremity. However, not enough evidence is available to permit these treatments to be recommended for the treatment of pressure injuries.

Discharge planning begins early in the hospital stay and requires an interdisciplinary approach. Knowledge of available resources facilitates smooth transitions through all levels of care. With more care being conducted in the home environment, education of the patient and caregiver in preventing and treating pressure injuries becomes increasingly important. Various methods can be used to facilitate the educational process, including charts, diagrams, photographs, and videos. This comprehensive approach can positively influence outcome.[77]

As a final note, some consideration should be given to the ethics of treating pressure injuries. For some individuals with pressure injuries, such as acutely hospitalized patients with a recoverable illness, aggressive treatment, as outlined in this article, is certainly indicated.

For other persons, however, such as chronically or terminally ill patients with long-standing or recurrent ulceration, aggressive treatment may not be in their best interests. In such instances, the wishes of the patient or the patient's family should be weighed carefully. It may prove to be the case that the patient's interests are better served by providing medical care and maintaining patient comfort than by instituting major invasive procedures.

In March 2015, the American College of Physicians (ACP) published clinical practice guidelines for risk assessment, prevention, and treatment of pressure ulcers (see Guidelines).[78, 79]

General Measures for Optimizing Medical Status

Spasticity should be controlled pharmacologically with medications such as diazepam, baclofen, or dantrolene sodium. Patients with spasticity refractory to medication may be candidates for neurosurgical ablation. Flexion contractures may also be relieved surgically.

Nutritional status should be evaluated and optimized to ensure adequate intake of calories, proteins, and vitamins.[80] Malnutrition is one of the few reversible contributing factors for pressure injuries, and establishing adequate caloric intake has been shown to improve healing of these lesions.

In a review of six clinical studies aimed at examining the effect of oral nutritional supplementation (ONS) enriched with arginine, vitamin C, and zinc in pressure ulcer care, ONS was found to have positive effects on pressure injury healing and potentially to reduce the risk of developing these injuries.[39, 81]

Implementation of more invasive methods of nutrient delivery becomes an ethical issue and must be weighed against the complications of such delivery. Goals of nutritional support should include adequate protein intake and the establishment of a positive nitrogen balance, with 1.0-2.0 g/kg/day being recommended for patients with pressure injuries.

Other important considerations include cessation of smoking, adequate pain control, maintenance of adequate blood volume, and correction of anaemia, the primary aims of which are to prevent vasoconstriction in the wound and to optimizing the oxygen-carrying capacity of the blood.

The wound and surrounding intact skin must be kept clean and free of urine and faeces through frequent inspection and cleansing. Appropriate evaluation of urinary or faecal incontinence is complex but must be performed thoroughly. Potentially reversible causes should be identified and treated. Urinary incontinence secondary to urinary tract infection (UTI) should be treated with antibiotics. Faecal incontinence secondary to diarrhoea may be related to an infectious cause (e.g. Clostridium difficilepseudomembranous colitis) that resolves with appropriate antibiotics.

Manual disimpassion and the addition of stool bulking agents to the diet may relieve overflow faecal incontinence. Urinary or faecal incontinence with no treatable cause may be minimized by establishing a bowel and bladder regimen. Constipating agents and a low residue diet also may be helpful.

Diapers and incontinence pads may be useful absorbing moisture away from the surface of the skin, provided that they are checked regularly and changed when soiled. If used inappropriately, these products may actually aggravate maceration and result in dermatitis. A bladder catheter or (in males) a condom catheter may be used to control urinary incontinence. In very severe cases involving chronic stool contamination, surgical diversion should be considered.

Bacterial contamination must be assessed and treated appropriately. Differentiation of infection from simple contamination through tissue biopsy (see Workup) helps ensure that antibiotics are used judiciously (ice, only in cases of actual infection) and, ideally, helps minimize the development of resistant species. Antibiotics also are indicated when accompanying osteomyelitis, cellulitis, bacteraemia, or sepsis is present.

A system of assessing wound healing must be in place to facilitate continuity of care among the various health care providers involved in the care of the patient. This often includes serial photography, detailed descriptions of the wound, and measurement of wound dimensions.

Pressure Reduction

The first step in healing a pressure injury is determination of the cause (ie, pressure, friction, or shear).[5, 82, 83] Turning and repositioning the patient remains the cornerstone of prevention and treatment through pressure relief. Patients who are capable of shifting their weight every 10 minutes should be encouraged to do so. Repositioning should be performed every 2 hours, even in the presence of a specialty surface or bed.

Patients who are bedbound should be positioned at a 30° angle when lying on their side to minimize pressure over the ischial tuberosity and greater trochanter. Efforts should be made to avoid sliding the patient over a surface to prevent shear forces and friction. Patients who develop a pressure injury while sitting should be placed on bed rest with frequent repositioning.

Pressure reduction may be achieved through the use of specialized support surfaces for bedding and wheelchairs that can keep tissue pressures below 32 mm Hg (the standard threshold value for evaluating support surfaces).[84, 85, 86, 87, 88] In theory, reduction of tissue pressures below capillary filling pressures should allow adequate tissue perfusion. Various different types of specialized support surfaces are available (see Table 1 below).

Table 1. Advantages and Disadvantages of Specialized Support Surfaces

Surface	Advantages	Disadvantages
Air	Low maintenance; inexpensive; multipatient use; durable	Can be punctured; requires proper inflation
Gel	Low maintenance; easy to clean; multipatient use; resists puncture	Heavy; expensive; little research
Foam	Lightweight; resists puncture; no maintenance	Retains heat and moisture; limited life
Water	Readily available in community; easy to clean	Requires heater; transfers are difficult; can leak; heavy; difficult to maintain; procedures difficult
Dynamic overlays	Easy to clean; moisture control; deflates for transfers; reusable pump	Noisy; can be damaged by sharp objects; requires assembly; requires power
Replacement mattresses	Reduced staff time; multipatient use; easy to clean; low maintenance	High initial cost; may not control moisture; loses effectiveness
Low air loss	Head and foot of bed can be raised; less frequent turning required; relieves pressure; reduces shear and friction; moisture control	Noisy; expensive; transfers are difficult; requires energy source; restricts mobility; requires skilled setup; rental charge
Air fluidized	Reduces shear and friction; lowest interface pressure; low moisture; less frequent turning required	Expensive; noisy; heavy; dehydration and electrolyte imbalances can occur; may cause disorientation; transfers are difficult; hot

These support surfaces may be divided into dynamic systems, which require an energy source to alternate pressure points, and static systems, which rely on redistribution of pressure over a large surface area and do not require an energy source. Each device may be further described as either

pressure-reducing or pressure-relieving. Pressure-relieving devices consistently reduce pressure below capillary closing pressure; pressure-reducing devices keep pressures lower than standard hospital beds but not consistently below capillary closing pressure.

These pressure-relief surfaces are often heavy, expensive, and difficult to clean, and they require ongoing maintenance to ensure proper function. In addition, they must be used properly to be effective. The patient's head and shoulders should be only minimally elevated on one pillow or a foam wedge to reduce shear forces and prevent the patient from "bottoming out" or having the sacrum or ischial tuberosities resting on the bed frame.

In a comparative study, two different cushions to prevent heel pressure injuries were investigated: a wedge-shaped, bed-wide, viscoelastic foam cushion and an ordinary pillow.[89] The patients using the wedge-shaped cushion had a decreased incidence of heel pressure injuries, and the probability of remain free of pressure injuries remained higher.

To date, relatively few clinical trials have been performed to evaluate the effectiveness of specialized support surfaces. Those that have been published have mostly been based on evaluation of tissue interface pressure, which is the force per unit area that acts perpendicularly between the body and the support surface and serves as an approximation of capillary closing pressure.[84, 85, 90, 86, 91, 92, 93, 94, 87, 88, 56]

Clinical trials for prevention and treatment of pressure injuries have been performed on air-fluidized and low-air loss beds.[55, 95, 96, 97] Although there is evidence that all of these surfaces can help prevent or treat pressure injuries can be prevented or improved, no data suggest that one support surface consistently performs better than all others in all circumstances.[55, 96, 98, 99, 100, 101] Therefore, patients should be actively treated on an individual basis to reduce specific risk factors.

