

Pressure Ulcers: Impact, Etiology, and Classification

Barbara Pieper

OBJECTIVES

1. Define pressure ulcer and terms associated with it.
2. Describe the incidence and prevalence of pressure ulcers.
3. Identify economic effects and quality-of-life impact of pressure ulcers.
4. Examine vulnerable populations (older adult, spinal cord injured, surgical, obese and underweight patients) in terms of pressure ulcer risk.
5. Describe the role of the causative factors (intensity of pressure, duration of pressure, and tissue tolerance [intrinsic and extrinsic]) for pressure ulcer formation.
6. Summarize the history of pressure ulcer classifications.
7. Describe the pathophysiologic consequences of pressure damage, including the changes that occur at the cellular level and the cone-shaped pressure gradient.
8. Define variables that influence the extent of tissue damage as a consequence of pressure.

A pressure ulcer is a localized injury to the skin and/or underlying tissue, usually over a bony prominence, as a result of pressure or pressure in combination with shear (NPUAP, EPUAP, PPIA, 2014). 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 *Prevention and Treatment of Pressure Ulcers: Clinical Practice Guideline* (NPUAP, EPUAP, PPIA, 2014), the Wound, Ostomy and Continence Nurses Society's™ (WOCN®) *Guideline for Prevention and Management of Pressure Ulcers* (WOCN®, 2010), the *Association for the Advancement of Wound Care's (AAWC) Venous and Pressure Ulcer Guidelines* (Bolton et al., 2014), the *American College of Physicians' Risk Assessment and Prevention of Pressure Ulcers: A Clinical Practice Guideline from the American College of Physicians* (Qaseem, Mir et al, 2015) and *Treatment of Pressure Ulcers: A Clinical Practice Guideline from the American College of Physicians* (Qaseem, Humphrey et al, 2015), the Registered Nurses Association of Ontario's (RNAO) *Risk Assessment and Prevention of Pressure Ulcers* (RNAO, 2011), *Guidelines for the Prevention of Pressure Ulcers* by the Wound

Healing Society (Stechmiller et al, 2008), the Canadian Association of Wound Care's *Best Practice Recommendations for the Prevention and Treatment of Pressure Ulcers: Update 2006* (Keast et al, 2006), *Healthy People 2020* (U.S. Department of Health and Human Services, 2010), and *Pan Pacific Clinical Practice Guideline for the Prevention and Management of Pressure Injury* (Australian Wound Management Association, 2012). For the Institute for Healthcare Improvement, pressure ulcers remain a serious threat to patient safety and is a key focus of their efforts (Institute for Healthcare Improvement, 2015).

SCOPE OF THE PROBLEM

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

Prevalence

Pressure ulcer *prevalence* is the proportion or percentage of patients in a defined population with at least one pressure ulcer at a given point in time (Berlowitz, 2012). In the United States across 15 studies, prevalence rates for acute care range from 0% to 15.8% (Goldberg, 2012). The prevalence of pressure ulcers in long-term care across 20 studies done in the United States has been reported as 8.2% to 32.2% (Pieper, 2012). The prevalence of pressure ulcers in home care ranges from 2.9% to 19.1% (Garcia, 2012). Pressure ulcer prevalence in pediatric populations ranges from 0.47% to 75%, with the highest occurrences in the spinal cord injured and outpatients with diagnoses of myelodysplasia and cerebral palsy; 44% of pressure ulcers were secondary to medical devices such as casts and orthoses (Baharestani, 2012). The discrepancies in prevalence can be attributed to the fact that some studies include intact pressure-damaged skin (suspected deep tissue injury or Stage I), 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 (Sieggreen, 2012a). Some skin conditions, such as candidiasis, incontinence-associated dermatitis, 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. Data collectors must accurately distinguish between pressure ulcers and other causes of erythema and skin ulcerations.

Incidence

Incidence is the number of patients in a defined population who initially were ulcer free but develop a pressure ulcer within a particular time period (Berlowitz, 2012). 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 clinical setting. The incidence of pressure ulcers in acute care ranges from 2.8% to 9% (Goldberg, 2012). The incidence in long-term care ranges from 3.6% to 59% (Pieper, 2012) and in home care from 4.5% to 6.3% (Garcia, 2012).

Considerable methodological 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

these kinds of data. The National Database of Nursing Quality Indicators (NDNQI) includes pressure ulcer prevention and hospital-acquired pressure ulcer reports from more than 1705 facilities in the United States plus 12 hospitals outside the United States designed for comparisons between hospitals of similar sizes and practice levels (Bergquist-Beringer et al, 2012). Utilizing this large database, the NDNQI reported the 2010 prevalence of pressure ulcers in acute care was 9.1% and the incidence of hospital-acquired pressure ulcers as 3.7% (Bergquist-Beringer et al, 2012).

Economic Effects

In fiscal year 2007, a preventable pressure ulcer was listed as a secondary diagnosis for 300,000 Medicare patients; each pressure ulcer added \$43,180 in costs to a hospital stay (AHRQ, 2011; Department of Health and Human Services, Centers for Medicare and Medicaid Services, 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. The CMS, along 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.

The 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 \$9.1 to \$11.6 billion annually (AHRQ, 2011). 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 with 12.7 days for a secondary pressure ulcer diagnosis. Theisen et al (2012) reported that in Germany, older adults with pressure ulcers had longer hospital stays (mean 19 vs. 9.9 days). According to the Healthcare Cost and Utilization Project, the average cost per hospital day for a principal pressure ulcer diagnosis was \$1200 compared with \$1600 for a secondary pressure ulcer diagnosis (Russo et al, 2008). Nearly three of four hospitalizations with a pressure ulcer diagnosis were billed to Medicare; Medicaid patients accounted for an 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. 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 (Bolton et al., 2014; NPUAP, EPUAP, PPIA, 2014; Pieper et al, 2009). Pain assessment and management are discussed in detail in Chapters 25 and 26.

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 (AHRQ, 2011). 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 septicemia 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). Flattau and Blank (2014) reported that risk factors for 90-day and 180-day mortality in hospitalized patients with pressure ulcers were diabetes, chronic renal failure, congestive heart failure, metastatic cancer, and low serum albumin.

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 a spinal cord injury, surgical patients, obese patients, underweight patients, children, and patients at the 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 three of four (72%) 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; Pieper, 2012). For older adults with a pressure ulcer, coexisting conditions were fluid and electrolyte disorders, nutritional disorders, diabetes mellitus without complications, and dementia (Bolton et al, 2014; Russo et al, 2008).

Pressure ulcer prevalence for persons with spinal cord injuries ranges from 5.8% to 23.3% (Brienza and Karg, 2012). 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. Reported factors associated with increased incidence and prevalence of pressure ulcers in the spinal cord injured are history of pressure ulcers, coexisting medical conditions (such as diabetes mellitus and depression), rehospitalizations, nursing home stays, less than high school education, older adult, male, African American, single, behavior factors such as smoking, and time since injury (Brienza and Karg, 2012; Schubart et al, 2008).

The incidence of pressure ulcers in the perioperative period ranges from 5% to 53.4% (Ganos and Siddiqui, 2012). Associated factors were at least one comorbidity (i.e., diabetes mellitus, congestive heart failure, decreased preoperative albumin, acute renal insufficiency, recent significant weight loss), managed with a warming device, receipt of three or more anesthetic agents, and median operative time of 4.48 hours (Aronovitch, 2007; Ganos and Siddiqui, 2012). Perioperative pressure ulcer definition includes appearance of the injury up to 72 hours post operating room (Ganos and Siddiqui, 2012). Thus 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 to 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.

