Frequently found on the sacrum, pressure ulcers develop due to prolonged periods of unrelieved pressure on soft tissues, but can occur anywhere there is pressure, including trochanters and especially heels. In the bedridden patient, constant pressure causes ischemia and necrosis of subcutaneous tissues and skin. Most patients are elderly, immobile and have neurologic impairments, often associated with inability to sense pain and discomfort and/or incontinence. Sacral ulcers can be treated with debridement, dressings and skin grafts. However, preventive efforts—including a regular turning schedule, proper assessments, moisturizers and adequate diet—are the most cost-effective and remain the foundation of management. Pressure ulcers can occur anywhere there is pressure, including trochanters and, especially, heels.

Key words: pressure ulcer, debridement, sacrum, risk factors, wound healing.

Introduction

Pressure ulcers develop under conditions of prolonged pressure and circulatory stasis, which damage the involved tissue by ischemia and necrosis. Typically seen in patients who are temporarily immobilized, these ulcers develop in areas of bony protuberances such as the sacrum, trochanters and heels, where pressure is concentrated on subcutaneous tissues and skin adjacent to the bone. The three major groups of patients at risk are the young and neurologically impaired, older adults and those who are hospitalized.

The prevalence of pressure ulcers ranges from 3–30% in hospitalized and long-term care patients. The acute-care setting varies in incidence from 3–5% on the general ward/patient room/care setting to 15% among those patients in the intensive care setting whose surgery or disease status may delay or prevent movement.

Pressure ulcers have been associated with increased morbidity and mortality rates in both acute and long-term care settings. One study demonstrated that patients who develop a new pressure ulcer within six weeks after hospitalization are three times as likely to die within a year as those who do not develop a pressure ulcer. Pressure ulcer development did not remain independently associated with decreased survival after adjusting for other predictors of mortality such as weight loss and hospital complications. The authors further reported that 67% of patients who developed a pressure ulcer during acute hospitalization died, compared to 15% of patients who did not have an ulcer but who were similarly at risk for primary disease (e.g., myocardial infarct, stroke, fractured hip, neurosurgery).

The average cost of treating one pressure ulcer is approximately $30,000; annual costs of treating pressure ulcers in the U.S. approach $8.5 billion. Pressure ulcers are categorized as chronic wounds. Acute wounds, the cause of which is usually a known and single event, tend to heal quickly and well, whereas chronic wounds heal slowly or do not heal at all. Four stages are used to describe the development of pressure ulcers. Stage I is nonblanchable erythema of intact skin. Although difficult to detect (especially in patients with dark skin), these changes usually do not advance to more serious lesions unless the precipitating event persists. Stage II ulcers involve the epidermis or dermis. The superficial skin is macerated or blistered. Stage III ulcers consist of full-thickness skin loss involving the subcutaneous fat, but not extending through the underlying fascia. Stage IV ulcers extend to muscle, bone or cartilage with extensive destruction of tissue and full-thickness skin loss.

These stages are not designed to evaluate a change in the status of a wound, but rather are steps in a process and describe the amount of tissue destroyed. The process is not reversible. The healed wound is more susceptible to breakdown than uninjured tissue.
Assessing wound depth (stage) is a reflection of experience rather than a result of some measurement. In the darkly pigmented individual, it may be difficult to distinguish red or purple color change (Stage I), such that partial thickness skin loss involving the dermis and epidermis (Stage II) will develop seemingly quickly.

**Normal Wound Healing and Assessment**

Wounds heal by primary or secondary intention, depending on the nature of the wound. A classic example of primary healing is a surgical incision with no tissue loss, for which healing begins almost immediately. The narrow incisional space is filled with clotted blood which forms a scab upon dehydration, followed by inflammation and wound closure by epithelialization and collagen deposition. Secondary healing occurs when loss of tissue and cells is extensive and wound edges remain open, such as in pressure ulcers. The repair process is more complicated because the wound must be filled. After inflammation has begun, healing occurs by deposition of tissue into the open space. This granulation tissue is rich in capillaries, fibroblasts and inflammatory cells. The feature that differentiates primary from secondary healing is wound contraction. Large wounds are reduced to 5–10% of their original size as the result of contraction caused by myofibroblasts—altered fibroblasts with the ultra-structure similar to smooth muscle cells.

