Pressure ulcers: Pathophysiology, epidemiology, risk factors, and presentation

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Learning objectives
After completing this learning activity, participants should be able to describe the burden that pressure ulcers pose to the individual and society; explain the pathophysiology of pressure ulcers, including the roles of pressure, shear, and friction; identify at-risk populations and discuss the elements of risk assessment and utility of risk assessment tools; and classify pressure ulcers according to the updated NPUAP staging system.

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Though preventable in most cases, pressure ulcers continue to pose a major burden to the individual and society, affecting ≤3 million adults annually in the United States alone. Despite increased national attention over the past 20 years, the prevalence of pressure ulcers has largely remained unchanged, while the associated costs of care continue to increase. Dermatologists can play a significant role in pressure ulcer prevention by becoming aware of at-risk populations and implementing suitable preventive strategies. Moreover, dermatologists should be able to recognize early changes that occur before skin breakdown and to properly identify and stage pressure ulcers to prevent delay of appropriate care. The aim of the first article in this continuing medical education series is to discuss the pathophysiology, risk factors, epidemiology, social and economic burdens, and clinical presentation of pressure ulcers. (J Am Acad Dermatol 2019;81:881-90.)

Key words: chronic wounds; epidemiology; pathophysiology; presentation; prevention; pressure injury; pressure sore; pressure ulcer; risk factors; staging; wound healing; wounds.

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Abbreviations used:

ICU: intensive care unit
NPUAP: National Pressure Ulcer Advisory Panel

Once thought to be an unavoidable consequence of paraplegia or infirmity, tissue damage caused by sustained pressure has long been known to exist. The renowned British surgeon Sir James Paget assessed this form of injury in 1873, noting: "The sloughing and mortification or death of a part produced by pressure…Sloughing follows these in the skin and subcutaneous tissue and fat. These latter die before the skin as sloughing proceeds faster in them, so when the skin comes away, the place formerly occupied by these tissues is empty."[1] This description is remarkably accurate given what we know today. Nonetheless, it was only with the start of World War I and the parallel modernization of nursing that people widely began to appreciate that pressure ulcers could be prevented and treated.[2]

Often referred to as pressure ulcers in the modern vernacular, many terms have been used to describe pressure-induced wounds, including decubitus ulcer, pressure sore, and bedsore. Notably, decubitus—"to lie down" in Latin—does not accurately describe these ulcers because they may occur in any position of prolonged pressure. In addition, in 2016, the National Pressure Ulcer Advisory Panel (NPUAP) released new terminology guidelines, redubbing the preferred name as "pressure injury" to better reflect all forms of tissue damage caused by pressure, including the stage before skin breakdown.[3] We will use "pressure ulcer" in this continuing medical education article because it is still the most widely used and accepted terminology.

The NPUAP defines a pressure ulcer as "localized damage to the skin and underlying soft tissue usually over a bony prominence or related to a medical or other device…as a result of intense and/or prolonged pressure or pressure in combination with shear."[3] The most common locations in adults are over the bony prominences of the sacral and hip regions, though the lower extremities are affected in ≤25% of cases.[4,5] While less often considered, neonatal and pediatric patients also suffer from pressure ulcers, which are most common over the occiput in these populations.[6]

Moreover, although pressure ulcers have been given substantial consideration within hospitals and long-term care facilities in recent decades, they remain a significant problem. In the United States alone, pressure ulcers affect ≤3 million adults annually and result in a diminished quality of life, high costs for the individual and health care system, and significantly increased morbidity and mortality. This purpose of the first article in this continuing medical education series is to discuss the pathophysiology, risk factors, epidemiology, social and economic burdens, and clinical presentation of pressure ulcers. The second article in this series will focus on prevention and treatment strategies.

