Pressure Ulcers present a significant health care threat to patients with restricted mobility or chronic disease and to older patients. Because of this threat, more documents about pressure ulcers are being published, such as the International Pressure Ulcer Guidelines for Prevention and Treatment (NPUAP and EPUAP, 2009), the Wound, Ostomy and Continence Nurses (WOCN) Society Guidelines for Prevention and Management of Pressure Ulcers (WOCN Society, 2003, 2010), the Registered Nurses Association of Ontario (RNAO) Risk Assessment and Prevention of Pressure Ulcers Guideline (RNAO, 2005), “Guidelines for the prevention of pressure ulcers” by the Wound Healing Society (Stechmiller et al, 2008), the Canadian Association of Wound Care “Best practice recommendations for the prevention and treatment of pressure ulcers: update 2006” (Keast et al, 2007), and Healthy People 2010 (U.S. Department of Health and Human Services, 2000). The Institute for Healthcare Improvement identified pressure ulcers as one of its primary goals in the “Save 5 Million Lives” campaign (Padula et al, 2008). The Cochrane Wounds Group also covers the prevention and treatment of pressure ulcers (Bell-Syer et al, 2007).

SCOPe OF THE PROBLEM

The scope of the pressure ulcer problem in the United States is examined in terms of the patient’s age and diagnosis and the setting. Statistics about pressure ulcers vary because of how data were collected, variations in terminology about prevalence and incidence, concern about litigation, and political and social events that changed American health care.

Prevalence

The WOCN Society (2004) defines pressure ulcer prevalence as the number of patients with at least one pressure ulcer who exist in a given patient population at a given point in time. In the United States, the 6-year sequential analysis of pressure ulcer prevalence ranges from 14% to 17% (Whittington and Briones, 2004). The prevalence of pressure ulcers in long-term care has been reported as 27.3%, with 8.5% being nosocomial (VanGilder et al, 2008; Whitney et al, 2008). The prevalence of pressure ulcers in home care ranges from 3% to 10% (Bolton et al, 2008). The 2003 National Pediatric Pressure Ucer and Skin Breakdown Prevalence Survey found a pressure ulcer prevalence of 4%; 92% of pressure ulcers were partial-thickness ulcers and 66% were facility acquired (McLane et al, 2004). The discrepancies in prevalence can be attributed to the fact that some studies include intact pressure-damaged skin (suspected deep tissue injury, or Stage 1), whereas other studies exclude such lesions. Prevalence is lower when intact pressure-damaged
skin is excluded from the sample. Pressure ulcers in dark-skinned persons may also be difficult to detect (Black et al, 2007). Some skin conditions, such as candidiasis and herpetic lesions, may be misclassified as pressure ulcers. In infants and children, the diagnosis of a pressure ulcer is carefully considered because the most common types of skin breakdown in this group include diaper dermatitis, skin tears, and intravenous extravasation (McLane et al, 2004). Data collectors must accurately distinguish between pressure ulcers and other causes of erythema and skin ulcerations.

**Incidence**

The WOCN Society (2004) defines incidence as the number of patients who initially were ulcer-free who develop a pressure ulcer within a particular time period in a defined population. Incidence measures new conditions (e.g., pressure ulcers) and therefore is considered more reflective of the quality of care within that setting. It is a measure used to evaluate the effects of preventive and therapeutic interventions. Determining the incidence of pressure ulcers is inherently difficult because such studies require longitudinal observations. As with prevalence, incidence will vary by setting. The incidence of pressure ulcers in acute care ranges from 7% to 9% (Whittington and Briones, 2004). The incidence in long-term care ranges from 3% to 31% (Shukla et al, 2008) and in home care ranges from 0% to 17% (NPUAP, 2001).

Considerable methodologic issues surround the calculation of incidence. For example, defining who is at risk (the number used in the denominator of the incidence formula) can have a significant influence on the resulting value, which actually may overestimate or underestimate the true frequency of the condition. Consequently, variation in reports of incidence may reflect a real difference in the frequency of the condition or simply different data collection techniques, definitions, and methods. Although differences in methodology make comparisons difficult, incidence remains an important measure. Consistency in data collection technique within the health care setting is essential to generate data that can be compared over time. National standards for the definition of terms and the process for conducting prevalence and incidence studies will also increase the comparability of this kind of data (WOCN Society, 2004). The National Database of Nursing Quality Indicators (NDNQI) includes pressure ulcer prevention and hospital-acquired pressure ulcer reports from more than 1,100 facilities in the United States designed for comparisons between hospitals of similar sizes and practice levels (Montalvo, 2007).

**Economic Effects**

In fiscal year 2007, a preventable pressure ulcer was listed as a secondary diagnosis for 257,412 Medicare patients. The average payment for admission in which a pressure ulcer was present was $43,180 (Armstrong et al, 2008). In 2008, the Centers for Medicare and Medicaid Services (CMS) ceased payment for hospital complications considered reasonably preventable, including Stage III or IV pressure ulcers. CMS with the Centers for Disease Control and Prevention (CDC) released new codes for pressure ulcers capturing wound severity (Krapfl, 2008).

Research on the costs incurred while a pressure ulcer is being managed must be viewed cautiously; the studies are not all comparable. Some studies account for all costs: room, nursing care, supplies, medications, physician fees, and so forth. Other studies examine only direct costs, such as the supplies or medications specifically indicated for that particular problem.