Surface area and volume of a chronic wound, such as a pressure ulcer, are clinical measurements recommended for monitoring wound healing. Although a simple ruler can be used to take these measurements, new developments are more precise and reliable. The use of enhanced digital photography and ultrasound scanners are sometimes used in the acute care setting (nursing homes rarely have this equipment). Enhanced digital photography provides accurate measurements by way of the system’s digital subpixel measuring techniques. Enhanced digital photography is cost effective, in part because the system uses natural light to capture the real image of the wound so does not require special lighting. Ultrasound scanners, such as Longport Inc. Soft Tissue Digital Ultrasound Scanner, capture and reproduce images of soft tissue at high resolution, generating accurate measurements on a computer. These new technological developments provide quantitative information on wound healing and add reliability to clinical research methodology. The equipment does not, however, compensate for appropriate care and is not necessary for accurate clinical assessment by the primary care physician. The use of digital photography and ultrasound scans are not practical for most primary care physicians; measuring length, width and depth is sufficient.

**Etiology and Pathophysiology**

An inverse relationship between time and pressure has been established in the pathogenesis of pressure ulcers: the more pressure applied over tissue, the less time it takes for skin ulceration to occur.12 The multitude of risk factors for pressure ulcers (e.g., diabetes, nutrition, paralysis) decrease the time required to develop a pressure ulcer.

Pressure ulcers are most common in the critically ill. Many patients admitted to Critical Care Units have reduced sensory perception and mobility, either from their condition or the medications they are taking. These deficits lead to prolonged periods of unrelieved pressure. Neurologically impaired patients also are not able to sense and respond meaningfully to pain and discomfort, facilitating conditions of unrelieved pressure and soft tissue ischemia and necrosis, common in the area of the sacrum. Moreover, critically ill patients often have hypotension, sepsis or low cardiac output. Such factors compromise soft tissue perfusion, thereby increasing the risk of pressure ulcers. The increased prevalence in critically ill patients in the acute hospital setting may be due to medical staff focusing on more life-threatening issues, which may preclude them from noticing the pressure ulcers. There are limitations to moving/turning some patients because of endotracheal tubes and ventilators, Foley catheters, gastric tubes and unstable cardiac status. The real issue, however, is attitude and education—patients can be turned if the staff want to do it.2

The sacrum, composed of five fused sacral vertebrae, provides strength and stability to the pelvis and transmits body weight to the pelvic girdle. The posterior surface of the sacrum is rough and convex; therefore, with constant pressure from the weight of the body against a hard bed underneath, the soft tissue in between begins to degrade due to ischemia and necrosis. This degradation is facilitated by the constant presence of moisture (e.g., from bouts of fecal and urinary incontinence or febrile episodes with diaphoresis), as well as from friction and shear, fatigue and decreased muscle strength.2

Shear, defined as the force that causes an opposite parallel sliding motion in the planes of the interested object,1 is a major contributor to the development of sacral pressure ulcers. In hospital settings, shear becomes a critical factor when the head of the bed is elevated. Increased pressure on sacral tissues is generated as the skeletal frames slide down toward the foot of the bed while sacral skin is held in place by friction on the bed sheets. The muscle and deeper fascia slide with the bone, but the superficial fascia remains with the dermis and epidermis of the sacrum, thereby stretching arteries supplying the skin from the deep fascia and muscles. This stretch decreases the pressure needed to stop the blood flow significantly and leads to ischemia and necrosis.1 Friction abrades the epidermis and, associated with shear, promotes widening of a pressure ulcer.

Constant moisture due to fecal or urinary incontinence or sweat also facilitates development of pressure ulcers of the sacrum.1,2 Moisture softens the skin and reduces its tensile strength. With such changes, the skin can be macerated easily by compression and is highly susceptible to erosion by friction. Fecal incontinence also can be a major source of infection in sacral ulcers.13
Pressure and Other Risk Factors

Although pressure is the principle determinant for development of sacral ulcers, underlying conditions can be important contributors. Disorders associated with pressure ulcers include diabetes mellitus, malnutrition and immune deficiencies.

Diabetes

In diabetic patients, a high blood glucose concentration may impair wound healing by altering the inflammatory response, fibroblast proliferation and collagen deposition. Tensile strength of the wounds is reduced. Diabetic patients also are more susceptible to pressure ulcers because of less efficient granulocyte phagocytosis and chemotaxis. In addition, increased incidences of atherosclerosis and microvessel disease in diabetes contribute to ischemia. Therefore, diabetic patients are not only at increased risk for developing ischemic ulcers, but also have impaired ability to heal wounds because of poor blood supply and less resistance to infection. Good control of blood glucose is the cornerstone of treatment.