A systematic review concluded that special foam mattresses designed to prevent pressure injuries were generally more effective than standard mattresses in patients at risk.[102] Organizations might consider the use of pressure-relief devices for high-risk patients in the operating room because this is associated with a reduction in the postoperative incidence of pressure injuries.

An updated systematic study not only confirmed the benefit of higher-specification foam mattresses but also suggested that sheepskin overlays were beneficial as well.[103] One of the trials included in this analysis determined that alternating-pressure mattresses may be more cost effective than alternating-pressure overlays. Several studies found only limited evidence when higher-technology products were compared.[104, 102]

Selection of a support surface should be based on the patient's management plan, his or her risk factors for developing pressure injuries, and the cost of obtaining and servicing the device.[90, 105] A dynamic management plan for each individual should include discontinuing the use of a support surface when it is determined that the patient is no longer at risk for developing pressure injuries.[55, 98, 99, 106, 107]

Any individual thought to be at risk for developing pressure injuries should be placed on a pressure-reducing device (e.g., foam, static air, alternating air, gel, or water) when lying in bed to relieve pressure on the heels.[108, 106, 109, 110, 111, 112] For persons who use a wheelchair, pressure-reducing devices of foam, gel, air, or a combination of these materials should be used.[113, 91, 114, 115, 116] Pressure-reducing devices should be used in addition to standard nursing care.[4, 117, 118]

The Agency for Healthcare Policy and Research (AHCPR) Pressure Ulcer Panel has developed guidelines for managing existing pressure ulcers (see Guidelines).[108, 31, 119, 120, 121, 96, 98, 101, 122, 7, 55, 122, 123]

Wound Management

Debridement and debriding agents

The purpose of wound debridement is to remove all materials that promote infection, delay granulation, and impede healing, including necrotic tissue, eschar, and slough (ie, the stringy yellow, green, or grey nonviable debris in an ulcer). Accurate injury staging cannot be accomplished until necrotic tissue is removed.[124] The following three debridement procedures are commonly used:

- Enzymatic debridement - This uses various chemical agents (proteolytic enzymes) that act by attacking collagen and liquefying necrotic wound debris without damaging granulation tissue [10, 76]
- Mechanical nonselective debridement - In this approach, necrotic tissue is loosened and removed is accomplished by means of whirlpool treatments, forceful irrigation, or the use of wet-to-dry dressings
- Sharp debridement - This consists of surgical removal of the eschar and any devitalized tissue within it (see Surgical Debridement); it is indiscriminate in the removal of vital and devitalized tissue and thus requires a great deal of clinical skill and judgment [125, 126]

Povidone-iodine solution can be used to debride infected wounds. Although the effervescent action of hydrogen peroxide results in wound debridement, it is not recommended for frequent or long-term use in pressure injuries, because it indiscriminately removes necrotic material and fragile granulation tissue and because it and other cleansing agents have been found to be toxic to fibroblasts.[127, 128, 10]

Once debridement has been completed and clean granulation tissue has been established, the use of debriding agents should be discontinued, and the site should be kept clean and moist.[128]

Solutions for wound cleansing

The major purpose of cleansing the wound is to decrease its bioburden and facilitate healing.[76, 127] When no germicidal action is required, normal saline is used. Saline solution should also be used as a rinse after other solutions are used to irrigate the wound and minimize fluid shifts within newly forming tissue. Normal saline solution can reduce the drying effects that some irrigants may have on tissue.[76, 129]

Povidone-iodine is useful against bacteria, spores, fungi, and viruses. Dilution is recommended, and this agent should be discontinued when granulation occurs.[76] Laboratory data demonstrate that povidone-iodine is toxic to fibroblasts in vitro, a finding that has theoretical implications for wound healing. Because povidone-iodine can affect thyroid function, it could be contraindicated for some patients.[129]

Acetic acid (0.5%) is specifically effective against Pseudomonas aeruginosa, a particularly difficult and common organism in fungating lesions. Acetic acid can change the colour of tissue and can mask potential superinfection. Rinsing with normal saline also is recommended.[129]

Sodium hypochlorite (2.5%) has some germicidal activity but is primarily used to debride necrotic tissue. Before it is used, zinc oxide should be placed around the edges of the wound to reduce the amount of irritation.[76] After cleansing with sodium hypochlorite, normal saline should be used as a rinse.[129] A multitude of cleansing agents are on the market, but none has been shown to be more efficacious than the others, and expert opinion still favours normal saline.[10]

Wound dressings

The choice of wound dressings varies with the state of the wound, the goal being to achieve a clean, healing wound with granulation tissue. A stage 1 pressure injury may not require any dressing. For more advanced injuries, various dressing options are available (see Table 2 below).

Table 2. Key Performance Characteristics of Major Wound Dressing Types

Major Dressing Type	Key Performance Characteristics
Alginates (sheets and fillers)	Exudate absorption; obliteration of dead space; autolytic debridement
Foams (sheets and fillers)	Obliteration of dead space; retention of moisture; exudate absorption; mechanical debridement
Gauzes (woven and nonwoven)	Obliteration of dead space; retention of moisture; exudate absorption; mechanical debridement
Hydrocolloids (wafers and fillers)	Occlusion; retention of moisture; obliteration of dead space; autolytic debridement
Hydrogels (sheets and fillers)	Retention of moisture; autolytic debridement
Transparent films	Occlusion; retention of moisture; autolytic debridement
Wound fillers	Obliteration of dead space; exudate absorption; retention of moisture; autolytic debridement
Wound pouches	Exudate control

Hydrocolloid dressings form an occlusive barrier over the wound while maintaining a moist wound environment and preventing bacterial contamination. A gel is formed when wound exudate comes in contact with the dressing. This gel can have fibrillolytic properties that enhance wound healing, protect against secondary infection, and insulate the wound from contaminants.[57, 130] Hydrocolloids help prevent friction and shear and may be used in stage 1, 2, 3, and some stage 4 pressure injuries with minimal exudate and no necrotic tissue.

Gel dressings are available in sheet form, in granules, and as liquid gel. All forms of gel dressings keep the wound surface moist as long as they are not allowed to dehydrate. Some gel dressings provide limited to moderate absorption, some provide insulation, and some provide protection against bacterial invasion. All gel dressings allow atraumatic removal.[10, 57, 131]

Transparent adhesive dressings are semipermeable and occlusive. They allow gaseous exchange and transfer of water vapor from the skin and prevent maceration of healthy skin around the wound. In addition, they are nonabsorptive, reduce secondary infection, and allow atraumatic removal. These dressings minimize friction and shear and may be used in shallow stage 1, 2, and 3 pressure injuries with minimal exudate and no necrotic tissue; however, they do not work well on patients who are diaphoretic or have wounds with significant exudation.[57]

Alginate dressings are semi occlusive, highly absorbent, and easy to use.[132, 133] They are natural, sterile, nonwoven dressings derived from brown seaweed. Alginate forms a gel when it comes into contact with wound drainage, and may be used in light to heavily draining stage 2, 3, and 4 injuries. It may be used in both infected and noninfected wounds[132] ; however, it should not be applied to dry or minimally draining wounds, as it can cause dehydration and delay wound healing.

Wounds with surface debris or fibrinous exudate may be mechanically debrided with wet-to-dry dressings incorporating normal saline or enzymatically debrided with collagenase. Wounds with a high level of bacterial contamination may benefit from wound irrigation. It has been shown that irrigation by low-pressure pulsatile lavage therapy with saline is more effective than continuous saline irrigation in decreasing bacterial loads within wounds.[134]

Vacuum-assisted closure (VAC) sponges conform to the wound surface by suction and stimulate wound contracture while removing exudate and oedema. Daily whirlpool therapy or pulse lavage therapy may be used to irrigate and mechanically debride the wound.

