Skin Fails Too: Acute, Chronic, and End-Stage Skin Failure

Diane K. Langemo, PhD, RN, FAAN, and Gregory Brown, BBA, BSN, RN, CWOCN

ABSTRACT

OBJECTIVE: To identify what has been published in the literature about acute and chronic skin failure and to propose a working definition of this phenomenon.

DESIGN: A systematic review of MEDLINE and CINAHL to determine what has been published in the literature on the topics of skin failure, acute skin failure, chronic skin failure, multiple organ failure, end-of-life skin deterioration, and pressure ulcers in hospice from 1984 to 2005.

MAIN OUTCOME MEASURES: Published papers were reviewed for content related to acute, chronic, and end-stage skin failure.

MAIN RESULTS: Seven articles were identified that referenced either acute, chronic, or end-stage skin failure. Additional information was identified that discussed the processes of acute and chronic skin failure and pressure ulcers in individuals in hospice care or at the end of life. Care considerations and dilemmas related to a curative versus palliative goal in wound healing were discussed.

CONCLUSIONS: Minimal literature exists on skin failure, yet caregivers and the public must be aware of, assess for, and consider this phenomenon in their care. Based on this literature review, skin failure was defined by the authors as an event in which the skin and underlying tissue die due to hypoperfusion that occurs concurrent with severe dysfunction or failure of other organ systems. Skin failure can be categorized as acute, chronic, or end stage. Pressure ulcers, a type of skin death, frequently occur in persons with a heavy disease burden, especially those at or near the end of life, despite good care.

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The skin is one of the largest organs of the body, and like other organs, it can fail. Organ failure is typically seen with advancing age. In the United States, the population is rapidly aging, with life expectancy over 76 years.[1] It is possible that failure of the skin will become a more common occurrence as the population ages.

Chronic illnesses are the most common cause of death in older adults,[1] with 3 of 4 80-year-olds having a disabling condition.[2] Functional decline occurs to some degree in every individual on an ongoing basis. It can occur quickly in older adults, especially those who are immobilized or on bed rest, as is seen in hospitalized patients.[3] With acute and chronic illnesses, body systems progressively fail, sometimes quite suddenly. Life can be prolonged with advancing medical technology, at times beyond the ability of the outer covering of the body—the skin—to maintain its integrity.

Some factors present in the deteriorating patient that are associated with pressure ulcer (PrU) development include poor tissue perfusion, poor nutritional status, and immobility. As each progressive body organ system is affected, the deleterious effects are compounded. Pressure ulcer development in older adults and the terminally ill is not always preventable, and at least 1 author has referred to this occurrence as a permissible PrU or skin failure.[4]

Skin failure is a term used in many articles and by a variety of practitioners, yet it has not been well defined. The purpose of this article is to present a systematic review of the literature on the topic of skin failure and to propose a working definition of this term.

SKIN AND AGING

The skin comprises between 10% and 15% of a person's body weight. The skin serves a variety of functions, including protection, storage of fat and water, regulation of temperature, management of waste exchange, vitamin D synthesis, and touch.[5,6] As an individual ages, changes take place in the skin. It becomes drier, less elastic, and less well perfused, making it vulnerable to damage from trauma, pressure, moisture, friction, shear, and malnutrition, to name a few.[7] Other changes are related to what is called replicative senescence; the epithelial and fatty layers become thinner and more prone to irritation and disruption, the collagen and elastic fibers shrink approximately 1% per year, and the sweat glands decrease in number and size.[8] The skin vascularity diminishes, arteriosclerotic changes occur in the small and large vessels, and the vessel walls thin. Consequently, the oxygen-carbon dioxide exchange decreases, tissue turnover slows, and ecchymosis occurs more

Diane K. Langemo, PhD, RN, FAAN, is Chester Fritz Distinguished Professor Emeritus of Nursing, University of North Dakota College of Nursing, Grand Forks, ND. Gregory Brown, BBA, BSN, RN, CWOCN, is a Surgical Case Manager, Dallas Veterans Medical Center, Dallas, TX. Submitted August 19, 2005; accepted in revised form December 14, 2005.