Facility-associated pressure ulcers add to the patient’s length of stay, delay the patient’s recuperation, and increase the patient’s risk for developing complications. In addition, pressure ulcers often necessitate hospitalization (in certain patient populations such as the elderly and patients with a spinal cord injury) because of sepsis or the need for debridement or surgical repair. At a time of increasingly scarce health care dollars, pressure ulcers consume intense resources in the form of dressing changes, nursing care, physical therapy, medications, nutritional support, and clinician services.

Literature reports a range of costs for pressure ulcer management. Approximately, 2.5 million patients are treated each year in U.S. acute care facilities for pressure ulcers, and the cost of treating pressure ulcers is estimated at $11 to $17.2 billion annually (Ayello and Lyder, 2008; Bolton et al, 2008). Pressure ulcers lead to loss of function, infection, and extended hospital stays, all of which can increase cost. Hospital length of stay for a principal pressure ulcer diagnosis was 14.1 days compared to 12.7 days for a secondary pressure ulcer diagnosis. The average cost per hospital day for a principal pressure ulcer diagnosis was $1,200 compared to $1,600 for a secondary pressure ulcer diagnosis. Three of four hospitalizations with a pressure ulcer diagnosis were billed to Medicare; Medicaid patients accounted for additional 12.5% of hospitalizations with a principal pressure ulcer. More than half of patients with pressure ulcer stays were discharged to long-term care, which is more than three times the rate of hospitalizations for all other causes (Russo et al, 2008).

**Impact on Quality and Duration of Life**

Pressure ulcers may affect psychosocial needs and quality of life in terms of occurrence, recurrence, ulcer characteristics, and ulcer demands. Pressure ulcers may cause social isolation and add burden and frustration for the patient, the family, and care providers. Aspects of quality of life include change in body image, pain, odor and drainage, and financial impact (Langemo, 2005). Quality-of-life research studies about persons with pressure ulcers...
tend to have small sample sizes, so additional research is needed. Important factors to assess are the patient’s social networks, the patient’s living space and environment, and the patient’s mental status, learning needs, and personal goals. Pain is an ever-present problem with pressure ulcers and must be assessed (Pieper et al, 2009).

Pressure ulcers have been examined in terms of their effects on mortality rates. In 2006, hospital mortality among persons with a secondary diagnosis of a pressure ulcer was 11.6% and was 4.2% among those with a pressure ulcer as a principal diagnosis (Russo et al, 2008). Approximately 60,000 patients die each year from pressure ulcer complications (Ayello and Lyder, 2008). Pressure ulcers were reported as a cause of death among 114,380 persons (1990–2001), and the age-adjusted mortality rate was 3.79 per 100,000 population. Pressure ulcers deaths occurred mostly in persons at least 75 years old, and sepsis was reported in 39.7%. Mortality rates were higher in African Americans than in persons of other racial/ethnic groups (Redelings et al, 2005). In a home care project in Italy, residents with a pressure ulcer had a relative risk of dying of 1.92 after adjusting for age, gender, and all significant variables between the two groups of patients (Landi et al, 2007).

**VULNERABLE PATIENT POPULATIONS**

A variety of specific patient populations have been discussed in the literature as being at increased risk for pressure ulcer formation: older adults, persons with spinal cord injury, surgical patients, obese patients, underweight patients, children, and patients at end of life. These particular patient populations are introduced here and discussed in greater detail throughout the text.

Historically, older adults admitted to acute and long-term care facilities have been a vulnerable population. Nearly 3 (72%) of 4 older adult patients hospitalized with a secondary pressure ulcer diagnosis and 56.5% of adults with a principal diagnosis of a pressure ulcer were 65 years of age or older (Russo et al, 2008). Among persons admitted to long-term care, 10.3% to 18.4% had one or more pressure ulcers on admission (Baumgarten et al, 2003; Siem et al, 2003). The presence of an existing pressure ulcer at the time of admission to acute care was 26.2% among persons admitted from a nursing home and 4.8% among those admitted from another living situation (Keelaghan et al, 2008). For older adults with a pressure ulcer, coexisting conditions were fluid and electrolyte disorders, nutritional disorders, diabetes mellitus without complications, and dementia (Russo et al, 2008).

Pressure ulcer prevalence for persons with spinal cord injuries ranges from 20% to 66% (Schubart et al, 2008). Paralysis and spinal cord injury were common coexisting conditions among younger adults hospitalized principally for pressure ulcers (Russo et al, 2008). Those with the greatest level of disability and mobility impairment have the highest pressure ulcer risk. Other risk factors are history of pressure ulcers, coexisting medical conditions, rehospitalizations, nursing home stays, less than high school education, older adult, male, African American, and single (Schubart et al, 2008).

In a study involving 37 facilities, the incidence of pressure ulcers related to the surgical event was 3.5% (Aronovitch, 2007). Associated factors were at least one comorbidity, managed with a warming device, receipt of three or more anesthetic agents, and median operative time of 4.48 hours (Aronovitch, 2007). Tissue damage may become apparent within hours or may be delayed for up to 3 days. Initial manifestations may be skin discoloration (e.g., bruising) that evolves into blister formation or necrosis. Because this process transpires over several days (i.e., 2–6 days), isolating the time of the original injury is complicated (Price et al, 2005). Because the length of surgery and other variables for the surgical patient cannot be changed, the surgical team must aim to decrease pressure and shear not only during the procedure but also when transferring the patient into position before and after the procedure (Schoonhoven et al, 2002).