PATHOPHYSIOLOGY

Key points

- **Sustained pressure over a bony prominence ultimately leads to tissue ischemia and necrosis**
- **Combination of shear and friction while lying at an incline may affect underlying capillary beds and contribute to local tissue hypoxia**
- **Excess moisture can lead to maceration and contribute to skin breakdown**

In individuals with normal sensation, mobility, and mental status, prolonged pressure elicits a feedback response that prompts a change in body position; however, when the feedback response is absent or impaired, sustained pressure ultimately leads to tissue ischemia, injury, and necrosis. Pressure ulcers typically begin when the individual's body weight exerts a downward force on the skin and subcutaneous tissue that lie between a bony prominence and an external surface, such as a mattress or wheelchair cushion. Sustained pressure from medical devices may also cause pressure injuries. It is generally thought that force that results in an external pressure more than the arterial capillary filling pressure, around 32 mm Hg, and more than the venous capillary outflow pressure, around 8 to 12 mm Hg, inhibits blood flow and results in local tissue hypoxia.[7] While some have questioned these particular threshold pressures, the centrality of ischemia and sustained pressure to the etiology of pressure ulcers is widely accepted.[8] Sustained external pressures above a threshold causes prolonged ischemia and sets the tissue down a path toward necrosis. Reperfusion injury, which occurs because of the return of blood supply after a period of ischemia, has been posited as an additional source of tissue damage leading to pressure ulcers.[9,10] Reperfusion of ischemic tissue may cause increased formation of reactive oxygen species and trigger an inflammatory response. In rats, multiple ischemia–reperfusion cycles have

been shown to cause more tissue damage than continuous ischemia alone.[11]

The highest pressures often occur at the interface of bone and muscle, causing necrosis at this depth while leaving the skin relatively spared.[12,13] Likewise, the effects of hypoxia and risk of tissue damage are initially greatest in muscle, followed by subcutaneous tissue and then skin, likely reflecting their respective metabolic requirements.[14] Therefore, at the point when skin ulceration is observed, extensive deep tissue injury is likely to have already occurred.

In addition, as reflected in the newest NPUAP guidelines,[3] shear and friction, as when lying at an incline, may affect local capillary beds and are thought to contribute to tissue hypoxia.[15] When lying at an angle, the downward force of gravity is countered by friction, which prevents the person from sliding down in the bed. Though the skin may not move down the bed, internal structures like muscle and bone that are not in contact with an external surface are displaced downward because of gravity. These shearing forces can disrupt blood flow as vessels caught between the skin and bone are distorted or compressed.[16,17]

Finally, excess moisture from either perspiration or incontinence can macerate the skin, making it more susceptible to breakdown with friction and repositioning.[18,19]

EPIDEMIOLOGY

Key points

- **Pressure ulcers are a significant problem worldwide and affect ≤3 million people in the United States**
- **The overall prevalence of pressure ulcers in hospitalized patients has been estimated to range from 5% to 15% but may be significantly higher in intensive care units and certain long-term care settings**

Pressure ulcers are a significant problem worldwide.[5,20-22] Recent epidemiologic data regarding pressure ulcers in the United States are somewhat limited, but the incidence has been estimated at around 1 to 3 million per year.[23-25] Among hospitalized patients, the reported prevalence rates vary significantly, affecting 5% to 15% of patients overall[5,26] but affecting consistently higher percentages of patients in intensive care units (ICUs).[27] The 1999 National Pressure Ulcer Prevalence Survey, which included >350 acute care facilities and 42,000 patients, found that the overall prevalence of pressure ulcers was 14.8%, with 7.1% of ulcers occurring during a hospital stay.[28] Pressure ulcers were seen in 21.5% of patients in ICUs, and the elderly were more at risk, with the highest prevalence at 29% among patients 71 to 80 years of age.[28] The National Pressure Ulcer Prevalence Survey was repeated 5 times between 1999 and 2005.[5] By 2005, data from 651 facilities with 85,838 patients, including acute care (533 facilities, 74,401 patients), long-term acute care (38 facilities, 1983 patients), and long-term care (52 facilities, 6242 patients), had been compiled. Between 1999 and 2005, the prevalence of all pressure ulcers was constant, at around 15% overall and 25% in ICUs.[5] Pressure ulcers were most prevalent in long-term acute care facilities (23-27%), while acute care and long-term care facility prevalence rates ranged from 13% to 15%. Hospital-acquired pressure ulcers were consistent around 7.5% overall and similar across facility type.[5] Another study of Medicare beneficiaries hospitalized between 2006 and 2007 found that 4.5% of patients developed a pressure ulcer during their hospital stays.[29] Moreover, Keelaghan et al[30] found that among newly hospitalized patients, ≤26.2% of those admitted from nursing homes compared with 4.8% of those admitted from other living situations were found to have pressure ulcers.