frequently. All of these damaging factors can, alone or in combination, result in skin breakdown. The inflammatory response decreases and regeneration of tissue is slower with these skin changes, delaying healing and often making tissue more prone to infection.[7]

PRESSURE ULCERS AND TISSUE PRESSURE

A PrU is a type of lesion caused by unrelieved pressure, resulting in damage of underlying tissue. Pressure ulcers usually occur over bony prominences.[9,10] These wounds are categorized into 4 stages, from Stage I, where skin is intact, to Stage IV, which is a full-thickness injury with damage to muscle, bone, or supporting structures.[11] The National Pressure Ulcer Advisory Panel (NPUAP) has recognized the occurrence of significant pressure-related deep tissue injury under intact skin, which can occur after a single episode of prolonged immobilization.[12] Deep tissue injury frequently evidences itself initially as a purplish nonblanchable area, yet may eventually evolve into a Stage IV ulcer.[12]

The baseline pressure in arterial skin capillaries needed to keep the capillary open for tissue perfusion ranges from 25 to 30 mm Hg at the arterial end and 5 to 10 mm Hg at the venous end.[13,14] Once the pressure on the tissue and capillaries exceeds these pressures for 2 or more hours, or exceeds it excessively for a shorter duration of time, tissue damage and, ultimately, necrosis can occur. This is particularly accentuated over bony prominences.[15] Sacral pressures can reach 70 mm Hg after short periods of immobilization, whereas pressure under an unsupported heel can reach 45 mm Hg.[16] Pressure-related damage occurs simultaneously from the skin down to the bone.[13] The damage is particularly detrimental in older individuals who already have diminished tissue integrity with decreased perception of pressure, light touch, and pain. When friction and shear forces and/or moisture are present, skin breakdown can occur more quickly and over a wider area.

The 25 to 30 mm Hg capillary closing pressure applies in general to healthy individuals. Individuals with a critical or chronic illness or general poor health could have lower pressures, which would further shorten the time required for tissue ischemia and necrosis to occur. In this instance, the skin may not be blanchable in the presence of skin failure where perfusion is quite low or entirely shutting down. The poorly perfused tissue is then squeezed between 2 unforgiving objects—the external support surface and the internal hard bone or other unyielding soft tissue—which further limits perfusion to the stressed area.

Pressure ulcers are observed in a wide variety of body locations, depending on the disease state of the individual and their body geometry. Guidelines from the Agency for Health Care Policy and Research[9,10] (AHCPR; now the Agency for Healthcare Research and Quality) and the Wound Ostomy and Continence Nurses (WOCN) Society[17] note that PrUs usually occur over bony prominences, implying that this may not always be the case. In patients who are morbidly obese, the phenomenon of PrUs occurring over a bony prominence located deep below the outer surface of tissue and between deep skin folds has been observed. Pressure ulcers occurring on nonbony prominences have also been observed and reported in persons with spinal cord injuries and individuals with atypical body geometry.[18]

PRESSURE ULCERS AND MORTALITY

Multiple studies examine the link between skin breakdown, particularly PrU development, and ultimate mortality. Berlowitz and Wilking[19] demonstrated in a study of 19,981 nursing home residents that although residents with PrUs were more likely to die, the increased risk was largely related to the frailty and high disease burden rather than to the PrU itself. Thomas et al[20] studied 286 hospitalized patients and determined that 59.5% of residents died within 1 year of developing a PrU. It appeared that development of a new PrU was a marker of coexisting illnesses, impaired nutrition, and functional status and not an independent risk factor for increased mortality. In a study of 301 nursing home admissions, Berlowitz and Wilking[21] discovered that the presence of a PrU on admission, the development of a new ulcer, and the failure of the PrU to heal were all associated with a 2- to 3-fold increase in the risk of dying during a 6-week period following admission. This study also suggested that the PrU itself did not appear to be the cause of the observed increased mortality.

Brown[22] examined outcomes of 74 individuals who developed a PrU in the intensive, acute, and long-term-care settings and found 180-day mortality rates of 66.7%, 75%, and 66.7%, respectively, after PrU onset. No deaths in this study were directly attributable to the presence of the PrU. Other research has shown that in acute hospitalizations, death occurs in 67% of patients who develop a PrU.[23] In nursing home residents, Kennedy[24] found that 55.7% of persons who died with a PrU died within 6 weeks of onset of the PrU. In Hanson et al,[25] 62.5% of PrUs in hospice patients occurred in the 2 weeks before death.