Both the obese and the underweight patient populations are vulnerable to pressure ulcer development. The morbidly obese are at risk for pressure ulcers due to their inability to turn themselves, underlying diseases, improper equipment, lack of adequate pressure redistribution, inadequate staff numbers, or staff not trained in how to turn and move such patients (Knudsen and Gallagher, 2003; Mathison, 2003). In a study of elderly patients admitted to acute care, the odds of developing a pressure ulcer in patients who were obese and those who were severely obese were very low (odds ratio 0.7 and 0.1, respectively). In contrast, the odds of the underweight patient developing a pressure ulcer was almost doubled (odds ratio 1.8) (Compfer et al, 2007).

Two additional vulnerable patient populations are children and patients at end of life.

**TERMINOLOGY**

Over the years, several terms have been used to describe pressure ulcers: bedsore, decubitus ulcer, decubiti, and pressure sore. Pressure ulcer is the accepted term because it is more accurate and descriptive. The origin of the term bedsore is not known, but it predates the term decubitus. Decubitus, a Latin word referring to the reclining position (Fox and Bradley, 1803), dates from 1747 when the French used it to mean bedsore. However, this term is inaccurate because it does not convey the tissue destruction associated with these lesions and because these lesions result from positions other than the lying position (such as sitting) (Arnold, 1983).
A pressure ulcer is defined as localized injury to the skin and/or underlying tissue usually over a bony prominence as a result of pressure or of pressure in combination with shear and/or friction. A number of contributing or confounding factors are associated with pressure ulcers, but the significance of these factors has not yet been elucidated (NPUAP, 2007).

Pressure ulcers occur most commonly over a bony prominence, such as the sacrum, ischial tuberosity, trochanter, and calcaneus; however, they may develop anywhere on the body (e.g., underneath a cast, splint, or cervical collar). Figure 1 shows common sites for pressure ulcers and frequency of ulceration per site. The majority of pressure ulcers occur in the pelvis, but other more common locations are the sacrum and the heels (VanGilder et al, 2008).

Bony locations are most prone to pressure ulcer formation because a person’s body weight is concentrated on these areas when resting on an unyielding surface. Those who have atrophy of the subcutaneous and muscle tissue layers are at even greater risk for the “mechanical load” of pressure and thus increased soft tissue and capillary compression. The coccyx, sacrum, and heel are particularly vulnerable because less soft tissue is present between the bone and skin.

CAUSATIVE FACTORS

Pressure is the major causative factor in pressure ulcer formation. However, several factors play a role in determining whether pressure is sufficient to create tissue ischemia proceeding to tissue death. The pathologic effect of excessive pressure on soft tissue can be attributed to (1) intensity of pressure, (2) duration of pressure, and (3) tissue tolerance (ability of skin and its supporting structures to endure pressure without adverse sequelae). Braden and Bergstrom (1987) presented a model of the factors that contribute to the intensity and duration of pressure ulcers (Figure 2), in combination with intrinsic and extrinsic factors that affect tissue tolerance.

Intensity of Pressure

To understand the importance of intensity of pressure, it is important to review the terms capillary pressure and capillary closing pressure. Capillary pressure tends to move fluid outward through the capillary membrane. Exact capillary pressure is not known because of the difficulty of obtaining the measurement. Various methods have been used to estimate capillary pressure. A normal hydrostatic pressure is approximately 32 mm Hg at the arterial end of a capillary bed and 12 mm Hg at the venous end (Figure 7-3) (Kumar et al, 2005a). The mean colloidal osmotic pressure in tissue is approximately 25 mm Hg.

The term capillary closing pressure, or critical closing pressure, describes the minimal amount of pressure required to collapse a capillary (Burton and Yamada, 1951). Tissue anoxia develops when externally applied pressure causes vessels to collapse. It is believed that the amount of pressure required to collapse capillaries must exceed capillary pressure, which is considered to be 12 to 32 mm Hg, the numerical “standard” for capillary closing pressure.

To quantify the intensity of pressure being applied externally to the skin, interface pressures are measured. Numerous studies measuring interface pressures have
of a healthy adult male in the supine, prone, side-lying, and sitting positions. Interface pressures ranged from 10 to 100 mm Hg. Interface readings as high as 300 mm Hg have been obtained over the ischial tuberosity of healthy, able-bodied male subjects when sitting in an unpadded chair (Kosiak, 1961).

Because head of bed elevation is important for mechanically ventilated patients in critical care and head of bed elevation greater than 30 degrees exposes the patient to increased shear injury and unrelieved pressure, Peterson et al. (2008) examined sacral interface pressures at elevations of 0, 10, 20, 30, 45, 60, and 75 degrees in 15 healthy subjects. The elevations 30 degrees or greater had peak interface pressures significantly higher than supine. In addition, elevations 45 degrees or higher had bed interface pressures greater than 32 mm Hg (Peterson et al, 2008).

Interface pressures in excess of capillary pressure will not routinely result in ischemia. Healthy people with normal sensation regularly shift their weight in response to the discomfort associated with capillary closure and tissue hypoxia. Unfortunately, pathologic processes such as spinal cord injury or sedation impair a person’s ability to recognize or respond to this discomfort. Tissue hypoxia then can develop and progress to tissue anoxia and cellular death.

**Duration of Pressure**

Duration of pressure is an important factor that influences the detrimental effects of pressure and must be considered in tandem with intensity of pressure. An inverse relationship exists between duration and intensity of pressure in creating tissue ischemia. Specifically, low-intensity pressures over a long period can create tissue damage just as high-intensity pressure can over a
short period (Figure 4). Husain (1953) underscored the significance of the relationship between duration and intensity of pressure. Husain found that a pressure of 100 mm Hg applied to rat muscle for 2 hours was sufficient to produce only microscopic changes in the muscle. However, the same pressure applied for 6 hours was sufficient to produce complete muscle degeneration.