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, and inadequate staff numbers or staff not trained in how to turn and move such patients (Blackett et al, 2011; Knudsen and Gallagher, 2003; Mathison, 2003). Risk factors include increased perspiration and moisture (including incontinence) and not fitting well in chairs or beds, so there is pressure against equipment (Sieggreen, 2012b). 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 comparison, the odds of the underweight patient developing a pressure ulcer were almost doubled (odds

ratio 1.8) (Compher et al, 2007). In contrast, Drake et al (2010) reported those with a body mass index of more than 40 and Braden scale score of 16 or less had an independent and statistically significant association with pressure ulcer occurrence.

Two additional vulnerable patient populations are children and patients at the end of life. Pressure ulcer prevalence, incidence, and risk factors for these two patient populations are presented in Chapters 36 and 37, respectively.

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 can 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, including pressure in combination with shear (NPUAP, EPUAP, PPPIA, 2014). A number of contributing or confounding factors are associated with pressure ulcers, but the significance of these factors has not yet been elucidated (NPUAP, EPUAP, PPPIA, 2014).

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., medical device related, such as underneath a cast, splint, or cervical collar). Figure 7-1 shows the 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/coccyx, buttocks, and the heels (Van Gilder et al, 2009).

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)

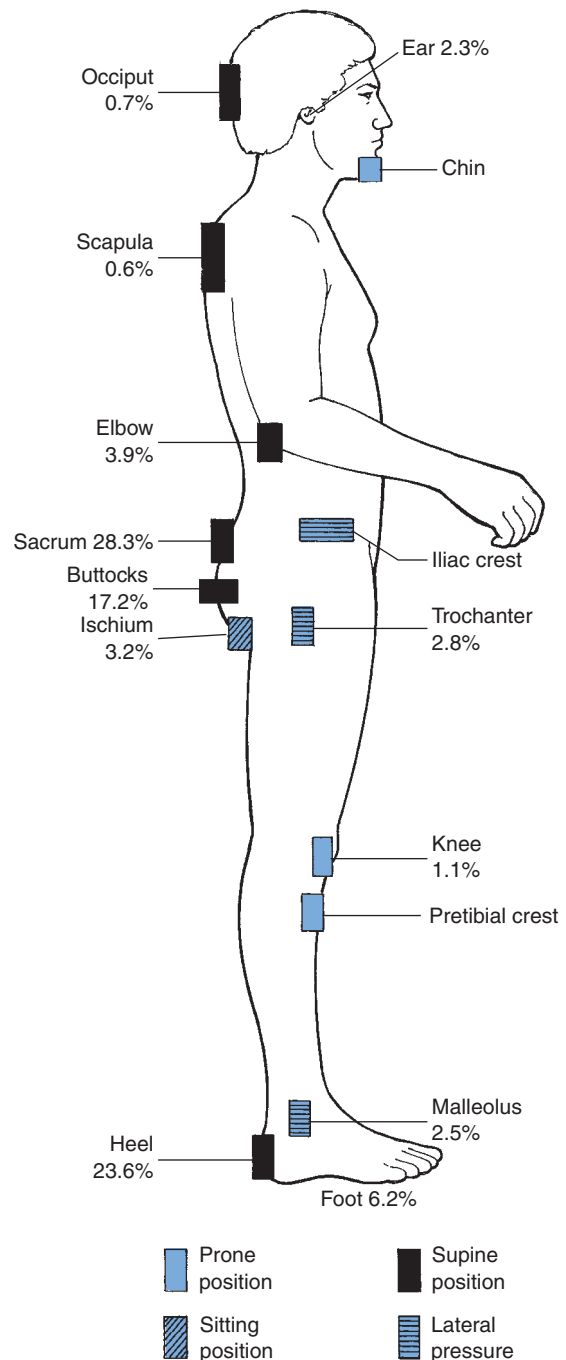


FIGURE 7-1 Sites for pressure ulcers and frequency of ulceration per site (N 5 85,838). Note: Sites for device-related pressure ulcers, which may not involve bony prominences, are not included. (Data from Van Gilder C et al: Results of the 2008–2009 International Pressure Ulcer Prevalence Survey and a 3-year, acute care, unit-specific analysis, *Ostomy Wound Manage* 55(11):39–45, 2009.)

presented a model of the factors that contribute to the intensity and duration of pressure ulcers (Figure 7-2), in combination with intrinsic and extrinsic factors that affect tissue tolerance. Defloor introduced a pressure ulcer conceptual scheme in 1999 arguing that only pressure (compressive force) and shear (shearing force) are known causative factors;

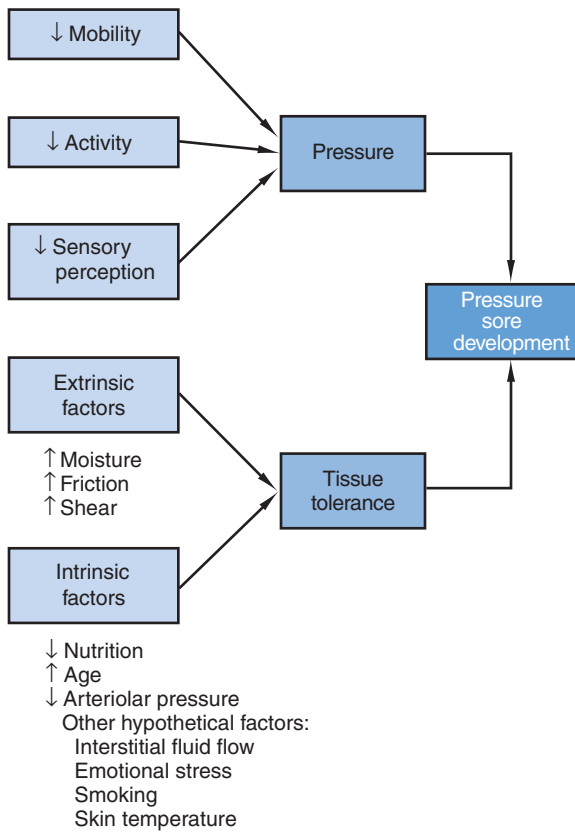


FIGURE 7-2 Factors contributing to the development of pressure ulcers. (From Braden B, Bergstrom N: A conceptual schema for the study of the etiology of pressure sores, *Rehabil Nurs* 12(1):8–12, 1987.)

therefore tissue tolerance is a modifying factor rather than a causative factor as depicted in the Braden and Bergstrom conceptual framework. Intensity and duration affect both compressive force and shearing force. Factors that affect intensity

of compressive force include support, posture, body build, and medical/nursing interventions, whereas factors affecting duration of compressive force include pain reaction, pain sensitivity, mobility/activity, and medical/nursing interventions. Intensity of shearing force is determined by support, posture, maceration, and friction, whereas duration of shearing force is determined by pain reaction, pain sensitivity, mobility/activity, and medical/nursing interventions. The DeFoor model also recognizes that different factors affect tissue tolerance for pressure and tissue tolerance for oxygen. Although preventive measures can be directed at pressure, shearing force, or tissue tolerance, pressure ulcer onset can only be eliminated by addressing the compressive force and the shearing force. The model consolidates known risk factors.

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.