Malnutrition

Malnutrition contributes to pressure ulcers in several ways. Malnourished patients are more likely to be immobile, have soilage from fistulas or incontinence, and are emaciated, with bony surfaces protruding on the skin. Normal cell division requires adequate protein supply, and wound healing requires an even greater protein supply. Low serum albumin levels are associated with pressure ulcers. Albumin is an essential building block in the wound healing process. In addition, hypoalbuminemia causes edema, which impedes cellular exchange of nutrients and waste products and contributes to microcirculatory ischemia. Fats are necessary for the development of cell membranes; without an adequate supply of proteins and fats, cell division at the site of pressure ulcers is impaired, which delays the healing process.

Vitamins and minerals are essential for normal cellular functions, and their requirements increase during wound healing. As an adequate supply of vitamin A is required for proper cell differentiation, this vitamin is required during epithelialization and cell-mediated immune responses. Several B vitamins such as riboflavin, pyridoxine and thiamine are cofactors in collagen cross-linking reactions. Deficiency of these vitamins leads to abnormal collagen synthesis that delays wound repair. Vitamin C promotes hydroxylation of major amino acids, lysine and proline, all of which are essential for collagen synthesis. Therefore, vitamin C deficiency can lead to inadequate production of collagen. Vitamin K has a central role in the synthesis of prothrombin and clotting factors, and its deficiency leads to excessive bleeding from wounds, which impairs healing. No evidence exists that any single vitamin or mineral improves healing in vivo; the best evidence points to protein and total calorie intake.

The minerals zinc and iron also impact proper healing. Zinc deficiency can cause delayed wound healing because it is an important cofactor in the synthesis of DNA and proteins, and for mitosis and cellular proliferation. Epithelialization and fibroblast proliferation are inhibited by a deficiency of zinc. Iron deficiency causes anemia, which contributes to an inadequate supply of oxygen to wound sites and delays healing.

Age

As the prevalence of disease tends to increase with age, older persons develop tissue ischemia more easily than younger persons and are more susceptible to shear while sitting. Aged skin is atrophic, and changes in collagen synthesis result in decreased strength and increased stiffness. Vascularity also is reduced. These factors render aged skin more vulnerable to ulceration. Older persons also tend to be thinner, which can result in higher pressures over bony prominences than over average-weight patients.

Oxygen

Lack of oxygen is a central factor in the development of pressure ulcers, and adequate oxygen delivery to the wound site is critical. Wounds in richly vascularized sites heal faster. A hypoxic state causes insufficiency of collagen production because oxygen is required for hydroxylation—an essential step in the production of collagen. Oxygen also is required for energy-dependent metabolic processes, oxygen radical production to combat bacteria, fibroblast proliferation and epithelialization.

Oxygen radicals are necessary to kill bacteria; therefore, hypoxic conditions facilitate infection. Bacteria not only deprive the healing tissue of nutrients, but also produce exo- and endotoxins that may damage newly generated as well as mature cells. Soft tissue infections prolong inflammation, further reducing delivery of oxygen to the wound site. This vicious cycle is another reason that pressure ulcers are more frequent in immunocompromised patients and older adults.

Table 1: Treatment Options for Pressure Ulcers

<table>
<thead>
<tr>
<th>Treatment Options for Pressure Ulcers</th>
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<tbody>
<tr>
<td>Keeping intact skin dry, maintain adequate moisture in wounds</td>
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<tr>
<td>Pressure relief</td>
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<tr>
<td>Control of urinary and fecal incontinence</td>
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<tr>
<td>Frequent repositioning</td>
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<tr>
<td>Debridement of necrotic tissue, e.g., autolysis, chemical or sharp</td>
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<tr>
<td>Proper cleansing of wound</td>
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<tr>
<td>Application of dehydrating dressings as needed</td>
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<tr>
<td>Application of growth factors</td>
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<tr>
<td>Skin grafting</td>
</tr>
<tr>
<td>Optimization of nutrition, e.g., vitamins A, B, C and K, zinc and iron</td>
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</tbody>
</table>

Skin grafting
Pressure Ulcers

Treatment
Pressure ulcer size is the most important predictor of healing; stage II ulcers are the most likely to heal, while stage IV ulcers are the least likely. Any treatment of ulcers includes keeping intact skin dry and providing wounds with enough moisture, pressure relief, debridements, dressings and skin grafts. Incontinence and immobility are important factors in delaying the healing of pressure ulcers. Urinary and fecal soiling must be controlled to avoid bacterial colonization and infection. Incontinence can be controlled by an indwelling Foley catheter or intermittent catheterization. Frequent repositioning is essential to prevent sustained pressure over the wound site.