**Tissue Tolerance**

Tissue tolerance is the third factor that determines the pathologic effect of prolonged pressure. It describes the condition or integrity of the skin and supporting structures that influence the skin’s ability to redistribute the applied pressure. Compression of tissue against skeletal structures and the resulting tissue ischemia can be prevented by effective redistribution of pressure.

The concept of tissue tolerance was first discussed with the need to identify how much pressure skin could “tolerate.” Later, Husain (1953) introduced the concept of sensitizing the tissue to pressure and consequently to ischemia. Rat muscle was sensitized with a pressure of 100 mm Hg applied for 2 hours. Seventy-two hours later, a mere 50 mm Hg pressure applied to the same tissue caused muscle degeneration in only 1 hour. This muscle destruction resulted during the second application of pressure, even though the intensity and duration of pressure were lower than the initial intensity and duration. This finding has significant implications for the patient population at risk for pressure ulcers. It indicates that episodes of deep tissue ischemia can occur without cutaneous manifestations and that such episodes can sensitize the patient’s skin. In vitro findings show that relatively small loads cause structural changes to the dermal component of tissue. Human tissue exhibits changes visible at the surface that often are minor compared to damage seen in deeper tissue layers (Edsberg, 2007). Small increments of pressure, even if only slightly above normal capillary pressure ranges, may then result in breakdown.

Tissue tolerance is influenced by the ability of the skin and underlying structures (e.g., blood vessels, interstitial fluid, collagen) to work together as a set of parallel springs that transmit load from the surface of the tissue to the skeleton inside (Krouskop, 1983). Several intrinsic and extrinsic factors can alter the ability of the soft tissue to perform this task.

**Extrinsic Factors that Affect Tissue Tolerance.**

**Shear.** Shear is caused by the interplay of gravity and friction. It exerts a force parallel to the skin and is the result of both gravity pushing down on the body and resistance (friction) between the patient and a surface, such as the bed or chair. For example, when the head of the bed is elevated, the effect of gravity on the body is to pull the body down toward the foot of the bed. In contrast, the resistance generated by the bed surface tends to hold the body in place. However, what is actually held in place is the skin, while the weight of the skeleton continues to pull the body downward.

Because the skin does not move freely, the primary effect of shear occurs at the deeper fascial level of the tissues overlying the bony prominence. Blood vessels, which are anchored at the point of exit through the fascia, are stretched and angulated when exposed to shear. This force also dissects the tissues, resulting in undermining.

Shear causes much of the damage often observed with pressure ulcers. In fact, some lesions that may result solely from shear are misinterpreted as pressure ulcers. Conversely, pressure ulcers may also be misinterpreted. Vascular occlusion is enhanced if shear and pressure occur together. For example, when the head of the bed is elevated more than 30 degrees, shear force occurs in the sacrococcygeal region. The sliding of the body transmits pressure to the sacrum and the deep fascia; the outer skin is fixed because of friction with the bed. The vessels in the deep superficial fascia angulate, leading to thrombosis and undermining of the dermis (see Figure 5-1). Dressings with a low-friction external surface have been reported to reduce shear force but do not significantly reduce interface pressures (Nakagami et al, 2006).

**Friction.** Friction is a significant factor in pressure ulcer development because it acts in concert with gravity to cause shear. Alone, its ability to cause skin damage is confined to the epidermal and upper dermal layers. In its mildest form, friction abrases the epidermis and dermis similar to a mild burn, and sometimes is referred to as “sheet burn.” This type of damage most frequently develops in patients who are restless. To prevent friction when moving up in bed, a patient who can lift independently should do so with a lift device or with use of the hands and arms. A patient who is dependent in care may

**FIGURE 4** Graph demonstrating relationship between intensity and duration of pressure. (From Kosiak M: Etiology of decubitus ulcers, *Arch Phys Med Rehabil* 42:191, 1961.)
need multiple caregivers to assist with moving up in bed while using a lift sheet or lift device to prevent the body from dragging.

When friction acts with gravity, the effect of the two factors is synergistic, and the outcome is shear. It is not possible to have shear without friction. However, it is possible to have friction without significant shear (such as from moving the heels repeatedly against the bed sheets).

**Moisture.** Moisture, specifically incontinence, is frequently cited in the literature as a predisposing factor to pressure ulcer development (Braden and Bergstrom, 1987). Persistent moisture alters the resiliency of the epidermis to external forces by weakening the lipid layer of the stratum corneum and collagen. Both shear and friction are increased in the presence of mild to moderate moisture but may be decreased in the presence of profuse moisture. The high-moisture environment created by urinary incontinence can impact the skin by alkalizing the skin’s pH, thereby altering normal skin flora. Persons with fecal incontinence are 22 times more likely to develop pressure ulcers than are persons without this condition (Thompson et al, 2005).

**Intrinsic Factors that Affect Tissue Tolerance.**

**Nutritional Debilitation.** Although good nutrition is necessary for wound healing, the role of significant nutritional debilitation in producing pressure ulcers is often less appreciated. Severe protein deficiency renders soft tissue more susceptible to breakdown when exposed to local pressure because hypoproteinemia alters oncotic pressure and causes edema formation. Oxygen diffusion and transport of nutrients in ischemic and edematous tissue are compromised. In addition, resistance to infection is decreased at low protein levels because of the effect on the immune system. Malnutrition has also been associated with altered tissue regeneration and inflammatory reaction, increased postoperative complications, increased risk of infection, sepsis, increased length of hospital stay, and death.

