The 18th Annual Cutting Edge Wound Care Symposium

November 14, 2025

11:15 AM - 12:15 PM

When Skin Fails Too: Pressure Ulcers and the Overlooked Organ

Pamela Scarborough, PT, DPT, MS, CWS, FAAWC

Pamela has been drawn to healthcare since her teenage years. Her journey in physical therapy has been fulfilling, providing opportunities in Orthopedics, including Sports Medicine, Total Joint and Spine care, Cardiac Rehabilitation, Geriatric physical therapy, and Wound Management. Pamela specialized in Wound Management with her Certified Wound Specialist (CWS) designation 24 years ago and has had an incredibly rewarding career providing wound care to patients, as well as teaching and mentoring clinicians in the specialty of wound management. She has supported healthcare professionals in acquiring their Board Certifications as wound specialists.

In addition, Pamela was credentialed for 20 years as a Certified Diabetes Educator (CDE), having a deep interest in diabetes prevention and management, understanding its connection to multiple health issues, including hard-to-heal wounds.

Northeast Surgical Wound Care

When the Skin Fails Too: Pressure Ulcers and the Overlooked Organ

Pamela Scarborough
PT, DPT, MS, CWS
Director of Education/Fellowship
United Wound Healing

Objectives

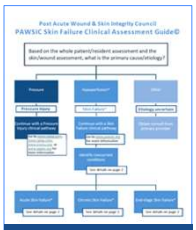
At the end of this presentation participants will be able to:

1. Define the terms associated with chronic and end-of-life skin failure;
2. Recognize how underlying organ failure (part of the PU/PI risk assessment) contributes to the etiology of skin failure;
3. Document clinical findings that identify residents at risk for unavoidable PU/PI/skin failure before an unavoidable lesion/injury/ulcer arises.

2

Post Acute Wound & Skin Integrity Council (PAWSIC) Resources Available to Download

Skin Failure Commentary John La Puma August 2025



PAWSIC Skin Failure Clinical Assessment Guide June 2025

References

Why This Topic???

- ALL care settings are taking care of people who are in various stages of organ failure
- We're **missing the big picture of organ involvement** and how skin is negatively affected as organs become compromised and continue their journey through impairment (chronic organ dysfunction) to failure
- **Recognizing organ impairment/failure as part of skin deterioration/damage is not in the forefront of the assessment processes currently in place in our care settings for PU/PI prevention or treatment**
- Not having this comprehensive awareness and clinical assessment processes in place to identify residents at risk for skin issues **early in the intake process** creates quality of care challenges related to overlooked risk factors for PU/PIs and SF
- Issues include: family/resident anxiety due to skin changes, survey readiness holes due to etiologies not captured that created the skin issue, and legal risks
- We must go **BEYOND** the skin assessment and the Braden to identify skin threats from organ systems' involvement for PU/PI and for hard to heal/stalled wounds!!!

Bottom Line

- Clinicians need better understanding of the pathophysiology and classification of Integument injuries **by underlying etiologies both avoidable and unavoidable**.
- More accurate review of **ALL** clinical diagnosis for people with skin failure or pressure injuries would lead to:
  - Appropriate treatment strategies,
  - Improved quality of life for affected patients,
  - Less wasted resources,
  - Reduced financial penalties for healthcare providers,
  - Decreased medical-legal claims.

2020 Article Available as Resource

REVIEW

The Pathophysiology of Skin Failure vs. Pressure Injury: Conditions That Cause Integument Destruction and Their Associated Implications

Michael Bain, MD, MMSc; Junko Hara, PhD; and Melissa J. Carter, PhD, MA*

ABSTRACT

Introduction. Although integument failure commonly is attributed to pressure alone, especially when a stage III develops over a bony prominence (pressure injury), all skin failure should not be attributed to pressure injuries. Objective. A systematic review of the literature was conducted to: (1) differentiate the types of integument injury and etiology; (2) describe the anatomic and pathophysiologic factors affecting integument failure; (3) differentiate avoidable vs. unavoidable integumentary injury of nonpressure-related sources; (4) describe factors leading to integument injury, including comorbid and risk factors; and (5) identify the clinical and economic importance of delineating pressure injuries from integument failure and associated risk factors in order to determine the pathophysiology underlying wound development and multiple factors capable of interacting with pressure to synergistically influence integumentary failure. Methods. The PubMed database was searched for English-language studies during March 2019 using the key words pathophysiology, etiology, pressure ulcers, pressure injury, pressure wounds, and risk factors. Results. The PubMed search yielded 636 publications in total; of these, 59 were selected for review based on their relevance, timeliness, and impact; in addition, 50 original studies of any study design, 3 review articles, and 4 public agency reports that addressed this study's specific components. Conclusions. Clinicians need to better understand the pathophysiology and classification of integument injuries by underlying etiologies both avoidable and unavoidable. A more accurate diagnosis was linked to more appropriate treatment strategies, an improved quality of care for affected patients, less wasted resources and reduced financial penalties for healthcare providers, and decreased medicolegal claims.

KEY WORDS

Integument failure, pressure injury, pressure ulcer, pathophysiology, skin failure

INDEX

Wounds 2020;32(11):319-327.