The choice of dressings is not as important as their appropriate application. The following points should be kept in mind:

- These dressings are not a substitute for sharp debridement in the presence of eschar or other necrotic material
- Dressings should be applied by trained individuals
- Care should be taken to keep the wound dressing within the boundaries of the wound to prevent maceration of the surrounding skin
- A hydrocolloid pad or skin sealant can be used to protect the surrounding skin and serve as a surface to which tape may be applied to hold dressings in place; tubular mesh gauze is an alternative for holding dressings in place in patients with extremely fragile skin

Antimicrobials and antibiotics

Antibiotic creams such as silver sulfadiazine may be applied to wounds to decrease bacterial load. Silver sulfadiazine has an excellent antimicrobial spectrum of activity, low toxicity, ease of application, and minimal pain. It inhibits DNA replication and modification of the cell membrane of Staphylococcus aureus; Escherichia coli; Candida albicans; Klebsiella, Pseudomonas, and Proteus species; and Enterobacteriaceae.

Mafenide, an antimicrobial agent that is bacteriostatic to many gram-positive and gram-negative organisms, including Pseudomonas aeruginosa, can penetrate an eschar and promote autolytic softening of the eschar prior to debridement.

Evaluation of a patient with an infected wound should follow an algorithmic approach. The following questions should be asked:

- Is the infection local (rubor, dolor, calor) or systemic (fever, tachycardia, hypotension, delirium, altered mental status)?
- Which antibiotic is most appropriate for the patient?
- Does the patient have any known allergies?
- Does the patient have any metabolic impairments that would alter the pharmacokinetics or pharmacodynamics of the drug?
- What are the effects of the drug on the hematopoietic system?

- What attributes does the drug possess for effective tissue penetration (ie, how much of the drug actually ends up in the tissue of interest)?
- How is the drug metabolized?
- What are the patient's total weight and lean body and fat mass?

The adverse effects of antibiotics are well known, and those that impede wound healing should be considered and counteracted. Antibiotic resistance is a major concern. Therefore, when antibiotic therapy is ordered, the wound care specialist must be alert to detect signs of antibiotic resistance, and he or she must be attentive to the results of the laboratory data, especially culture and sensitivity results.

Patients who are immunocompromised or have impaired chemotaxis resulting in bacterial overgrowth or candidiasis need concomitant treatment with selected antimycotic or antifungal agents.

Other considerations in prescribing an antibiotic include the patient's length of hospital stay, the availability of home health services and infusion services, the influence of the pharmacy and therapeutics committee, the hospital's formulary, and the influence of the payer's approval of prescription benefits.

Other wound treatments

A wide variety of additional therapeutic methods are being evaluated for the treatment of chronic wounds, specifically for pressure injury management.[135] These include electrotherapy,[136, 137] application of growth factors,[138, 139, 140, 62] and preventive use of free radical scavengers and special drug delivery systems.[141, 142, 143]

The recombinant human platelet-derived growth factor becaplermin has been approved by the US Food and Drug Administration (FDA) for the treatment of lower-extremity diabetic neuropathic ulcers that extend into the subcutaneous tissue or beyond. Studies are underway to explore the possibility of expanding its approved indications to include other wounds. Other growth factors also are being evaluated for use in human clinical settings.

Another potentially promising treatment option is NPWT using VAC.[144, 145, 146] NPWT enhances wound healing by reducing oedema, increasing the rate of granulation tissue formation, and stimulating circulation. Increased blood flow translates into a reduction in the bacterial load (removal of interstitial tissue) and delivery of infection-fighting leukocytes.[147]

The following are general indications for NPWT[148] :

- Chronic wounds
- Acute wounds
- Traumatic wounds
- Partial-thickness wounds
- Dehisced wounds
- Diabetic ulcers
- Pressure injuries
- Flaps
- Grafts

The following are general contraindications for NPWT[148] :

- Malignancy of the wound
- Untreated osteomyelitis
- Nonenteric or unexplored fistulas
- Known allergies or sensitivity to acrylic adhesives
- Placement of negative-pressure dressings directly in contact with exposed blood vessels, organs, or nerves

Principles of Surgical Intervention

Even with optimal medical management, many patients require a trip to the operating room for debridement, diversion of the urinary or faecal stream, release of flexion contractures, wound closure, or amputation.

Debridement is aimed at removing all devitalized tissue that serves as a reservoir for ongoing bacterial contamination and possible infection. Extensive debridement should be done in the operating room, but minor debridement is commonly performed at the bedside. Although many of these patients are insensate, others are unable to communicate pain sensation. Pain medication should be administered liberally, and vital signs may indicate pain perception. Care should be taken with bedside debridement because wounds may bleed significantly.

Urinary or faecal diversion may be necessary to optimize wound healing. Many of these patients are incontinent and their wounds are contaminated with urine and faeces daily. Patients with loose stools benefit from constipating agents and a low-residue diet.

Release of flexion contractures resulting from spasticity may assist with positioning problems, and amputation may be necessary for a nonhealing wound in a patient who is not a candidate for reconstructive surgery.

Reconstruction of a pressure injury is aimed at improvement of patient hygiene and appearance, prevention or resolution of osteomyelitis and sepsis, reduction of fluid and protein loss through the wound, and prevention of future malignancy (Marjolin ulcer). In general, stage 3 and 4 pressure injuries tend to require flap reconstruction.

The first step is adequate excision of the injury, including the bursa or lining, surrounding scar tissue, and any heterotopic calcification found. Underlying bone must be adequately debrided to ensure that there is no retained nidus of osteomyelitis. Some evidence in the literature indicates that pulsed lavage can be beneficial in reducing bacterial counts in wounds, and some surgeons routinely use this method after debridement.

Once the wound has been appropriately debrided, it may be closed in a variety of ways, depending on the location of the pressure injury, any previous scars or operations, and the surgeon’s individual preference. However, the basic tenets of reconstruction remain the same in all pressure injury reconstructions.

Very few pressure injuries can or should be closed primarily after debridement, given the unacceptably high complication rates. A well-vascularized pad of tissue should be placed in the wound, usually a musculocutaneous flap transposed or rotated on a pedicle containing its own blood

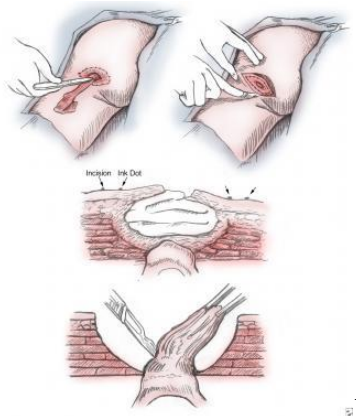
supply. This also may involve the use of tissue expansion or a free flap with microvascular anastomosis. The goals are to eliminate dead space in the wound, enhance perfusion, decrease tension on the closure, and provide a new source of padding over the bony prominence.

Before wound closure, drains should be placed in the bed of the wound. This allows external drainage of any fluid that may accumulate beneath the flap and should help minimize wound complications such as hematoma and seroma.

Surgical Debridement

Once the decision has been made to reconstruct, the wound is debrided. It should be noted that debridement of a pressure injury that will be reconstructed is different from debridement of a pressure injury that will be treated conservatively (i.e., allowed to heal by secondary intention).

Pressure injuries that are treated conservatively are not radically debrided; they need only be debrided of obvious necrotic tissue. For pressure injuries that will be reconstructed, a radical bursectomy is performed to prevent the development of infection or seroma under the flap. This radical bursectomy is technically achieved by placing a methylene blue–moistened sponge in the bursa and excising the pressure injury circumferentially, removing all granulation tissue, even from the wound base (see the image below).



Radical bursectomy is performed by placing methylene blue–moistened sponge in bursa and excising pressure ulcer circumferentially, removing all granulation tissue, even from wound base.

After the bursectomy, primary closure of the pressure injury is almost always under tension and is therefore doomed to fail if attempted. Other technical points of pressure injury reconstruction include radically removing underlying necrotic bone, padding of the bone stump, filling the dead space with muscle, using a large flap, achieving adequate flap mobilization to avoid tension, and avoiding adjacent flap territories to preserve options to reconstruct other locations.

Options for Wound Closure

Several options are available for surgical management of pressure injuries, including direct closure, skin grafting, skin flaps, and musculocutaneous flaps. Such management can provide skin coverage as well as soft tissue coverage. Flaps containing muscle provide a physiologic barrier to infection, eliminate dead space in the wound, and improve vascularity. Improved vascularity enhances local

oxygen tension, provides extended soft-tissue penetration for antibiotics, and improves total lymphocyte function.[149, 150, 151]

The patient should be medically stable and able to benefit from the procedure. The patient should also participate in the decision. The nutritional status of the patient must be considered because good nutritional parameters are required for good wound healing and immune function. Involuntary muscle spasms should be controlled preoperatively with baclofen or diazepam.