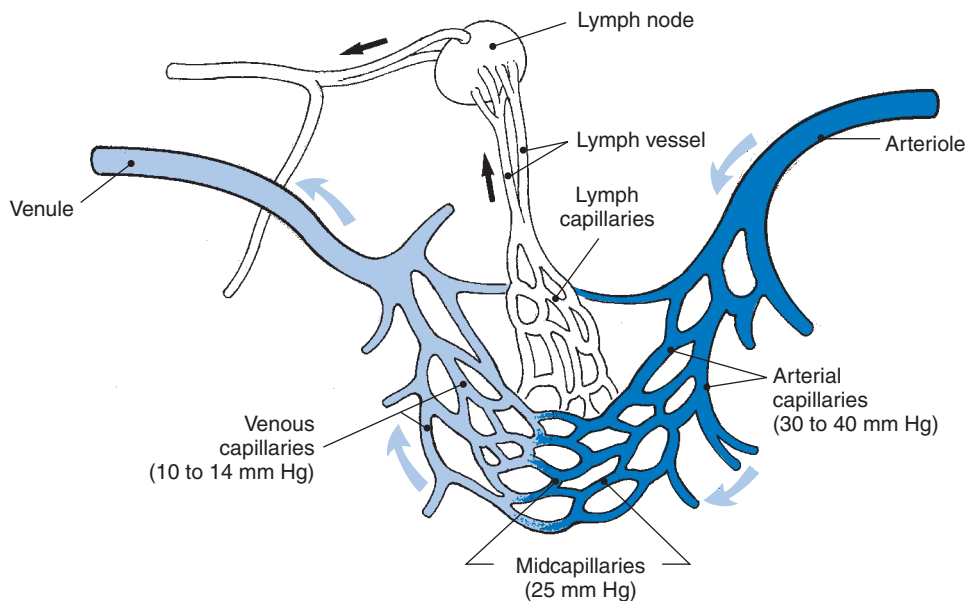


FIGURE 7-3 Capillary pressure within the capillary bed.

To quantify the intensity of pressure being applied externally to the skin, interface pressures are measured. Numerous studies measuring interface pressures have been conducted (Kosiak, 1961; Kosiak et al, 1958; Lindan, 1961). These studies showed that interface pressures attained while a person is in the sitting or supine position commonly exceed capillary pressures (Bennett et al, 1984). In 1961 Lindan used an experimental “bed” to calculate the pressure distribution over the skin 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.

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 can then develop and progress to tissue anoxia and cellular death. Olesen et al (2010) reviewed literature on etiology of sitting-acquired deep tissue pressure ulcers. They reported (1) high interface pressure and shear forces are risk factors for wheelchair users; (2) interface pressure leads to strain and stress concentrations in the interface between bone and muscle under the buttocks; (3) ischemia experiments show pressure impinges arteries, which does not quickly lead to necrosis, but impingement of the veins does; and (4) deformation rather than hypoxia kills the cells. Gefen (2010) described a biomechanical investigation of heel pressure ulcers using a model that characterized the internal mechanical loading at the soft tissues of a supported heel. He reported a heavy-weighted foot, sharp posterior calcaneus and thin soft tissue padding, and diabetes and edema imposed risks for heel ulcers.

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

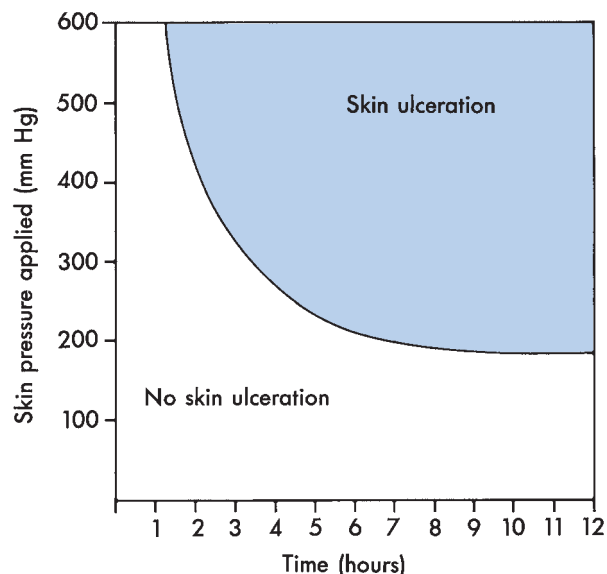


FIGURE 7-4 Graph demonstrating relationship between intensity and duration of pressure. (From Kosiak M: Etiology of decubitus ulcers, *Arch Phys Med Rehabil* 42:19–29, 1961.)

(Figure 7-4). 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. The duration of time that tissues can resist periods of ischemia differs for muscle, fat, and skin; muscle tissues appear to be more susceptible to damage than skin tissue (NPUAP, EPUAP, PPIA, 2014). Sprigle and Sonenblum (2011) noted both magnitude and duration of load must be considered; the use of different size and shape indenters, different loading parameters, and different animal models explains why a range of magnitudes and duration is linked to pressure ulcers.

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 with 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. High shear forces at the interface between the body and the supporting surface may exacerbate the damage caused by normal stresses alone (NPUAP, EPUAP, PPIA, 2014).

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. The National Pressure Ulcer Advisory Panel (NPUAP) deleted the word *friction* from the definition of a pressure ulcer (NPUAP, EPUAP, PPIA, 2014) to reinforce that skin injuries caused by friction were not to be considered

pressure ulcers (Antokal et al, 2012). Friction can cause minor to substantial skin impairment, and these lesions can occur in the same location as pressure ulcers. Friction is not a direct cause of a pressure ulcer, but is a risk factor that may contribute to or exacerbate pressure ulcer development due to the shear it creates (Antokal et al, 2012).

Alone, friction's ability to cause skin damage is confined to the epidermal and upper dermal layers and disturbs the barrier function of the stratum corneum (NPUAP, EPUAP, PPIA, 2014). In its mildest form, friction abrades 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 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 from incontinence, is frequently cited in the literature as a predisposing factor to pressure ulcer development. 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 affect the skin by alkalinizing the skin's pH, thereby altering normal skin flora. Persons with fecal incontinence are more likely to develop pressure ulcers than are persons without this condition (Bergquist-Beringer and Gajewski, 2011). Clinicians need to be able to differentiate among lesions caused by moisture, moisture and friction, and pressure and shear (Doughty et al, 2012; Gray et al, 2011; Gray et al, 2012).

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. Poor nutritional intake and poor nutritional status correlate with the development of pressure ulcers and should be assessed (Bolton et al, 2014; Dorner et al, 2009; NPUAP, EPUAP, PPIA, 2014). Banks et al (2009) reported malnutrition was associated with at least twice the odds ratio of having a pressure ulcer in a public health care facility. 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). Vitamin D deficiency was not found to be an independent risk factor for pressure ulcers, but may be a marker of comorbid illnesses, which increase the risk of pressure ulcers (Kalava et al, 2011).

All nutrients have an important role in maintaining skin integrity. Cowan et al (2012) found that malnutrition, along with three other medical factors, had stronger predictive value for predicting pressure ulcers in acutely ill veterans than Braden scale total scores alone. Still, questions remain regarding how much supplementation of nutrients will positively affect outcomes. Metaanalyses 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 with 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 methodological issues with the studies (Langer et al, 2003). However, they reported that nutritional interventions may be able to reduce the number of people who develop pressure ulcers. 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 (Qaseem et al, 2015).