Can share this article per the publisher.

Bain M, Hara J, Carter JF. The Pathophysiology of Skin Failure vs. Pressure Injury: Conditions That Cause Integument Destruction and Their Associated Implications. Wounds 2020;32(11):319-327.

Multiple Organ Dysfunction Syndrome (MODS)

Leads to:

Skin failure aka Kennedy Terminal Lesion/Ulcer

aka

End of Life Wounds (EOL)
a type of Unavoidable Pressure Injury
Per CMS

Multiple Terms Currently Used to Describe Unavoidable Skin Changes associated with Multi-Organ Dysfunction and Skin Failure

- Several classifications/terms for similar/overlapping clinical syndromes
  - Kennedy Lesion/Terminal Ulcer (KTU) (CMS SOM-F684 and F686)
  - Skin Changes at Life's End (SCALE) - 2008
  - Trombley-Brennan Terminal Tissue Injury (TB-TTI) - 2010
  - Skin Failure - 2016
  - Unavoidable pressure ulcer/injuries (PU/PI) CMS SOM F686)
- May be a heralding sign of organ failure where the skin is failing at the same time as other body systems
- Recognizing residents in multi-organ pathologies is important for care planning for **potential** end-of-life skin failure

Often the Fault Lies with the Body, NOT the Building

In 1991 an author asked this question, which was related to the ethics of pressure ulcer development and blame

The author stated: *"The skin is the largest organ of the body. If the heart, lungs, and kidneys are showing signs of failing, isn't it logical that the skin would also show signs of failing? Why is a pressure ulcer considered a sign of inadequate healthcare, when manifestations of heart disease or lung disease or kidney disease are not?"*

LaPuma J. The ethics of pressure ulcers. Decubitus 1991;4(2):43-4.

An Avoidable Pressure Injury is a Symptom of a Failed System

Did the facility PI/PU prevention system fail or is the resident's BODY in failure?

La Puma – 2025 Commentary of Skin Failure

Terminal End-of-Life Skin Definitions

Skin Failure: 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.

Kennedy Terminal Ulcer: Can emerge suddenly, often within a matter of hours. Typically manifest in areas such as the sacrum and coccyx, but they can also develop on the heels, posterior calf muscles, arms, and elbows. Their edges tend to be irregular and can change in color as the ulcer advances, sometimes resembling the shape of a pear, butterfly, or horseshoe. Initially, they might resemble abrasions, blisters, or darkened areas, and they can rapidly progress s to more severe stages, such as Stage 2, , or 4 ulcers.

Attribution: Dot Weir

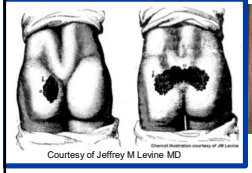
Trombley-Brennan Terminal Tissue Injury (TB-TTI): spontaneously appearing skin alterations, with rapid evolution, speed of enlargement and progression, and appearance in areas of little to no pressure, such as shins, thighs, and mirror imaging found in patients at the end-of-life

Skin Changes at Life's End: Observable changes in the skin that typically occur at the end-of-life (days to weeks, months) or may also occur with multiple organ failure in acute or chronic illness.

Unavoidable PU/PI: Unavoidable PU/PI to mean that "the resident developed a PU/PI even though the facility has evaluated the resident's clinical condition and risk factors; defined and implemented interventions that are consistent with resident needs, goals, and professional standards of practice; monitored and evaluated the impact of the interventions; and revised the approaches as appropriate".

Decubitus Acutus/ Ominosus Historical Medical Term

Decubitus Acutus (Unilateral) | Decubitus Ominosus (Bilateral)



Courtesy of Jeffrey M Levine MD

Attribution: Karen Kennedy
Termed 3:30 Syndrome

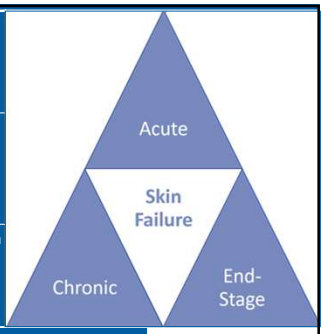
Jean-Martin Charcot
1825-1893

- Skin breakdown heralding impending death of patient - decubitus ominosus/acutus.
- This name was forgotten until late 20th century when Karen Kennedy recognized and published information on what became known as the Kennedy Lesion in 1989.
- The name was changed to Kennedy Terminal Ulcer (KTU) and is now recognized as a form of skin failure.

Acute Skin Failure: "an event in which skin and underlying tissue die due to **hypoperfusion** concurrent with a critical illness" (e.g., acute respiratory, renal failure)

Chronic Skin Failure: "an event in which skin and underlying tissue die due to **hypoperfusion** with a chronic disease state" (e.g., chronic respiratory, renal, heart failure).

End-Stage Skin Failure: "an event in which skin and underlying tissue dies due to **hypoperfusion** concurrent with the end-of-life" (e.g., terminal cancer terminal or end-stage diagnosis, failure–to-thrive).