Factors associated with impaired healing should be corrected preoperatively. Tobacco use and smoking are associated with intrinsic factors that compromise wound healing.[152] For example, carbon monoxide and nicotinic acid are potent vasoconstrictors that increase blood viscosity.[153] These factors predispose tissue to excessive oxidase activity and free radical injury.

Under normal conditions, the body is able to handle normal oxidative stress. However, with excessive stress comes increased risk for development of pressure injuries and impaired wound healing. A neutrophil-mediated free radical injury results in excessive oxidase activity, which can cause vascular damage and thrombosis, leading to cell death and tissue destruction.[141, 142, 143]

Patient positioning is dictated by the location of the ulcer and the planned reconstruction. Many pressure ulcers occur in the gluteal region and require prone positioning. Most anaesthesiologists choose to use general endotracheal anaesthesia, particularly if the patient is prone, but ulcer closure may be performed under regional or local anaesthesia if necessary. Significant blood loss is possible; accordingly, 2 units of type-specific packed red blood cells should be available during the operation.

Arrangements should be made to have a pressure-reducing mattress available for the postoperative period to reduce the risk of immediate recurrence or dehiscence. If urinalysis and urinary culture findings (i.e., nitrites, leukocyte esterase) confirm the presence of a urinary tract infection (UTI), appropriate treatment should be provided.

Direct closure

Although direct closure is the simplest approach, pressure injuries considered for surgical treatment are usually too large to be amenable to direct primary closure. Because these wounds are tense as a result of large soft-tissue defects, direct closure can lead to wound defects, excessive wound tension, and a paucity of soft-tissue coverage. Tissue expanders have been used to provide more skin surface and to facilitate closure.[154]

Skin grafts

Split-thickness skin grafts can be used to repair shallow defects and pressure injuries, but their main disadvantage is that they provide only a skin barrier. When applied directly to granulating bone, skin grafts quickly erode, thus precluding healing. They also cause scars in the area from which the skin is harvested, and the transplanted skin is never as tough as the original skin.

Skin flaps

Before the 1970s, repair using local full-thickness skin flaps was the standard surgical treatment for pressure injuries; today, it is typically employed as an alternative to secondary repair.[155] Local skin flaps have a random vascular supply, and the tissue repair is essentially a redistribution of

inadequately perfused tissue rather than a planned revascularization that makes use specific blood vessels.

Myocutaneous flaps

Myocutaneous (musculocutaneous) flaps are usually the best choice for patients with spinal cord injuries (SCIs) and for those who have a loss of muscle function that does not contribute to a comorbidity. For patients who are ambulatory, the choice is less clear, in that the improved blood supply and reliability of the muscle flap must be balanced against the need to sacrifice functional muscle units.[156, 157, 158]

Myocutaneous flaps can help heal osteomyelitis and limit the damage caused by shearing, friction, and pressure.[159, 160, 161] They bring muscle and skin to the area of the defect and are probably as resistant to future pressure injuries as the original skin.

Free flaps

Free flaps are muscle-type flaps in which the vein and artery are disconnected at the donor site and subsequently reconnected to the vessels at the recipient site with the aid of a microscope. This is the most complex method of wound closure and would be considered only after all other options for reconstruction have been exhausted. In paraplegic patients dependent on their upper body for mobility, the latissimus dorsi would typically be an unacceptable donor for free tissue transfer; however, a portion of the muscle may be used with limited donor site morbidity. A protocol for postoperative care must be followed strictly for free flap survival.[162]

Surgical Management of Specific Pressure Injury Types

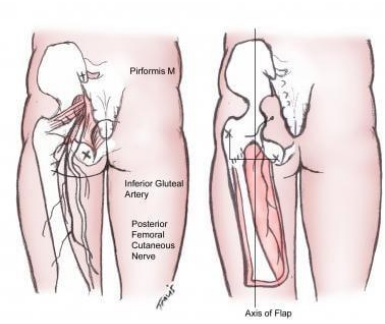
The choice of reconstruction approach depends on the location of the pressure injury.

Ischial pressure injury

The ischial location is the most common site of pressure injury in individuals with paraplegia. In the course of excisional debridement in preparation for flap repair of an ischial wound, aggressive resection of the ischial tuberosity may raise the risk of a contralateral ischial pressure injury from increased contralateral pressure. Bilateral ischiectomy increases pressure on the perineum and thus increases the risk of perineal pressure injury.

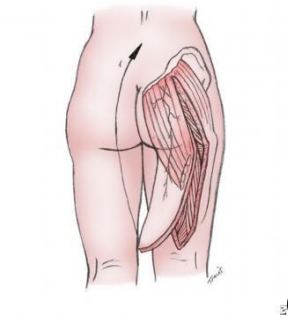
Recurrence of the pressure injury is common in the ischial location.[163] Therefore, the first option for reconstruction of ischial wounds is the gluteal thigh rotation flap, which does not preclude the future use of the inferior portion of the gluteus maximus muscle.[164, 165]

The gluteal thigh rotation flap is an axial flap based on the inferior gluteal artery. Both the biceps femoris flap and the hamstring myocutaneous flap transect the inferior gluteal artery. With the gluteal thigh flap, a superiorly based flap is elevated, with its axis being the inferior gluteal artery located between the greater trochanter and the ischial tuberosity (see the image below).

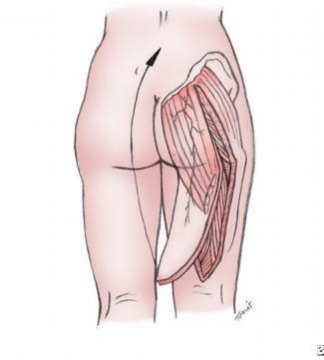


With gluteal thigh flap, superiorly based flap is elevated, with inferior gluteal artery located between greater trochanter and ischial tuberosity as its axis.

The gluteal thigh rotation flap is raised as a fasciocutaneous flap superiorly to the gluteal crease (see the image below).


Gluteal thigh rotation flap is raised as fasciocutaneous flap superiorly to gluteal crease.

The gluteal thigh flap may be raised to include the inferior portion of the gluteus maximus. This increases the arc of rotation and allows the flap to be used to reconstruct sacral defects (see the image below).[164]

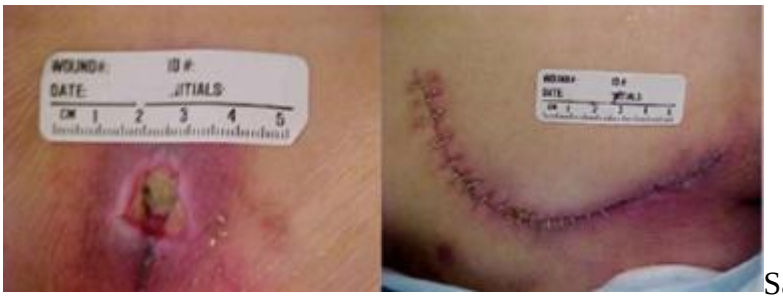

Gluteal thigh flap may be raised to include inferior portion of gluteus maximus, which increases arc of rotation to allow flap also to be used to reconstruct sacral defects.

Another popular option for ischial reconstruction, the inferior gluteus maximus myocutaneous flap, limits options for reconstruction of sacral wounds. Bilateral V-Y advancement flaps, inferiorly based random flaps, and superior gluteal myocutaneous flaps are not options for sacral reconstruction if an inferior gluteal myocutaneous flap has been used.

Additional options described for ischial reconstruction include the hamstring myocutaneous flap, the biceps femoris myocutaneous flap, the tensor fasciae latae (TFL) flap, the gracilis myocutaneous flap,[163] and the medially based posterior thigh skin flap with or without the biceps femoris.