Certain vitamin deficiencies, particularly of vitamins A, C, and E, are a concern when assessing pressure ulcer risk. Vitamin A has a role in epithelial integrity, protein synthesis, and immune function; therefore a deficiency of vitamin A delays reepithelialization, collagen synthesis, and cellular cohesion. Vitamin C plays a role in collagen synthesis, enhanced activation of leukocytes and macrophages at a wound site, and immune function. Specific to wound healing, vitamin E aids in collagen synthesis, metabolism of fat, and stabilization of cell membranes (Posthauer, 2006).

All nutrients have an important role in maintaining skin integrity and in wound repair. Still, questions remain regarding how much supplementation of nutrients will positively affect outcomes. Meta-analyses of the clinical benefits of nutritional support in patients with or at risk for pressure ulcers showed an oral nutritional supplement was associated with a significantly lower incidence of pressure ulcer development in at-risk patients of 25% compared to routine care (Stratton et al, 2005). A Cochrane evaluation of enteral and parenteral nutrition on pressure ulcer prevention and treatment was not able to draw conclusions about the effect of such nutrition because of the small number of studies and methodologic issues with the studies (Langer et al, 2004). Researchers conclude that more research is needed about the impact of oral nutritional supplements and enteral tube feeding on prevention and treatment of pressure ulcers.

**Advanced Age.** Several changes occur in the skin and its supporting structures with aging. The dermoepidermal junction flattens, less nutrient exchange occurs, and less resistance to shear force is present (Pittman, 2007; Reddy, 2008). With aging, gradual atrophy and greater heterogeneity of blood and lymph vessels of human skin occur (Fore, 2006; Reddy, 2008). Changes in the cutaneous nerves lead to impaired early pain warning (Fore, 2006). Skin tears occur more commonly. Loss of dermal thickness occurs; the skin appears paper-thin and nearly transparent. Aging skin experiences decreased epidermal turnover, decreased surface barrier function, decreased sensory perception, decreased delayed and immediate hypersensitivity reaction, increased vascular fragility, loss of subcutaneous fat, and clustering of melanocytes (Fore, 2006; Pittman, 2007). With these changes, the ability of the soft tissue to distribute the mechanical load without compromising blood flow is impaired. These changes combine with many other age-related changes that occur in other body systems to make the skin more vulnerable to pressure, shear, and friction (Pittman, 2007). For example, studies have shown that blood flow in the area of the ischial tuberosity while sitting on an unpadded surface is lower in paraplegic and geriatric populations than in normal patients.

**Low Blood Pressure.** Mayrovitz et al (2003) noted in a study about heels that persons with lower blood pressure need lower levels of pressure to cause breakdown to the heels. When interface pressures are near diastolic pressure, little if any functional pressure redistribution is realized. When perfusion is decreased by hypotension, shock, or dehydration, blood flow to the skin is likely to be compromised, thus increasing ischemia; deep tissues may be particularly vulnerable because of their extensive vascular supply (Berlowitz and Brienza, 2007). Hypotension may shunt blood flow away from the skin to more vital organs, thus decreasing the skin’s tolerance for pressure by allowing capillaries to close at lower levels of interface pressure.

**Stress.** Early research identified psychosocial issues, such as emotional stress, as having an association with pressure ulcers. Cortisol may alter the mechanical properties of the skin by disproportionately increasing the rate of collagen degradation over collagen synthesis.
Glucocorticoids may trigger structural changes in connective tissue and may affect cellular metabolism by interfering with the diffusion of water, salt, and nutrients between the capillary bed and the cells. Hospitalization in acute or long-term care is stressful. In examining the relationship between stress and wound healing, stress has been negatively associated with healing. Cortisol may be the trigger for lowered tissue tolerance when a person is under stress. Cortisol is the primary glucocorticoid secreted when a person is exposed to a stressor and lacks appropriate coping mechanisms to mediate the stress-related hormonal response. Higher cortisol levels were related to longer time to heal (Ebrecht et al, 2004; Gouin et al, 2008). Many factors affect cortisol; they include advanced age, immobility, body fat, recent surgery, stroke, and malnutrition.

**Smoking.** Smoking is associated with tissue hypoxia, nicotine-induced stimulation of the sympathetic nervous system resulting in epinephrine that causes peripheral vasconstriction and decreased circulation, carbon monoxide shift of the oxygen dissociation curve, and hydrogen cyanide interference with cellular oxygen metabolism (Ahn et al, 2008). Smoking must be considered in patients at risk for pressure ulcers. For patients with spinal cord injury, cigarette smoking was associated with a 1.16 incidence rate ratio for one or more pressure ulcers during the previous year (Smith et al, 2008).

**Elevated Body Temperature.** The body experiences a 10% increase in tissue metabolism with each 1°C rise in skin temperature (Aronovitch, 2007). Elevated body temperatures increase metabolic rates and subsequently increase oxygen consumption rates. Elevated skin temperature exacerbate the effects of ischemia by increasing the need for oxygen (Berlowitz and Brienza, 2007).

**Miscellaneous Factors.** Other conditions, such as those that create sluggish blood flow, anemia, blood dyscrasias, or poor oxygen perfusion, may be significant intrinsic factors jeopardizing tissue tolerance. For example, greater tissue damage has been associated with increased blood viscosity and high hematocrit level. This may explain why dehydration is sometimes mentioned as a contributing factor in pressure ulcer development.