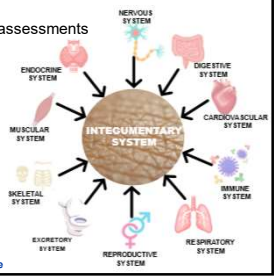


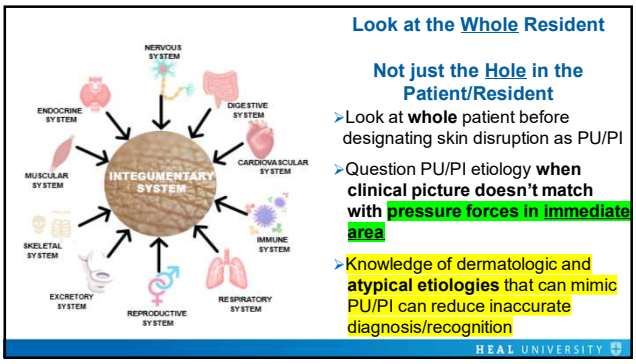
Langemo, D. K., & Brown, G. Skin fails too: Acute, chronic, and end stage skin failure. Adv Skin Wound Care 2006;19(4):206–211

End-of-Life Skin and Pressure-Related Wounds Differentiation

- Distinguishing between EOL skin manifestations and pressure-related wounds **require attention to clinical assessments**
- Assessment must go **beyond** the skin & Braden assessments
- Development of wounds not related to trauma, or wounds that do not heal – **is a symptom of failing physiology from comorbidities**, or **an infection**
- Wounds **not** healing as expected, reassess overall physiology of patient
  - 11-organ systems including the skin
  - Skin failure/EOL wounds/pressure injuries are associated with multi-organ impairment/failure
  - Providers must assist with recognition and documentation of EOL wounds

EOL = End-of-Life





Look at the Whole Resident

Not just the Hole in the Patient/Resident

- Look at **whole** patient before designating skin disruption as PU/PI
- Question PU/PI etiology **when clinical picture doesn't match with pressure forces in immediate area**
- Knowledge of dermatologic and **atypical etiologies** that can mimic PU/PI can reduce inaccurate diagnosis/recognition

HEAL UNIVERSITY

Two of These are Forms of Skin Failure

Moisture Associated Skin Damage	Deep Tissue Pressure Injury	KTU (AKA)	Chronic Tissue Injury	COVID-19 Skin Manifestation
Superficial Partial thickness	Deep damage at muscle/bone interface	Form of Skin Failure From MOF Closely mimics DTI	Superficial Aka Recliner Butt (Venous extravasation)	Associated with coagulopathy issues (clots) from cytokine storm

MOF=Multiorgan Failure

Statements in paper:

- Integument failure of various etiologies is a significant issue for severely ill patients in both acute and chronic care settings (e.g. LTC, HH)
- The pathology and **anatomic etiology** underlying integument failure are not clearly described *in the literature*
- Because of the complexity of the underlying mechanism leading to integument failure, it is imperative not only to better understand its underlying pathophysiology, but also to accurately classify integument injury by the avoidable and unavoidable factors responsible for injury (primary cause) including pressure
- NOTE: Please read this paper to have an awareness of angiosomes anatomy and how this vascular structure is associated with tissue damage

The Pathophysiology of Skin Failure vs. Pressure Injury: Conditions That Cause Integument Destruction and Their Associated Implications

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KEY WORDS

integument failure, pressure injury, pressure ulcer, pathophysiology, skin failure

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Assessment, Diagnosis, and Differential Diagnosis of Skin Failure

Medical Diagnosis

- Skin Failure (SF) must be diagnosed by a *qualified medical provider*:
  - Physician (MD, DO)
  - Podiatric physician (DPM)
  - Nurse practitioner (NP)
  - Physician associate (PA)
- **Not authorized** to diagnose: RNs, LPNs/LVNs, coders, PTs, MDS coordinators
- Coders and documentation staff must follow the provider's diagnosis per the ICD coding process

The Examination

- Assessing the person with hard-to-heal wounds:

Holistic assessment – essential

- **Systemic factors** (the overall health and comorbid conditions of the patient),
- **Regional factors** (the condition of the affected limb or region, including circulation and tissue quality),
- **Local factors** (the wound's characteristics).

- International consensus guidelines emphasize that accurate wound diagnosis and treatment planning must **begin by addressing systemic and regional etiologies before focusing on the local wound bed**

- Eriksson E, et al. *Wound Repair Regen*. 2022;30(2):156-171. Erratum in: *Wound Repair Regen*. 2022;30(4):536.
- **American Diabetes Association (ADA), American Heart Association (AHA), American College of Cardiology (ACC)**. 2024 AHA/ACC/ADA Guideline for the Management of Lower Extremity Peripheral Artery Disease: A Report of the American College of Cardiology/American Heart Association Joint Committee on Clinical Practice Guidelines. *J Am Coll Cardiol*. 2024;83(11):1171–1274. doi:10.1016/j.jacc.2023.11.021

Systemic Assessments

- Evaluating patient's overall health, underlying diseases, and general risk factors that might cause a wound or impede healing is critical to care planning
- Best practice guidelines call for identification and optimization of these **whole-body factors** as a **first step in wound management**
- Without resolving/managing the primary cause/s, wound healing often impaired: stalls, worsens or recurs
- Do you think systemic assessments are performed consistently?
- Do you think systemic conditions are being **managed consistently** to optimize wound healing by the PCPs and other specialists?