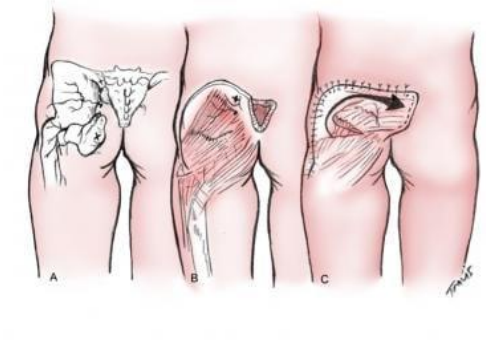
Sacral pressure injury

Sacral pressure injuries (see the image below) are common in patients who have been on prolonged bed rest. Treatment involves complete excision, including the entire bursa, and conservative ostectomy.

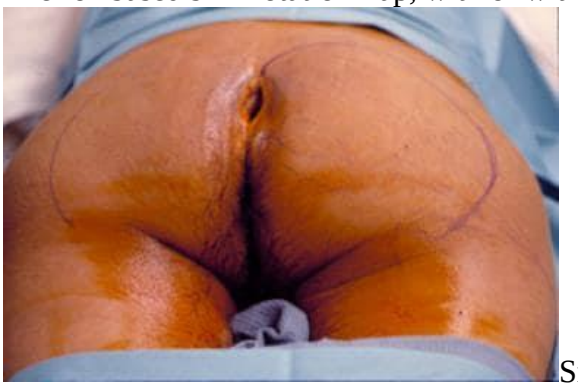


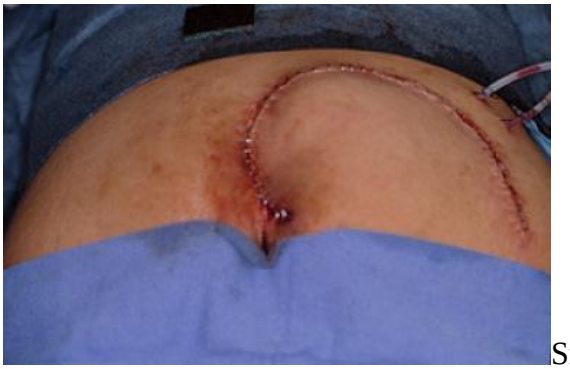
Sacral pressure ulcer before and after flap closure.

Small sacral pressure injuries can be reconstructed with an inferiorly based skin rotation flap, with or without a superior gluteus maximus myocutaneous flap (see the images below). The use of the random skin rotation flap does not preclude later use of the gluteus maximus. When a random skin rotation flap is used, it is essential to design a large and wide flap with an axis of rotation that permits tension-free closure.

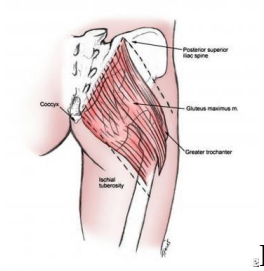


Small sacral pressure sores can be reconstructed with the inferior-based skin rotation flap, with or without the superior gluteus maximus myocutaneous flap.


Small sacral pressure ulcer reconstructed with inferiorly based skin rotation flap.

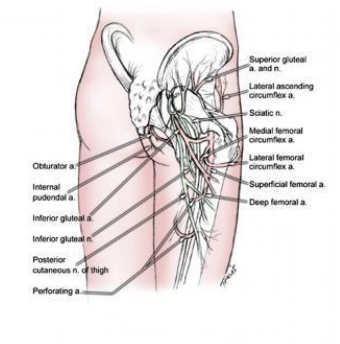
 Small sacral pressure ulcer reconstructed with inferiorly based skin rotation flap.

With a superior gluteal myocutaneous flap, a wide skin rotation flap is elevated with the superior portion of the gluteus maximus. Landmarks for the superior gluteal artery on which this flap is based include the posterior superior iliac spine (PSIS) and the ischial tuberosity (see the image below).



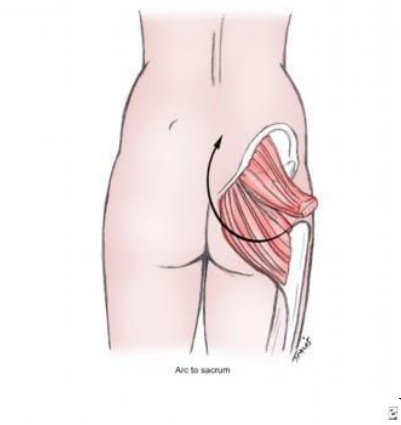
Landmarks for superior gluteal artery, on which superior gluteus maximus muscle flap is based, include posterior superior iliac spine and ischial tuberosity.

The superior and inferior gluteal arteries branch from the internal iliac artery superior and inferior to the piriformis approximately 5 cm from the medial edge of the origin of the gluteus maximus from the sacrococcygeal line (from PSIS to coccyx; see the image below).



Superior and inferior gluteal arteries branch from internal iliac superior and inferior arteries to piriformis approximately 5 cm from medial edge of origin of gluteus maximus from sacrococcygeal line.

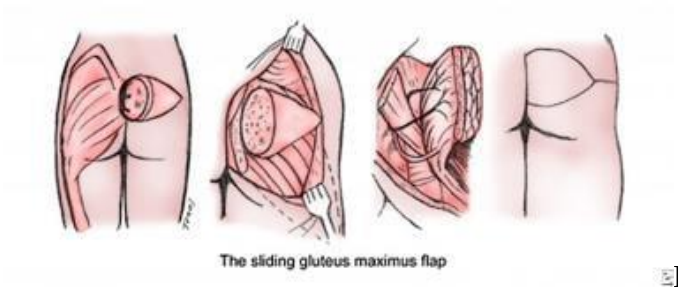
When the superior portion of the gluteus maximus muscle is used as a flap, it is elevated in a lateral-to-medial direction to keep from injuring the superior gluteal artery, which can be difficult to identify from the medial direction because of the inflammation and scarring associated with the sacral pressure injury. The insertion of the superior portion of the gluteus maximus is the iliotibial tract; this insertion is released.



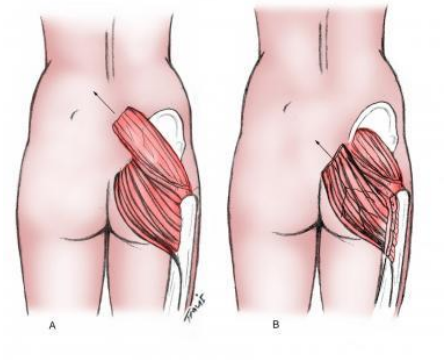
When superior portion of gluteus maximus is used as flap, it is elevated in lateral-to-medial direction to avoid injury to superior gluteal artery. Insertion of superior portion of gluteus maximus into iliotibial tract is released. Harvesting entire length of muscle may be necessary to allow rotation or turnover into defect without tension.

The superior gluteal artery is only 4 cm long, which limits the rotation of the muscle. Thus, harvesting the entire length of the muscle may be necessary to allow for rotation or turnover into the defect without tension.

Larger sacral pressure injuries require the use of bilateral flaps such as bilateral V-Y myocutaneous advancement flaps (see the first image below). V-Y flaps can be based on the superior, inferior, or entire gluteus maximus, depending on the location of the pressure injury (see the second image below).


The sliding gluteus maximus flap

Larger sacral ulcers require use of bilateral flaps, such as bilateral V-Y advancement flaps.



V-Y flaps can be based superiorly or inferiorly or on entire gluteus maximus.

The V should be fashioned wide enough and long enough to permit closure as a Y without tension. The medial edge of the origin of the gluteus maximus is elevated in a medial-to-lateral direction for

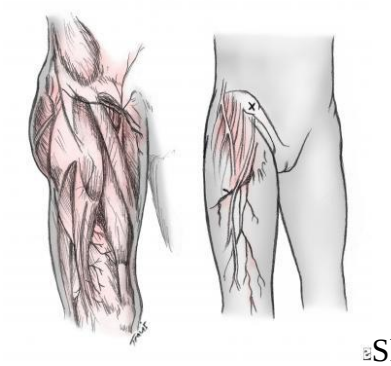
approximately 4 cm because the superior and inferior gluteal arteries enter the gluteus maximus 5 cm from its origin.