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. Compared with young skin, aged skin showed subepidermal separations and altered orientation of collagen fibers at an earlier time point (Stojadinovic et al, 2013). Researchers concluded that aging contributes to rapid morphologic changes and a decline in the innate inflammatory response in elderly skin, possibly contributing to pressure ulcer pathogenesis. Sopher and Gefen (2011) reported theoretically that wetness, skin aging, and/or skin wrinkling are risk factors for superficial pressure ulcers.

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, Wong (2011) reported for older adults who needed help turning and repositioning that repositioning from a supine to lateral position did not cause the transcutaneous oxygen to return to preload levels. She concluded the efficacy of 2-hour repositioning needs further investigation. Seating and repositioning can affect pressure even when participants are young. For example, pressure relief maneuvers and tilting can result in reductions in interface pressure and increases in buttocks blood flow (Sonnenblum and Sprigle, 2011; Sonnenblum et al, 2014).

Low blood pressure. Mayrovitz et al (2003) noted in a study about heels that persons with lower blood pressure require lower levels of pressure to the heels to cause breakdown. 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. Hypotension was a significant factor in patients with pressure ulcers in intensive care units (Terekechi et al, 2009). Man and Au-Yeung (2013) reported the odds of developing a pressure ulcer in a patient with hypotension in the acute care setting was 6.71 ($p = .001$). That said, vasopressor medications are a first-line treatment modality for hypotension (Cox, 2013). In a literature review, 7 of 10 studies reported significant associations among the broad category of vasopressors as a pressure ulcer risk factor (Cox, 2013). Research is needed to elucidate vasopressors as an independent risk factor for pressure ulcer development.

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: 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 vasoconstriction 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. Using an animal model, Tsutakawa et al (2009) reported the increased risk of pressure ulcers due to cigarette smoking is mediated, in part, by nicotine, which is elicited via an increase of inflammatory mediators in the ischemic reperfusion-treated skin. For veterans with spinal cord injury, those who smoked cigarettes had a slightly higher incidence (incidence rate ratio = 1.16) for one or more pressure ulcers during the previous year (Smith et al, 2008). In contrast, current smoking did not differ between veterans with and without pressure ulcers (Rabadi and Vincent, 2011). Bolton et al (2014) stated smoking and other substance abuse issues that affect the skin should be assessed.

Elevated body temperature. The body experiences a 10% increase in tissue metabolism with each 1° C (33.8° F) rise in skin temperature (Aronovitch, 2007). Elevated body temperatures increase metabolic rates and subsequently increase oxygen consumption rates. Elevated skin temperature exacerbates the effects of ischemia by increasing the need for oxygen (Berlowitz and Brienza, 2007). Findings on elevated body temperature are mixed. Dhandapani et al (2014) reported prolonged fever had no significant association with pressure ulcer development in patients with severe traumatic brain injury. Systemic infection or fever was associated with mortality in 90 days for hospitalized patients with pressure ulcers; it did not show a confounding effect on other variables (Flattau and Blank, 2014).

Rapp et al (2009) examined skin temperature in a small sample of nursing facility residents. Skin temperature was lowest in those who developed pressure ulcers and between low-risk and high-risk residents. They concluded the findings support skin temperature regulation as a component of tissue tolerance to pressure.

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. As observed by Hussain in 1953, 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, a top-to-bottom model for pressure ulcer formation proposes that skin destruction 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 unrelieved 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 7-5). 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 7-6). Thus deep tissue damage may occur with relatively little initial superficial evidence of damage. The skin damage seen with 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

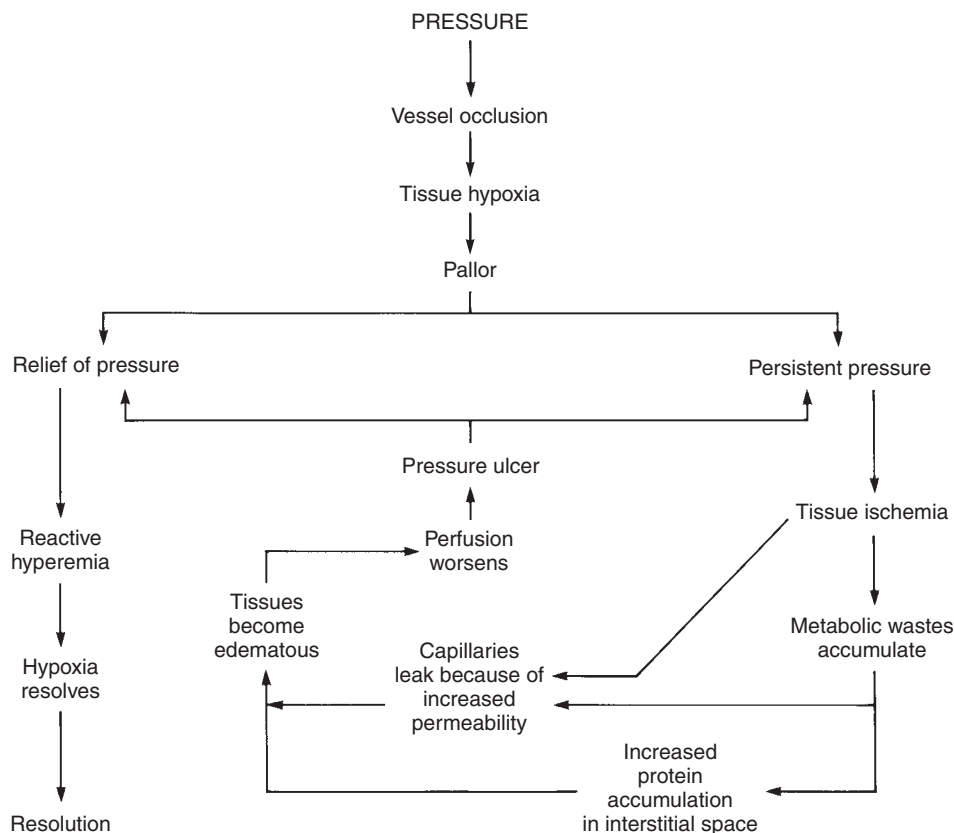


FIGURE 7-5 Cellular response to pressure.