### PATHOPHYSIOLOGIC CHANGES

Two primary theories explain the mechanism of pressure ulcer formation and progression (Niezgoda and Mendez-Eastman, 2006). The deep tissue injury theory holds that pressure ulcers begin from the bone and move outward. Deep tissue injury occurs first near the bone, with ischemic injury and tissue destruction continuing in an outward manner. Deep muscle tissue appears to be more susceptible to pressure damage than are skin and fat (Berlowitz and Brienza, 2007). Although it is the less favored model of pressure ulcer development, the top-to-bottom model states pressure ulcer formation results from skin destruction that occurs at the epidermis and proceeds to deeper tissue (Niezgoda and Mendez-Eastman, 2006).

If pressure is not relieved, ischemic changes occur as a consequence of decreased perfusion; however, the occlusion also triggers a cascade of events that intensifies the extent of tissue ischemia. Hence the tissue damage typically seen with pressure is precipitated by pressure but then worsened by a series of events, such as venous thrombus formation, endothelial cell damage, redistribution of blood supply in ischemic tissue, alteration in lymphatic flow, and alterations in interstitial fluid composition.

Berlowitz and Brienza (2007) listed the four commonly hypothesized pathophysiologic explanations for pressure ulcers: (1) ischemia caused by capillary occlusion; (2) reperfusion injury; (3) impaired lymphatic function that results in accumulation of metabolic waste products, proteins, and enzymes; and (4) prolonged mechanical deformation of tissue cells. Prolonged mechanical deformation of tissue cells refers to unrelied pressure. The remaining three hypothesized pathophysiologic explanations for pressure ulcers are described here.

### Ischemia Caused by Capillary Occlusion

Obstruction of capillary blood flow by externally applied pressure creates tissue ischemia (hypoxia). If the pressure is removed in a short period, blood flow returns and the skin can be seen to flush. This phenomenon, known as *reactive hyperemia*, is a compensatory mechanism whereby blood vessels in the pressure area dilate in an attempt to overcome the ischemic episode. Reactive hyperemia by definition is transient and may also be described as blanching erythema. Blanching erythema is an area of erythema that becomes white (blanches) when compressed with a finger. The erythema promptly returns when the compression is removed. The site may be painful for the patient with intact sensation. Blanching erythema is an early indication of pressure and usually will resolve without tissue loss if pressure is reduced or eliminated.

When hyperemia persists, deeper tissue damage should be suspected. Nonblanching erythema is a more serious sign of impaired blood supply and suggests that tissue destruction is imminent or has already occurred; it results from damage to blood vessels and extravasation of blood into the tissues. The color of the skin can be an intense bright red to dark red or purple. Many providers misdiagnose pressure-induced nonblanching erythema as hematoma or ecchymosis. When deep tissue damage is also present, the area is often either indurated or boggy when palpated.

When pressure occludes capillaries, a complex series of events is set into motion. Surrounding tissues become deprived of oxygen, and nutrients and metabolic wastes begin to accumulate in the tissue. Damaged capillaries become more permeable and leak fluid into the interstitial space, causing edema. Because perfusion through edematous tissue is slowed, tissue hypoxia worsens. Cellular
death ensues, and more metabolic wastes are released into the surrounding tissue. Tissue inflammation is exacerbated, and more cellular death occurs (Figure 5). Considering data from surgical patients, animal models, and in-vitro cell culture models, pressure ulcers in subdermal tissue under bony prominences very likely occur approximately between the first hour and 4 to 6 hours after sustained loading (Gefen, 2008).

Muscle damage may occur with pressure ulcers and is more significant than cutaneous damage. Pressure is highest at the point of contact between the soft tissue (e.g., muscle or fascia) and the bony prominence. This cone-shaped pressure gradient indicates that deep pressure ulcers initially form at the bone–soft tissue interface, not the skin surface, and extend outward to the skin (Figure 6). Thus deep tissue damage may occur with relatively little initial superficial evidence of damage to alert caregivers of its extensiveness. The skin damage seen in pressure ulcers is often referred to as the “tip of the iceberg” because a larger area of necrosis and ischemia is assumed to be present at the tissue–bone interface. Muscle and fat tissue loading over a bony prominence is substantially higher during sitting than lying down, so pressure ulcer and deep tissue injury development are likely to occur sooner while sitting versus lying down (Gefen, 2008).

Muscle tissue is the most vascularized tissue layer between bone and skin. It is the tissue with the highest metabolic demand and the lowest tolerance to mechanical compression (Gefen, 2008). In addition, atrophied, scarred, or secondarily infected tissue has an increased susceptibility to pressure because of injured cells (Kumar et al., 2005b). An understanding of the structure of the vascular system allows formation of a rationale for this enhanced muscle damage.

The vascular circulation can be divided into three sections: segmental, perforator, and cutaneous. The segmental system is composed of the main arterial vessels arising from the aorta. The perforator system supplies the muscles but also serves as an interchange supply to the skin. The cutaneous system consists of arteries, capillary beds, and veins draining at different levels of the skin; it serves to provide thermoregulation and limited nutritional support. This indicates that occlusion of the perforator system may initiate muscle damage and may also create some of the cutaneous ischemia. The significance of perforator blood flow to skin damage has been demonstrated when musculocutaneous flaps have been elevated surgically.