Patients with neurologic impairments have a lifetime risk of developing a pressure ulcer that ranges from 25% to 85%.[31] Up to middle age, pressure ulcers are more prevalent in men because of the increased number of men with traumatic spinal cord injuries; however, among the elderly, prevalence between sexes is nearly equal, which likely reflects longer life expectancy in women.[5] Some data suggest that darker-skinned patients have a higher risk of pressure ulcer development,[32] which may in part be explained by increased difficulty in recognizing nonblanching erythema before skin breakdown.[33]

Up to roughly 25% of patients in neonatal and pediatric ICUs may develop pressure ulcers, while incidence rates among noncritical hospitalized children have been reported to range from 0.3% to 6%.[6]

AT-RISK POPULATIONS

Key points

- **Anyone, including children and neonates, is susceptible to pressure ulcers in the setting of sustained pressure**
- **The greatest risk for pressure ulcers is in people with impaired mobility or sensation who are generally bed- or wheelchair-bound**
- **Natural skin changes with aging are an additional risk factor in elderly patients**

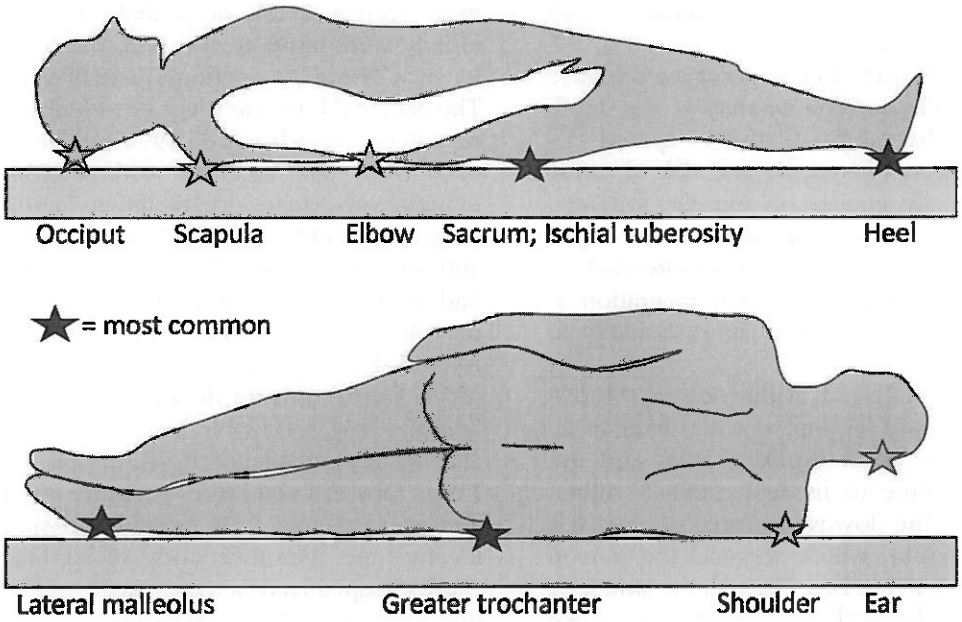

Fig 1. Braden scale for the risk assessment of pressure ulcers.

Anyone experiencing sustained pressures over the skin that are strong enough to cause underlying tissue ischemia is susceptible to pressure ulcers. Typically, this precondition of sustained pressure occurs in people with impaired mobility or sensation, possibly because of spinal cord injury, other neurologic impairment, sedation, peri- or postoperative immobilization, hospitalization, and frailty, among other reasons. Poor nutrition with a subsequent loss of muscle bulk and body mass, commonly seen in both immobilized and elderly populations, accentuates bony prominences and may increase risk of ulceration, either directly because of pressure effects or because of malnutrition. The elderly also have additional risk factors inherent to natural skin aging, including dermal and epidermal thinning, decreased epidermal turnover, and loss of dermal papillae resulting in flattening of the dermoepidermal junction.[34] Consequently, aging skin has less resistance to shear forces and a reduced contiguous surface area between the dermis and epidermis through which nutrient and oxygen transport can occur.[34] Among neonatal and pediatric populations, pressure ulcers are more likely to be related to medical equipment.[35]

