Pressure Injury Impostors on Buttocks

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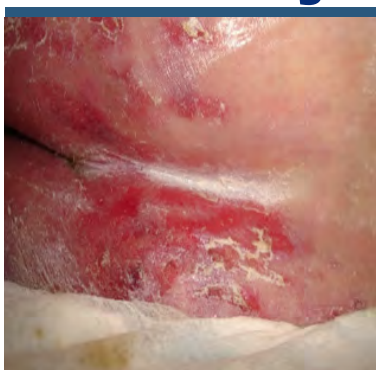
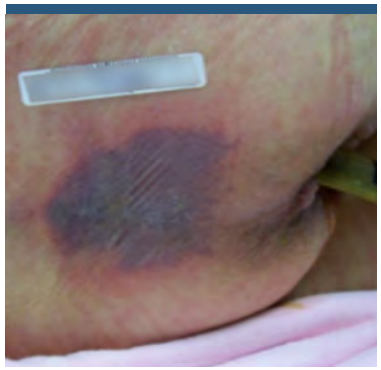
Can your teams differentiate these etiologies?



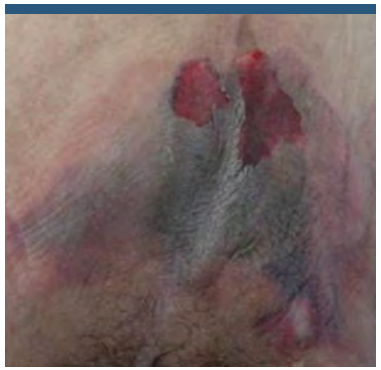
Moisture Associated Skin Damage

Prevalent in the LTC and home health settings**.** Due to sweating, episodes of incontinence, or wound drainage, or effluent from ostomies that leak onto the skin. NOT pressure etiology.



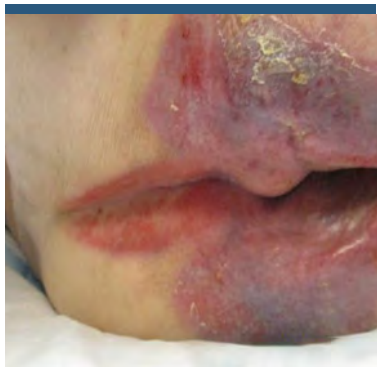
Deep Tissue Pressure Injury

Tissue and cellular damage at the bone-muscle interface creating full-thickness wounds. This IS pressure etiology.



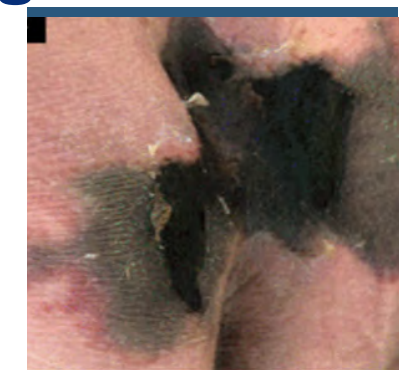
Kennedy Terminal Ulcer Skin Failure

Also known as skin failure, the KTU is recognized by CMS as a form of unavoidable pressure ulcer/injury that happens when a person is experiencing organ failure and the skin, the largest organ, also begins to fail. NOT pressure etiology.



Chronic Tissue Injury

Believed to result from venous pooling/ engorgement. Occurs on the fleshy portion of the buttocks. Patients and residents may be somewhat mobile. May be present for months. NOT pressure etiology.



COVID-19 Skin Manifestations

Associated with the disease COVID-19. May occur before, during, or after the disease or may be the only symptom of COVID-19. NOT pressure etiology.

Why is Accurate Recognition, Diagnosis and Reporting Important?

Pressure injuries and the associated stages and wound characteristics have clinical, regulatory (survey), costs, reimbursement, and legal implications.

Misidentification or misdiagnosis can lead to inappropriate documentation, mistakes in reporting to CMS, ineffective treatments and inappropriate use of resources.

Do NOT default to a pressure ulcer/ injury diagnosis, unless all the clinical criteria point to the PU/PI etiology.

References

- WOUNDVISION.com/npiap-imposter
- State Operations Manual. Appendix PP-Guidance to Surveyors for Long Term Care Facilities. 173, Rev. 11-22-17.
- https://link.zixcentral.com/u/c7bbfdc3/4l85PjBz7BGw_fUBh3soMg?u=http%3A%2F%2Fwww.kennedyterminalulcer.com%2F%23Q1.
- Mahoney MF, Rozenboom BJ. Definition and Characteristics of Chronic Tissue Injury. J Wound Ostomy Continence Nurs. 2019;46(3):187-191.
- Howell M, Loera S, Tickner A, Maydick D, Faust E, et al. Conditions That Mimic Pressure Ulcer/Injuries-To Be or Not To Be. Wound Management & Prevention 2021;67(2):12–38 doi:10.25270/wmp.2021.2.1238.

Rev03-2022

PRESSURE INJURY IMPOSTERS

AND

THE UNAVOIDABLE PRESSURE INJURY

Pamela Scarborough

PT, DPT, CWS, FAAWC

Vice President Clinical Affairs
Restorix Health / AMT

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DISCLAIMER

- This information is provided for informational purposes only. Patient management decisions should be based on a number of factors, including (but not limited to) professional society guidelines and published clinical literature relevant to a patient's condition. Providers are encouraged to rely on their training and expertise, as well as any and all available information, prior to making management or treatment decisions for any individual patient."

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OBJECTIVES

At the end of this educational session participants should be able to:

- Identify etiologies that look like but are not related to pressure forces.
- Describe the proposed etiology and appearance of chronic tissue injury.
- Verbalize the description of the Unavoidable Pressure Injury aka the Kennedy Terminal Ulcer/skin failure.
- Discuss potential issues with misidentifying/misdiagnosing an etiology as pressure-related when the lesion is NOT related to pressure forces.

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HANDOUTS THAT INFORMED PRESENTATION

1. PPT presentation PDF
2. Definition and Characteristics of Chronic Tissue Injury: A Unique Form of Skin Damage
3. Practice Dilemmas: Conditions that Mimic Pressure Ulcer/Injuries-To Be or Not to Be
4. The Pathophysiology of Skin Failure vs. Pressure Injury: Conditions That Cause Integument Destruction and Their Associated Implications
5. Differentiating PU from Acute Skin Failure
6. SCALE-Final Version
7. Deep Tissue Pressure Injury or an Imposter-NPIAP Document
8. Pressure Injury Imposter on the Buttocks-Differential Recognition
9. The Pathophysiology of Skin Failure vs. Pressure Injury: Conditions That Cause Integument Destruction and Their Associated Implications

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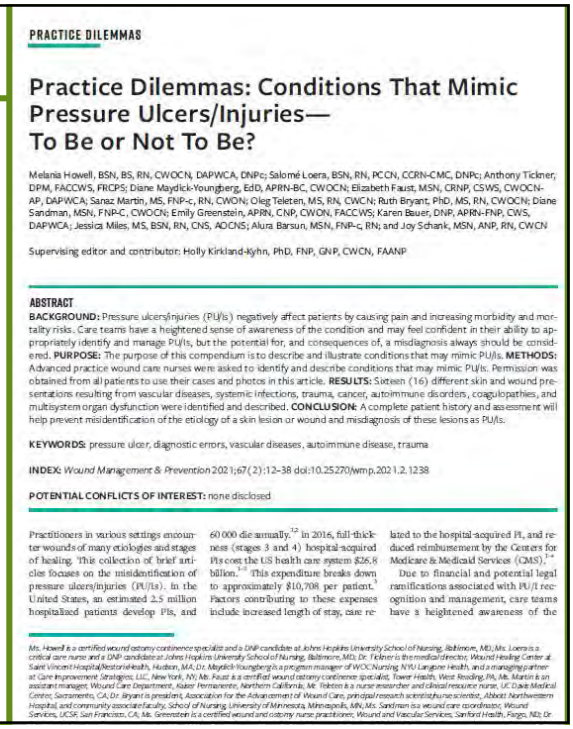

PRACTICE DILEMMAS

Practice Dilemmas: Conditions That Mimic Pressure Ulcers/Injuries— To Be or Not To Be?

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ABSTRACT

BACKGROUND: Pressure ulcers/injuries (PU/Is) negatively affect patients by causing pain and increasing morbidity and mortality risks. Care teams have a heightened sense of awareness of the condition and may feel confident in their ability to appropriately identify and manage PU/Is, but the potential for, and consequences of, a misdiagnosis always should be considered. **PURPOSE:** The purpose of this compendium is to describe and illustrate conditions that may mimic PU/Is. **METHODS:** Advanced practice wound care nurses were asked to identify and describe conditions that may mimic PU/Is. Permission was obtained from all patients to use their cases and photos in this article. **RESULTS:** Sixteen (16) different skin and wound presentations resulting from vascular diseases, systemic infections, trauma, cancer, autoimmune disorders, coagulopathies, and multisystem organ dysfunction were identified and described. **CONCLUSION:** A complete patient history and assessment will help prevent misidentification of the etiology of a skin lesion or wound and misdiagnosis of these lesions as PU/Is.

KEYWORDS: pressure ulcer, diagnostic errors, vascular diseases, autoimmune disease, trauma

INDEX: *Wound Management & Prevention* 2021;67(2):12–38 doi:10.25270/wmp.2021.2.1238

POTENTIAL CONFLICTS OF INTEREST: none disclosed

Practitioners in various settings encounter wounds of many etiologies and stages of healing. This collection of brief articles focuses on the misidentification of pressure ulcers/injuries (PU/Is). In the United States, an estimated 2.5 million hospitalized patients develop PIs, and 60 000 die annually.[1,2] In 2016, full-thickness (stages 3 and 4) hospital-acquired PIs cost the US health care system $26.8 billion.[1,2] This expenditure breaks down to approximately $10,708 per patient.[3] Factors contributing to these expenses include increased length of stay, care related to the hospital-acquired PI, and reduced reimbursement by the Centers for Medicare & Medicaid Services (CMS).[3,4]

Due to financial and potential legal ramifications associated with PU/I recognition and management, care teams have a heightened awareness of the

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INTRODUCTION

- True incidence of PI misdiagnosis unknown
- There are MANY conditions that mimic PU/PIs
- Program will identify some of the confounding conditions that are reported as pressure, including chronic tissue injury
- Today's program is primarily to introduce the term chronic tissue injury and to highlight other etiologies that mimic pressure-related damage
- Please read the accompanying handouts for more in-depth information related to these issues
- Support for conversations with your clinical teams, providers, surveyors, and attorneys.

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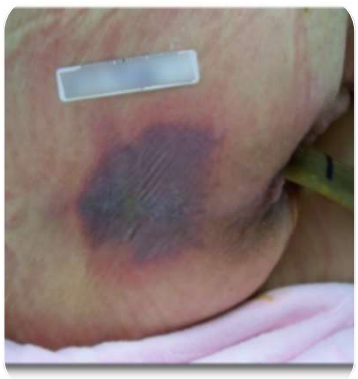
WHY IS THIS TALK IMPORTANT

- Pressure injuries, associated stages and wound characteristics have clinical, regulatory (survey), costs, reimbursement, and legal implications
- Misidentification or misdiagnosis may lead to:
  - Inappropriate documentation,
  - Mistakes in reporting to CMS,
  - Ineffective treatments,
  - Inappropriate use of resources,
  - Issues with the survey process,
  - Complicate/negatively impact legal defense.



- **DTPI Over Sacrum**
- **Mobility impaired Resident**
- **Document & Report as PI**

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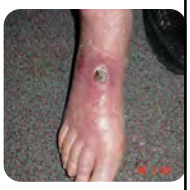
CMS SOM F684

GUIDANCE FROM F-TAG 684

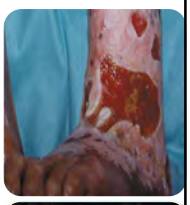
REVIEW OF A RESIDENT WITH **NON PRESSURE-RELATED** SKIN ULCER/WOUND

- Residents may develop various types of skin ulceration.
- At the time of the assessment and diagnosis of a skin ulcer/wound, the clinician is **expected to document the clinical basis (e.g., underlying condition contributing to the ulceration**, ulcer edges and wound bed, location, shape, condition of surrounding tissues) which permit differentiating the ulcer type, **especially if the ulcer has characteristics consistent with a pressure ulcer, but is determined not to be one.**

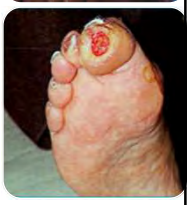
Arterial



Venous



Diabetic Neuropathic



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CHRONIC TISSUE INJURY

Recognition and Definition

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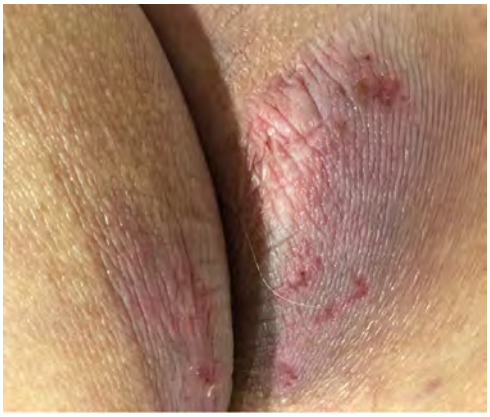
WHAT IS CHRONIC TISSUE INJURY (CTI)? SENILE GLUTEAL DERMATOSIS?