Importance of Lab Values and Organ Cross Talk in Relation to Organs' Function

Can be applied to organ impairments associated with:

1. Etiology of wound
2. Healing opportunities
3. Explain failure to heal

Lab Values
Basic vs Comprehensive Metabolic Panels

Basic Metabolic Panel

- BMP is typically used for:
- Basic screening (e.g., hydration, kidney status, glucose)
- Monitoring electrolyte levels in acutely ill or hospitalized patients

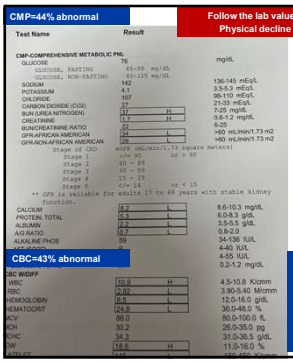
Comprehensive Metabolic Panel

- CMP is preferred when:
- You need a broader metabolic assessment
- You're monitoring patients with liver disease, chronic illness, or nutritional concerns
- To identify organ failure the **CMP is more comprehensive**

Case Study

CMP=44% abnormal

Follow the lab values over months
Physical decline follows labs



Test Name	Result	Flag	Reference Range
GLUCOSE	76		mg/dL
GLUCOSE, FASTING			65-99 mg/dL
GLUCOSE, NON-FASTING			65-125 mg/dL
SODIUM	142		136-145 mEq/L
POTASSIUM	4.1		3.5-5.3 mEq/L
CHLORIDE	107		98-110 mEq/L
CARBON DIOXIDE (CO2)	27		21-33 mEq/L
BUN (UREA NITROGEN)	37	H	7-25 mg/dL
CREATININE	1.7	H	0.6-1.2 mg/dL
BUN/CREATININE RATIO	22		6-25
GFR-AFRICAN AMERICAN	34	L	>60 mL/min/1.73 m2
GFR-NON-AFRICAN AMERICAN	28	L	>60 mL/min/1.73 m2
Stage of CKD	eGFR (mL/min/1.73 square meters)		
Stage 1	>/= 90		or > 90
Stage 2	60 - 89		
Stage 3	30 - 59		
Stage 4	15 - 29		
Stage 5	</= 14		or < 15
** GFR is reliable for adults 17 to 69 years with stable kidney function.			
CALCIUM	8.2	L	8.6-10.3 mg/dL
PROTEIN, TOTAL	5.3	L	6.0-8.3 g/dL
ALBUMIN	2.2	L	3.5-5.5 g/dL
A/G RATIO	0.7	L	0.8-2.0
ALKALINE PHOS	59		34-136 IU/L
			4-40 IU/L
			4-65 IU/L
			0.2-1.2 mg/dL

CBC=43% abnormal

CBC W/DIFF			
WBC	10.9	H	4.5-10.8 K/cmm
RBC	2.62	L	3.90-5.40 M/cmm
HEMOGLOBIN	8.5	L	12.0-18.0 g/dL
HEMATOCRIT	24.8	L	36.0-48.0 %
MCV	88.0		80.0-100.0 fL
MCH	30.2		26.0-35.0 pg
MCHC	34.3		31.0-36.5 g/dL
RDW	18.6	H	11.0-16.0 %

Use labs to give family/representatives an indication of trajectory towards end-of-life and potential for skin failure or lack of healing

Test Name	Result	Flag	Normal Range
MPV	8.4		6.5-12.0 fL
ANISOCYTOSIS	1+	ABN	NEGATIVE
NEUTROPHILS	73.6		40.0-80.0 %
LYMPHS	17.6		13.0-48.0 %
MONOCYTES	7.4		2.0-12.0 %
EOS	1.1		0.0-8.0 %
BASO	0.3		0.0-2.0 %
NEUTS (ABSOLUTE)	6.00	H	1.50-7.80 K/uL
LYMPHS (ABSOLUTE)	1.90		0.90-5.50 K/uL
MONOCYTES (ABSOLUTE)	0.80		0.15-1.10 K/uL
EOS (ABSOLUTE)	0.10	L	0.20-0.80 K/uL
NUCLEATED RBC	0.3		<1.0 NRBC/100 W
PREALBUMIN	8	L	16-45 mg/dL

Organs in Failure

- Brain
- Kidney
- Skin

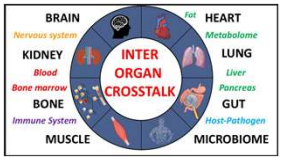
Conditions

- Anemia
- Inflammation
- Autoimmune disease
- Protein Calorie Malnutrition

Organ Crosstalk

"Organ crosstalk" refers to the **bi-directional communication between organ systems**, mediated by:

- Cytokines and inflammatory mediators
- Neurohormonal signaling
- Metabolic intermediates
- Vascular-arterial, venous, lymphatic networks
- Autonomic nervous system signaling



➢ In times of **systemic stress**, this communication becomes **dysregulated**, leading to **co-amplified organ damage**, including the skin.