Other medical conditions that have been associated with pressure ulcers include cognitive impairment, deep venous thrombosis, impaired microcirculation, congestive heart failure, lower extremity edema, diabetes, and rheumatoid arthritis.[36]

ECONOMIC IMPACT

Key points

- **Medicare and Medicaid have not paid for hospital-acquired pressure ulcers since 2008, costing hospitals >$11 billion annually**
- **Models have shown that implementing preventive strategies ultimately lowers costs**

A recent analysis of a Medicare data set found that after arterial ulcers, pressure ulcers are the costliest chronic wounds.[37] Since 2008, Medicare and Medicaid have not paid for hospital-acquired pressure ulcers, putting the onus on hospitals to focus on prevention. Hospital-acquired pressure ulcers alone cost >$11 billion annually.[23] The average cost of a hospital stay for patients with pressure ulcers is $72,000 compared with $32,000 for those without pressure ulcers.[38] A study from the United Kingdom found that average individual cost of pressure ulcer treatment ranged from $1500 for stage 1 to $18,000 for stage 4 ulcers.[39]

Multiple cost-effectiveness analyses have found that the cost of prevention strategies is less than the cost of treatment.[23,40,41] One model has shown that implementation of effective prevention methods could lower costs ($7300 vs $10,100 in standard care approach) and increase quality-adjusted life years (11.2 vs 9.3).[23] The implementation of prevention strategies, however, may be challenging, given the increased up-front costs and necessary changes to established protocols and workflow.

BRADEN SCALE FOR PREDICTING PRESSURE SORE RISK

Patient's Name _____ Evaluator's Name _____ Date of Assessment

SENSORY PERCEPTION ability to respond meaningfully to pressure-related discomfort	**1. Completely Limited** Unresponsive (does not moan, flinch, or grasp) to painful stimuli, due to diminished level of consciousness or sedation. OR limited ability to feel pain over most of body	**2. Very Limited** Responds only to painful stimuli. Cannot communicate discomfort except by moaning or restlessness OR has a sensory impairment which limits the ability to feel pain or discomfort over ½ of body.	**3. Slightly Limited** Responds to verbal commands, but cannot always communicate discomfort or the need to be turned. OR has some sensory impairment which limits ability to feel pain or discomfort in 1 or 2 extremities.	**4. No Impairment** Responds to verbal commands. Has no sensory deficit which would limit ability to feel or voice pain or discomfort..				
MOISTURE degree to which skin is exposed to moisture	**1. Constantly Moist** Skin is kept moist almost constantly by perspiration, urine, etc. Dampness is detected every time patient is moved or turned.	**2. Very Moist** Skin is often, but not always moist. Linen must be changed at least once a shift.	**3. Occasionally Moist:** Skin is occasionally moist, requiring an extra linen change approximately once a day.	**4. Rarely Moist** Skin is usually dry, linen only requires changing at routine intervals.				
ACTIVITY degree of physical activity	**1. Bedfast** Confined to bed.	**2. Chairfast** Ability to walk severely limited or non-existent. Cannot bear own weight and/or must be assisted into chair or wheelchair.	**3. Walks Occasionally** Walks occasionally during day, but for very short distances, with or without assistance. Spends majority of each shift in bed or chair	**4. Walks Frequently** Walks outside room at least twice a day and inside room at least once every two hours during waking hours				
MOBILITY ability to change and control body position	**1. Completely Immobile** Does not make even slight changes in body or extremity position without assistance	**2. Very Limited** Makes occasional slight changes in body or extremity position but unable to make frequent or significant changes independently.	**3. Slightly Limited** Makes frequent though slight changes in body or extremity position independently.	**4. No Limitation** Makes major and frequent changes in position without assistance.				
NUTRITION usual food intake pattern	**1. Very Poor** Never eats a complete meal. Rarely eats more than ⅓ of any food offered. Eats 2 servings or less of protein (meat or dairy products) per day. Takes fluids poorly. Does not take a liquid dietary supplement OR is NPO and/or maintained on clear liquids or IV's for more than 5 days.	**2. Probably Inadequate** Rarely eats a complete meal and generally eats only about ½ of any food offered. Protein intake includes only 3 servings of meat or dairy products per day. Occasionally will take a dietary supplement. OR receives less than optimum amount of liquid diet or tube feeding	**3. Adequate** Eats over half of most meals. Eats a total of 4 servings of protein (meat, dairy products per day. Occasionally will refuse a meal, but will usually take a supplement when offered OR is on a tube feeding or TPN regimen which probably meets most of nutritional needs	**4. Excellent** Eats most of every meal. Never refuses a meal. Usually eats a total of 4 or more servings of meat and dairy products. Occasionally eats between meals. Does not require supplementation.				
FRICTION & SHEAR	**1. Problem** Requires moderate to maximum assistance in moving. Complete lifting without sliding against sheets is impossible. Frequently slides down in bed or chair, requiring frequent repositioning with maximum assistance. Spasticity, contractures or agitation leads to almost constant friction	**2. Potential Problem** Moves feebly or requires minimum assistance. During a move skin probably slides to some extent against sheets, chair, restraints or other devices. Maintains relatively good position in chair or bed most of the time but occasionally slides down.	**3. No Apparent Problem** Moves in bed and in chair independently and has sufficient muscle strength to lift up completely during move. Maintains good position in bed or chair.					
					Total Score			