- Distinct form of skin damage affecting buttocks
- Presentation:
  - Purple-maroon discoloration
  - Thinning epidermis
  - With or without open injury
  - Lichenification often present
- Location
  - Fleshy part of buttocks
  - Usually **NOT** on boney prominences



- CTI on 80 y/o male, COPD, O_2 use, ambulatory, most of day spent sitting
- Purple/maroon discoloration for 6 months with multiple partial-thickness skin disruptions that close and acquire new small lesions in different locations

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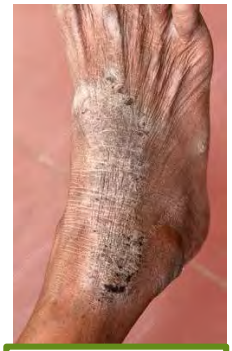
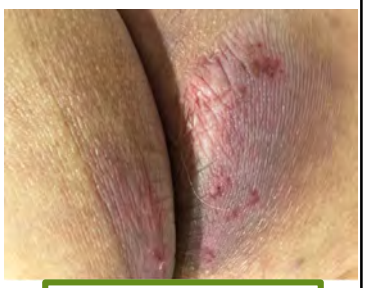
DOES THE SKIN HAVE LICHENIFICATION?

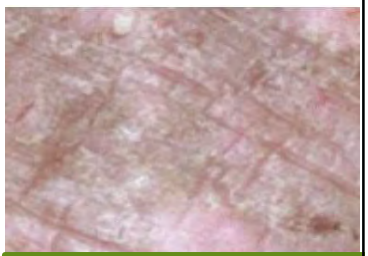
- Thickening and induration of skin
- Characterized by **exaggerated skin markings ("wrinkles")**
- Usually due to chronic trauma
- Often accompanied by hyperpigmentation
- Many clinical reasons for lichenification



Eczema induced Lichenification



Chronic tissue injury induced Lichenification



Scabies induced Lichenification

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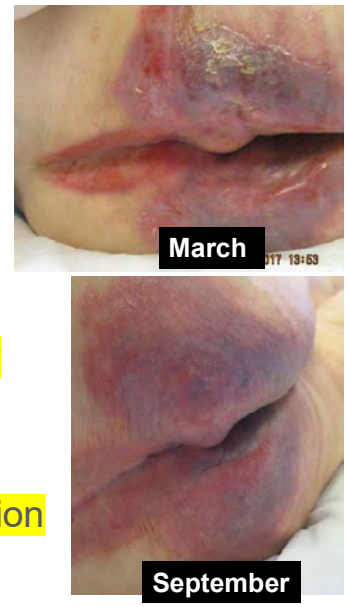
CHRONIC TISSUE INJURY ETIOLOGY

- Etiology not well understood
- Differential diagnosis confusing
- May occur with other skin injuries/issues
- Venous pooling and engorgement possible etiology
- Hemosiderosis-reddish/purple pigmentation
  - From extravasated red blood cells and inflammation
- NOT related to pressure



Courtesy: Dot Weir, RN, CWON, CWS

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WHY DO WE NEED THIS INFORMATION?

- No recognized 'label' for chronic tissue injury
- May be misidentified as pressure injury
- Clinical teams may choose wound type most closely resembling this tissue change when tissue damage is **NOT related to pressure**
- Results in inaccurate and inconsistent classification of soft tissue damage
- **Code for CTI/senile dermatosis: L98.8 / L98.9-discuss with your billing expert!!!**

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DISCUSS USING ONE OF THESE CODES FOR CTI WITH YOUR CODING EXPERT

L98.8 Other specified disorders of the skin and subcutaneous tissue

L98.9 Disorder of the skin and subcutaneous tissue, unspecified

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ACCURATE IDENTIFICATION OF WOUND TYPES CRITICAL

- Accurate identification of wound types required to guide appropriate care planning
- Incorrect identification of wound and skin disruption etiologies causes confusion with regulatory mandates for reporting pressure and other wound etiologies
- Creates issues with the survey process
- May set facilities up for legal issues

- **Accurate identification**
- **Critical for appropriate care planning**

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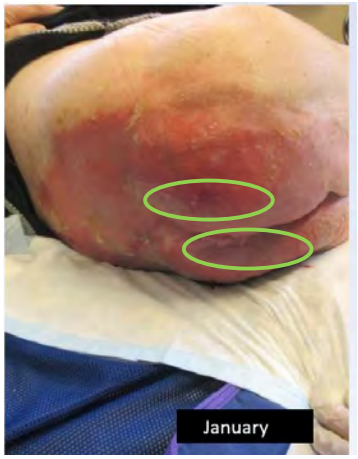
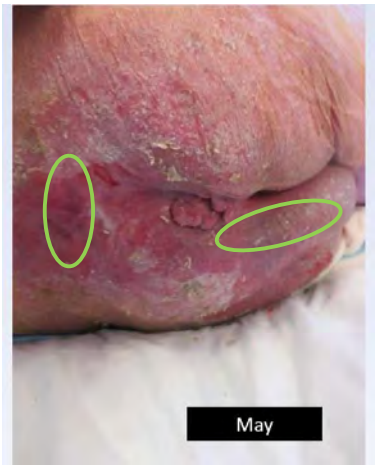
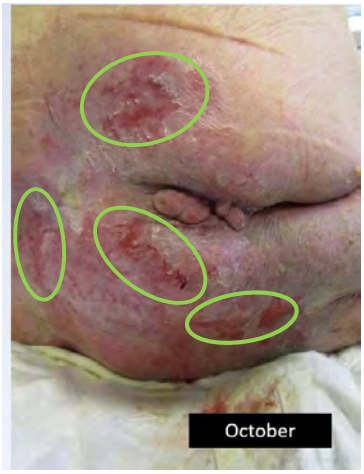
IS THIS PRESSURE RELATED???







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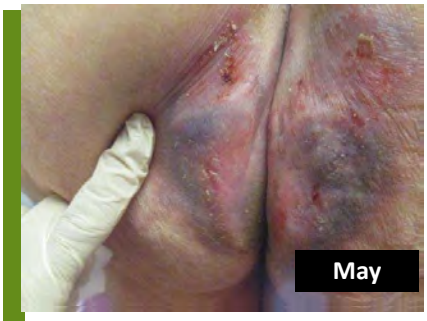
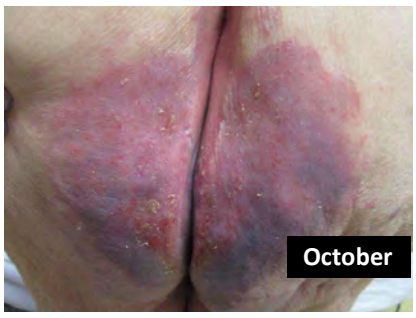
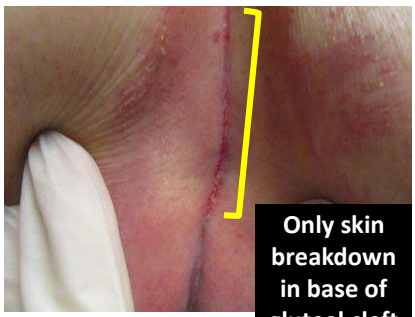
CHRONIC TISSUE INJURY VS CTI







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Courtesy: Dot Weir, RN, CWON, CWS

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ISSUES WITH DOCUMENTATION AND REPORTING OF CHRONIC TISSUE INJURY

- Lack of EMR identification
- No MDS reporting mechanism for this issue in LTC
- Usually reported as:
  - Stage 1,
  - Stage 2,
  - DTPI,
  - Skin failure (unavoidable pressure injury/KTU),
  - MASD
  - Trauma,
  - Inflammatory lesions

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DO NOT DEFAULT TO PU/PI DIAGNOSIS WHEN SKIN MANIFESTATION UNKNOWN

- Wide variety of etiologies produce wounds in vicinity of pressure-shear prone anatomical locations
- Be thorough in your assessment
- Nurses partner with providers **before** designating a wound as a PU/PI if clinical picture doesn't correlate with pressure forces as cause
- Once a PU/PI has been documented as Dx in medical record that Dx is often **maintained regardless of accuracy**
- **If you think the issue is chronic tissue injury and the provider or surveyor doesn't have this information yet, educate them.**
- **Support accurate diagnosis, reporting, and treatment**

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- **FRICTION/CTI**
- **MASD/CTI**

Differential Recognition - Diagnosis

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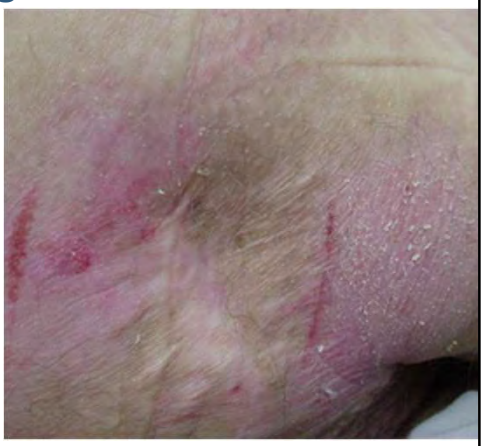
FRICTION INJURY VS CTI

DIFFERENTIAL RECOGNITION / DIAGNOSIS

- Superficial skin damage
- Immobility issues
- Lichenification with ridges often present
- May accompany pressure injury
- Not related to pressure
- Often identified as Stage 1 or 2 pressure injury
- Usually resolves with good repositioning techniques (no friction), and good skin care



Courtesy: Dot Weir, RN, CWON, CWS

Friction Injury Superficial Wounds

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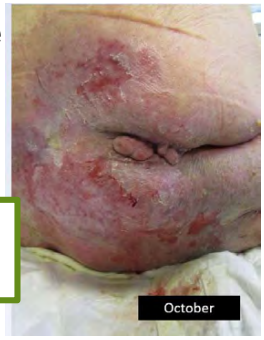
MOISTURE ASSOCIATED SKIN DAMAGE VS CTI
DIFFERENTIAL RECOGNITION / DIAGNOSIS

- Presents with **inflammation**, erosion; **not** purple-maroon discoloration
- Associated with moisture damage
  - Incontinence
  - Perspiration
  - Wound exudate
  - Ostomy effluent
- May have denuding/erosion from friction in combination with moisture
- In addition to moisture, MASD may be associated with immobility



CTI with erosion

MASD + Erosion
Document Denuding

Document: skin erosion with denuding present and dermal tissue exposed

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CHRONIC TISSUE INJURY VS PRESSURE INJURIES

Pressure injuries occur as a result of intense and/or prolonged pressure or pressure in combination with shear.

These forces create an ischemia/reperfusion injury that may or may not result in an open wound – NPIAP 2019

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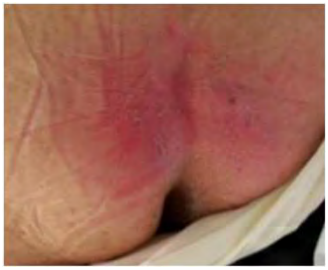
STAGE 1 PRESSURE INJURY VS CTI
DIFFERENTIAL RECOGNITION / DIAGNOSIS

- Nonblanchable erythema
- Skin intact
- NO maroon discoloration
- Associated with immobility
- Usually on or near boney prominence



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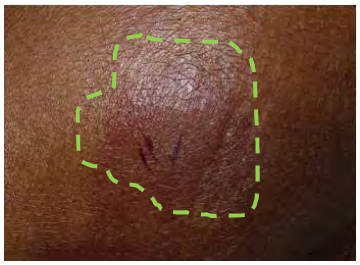
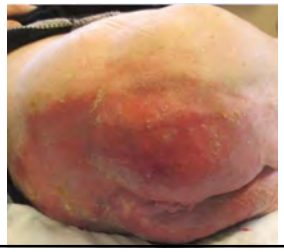
Stage 1 PU/PI Light SOC



Stage 1 PU/PI Dark SOC



Stage 1 or CTI?

SOC=Skin of Color

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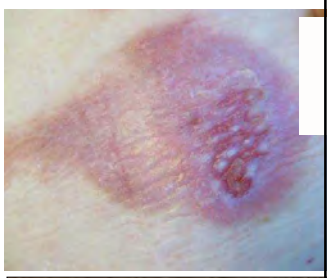
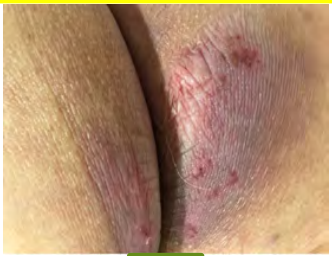
STAGE 2 PRESSURE INJURY VS CTI
DIFFERENTIAL RECOGNITION / DIAGNOSIS

- Partial-thickness skin loss
- Shallow open ulcer/pink red wound bed (i.e., dermal tissue exposed)
- Without slough
- Without bruising (i.e., No purple-maroon discoloration
- Usually over boney prominences
- Mobility issues
- May be confusion between CTI skin changes and stage 2 PU/PI



Stage 2 PIs



CTI

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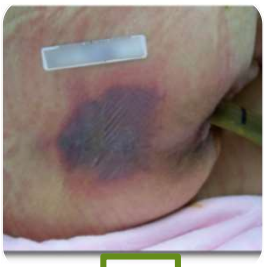
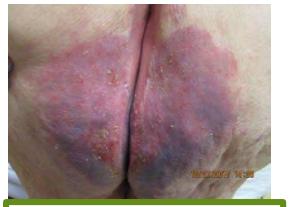
DTPI VS CTI
DIFFERENTIAL RECOGNITION / DIAGNOSIS

- DTPIs characterized by purple discoloration of affected skin
- Discoloration of DTPI appears as deeper violet hue, which may reflect the intense tissue load believed to cause DTPIs
- DTPI follows a variable course of deterioration or healing
- Severity of CTI persists over time; often months
- DTPI often associated with pain not seen in patients with CTI