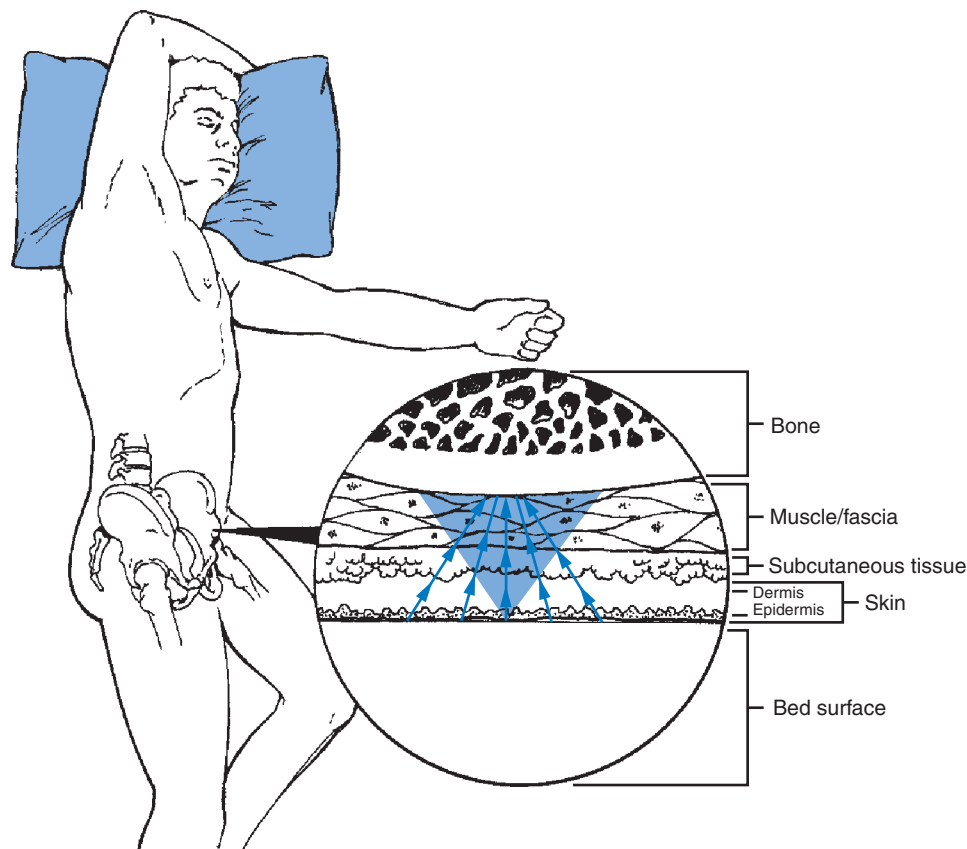


FIGURE 7-6 Amount of pressure exerted at skin level is dispersed over a large area and tapers to a point at the bone level (as indicated by the arrows), whereas the amount of tissue damage at the skin level is a pinpoint and broadens to a larger amount of tissue damage at the bone interface (indicated by the shaded area).

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

Pressure ulcer staging has a long history (Black and Langemo, 2012). 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®, 2010). In 2007 the NPUAP released an updated staging system (Box 7-1). The new system improved

clarity and accuracy by adding more descriptors and creating definitions for suspected deep tissue injury and unstageable pressure ulcers (NPUAP, 2012). In 2014 the NPUAP/EPUAP international pressure ulcer classification system was released. The classification uses the same definitions, but added the term *category* (NPUAP, EPUAP, PPPIA, 2014). Some have challenged the current staging system and propose a paradigm that delineates superficial skin changes and deep pressure ulcers (Sibbald et al, 2011).

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.

BOX 7-1 International NPUAP/EPUAP Pressure Ulcer Classification System

Pressure Ulcer Stages/Categories

Category/Stage I: Nonblanchable Erythema

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. The area may be painful, firm, soft, warmer, or cooler compared with adjacent tissue. Category I may be difficult to detect in individuals with dark skin tones. May indicate "at risk" persons.

Category/Stage II: Partial Thickness

Partial-thickness loss of dermis presenting as a shallow open ulcer with a red-pink wound bed, without slough. May also present as an intact or open/ruptured serum-filled or serosanguineous-filled blister. Presents as a shiny or dry shallow ulcer without slough or bruising.* This category should not be used to describe skin tears, tape burns, incontinence-associated dermatitis, maceration, or excoriation.

Category/Stage III: Full-Thickness Skin Loss

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. The depth of a Category/Stage III pressure ulcer varies by anatomic location. The bridge of the nose, ear, occiput, and malleolus do not have (adipose) subcutaneous tissue, and Category/Stage III ulcers can be shallow. In contrast, areas of significant adiposity can develop extremely deep Category/Stage III pressure ulcers. Bone/tendon is not visible or directly palpable.

Category/Stage IV: Full-Thickness Tissue Loss

Full-thickness tissue loss with exposed bone, tendon, or muscle. Slough or eschar may be present. Often includes undermining

and tunneling. The depth of a Category/Stage IV pressure ulcer varies by anatomic location. The bridge of the nose, ear, occiput, and malleolus do not have (adipose) subcutaneous tissue and these ulcers can be shallow. Category/Stage IV ulcers can extend into muscle and/or supporting structures (e.g., fascia, tendon, or joint capsule) making osteomyelitis or osteitis likely to occur. Exposed bone/muscle is visible or directly palpable.

Unstageable/Unclassified: Full-Thickness Skin or Tissue Loss – Depth Unknown

Full-thickness tissue loss in which actual depth of the ulcer is completely obscured by slough (yellow, tan, gray, green, or brown) and/or eschar (tan, brown, or black) in the wound bed. Until enough slough and/or eschar is removed to expose the base of the wound, the true depth cannot be determined, but it will be either a Category/Stage III or IV. Stable (dry, adherent, intact without erythema or fluctuance) eschar on the heels serves as "the body's natural (biological) cover" and should not be removed.

Suspected Deep Tissue Injury – Depth Unknown

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 with adjacent tissue. 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.

*Bruising indicates deep tissue injury.

National Pressure Ulcer Advisory Panel (NPUAP), European Pressure Ulcer Advisory Panel (EPUAP) and Pan Pacific Pressure Ulcer Injury Alliance (PPPIA): Prevention and treatment of pressure ulcers: clinical practice guideline. E Haesler, editor: Cambridge Media: Osborne Park, Western Australia, 2014.

In such situations, “unstageable” should be documented. Examples of Stage I to IV pressure ulcers, suspected deep tissue injury, and unstageable pressure ulcers are provided in Plates 27 through 33. Box 7-2 gives the differential diagnoses of suspected deep tissue injury due to pressure (Ankrom et al, 2005).

Darker Skin Tones

Identifying suspected deep tissue injury 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. Skin inspection should include assessment of changes in skin tissue consistency (firm vs. boggy when palpated), sensation (pain), edema, and warmer or cooler temperature (Bolton et al, 2014; NPUAP, EPUAP, PPIA, 2014). Box 3-1 lists unique characteristics of darker versus lighter pigmented skin, and Checklist 6-1 gives points to consider when assessing darkly pigmented skin.

Medical Device–Related Pressure Ulcers

Pressure ulcers can develop beneath medical devices. Medical device–related (MDR) pressure ulcers have a reported incidence of 9.1%; as many as 34.5% of hospital-acquired pressure ulcers reported as MDR (Goldberg, 2012). The most common stages are I and II and common sites are ears, heels, and lower legs. Patients with a medical device are 2.4 times more likely to develop a pressure ulcer of any kind (Black et al, 2010). Although numerous risk factors have

been identified, they are not unique to MDR pressure ulcers (Black et al, 2010). Medical device–related ulcers also affect children (Baharestani, 2012).

Mucosal Pressure Ulcers

Mucous tissues (i.e., lining of the gastrointestinal 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 or classified as partial or full thickness because the histology of mucous membrane tissue is different from 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.

BOX 7-2 Differential Diagnoses of Suspected Deep Tissue Injury Due to Pressure

Bruise: Extravasation of blood in the tissues as a result of blunt-force impact to the body. Usually about 2 weeks is required for a bruise to heal under normal conditions. History of trauma is common.

Calciophylaxis: Vascular calcification and skin necrosis most common in patients with long-standing history of chronic renal failure and renal replacement therapy. Lesions may have a violaceous hue and be excruciatingly tender and extremely firm. Lesions are most commonly seen on the lower extremities, not over bony prominences. The incidence of these lesions is very low in general patient populations.

Fournier’s gangrene: Intensely painful necrotizing fasciitis of the perineum. May manifest initially as cellulitis.

Hematoma: Deep-seated purple nodule from clotted blood; usually associated with trauma.

Perirectal abscess: First sign commonly is a dull, aching, or throbbing pain in the perianal area. The pain worsens when sitting and immediately before defecation; the pain abates after defecation. A tender, fluctuant mass may be palpated at the anal verge. These abscesses can open to reveal large cavities, which can be confused with deep pressure ulcers.