MULTIPLE ORGAN FAILURE AND SKIN FAILURE

Seven articles, including 1 editorial, concerning skin failure were identified through the literature review (Table 1). As early as 1989, Goode and Allman[26] noted multiple/multiorgan failure (MOF) as a terminal stage of many diseases that occurs as the body wastes away. In 1991, the term skin failure was used in

Table 1.

RESULTS OF LITERATURE REVIEW RELATED TO SKIN FAILURE

Article Reviewed	Criteria for Inclusion
Witkowski JA, Parish LC[4]	Asserted that pressure ulcers that develop in older adults and the terminally ill are not always preventable. Some pressure ulcers are permissible because skin failure is present.
Brown G[22]	Found high mortality rates in persons developing pressure ulcers, indicating an internal pathologic process of development rather than an external care issue.
La Puma L[26]	Used the term skin failure.
Witkowski JA, Parish LC[27]	Discussed skin failure and pressure ulcers at the end of life and in patients in intensive care settings.
Hobbs L, Spahn JG, Duncan C[28]	Discussed the condition of skin failure.
Goode PS, Allman RM[29]	Found that multiple/multiorgan failure is seen in terminal stages of many diseases as the body wastes away. Concluded that the skin, as the largest organ of the body, is susceptible to failure and death, must be considered in multiorgan death syndrome.
Leijten FS, De Weerd AW, Poortvliet DC, De Ridder VA, Ulrich C, Harink-De Weerd JE[36]	Discussed chronic skin failure as occurring in older adults and in patients with multiple comorbidities that can result in a pressure ulcer, especially at the end of life.

the literature by La Puma.[27] Witkowski and Parish[28] discussed skin failure and PrUs in a 1993 editorial. They described skin and underlying tissue damage that occur at the end stages of life and in the intensive care setting. Skin failure was addressed again in 2000 by Hobbs et al,[29] but it was not clearly defined.

Some authors have referred to multiple organ death syndrome (MODS), whereas other authors have made reference to MOF. Goode and Allman[26] said that in MOF, the damaged tissue loses its tolerance to pressure and trauma and the tissue increasingly cannot assimilate nutrients, causing more tissue damage and, ultimately, necrosis. As a body shunts blood and nutrients to vital organs such as the heart, lungs, and kidneys, it shunts blood away from the periphery or the skin. Goode and Allman[26] concluded that ''the skin, as the largest organ of the body, is susceptible to failure and death must be considered in multiorgan death syndrome.''

Skin failure, as defined by the authors of the present article, is an event in which the skin and underlying tissue die due to hypoperfusion that occurs concurrent with severe dysfunction or failure of other organ systems. A PrU, in the classic definition, can result from any unrelieved pressure that results in tissue ischemia and resulting necrosis; it can occur in healthy persons.

The distinction between skin failure and PrUs, therefore, lies in the health status of the individual. The authors of this article believe that the coexistence of a significant disease process or organ failure during development of the external lesion is the crucial factor in determining the ability to avoid such lesions and its classification as either a PrU or skin failure. Indeed, in the presence of a significant disease process or organ failure, the normal healing process is significantly impaired and closure of the ulcer is less likely to occur. Although the mortality rate of persons with PrUs and coexisting illnesses is high, death does not necessarily have to occur within a specified amount of time for a lesion to be considered skin failure.

Internal organs and organ systems are often stratified according to the degree of dysfunction or failure (eg, acute, chronic and end stage). So, too, can skin and the underlying tissue be classified according to their degree of dysfunction in the broader context of multiple organ system failure. The degree of internal organ failure can often be monitored by a broad array of laboratory tests and imaging techniques. Decline of skin and underlying fat, fascia, and muscle, however, does not have such a wealth of tests to monitor declining function.[4,28] Gross examination of such variables as muscle mass, subcutaneous tissue thickness, wound granulation, and tissue necrosis is the current standard. Nevertheless, stratifying external organ system failure according to the patient's past, current, and future medical condition can be a useful method for planning interventions and setting realistic goals for treatment and healing.

