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The VCU Pressure Ulcer Summit

The Search for a Clearer Understanding and More Precise Clinical Definition of the Unavoidable Pressure Injury

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ABSTRACT

This article reports the findings of the Unavoidable Pressure Ulcer Committee (of the VCU Pressure Ulcer Summit) that was tasked with addressing key issues associated with pressure injuries that are unavoidable or unpreventable. Our goals were (1) to clarify nomenclature and descriptions surrounding "terminal ulceration," (2) to describe the medical complications and comorbid conditions that can lead to skin failure and/or terminal ulceration, (3) to describe the variable possible causes of unavoidable pressure injuries, and (4) to present clinical cases to exemplify pressure injuries considered to be unavoidable.

KEYWORDS: Charcot's decubitus ominosus, Deep-tissue injury, Kennedy terminal ulcer, Pressure ulcers, Skin Changes at Life's End (SCALE), Skin failure, Trombley-Brennan terminal tissue injury, Unavoidable pressure injuries.

INTRODUCTION

The primary purpose of the VCU Pressure Ulcer Summit (held in March 2014) was to extend the process of ensuring quality healthcare delivery in pressure ulcer minimization spanning all healthcare settings, using the power of the Magnet model to unify and deliver a framework for change.[1] This article discusses findings of a committee asked to review, better define, and clinically describe conditions where pressure injury development is unavoidable.

The National Pressure Ulcer Advisory Panel (NPUAP) hosted 2 consensus conferences, one was held in 2010 and a second in 2013, in an attempt to establish consensus around factors related to unavoidable pressure injuries.[2,3] Unanimous consensus was established for the following statements: (1) most (but not all) pressure injuries are avoidable; (2) pressure redistribution surfaces cannot replace turning and repositioning; (3) if enough pressure was removed from the external body, the skin cannot always survive; and (4) skin failure is not the same as a pressure injury. It was also agreed that that there are clinical scenarios and comorbid conditions that may make pressure injury development unavoidable. These include hemodynamic instability worsened with physical movement, inability to maintain nutrition and hydration status, and the presence of an advanced directive prohibiting artificial nutrition and hydration.

The NPUAP panelists reviewed existing definitions of avoidable and unavoidable pressure injuries originally presented in the Centers for Medicare & Medicaid Services Guidance to Surveyors for Long Term Care Facilities.[4] Because the definition was specific to long-term care, the NPUAP advocated expanding it to include all care settings. According to the NPUAP, an unavoidable pressure injury is one that develops even though the provider has evaluated the patient's clinical condition and pressure injury risk factors; defined and implemented interventions that are consistent with the patient's needs and goals, and formulated with recognized standards of practice; monitored and evaluated the impact of interventions; and revised these approaches as appropriate.[3]

To the best of our knowledge, there are no definitive clinical studies that enable determination of which pressure injuries are unavoidable. In addition, there are currently no validated and reliable decision algorithms to determine unavoidability.[4] Therefore, clinicians and other interested parties must rely on expert opinion. Etiologic factors associated with pressure injuries are pressure and shear, but they are hypothesized to interact with multiple contributing factors that add to the complexity of pressure injury prevention and management. In the unavoidable pressure injury, pressure and/or shear are not the only causative factors; rather, other pathology exists that specifically impairs the integrity of the skin.[3] It is the opinion of this expert panel that, in most instances, preventive interventions can correct or improve factors involved in

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pressure-related skin uleration. However, circumstances do exist where causative factors cannot be corrected or improved, rendering prevention unrealistic.

Another challenge is attempting to define whether unavoidable pressure injurie are a separate entity or similar to previously described unavoidable ulcer development that occurs at life's end such as the Kennedy terminal ulcer (KTU),[7] Charcot's decubitus ominosus,[8] Skin failure,[9] Trombley-Brennan terminal tissue injury (TB-TTI),[10] or Skin Changes at Life's End (SCALE).[11] This article provides an updated review of unavoidable pressure injuries that have been identified with end-of-life situations, along with pressure injuries occurring in the context of permanent or temporary medical complications where pressure cannot be relieved and the skin (as an organ) fails.

PRESSURE ULCERS ASSOCIATED WITH TERMINAL TISSUE INJURY OR SKIN FAILURE

Skin failure was defined by Langemo and Brown as "an event in which the skin and underlying tissue die due to hypoperfusion that occurs concurrently with severe dysfunction or failure of other organ systems."[9(p208)] Patients who are critically ill with multiorgan failure (MOF) or mutliorgan dysfunction syndrome (MODS) are at high risk for hypoperfusion resulting from microvascular dysfunction, increased demand for oxygen, and vasoconstriction.[3,9] This is only intensified when MOF occurs concurrently with sepsis. In addition, compromised skin integrity and skin failure are associated with mortality.[12]

Individuals with MOF have decreased sympathetic vasomotor tone and endothelial dysfunction, causing vasodilation and hypotension.[13,14] While tissue blood flow may be increased, oxygen delivery to, and uptake by, the cells is diminished,[15] leading to hypoperfused tissue and compromised skin integrity.

Multiorgan dysfunction syndrome is defined as the presence of altered organ function in an acutely ill patient such that homeostasis cannot be maintained without an intervention.[16] A systemic inflammatory response syndrome event usually precedes MODS; this response is attributable to infectious or noninfectious triggers. Other precipitating factors for MODS are severe illness, severe trauma, major surgery, age greater than 65 years, persistent hypoxemia after circulatory shock, and liver failure. Systemic inflammatory response syndrome can independently cause organ damage and is the most common condition leading to MODS and death of intensive care unit (ICU) patients.[17,18]

In an 18-month prospective study of 29 patients with acute skin failure (mean age = 59 years), Varon and Marik[18] observed that the average time between ICU admission and onset of signs of skin failure was 7.7 days. They noted that all patients had failure of at least 2 organ systems or a single organ system plus sepsis. They also observed that 90% of patients had failure of more than 1 organ system besides the skin; 17.2% had failure of 2 organ systems plus the skin; 51.7% had 3 or more organ systems that failed or 2 organ systems plus sepsis; and 31% had failure of 4 or more organ systems and/or sepsis. More than 75% of patients with skin failure also had a mean arterial pressure of less than 70 mm Hg, required respiratory ventilation, and had generalized edema. Ninety percent had a serum albumin of less than 3.5 mg/dL. Based on this combination of factors, Varon and Marik concluded that a clinician can expect to observe nonskin organ system failure and skin failure concurrently.