CLINICAL CONSULT

A: Referral received to evaluate a large purple discolored area over left trochanter. An 87-year-old male admitted to general medicine after falling at home and lying trapped between his toilet and bathtub on the floor for between 24 and 36 hours. Trochanter painful to light palpation. No fractures per radiologic evaluation. Purple discolored area 24 cm × 12 cm, boggy upon palpation.

D: Suspected deep tissue injury (sDTI).

P: Pressure relief to trochanter; treat dehydration and restore fluid imbalance.

I: (1) Initiate low air loss mattress. (2) Reposition every 2 hours in 30 degree side position to offload coccyx and trochanters. (3) Physical therapy to assess balance and safety of ambulation. (4) No topical dressing over trochanter; monitor site twice daily for changes in appearance.

E: The definitive pressure ulcer diagnosis will be made over time, with reassessment determining if the purple tissue returns to normal color and remains intact or sloughs to reveal existing tissue damage, Stage III, IV, or unstageable 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. Although not all pressure ulcers are avoidable, pressure ulcers now are more commonly considered preventable and unacceptable (in most cases) and are considered an indicator of quality care.

REFERENCES

- Ahn C, Mulligan P, Salcido RS: Smoking—the bane of wound healing: biomedical interventions and social influences, *Adv Skin Wound Care* 21(5):227–236, 2008.
- Agency for Healthcare Research and Quality (AHRQ): *Preventing pressure ulcers in hospitals: a toolkit for improving quality of care*, Rockville, MD, April 2011, AHRQ. <http://www.ahrq.gov/professionals/systems/long-term-care/resources/pressure-ulcers/pressureulcertoolkit/putool1.html>. Accessed Feb 8, 2014.
- Ankrom MA, Bennett RG, Sprigle S, et al: Pressure-related deep tissue injury under intact skin and the current pressure ulcer staging systems, *Adv Skin Wound Care* 18(1):35–42, 2005.
- Antokal S, Brienza D, Bryan N, et al: *Friction induced skin injuries – are they pressure ulcers?* (National Pressure Ulcer Advisory Panel white paper), Nov 20, 2012. <http://www.npuap.org/wp-content/uploads/2012/01/NPUAP-Friction-White-Paper.pdf>. Accessed May 27, 2014.
- Arnold HL, Decubitus: the word. In: Parish LC, Witkowski JA, Crissey JT (1983) *The decubitus ulcer*. Masson, New York, pp 1–3
- Aronovitch SA: Intraoperatively acquired pressure ulcers: are there common risk factors? *Ostomy Wound Manage* 53(2):57–69, 2007.
- Australian Wound Management Association: *Clinical practice guideline for the prevention and management of pressure injury*, Osborne Parke, Western Australia, 2012, Cambridge Media.
- Baharestani MM: Pressure ulcers in pediatric populations. In Pieper B, editor (with the National Pressure Ulcer Advisory Panel [NPUAP]): *Pressure ulcers: prevalence, incidence, and implications for the future*, Washington, DC, 2012, NPUAP, pp 151–171.
- Banks M, Bauer J, Graves N, et al: Malnutrition and pressure ulcer risk in adults in Australian health care facilities, *Nutrition* 26(9):896–901, 2010.
- Baumgarten M, Margolis D, Gruber-Baldini AL, et al: Pressure ulcers and the transition to long-term care, *Adv Skin Wound Care* 16(6):299–304, 2003.
- Bennett LM, Kavner D, Lee BY, et al: Skin stress and blood flow in sitting paraplegic patients, *Arch Phys Med Rehabil* 65(4):186–190, 1984.
- Bergquist-Beringer S, Gajewski BJ: Outcome and assessment information set data that predict pressure ulcer development in older adult home health patients, *Adv Skin Wound Care*, 24(9):404–414, 2011.
- Bergquist-Beringer S, Gajewski BJ, Davidson J: Pressure ulcer prevalence and incidence: report from the National Database of Nursing Quality Indicators (NDNQI). In Pieper B, editor (with the National Pressure Ulcer Advisory Panel [NPUAP]): *Pressure ulcers: prevalence, incidence, and implications for the future*, Washington, DC, 2012, NPUAP, pp 175–188.
- Berlowitz D: Prevalence, incidence, and facility-acquired rates. In Pieper B, editor (with the National Pressure Ulcer Advisory Panel [NPUAP]): *Pressure ulcers: prevalence, incidence, and implications for the future*, Washington, DC, 2012, NPUAP, pp 19–24.
- Berlowitz DR, Brienza DM: Are all pressure ulcers the result of deep tissue injury? A review of the literature, *Ostomy Wound Manage* 53(10):34–38, 2007.
- Black J, Baharestani MM, Cuddigan J, et al: National Pressure Ulcer Advisory Panel's updated pressure ulcer staging system, *Adv Skin Wound Care* 20(5):269–274, 2007.
- Black J, Langemo D: Pressure ulcer staging/categorization. In Pieper B, editor (with the National Pressure Ulcer Advisory Panel [NPUAP]): *Pressure ulcers: prevalence, incidence, and implications for the future*, Washington, DC, 2012, NPUAP, pp 5–14.
- Black JM, Cuddigan JE, Walko MA, et al: Medical device related pressure ulcers in hospitalized patients, *Int Wound J* 7(5):358–365, 2010.
- Blackett A, Gallagher S, Dugan S, et al: Caring for persons with bariatric health care issues, *J Wound Ostomy Continence Nurs* 38(2):133–138, 2011.
- Bolton II, Girolami S, Corbett L, et al: The Association for the Advancement of Wound Care (AAWC) venous and pressure ulcer guidelines, *Ostomy Wound Manage* 60(11):24–66, 2014.
- Braden BJ, Bergstrom N: A conceptual schema for the study of the etiology of pressure sores, *Rehabil Nurs* 12(1):8–12, 1987.
- Brienza D, Karg P: Pressure ulcers in persons with spinal cord injury. In Pieper B, editor (with the National Pressure Ulcer Advisory Panel [NPUAP]): *Pressure ulcers: prevalence, incidence, and implications for the future*, Washington, DC, 2012, NPUAP, pp 107–112.
- Burton AC, Yamada S: Relation between blood pressure and flow in the human forearm, *J Appl Physiol* 4(5):329–339, 1951.
- Compher C, Kinosian BP, Ratcliffe SJ, et al: Obesity reduces the risk of pressure ulcers in elderly hospitalized patients, *J Gerontol A Biol Sci Med Sci* 62(11):1310–1312, 2007.
- Cowan LJ, Stechmiller JK, Rowe M, et al: Enhancing Braden pressure ulcer risk assessment in acutely ill adult veterans, *Wound Repair Regen* 20(2):137–148, 2012.
- Cox J: Pressure ulcer development and vasopressor agents in adult critical care patients: a literature review, *Ostomy Wound Manage* 59(4):50–54, 56–60, 2013.
- Defloor T: The risk of pressure sores: a conceptual scheme, *J Clin Nurs* 8:206–216, 1999.
- Department of Health and Human Services, Centers for Medicare and Medicaid Services: Medicare program; changes to the hospital inpatient prospective payment systems and fiscal year 2008 rates; final rule, *Federal Register* 72(162), pp 1–12, 2007.
- Dhandapani M, Dhandapani S, Agarwal M, et al: Pressure ulcer in patients with severe traumatic brain injury: significant factors and association with neurological outcome, *J Clin Nurs* 23(7–8):1114–1119, 2014.
- Dorner B, Posthauer ME, Thomas D: The role of nutrition in pressure ulcer prevention and treatment: National Pressure Ulcer