DTPI
Deep
Bone/Muscle Interface



CTI
Superficial

25

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MULTIPLE ORGAN DYSFUNCTION SYNDROME (MODS)
AND SKIN FAILURE

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FAILING ORGAN SYSTEMS

- Skin failure and non-healing wounds often evidence of failing organ systems
- Have you documented **EVERY failing system** in the body?
- Documentation of other systems failing will support the facility:
  - With surveyors when using unavoidable PU/PI designation
  - With attorneys when there's a lawsuit claiming negligence

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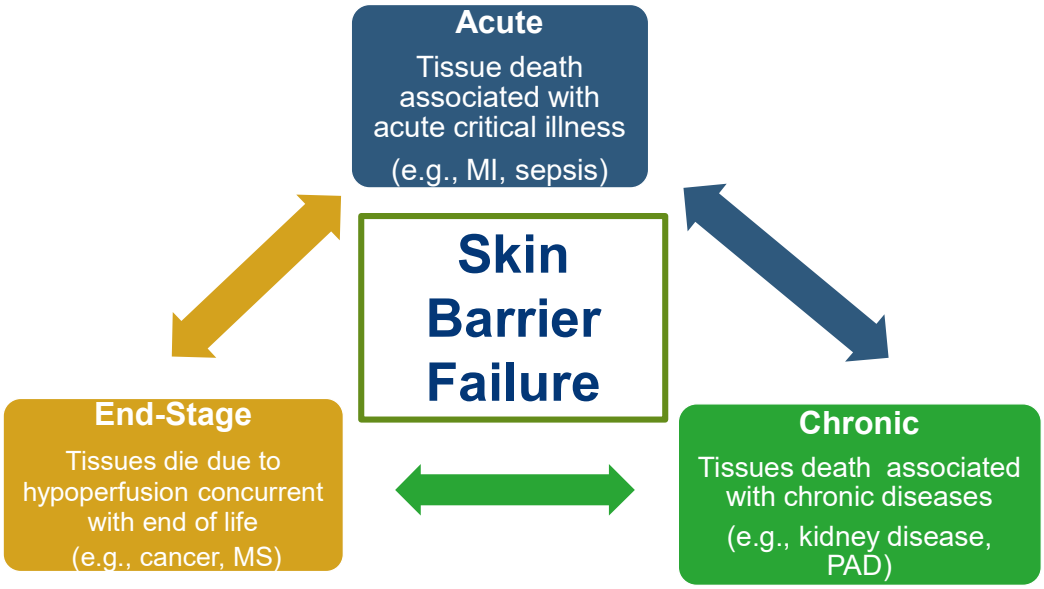
ORGAN FAILURE STRATIFICATION



Langermo DK, Brown G; Skin Fails Too: Acute, Chronic and End-Stage Skin Failure. Adv in Skin & Wound Care, Vol 19, No 4, 206-211, May 2006

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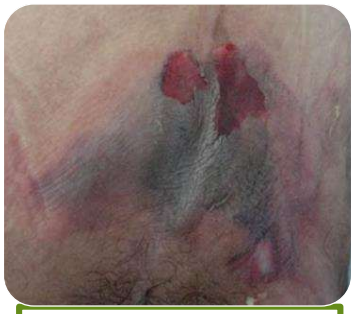
KTU/SKIN FAILURE
DIFFERENTIAL RECOGNITION / DIAGNOSIS

- Associated with organ failure
- Person in the dying process
- Usually pass within 6-weeks
- Wound usually demarcates in a short period of time, usually days
- Superficial or deep wounds
- Whereas CTI may be present for months



KTU/Skin Failure

May be Superficial
May be Deep

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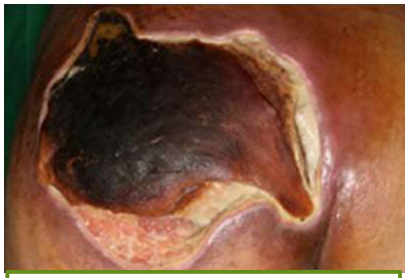
ACUTE SKIN FAILURE (ASF) – SEEN MOST OFTEN IN ACUTE CARE/ICU

- Etiology = hypoperfusion, associated with acute illness
- Hemodynamic instability associated with major organ system dysfunction/failure
- Skin requires 25-33% of cardiac output
- Predictors for ASF:
  - Respiratory failure
  - Liver failure
  - Severe sepsis/septic shock
  - Mechanical ventilation greater than 72 hours
  - Renal failure
  - Low albumin in combination with renal failure, plus respiratory failure or failure of more than one organ system (not including skin)
- May be called KTU, skin failure or Skin Changes at Life's End



Acute Skin Failure in ICU patient

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HINTS FOR DIFFERENTIAL DIAGNOSIS / RECOGNITION BETWEEN KTU/SKIN FAILURE AND OTHER SIMILAR ETIOLOGIES

- Timing, timing, timing – how long between appearance of skin manifestation and death
  - Can be hours, days or weeks
  - Usually, not months
- Location – lesions appearing on non-boney prominences **not associated with pressure forces** are probably NOT related to pressure
- Patient/residents in **physical decline with multi-organ involvement** more likely to acquire unavoidable skin failure aka Kennedy Terminal Ulcers

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SKIN FAILURE/KTU DOCUMENTATION TIPS

- SCALE document and NPIAP position statements
- **Two conditions necessary for establishing the diagnosis of skin failure are skin hypoperfusion and severe organ dysfunction or failure**
- ICD-10 diagnosis of skin failure: L98.9 Disorders of the skin
- When it appears skin failure/KTU is involved in failing skin integrity provider should collaborate with facility staff a.s.a.p.
- Provider needs to document ICD-10 codes that **corroborate organ failure to skin failure**.


SCALE
SKIN CHANGES AT LIFE'S END
Final Consensus Statement
Abstract

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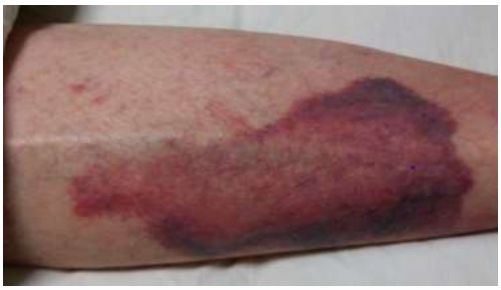
TROMBLEY-BRENNAN TERMINAL TISSUE INJURY (TB-TTI)

- Purple maroon discoloration
- **Patient will exhibit these skin changes on bony and non-bony prominence**
- Do not evolve into full thickness wounds with non-viable tissue
- Increase in surface area
- No drainage present
- Linear and mirror images may appear on lower extremities
- No complaints of discomfort
- Do not follow the same course as the KTU



DX: metastatic breast cancer
Large purpuric macular lesion on right leg
Appeared 8 days before death

Alvarez O, Brindle T, Langemo, D, Kennedy-Evans KL, Krasner Diane, Brennan M, Levine J. (2016). The VCU Pressure Ulcer Summit: The Search for a Clearer Understanding and More Precise Clinical Definition of the Unavoidable Pressure Injury. Journal of Wound, Ostomy and Continence Nursing. 43. 1. 10.1097/WON.

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TROMBLEY-BRENNAN TERMINAL TISSUE INJURY (TB-TTI)

- Spontaneously appearing skin alterations (rapid evolution, speed of enlargement and progression, appearance in areas of little to no pressure such as shins, thighs, and mirror imaging found in patients at the end of life.
- Trombley Brennan (TB-TTI) (2010)

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18 MINUTE MINI LECTURE ON MODS
EXCELLENT REVIEW FOR FACILITY CLINICAL TEAMS

Multiple Organ Dysfunction Syndrome (MODS)

- Defined as 2+ organ systems failing
  - 3+ failing = 80-90% mortality

https://www.youtube.com/watch?v=0o_jFKbEbTg

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OTHER PRESSURE INJURY IMPOSTERS

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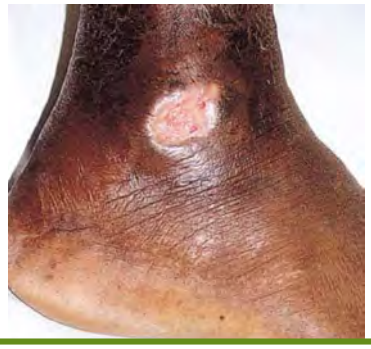
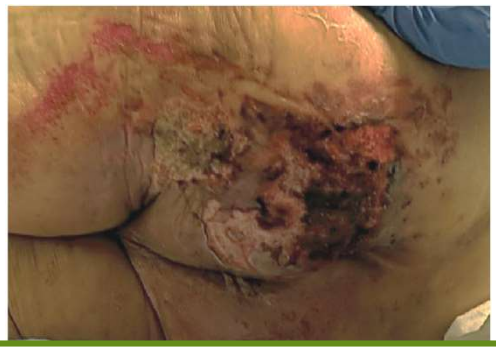
VASCULAR ULCERS MIMICKING PU/PI



Arterial ulcer on lateral malleolus of bedbound patient
Initially identified as PU/PI



- Vascular compromise of iliac arteries manifesting as full-thickness lesions on the buttock
- Initially identified a PU/PI

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Howell M, Loera S, Tickner A, Maydick D, Faust E, et al. ***Conditions That Mimic Pressure Ulcer/Injuries-To Be or Not To Be***. *Wound Management & Prevention* 2021;67(2):12–38 doi:10.25270/wmp.2021.2.1238

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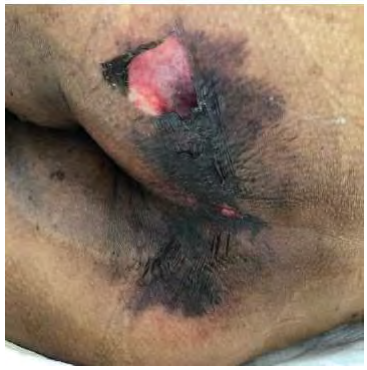
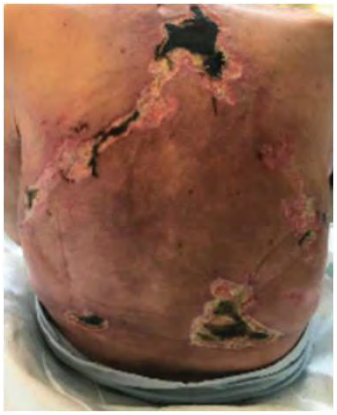
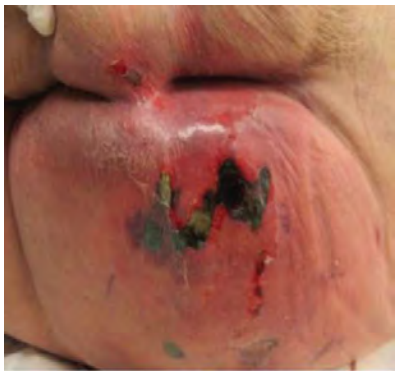
OTHER ETIOLOGIES THAT MAY MIMIC PU/PI



- COVID-19 skin manifestation
- May be mistaken for DTI or KTU



- Calciphylaxis lesions on posterior torso
- Initially identified as unstageable PU/PI



- Necrotizing soft tissue infection
- May be mistaken for DTPI or KTU

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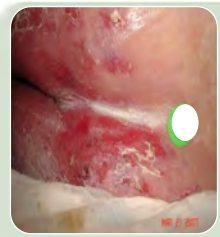
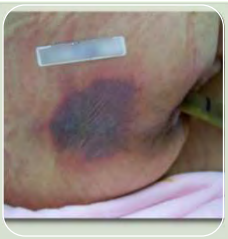
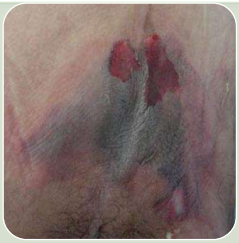
CAN YOUR TEAMS DIFFERENTIATE THESE ETIOLOGIES ON THE BUTTOCKS?



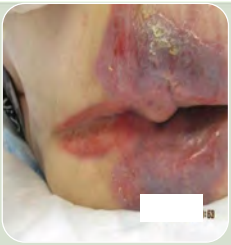
Moisture Associated Skin Damage



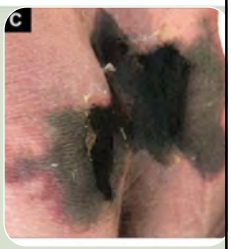
Deep Tissue Pressure Injury



Kennedy Terminal Ulcer (KTU) AKA Skin Failure



Chronic Tissue Injury



COVID-19 Skin Manifestation

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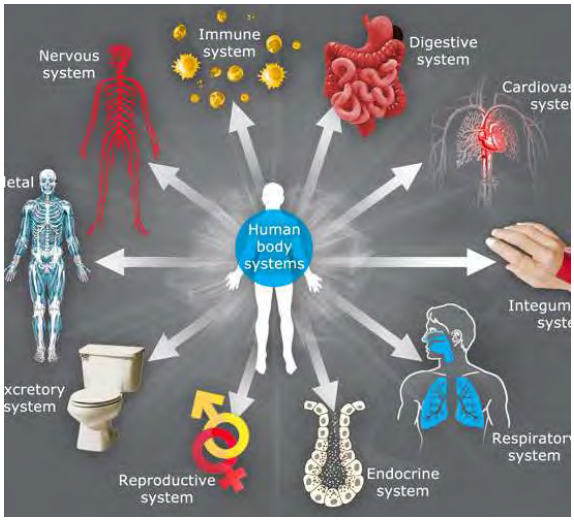
SUMMARY

- Look at **whole patient** before designating skin disruption as PU/PI
- Question PU/PI etiology when clinical picture does **not** match with a pressure etiology
- Knowledge regarding common or rare conditions that can mimic PU/PI **can reduce** inaccurate diagnosis



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SUMMARY

- Ensure accurate recognition, assessment, documentation and reporting of pressure injuries
- Complete patient/resident history review with comprehensive assessments result in :
  - Appropriate care plans
  - Improved outcomes
  - Reduced costs
  - Decreased deficiencies from surveys
  - Potentially better outcomes in lawsuits

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Pressure Injury Impostors on Buttocks

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Can your teams differentiate these etiologies?