Skin Breakdown and Non-Healing Wounds and End-of-Life Wounds

Skin integrity highly dependent on:

- **Perfusion** (cardiac output, blood pressure)
- **Oxygenation** (pulmonary exchange)
- **Nutritional supply** (gastrointestinal & **hepatic function**)
- **Waste clearance** (renal function)
- **Immune surveillance** (lymphoid & marrow-derived cells)

When these systems are failing in synchrony, skin compromised resulting in:

- Loss of **barrier function**
- Dysfunction in **cellular turnover**
- Unable to mediate **immune response**
- Resulting in **hard-to-heal / stalled wounds or skin failure / end-of life wounds**

Proposed Objective Indicators of Synchronous Organ Failure Affecting the Skin

Organ System	Measurable Marker	Effect on Skin/Wounds
Cardiac	↓ Cardiac output, ↑ BNP, low MAP	Hypoperfusion → ischemia, DTI, mottling
Pulmonary	↓ PaO_2, ↑ CO_2, ↓ SpO_2	Hypoxia impairs fibroblast & collagen synthesis
Renal	↑ BUN/Creatinine, ↓ urine output	Uremic toxins → poor wound granulation, fluid overload/edema
Hepatic	↑ LFTs, ↓ albumin, coagulopathy	Edema, impaired detox → inflammation & poor protein reserves
GI/Nutrition	↓ Prealbumin, weight loss, cachexia	Impaired collagen matrix, slowed repair
Immune/Inflammation	↑ CRP, IL-6, neutrophilia or lymphopenia	Chronic inflammation → delayed healing, higher infection risk
Endocrine/Glucose	↑ Cortisol, persistent hyperglycemia	Impaired neutrophil function, high glucose toxic to tissue

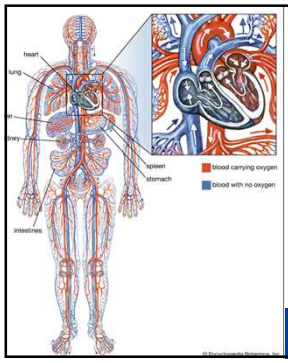
Recognizing Proposed Thresholds for Synchronous Failure

Skin failure represents the cutaneous manifestation of multi-organ dysfunction. Applying objective, measurable criteria ensures diagnostic accuracy, clinical consistency, and defensible documentation.

Objective indicators:

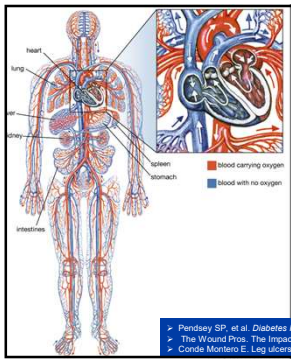
- **Cardiac**: ↓ CO, ↑ BNP, MAP < 65 mmHg
- **Renal**: Creatinine > 2.0 mg/dL, oliguria
- **Pulmonary**: PaO2 < 60 mmHg, SpO2 < 88%
- **Hepatic**: ↑ LFTs, INR > 1.5, Albumin < 2.5 g/dL
- **Endocrine**: Glucose > 180 mg/dL, ↑ Cortisol,
- **Inflammatory**: CRP > 100 mg/L, IL-6 > 40 pg/mL

Langley RG, Dutz JP, Lynde C, et al. *Skin as a barometer of internal organ dysfunction.* ***Journal of the American Academy of Dermatology*** *(JAAD).* 2015;72(3):481–490.



- Sources collectively emphasize that **impaired perfusion in heart failure is a central factor linking cardiac dysfunction to healing impairments.**

- Rhou YJJ, et al. *J Diabetes Complications*. 2015;29(4):556-62.2015;29. 10.1016/j.jdiacomp.2015.02.009
- Itzhak B, et al. *Diabetes Res Clin Pract*. 2021;176:108849. 10.1016/j.diabres.2021.108849



Vasculature to Organs

- Heart failure-of any etiology-reduces cardiac output and **redirects blood flow to essential organs** at the expense of other tissues
- **This adaptive redistribution preserves perfusion of brain, myocardium**
- Leads to chronic under-perfusion of **kidneys, gut, skin, skeletal muscles (muscle tissue most sensitive to hypoxia)**

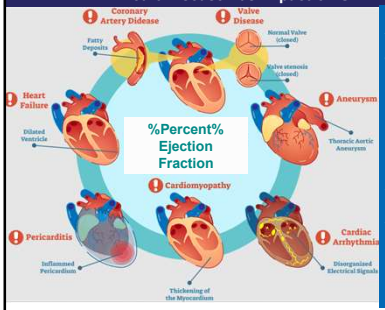
- Pendsey SP, et al. *Diabetes Res Clin Pract*. 2015;107(3):372-377. Sen CK. *Wound Repair Regen*. 2009;17(1):1-18.
- The Wound Pros. The Impact of Cardiovascular Diseases on Wound Healing. Published October 2021.
- Conde Montero E. Leg ulcers due to heart failure? Published July 17, 2022.