Fig 2. Common sites of pressure ulcers.

PSYCHOSOCIAL IMPACT

Key point

- **Pressure ulcers have significant physical, social, and psychological impacts that can significantly affect quality of life**

Apart from the serious medical complications that can arise, living with a pressure ulcer can have physical, social, and psychological impacts that significantly affect quality of life.[42-44] Undergoing treatment, be it in the hospital, clinic, or home, often necessitates reduced physical activity and time away from one's daily routine and usual social activities. Bauer et al[38] found that hospitalized patients with pressure ulcers had a median length of stay of 7 days versus 3 days for those without pressure ulcers. Decreased independence, social isolation, pain, fear, and anxiety have all been reported to be common to the experience of living with a pressure ulcer.[44-46]

RISK ASSESSMENT

Key points

- **Risk-assessment tools can help identify at-risk patients, but evidence for their efficacy in lower pressure ulcer incidence is lacking**
- **Clinical judgment may be as valuable as the commonly used risk-assessment tools currently available**

At-risk patients require a thorough assessment that incorporates a detailed medical history, skin examination, and evaluation of patient support systems. Risk assessment instruments have been developed to identify individuals who are at greatest risk and to reduce the incidence of pressure ulcers, with the idea being that at-risk individuals may then benefit from more rigorous interventions.[47] Agreement on the predictive risk factors is lacking, however,[48] which has led to the proliferation of various tools that include diverse variables of

Table I. National Pressure Ulcer Advisory Panel staging system*

Pressure injury stage	Description	Other notes
1	Nonblanchable erythema of intact skin	Blanchable erythema or sensory changes may precede development of stage 1 injury; purple or maroon discoloration indicates deep tissue pressure injury
2	Partial-thickness skin loss with exposed dermis	Adipose or deeper tissues are not exposed; often caused by adverse microclimate and shear
3	Full-thickness skin loss	Adipose tissue is visible in the ulcer bed, which may have undermining and tunneling; fascia, muscle, tendon, ligament, cartilage, or bone is not exposed
4	Full-thickness skin and tissue loss	Fascia, muscle, tendon, ligament, cartilage, or bone is exposed; undermining, tunneling, and epibole may be present
Unstageable pressure injury	Obscured full-thickness skin and tissue loss	Extent of tissue damage within the ulcer is obscured by slough or eschar and cannot be determined; removal of slough or eschar reveals a stage 3 or 4 pressure injury
Deep tissue pressure injury	Persistent nonblanchable deep red, maroon, or purple discoloration	May be seen with intact or nonintact skin