Moisture Associated Skin Damage

Prevalent in the LTC and home health settings. Due to sweating, episodes of incontinence, or wound drainage, or effluent from ostomies that leak onto the skin. **NOT** pressure etiology.

Deep Tissue Pressure Injury

Tissue and cellular damage at the bone-muscle interface creating full-thickness wounds. This **IS** pressure etiology.

Kennedy Terminal Ulcer Skin Failure

Also known as skin failure, the KTU is recognized by CMS as a form of unavoidable pressure ulcer/injury that happens when a person is experiencing organ failure and the skin, the largest organ, also begins to fail. **NOT** pressure etiology.

Chronic Tissue Injury

Believed to result from venous pooling/ engorgement. Occurs on the fleshy portion of the buttocks. Patients and residents may be somewhat mobile. May be present for months. **NOT** pressure etiology.

COVID-19 Skin Manifestations

Associated with the disease COVID-19. May occur before, during, or after the disease or may be the only symptom of COVID-19. **NOT** pressure etiology.

Why is Accurate Recognition, Diagnosis and Reporting Important?

Pressure injuries and the associated stages and wound characteristics have clinical, regulatory (survey), costs, reimbursement, and legal implications.

Misidentification or misdiagnosis can lead to inappropriate documentation, mistakes in reporting to CMS, ineffective treatments and inappropriate use of resources.

Do NOT default to a pressure ulcer/ injury diagnosis, unless all the clinical criteria point to the PU/PI etiology.

References

- WOUNDVISION.com/hplap-Imposter
- State Operations Manual. Appendix PP-Guidance to Surveyors for Long Term Care Facilities. 173, Rev. 11-22-17.
- https://link.ztcentral.com/a/c7bbfdc3/4185PJBz7BGw_fU8h3soMg?u=http%3A%2F%2Fwww.kennedyterminalulcer.com%2F%23G1.
- Mahoney MF, Rozenboom BJ. Definition and Characteristics of Chronic Tissue Injury. J Wound Ostomy Continence Nurs. 2019;46(3):187-191.
- Howell M, Loera S, Tickner A, Maydick D, Faust E, et al. Conditions That Mimic Pressure Ulcer/Injuries-To Be or Not To Be. Wound Management & Prevention 2021;67(2):12-38 doi:10.25270/wmp.2021.2.1238.

R+01-2022

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NPIAP DOCUMENT ON DTPI AND IMPOSTERS PROVIDED AS A HANDOUT

Some of these Pressure Injury Imposters may also look like an unavoidable pressure injury.

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REFERENCES

- Mahoney MF, Rozenboom BJ. ***Definition and Characteristics of Chronic Tissue Injury***. J Wound Ostomy Continence Nurs. 2019;46(3):187-191.
- Howell M, Loera S, Tickner A, Maydick D, Faust E, et al. ***Conditions That Mimic Pressure Ulcer/Injuries-To Be or Not To Be***. *Wound Management & Prevention* 2021;67(2):12–38 doi:10.25270/wmp.2021.2.1238
- Kim JH, Shin HK, Jung GY, Lee DL. ***A case of acute skin failure misdiagnosed as a pressure ulcer, leading to a legal dispute.*** Arch Plast Surg 2019;46:75-78.
- Langermo DK, Brown G; ***Skin Fails Too: Acute, Chronic and End-Stage Skin Failure***. Adv in Skin & Wound Care, Vol 19, No 4, 206-211, May 2006
- National Pressure Ulcer Advisory Panel, European Pressure Ulcer Advisory Panel and Pan Pacific Pressure Injury Alliance. ***Prevention and Treatment of Pressure Ulcers: Clinical Practice Guideline***. Emily Haesler (Ed.). Cambridge Media: Perth, Australia; 2019.

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REFERENCES

- Alvarez O, Brindle T, Langemo, D, Kennedy-Evans KL, Krasner Diane, Brennan M, Levine J. (2016). The VCU Pressure Ulcer Summit: The Search for a Clearer Understanding and More Precise Clinical Definition of the Unavoidable Pressure Injury. Journal of Wound, Ostomy and Continence Nursing. 43. 1. 10.1097/WON.0000000000000255.
- Practice Dilemmas: Conditions that Mimic Pressure Ulcer/Injuries-To Be or Not to Be
- The Pathophysiology of Skin Failure vs. Pressure Injury: Conditions That Cause Integument Destruction and Their Associated Implications
- Differentiating PU from Acute Skin Failure
- SCALE-Final Version
- Deep Tissue Pressure Injury or an Imposter-NPIAP Document
- Pressure Injury Imposter on the Buttocks-Differential Recognition

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THE UNAVOIDABLE PRESSURE ULCER/INJURY

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RESOURCES PROVIDED

- State Operations Manual. Appendix PP-Guidance to Surveyors for Long Term Care Facilities. 173, Rev. 11-22-17
- Skin Changes at Life's End: final consensus statement: October 1, 2009.
- NPIAP: Deep Tissue Pressure Injury or an Imposter. https://cdn.ymaws.com/npiap.com/resource/resmgr/online_store/posters/Feb2021_-_NPIAP_DTPI_and_Imp.pdf. Accessed 1/11/22.
- Preventing Pressure Ulcers in Hospitals: A Toolkit for Improving Quality of Care. AHRQ
- 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.

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PURPOSE OF PROGRAM

- Review different definitions for similar conditions related to end-of-life wounds
- Describe conditions associated and situations that may lead to an unavoidable pressure injury
- Describe interventions for people with skin failure
- Review documentation related to end-of-life skin failure
- Provide literature and resources for education related to Unavoidable skin failure

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An **Avoidable** Pressure Injury is a Symptom of a **Failed System**

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Major Question:

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

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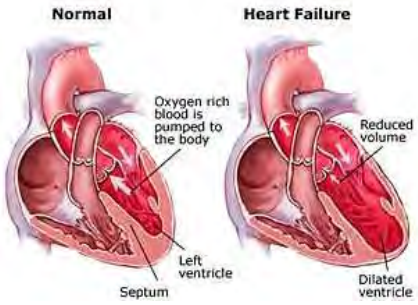
INTRODUCTION

- Skin - largest organ of the body
- Fails same as other organs: e.g. heart, kidneys, liver, etc.
- With acute and chronic illnesses body systems can fail; suddenly or slow decline
- Skin failure is an unavoidable condition
- Older adults have higher risk for skin failure due to more fragile overall organ physiology, including the skin
- When patients/residents deteriorating physically, skin failure **may NOT be preventable**



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AVOIDABILITY/UNAVOIDABILITY OF SKIN BREAKDOWN

- Terminal (end of life) ulceration is NOT new concept
- Over 100 years old and documented in historical medical literature
- Lack of complete understanding of skin failure
- Some people think, erroneously, that ALL PU/PIs are avoidable
- CMS agrees not all PU/PIs are avoidable (both F684 and F686 mention the Kennedy Terminal Ulcer (KTU)

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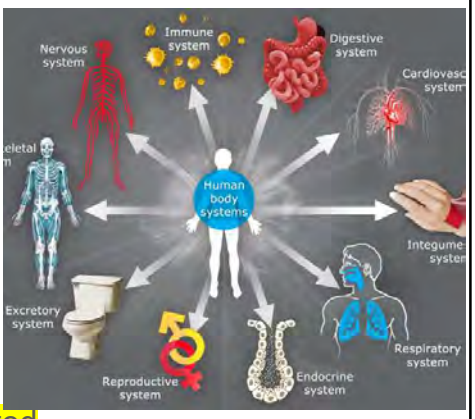
EVERYTHING IS INTERCONNECTED IN THE BODY

- Organ systems work together to maintain homeostasis with normal body functions
- Endocrine disorders such as diabetes affect glucose levels in the body
  - Altered blood glucose levels can negatively affect many organ systems
    - (e.g. elevated blood glucose impairs immune system creating increased risk of infections)
- Urinary system may experience kidney damage from elevated blood glucose or other insults
  - (e.g. kidney stones, hypertension)
- Cardiovascular system can experience damage from different diseases
  - (e.g. CAD, cardiopulmonary diseases → CHF)
- Pulmonary diseases affect tissue oxygenation
- Document ALL risk factors: Everything is interconnected



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BOTTOM LINE

- Clinicians need to better understand the pathophysiology and classification of Integument injuries **by underlying etiologies both avoidable and unavoidable**.
- More accurate diagnosis 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.

REVIEW

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

Michael Bain, MD, MMS; Junko Hara, PhD; and Marissa J. Carter, PhD, MA

ABSTRACT

KEY WORDS

INDEX

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.

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END-OF-LIFE SKIN/INTEGUMENT FAILURE

First described in medical literature in the mid-1800s

Resurfaced in modern literature in 1989

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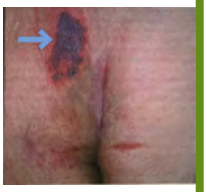
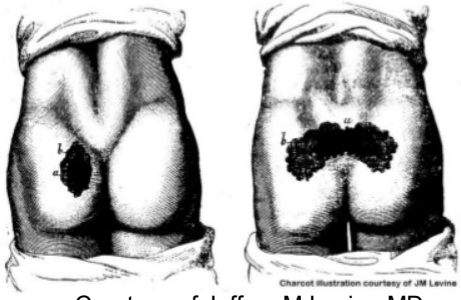
DECUBITUS OMINOSUS





Courtesy of Jeffrey M Levine MD

Jean-Martin Charcot

1825-1893

- Skin breakdown heralding impending death of patient - decubitus ominosus.
- This name was forgotten until late 20th century when Karen Kennedy recognized and published information on what became known as the Kennedy Terminal Ulcer (KTU) in 1989.

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MULTIPLE TERMS CURRENTLY USED TO DESCRIBE UNAVOIDABLE SKIN CHANGES

- Several classifications/terms for similar/overlapping clinical syndromes
  1. Kennedy Terminal Ulcer (CMS SOM-F684 and F686)
  2. Trombley-Brennan Terminal Tissue Injury
  3. Skin Changes at Life's End
  4. Skin Failure
  5. Unavoidable pressure ulcer/injuries (CMS SOM F686)
     - May be a heralding signs of organ failure where skin is failing at the same time as other body systems
     - Recognizing the resident is in organ failure is important for care planning potential end-of-life skin deterioration

SOM=State Operations Manual-Guidance for Surveyors

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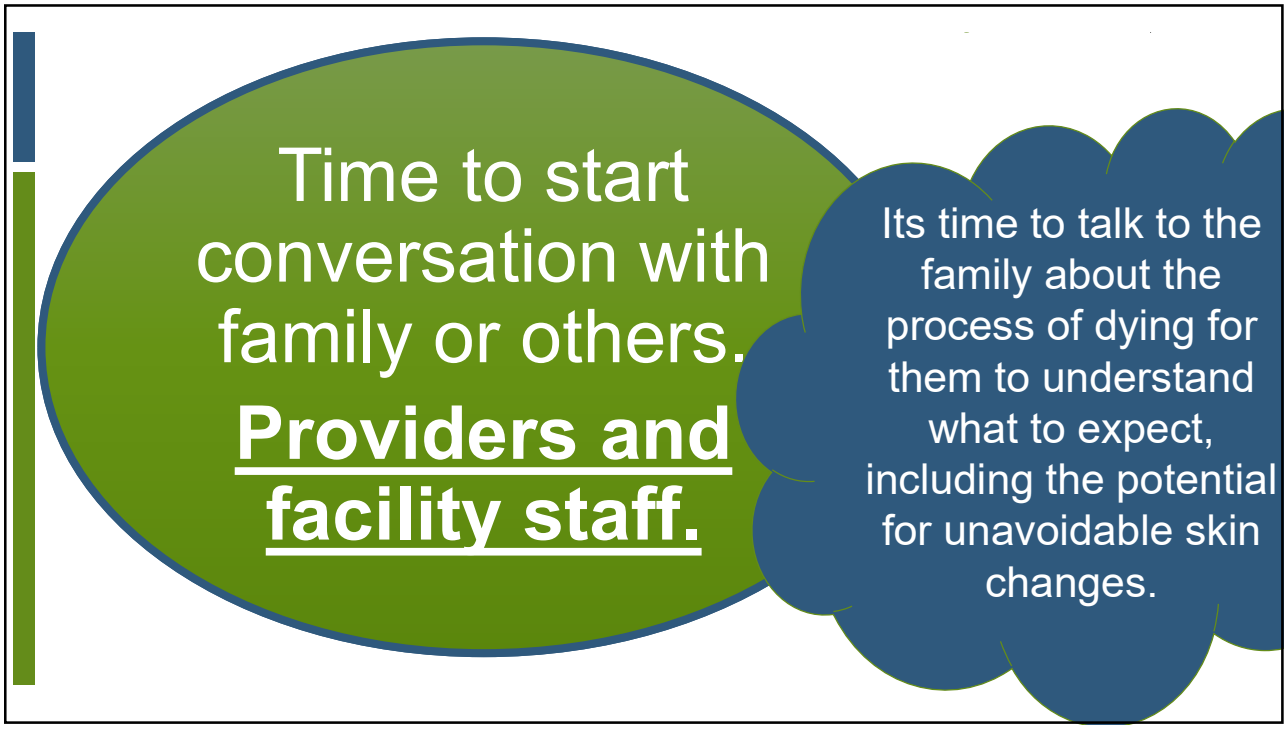

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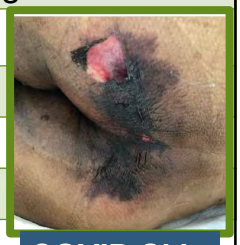
KENNEDY TERMINAL ULCER

Unavoidable skin breakdown or skin failure that occurs as part of dying process

- Not a cause of a patient's death
- Occurs in spite of good quality care
- May start out superficially as a blister or what appears to be a Stage 2
- Appears quickly and progresses rapidly to full-thickness
- May have early characteristics of a DTPI
- Many patients die within 6 weeks
- Can mimic COVID skin manifestation

KTU



COVID Skin

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CMS SOM F686

THE 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 preventive measures in place to prevent **avoidable** PU/PI

PI Prevention Plan → Prevention care plan in place **BEFORE** KTU can be called Unavoidable PI

All Risk Factors Identified? → Show KTU related to organ failure or other non-modifiable risk factors **providers involved**

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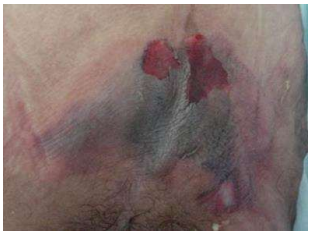
CMS SOM F686

CHARACTERISTIC OF KENNEDY TERMINAL ULCERS F686

KNOW WHEN TO USE THIS DESIGNATION!!!