Pressure Injuries REQUIRE Local Pressure as Part of Their Etiology

Clinical Implication in Pressure Injuries

- Pressure ulcer/injuries begins in muscles and progresses outward
- Off-loading too late allows ongoing damage from edema and reperfusion injury
- Skin may appear intact while deeper tissue necrosis evolves
- **Key Point:** Muscle fails before skin because it experiences higher deformation, higher oxygen demand, and greater stress concentration—a localized hypoxia problem, not a systemic one

Heart Disease has Impact on Skin Health and Healing



A decreased **ejection fraction (EF)**—a measure of the heart's pumping efficiency—**can adversely affect skin health and wound healing**, particularly when it falls below **40%**, with more severe effects observed below **30%**.

Lee YJ, Ahn CM, Ko YG, Park KH, Lee JW, Lee SJ, Hong SJ, Kim JS, Kim BK, Choi D, Hong MK, Jang Y. Skin Perfusion Pressure Predicts Early Wound Healing After Endovascular Therapy in Chronic Limb-Threatening Ischaemia. Eur J Vasc Endovasc Surg. 2021 Dec;62(6):909-917.

AMERICAN JOURNAL OF PHYSIOLOGY
HEART AND CIRCULATORY PHYSIOLOGY

• Am J Physiol Heart Circ Physiol. 2022 Jan 28;322(4):H537-H548. doi: 10.1152/ajpheart.00612.2021

Faster skin wound healing predicts survival after myocardial infarction

Medina Becirovic-Agic [1,2], Upendra Chalise [1,2], Mira Jung [3], Jocelyn R Rodriguez-Paar [1,2], Shelby R Konfrst [1,2], Elizabeth R Flynn [4], Jeffrey D Salomon [1,5], Michael E Hall [6], Merry L Lindsey [1,2,85]

Findings:

In conclusion, wound healing **across organs** shares common pathways.

Tissue repair efficiency in one organ can predict tissue repair efficiency in a different organ.

Study showed that **faster skin wound healing** rate is a **predictor of more favorable outcome after MI.**

Skin wound healing may be used to screen for patients that are at risk of developing heart failure.

MI=myocardial infarction

Becirovic-Agic M, et al. *Am J Physiol Heart Circ Physiol*. 2022;322(4):H537-H548.

Congestive Heart Failure/Liver Disease Presence Predicts Delayed Healing of Foot Ulcers in Diabetes

- Comorbid **congestive cardiac failure is predictive of delayed foot ulcer healing rate**
- In addition - lower probability of healing overall
- **Liver enzyme abnormalities also predicted delayed ulcer healing outcomes**
- Mechanisms underlying these associations with foot ulcer outcomes in diabetes are unclear
- Further studies are needed to determine the role of systematic routine documentation of heart failure and its severity, and then targeting of heart failure to potentially aid the management of foot ulcers in diabetes

Rhou YJ, et al. *J Diabetes Complications*. 2015;29(4):556-62. Xu L, et al. *Journal of Diabetes*. 2013;5.

Hypotension and Wounds

- *Hypotension which can lead to **hypoperfusion** is associated with wound development and decreased healing rates.*
- *When blood pressure is <100 mmHg systolic and <60 mmHg diastolic the capillary closing pressure is diminished.*
- *Low blood pressure can compromise blood flow to the soft tissues and organs, causing tissue hypoxia.*
- *Resulting hypoxia can create a detrimental physiological cascade, causing cellular death resulting in soft tissue necrosis and eventual ulceration.*

Pittman J, et al. *Am J Crit Care*. 2019;28(5):338- 350. Bergstrom N, et al. *Nurs Res*. 1987;36(4):205-210.
Sibbald RG, et al. *Adv Skin Wound Care*. 2023;36(3):151-157.

Why Recognition of Skin Failure Matters

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Why Recognize Skin Failure?

Clinically

Recognizing skin failure ensures:

- Appropriate diagnosis and treatment planning
- Prevents mislabeling as pressure injury or other similar looking etiology
- Supports accurate prognostic discussions with the patient (if appropriate), care team, and family.

Example: A patient in multi-organ failure develops sacral skin necrosis despite optimal repositioning, support surfaces, etc.

- Recognizing skin failure guides realistic goals of care rather than escalation of futile interventions.

Why Recognize Skin Failure?

Ethically

- Aligns clinical decisions with patient-centered care and transparency—acknowledging when skin breakdown reflects underlying organ failure rather than neglect or poor care.
- **Example:** Transparent discussion with family acknowledges that tissue breakdown is part of organ decline, not neglect—maintaining trust and moral integrity of the care team.

Why Recognize Skin Failure?

Policy-Wise

- Recognition supports accurate documentation and coding practices that reflect **the true clinical picture**, guiding institutional protocols, quality indicators, and outcome tracking.
- **Example:** Accurate coding of skin failure (not Stage 3 PI) informs facility quality metrics and prevents distortion of pressure-injury prevalence data used for public reporting.

Why Recognize Skin Failure?