Varon and Marik[18] further observed that the renal and respiratory systems failed most frequently (89.7% each), followed by cardiac (34.5%), and hepatic systems (27.6%). Sepsis was present in 62.1% of patients. Multiorgan failure was also observed; 15 of 29 patients (51.7%) experienced failure of 3 organs or failure of 2 organs plus sepsis. Seventeen percent experienced 2 organs failing in conjunction with skin failure. Vasopressors were being administered to 69% of subjects, and 37.9% were receiving 2 vasopressors. The researchers concluded that the skin does not fail on its own but is dependent on various others factors. In patients with renal failure, respiratory failure, or failure of more than 1 organ system other than skin, skin failure can be expected and therefore pressure injuries often develop that are probably unavoidable.

Curry and associates[19] conducted a prospective, descriptive study of the characteristics associated with skin failure in critically ill adults. They reported that the common sequence of failure of organs was usually respiratory, followed in order by hepatic, intestinal, and then renal. Deitch[20] reviewed 8 studies and concluded that the respiratory system commonly fails. Initial respiratory failure may be partly attributable to complicating factors of immobility imposed by intubation and ventilator dependence.

Lee and colleagues[21] retrospectively reviewed records of 3324 patients with a sacral pressure injury in order to identify predictive risk factors related to different stages of pressure injuries. They found that that 72% of patients with sacral pressure injuries were older than 65 years and more than 65% had dysfunction of at least 1 organ or other major illness. A study of 94,758 US residents who were discharged from hospital with a pressure injury also found a significant association between ulcer formation and organ failure or serious infection.[22] In a prospective study of 142 ICU patients, high APACHE-II scale score (22.3 vs 18.2), a reflection of severity of illness, was significantly related to pressure injury development ($P < .05$).[23]

Individuals who are critically ill and experiencing failure of multiple organs are particularly susceptible to cutaneous breakdown and failure.[19,24] These patients have altered tissue perfusion, which, in combination with failed organ system(s), may compromise the individual's ability to maintain homeostasis, leading to necrosis of skin and underlying structures.

Our knowledge of the pathophysiology of skin failure and its consequences is incomplete. As technological advances progress and we are able to maintain life longer via artificial means, the occurrence of skin failure and pressure injury can be unavoidable in some instances.[25] A photographic example of skin failure is shown in Figure 1. Similar to our conclusion stated earlier in this article, Edsberg and colleagues,[3] on behalf of the NPUAP, opined that in the vast majority of cases, appropriate identification and mitigation of risk factors can prevent or minimize pressure ulcer formation; nevertheless, there are some pressure injuries that are unavoidable.[3] The Table describes the etiology, presentation, and differential diagnoses of skin changes that may occur at end of life.

Decubitus Ominosus

Jen-Martin Charcot[27] observed that some pressure injuries occur near death; he labeled theses wounds as decubitus

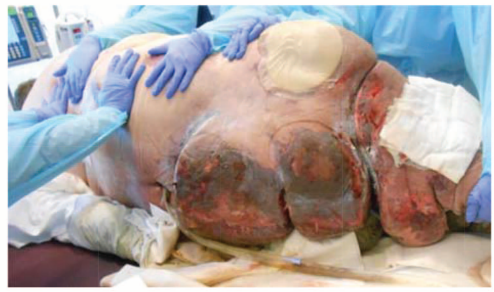
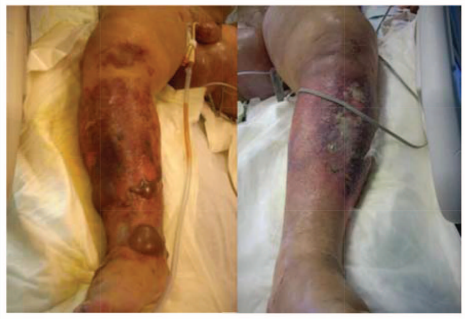
	This is a 38 y.o. 785 lbs (357 kg) Caucasian male who was admitted to the respiratory ICU for 4 months due to pneumonia and respiratory failure. He remained in ICU for 4 months with no skin breakdown. 24 hours after a cardiac arrest with low MAP despite multiple pressors, the patient developed skin failure, manifested as: sudden purple, non-blanchable discoloration with rapid epidermal sloughing to the bilateral buttocks, thighs, anterior pannus and bilateral trochanters. Necrotizing infections were ruled out. The patient expired 48 hours later after initial arrest.
	This patient is a 53 y.o. Caucasian male who was admitted for acute pancreatitis and cirrhosis in the context of alcohol consumption, who subsequently developed septic shock. Following critical drops in mean arterial pressure, the patient was placed on multiple vasopressors. Despite this treatment, he continued to experience hemodynamic instability. The patient's skin was intact, including sacrum and heels, until 4 days before the patient ultimately expired when skin failure developed as evidenced by his bilateral lower extremities presenting with progressive purpura, hemorrhagic bullae formation and epidermal sloughing.

Figure 1. Photographic examples and case summary of patients with skin failure.

ominosus. The decubitus ominosus was one component of a larger classification system for pressure injury that included decubitus acutus and chronicus; this taxonomy is the first known attempt to classify pressure injuries promulgated in modern history. Charcot, who was a neurophysiologist, believed that fibers that connected the central nervous system to the skin, which when interrupted, resulted in skin breakdown over the buttocks and sacral areas. As a result of this belief, Charcot hypothesized that all pressure injuries were unavoidable when the brain or spinal cord suffered damage, while pressure had little or no affect as a contributing factor. The neurotrophic theory of skin ulceration was discredited in the 20th century when Charcot's postulated fibers were not demonstrated.[28]

Kennedy Terminal Ulcer

The KTU was first described in 1989 in a 5-year retrospective study on pressure injuries in a 500-bed long-term care facility.[7] Kennedy reported that 55.7% of patients who died with pressure injuries expired within 6 weeks of the onset of the ulcer. This finding sparked the development of a facility committee to investigate the possibility that certain pressure injuries could be a sign of impending death or possibly an indicator of increased morbidity and rapid deterioration. Originally named the Kennedy terminal lesion, the KTU was initially described as a type (subgroup) of pressure injury that may develop during the dying process.[7,30]

Two distinct presentations of the KTU have been described in the literature.[7,30] The most common is a bilateral presentation affecting both buttocks. It has been characterized as having 5 descriptive features: (1) a shape often resembling a pear, butterfly, or horseshoe with irregular margins, (2) typically located on the coccyx or sacrum, (3) initially erythematous and/or purpuric with or without epidermal erosion progressing to a yellow and/or black color, (4) of sudden onset, and (5) "occurs within 2 weeks to several months of the patients death."[7(p45)]

The second presentation is a unilateral ulcer affecting the right or left buttock that develops more rapidly than the bilateral KTU.[30] This lesion is described as typically macular; it tends to present as a small black or purple area with irregular margins. The lesion progresses rapidly but does not usually erode or open before death. The time from onset to death in this unilateral presentation is significantly less than in the bilateral KTU presentation. It typically occurs between 8 and 24 hours before death.[30] Kennedy called the unilateral presentation "the 3:30 syndrome" since that seemed to be the time of day the staff noted the skin discoloration. Figure 2 displays photographs showing examples of the 2 KTU presentations. These presentations correlate with Charcot's description of "decubitus chronicus" (bilateral sacral ulcer) and "decubitus acutus" (an ulcer that presents unilaterally) first reported in 1877.[8]

Diagnosis of a KTU relies on knowledge of the clinical manifestations and the patient's medical history. Prevalent comorbid conditions include vascular insufficiency and organ failure.