Release of the gluteal muscle insertion laterally is important for medial advancement and tension-free approximation of the muscles medially. Inflamed fibrous tissue along the medial muscle edge can be preserved and used to hold sutures for midline muscle approximation. Another option for sacral reconstruction is the transverse lumbosacral flap.[166]

Trochanteric pressure injury

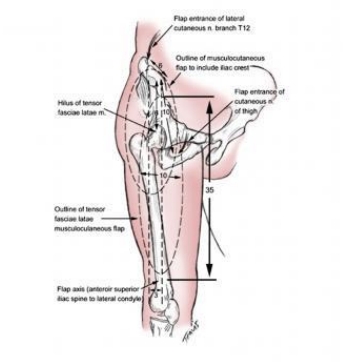
Trochanteric pressure injuries are less common and are typically associated with minimal skin loss. Excisional debridement of these injuries in preparation for flap repair involves resection of the entire bursa and greater trochanter of the femur. The first option for reconstruction of trochanteric pressure injuries is the TFL flap, a myocutaneous flap based on the lateral femoral circumflex artery.[167] The TFL is 13 cm long, 3 cm wide, and 2 cm thick, and it originates from the anterior superior iliac spine (ASIS) and the iliac crest and inserts into the iliotibial tract.

The skin paddle is harvested in a width of 10 cm and designed over the muscle along an axis from the ASIS to the lateral tibial condyle (see the image below).



Skin paddle is harvested 10 cm in width and designed over muscle along axis from anterior superior iliac spine to lateral tibial condyle.

The inferior limit of the cutaneous territory can be extended to 6 cm above the knee and 25-35 cm in length (see the image below). The lateral femoral circumflex artery can be found approximately 6-8 cm inferior to the ASIS. In patients with lumbar lesions, a sensate TFL flap can be designed to include the T12 dermatome by fashioning the flap to include the area 6 cm posterior to the ASIS.

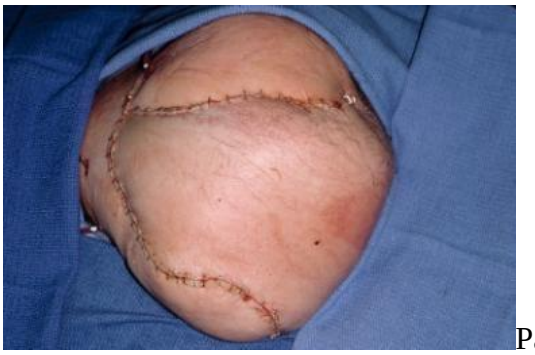


Inferior limit of cutaneous territory can be extended to 6 cm above knee and 25-35 cm in length. Lateral femoral circumflex artery can be found approximately 6-8 cm inferior to anterior superior iliac spine.

Other described modifications of the TFL flap include the retroposition V-Y flap and the bipedicled TFL flap. Other options for trochanteric pressure injury reconstruction include the vastus lateralis myocutaneous flap, the gluteal thigh flap, and the anterior thigh flap.

Multiple pressure injuries

Multiple pressure injuries may be observed in the same patient. Reconstruction of multiple injuries may require the use of a total thigh flap (see the image below). The total thigh flap is a long and formidable operation that typically involves the transfusion of 6-20 units of blood. It should be reserved for use as a salvage procedure when other attempts have been unsuccessful. Patients who have undergone a unilateral total thigh flap can sit a wheelchair (see image below).


Patient required reconstruction of extremely large pressure ulcer with fillet total thigh flap procedure.

Postoperative Care

In terms of the ultimate success or failure of pressure injury reconstruction, the work done in the operating room is only the first step. Once a pressure injury has been successfully closed, appropriate postoperative care must be initiated to encourage wound healing and to reduce the risk of complications such as recurrence.

From the time of transfer from the operating table to the air-fluid bed, care must be exercised to prevent shearing and tension across the flap repair. Patients are positioned flat in the air-fluid bed for 4 weeks. After 4 weeks, the patient can be placed carefully into a semi sitting position.

At 6 weeks after surgery, the patient begins sitting, initially for only 10 minutes at a time. After these sitting periods, the flap should be evaluated for discoloration and wound edge separation. Over 2 weeks, the sitting periods are increased in 10-minute increments until they reach 2 hours. Patients are taught to lift themselves to relieve pressure for 10 seconds every 10 minutes.

Skin care must be performed daily. This involves careful inspection of all skin surfaces to identify areas of impending breakdown before the breakdown occurs. (An often overlooked detail is to remove compression stockings and inspect the heels.) Skin should be washed with soap and water and completely dried. Moisture should not be allowed to accumulate on the skin or in clothing or bedding, nor should the skin be allowed to become overly dry and scaly. Skin moisturizers are useful to maintain the appropriate level of moisture at the skin surface.

Patients may benefit from transfer to a subacute or rehabilitation facility after wound closure. This allows them to receive ongoing education, observation, and rehabilitative therapies before returning to their usual place of residence.

Complications

Complications as a result of reconstructive surgery are, unfortunately, considerable. Such complications include the following:

- Hematoma
- Seroma
- Wound dehiscence
- Wound infection
- Recurrence

With the use of well-vascularized flaps, flap necrosis is infrequent.

Treatment of patients with pressure injuries involves several perioperative considerations to minimize the risk of adverse outcomes of the reconstruction, as follows:

- Preoperatively, patients must be meticulously and compulsively prepared, with nutritional deficiency, anaemia, spasms, and coexisting urinary infection corrected; adequate social resources, including pressure-release beds, wheelchair mattresses, and a compliant attitude, should be present
- Intraoperatively, key technical points must be carefully addressed, including tension-free flap reconstruction, suction catheter drainage, meticulous haemostasis, and aggressive debridement
- Postoperatively, a strict and careful regimen for the transition from flat bed rest to sitting and for weight-shifting into and out of the wheelchair in the return to daily living must be implemented; pressure-reducing mattresses and pressure-release techniques should be used

Even with close adherence to these guidelines, pressure injury recurrence rates are high. In caring for patients with chronic pressure ulcers, it is essential to plan flap procedures carefully and provide social resources unstintingly to reduce the high risk of adverse outcomes in this complication-prone population.

Activity

After successful wound closure, ambulatory patients should be out of bed with assistance as soon as possible. More strenuous physical activity should be delayed for approximately 6 weeks.

In patients with ischial tuberosity pressure injuries, sitting may be resumed 6 weeks after a healed wound is achieved. Sitting may be gradually reintroduced over several weeks, and detailed guidelines have been published. Because of the extremely high pressures generated over the ischial tuberosities during sitting, wheelchair patients should lift themselves out of their seat or rock back in the chair every 15 minutes.

These recommendations regarding the resumption of activity vary according to the clinical situation and are implemented at the discretion of the treating physician.

Prevention

Although in principle, pressure injuries are preventable and should not occur, they continue to be among the most pervasive and perplexing problems encountered in the treatment of persons who are ill, recovering from illness, or functionally impaired.

In 2016, the Wound, Ostomy and Continence Nurses Society (WOCN) issued guidelines for the prevention and management of pressure ulcers (injuries).[168] (See Guidelines.)

In 2017, the WOCN Society issued an updated position statement on the topic of avoidable and unavoidable pressure ulcers (injuries).[169] This statement defined unavoidable pressure ulcers as those that develop even though the patient's clinical condition and pressure ulcer risk factors were properly evaluated and interventions consistent with patient needs, management goals, and standards of practice were implemented, monitored, and revised as appropriate.

To the extent that prevention is achievable, it is the optimal form of treatment. Prevention of pressure ulcers has two main components: identification of patients at risk and interventions designed to reduce the risk.