- Advisory Panel white paper, *Adv Skin Wound Care* 22(5): 212–221, 2009.
- Doughty D, Junkin J, Kurz P, et al: Incontinence-associated dermatitis: consensus statements, evidenced-based guidelines for prevention and treatment, and current challenges, *J Wound Ostomy Continence Nurs* 39(3):303–315, 2012.
- Drake DJ, Swanson M, Baker G, et al: The association of BMI and Braden Total Score on the occurrence of pressure ulcers, *J Wound Ostomy Continence Nurs* 37(4):367–371, 2010.
- Ebrecht M, Hextall J, Kirtley LG, et al: Perceived stress and cortisol levels predict speed of wound healing in healthy male adults, *Psychoneuroendocrinology* 29(6):798–809, 2004.
- Edsberg LE: Pressure ulcer tissue histology: an appraisal of current knowledge, *Ostomy Wound Manage* 53(10):40–49, 2007.
- Farid KJ: Applying observations from forensic science to understanding the development of pressure ulcers, *Ostomy Wound Manage* 53(4):26–28, 30, 32 passim, 2007.
- Flattau A, Blank AE: Risk factors for 90-day and 180-day mortality in hospitalized patients with pressure ulcers, *Int Wound J* 11(1):14–20, 2014.
- Fore J: A review of skin and the effects of aging on skin structure and function, *Ostomy Wound Manage* 52(9):24–35, 2006.
- Fowler E, Scott-Williams S, McGuire JB: Practice recommendations for preventing heel pressure ulcers, *Ostomy Wound Manage* 54(10):42–48, 50–52, 54–57, 2008.
- Fox J, Bradley R: *A new medical dictionary*, London, 1803, Darton & Harvey.
- Ganos D, Siddiqui A: Operating room. In Pieper B, editor (with the National Pressure Ulcer Advisory Panel [NPUAP]): *Pressure ulcers: prevalence, incidence, and implications for the future*, Washington, DC, 2012, NPUAP, pp 57–64.
- Garcia AD: Home care. In Pieper B, editor (with the National Pressure Ulcer Advisory Panel [NPUAP]): *Pressure ulcers: prevalence, incidence, and implications for the future*, Washington, DC, 2012, NPUAP, pp 97–104.
- Gefen A: Bioengineering models of deep tissue injury, *Adv Skin Wound Care* 21(1):30–36, 2008.
- Gefen A: The biomechanics of heel ulcers, *J Tissue Viability* 19(4):124–131, 2010.
- Goldberg M: General acute care. In Pieper B, editor (with the National Pressure Ulcer Advisory Panel [NPUAP]): *Pressure ulcers: prevalence, incidence, and implications for the future*, Washington, DC, 2012, NPUAP, pp 27–46.
- Gouin JP, Kiecolt-Glaser JK, Malarkey WB, et al: The influence of anger expression on wound healing, *Brain Behav Immun* 22(5):699–708, 2008.
- Gray M, Beekman D, Bliss DZ, et al: Incontinence-associated dermatitis: a comprehensive review and update, *J Wound Ostomy Continence Nurs* 39(1):61–74, 2012.
- Gray M, Black JM, Baharestani MM, et al: Moisture-associated skin damage: overview and pathophysiology, *J Wound Ostomy Continence Nurs* 38(3):233–241, 2011.
- How-to Guide: *Prevent Pressure Ulcers*, Cambridge, MA, 2011, Institute for Healthcare Improvements. (Available at www.ihl.org).
- Husain T: An experimental study of some pressure effects on tissues, with reference to the bedsore problem, *J Pathol Bacteriol* 66(2):347–358, 1953.
- Institute for Healthcare Improvement: Pressure ulcers. <http://www.ihl.org/Topics/PressureUlcers/Pages/default.aspx>. Accessed July 18, 2015.
- Kalava UR, Cha SS, Takahashi PY: Association between vitamin D and pressure ulcers in older ambulatory adults: results of a matched case-control study, *Clin Interv Aging* 6:213–219, 2011.
- Keast DH, Parslow N, Houghton PE, et al: Best practice recommendations for the prevention and treatment of pressure ulcers: update 2006, *Adv Skin Wound Care* 20(8):337–360, 2007.
- Keelaghan E, Margolis D, Zhan M, et al: Prevalence of pressure ulcers on hospital admission among nursing home residents transferred to the hospital, *Wound Repair Regen* 16(3):331–336, 2008.
- Knudsen A, Gallagher S: Care of the obese patient with pressure ulcers, *J Wound Ostomy Continence Nurs* 30(2):111–118, 2003.
- Kosiak M: Etiology of decubitus ulcers, *Arch Phys Med Rehabil* 42:19–29, 1961.
- Kosiak M, Kubicek WG, Olson M, et al: Evaluation of pressure as a factor in the production of ischial ulcers, *Arch Phys Med Rehabil* 39(10):623–629, 1958.
- Krapfl LA: Inpatient prospective payment changes: a guide for the WOC nurse, *WOC News* 3:16, 2008.
- Krouskop TA: A synthesis of the factors that contribute to pressure sore formation, *Med Hypotheses* 11(2):255–267, 1983.
- Kumar V, Fausto N, Abbas A: Acute and chronic inflammation. In *Robbins and Cotran pathologic basis of disease*, ed 7, Philadelphia, 2005a, Saunders.
- Kumar V, Fausto N, Abbas A: Cellular adaptations, cell injury, and cell death. In *Robbins and Cotran pathologic basis of disease*, ed 7, Philadelphia, 2005b, Saunders.
- Landi F, Onder G, Russo A, et al: Pressure ulcer and mortality in frail elderly people living in community, *Arch Gerontol Geriatr* 44(Suppl 1):217–223, 2007.
- Langer G, Schloemer G, Knerr A, et al: Nutritional interventions for preventing and treating pressure ulcers, *Cochrane Database Syst Rev* (4):CD003216, 2003.
- Lindan O: Etiology of decubitus ulcers: an experimental study, *Arch Phys Med Rehabil* 42:774–783, 1961.
- Man SP, Au-Yeung TW: Hypotension is a risk factor for new pressure ulcer occurrence in older patients after admission to an acute care hospital, *J Am Med Dir Assoc* 14(8):627.e1–5, 2013.
- Mathison CJ: Skin and wound care challenges in the hospitalized morbidly obese patient, *J Wound Ostomy Continence Nurs* 30(2):78–83, 2003.
- Mayrovitz HN, Sims N, Taylor MC, et al: Effects of support surface relief pressures on heel skin blood perfusion, *Adv Skin Wound Care* 16(3):141–145, 2003.
- Muthuchamy M, Zawieja D: Molecular regulation of lymphatic contractility, *Ann N Y Acad Sci* 1131:89–99, 2008.
- Nakagami G, Sanada H, Konya C, et al: Comparison of two pressure ulcer preventive dressings for reducing shear force on the heel, *J Wound Ostomy Continence Nurs* 33(3):267–272, 2006.
- National Pressure Ulcer Advisory Panel (NPUAP): *Mucosal pressure ulcers: an NPUAP position statement*, 2009. http://www.npuap.org/Mucosal_Pressure_Ulcer_Position_Statement_final.pdf. Accessed May 27, 2014.
- National Pressure Ulcer Advisory Panel (NPUAP), European Pressure Ulcer Advisory Panel (EPUAP) and Pan Pacific Pressure Ulcer Injury Alliance (PPPIA): Prevention and treatment of pressure ulcers: clinical practice guideline. In Haesler E, editor: Cambridge Media:Osborne Park, Western Australia, 2014.
- Niezgoda JA, Mendez-Eastman S: The effective management of pressure ulcers, *Adv Skin Wound Care* 19(Suppl 1):3–15, 2006.