- "KTUs have certain characteristics which differentiate them from pressure ulcers such as the following:
  - KTUs appear suddenly and within hours;
  - Usually appear on the sacrum and coccyx but can appear on the heels, posterior calf muscles, arms and elbows;



  - Edges are usually irregular and are red, yellow, and black as the ulcer progresses, often described as pear, butterfly or horseshoe shaped; and
  - Often appear as an abrasion, blister, or darkened area and may develop rapidly to a Stage 2, Stage 3, or Stage 4 injury."

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CMS SOM F684

F684: QUALITY OF LIFE
KENNEDY'S TERMINAL ULCER: PRESSURE ULCER

- Kennedy Terminal Ulcers are considered PRESSURE ULCER/INJURY per CMS
- Pressure ulcers that generally occur at the end of life
- For concerns related to Kennedy Terminal Ulcers, refer to F686, Pressure Ulcers
- NOTE: From Presenter…**not CMS statement**, but reality.
- Skin changes from organ failure are not pressure ulcers
- Skin failure due to dying process or during multi-organ failure.
- E.g., Resident in dying process and the skin…largest organ of the body begins to fail.
- Providers can help by **documenting medical issues contributing to skin failure**
- Can document them as unavoidable pressure ulcer/injuries
- Avoid F-tag, avoid civil money penalties, give attorneys defensible documentation should a lawsuit be brought against the facility/staff

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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."*

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PRESSURE ULCER/INJURY DEVELOPMENT

- More than **100 risk factors** cited in literature related to PU/PI development
- Affirms multifactorial etiology of PU/PI development
- Braden captures SOME (6) of these factors; not all
- Comorbidities listed as contributory include:
- Diabetes, infection, PAD, cardiovascular disease, anemia, hypotension, advancing age, vasopressor medications, and many more…
- The research, literature, and experience of clinicians over the decades agree that ALL pressure ulcer/injuries are NOT preventable

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CMS -SOM F686

UNAVOIDABLE PRESSURE ULCER IN STATE OPERATIONS MANUAL GUIDANCE TO SURVEYORS

- *F686*
- *Based on the comprehensive assessment of a resident, the facility must ensure that—*
- *(i) A resident receives care, consistent with professional standards of practice, to prevent pressure ulcers and does not develop pressure ulcers unless the individual's clinical condition demonstrates that they were unavoidable; and*
- *(ii) A resident with pressure ulcers receives necessary treatment and services, consistent with professional standards of practice, to promote healing, prevent infection and prevent new ulcers from developing.*

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CMS SOM F686

AVOIDABLE PRESSURE ULCER/INJURY PER CMS

- ***"Avoidable"*** *means that the resident developed a pressure ulcer/injury and that the facility did not do one or more of the following:*
  - *evaluate the resident's clinical condition and risk factors;*
  - *define and implement interventions that are consistent with resident needs, resident goals, and professional standards of practice;*
  - *monitor and evaluate the impact of the interventions; or revise the interventions as appropriate.*
  - Example of true unavoidable Pressure Ulcer/Injury

66

EXAMPLE OF INCREASED RISK FOR 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 ulcer 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

R-sided heart failure
Pulmonary edema/congestion

Preventing Pressure Ulcers in Hospitals: A Toolkit for Improving Quality of Care. AHRQ

67

CMS SOM F686

UNAVOIDABLE PRESSURE ULCER/INJURY PER CMS

- *"**Unavoidable**" means that the resident developed a pressure ulcer/injury even though the facility had:*
  - *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.*
  - *NOTE: Facility documentation MUST show the assessment and prevention strategies in place **before** the unavoidable PI develops.*

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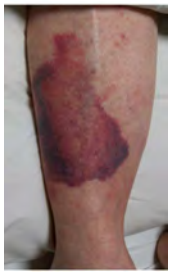
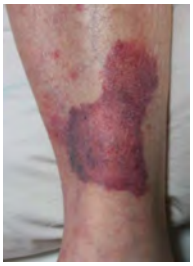
TROMBLEY-BRENNAN TERMINAL TISSUE INJURY (TB-TTI)

- Spontaneously appearing skin alterations (rapid evolution, speed of enlargement and progression, appearance in areas of little to no pressure such as shins, thighs, and **mirror imaging** found in patients at the end of life.
  Trombley & Brennan (TB-TTI) (2010)
- NOTE: not the same issue as senile purpura



Right Leg



Left Leg

Attribution: Photos from presentation- End of Life Care: Current Knowledge and Future Research
Mary R Brennan, RN, MBA, WOCN

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DIFFERENTIATING TB-TTI AND SENILE PURPURA

TB-TTI

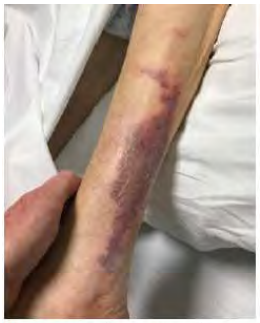
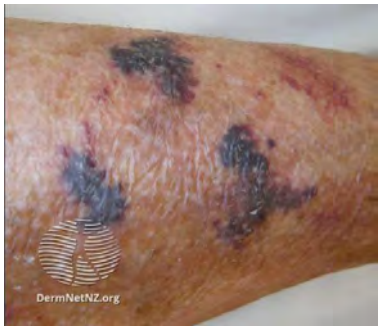


Figure 6. Trombley-Brennan terminal tissue injury. Photograph courtesy of Mary R Brennan, RN, MBA, CWON.

- Directly associate with dying process

Senile Purpura



- Associate with connective tissue damage/atrophy
- Caused by chronic sun exposure, aging and drugs

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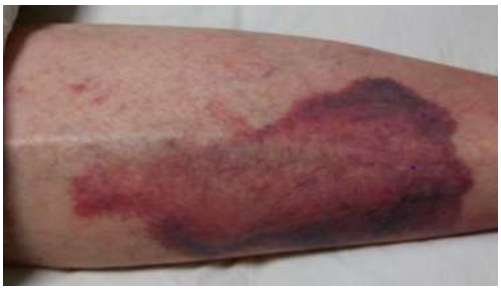
TROMBLEY-BRENNAN TERMINAL TISSUE INJURY CHARACTERISTICS (TB-TTI)

- Purple maroon discoloration – may appear at end of life
- **Patient will exhibit these skin changes on bony and non-bony prominence**
- Do not evolve into full thickness wounds with non-viable tissue
- Increase in surface area
- No drainage present
- Linear and mirror images may appear on lower extremities
- No complaints of discomfort
- Do not follow the same course as the KTU

71



DX: metastatic breast cancer
Large purpuric macular lesion on right leg
Appeared 8 days before death

Alvarez O, Brindle T, Langemo, D, Kennedy-Evans KL, Krasner Diane, Brennan M, Levine J. (2016). The VCU Pressure Ulcer Summit: The Search for a Clearer Understanding and More Precise Clinical Definition of the Unavoidable Pressure Injury. Journal of Wound, Ostomy and Continence Nursing. 43. 1. 10.1097/WON.

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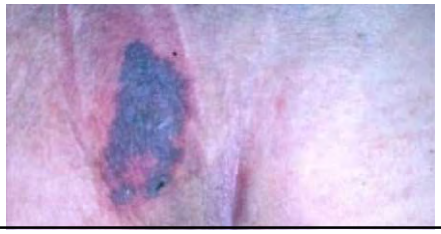
SKIN FAILURE DEFINITION

- *"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**" (Langemo, 2005, Langemo & Brown, 2006)*
- *"Skin Failure and pressure ulcers are 2 distinct, yet related clinical phenomena" (Delmore, Cox, Rolnitzky et al, 2015)*



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SKIN FAILURE - WORKING DEFINITION DR. JEFF LEVINE

- Skin failure is the state in which tissue tolerance is so compromised that cells can no longer survive in zones of physiological impairment that includes hypoxia, local mechanical stresses, impaired delivery of nutrients, and buildup of toxic metabolic byproducts.
- This includes pressure injuries, wounds that occur at life's end, and in the setting of multi-system organ failure.

Levine JM. Skin Failure: an emerging concept. J Am Med Dir Assoc 2016;17:666-9.

73

PHYSICAL MANIFESTATIONS OF SKIN FAILURE

- Hemodynamic changes
  - **Hypoperfusion of skin** – shunting of blood to vital organs to preserve life
  - Decreased blood flow to skin organ
- Impaired t**hermoregulatory** control
  - From aging and comorbidities
- Metabolic abnormalities of **toxic metabolites from catabolism**

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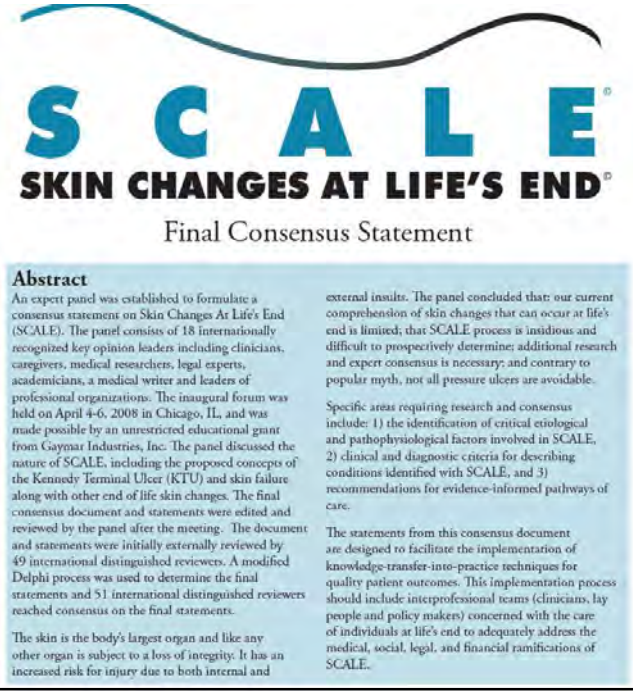

SCALE®

SKIN CHANGES AT LIFE'S END®

Final Consensus Statement

Abstract

An expert panel was established to formulate a consensus statement on Skin Changes At Life's End (SCALE). The panel consists of 18 internationally recognized key opinion leaders including clinicians, caregivers, medical researchers, legal experts, academicians, a medical writer and leaders of professional organizations. The inaugural forum was held on April 4-6, 2008 in Chicago, IL, and was made possible by an unrestricted educational grant from Gaymar Industries, Inc. The panel discussed the nature of SCALE, including the proposed concepts of the Kennedy Terminal Ulcer (KTU) and skin failure along with other end of life skin changes. The final consensus document and statements were edited and reviewed by the panel after the meeting. The document and statements were initially externally reviewed by 49 international distinguished reviewers. A modified Delphi process was used to determine the final statements and 51 international distinguished reviewers reached consensus on the final statements.

The skin is the body's largest organ and like any other organ is subject to a loss of integrity. It has an increased risk for injury due to both internal and external insults. The panel concluded that: our current comprehension of skin changes that can occur at life's end is limited; that SCALE process is insidious and difficult to prospectively determine; additional research and expert consensus is necessary; and contrary to popular myth, not all pressure ulcers are avoidable.

Specific areas requiring research and consensus include: 1) the identification of critical etiological and pathophysiological factors involved in SCALE, 2) clinical and diagnostic criteria for describing conditions identified with SCALE, and 3) recommendations for evidence-informed pathways of care.

The statements from this consensus document are designed to facilitate the implementation of knowledge-transfer-into-practice techniques for quality patient outcomes. This implementation process should include interprofessional teams (clinicians, lay people and policy makers) concerned with the care of individuals at life's end to adequately address the medical, social, legal, and financial ramifications of SCALE.

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SCALE

SKIN CHANGES AT LIFE'S END

- ***Physiologic changes that occur as a result of the dying process (days to weeks) may affect the skin and soft tissues and may manifest as observable (objective) changes in skin color, turgor, or integrity, or as subjective symptoms such as localized pain.***
- ***These changes can be unavoidable and may occur with the application of appropriate interventions that meet or exceed the standard of care.***

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SCALE

SKIN CHANGES AT LIFE'S END

- Skin changes at life's end are a reflection of compromised skin:
- Reduced soft tissue perfusion,
- Decreased tolerance to external insults,
- Impaired removal of metabolic wastes.

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SKIN FAILURE

- Based on the SCALE document (2008) and NPUAP position statements (2011, 2014), two conditions necessary for establishing the diagnosis of skin failure are skin hypoperfusion and severe organ dysfunction or failure (White-Chu & Langemo, 2012)
- ICD-10 diagnosis of skin failure: L98.9 Disorders of the skin
- When it appears skin failure/KTU involved in failing skin integrity have provider collaboration a.s.a.p.