**Reperfusion Injury**

As blood returns to tissue where it was occluded, an accumulation of damaged cellular byproducts and white blood cells obstructs the capillaries, and free radicals are released. The free radicals damage cellular proteins, DNA, and cell membranes and contribute to cell death (Fowler et al., 2008). Tissue injury increases with each ischemia–reperfusion cycle, the duration of ischemia, and the frequency of ischemia–reperfusion cycles (Farid, 2007).
Externally applied high pressures, even when applied for a short duration, damage the blood vessels directly, which in turn causes tissue ischemia. The changes in larger vessels and the formation of venous thrombi impair the normal reactive hyperemia that should occur once pressure is removed. Tissue remains ischemic even after the pressure has been alleviated.

Compression of the capillary wall also damages the endothelium. Complex pathologic changes in diverse cellular systems occur with ischemia. Up to a certain point and varying among different cell types, injury is amenable to repair, but with extension of ischemic duration, cell structures continue to deteriorate (Kumar et al, 2005b). Once pressure is removed and reperfusion begins, injury can be paradoxically exacerbated and proceed at an accelerated pace, and loss of additional cells occurs. As the endothelium is shed, platelets are activated by the underlying collagen, and clot formation is triggered. Furthermore, damaged endothelial cells lose their usual anticoagulant characteristics and release thrombogenic substances that exacerbate vessel occlusion and ultimately cause increased tissue ischemia.

The redistribution of the blood supply that occurs in ischemic skin further aggravates pressure-induced tissue hypoxia. Because of the externally applied pressure, blood flow to surface capillaries is reduced, and the reduction renders these vessels more vulnerable and more permeable than before.

**Impaired Lymphatic Function**

The lymphatic system has a critical role in body fluid and many other functions. It must act as a conduit that directs and regulates lymph flow and as a pump that generates lymph flow (Muthuchamy and Zawieja, 2008). Thus, the lymphatics are affected by pressure-induced ischemia. Lymphatic flow in pressure-damaged skin ceases. Likewise, the normal movement of interstitial fluid is inhibited by both pressure and ischemia. Consequently, protein is retained in the interstitial tissues, causing increased interstitial oncotic pressure, edema formation, dehydration of cells, and tissue irritation.

In summary, extensive or extended pressure occludes blood flow, lymphatic flow, and interstitial fluid movement. Tissues are deprived of oxygen and nutrients, and toxic metabolic products accumulate. Interstitial fluids retain proteins that dehydrate cells and irritate tissues. The ensuing tissue acidosis, capillary permeability, and edema contribute to cellular death.

**CLASSIFICATION OF PRESSURE ULCERS**

**History and Purpose**

During the 1980s the International Association for Enterostomal Therapy, now known as the WOCN Society, modified the Shea staging system, which originally was developed in 1975 (WOCN Society, 2003). In 2007, the...
National Pressure Ulcer Advisory Panel (NPUAP) released an updated staging system (Box 1). The new system improved clarity and accuracy by adding more descriptors and creating definitions for suspected deep tissue injury and unstageable pressure ulcers (NPUAP, 2007). In 2009, international pressure ulcer guidelines were released. These guidelines use the same definitions but have added the term category and specify that the suspected deep tissue injury and unstageable categories used in the United States (NPUAP and EPUAP, 2009).

Staging of tissue layers provides increased uniformity of language and a beginning basis for evaluation of protocols. Accurate staging requires knowledge of the anatomy of skin and deeper tissue layers, the ability to recognize these tissues, and the ability to differentiate among them. Careful evaluation of the wound bed facilitates accurate staging. Staging wounds is a complex skill that can take time to develop. The staging system, which is designed for use with pressure-induced ulcers only, is based on the ability to assess the type of tissue in the wound bed. Therefore a wound bed in which the base is covered with necrotic tissue cannot be accurately staged because of the inability to visualize the normal architecture of the wound bed. In such situations, “unstageable” should be documented. Box 7-2 gives the differential diagnosis of suspected deep tissue injury due to pressure (Ankrom et al, 2005).

**Dark Skin Tones**

Identifying deep tissue injury (suspected) and Stage I pressure ulcers in darker skin tones is difficult. Redness and other color changes are not as detectable with darker skin tones. Therefore other observable, pressure-related alterations of intact skin compared with the adjacent or opposite area on the body should be documented. Such alterations of intact skin compared with the adjacent or opposite area on the body should be documented. Such alterations of intact skin compared with the adjacent or opposite area on the body should be documented. Such