Acute skin failure

Acute skin failure is an event in which skin and underlying tissue die due to hypoperfusion concurrent with a critical illness. An individual with few PrU risk factors can undergo an event that places him or her in an extreme medical condition for an extended period of time. Such events can include myocardial infarction, stroke, sepsis, trauma, or complications from surgery, among others. Significant risk factors for PrU formation that begin to prevail include sensory deprivation, immobilization, decreased perfusion, anemia, and malnutrition.

Immobilization often occurs during a critical or extended illness due to intubation, sedation, restraints, and consequences of disease processes. Prolonged low blood pressure is also commonly observed from volume depletion and/or sepsis, along with the use of vasopressors. During an extended stay in an intensive care setting, anemia often arises from blood loss through internal and/or external bleeding, procedures and

surgeries, or renal failure and/or dialysis—all common events in an intensive care setting.[30] Malnutrition is also a common occurrence due to sedation, intubation, and concern for aspiration.[31]

Pressure ulcers are commonly observed in an intensive care setting, with prevalence rates ranging from 33% to 56%.[32,33] Interventions to reduce the incidence of skin failure in an intensive care setting can be quite challenging. Sedation and immobilization are often required to keep the person breathing regularly and effectively and to prevent inadvertent self-harm. Vasopressors are required to maintain adequate core internal organ perfusion. The simple act of repositioning from side to side can significantly impact hemodynamic stability, as well as blood pressure and ventilator management. The use of pressure-redistributing mattresses can lower the interface pressure between the bed surface and the tissue, but an individual in such a critical condition may not have sufficient blood pressure and adequately nourished blood to maintain viability of the stressed tissue, despite the most advanced surfaces.[34]

Even with consistent, ongoing assessment of the stressed areas, early identification of an ischemic lesion, and prompt interventions to mitigate damage, skin and underlying tissue failure can become inevitable or unavoidable in such critically ill persons. Dysfunction of internal organs, such as acute renal failure and critical illness neuromyopathy, is a well-documented and accepted complication of complex and extended stays in an intensive care setting.[35,36] Skin failure in critically ill persons, after well-documented preventative measures have been implemented, should be viewed in this context as well. Mortality rates range from 33% within 30 days to 73.3% within 1 year of onset of skin failure in the intensive care population.[22]

Chronic skin failure

Chronic skin failure is an event in which skin and underlying tissue die due to hypoperfusion concurrent with an ongoing, chronic disease state. In contrast to acute skin failure, chronic skin failure occurs in a more steady fashion over time in the presence of chronic illness. Individuals experiencing chronic skin failure are usually older and have multiple comorbidities that predispose them to increased PrU risk. One by one, internal organ systems increasingly and irreversibly lose their ability to function as the end of life nears. A progressive decline in mentation, which leads to decreased functional ability, is commonly observed. In turn, decline in functional ability often results in malnutrition and inevitable loss of fat and muscle mass. Degeneration in overall functioning can be accelerated by a number of comorbid conditions and can occur to a point at which the combination of age-related declines and chronic comorbidities accelerate the loss in functioning in the end stages of life.[37]

Deterioration of internal organ systems is often manifested in the external organ system—the skin. Ultimately, the skin and underlying tissue will fail when they can no longer be supported by multiple chronically failing organ systems. Interventions to prevent such external skin failure present challenges to multiple disciplines. Nutritional support, hydration, medical management, hygiene, functional rehabilitation, and pressure redistributing surface selection all play an important role in preventing or mitigating skin failure in the chronically ill person.[38] Interventions to prevent skin failure must be appropriately attempted and well documented to determine their ability to be avoided. Given the dying condition that often accompanies skin failure, healing is not usually a reasonable goal.

End-stage skin failure

End-stage skin failure is an event in which skin and underlying tissue die due to hypoperfusion concurrent with the end of life. Mortality rates range from 20.8% within 30 days of first detection of skin failure to 73.3% at 1 year after onset of skin failure in the long-term-care setting.[22] The occurrence of skin failure in the chronically ill—like any other organ system failure, such as renal, respiratory, and cardiac—should be used to establish a dialogue among all involved parties regarding the pros and cons of future aggressive medical interventions.