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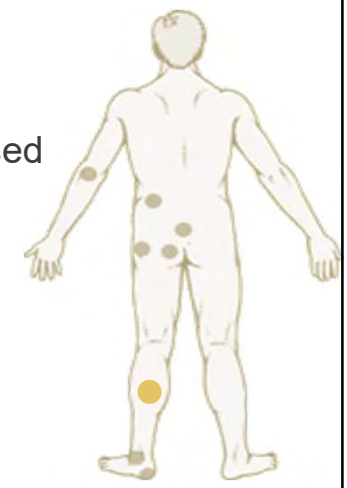
END-STAGE ORGAN DECOMPENSATION AND FAILURE

- Large and unusual presentations of skin failure
- Body shunts blood to vital organs
- Widespread and deep tissue destruction over stressed areas can appear in a matter of hours or less
  - Sacrum
  - Heels
  - Posterior calf muscles
  - Arms
  - Elbows



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SKIN FAILURE IN INDIVIDUALS WITH ADVANCED OR TERMINAL DISEASES

- These patient/residents at significant risk for KTU/Skin Failure
- Full-thickness (appearance of Stage 3 and 4 pressure injuries common; but in reality are KTUs/Skin Failure)
- Majority of skin failure in hospice occur ~2 weeks before death
- Correlates with physiological shut down of body systems 10-14 days before death

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DIFFICULT TO TELL THE DIFFERENCE BETWEEN PU/PI AND SKIN FAILURE

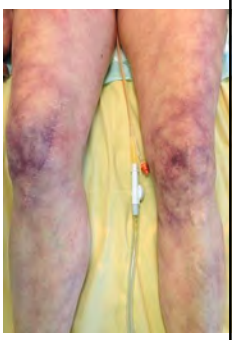
Pressure Ulcer/Injury

- Necrosis
- Ulceration
- Blistering
- Usually over bony prominences
- Over 100 risk factors for PIs
- Immobility, decreased nutrients/fluids, decreased oxygenation, etc.

Skin Failure

- Necrosis
- Ulceration
- Blistering
- Mottling-shunting blood from skin
- Gangrene
- Anywhere on the body
- In association with organ failure

Signs of Organ Failure



Skin Mottling. Pt. in respiratory failure and hypotension

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CMS SOM F686

PRESSURE ULCER/INJURIES AT END OF LIFE F686 GUIDANCE TO SURVEYORS

- *"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 indicated **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."***

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GOALS FOR TREATMENT OF KTU/SKIN FAILURE WOUNDS AT END-OF-LIFE

- Prevent wound deterioration as much as possible using current evidence-based wound care practices
- Conservative interventions often more appropriate (e.g. collagenase/Santyl for debridement instead of sharp/surgical)
- Pain assessment and management – do NOT undertreat pain unless requested by resident
- Odor management
- Infection prevention/management
- Maximize ADLs to resident's tolerance and wishes
- POC should enhance QoL even though the wound may not improve or heal

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PREVENTION OF AVOIDABLE PU/PI

- Comprehensive assessment for PU/PI risk factors – beyond Braden - **Care planning for every risk factor that is modifiable**
- Documentation of non-modifiable risk factors **by providers**
- Specialty mattresses/bed surfaces for bedbound residents at high risk for PU/PI due to documented risk factors
- Heel protectors for all bedbound residents…heel-lift devices that distribute pressure over the entire calf are recommended over pillows

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PREVENTION OF AVOIDABLE PU/PI

- Nutritional supplements
- Adherence to prevention protocols by facility staff
- Timely/complete skin and risk assessments with change of condition
- Education for residents and family, where applicable
- **If all of these steps are implemented, the resident/patient may develop an unavoidable pressure injury, BUT the facility will be in compliance with the regulations**

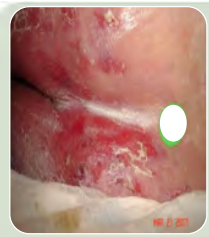
85

DOCUMENTATION: WHAT TO INCLUDE

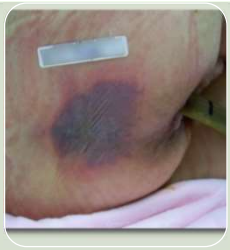
- Accurate and timely documentation
- Adherence to facility PU/PI prevention protocols
- Consistency in care with supporting documentation of interventions
- Documentation of skin and wound assessments
- Documentation of interdisciplinary care
- Documentation of education with residents/patients and family

86

CAN YOUR TEAMS DIFFERENTIATE THESE ETIOLOGIES ON THE BUTTOCKS?

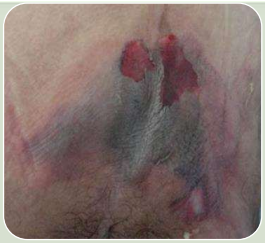


Moisture Associated Skin Damage



Deep Tissue Pressure Injury

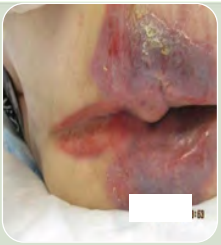
Pressure Forces



Kennedy Terminal Ulcer

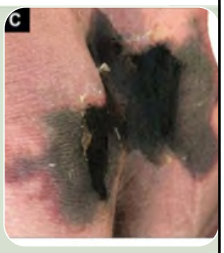
Skin Failure

CMS Unavoidable PU/PI



Chronic Tissue Injury

Not Pressure Related



COVID-19 Skin Manifestation

Coagulopathy (Clots) Damage

87

NPIAP DOCUMENT ON DTPI AND IMPOSTERS PROVIDED AS A HANDOUT

Some of these Pressure Injury Imposters may also look like an unavoidable pressure injury.

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SUMMARY

- Skin failure - a subset of multiple organ dysfunction syndrome (MODS) (Bone et al, 1992)
- These skin disruptions are NOT pressure ulcers (Langemo & Brown, 2006, White-Chu & Langemo, 2012, Delmore et al. 2015)
- Skin failure and PU/PI are 2 distinct phenomena, yet interrelated & may occur simultaneously *(Delmore, Cox, Rolnitzky et al, 2015)*
- Skin Failure occurs without the presence of pressure and/or shear. (White-Chu & Langemo, 2012)
- PU/PI can occur in people not chronically ill or at life's end (e.g. paraplegics /quadriplegics)
- Skin failure can occur acutely, in chronically ill residents, or at life's end (Langemo 2006)
- Respiratory failure significantly associated with skin failure (Curry et al, 2012, Levine et al, 2009)
- Curry et al also found 2 or more failed organ systems resulted in skin failure

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SUMMARY

- We need good quality studies that confirm the physiological skin changes, what we see on the skin AND what is under the skin, that validates skin failure resulting from MOF as a different mechanism of injury from pressure forces.
- In the meantime…document ALL of the patients' and residents' multiple organ pathology that may lead to skin failure/KTU aka unavoidable PU/PI
- Care plan for each identified risk factor for PU/PI
- Use the Unavoidable PI/PU designation provided by CMS to soften the survey issues when you cannot stop the overall failure of the skin that fails in tandem with the other organs at life's end.

MOF=Multi-Organ Failure

90

An **Avoidable** Pressure Injury is a Symptom of a **Failed System**

91

Unavoidable skin changes are evidence of the failing organs

92

THANK YOU!!!

93

REFERENCES/RESOURCES

- State Operations Manual. Appendix PP-Guidance to Surveyors for Long Term Care Facilities. 173, Rev. 11-22-17
- Langemo, D. K., & Brown, G. Skin fails too: Acute, chronic, and end stage skin failure. Adv Skin Wound Care.2006:19(4):206–211.
- Ayello, EA, Sibbald GA. Report on NPUAP Session: Untangling the Terminology of Unavoidable Pressure Injuries, Terminal Ulcers and Skin Failure. Adv in Skin and Wound Care, Vol 30 No. 5 198, 2017.
- Kennedy KL. The prevalence of pressure ulcers in an intermediate care facility. Decubitus 1989;2(2):44-7.
- http://www.kennedyterminalulcer.com/#Q1. Accessed 1/21/22
- Sibbald RG, Krasner DL, Lutz J. SCALE: Skin Changes at Life's End: final consensus statement: October 1, 2009. Adv Skin Wound Care 2010;23:225-36.
- Levine JM. Unavoidable Pressure Injuries, Terminal Ulceration, and Skin Failure: In Search of a Unifying Classification System. *Adv Skin Wound Care*. 2017;30(5):200-202. doi:10.1097/01.ASW.0000515077.61418.44

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REFERENCE/RESOURCES

- Levine, JM, Delmore B, Cox J. Skin Failure, Advances in Skin & Wound Care: November 1, 2021 - Volume - Issue -doi: 10.1097/01.ASW.0000798464.03618.d9
- Brennan MR, Trombley K. Kennedy Terminal Ulcers. Va palliative care unit's experience over a 12-month period of time. World Council Enterostomal Ther J 2010;30(3):20-2.
- Trombley K, Brennan MR. Prelude to Death or Practice Failure? Trombley-Brennen Terminal Tissue Injuries. Am J Hospice & Palliative Medicine, 2012, 29 (7), 541-545.
- Brennan MR, Thomas L, Kline M. Prelude to Death or Practice Failure? Trombley-Brennan Terminal Tissue Injury Update. *Am J Hosp Palliat Care*. 2019;36(11):1016-1019. doi:10.1177/1049909119838969
- Levine JM. Skin Failure: an emerging concept. J Am Med Dir Assoc 2016;17:666-9.
- NPIAP: Deep Tissue Pressure Injury or an Imposter. https://cdn.ymaws.com/npiap.com/resource/resmgr/online_store/posters/Feb2021_-_NPIAP_DTPI_and_Imp.pdf. Accessed 1/11/22.
- Schank J. Making a case for retaining Kennedy terminal ulcer and other end-of-life ulcer terminology: a review of the literature. *Wounds*. 2021;33(12)309-320. doi:10.25270/wnds/2021309320
- Preventing Pressure Ulcers in Hospitals: A Toolkit for Improving Quality of Care. AHRQ

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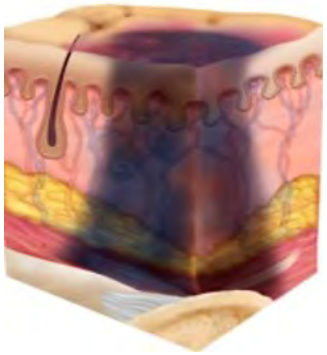
NATIONAL PRESSURE INJURY ADVISORY PANEL
Improving Patient Outcomes Through Education, Research and Public Policy

WOUNDVISION
woundvision.com/npiap-imposter

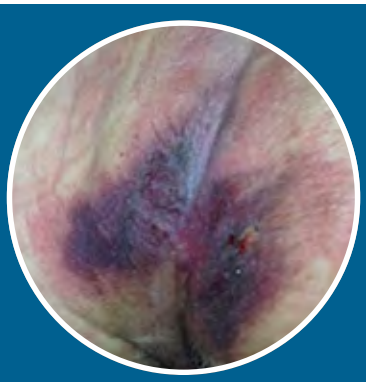
DEEP TISSUE PRESSURE INJURY OR AN IMPOSTER?

Intact or non-intact skin with localized area of persistent non-blanchable deep red, maroon, purple discoloration or epidermal separation revealing a dark wound bed or blood-filled blister. Pain and temperature change often precede skin color changes. Discoloration may appear differently in darkly pigmented skin. This injury results from intense and/or prolonged pressure and shear forces at the bone-muscle interface.

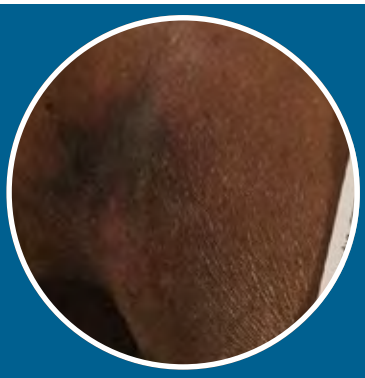
The wound may evolve rapidly to reveal the actual extent of tissue injury or may resolve without tissue loss. If necrotic tissue, subcutaneous tissue, granulation tissue, fascia, muscle or other underlying structures are visible, this indicates a full thickness pressure injury (Unstageable, Stage 3 or Stage 4).