- Olesen CG, de Zee M, Rasmussen J: Missing links in pressure ulcer research—an interdisciplinary overview, *J Appl Physiol* 108(6):1458–1464, 2010.
- Peterson M, Schwab W, McCutcheon K, et al: Effects of elevating the head of bed on interface pressure in volunteers, *Crit Care Med* 36(11):3038–3042, 2008.
- Pieper B: Long term care/nursing homes. In Pieper B, editor (with the National Pressure Ulcer Advisory Panel [NPUAP]): *Pressure ulcers: prevalence, incidence, and implications for the future*, Washington, DC, 2012, NPUAP, pp 65–88.
- Pieper B, Langemo D, Cuddigan J: Pressure ulcer pain: a systematic literature review and National Pressure Ulcer Advisory Panel white paper, *Ostomy Wound Manage* 55(2):16–31, 2009.
- Pittman J: Effect of aging on wound healing, *J Wound Ostomy Continence Nurs* 34(4):412–415, 2007.
- Posthauer ME: The role of nutrition in wound care, *Adv Skin Wound Care* 19(1):43–52, 2006.
- Price MC, Whitney JD, King CA, et al: Development of a risk assessment tool for intraoperative pressure ulcers, *J Wound Ostomy Continence Nurs* 32(1):19–30, 2005.
- Qaseem A, Humphrey LL, Forcica MA, et al: Treatment of pressure ulcers: a clinical practice guideline from the American College of Physicians, *Ann Intern Med* 162:370–379, 2015.
- Qaseem A, Mir TP, Starkey M, et al: Risk assessment and prevention of pressure ulcers: a clinical practice guideline from the American College of Physicians, *Ann Intern Med* 162:359–369, 2015.
- Rabadi MH, Vincent AS: Do vascular risk factors contribute to the prevalence of pressure ulcer in veterans with spinal cord injury? *J Spinal Cord Med* 34(1):46–51, 2011.
- Rapp MP, Bergstrom N, Padhye NS: Contribution of skin temperature regularity to the risk of developing pressure ulcers in nursing facility residents, *Adv Skin Wound Care* 22(11):506–513, 2009.
- Reddy M: Skin and wound care: important considerations in the older adult, *Adv Skin Wound Care* 21(9):424–436, 2008.
- Redelings MD, Lee NE, Sorvillo F: Pressure ulcers: more lethal than we thought? *Adv Skin Wound Care* 18(7):367–372, 2005.
- Registered Nurses Association of Ontario (RNAO): *Risk assessment and prevention of pressure ulcers*, Toronto, ON, 2011, RNAO.
- Russo CA, Steiner C, Spector W: *Hospitalizations related to pressure ulcers among adults 18 years and older*, 2006 (HCUP statistical brief no. 64), 2008. <http://www.hcup-us.ahrq.gov/reports/statbriefs/sb64.pdf>. Accessed Dec 20, 2013.
- Schubart JR, Hilgart M, Lyder C: Pressure ulcer prevention and management in spinal cord-injured adults: analysis of educational needs, *Adv Skin Wound Care* 21(7):322–327, 2008.
- Sibbald RG, Krasner DL, Woo KY: Pressure ulcer staging revisited: superficial skin changes & deep pressure ulcer framework, *Adv Skin Wound Care* 24(12):571–580, 2011.
- Sieggreen M: Pressure ulcers in persons of color: race and ethnicity. In Pieper B, editor (with the National Pressure Ulcer Advisory Panel [NPUAP]): *Pressure ulcers: prevalence, incidence, and implications for the future*, Washington, DC, 2012a, NPUAP, pp 141–146.
- Sieggreen M: Pressure ulcers in morbidly obese patients. In Pieper B, editor (with the National Pressure Ulcer Advisory Panel [NPUAP]): *Pressure ulcers: prevalence, incidence, and implications for the future*, Washington, DC, 2012b, NPUAP, pp 147–150.
- Siem CA, Wipke-Tevis DD, Rantz MJ, et al: Skin assessment and pressure ulcer care in hospital-based skilled nursing facilities, *Ostomy Wound Manage* 49(6):42–44, 46, 48 passim, 2003.
- Smith BM, Guihan M, LaVela SL, et al: Factors predicting pressure ulcers in veterans with spinal cord injuries, *Am J Phys Med Rehabil* 87(9):750–757, 2008.
- Sonenblum SE, Sprigle SH: The impact of tilting on blood flow and localized tissue loading, *J Tissue Viability* 20(1):3–13, 2011.
- Sonenblum SE, Vonk TE, Janssen TW, et al: Effects of wheelchair cushions and pressure relief maneuvers on ischial interface pressure and blood flow in people with spinal cord injury, *Arch Phys Med Rehabil* 2014;95(7):1350–1357.
- Sopher R, Gefen A: Effects of skin wrinkles, age and wetness on mechanical loads in the stratum corneum as related to skin lesions, *Med Biol Eng Comput* 49(1):97–105, 2011.
- Sprigle S, Sonenblum S: Assessing evidence supporting redistribution of pressure for pressure ulcer prevention: a review, *J Rehabil Res Dev* 48(3):203–213, 2011.
- Stechmiller JK, Cowan L, Whitney JD, et al: Guidelines for the prevention of pressure ulcers, *Wound Repair Regen* 16(2):151–168, 2008.
- Stojadinovic O, Minkiewicz J, Sawaya A, et al: Deep tissue injury in development of pressure ulcers: a decrease of inflammasome activation and changes in human skin morphology in response to aging and mechanical load, *PLoS One* 8(8):e69223, 2013.
- Stratton RJ, Ek AC, Engfer M, et al: Enteral nutritional support in prevention and treatment of pressure ulcers: a systematic review and meta-analysis, *Ageing Res Rev* 4(3):422–450, 2005.
- Terekeci H, Kucukardali Y, Top C, et al: Risk assessment study of the pressure ulcers in intensive care unit patients, *Eur J Intern Med* 20(4):394–397, 2009.
- Theisen S, Drabik A, Stock S: Pressure ulcers in older hospitalized patients and its impact on length of stay: a retrospective observational study, *J Clin Nurs* 21(3–4):380–387, 2012.
- Tsutakawa S, Kobayashi D, Kusama M, et al: Nicotine enhances skin necrosis and expression of inflammatory mediators in a rat pressure ulcer model, *Br J Dermatol* 161(5):1020–1027, 2009.
- U.S. Department of Health and Human Services: *Healthy People, 2010*, Washington, DC, 2000, HHS.
- Van Gilder C, Amlung S, Harrison P, et al: Results of the 2008–2009 International Pressure Ulcer Prevalence Survey and a 3-year, acute care, unit-specific analysis, *Ostomy Wound Manage* 55(11):39–45, 2009.
- Wong V: Skin blood flow response to 2-hour repositioning in long-term care residents: a pilot study, *J Wound Ostomy Continence Nurs* 38(5):529–537, 2011.
- Wound, Ostomy and Continence Nurses Society™ (WOCN®): *Guideline for prevention and management of pressure ulcers*, Glenview, IL, 2010, WOCN®.