TABLE.
Etiology, Presentation, and Differential Diagnoses of Avoidable and Unavoidable Skin Changes That May Occur at End of Life

Name of Skin Change	Etiology	Pressure Ulcer	Presentation	Avoidable	Differential Diagnosis
Skin failure	Hypoperfusion from organ dysfunction/failure; microvascular dysfunction, abnormal capillary vasomotion	Maybe, not necessarily	Diffuse dermal edema possibly with epidermal erosion or dissociation (bullae). The appearance of the skin may be hemorrhagic.	No	Coumadin-induced skin necrosis, candidiasis, calciphylaxis
Decubitus ominosus (neurotropic theory)	Failure of fibers connecting the nervous system to skin[26]	Unlikely but not enough is known	Vesicles and bullae, acute pain, intense remittent fevers, remove pulmonary emboli, involvement of deep spinal cord structures.[28] Phlegmonous tumefaction and gangrenous ichor.	No, if brain and/or spinal cord injury, then decubitus ominosus.	Pressure injury, DTI, occlusive vasculopathy/vasculitis cryoglobulinemia, Coumadin-induced skin necrosis
Kennedy terminal ulcer	Comorbidity of, hypoperfusion (localized vascular insufficiency), and multiorgan failure	likely, but not enough is known[6,30]	1. Bilateral buttocks, pear-shaped, irregular margins; on coccyx or sacrum; initially erythematous and/or purpuric with or without epidermal erosion progressing to yellow or black color; sudden onset 2 weeks to several months prior to death.[6,30] 2. Unilateral one that develops acutely, with macular lesion <1 cm^2, purpuric or black irregular margins, rapid progression but does not open prior to death[29]; progresses to death.	No	DTI, occlusive vasculopathy/vasculitis calciphylaxis, glucagonoma, Coumadin-induced skin necrosis
Trombley-Brennan terminal tissue injury at end of life	Physiological changes occurring with death; manifested as purplish-reddish discoloration	Maybe	Rapid onset, increase in surface volume without wound opening or evolving through pressure injury staging system (like Kennedy terminal ulcer). Skin is intact, purplish-reddish discoloration. Onset 20 min to several days prior to death. Does NOT progress to open wound.	No	Senile purpura, hematoma, vasculitis, fixed drug eruption, Coumadin-induced skin necrosis, DTI
SCALE	Physiological changes occurring with the dying process; manifesting as changes in skin color, turgor, or integrity, or localized pain[10,32,33]; decreased tissue perfusion, impaired skin oxygenation	May manifest as pressure injury or wounds of other etiologies	Mottled discoloration, impaired skin integrity, partial- or full-skin thickness ulceration, possible skin necrosis, fungating tumors.[11,31,32]	No	Not applicable since any wound that is present at life's end can constitute SCALE
Deep-tissue injury	Full-thickness skin damage secondary to pressure and/or shear forces	Yes	Initially presents as an erythematous, purpuric, or hemorrhagic lesion over a bony prominence that (with time days to weeks) progresses to a necrotic lesion involving the skin and subcutaneous tissues.	Maybe avoidable or unavoidable depending on other medical complications	Hematoma, Coumadin-induced skin necrosis, occlusive vasculopathy
Gangrene	Permanent damage to skin and (death of tissue) due to a lack of blood flow or a bacterial infection; most commonly affects the extremities, including the toes, fingers, and limbs, but it can also occur in the muscles and internal organs	May be associated with a pressure injury most likely on the foot or lower extremity.	There are 2 main types of gangrene: Dry gangrene—More common in people with diabetes and autoimmune diseases; dry gangrene usually affects the extremities. In dry gangrene, the tissue dries up and may become darker; it can be purple or blue to black in color. Unlike other types of gangrene, infection is typically not present in dry gangrene. However, dry gangrene can lead to wet gangrene if it becomes infected. Wet gangrene involves an infection. The tissue swells and blisters and is called "wet" because of pus. Infection from wet gangrene can spread quickly throughout the body.	Maybe avoidable or unavoidable	Benign acrocyanosis, calciphylaxis, medium vessel or occlusive vasculopathy, large vessel vasculitis, thrombus, or emboli
Mottling	Mottled skin refers to blood vessel changes in the skin that cause a patchy appearance	Unlikely	A reticulated vascular pattern that appears as a lace-like purplish discoloration of the skin. The discoloration is caused by swelling of the venules owing to obstruction of capillaries.	Maybe avoidable or unavoidable	Livedo reticularis, livedo racemosa, Sneddon's syndrome

Abbreviations: DTI, deep-tissue injury; SCALE, Skin Changes at Life's End.

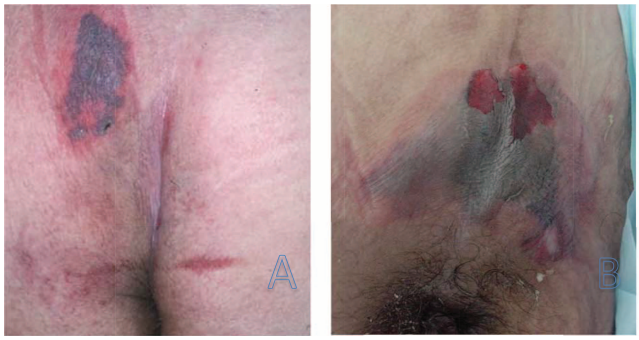


Figure 2. The Kennedy terminal ulcer: "3:30 syndrome" unilateral (A) and bilateral (B) presentations.

Measures typically used to prevent progression of a common pressure injury such as off-loading often fail to prevent or reverse tissue damage. Instead, the KTU may progress to form a full-thickness wound associated with necrotic tissue.[7,11,30] Currently, there is no official stage for a KTU.[31]

The etiology of a KTU is unknown but most likely associated with end-stage disease and multiorgan system failure causing unavoidable skin changes at end of life. Given the location of the wound, we hypothesize that pressure and shear are likely involved but are not the only contributing factors.

SCALE: Skin Changes at Life's End

In 2008, Stewart convened an expert panel to discuss issues surrounding alterations in skin and tissue integrity that develop at end of life. The 18-member interdisciplinary panel elected to develop a consensus document using a 3-phase modified Delphi method approach.[32] The result of their effort is the SCALE Consensus Statement. Input was solicited from numerous groups of wound care practitioners and experts via conferences and journal publications. Initially, the document and consensus statements were externally reviewed by 49 international reviewers and 51 reviewers participated and reached agreement on the final 10 consensus statements.