<table>
<thead>
<tr>
<th>BOX 1</th>
<th>National Pressure Induced Ulcer Advisory Panel (NPUAP) Pressure Ulcer Stages</th>
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</thead>
<tbody>
<tr>
<td><strong>Suspected Deep Tissue Injury</strong></td>
<td>Purple or maroon localized area of discolored intact skin or blood-filled blister due to damage of underlying soft tissue from pressure and/or shear. The area may be preceded by tissue that is painful, firm, mushy, boggy, warmer or cooler compared to adjacent tissue.</td>
</tr>
<tr>
<td><strong>Further Description</strong></td>
<td>Deep tissue injury may be difficult to detect in individuals with dark skin tones. Evolution may include a thin blister over a dark wound bed. The wound may further evolve and become covered by thin eschar. Evolution may be rapid, exposing additional layers of tissue even with optimal treatment.</td>
</tr>
<tr>
<td><strong>Stage I</strong></td>
<td>Intact skin with nonblanchable redness of a localized area usually over a bony prominence. Darkly pigmented skin may not have visible blanching; its color may differ from the surrounding area.</td>
</tr>
<tr>
<td><strong>Further Description</strong></td>
<td>The area may be painful, firm, soft, warmer or cooler compared to adjacent tissue. Stage I may be difficult to detect in individuals with dark skin tones. May indicate “at risk” persons (a heralding sign of risk)</td>
</tr>
<tr>
<td><strong>Stage II</strong></td>
<td>Partial thickness loss of dermis presenting as a shallow open ulcer with a red–pink wound bed, without slough. May also present as intact or open/ruptured serum-filled blister.</td>
</tr>
<tr>
<td><strong>Further Description</strong></td>
<td>Presents as shiny or dry shallow ulcer without slough or bruising.* This stage should not be used to describe skin tears, tape burns, perineal dermatitis, maceration, or excoriation.</td>
</tr>
<tr>
<td><strong>Stage III</strong></td>
<td>Full-thickness tissue loss. Subcutaneous fat may be visible, but bone, tendon, or muscle is not exposed. Slough may be present but does not obscure the depth of tissue loss. May include undermining and tunneling.</td>
</tr>
<tr>
<td><strong>Further Description</strong></td>
<td>The depth of a Stage III pressure ulcer varies by anatomic location. The bridge of the nose, ear, occiput, and malleolus do not have subcutaneous tissue, and Stage III ulcers can be shallows. In contrast, areas of significant adiposity can develop in extremely deep Stage III pressure ulcers. Bone/tendon is not visible or directly palpable.</td>
</tr>
<tr>
<td><strong>Stage IV</strong></td>
<td>Full-thickness tissue loss with exposed bone, tendon, or muscle. Slough or eschar may be present on some parts of the wound bed. Often include undermining and tunneling.</td>
</tr>
<tr>
<td><strong>Further Description</strong></td>
<td>The depth of a Stage IV pressure ulcer varies by anatomic location. The bridge of the nose, ear, occiput, and malleolus do not have subcutaneous tissue, and these ulcers can be shallow. Stage IV ulcers can extend into muscle and/or supporting structures (e.g., fascia, tendon, joint capsule), making osteomyelitis possible. Exposed bone/tendon is visible or directly palpable.</td>
</tr>
<tr>
<td><strong>Unstageable</strong></td>
<td>Full-thickness tissue loss in which the base of the ulcer is covered by slough (yellow, tan, gray, green, or brown) and/or eschar (tan, brown, or black) in the wound bed.</td>
</tr>
<tr>
<td><strong>Further Description</strong></td>
<td>Until enough slough and/or eschar is removed to expose the base of the wound, the true depth, and therefore the stage, cannot be determined. Stable (dry, adherent, intact without erythema or fluctuance) eschar on the heels serves as the “body’s natural (biologic) cover” and should not be removed.</td>
</tr>
</tbody>
</table>

From National Pressure Ulcer Advisory Panel (NPUAP) and European Pressure Ulcer Advisory Panel (EPUAP); Treatment of pressure ulcers, Washington DC, 2009, National Pressure Ulcer Advisory Panel.

*Bruising indicates suspected deep tissue injury.
indicators include changes in skin tissue consistency (firm versus boggy when palpated), sensation (pain), and warmer or cooler temperature (Black et al, 2007). Box 1 lists unique characteristics of darker versus lighter pigmented skin, and Checklist 1 gives points to consider when assessing darkly pigmented skin.

**Mucosal Pressure Ulcers**

Mucous tissues (i.e., lining of the gastrointestinal [GI] tract, oral cavity, nares, etc.) are also vulnerable to pressure ulcers from the presence of medical devices such as oxygen tubing, endotracheal tubes, bite blocks, nasogastric tubes, urinary catheters and fecal containment devices. These ulcers cannot be staged using the pressure ulcer staging system nor classified as partial or full thickness because the histology of mucous membrane tissue is different than skin. Therefore, pressure ulcers on mucous membranes should be documented simply as mucosal pressure ulcers (NPUAP, 2009).

**Reverse Staging**

The practice of reverse staging, in which the wound is described as progressing from a Stage III to a Stage II to a Stage I pressure ulcer, is incorrect. Once layers of tissue and supporting structures are gone (such as with full-thickness wounds), they are not replaced. Instead, the wound is filled with granulation tissue. This staging system is to be used for describing wounds in their most severe state and once the wounds are accurately described, these descriptor levels endure, even in the presence of healing (Black et al, 2007). Negative outcomes of reverse staging can lead to (1) denial of acute or skilled care after Stage IV ulcers have been restaged as Stage II ulcers; (2) withdrawal of pressure-reducing support surfaces when ulcers have “healed” from Stage III or Stage IV to Stage II; and (3) lower fees paid to extended-care facilities for care of patients with healing Stage III and Stage IV ulcers that have been reclassified as Stage II or Stage I pressure ulcers. Therefore a Stage III pressure ulcer that appears to be granulating and resurfacing is described as a healing Stage III pressure ulcer.

**SUMMARY**

Pressure ulcers present a significant economic, quality-of-life, and overall health care threat worldwide. Standards for assessment and care initially presented by the WOCN Society have now been published by a number of dedicated disciplines and groups around the world. Once a problem considered a side effect of aging, pressure ulcers have captured the attention of payers and regulators. Pressure ulcers now are more commonly considered preventable and unacceptable (in most cases) and are considered an indicator of quality care.

**REFERENCES**


Farid KJ: Applying observations from forensic science to understanding the development of pressure ulcers, Ostomy Wound Manage 53:26, 2007.


Darton & Harvey.