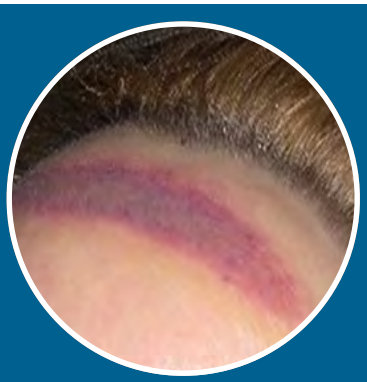
Initial DTPI
Initially intact purple or maroon skin or blood blister



Sacral DTPI after cardiac surgery in supine position 48 hours ago

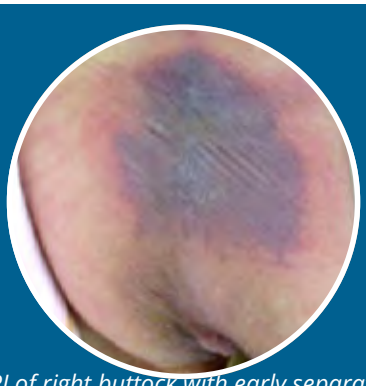


Low sacral-coccygeal DTPI in a patient sitting in High-Fowler's position

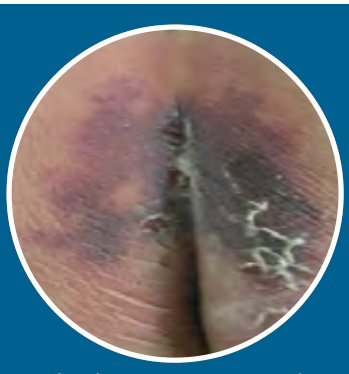


Forehead DTPI after surgery in prone position 24 hours ago

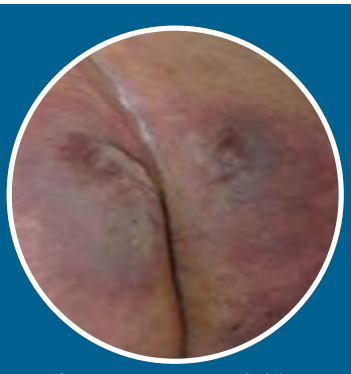
Evolving DTPI
Blistered appearance as epidermis sloughs



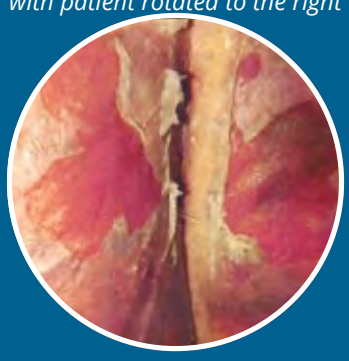
DTPI of right buttock with early separation of the dermis, 72 hours after surgery done with patient rotated to the right



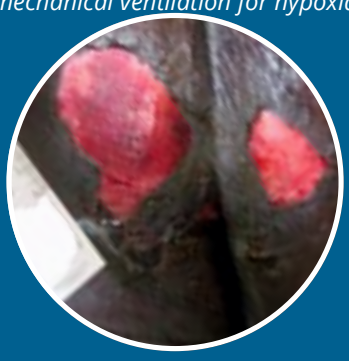
DTPI of right para-sacrum with early separation of the dermis, 72 hours after mechanical ventilation for hypoxia



DTPI of para-sacrum with blistering, 72 hours after cardiac surgery in supine position



DTPI of para-sacrum with blistering, 72 hours after cardiac surgery in supine position



DTPI of buttocks with blistering, 72 hours after mechanical ventilation for hypoxia

Blood blister - Tissue may be hard to the touch or boggy

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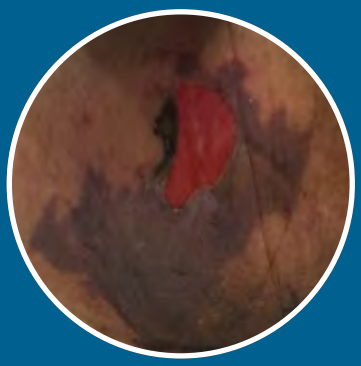
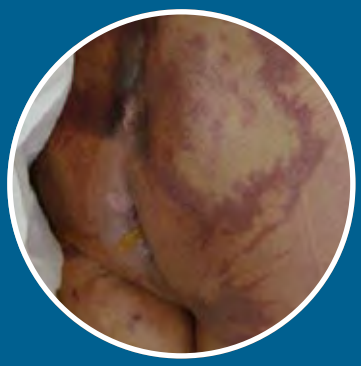
DEEP TISSUE PRESSURE INJURY OR AN IMPOSTER?

Many conditions can lead to purple or ecchymotic skin and rapidly developing eschar. Some of the most common differential diagnoses are shown below.

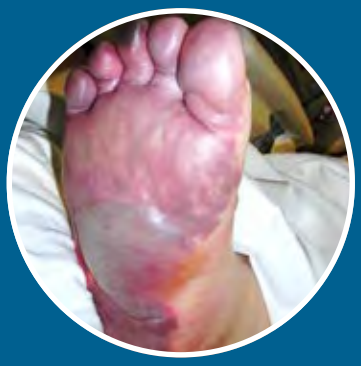
Ischemia



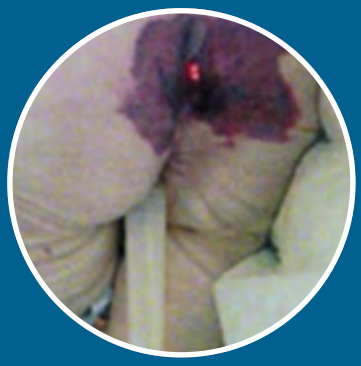
COVID-19
COVID-19 accelerates clotting in small vessels Skin color change is not always on pressure bearing tissues



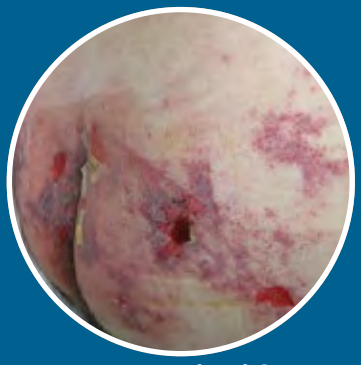
Embolic Disease
Marked disease of internal iliacs or postoperative aorto-iliac bypass with emboli



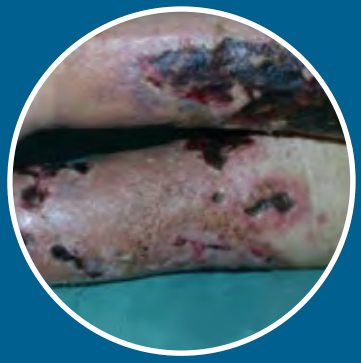
Vasopressor Induced Peripheral Ischemia
Levophed in use - Ischemia of ears, nose, fingers also common



Ischemia From Hypotension
Sudden purpura near end of life, no pressure events had occurred. Patient died 4 days later

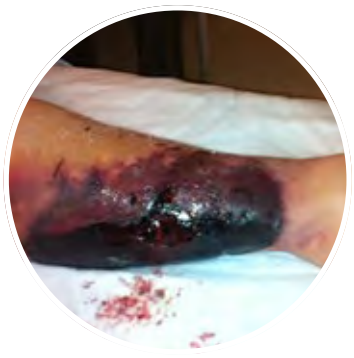


DIC/Sepsis with Microvascular Emboli
Reticular presentation Spontaneous onset, rapidly necrotic

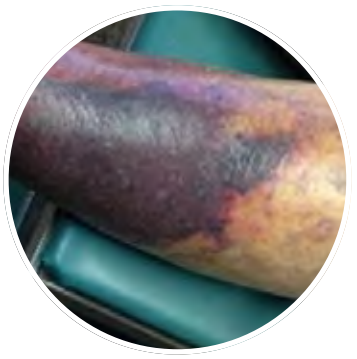


Calciphylaxis (AKA Calcific Uremic Arteriopathy)
Seen in patients in dialysis dependent renal failure due to hyper-parathyroidism, hypercalcemia and hyper-phosphatemia

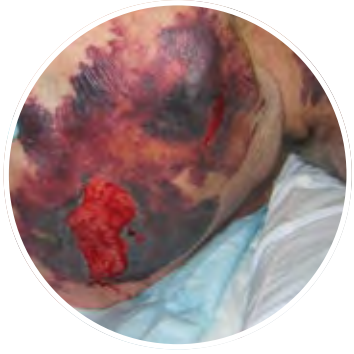
Trauma



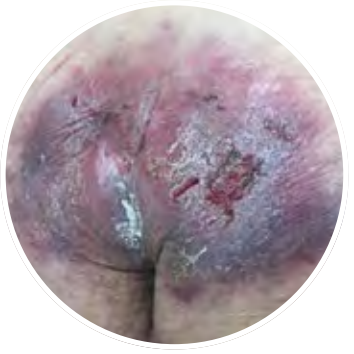
Warfarin Induced Skin Necrosis
Erythematous flushing then progressing within 24 hours to full thickness hemorrhagic bullae several days after high loading doses of Warfarin.



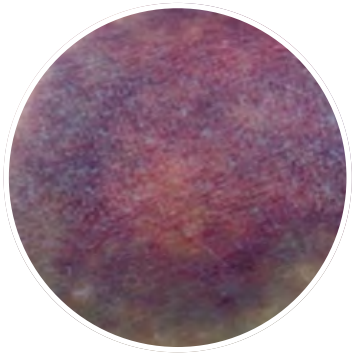
Hematoma
History of trauma to area, often anticoagulated - Area is palpable and often tender



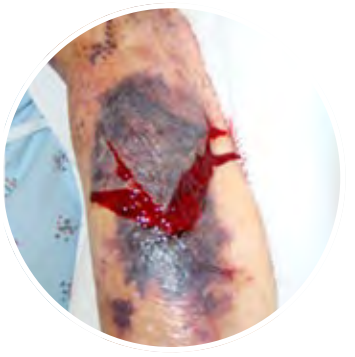
Blunt Trauma
History of traumatic injury Irregular shape Painful to touch. Morel Lavallée Lesions are possible



Chronic Friction Injury
Immobile or chairbound patient who uses a slide board Skin thick and irregular lesions



Bruise
History of trauma in the area Color changes to yellow and green in a few days



Skin Tear
Patient fell attempting to ambulate. Usually, profuse bleeding.

SCALE©
SKIN CHANGES AT LIFE'S END©

Final Consensus Statement

Abstract

An expert panel was established to formulate a consensus statement on Skin Changes At Life's End (SCALE). The panel consists of 18 internationally recognized key opinion leaders including clinicians, caregivers, medical researchers, legal experts, academicians, a medical writer and leaders of professional organizations. The inaugural forum was held on April 4-6, 2008 in Chicago, IL, and was made possible by an unrestricted educational grant from Gaymar Industries, Inc. The panel discussed the nature of SCALE, including the proposed concepts of the Kennedy Terminal Ulcer (KTU) and skin failure along with other end of life skin changes. The final consensus document and statements were edited and reviewed by the panel after the meeting. The document and statements were initially externally reviewed by 49 international distinguished reviewers. A modified Delphi process was used to determine the final statements and 51 international distinguished reviewers reached consensus on the final statements.

The skin is the body's largest organ and like any other organ is subject to a loss of integrity. It has an increased risk for injury due to both internal and external insults. The panel concluded that: our current comprehension of skin changes that can occur at life's end is limited; that SCALE process is insidious and difficult to prospectively determine; additional research and expert consensus is necessary; and contrary to popular myth, not all pressure ulcers are avoidable.

Specific areas requiring research and consensus include: 1) the identification of critical etiological and pathophysiological factors involved in SCALE, 2) clinical and diagnostic criteria for describing conditions identified with SCALE, and 3) recommendations for evidence-informed pathways of care.

The statements from this consensus document are designed to facilitate the implementation of knowledge-transfer-into-practice techniques for quality patient outcomes. This implementation process should include interprofessional teams (clinicians, lay people and policy makers) concerned with the care of individuals at life's end to adequately address the medical, social, legal, and financial ramifications of SCALE.

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The content of this document is based on the results of a two-day round table discussion held on April 4-6, 2008 in Chicago, IL, and was made possible by an unrestricted educational grant from Gaymar Industries, Inc. Additional input was received from an internationalfrom international panels of 49 and 51 distinguished reviewers using a modified Delphi Method process. The information contained herein does not necessarily represent the opinions of all panel members, distinguished reviewers, or Gaymar Industries, Inc.

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Background for Skin Changes At Life's End (SCALE)

Organ dysfunction is a familiar concept in the health sciences, and can occur at any time but most often occurs at life's end, during an acute critical illness or with severe trauma. Body organs particularly the heart and kidneys undergo progressive limitation of function as a normal process related to aging and the end of life. End of life is defined as a phase of life when a person is living with an illness that will often worsen and eventually cause death. This time period is not limited to the short period of time when the person is moribund.[1] It is well accepted that during the end stages of life, any of a number of vital body systems (e.g. the renal, hepatic, cardiac, pulmonary, or nervous systems) can be compromised to varying degrees and will eventually totally cease functioning. The process of organ compromise can have devastating effects, resulting in injury or interference with functioning of other organ systems that may contribute to further deterioration and eventual death.

We propose that the skin, the largest organ of the body, is no different, and also can become dysfunctional with varying degrees of resultant compromise. The skin is essentially a window into the health of the body, and if read correctly, can provide a great deal of insight into what is happening inside the body. Skin compromise, including changes related to decreased cutaneous perfusion and localized hypoxia (blood supply and local tissue factors) can occur at the tissue, cellular, or molecular level. The end result is a reduced availability of oxygen and the body's ability to utilize vital nutrients and other factors required to sustain normal skin function. When this compromised state occurs, the manifestations are termed, Skin Changes At Life's End (SCALE). It should be noted that the acronym SCALE is a mnemonic used to describe a group of clinical phenomena, and should not be confused with a risk assessment tool. The term applies to all individuals across the continuum of care settings.

Skin organ compromise at life's end is not a new concept in the literature. The first clinical description in modern medical literature appeared in 1989 with the Kennedy Terminal Ulcer (KTU).[2] Kennedy

The skin is essentially a window into the health of the body, and if read correctly, can provide a great deal of insight into what is happening inside the body.

described the KTU as a specific subgroup of pressure ulcers that some individuals develop as they are dying. They are usually shaped like a pear, butterfly, or horseshoe, and are located predominantly on the coccyx or sacrum (but have been reported in other anatomical areas). The ulcers are a variety of colors including red, yellow or black, are sudden in onset, typically deteriorate rapidly, and usually indicate that death is imminent.[2] This initial report was based on retrospective chart reviews of individuals with pressure ulcers. It sparked further inquiry into how long these individuals within the facility lived after occurrence of a pressure ulcer. Just over half (55.7%) died within six weeks of discovery of their pressure ulcer(s). The observations were further supported by Hanson and colleagues (1991), who reported that 62.5% of pressure ulcers in hospice patients occurred in the 2 weeks prior to death.[3] Further evidence for the existence of the KTU is mostly observational in nature, but is consistent with the premise that skin function can become compromised at life's end.