The final SCALE document includes 10 consensus statements, 2 tables, an algorithm for practice, and an extensive glossary of terms.[11] Two of the 10 SCALE consensus statements are most relevant to this summary of unavoidable wounds. Statement 1 notes that physiologic changes that can occur as a result of the dying process can be unavoidable and may occur with the application of appropriate interventions that meet or exceed the standard of care. Statement 6 discusses risk factors, symptoms, and signs associated with SCALE, such as suboptimal nutrition and diminished perfusion.

The SCALE acronym was designed to incorporate the range of alterations in skin and tissue integrity that individuals experience at end of life. Types of SCALE wounds include cancer wounds, deep-tissue injuries, gangrene, hemorrhagic wounds, ischemic wounds, KTUs, pressure injuries, skin tears, skin

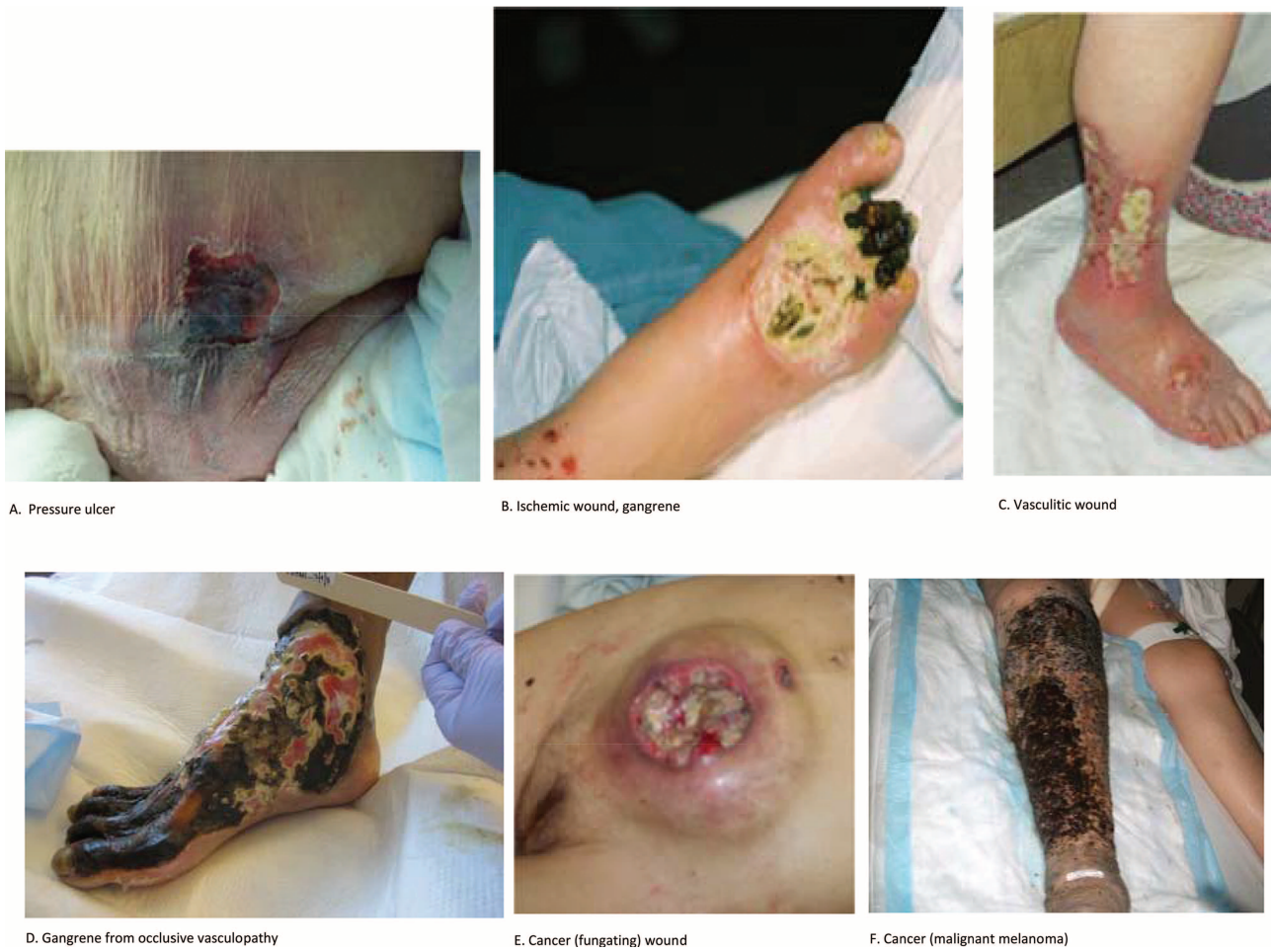


Figure 3. Skin Changes at Life's End–associated chronic wounds of varying etiologies.

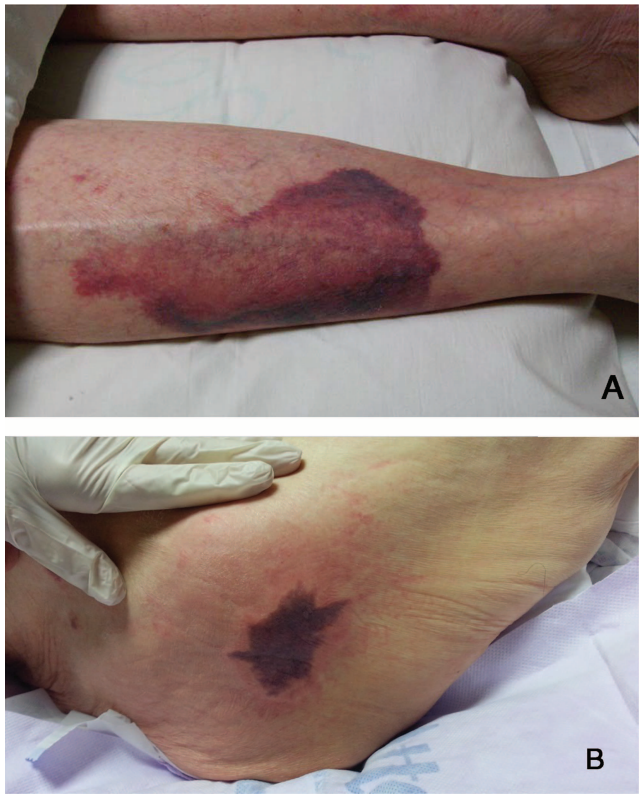


Figure 4. Photographic examples of Trombley-Brennan terminal tissue injury. (A) Note purpuric macular lesion with irregular margins that appeared 192 hours prior to death on a palliative care patient with metastatic breast cancer. (B) Note a larger purpuric lesion with telangiectasia on the right trochanter of a palliative care patient with a subdural hematoma with midline shift. This lesion was noted at 58.5 hours prior to death and an LLE (mirror image) presented 36 hours prior to death.