Identification of patients at risk

Various approaches to the identification of persons at risk for the formation of pressure injuries have been tested. A person who uses a wheelchair, is in bed for most of the day, or has impaired ability to reposition the body should be assessed for additional factors that increase risk of pressure injuries. General physical and mental condition, nutritional status, activity level, mobility, and degree of bowel and bladder control are all known to affect this risk.[9, 56, 170, 125, 117]

A simple clinical prediction rule based on five patient characteristics may help identify patients who are at increased risk for pressure injury development and thus in need of preventive measures. Detection of a stage 2 or worse pressure injury during admission to the hospital is directly related to the following independent predictors of pressure injuries[171] :

- Age
- Weight at admission
- Abnormal appearance of the skin
- Friction and shear
- Planned surgery in the coming week

A systematic assessment of pressure injury risk can be accomplished by using a assessment tool such as the Braden scale or the Norton scale (see Table 3 below). No information is currently available to suggest that adaptations of these risk assessment tools or the assessment of any single risk factor or a combination of risk factors predicts risk as well as the overall scores obtained with these tools.[172, 173]

Table 3. Norton and Braden Scales for Assessing Pressure Ulcer Risk

Area of Comparison	Norton Scale	Braden Scale
Assessment criteria	Physical condition; mental condition; activity; mobility; incontinence (score ≥12 is at risk)	Activity; mobility; sensory perception; moisture; nutrition; friction; shear

Attributes	Tested on elderly persons in hospital settings	Evaluated in diverse sites (eg, medical-surgical, intensive care units, nursing homes)
Replications	Tested extensively	Tested extensively
Reliability	Not available	Good interrater reliability

In 1992, the Agency for Health Care Policy and Research (AHCPR), now known as the Agency for Healthcare Research and Quality (AHRQ), developed guidelines for the prediction and prevention of pressure ulcers in adults.[15] In 1994, these guidelines were followed by guidelines for the treatment of these lesions.[10]

According to the AHPCR prevention guidelines, risk assessment should include the following[15] :

- Complete medical history taking
- Determination of Norton (or Braden) score (see above)
- Skin examination
- Identification of previous pressure ulcer sites

Prime candidates for pressure ulcers include the following[15] :

- Elderly persons
- Persons who are chronically ill (e.g., those with cancer, stroke, or diabetes)
- Persons who are immobile (e.g., as a consequence of fracture, arthritis, or pain)
- Persons who are weak or debilitated
- Patients with altered mental status (e.g., from the effects of narcotics, anaesthesia, or coma)
- Persons with decreased sensation or paralysis

Secondary factors include the following[15] :

- Illness or debilitation increases pressure ulcer formation
- Fever increases metabolic demands
- Predisposing ischemia
- Diaphoresis promotes skin maceration
- Incontinence causes skin irritation and contamination
- Other factors, such as oedema, jaundice, pruritus, and xerosis (dry skin)

The WOCN Society has also issued guidelines on preventing pressure injuries (see Guidelines).[168]

Interventions for minimizing risk

Effective prevention of pressure injuries depends on a comprehensive care plan that includes strategies and practices aimed at reducing or eliminating the risk of these injuries. Elements of such a plan may include the following:

- Scheduled turning and body repositioning - Although numerous factors are known to contribute to the development of pressure injuries, it remains essential to establish a regimen in which pressure is completely relieved on all areas of the body at regular intervals [174, 175]
- Appropriate bed positioning - Patients can benefit from lying prone; shearing forces can be minimized by keeping the head of the bed lower than 45°

- Protection of vulnerable bony prominences - Positioning devices such as pillows or foam wedges (not donut-type devices [108]) should be used to prevent direct contact between bony prominences (e.g., knees and ankles); massage of body prominences should be avoided [9, 10, 113]
- Skin care - Removal of skin secretions and excretions; avoidance of hot water; use of nonirritating, non-drying skin-cleansing agents; use of moisturizers; use of topical agents such as moisture barriers; use of dry, wrinkle-free sheets
- Alertness for skin changes that might indicate an impending breakdown (e.g., inflammation of the skin that blanches on application of digital pressure [9]), particularly in elderly or immunocompromised patients
- Control of spasticity and prevention of contractures
- Use of support surfaces and specialty beds (see Pressure Reduction)
- Nutritional support as required - This may involve enteral or parenteral nutrition or vitamin therapy
- Maintenance of current levels of activity, mobility, and range of motion; persons who use a wheelchair should be taught to perform push up exercises and to lean side to side for pressure relief

All interventions should be monitored and documented. Specific details that are required include who should provide the care, how often it should be provided, and the supplies and equipment needed. How the care is to be undertaken should be individualized, written down, and readily available. Results of the interventions and the care being rendered should be documented. To ensure continuity, documentation of the plan of care should be clear, concise, and accessible to every caregiver. Patient education is also essential.[9]

In a study assessing the results of a long-term acute care hospital's program to reduce the occurrence of pressure injuries, the hospital traced its apparently above-average ulcer prevalence rates to the lack of wound care professionals, methods for consistently documenting prevention and wound data, and an interdisciplinary wound care team approach.[176] By addressing these issues, the hospital was able to reduce the prevalence of facility-acquired pressure injuries from 41% to an average of 4.2% over a 12-month period.

Consultations

A multidisciplinary approach can yield maximal benefit. Neurosurgery, urology, plastic surgery, orthopaedic surgery, and general surgery consultations all may be indicated in a given case. Rehabilitation medicine specialists, social workers, and psychologists or psychiatrists may work with geriatricians and internists to improve the patient's health, attitude, support structure, and living environment. Plastic surgeons perform most pressure injury reconstructions; a plastic surgery consultation is appropriate with any complex or chronic wound.

Long-Term Monitoring

Follow-up should be performed every 3 weeks for the first several months. The interval may then be increased to every 6 months and then yearly. Early issues include suture removal, drain removal, and when to allow the patient to exercise or sit up.

Concise documented measurement of the wound healing process contributes to efficient management. The most common method of monitoring the healing of pressure injuries utilizes

photography and diagrams.[62] Another method is to measure the volume (volumetrics) and the dimensions of the pressure injury wound (e.g., by using a measured amount of saline to infer the volume of the wound). Sophisticated radiographic techniques are available for this purpose as well, but they are too expensive for routine use.

Once healing is complete, long periods of uninterrupted pressure must be avoided. Patients must be repositioned frequently, either by their own efforts or with help from their support group. Seated patients with upper-extremity function should lift themselves from their wheelchair for at least 10 seconds every 10-15 minutes. Patients in bed should be repositioned at least every 2 hours.

Pressure dispersion, through the application of specialized support surfaces on beds and wheelchairs, should be extended through the wound healing period and into the outpatient setting if available and tolerated by the patient. This is an adjunct to the alternating of weightbearing surfaces and maintains low pressures on the tissues at all times. Control of spasticity and maintenance of adequate nutrition also must be continued into the outpatient setting to prevent recurrence.

After they return home, patients may benefit from visits from a home health care organization. Such visits may ease the transition and ensure that pressure avoidance strategies are adapted to the home and continued over the long term.

Guidelines

ACP Clinical Practice Guidelines

The 2015 American College of Physicians (ACP) clinical practice guidelines for risk assessment, prevention, and treatment of pressure ulcers included the following recommendations and statements[78, 79] :

- Perform a risk assessment to identify patients who are at risk of developing pressure ulcers
- Choose advanced static mattresses (made of foam or gel that stays put when a person lies down) or advanced static overlays (a material attached to the top of a mattress such as sheepskin or a pad filled with air, water, gel, or foam) in patients who are at an increased risk of developing pressure ulcers
- ACP recommends against using alternating-air mattresses or alternating-air (also called dynamic) overlays in patients who are at an increased risk of developing pressure ulcers
- Use protein or amino acid supplementation in patients with pressure ulcers to reduce wound size
- Use hydrocolloid or foam dressings in patients with pressure ulcers to reduce wound size; the evidence also showed that hydrocolloid dressings are better than gauze for reducing wound size and resulted in similar complete wound healing as foam dressings
- Although radiant heat dressings accelerated wound healing, there was no evidence they were better than other dressings for improving complete wound healing
- Use electrical stimulation as adjunctive therapy in patients with pressure ulcers to accelerate wound healing; the most common adverse effect for this stimulation was skin irritation, and frail elderly patients were more susceptible to harms from electrical stimulation
- Those at higher risk for pressure ulcers include blacks or Hispanics and those with lower body weight, cognitive or physical impairments, and other comorbid conditions that affect soft tissue(e.g., incontinence, oedema, malnutrition, and diabetes)

AHCPR Pressure Ulcer Panel Guidelines

Guidelines developed by the Agency for Healthcare Policy and Research (AHCPR) Pressure Ulcer Panel for managing existing pressure ulcers include the following:

- Use positioning devices to raise a pressure ulcer off the support surface; if the patient is no longer at risk for pressure ulcers, these devices may reduce the need for pressure-reducing overlays, mattresses, and beds; avoid using donut-type devices [108]
- Assess all patients with existing pressure ulcers to determine their risk for developing additional pressure ulcers; if the patient remains at risk, use a pressure-reducing surface [31, 119, 120, 121]
- If patients can assume a variety of positions without bearing weight on the lesion and without "bottoming out," a static support surface should be used [96, 98, 101, 122]
- If the patient cannot assume a variety of positions without bearing weight on the ulcer, if the patient fully compresses the static support surface, or if the pressure ulcer does not show evidence of healing, a dynamic surface should be used [96]
- Finally, if the patient has large stage III or stage IV pressure ulcers on multiple turning surfaces, a pressure-relieving product is warranted [7, 55, 96, 101, 122, 123]

WOCN Guidelines

In 2016, the Wound, Ostomy and Continence Nurses Society (WOCN) issued guidelines on the prevention and management of pressure ulcers (injuries).[168] Recommendations for prevention included the following:

- Implement measures to reduce the risk of developing pressure ulcers: minimize/eliminate pressure, friction, and shear.
- Minimize/eliminate pressure from medical devices such as oxygen tubing, catheters, cervical collars, casts, and restraints.
- Maintain the head-of-bed elevation at/or below 30°, or at the lowest degree of elevation consistent with the patient's medical condition to prevent shear-related injury, and use a 30° side-lying position.
- Schedule regular repositioning and turning for bedbound and chairbound individuals, taking into consideration the condition of the patient and the pressure redistribution support surface in determining the repositioning strategy.
- Position sitting patients with special attention to the individual's anatomy, postural alignment, distribution of weight, and support of the feet.
- Consider prophylactic dressings to prevent sacral and heel ulcers in at-risk patients.
- Use heel suspension devices for patients who are at risk for pressure ulcers that elevate (float) and offload the heel completely, and redistribute the weight of the leg along the calf without putting pressure on the Achilles tendon.
- Utilize support surfaces (on beds and chairs) to redistribute pressure. Pressure redistribution devices should serve as adjuncts and not replacements for repositioning protocols.
- Place individuals who are at risk for pressure ulcers on a pressure redistribution surface.
- Consider using the WOCN Evidence- and Consensus-Based Support Surface Algorithm (http://algorithm.wocn.org) to identify the appropriate support surface (overlay, mattress, or integrated bed system) for adults (≥16 years) and bariatric patients in care settings where the length of stay is 24 hours or more.

- Use a high-specification reactive or alternating pressure support surface in the operating room for individuals at high risk for developing pressure ulcers.
- Avoid foam rings, foam cut-outs, or donut-type devices for pressure redistribution because they concentrate pressure on the surrounding tissue.
- Use incontinence skin barriers such as creams, ointments, pastes, and film-forming skin protectants as needed to protect and maintain intact skin in individuals who are incontinent and at risk for pressure ulcers.
- Offer individuals with nutritional and pressure ulcer risks a minimum of 30-35 kcal/kg body weight per day, 1.25-1.5 g of protein/kg body weight per day, and 1 ml of fluid intake/kcal per day.
- Educate the patient/caregiver(s) about the causes and risk factors for developing pressure ulcers and ways to minimize the risk.

Recommendations for management included the following:

- Float/elevate the heel(s) completely off the surface with a pillow or heel suspension device for stage 1 and 2 pressure ulcers or a heel suspension device for stage 3 and 4 heel pressure ulcers.
- Turn and reposition the patient regularly and frequently.
- Utilize support surfaces for patients with pressure ulcers (i.e., mattresses, mattress overlays, integrated bed systems, seat cushions or seat cushion overlays) that meet the individual's needs, and are compatible with the care setting.
- Consider using the WOCN Society's Evidence-and Consensus-Based Support Surface Algorithm (http://algorithm.wocn.org) to identify the appropriate support surface for adults (≥16 years) and bariatric patients in care settings where the length of stay is 24 hours or more.
- Utilize seating redistribution support surfaces that meet the needs of sitting individuals who have a pressure ulcer.
- Establish an individualized bowel/bladder management program for the patient with incontinence.
- Screen for nutritional deficiencies at the patient's admission to the care setting, when their condition changes, and/or if the pressure ulcer is not healing.
- Provide daily calorie and protein intake for adult patients with pressure ulcers: 30-35 kcal/kg and protein 1.25-1.5 g/kg.
- Consider evaluation of laboratory tests such as albumin and prealbumin as only one part of the ongoing assessment of nutritional status.
- Cleanse the wound and peri wound at each dressing change, minimizing trauma to the wound.
- Choose appropriate solutions for cleaning pressure ulcers, which may include potable tap water, distilled water, cooled boiled water, or saline/salt water.
- Determine the bacterial bioburden by tissue biopsy or Levine quantitative swab technique.
- Consider a 2-week course of topical antibiotics for nonhealing, clean pressure ulcers.
- Consider use of antiseptics for "maintenance wounds," which are defined as wounds that are not expected to heal, or for wounds that are critically colonized.
- Use systemic antibiotics in the presence of bacteraemia, sepsis, advancing cellulitis, or osteomyelitis.
- Debride the pressure ulcer of devitalized tissue, or when there is a high index of suspicion that biofilm is present (i.e., wound fails to heal despite proper wound care and antimicrobial therapy), and when consistent with the patient's condition and goals of therapy.

- Modify the type of dressing as appropriate due to changes in the wound during healing or if the pressure ulcer deteriorates. Monitor and assess the wound on a regular basis and at every dressing change to determine if the type of dressing is appropriate or should be modified.
- Consider adjunctive therapies as indicated: platelet-derived growth factor (PDGF); electrical stimulation; negative-pressure wound therapy (NPWT).
- Evaluate the need for operative repair for patients with stage 3 and 4 ulcers that do not respond to conservative medical therapy.
- Implement measures to eliminate or control the source of pressure ulcer pain.
- Implement appropriate treatment of pressure ulcers to optimize healing, recognizing that complete healing may be unrealistic in some patients.
- Educate the patient/caregiver(s) about strategies to prevent pressure ulcers, promote healing, and prevent recurrences of ulcers; and emphasize these are lifelong interventions.

Medication

Medication Summary

Relief of spasticity (if present) is essential in the treatment and prevention of pressure ulceration. The medications most commonly employed for this purpose are muscle relaxants.

Skeletal Muscle Relaxants (Centrally Acting)

Class Summary

Centrally acting skeletal muscle relaxants inhibit reflexes at the spinal level.

Baclofen (Lioresal, Gablofen)

Baclofen may induce hyperpolarization of afferent terminals and inhibit both monosynaptic and polysynaptic reflexes at the spinal level.

Diazepam (Valium, Diastat)

Diazepam depresses all levels of the central nervous system (CNS), including the limbic and reticular formations, possibly by increasing the activity of gamma-aminobutyric acid (GABA), which is a major inhibitory neurotransmitter. To prevent adverse effects, the dosage should be individualized and increased cautiously.

Skeletal Muscle Relaxants (Direct Acting)

Class Summary

Direct-acting skeletal muscle relaxants inhibit muscle contraction by decreasing calcium release from the sarcoplasmic reticulum in muscle cells.

Dantrolene (Dantrium, Revonto)

Dantrolene stimulates muscle relaxation by modulating the skeletal muscle contractions at a site beyond the myoneural junction and by acting directly on the muscle itself. Most patients respond to a dosage of 400 mg/day or less.

Antibiotics

Class Summary

Empiric antibiotics should be started immediately. Initial antimicrobial therapy should be broad-based, to cover aerobic gram-positive and gram-negative organisms and anaerobes.

Silver sulfadiazine (Silvadene, SSD, Thermazene)

Silver sulfadiazine has broad-spectrum antibacterial activity and is associated with relatively few complications in these wounds.

The current formulation contains a lipid-soluble carrier, polypropylene glycol, which has certain disadvantages, including pseudoeschar formation. When this antibacterial agent is formulated with poloxamer 188, the silver sulfadiazine can be washed easily from the wound because of its water solubility, making dressing changes considerably more comfortable.

Mafenide (Sulfamylon)

Mafenide is an alternate agent that penetrates eschar more effectively than silver sulfadiazine. Consequently, it is frequently used on infected wounds that do not respond to silver sulfadiazine. Use mafenide with caution because it can induce metabolic acidosis.

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