Proper treatment of sacral pressure ulcers may require debridement of necrotic tissue, cleansing of the wound and application of appropriate dressings (Table 1). Debridement is a naturally occurring process in wound repair; however, when pressure persists, an accumulation of dead tissue occurs, increasing the risk of infections.

Types of debridement procedures include autolysis, chemical or sharp. Autolysis promotes sloughing off of necrotic tissue. A hydrocolloid dressing promotes autolysis by hyperhydration, which helps in the sloughing of dead tissue in a pressure ulcer. Chemical debridement uses enzymes, such as collagenase, accuzyme, fibrinolysin or trypsin, which dissolve the collagen holding the necrotic cells to the soft tissue bed. Sharp debridement involves the surgical removal of necrotic tissue. This may be the most effective method of debridement, as well as the highest risk for sepsis and bleeding.

A drastic yet effective treatment of sacral pressure ulcers is grafting of a skin flap. Use of a muscle flap is usually more effective because it is better vascularized, more resistant to pressure and more metabolically active than skin. Various skin grafts have been developed. These grafts are bioengineered tissues made from human keratinocytes, fibroblasts and type I bovine collagen. Any treatment, however, can only be effective when the patient is on a turning schedule to relieve pressure on the ulcer. Surgical intervention for the sacral ulcer usually requires an appropriately trained and interested general or plastic/reconstructive surgeon. Surgical intervention can be considered for stage III or IV ulcers.

General care of pressure ulcers includes treatment of the underlying disease. Therefore, controlling the blood sugar of diabetic patients, improving nutrition through diet and administering antibiotics for infection are paramount toward healing of pressure ulcers.

Prevention
Ulcers may be overlooked until they become a major problem. Pressure ulcers on heels often lead to amputations, while pressure ulcers on the sacrum can lead to death. Major preventive measures include identification of high-risk patients, frequent assessment, regular repositioning, pressure-relief bedding, moisture barriers and adequate diet (Table 2). Risk assessment scales such as the Braden Scale should be used to identify patients at risk for pressure sores. Patients with pressure ulcers have significantly lower total Braden Scale scores: a score of 16 or 17 represents a mild risk of developing pressure ulcers, and a score below 16 indicates moderate to high risk.

Not only total scores, but the scores for each component (moisture, activity, mobility, sensory perception, nutrition, and friction/shear) were considerably lower in patients with pressure ulcers. A risk assessment tool such as the Braden Scale should be used at baseline, but also at regular intervals thereafter.

Skin should be assessed periodically for ulcers. Regular use of lotions is encouraged. Lotions have the highest water content of all moisturizers but evaporate rapidly and so must be applied frequently. The goal is to keep the skin well lubricated without over-saturating the epidermis. Products that have fragrances or skin sensitizers should not be used (avoid neomycin, lanolin and bacitracin). Pressure-relieving devices, along with a repositioning schedule and cushioning, are important preventive measures. A two-hour turning schedule, keeping in mind individual tolerance to pressure, is widely accepted and used in medical and nursing practices. Pressure also can be relieved using special mattresses that alternate periods of high and low pressure. Low-air-loss beds, waterbeds and air-fluidized beds all take the pressure away from the bony prominences. However, these mattresses and oscillatory beds are costly and not usually available for all patients at risk.

The best method of pressure ulcer prevention remains the identification of high-risk conditions. High-risk patients should be started on a regular turning schedule and have frequent assessments. Areas at risk should be kept dry while applying lotions periodically. All these measures should be implemented in conjunction with an adequate diet and general medical care.

Table 2: Major Preventative Measures for Pressure Ulcers

<table>
<thead>
<tr>
<th>Identification of high-risk patients</th>
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<tr>
<td>Risk assessment scales, e.g., Braden Scale</td>
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<tr>
<td>Frequent assessment</td>
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<td>Regular repositioning</td>
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<td>Pressure-relief bedding</td>
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<td>Cushioning, special mattresses</td>
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<td>Moisture barriers</td>
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<td>Regular use of moisturizing lotions</td>
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<td>Adequate diet</td>
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