tears that evolve into ulcers, vasculitic (inflammatory) wounds, wound infection, and wounds of unknown etiology. Examples of SCALE-associated chronic wounds of varying etiologies are presented in Figure 3. Statements resulting from the SCALE consensus document were designed to facilitate the implementation of knowledge-transfer-into-practice techniques for quality patient outcomes. Developers also recommended that implementation include multidisciplinary professional teams (clinicians, lay people, and policy makers) concerned with the care of individuals at life's end to adequately address its medical, social, legal, and financial ramifications.[33]

Trombley-Brennan Terminal Tissue Injuries

In 2009, a team of palliative care nurses in a tertiary care hospital on Long Island were alarmed by skin changes and wounds forming on terminally ill patients.[10] They initially classified these wounds as pressure injuries characterized by a rapid onset that failed to respond to preventive or ameliorative interventions. Trombley and Brennan noted these skin changes did not follow the same course of skin changes as the KTUs; instead, the skin remained intact and the purple-red discoloration expanded in size without development of an open wound and deeper tissue damage as Kennedy described.[34] Comorbid conditions seen in these patient were cardiovascular disease (42%), cancer (15%), along with gastrointestinal (10%), respiratory disease (8%), endocrine (4%), neurologic (8%), renal (4%), sepsis (1%), orthopedic (1%), and other disorders (6%). Terminal tissue injuries occurred in patients ranging in age from 35 to 95 years. Their onset occurred from 20 minutes to several days preceding death. Trombley and Brennan concluded that this tissue damage was a potentially new, unstudied type of wound and named them "Trombley-Brennan terminal tissue injury" or TB-TTI. The findings were used as a prognostic indicator for death and impacted end-of-life care in the unit (Figure 4).

We identified 3 articles that discussed the pathophysiology of skin changes occurring near the end of life. Langemo and Brown[9] hypothesized that skin failure was attributable to hypoperfusion associated with severe organ dysfunction or failure occurring near death.[9] Tippett[35] postulated that development of wounds as a person nears the end of life is an indicator of overall frailty. Trombley and colleagues[10] theorized that the skin discoloration they observed was caused by death of the underlying tissue. Additional research into the epidemiology and pathophysiology of skin changes occurring near the end of life is clearly needed.

Gluteal Compartment Syndrome

Compartment syndrome is a condition characterized by increased hydraulic pressure within a closed and nonexpandable anatomic space.[36] It leads to a vascular insufficiency that becomes critical once vascular flow cannot return blood to the venous system. Compartment syndrome causes potentially irreversible damage to contents of the affected compartment, including any muscle. Gluteal compartment syndrome is a rare condition; scant case reports suggest that gluteal compartment syndrome may cause permanent damage to the sciatic nerve and resulting myoglobinuria may lead to renal failure.[37] Gluteal compartment syndrome has been associated with prolonged immobilization due to surgery, trauma, drug overdose, or complications experienced during surgery to repair pelvic trauma or resection of a malignant tumor.[38,39] It has also been associated with coagulation disorders caused by myeloproliferative disease or anticoagulation therapy.[40]

UNAVOIDABLE PRESSURE ULCERS IN NONTERMINAL PATIENTS

In certain patients, clinical conditions exist (in all care settings) where it may be impossible to provide adequate pressure relief. There are also certain dermatologic conditions that weaken the structure and function of the skin, making it more vulnerable to injury. A case scenario is provided to illustrate a clinical situation where pressure injuries deemed irreversible occurred in a nonterminal patient.

Case 1

C.H. is a 73-year-old woman who suddenly became dizzy, developed a severe headache, and experienced nausea and vomiting while getting ready for bed. Her husband became alarmed and called 911. By the time C.H. reached her local emergency department, she was unresponsive. She was immediately placed on a ventilator and transferred to the ICU where she was treated for shock with vasopressors. C.H. medial history included type 2 diabetes mellitus, hypertension, rheumatoid arthritis, hyperlipidemia, morbid obesity, and cardiovascular disease. Her surgical history included bilateral knee replacement, bariatric surgery (gastric bypass), and angioplasty with placement of stents. Physical examination and imaging (computed tomographic scan) revealed acute hemorrhagic stroke.

She was managed with left frontal burr hole and placement of a ventriculostomy. C.H. experienced a prolonged hospital course complicated by respiratory failure and inability to eat, resulting in tracheostomy and percutaneous endoscopic gastrostomy (PEG) tube placement. She also experienced prolonged loss of consciousness (which required another ventriculostomy), urinary tract infection, renal insufficiency (causing generalized edema), *Clostridium difficile* infection, bacteremia, and lower extremity bilateral deep venous thrombosis (DVT). While in the ICU, she was on a critical care bed with a static pressure redistributing foam mattress. Due to difficulty breathing, the head of the bed (HOB) was maintained at greater than 30°. After 20 days in the ICU, she was transferred to a respiratory care unit where she was placed on an alternating pressure support surface. Routine skin care was provided via a pressure injury prevention protocol developed by the hospital; it comprised standard cleansing with a mild soap and application of a skin protectant; she was turned and repositioned according to hospital policy (every 2 hours and as needed). Twenty-three days after admission, a sacral pressure injury was identified. The pressure ulcer was described as erythematous with areas of purpura. Four days later, the sacral wound was documented as stage 2; the ulcer was described as erythematous, hemorrhagic with epidermal erosion. Although unidentified at this time, this lesion was likely a deep-tissue injury. Her overall health improved, and she was discharged to a long-term acute care facility 34 days after she was admitted. Upon admission to the long-term acute care facility, the pressure injury was described as unstageable and covered with soft black eschar.