Regulations

- Correctly identifying skin failure prevents inappropriate citations and reimbursement denials, aligns with CMS and surveyor guidance, and ensures reimbursement reflects the patient's medical complexity rather than a preventable event.
- **Example**: Correct classification prevents citation under **F686** and aligns with **CMS guidance** that not all skin breakdowns are avoidable; ensures appropriate payment for medically complex care.

Why Recognize Skin Failure?

Legal Implications

- Failure to distinguish skin failure from pressure injury can lead to misinterpretation in investigations and litigation; accurate recognition supports defensible documentation and protects providers and organizations.
- Example: Clear documentation of skin failure etiology protects providers in post-survey or legal review by showing the wound was *clinically unavoidable* due to underlying systemic failure.

Why Recognize Skin Failure?

Research and Data Integrity

- Accurate identification and classification of skin failure are essential for valid research and data collection, ensuring that outcomes, prevalence rates, and prevention metrics truly reflect clinical reality rather than being confounded by **misclassified pressure injuries.**
- **Example:** When skin failure is **incorrectly coded** as a pressure injury, national prevalence data, quality benchmarks, and funding priorities become distorted—obscuring the true incidence of avoidable versus unavoidable wounds and undermining evidence-based policy and practice development.

Regulations

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Regulations

- Why is it important to differentiate skin failure from pressure injuries?
- National Quality Forum: (Acute Care)
  - Pressure injuries are **never events in *acute care***,
  - Acute care facilities are penalized as a result
- FTag 686: (Long Term Care)
  - ***"Kennedy Terminal Ulcers are considered to be pressure ulcers that generally occur at the end of life.***
- MDS-Section M: "***Skin changes at end of life (SCALE), also referred to as Kennedy Terminal Ulcers (KTU's) and skin failure, are not primarily caused by pressure and are not coded in Section M".***
- We need ICD-10 codes for skin failure for appropriate coding, reimbursement, and to facilitate research.

Acute Care Narrative Documentation for Medical Record

- Since no code exists, the *provider's narrative* becomes the authoritative defense.
- "Patient exhibits skin and tissue changes consistent with *acute skin failure* in the setting of multi-organ dysfunction and terminal decline. Despite appropriate preventive interventions (offloading, moisture management, support surface, repositioning as tolerated), tissue tolerance was exceeded.
- Lesion is *not consistent with a preventable pressure injury* and reflects systemic hypoperfusion associated with end-of-life physiology."

This language should appear in:

- Daily progress notes (MD/DO/NP/PA),
- Wound/skin specialist notes,
- Discharge summary or death summary, and
- Interdisciplinary notes (nursing, wound care, palliative care).

Use of Proxy Diagnostic Codes

Although not ideal, coders can pair *descriptive terms* with **existing ICD-10-CM codes** that represent the **systemic failure and resulting tissue necrosis**:

Clinical Phenomenon	ICD-10-CM Coding Options (Examples)
Multi-organ failure / systemic decline	R57.9 (Shock, unspecified), R65.21 (Severe sepsis with septic shock), R65.10 (Systemic inflammatory response), R09.02 (Hypoxemia)
Cutaneous necrosis / ischemia (non-pressure)	L98.49 (Other non-pressure chronic ulcer of skin, other specified sites), L98.8 (Other specified disorders of skin and subcutaneous tissue), L98.9 (Disorder of skin, unspecified)
End-stage / terminal decline	Z51.5 (Encounter for palliative care), Z66 (Do not resuscitate status)

NOTE: Presenter is NOT a coding specialist. Please review any suggestion on coding with your coding team.

CMS SOM F686

INTENT of F686 Related to PU/PIs

- *"The intent of this requirement is that the resident does not develop pressure ulcers/injuries (PU/PIs) unless clinically unavoidable and that the facility provides care and services consistent with professional standards of practice to:*
  - *Promote the prevention of pressure ulcer/injury development;*
  - *Promote the healing of existing pressure ulcers/injuries (including prevention of infection to the extent possible); and*
  - *Prevent development of additional pressure ulcer/injury."*

F686 Take of End-of-Life Wounds

- Residents at the **end of life**, in **terminal stages** of an illness or having **multiple system failures** may have written directions for his or her treatment goals (or a decision has been made by the resident's representative, in accordance with State law).
- It is **important for surveyors to understand** that when a facility has **implemented individualized approaches for end-of-life care** in accordance with the resident's wishes, the development, continuation, or worsening of a PU/PI may be considered unavoidable.
- If the facility has implemented appropriate efforts to stabilize the resident's condition (or indicted why the condition cannot or should not be stabilized) and has provided care to prevent or treat existing PU/PIs (including pertinent, routine, lesser aggressive approaches, such as, cleaning, turning, repositioning), the **PU/PI may be considered unavoidable and consistent with regulatory requirements.**

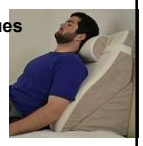
CMS SOM F686

Kennedy Terminal Ulcer (KTU) per SOM

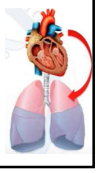
"The facility is responsible for accurately assessing and classifying an ulcer as a KTU or other type of PU/PI and demonstrate that appropriate preventative measures were in place to prevent non-KTU pressure ulcers."