*Data from Edsberg et al.[3]

interest. The Braden (Fig 1), Norton, and Waterlow scales are the most commonly used risk assessment tools for lowering the incidence of pressure ulcers.[49-52] Studies of the effectiveness of risk assessment instruments have yielded mixed results.[53] In general, these scales have all shown low sensitivity and specificity in identifying at-risk patients.[54] Likewise, there is no current evidence that these tools are superior to clinical judgment in lowering pressure ulcer incidence,[47] though few high-quality studies have been carried out. A randomized comparison of nurses using the Braden scale (n = 74), unstructured risk assessment (n = 106), or training plus unstructured risk assessment (n = 76) found no statistical difference in pressure ulcer incidence among hospitalized patients.[55] A single-blinded randomized controlled trial comparing the Waterlow scale (n = 411), the Ramstadius screening tool (n = 420), and nurses' clinical judgment (n = 420) revealed no difference in pressure ulcer incidence in hospitalized patients.[56] Moreover, studies have not stratified by care setting or patient subgroups.[54] The ability to develop one risk assessment instrument that has validity across all care settings and patient populations is unlikely, particularly given that predisposing risk factors may vary by clinical setting.[47,57]

CLINICAL PRESENTATION

Key points

- **Common locations for pressure ulcers include over the sacrum, ischial tuberosity, greater trochanter, heel, and lateral malleolus**
- **All pressure ulcers should be staged according to the most recent NPUAP staging system**
- **Undermining and tunneling should always be assessed along with standard wound measurements**

Approximately 70% of pressure ulcers occur over the sacrum, ischial tuberosity, or greater trochanter, while 15% to 25% occur on the lower extremities, typically the heel or lateral malleolus (Fig 2).[4,5] Though these locations are the most classic, pressure ulcers can occur at any site of prolonged pressure, including the elbow, ear, nose, chest, and back.

Staging

Several pressure ulcer classification scales have been used,[58-60] but the NPUAP staging system, first devised in 1989 and most recently revised in 2016,[3] has been widely adopted. The newest system defines 6 classifications (Table I; Figs 3 and 4). Pressure ulcers should be staged after cleaning the wound bed to ensure optimal visualization of the anatomy. If obscured by adherent slough or eschar, the pressure ulcer is classified as "unstageable."

In addition to the 6 defined stages, 2 types of pressure injury are newly defined by the NPUAP.[3] "Medical device-related pressure injury" refers to prolonged pressure from diagnostic or therapeutic devices and should be staged no differently than other pressure ulcers. "Mucosal membrane pressure injury" is caused by the presence of a medical device over a mucous membrane and cannot be staged.

Of note, pressure ulcers should be staged according to their maximum historical depth.