Multiple factors were present during C.H.'s hospital course that, when combined, make it unlikely her sacral pressure injury could have be prevented. The HOB was elevated more than 30° making it impossible to distribute her weight and avoid pressure and possibly shearing forces over the sacrum.[41] In addition, C.H. had been taking oral corticosteroids for treatment of rheumatoid arthritis. An adverse effect of prolonged corticosteroid use is dermal atrophy and muscle wasting, making her skin and muscle more susceptible to injury from pressure, shear and friction.[42,43] C.H. also received vasopressors during her hospital course that increase blood pressure by constricting the arterioles. A common adverse side effect of this class of drugs is decreased skin perfusion.[44] C.H. also experienced renal insufficiency and generalized edema; dermal edema increases local skin temperature and reduces skin turgor and perfusion, rendering the skin more vulnerable to pressure injury.[45-49] Finally, C.H. experienced diarrhea and fecal incontinence associated with a *Clostridium difficile* infection; both the diarrhea and incontinence-associated dermatitis have been linked to an increased likelihood of sacral pressure injury.[50-54]

Situational Unavoidability

We assert that all unavoidable pressure injuries occur due to a combination of etiologic and contributing factors. Intrinsic factors associated with pressure injury occurrences include immobility, chronic illness, cognitive deficit, malnutrition, aging, and the use of certain medicines that affect the skin.[55] Extrinsic factors include pressure, shearing forces, and moisture. Predominantly, damage to the skin in an unavoidable pressure injury is full thickness but we believe there may be situations when partial thickness or epidermal erosion occurs that is unavoidable. When pressure and shear are part of the causative equation, there is loss of perfusion, causing deep-tissue damage leading to ischemia and necrosis of the entire dermis and often subcutaneous tissues (fat, muscle, and sometimes bone). The anatomic location of these ulcers makes it apparent that pressure is acting as an etiologic factor. Additional factors such as poor dermal perfusion, dermal edema, dermal atrophy, dermal fibrosis, and dermatologic toxicities may contribute to pressure injury development.[56] Poor skin perfusion may be a contributing factor to development of an unavoidable pressure injury and can occur in patients with MOF, MODS, sepsis, and shock.[9] Diminished skin perfusion can also occur in patients with hypotension who are given vasoconstrictors.[44,57] Kidney failure or fluid overload frequently leads to generalized and dermal edema. Dermal edema weakens the dermal-epidermal junction, causing the skin to blister. Prolonged dermal edema can also lead to poor blood flow.[45,48]

Skin atrophy may be another contributing factor.[58] Atrophy of the skin is irreversible; it is characterized by the reduction of its volume, as well as qualitative changes in the tissue, especially elastic fibers. Senile atrophy of the skin develops as a result of age-related involution. Pathologic skin atrophy occurs as a result of infringement of metabolism in the skin and reduces the activity of enzymes.[59] Generalized, secondary skin atrophy can develop as a result of aging, prolonged corticosteroid use, and radiation.[59,60] Dermal disorders that can lead to atrophy are dermatomyositis, lupus, necrobiosis lipoidica, scleroderma, and mixed connective tissue disease.[61]

Dermal fibrosis, characterized by excessive scarring of the skin owing to chronic inflammation and a pathologic wound healing response, also may contribute to development of an unavoidable pressure injury.[62] A variety of immune, autoimmune, and inflammatory mechanisms may adversely affect the balance of collagen production and degradation in the skin, resulting in dermal fibrosis. Fibrotic skin diseases include scleroderma, nephrogenic fibrosing dermopathy, mixed connective tissue disease, scleromyxedema, scleredema, eosinophilic fasciitis, and lymphedema.[62] Chronic exposure to chemicals or physical agents also may contribute to dermal fibrosis.

Dermatologic toxicities secondary to drug therapy are particularly prevalent in older patients, and they may act as a situational factor affecting the likelihood for developing an unavoidable pressure injury.[56,63,64] The cause of skin reactions is often unknown, although many have an allergic or toxic basis. Skin reactions associated with the use of a specific drug can be dose independent and may persist long after the drug causing the allergic reaction has been discontinued. For example, with a hypersensitivity reaction to penicillin, a dermatologic toxicity may worsen for 7 to 10 days after the drug has been stopped. Toxic reactions, in contrast, are dose dependent and skin reactions generally resolve fairly soon after the drug causing the reaction is stopped. Diseases that may increase the risk of developing skin reactions are liver disease, kidney disease, lupus, and AIDS. Dermal toxicities associated with chemotherapy and/or radiation may persist for months or years after therapy has stopped.

We believe that the process of prioritizing multiple life-threatening medical conditions or complications also acts as a situational factor influencing development of unavoidable pressure injuries. For example, respiratory failure or gastric intubation may require HOB elevation higher than 30°. Depending on the patient's weight and anatomy, elevation can distribute significant mass over a much smaller area, causing a marked increase in interfacial pressure over the buttocks and sacrum.[65-68] When combined with shearing forces, the skin

becomes stretched and angulates underlying blood vessels that may become occluded.[67,68] Other situational factors that may contribute to occurrence of an unavoidable pressure injury include contractures, amputations, spasticity, ostomies, surgical drains, other skin injuries, fractures requiring traction, and intractable pain.

SUMMARY

The purpose of this article was to provide a detailed description for identifying how the unavoidable ulcers may occur near the end of life (decubitus ominosus, KTU, SCALE, skin failure, and TB-TTI) or when temporary or permanent medical conditions render pressure injury truly unavoidable. Our panel agrees with the NPUAP and the Wound, Ostomy and Continence Nurses Society that most pressure injuries are preventable, and we support the NPUAP's definition of unavoidable pressure injuries.[69] We also sought to provide a focused description of unavoidable pressure injuries that enables colleagues to enhance our understanding of the situational and comorbid conditions that lead to an unavoidable pressure injury.

We acknowledge that identification of an unavoidable pressure injury is not based exclusively on individual medical determination. Public policy regarding unavoidable pressure injuries is inconsistent, and government language is confusing by differentiating "unavoidability" between healthcare settings.[70,71] In acute care, pressure injuries are deemed "reasonably preventable," whereas unavoidability in the long-term care facility is based on compliance with guidance documents promulgated by the US Centers for Medicare & Medicaid Services. The language in these documents is written in an attempt to control civil monetary penalties associated with the development of pressure injuries. Nevertheless, patients who develop pressure injuries have the same immobility problems and similar comorbidities regardless of where they are cared for. The definitions and descriptions used in this document are intended to apply equally to any setting in which immobile patients are cared for.

Proper characterization of terminal unavoidable has not been achieved. Histopathologic and laboratory investigations are necessary in order to describe these skin changes at the metabolic and cellular levels. In the case of the unavoidable pressure injury associated with medical and situational circumstances in the nonterminal patient, awareness and proper communication of the various medical conditions that make prevention unlikely are paramount.