The chronically critically ill population[39,40] present many challenges to maintaining skin integrity. The individual may not follow a continuum from acutely to chronically ill to skin failure. In one individual, the issue may be the effects of age and chronic disease superimposed on an acute illness and a stay in an intensive care setting. Another middle-aged individual may develop acute septicemia, septic shock, and MOF over a period of several hours. Organ decompensation and failure in the final days or weeks of life can result in large and unusual presentations of skin failure as the body organs shut down and the body naturally shunts blood to vital organs in a last resort attempt to maintain internal organ system function. Widespread and deep tissue destruction over stressed areas, such as the sacrum, heels, posterior calf muscles, arms, and elbows, can appear in a matter of hours or less. One cannot grossly observe internal organ system death, yet multiple organ death eventually reveals itself on the external skin as the underlying tissue death occurs. In such grave situations, few interventions can lessen the external skin damage as the body actively begins to shut down.[34] Clear, honest communication of medical assessments and prognosis among health care providers and the patient, significant others, and caregivers

can assist in establishing realistic goals for treatment and the acceptance of unavoidable complications at the end stages of life.[39]

A DILEMMA

Health care providers have at their disposal a wide variety of objective clinical data, laboratory results, and imaging studies with which to assess for and determine the extent of internal organ failure. Data on failing internal organs are invaluable in assisting the clinician, patient, family, and significant others in understanding the extent of damage. Laboratory values and black and white imaging of internal organs are objective and emotionally detached and generally do not invoke a negative gut response.

External organ failure, specifically skin failure, however, is visible to the eye. It tends to cause a more emotional response because wounds are generally esthetically displeasing, painful, and, oftentimes, malodorous. Methods for healing and assessing progress or digress are still subject to significant debate. Skin and underlying tissue repair, especially full-thickness repair, can be a lengthy and difficult process. Skin failure, whether acute, chronic, or end stage, occurs at an emotional time in life for the patient and the caregiver. Due to the often unpleasant nature of wounds and the negative emotional response to them by the professional and lay person alike, the goal is often to completely eliminate or heal them, regardless of how much it costs and whether the goal is achievable.

Society has tended toward presumed negligence on the part of health care providers when PrUs occur. Concurrently, the expectation of healing all wounds continues to exist. The 2004 revised guidelines from the Centers for Medicare & Medicaid Services recognize PrUs as unavoidable when appropriate assessment and interventions are implemented and documented.[41] This recognition has helped to foster a movement toward a more palliative-focused care approach for wounds, rather than the typical curative focus.

As society continues to be cure-oriented, one must question if advancing medical technology prolongs dying at the cost of more patients developing a PrU. Pressure ulcers will occur as the result of MOF because the skin is in the active dying process with the other organ systems. This is not often preventable, but must be prevented or limited to the most reasonable extent possible.

Patients have the right to choose their treatments, and health care providers have a responsibility to respect these choices. If an individual chooses, for example, not to use a pressure-reducing device or a supplemental feeding device and a PrU develops, it is not the result of poor care but rather the result of the right of the individual patient to elect or reject treatments. Respecting wishes of an individual patient who understands the consequences of these decisions is care with dignity.

CONCLUSION

The term skin failure was defined by the authors, based on this literature review, as an event in which the skin and underlying tissue die due to hypoperfusion that occurs concurrent with severe dysfunction or failure of other organ systems. This definition recognizes that the skin and underlying tissue will, in many cases, ultimately fail, despite the most modern and comprehensive interventions in the presence of a heavy disease burden. Assessment and diagnostic technology is not yet available to detect tissue necrosis in its most formative stages so that such damage can be minimized or prevented. Pressure ulcer detection is still a highly visual exercise that inevitably reveals damage after the fact.

The medical and nursing literature abound with studies on the prevention and treatment of PrUs. Far fewer, yet relevant studies conclude that a PrU is oftentimes an end-stage, pathologic condition that may not always be preventable or treatable. Preventing any untoward event detrimental to the health and well-being of an individual is an important goal. Prevention and treatment must, however, be implemented with a realistic understanding of what can be achieved, given the health status of individuals with irreversible acute illness or multiple chronic illnesses or those at the end stages of life. ●

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