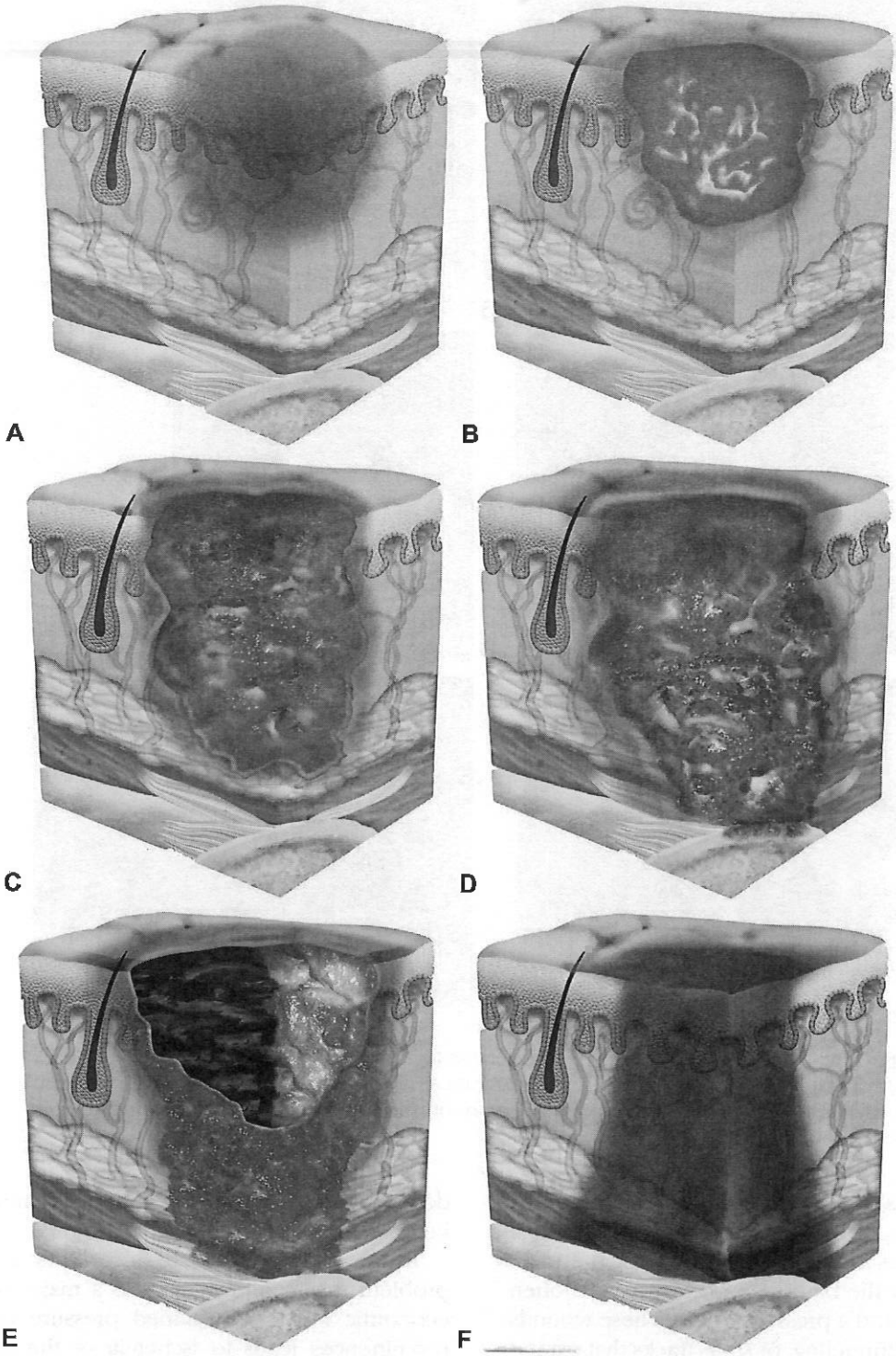

Fig 3. Pressure ulcer stage diagrams. **A**, Stage 1. **B**, Stage 2. **C**, Stage 3. **D**, Stage 4. **E**, Unstageable pressure injury. **F**, Deep tissue pressure injury. Used with permission of the National Pressure Ulcer Advisory Panel.

Accordingly, a pressure ulcer that is initially stage 3 but progresses to stage 4 over the course of treatment is now classified as stage 4; however, as this ulcer heals, it should not again be referred to as a stage 3 or lesser ulcer. The NPUAP has advised against such "reverse staging"[61] because reepithelialization may precede the healing of deeper tissue.[62]