REFERENCES

1. Brindle CT, Creehan S, Black J, Zimmerman D. The VCU Pressure Ulcer Summit: collaboration to operationalize hospital-acquired pressure ulcer prevention best practice recommendations. *J Wound Ostomy Continence Nurs.* 2015;42(4):331-337.
2. Black J, Edsberg LE, Baharastani MM, et al. National Pressure Ulcer Advisory Panel. Pressure ulcers: avoidable or unavoidable? Results of the National Pressure Ulcer Advisory Panel Consensus Conference. *Ostomy Wound Manage*. 2011;57(2):24-37.
3. Edsberg LE, Langemo D, Baharestani MM, Posthauer ME, Goldberg M. Unavoidable pressure ulcer injury: state of the science and consensus outcomes. *J Wound Ostomy Continence Nurs*. 2014;41(4):313-334.
4. Department of Health and Human Services. *CMS Manual System. Centers for Medicare and Medicaid Guidance to Surveyors for Long-Term Care Facilities*. Transmittal 4. DHHS Pub No. 100-07. http://www.hsag.com/App_Resources/Documents/PrU_LS1_F_314.pdf. Published November 12, 2004. Accessed January 25, 2011.
5. Levine JM, Zulkowski K. Secondary analysis of Office of Inspector General's pressure ulcer data: incidence, avoidability, and level of harm. *Adv Skin Wound Care.* 2015;28(9):420-428.
6. National Pressure Ulcer Advisory Panel and European Pressure Ulcer Advisory Panel; Cuddigan JE, Langemo D, Dealey C, eds. *Prevention and Treatment of Pressure Ulcers: Clinical Practice Guideline*. Washington, DC: National Pressure Ulcer Advisory Panel; 2009.
7. Kennedy KL. The prevalence of pressure ulcers in an intermediate care facility. *Decubitus.* 1989;2(2):44-45.
8. Charcot JM. *Lectures on the Diseases of the Nervous System*. Sigerson G, trans. London, England: The New Sydenham Society; 1877.
9. Langemo DK, Brown G. Skin fails too: acute, chronic, and end-stage skin failure. *Adv Skin Wound Care.* 2006;19(4):206-211.
10. Trombley K, Brennan MR, Thomas L, Kline M. Prelude to death or practice failure? Trombley-Brennan terminal tissue injuries. *Am J Palliat Care*. 2012;29(7):541-545.
11. Sibbald RG, Krasner DL, Lutz J. SCALE: Skin Changes at Life's End: final consensus statement: October 1, 2009. *Adv Skin Wound Care.* 2010;23(5):225-236.
12. Brown G. Long-term outcomes of full-thickness pressure ulcers: healing and mortality. *Ostomy Wound Manage*. 2003;49(10):42-50.
13. Holloway GA, Tolentino G, DeLateur BJ. Cutaneous blood flow responses to wheelchair cushion pressure loading measured by laser Doppler flowmetry. In: Lee BY, ed. *Chronic Ulcers of the Skin*. New York, NY: McGraw-Hill;1985:57-67.
14. Young JD, Cameron EM. Dynamics of skin blood flow in human sepsis. *Intensive Care Med.* 1995;21:669-674.
15. Beal AL, Cerra FB. Multiple organ failure syndrome in the 1990's. *JAMA.* 1994;271:226-233.
16. Bone R, Balk R, Cerra F, et al. Definitions for sepsis and organ failure and guidelines for the use of innovative therapy in sepsis. *Chest*. 1992;101(6):1644-1655.
17. Schulman C, Hare K. New thoughts on sepsis: the unifier of critical care. *Dimens Crit Care Nurs*. 2003;22(1):22-30.
18. Varon J, Marik P Multiple organ dysfunction syndrome. In: Irwin RS, Rippe J, eds. *Intensive Care Medicine*. Philadelphia, PA: Lippincott Williams & Wilkins; 1999, p. 174.
19. Curry K, Kutash M, Chambers T, Evans A, Holt M, Purcell S. A prospective, descriptive study of characteristics associated with skin failure in critically ill adults. *Ostomy Wound Manage*. 2012;58(5):36-43.
20. Deitch E. Multiple organ failure: pathophysiology and potential future therapy. *Ann Surg*. 1992;216(2):117-134.
21. Lee T, Lin K, Mills ME, Kuo Y. Factors related to the prevention and management of pressure ulcers. *Comput Inform Nurs.* 2012;9:489-495.
22. Fogerty MD, Abumrad NN, Nanney L, Arbogast PG, Poulose B, Barbul A. Risk factors for pressure ulcers in acute care hospitals. *Wound Rep Regen*. 2008;16:11-18.
23. Terecki H, Kucukardali Y, Top C, Onem Y, Celik S, Oktenli C. Risk assessment study of pressure ulcers in intensive care unit patients. *Eur J Int Med*. 2009;20(4):394-397.
24. Shahin ES, Dassen T, Halfens P, Meyer S. Results of the 2008-2009 International Pressure Ulcer Prevalence Survey and a 3-year acute care, unit-specific analysis. *Ostomy Wound Manage.* 2009;55(11):39-45.
25. Witkowski JA, Parish LC. Skin failure and the pressure ulcer [Editorial]. *Decubitus.* 1992;6(5):4.
26. Goode PS, Allman RM. The prevention and management of pressure ulcers. *Med Clin N Am*. 1989;73:1511-1524.
27. Levine JM. Historical perspective: the neurotrophic theory of skin ulceration. *J Am Geriatr Soc*. 1992;12:1281-1283.
28. Freeman L. Ulceration and ulcers. In: Keen WW, ed. *Surgery: Its Principles and Practices*. Vol 1. Philadelphia, PA: Saunders; 1909:295.
29. Levine JM. Historical notes on pressure ulcers: the decubitus ominosus of Jean-Martin Charcot. *J Am Geriatr Soc.* 2005;53:1248-1251.
30. Kennedy KL. The Kennedy terminal ulcer. In: Milne CT, Corbett LQ, Bubuc DL, eds. *Wound, Ostomy, and Continence Nursing Secrets*. Philadelphia, PA: Hanley & Belfus Inc; 2003:198-199.
31. American Medical Directors Association. *Pressure Ulcers in the Long-Term-Care Setting Clinical Practice Guideline*. Columbia, MD: American Medical Directors Association; 2008:11.
32. Woo KY, Krasner DL, Sibbald RG. Palliative wound care and treatment at end of life. In: Thomas DR, Compton G, eds. *Pressure Ulcers in the Aging Population: A Guide for Clinicians. Aging Medicine Series*. New York, NY: Humana Press-Springer; 2014:chap 10.