- **Document** → Appropriate measures in place to prevent **avoidable** PU/PI
- **PI Prevention Plan** → Prevention care plan in place **BEFORE** KTU/SF can be called Unavoidable PI
- **All Risk Factors Identified?** → Show KTU/SF related to organ failure or other non-modifiable risk factors **Providers involvement**

Example of Increased Risk for an Unavoidable Pressure Ulcer/Injury



- Patients/residents with **feeding tubes** or **respiratory issues** often need head-of-bed elevated more than 30° degrees
- Contrary to usual pressure injury prevention care plans
- Care plans and documentation in the medical record will need to address why HOB with high elevation
- Ex. **Resident with end-stage CHF/pulmonary edema**
- **Provider** write order for higher elevation of HOB if needed to facilitate breathing
- Providers write: PU/PI risk very high due to positioning requirements to facilitate breathing



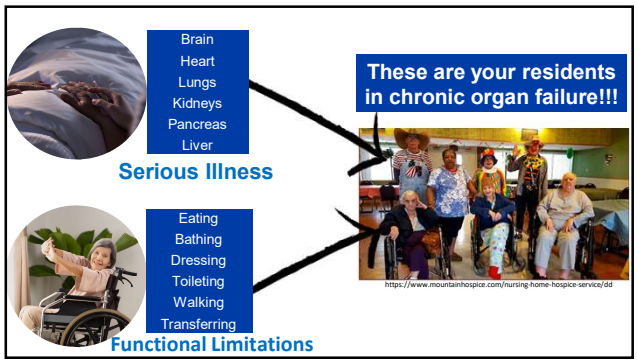
- One example…many illness and conditions put people at risk for unavoidable skin changes and breakdown

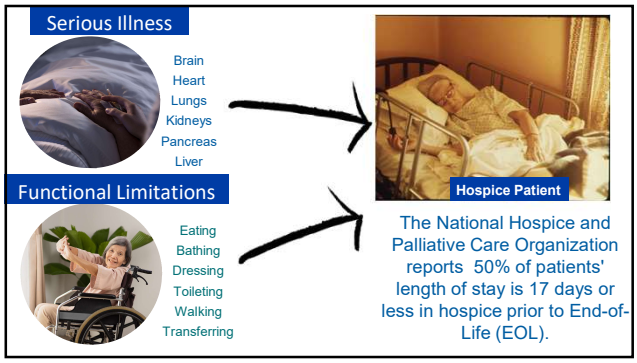
Clinical Pearls

Until we have codes for Skin Failure – Skin Changes and Wounds due to skin failure:

- Document the assessment and description of the skin changes/wounds thoroughly in the healthcare record
- If permitted by your facility policy, photograph the skin changes/wounds
- Consider using the following diagnosis code:

L98.89 Other specified disorders of the skin and subcutaneous tissue





In closing:

Statement from Dr. John La Puma, MD, FACP

Skin Failure Shared Interest Group
August 2025
Commentary on Skin Failure by John La Puma, MD
34-years after his 1991 Statement

"The scarcity of dedicated data on Skin Failure (SF) is a direct consequence of the problem we aim to solve, but there is a growing body of literature that, when synthesized, makes a powerful case.

My thoughts on the matter are that we must reframe Skin Failure not as a severe type of pressure injury (PI), but as a distinct clinical syndrome . . . SF is a systemic phenomenon where the skin, as an organ, begins to fail due to hypoperfusion, often in the context of critical illness, multi-organ dysfunction, or the dying process. This conceptual shift recognizes SF across its acute, chronic, and end-stage presentations.

The persistent misclassification of SF as a PI is a categorical error with devastating ethical, economic, and personal consequences. Ethically, it robs patients, particularly those at the end of life, of a dignified experience by subjecting them to painful, futile interventions aimed at "healing" an organ that is shutting down, rather than shifting to palliative goals focused on comfort. This misdiagnosis creates a false and painful narrative of neglect for families and inflicts a profound moral distress on clinicians who are wrongly blamed for an unavoidable outcome. Economically, this confusion fuels a deeply irrational system, wasting billions on inappropriate treatments and triggering unjust financial penalties for healthcare institutions under policies never intended for unavoidable organ failure.

The literature specifically using the term "Skin Failure" is indeed fragmented, as much of the data is incorrectly buried within studies on pressure injuries, thereby contaminating the evidence base. However, a coherent picture emerges when we analyze research on related phenomena now understood as manifestations of SF, such as the Kennedy Terminal Ulcer (KTU), Skin Changes at Life's End (SCALE), and Trombley-Brennan Terminal Tissue Injury (TB-TTI).

The path forward requires a concerted advocacy effort focused on two main goals.

First, we need robust educational initiatives to instill the principles of differentiation in all clinicians.

Second, and most critically, we must establish a unique ICD code for Skin Failure. This single policy change is the key that will unlock accurate data collection, enable fair quality metrics, drive targeted research, and, most importantly, ensure that our care for the most vulnerable patients is both clinically sound and profoundly compassionate."

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