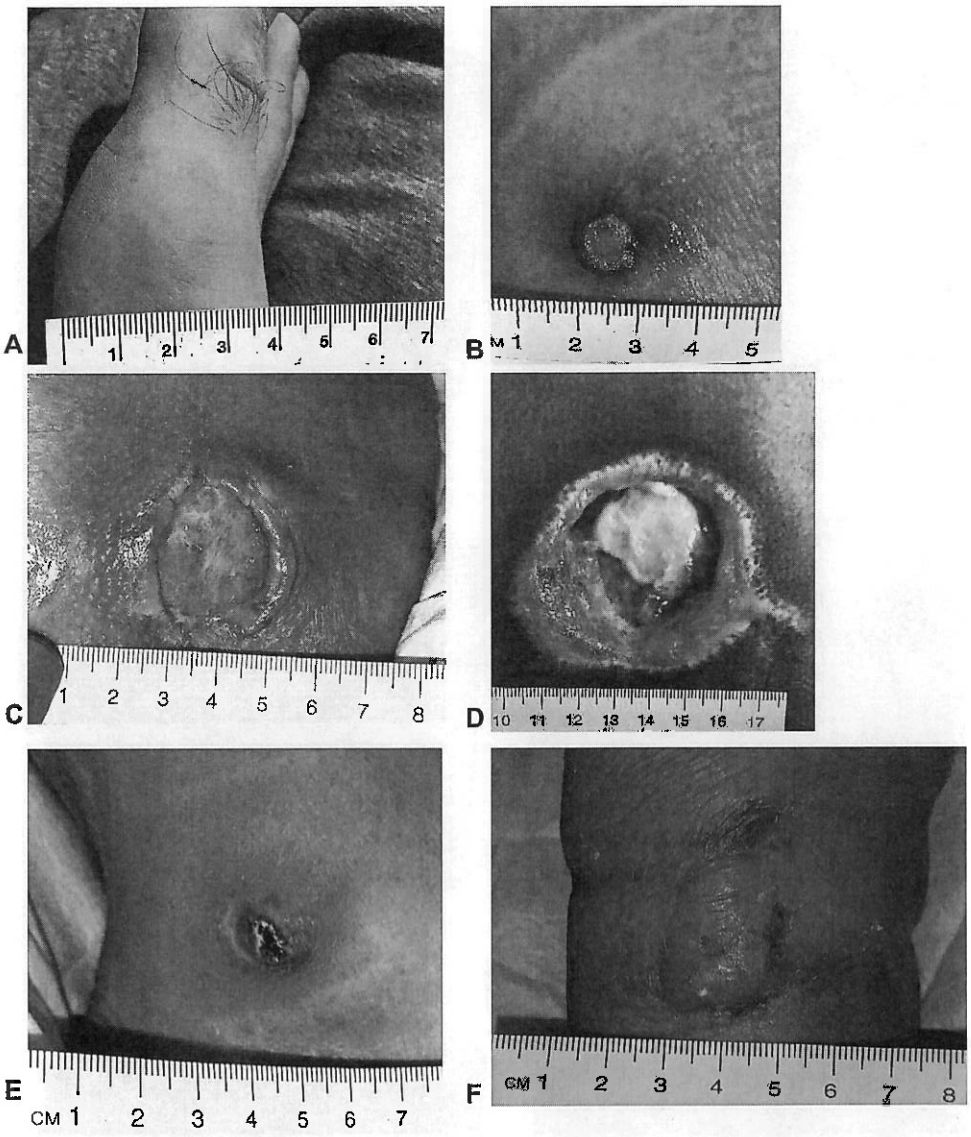

Fig 4. Pressure ulcers. **A**, Stage 1 over the metatarsophalangeal joint. **B**, Stage 2 on the heel. **C**, Stage 3 on the sacrum. **D**, Stage 4 on the sacrum. **E**, An unstageable pressure injury on the lateral malleolus. **F**, Deep tissue pressure injury on the lower leg. Photographs courtesy of Robert S. Kirsner, MD, and Luis J. Borda, MD.

Other features

Undermining, or extension of tissue damage under the edges of intact skin such that the ulcer area is larger at the base than skin surface, is often seen in stage 3 and 4 pressure ulcers. These wounds may also show tunneling, or sinus tracks that extend into and through subcutaneous tissue, typically beyond the edges of intact skin. The depth and location of undermining and tunneling can be assessed using a cotton-tipped applicator and should be regularly recorded along with standard wound measurements. Epibole, which refers to rolled wound borders caused by the downward extension of epithelium over the ulcer edges, may occur in deeper pressure ulcers and impede the migration of keratinocytes from the wound margins.

In conclusion, pressure ulcers are a common problem that continue to pose a major social and economic burden. Sustained pressure over bony prominences leads to ischemia of the underlying tissue and skin. Pressure ulcers occur in people who are immobilized or lack sensation, most often seen in association with spinal cord injury, other neurologic dysfunction, or hospitalization. The newest NPUAP guidelines now define 6 classes of pressure injury that better reflect the clinical presentations of tissue ischemia and necrosis that may occur in the absence of skin breakdown. Prevention and treatment of

pressure ulcers is the focus of the second article in this continuing medical education series.

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