33. The SCALE Expert Panel. SCALE: Skin Changes at Life's End. In: Krasner DL, ed. *Chronic Wound Care: The Essentials*. Wayne, PA: HMP Communications; 2014:chap 23.
34. Kennedy Terminal Ulcer. Understanding the Kennedy terminal ulcer. http://www.kennedyterminalulcer.com. Accessed March 6, 2011.
35. Tippett AW. Wounds at the end of life. *Wounds.* 2005;17(4):91-98.
36. Twaddle C, Amendola A. Compartment syndromes. In: Browner B, Levine A, Jupiter J, Trafton P, Krettek C, eds. *Skeletal Trauma*. 4th ed. Saunders/Elsevier, Phila PA, p. 353-63, 2009:chap 13.
37. Iizuka S, Miura N, Fukushima T, Seki T, Sugimoto K, Inokuchi S. Gluteal compartment syndrome due to prolonged immobilization after alcohol intoxication: a case report. *Tokai J Exp Clin Med.* 2011;36(2): 25-28.
38. Mustafa M, Hyun A, Kumar JS, Yekkirala A. Gluteal compartment syndrome: a case report. *Cases J.* 2009;2(11):190.
39. Liu HJ, Wong DSY. Gluteal compartment syndrome after prolonged immobilisation. *Asian J Surg.* 2009;32(2):123-126.
40. Bain J. Bone marrow biopsy morbidity and mortality. *Br J Haematol.* 2003;121(6):949-951.
41. Metheny A, Frantz RA. Head-of-bed elevation in critically ill patients: a review. *Crit Care Nurse.* 2013;33(3):53-67.
42. Kolbe L, Kligman AM, Schreiner V, Stoudemayer T. Corticosteroid-induced atrophy and barrier impairment measured by non-invasive methods in human skin. *Skin Res Technol.* 2001;7(2):73-77.
43. Hasselgren P-O, Alamdari N, Aversa Z, Gonnella P, Smith AJ, Tizio S. Corticosteroids and muscle wasting: role of transcription factors, nuclear cofactors, and hyperacetylation. *Curr Opin Clin Nutr Metab Care.* 2010;13(4):423-428.
44. Overgaard CB, Džavíkm V. Inotropes and vasopressors: review of physiology and clinical use in cardiovascular disease. *Circulation*. 2008;118:1047-1056.
45. Trayes KP, Studdiford JS, Pickle S, Tully AS. Edema: diagnosis and management. *Am Fam Physician.* 2013;88:102-110.
46. Maurice PD. Acquired perforating dermatosis in renal patients. *Nephrol Dial Transpl.* 1997;12(12):2774-2775.
47. Farrell AM. Acquired perforating dermatosis in renal and diabetic patients. *Lancet.* 1997;349(9056):895-896.
48. Lupi O, Rezende L, Zangrando M, et al. Cutaneous manifestations in end-stage renal disease. *An Bras Dermatol*. 2011;86(2):319-326.
49. Lee A. Skin manifestations of systemic disease. *Aust Fam Physician.* 2009;38(7):498-505.
50. Gray M. Incontinence associated dermatitis in the elderly patient: assessment, prevention and management. *J Geriatr Care Manage.* Spring 2014. http://www.gcmjournal.org/2014/05/14/incontinence-associated-dermatitis-in-the-elderly-patient-assessment-prevention-and-management.
51. Bell S. Investigation and management of chronic diarrhoea in adults. In: Norton C, Chelvanayagam S, eds. *Bowel Continence Nursing*. Beaconsfield, England: Beaconsfield Publishers; 2004. p. 92-102
52. Emmanuel A. The physiology of defecation and continence. In: Norton C, Chelvanayagam S, eds. *Bowel Continence Nursing*. Beaconsfield, England: Beaconsfield Publishers; 2004. p. 8-13
53. Gray M, Beeckman D, Bliss DZ, et al. Incontinence-associated dermatitis: an update. *J Wound Ostomy Continence Nurs.* 2012;39(1): 61-74.
54. Lachenbruch C, Ribble D, Emmons K, VanGilder C. Pressure ulcer risk in the incontinent patient: analysis of incontinence and hospital-acquired pressure ulcers from the International Pressure Ulcer Prevalence™ Survey. *J Wound Ostomy Continence Nurs.* 2016;43(3):235-241.
55. Braden B, Bergstrom N. A conceptual schema for the study of the etiology of pressure sores. *Rehabil Nurs.* 1987;12(1):8-16.
56. Rasmussen L, Arvin A. Chemotherapy-induced immunosuppression. *Environ Health Perspect.* 1982;43:21-25.
57. Kanter J, DeBlieux P. Pressors and inotropes. *Emerg Med Clin N Am.* 2014;32(4):823-834.
58. Klaus W, Kibbi AG, Mihm MC. Basic pathologic reactions of the skin. In: Fitzpatrick TB, Eisen AZ, Wolf K, Fredberg IM, Austen KF, eds. *Dermatology in General Medicine*. 3rd ed. McGraw Hill Inc Publ New York, NY; 1987:49-68.
59. Pinkus H, Mehregan AH. *A Guide to Dermatopathology*. New York, NY: Appleton-Century-Crofts; 1981.
60. Fitzpatrick TB, Bernhard JD, Soter NA. Correlation of pathophysiology of the skin. In: Fitzpatrick TB, Eisen AZ, Wolf K, Fredberg IM, Austen KF, eds. *Dermatology in General Medicine*. 3rd ed. McGraw Hill Inc Publ New York, NY; 1987:69-86.
61. Flour M. Pathophysiology of vulnerable skin. *World Wide Wounds*. http://www.worldwidewounds.com/2009/September/Flour/vulnerable-skin-1.html. Published September 2009.
62. Luzina IG, Atamas SP. Fibrotic skin diseases. In: Gaspari AA, Tyring SK eds. *Clinical and Basic Immunodermatology*. Springer Verlag, London 2008:721-737.
63. Riedl MA, Casillas AM. Drug reactions: types and treatment options. *Am Fam Physician.* 2003;68(9):1781-1791.
64. Bryce J, Jennifer J, Lacouture ME; Multinational Association of Supportive Care in Cancer (MASCC). MASCC Skin Toxicity Study Group. http://www.mascc.org/skin-toxicities. Accessed March 1, 2016.
65. Alvarez OM, Childs EJ. Pressure ulcers: physical, supportive and local aspects of management. In: Albert SF, Mulder GD, eds. *Clinics of Podiatric Medicine and Surgery—Wound Healing Edition*. Philadelphia, PA: WB Saunders & Co; 1991:869-890.
66. Alvarez OM. Pressure ulcers: critical consideration in prevention and management. *Clin Mater Spec Issue Wound Healing.* 1991;8: 209-222.
67. Alvarez OM, Massac E, Brown B, Grauer K. Prevention, initial assessment and supportive care of pressure ulcers. *Fam Pract Recertification.* 1990;12:15-35.
68. Alvarez OM, Massac E, Brown B, Grauer K. Management issues in the critical care of pressure ulcers. *Fam Pract Recertification.* 1990;12: 78-104.
69. Wound, Ostomy and Continence Nurses Society. New position paper on avoidable versus unavoidable pressure ulcers. http://www.wocn.org/About_Us/News/37. Published May 7, 2009. Accessed March 1, 2010.
70. Centers for Medicare & Medicaid Services, Department of Health and Human Services. Medicare program; changes to the hospital inpatient prospective payment system for acute care hospitals and fiscal year 2010 rates; and changes to the long-term care hospital prospective payment system and rate years 2010 and 2009 rates. Final rules and interim final rule with comment period. *Fed Regist.* 2009;74:43753-44236.
71. Ayello EA, Capitulo KL, Fife CE, et al. Legal issues in the care of pressure ulcer patients: key concepts for health care providers: a consensus paper from the international expert wound care advisory panel. *J Palliat Med.* 2009;12:995-1008.

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