It is noteworthy that while Kennedy independently described the KTU in 1989, a similar condition was actually first described much earlier in the French medical literature by Jean-Martin Charcot (1825-1893).[4, 5] In a medical textbook written in 1877, Charcot described a specific type of ulcer that is butterfly in shape and occurring over the sacrum. Patients that developed these ulcers usually died shortly thereafter, hence he termed the ulcer Decubitus Ominosus. However, Charcot attributed the ulcers to being neuropathic rather than pressure in origin. Charcot's writings of Decubitus Ominosus

were all but forgotten in the medical literature until recently with renewed interest in skin organ compromise.[4] The fact that two experts in the field of chronic wounds independently reported the same clinical phenomenon, with very similar descriptions, 112 years apart, lends credence to the possible existence of terminal pressure ulcers as a result of end-of-life skin organ compromise.

Also of historical interest is the original work of Dr. Alois Alzheimer in Germany. He was on call in 1901 when a 51 year old woman, Frau August D, was admitted to his asylum for the insane in Frankfort. Dr. Alzheimer followed this patient, studied her symptoms and presented her case to his colleagues as what came to be known as Alzheimer's Disease. When Frau Auguste D. died on April 8, 1906, her medical record listed the cause of death as "septicemia due to decubitus."[6] Alzheimer noted, "at the end, she was confined to bed in a fetal position, was incontinent and in spite of all the care and attention given to her, she suffered from decubitus." So, here we have the first identified patient with Alzheimer's Disease having developed immobility and two pressure ulcers with end stage Alzheimer's. In our modern times, end stage Alzheimer's Disease has become an all-too-frequent scenario with multiple complications including SCALE.

In 2003, Langemo proposed a working definition of skin failure; that it is a result of hypoperfusion, creating an extreme inflammatory reaction concomitant with severe dysfunction or failure of multiple organ systems.[7] Three years later, Langemo and Brown (2006) conducted a comprehensive review of the literature on the concept of skin failure that focused largely on pressure ulcer development.[8] They presented a discussion of changes in the skin that can occur with aging, the development of pressure ulcers, multiorgan failure, and "skin failure" (both acute and chronic as well as end of life).[9-15] In the early 1990's two publications by Parish & Witkowski had presented logical arguments about the mechanism of pressure ulcer occurrence at the end of life, suggesting that they may not be preventable in those individuals with multiple organ failure.[11, 16] Although the term skin failure has been introduced, it is not currently a widely accepted term in the dermatological or the wound literature.

Despite the limited scientific literature, there is consensus from the narrative literature that some pressure ulcers may be unavoidable including those that are manifestations of SCALE. We propose that at the end of life, failure of the homeostatic mechanisms that support the skin can occur, resulting in a diminished reserve to handle insults such as minimal pressure. Therefore, contrary to popular myth, not all pressure ulcers are avoidable.[17, 18] Many members of the SCALE Panel acknowledge the need for systematic study of the phenomenon.

Goals and Objectives of the SCALE Panel

The overall goal of the SCALE Expert Panel was to initiate stakeholder discussion of skin changes at the end of life, a phenomenon that we have termed SCALE. An objective was to examine the concept of unavoidable pressure ulcers that can occur as a result of SCALE. While reaching consensus on the various aspects of this topic is an important outcome, this endeavor will require a more rigorous scientific investigative approach that was beyond the scope of this ground breaking meeting. The purpose of this initial meeting was to generate a series of statements that will serve as a platform for future consensus discussions. The objective of this document is to present these panel statements, disseminate them for public discussion, and to further the development of the body of scientific knowledge on this important topic.

Methodology

A modified three phase Delphi Method approach was used to reach consensus on the 10 statements reported in this document. The Delphi Method relies on expert panel input to reach consensus on a topic of interest.[19] Our approach consisted of three separate phases of consensus building involving an international group of 69 noted experts in the field

of wound care.

Phase 1: A panel of 18 experts in the field of wound care with expertise in wound and skin care convened in a round table format on April 4-6, 2008 in Chicago, IL, USA. Audio proceedings and written notes from this round table discussion were used to generate a Preliminary Consensus Document (PCD). This PCD was returned to the original panel for review and was modified as necessary to reach panel consensus.

Phase 2: The PCD was presented and distributed at numerous international conferences seeking public comment from September 2008 through June 2009. The document was published,[20] and also available for public download from the web site of the panel sponsor (Gaymar Industries, Inc.).[21] The PCD was further reviewed by a selected international panel of 49 Distinguished Reviewers with noted expertise in wound care and palliative medicine.

Phase 3: Written input received from the panel of Distinguished Reviewers and from the various public presentations was used to generate A Final Consensus Document (FCD). This FCD was then returned to the original 18-member Expert Panel and a 52-member Distinguished Reviewer Panel for voting on each of the 10 statements for consensus. A quorum of 80% that *strongly agree* or *somewhat agree* with each statement was used as a pre-determined threshold for having achieved consensus on each of the statements. Fifty two individuals voted on the final consensus process.

In addition to the PCD and FCD documents, an annotated bibliography of literature pertinent to SCALE was generated and is available for download from the web site of the panel sponsor (Gaymar Industries, Inc.).[21]

Panel Statements

As a result of the two-day panel discussion and subsequent panel revisions, and with input from 69 noted wound care experts in a modified Delphi Method approach, the following 10 statements are proposed by the SCALE Expert Panel:

Statement 1

Physiologic changes that occur as a result of the dying process (days to weeks) may affect the skin and soft tissues and may manifest as observable (objective) changes in skin color, turgor, or integrity, or as subjective symptoms such as localized pain. These changes can be unavoidable and may occur with the application of appropriate interventions that meet or exceed the standard of care.

When the dying process compromises the homeostatic mechanisms of the body, a number of vital organs may become compromised. The body may react by shunting blood away from the skin to these vital organs, resulting in decreased skin and soft tissue perfusion and a reduction of the normal cutaneous metabolic processes. Minor insults can lead to major complications such as skin hemorrhage, gangrene, infection, skin tears and pressure ulcers that may be markers of SCALE. See Statement 6 for further discussion.

Statement 2

The plan of care and patient response should be clearly documented and reflected in the entire medical record. Charting by exception is an appropriate method of documentation.

The record should document the patient's clinical condition including co-morbidities, pressure ulcer risk factors, significant changes, and clinical interventions that are consistent with the patient's wishes and recognized guidelines for care.[22] Facility policies and guidelines for record keeping should be followed and facilities should update these policies and guidelines as appropriate. The impact of the interventions should be assessed and revised as appropriate. This documentation may take many forms. Specific approaches to documentation of care should be consistent with professional, legal, and regulatory guidelines, and may involve narrative documentation, the use of flow sheets, or other documentation systems/tools.

If a patient is to be treated as palliative, it should be stated in the medical record, ideally with a reference to a family/caregiver meeting, and that consensus was reached. If specific palliative scales such as the Palliative Performance Scale,[23] or other palliative tools were utilized,[24] they should be included in the medical record. Palliative care must be patient-centered, with skin and wound care being only a part of the total plan of care.

It is not reasonable to expect that the medical record will be an all-inclusive account of the individual's care. Charting by exception is an appropriate method of documentation. This form of documentation should allow the recording of unusual findings and pertinent patient risk factors. Some methods of clinical documentation are antiquated in light of today's complexity of patient care and rapidly changing interprofessional healthcare environment; many current documentation systems need to be revised and streamlined.

Statement 3

Patient centered concerns should be addressed including pain and activities of daily living.

A comprehensive, individualized plan of care should not only address the patient's skin changes and co-morbidities, but any patient concerns that impact quality of life including psychological and emotional issues. Research suggests that for wound patients, health-related quality of life is especially impacted by pain, change in body image, odors and mobility issues. It is not uncommon for these factors to have an effect on aspects of daily living, nutrition, mobility, psychological factors, sleep patterns and socialization.[25, 26] Addressing these patient-centered concerns optimizes activities of daily living and enhance a patient's dignity.

A comprehensive, individualized plan of care should not only address the patient's skin changes and co-morbidities, but any patient concerns that impact quality of life including psychological and emotional issues.

Statement 4

Skin changes at life's end are a reflection of compromised skin (reduced soft tissue perfusion, decreased tolerance to external insults, and impaired removal of metabolic wastes).

When a patient experiences SCALE, tolerance to external insults (such as pressure) decreases to such an extent that it may become clinically and logistically impossible to prevent skin breakdown and the possible invasion of the skin by microorganisms. Compromised immune response may also play an important role, especially with advanced cancer patients and with the administration of corticosteroids and other immunosuppressant agents.

Skin changes may develop at life's end despite optimal care, as it may be impossible to protect the skin from environmental insults in its compromised state. These changes are often related to other cofactors including aging, co-existing diseases and drug adverse events. SCALE, by definition occurs at life's end, but skin compromise may not be limited to end of life situations; it may also occur with acute or chronic illnesses, and in the context of multiple organ failure that is not limited to the end of life.[8, 27] However, these situations are beyond the scope of this panel's goals and objectives.

Statement 5

Expectations around the patient's end of life goals and concerns should be communicated among the members of the interprofessional team and the patient's circle of care. The discussion should include the potential for SCALE including other skin changes, skin breakdown and pressure ulcers.

It is important that the provider(s) communicate and document goals of care, interventions, and outcomes related to specific interventions (See Statement 2). The patient's circle of care includes the members of the patient unit including family, significant others, caregivers, and other healthcare professionals that may be external to the current interprofessional team. Communication with the interprofessional team and the patient's circle of care should be documented. The education plan should include realistic expectations surrounding end of life issues with input from the patient if possible. Communication of what to expect during end of life is important and this should include changes in skin integrity.

Being mindful of local protected health information disclosure regulations (e.g. USA: HIPAA, 1996),[28] the patient's circle of care needs to be aware that an individual at the end of life may develop skin breakdown, even when care is appropriate. They need to understand that skin function may be compromised to a point where there is diminished reserve to tolerate even minimal pressure or external insult. Educating the patient's circle of care up front may help reduce the chances of shock and emotional reactions if end of life skin conditions occur.

This education includes information that as one nears end of life, mobility decreases. The individual frequently has a "position of comfort" that the patient may choose to maintain, resulting in a greater potential for skin breakdown. Some patients elect to continue to lie on the pressure ulcer, stating it is the most comfortable position for them. Respecting the coherent patient's wishes is important.

With the recognition that these skin conditions are sometimes a normal part of the dying process, there is less potential for assigning blame, and a greater understanding that skin organ compromise may be an unavoidable part of the dying process.

The patient's circle of care includes the members of the patient unit including family, significant others, caregivers, and other healthcare professionals that may be external to the current interprofessional team.

Discussions regarding specific trade-offs in skin care should be documented in the medical record. For example, patients may develop pressure ulcers when they cannot be (or do not want to be) turned due to pain or the existence of other medical conditions. Pressure ulcers may also occur in states of critical hypoperfusion due to underlying physical factors such as severe anemia, hypoxia, hypotension, peripheral arterial disease, or severe malnutrition. Care decisions must be made with the total goals of the patient in mind, and may be dependent on the setting of care, trajectory of the illness, and priorities for the patient and family. Comfort may be the overriding and acceptable goal, even though it may be in conflict with best skin care practice. In summary, the patient and family should have a greater understanding that skin organ compromise may be an unavoidable part of the dying process.

Statement 6

Risk factors symptoms and signs associated with SCALE have not been fully elucidated, but may include:

- ***Weakness and progressive limitation of mobility.***
- ***Suboptimal nutrition including loss of appetite, weight loss, cachexia and wasting, low serum albumin/pre-albumin, and low hemoglobin as well as dehydration.***

- ***Diminished tissue perfusion, impaired skin oxygenation, decreased local skin temperature, mottled discoloration, and skin necrosis.***
- ***Loss of skin integrity from any of a number of factors including equipment or devices, incontinence, chemical irritants, chronic exposure to body fluids, skin tears, pressure, shear, friction, and infections.***
- ***Impaired immune function.***

Diminished tissue perfusion is the most significant risk factors for SCALE and generally occurs in areas of the body with end arteries, such as the fingers, toes, ears, and nose. These areas may exhibit early signs of vascular compromise and ultimate collapse, such as dusky erythema, mottled discoloration, local cooling, and eventually infarcts and gangrene.

As the body faces a critical illness or disease state, a normal protective function may be to shunt a larger percentage of cardiac output from the skin to more vital internal organs, thus averting immediate death. Chronic shunting of blood to the vital organs may also occur as a result of limited fluid intake over a long period of time. Most of the skin has collateral vascular supply but distal locations such as the fingers, toes, ears and nose have a single vascular route and are more susceptible to a critical decrease in tissue oxygenation due to vasoconstriction. Furthermore, the ability to tolerate pressure is limited in poorly perfused body areas.

Additional literature reviews and clinical research are needed to more thoroughly comprehend and document all of the potential risk factors associated with SCALE and their clinical manifestations.

For pressure ulcers, it is important to determine if the ulcer may be (i) healable within an individual's life expectancy, (ii) maintained, or (iii) non-healable or palliative.

Statement 7

A total skin assessment should be performed regularly and document all areas of concern consistent with the wishes and condition of the patient. Pay special attention to bony prominences and skin areas with underlying cartilage. Areas of special concern include the sacrum, coccyx, ischial tuberosities, trochanters, scapulae, occiput, heels, digits, nose and ears. Describe the skin or wound abnormality exactly as assessed.

It is important to assess the whole body because there may be signs that relate to skin compromise. Table 1 provides a limited list of dermatologic terms that may be useful when describing areas of concern. Table 2 provides descriptive terms for lesions based on characteristics and size.

Statement 8

Consultation with a qualified health care professional is recommended for any skin changes associated with increased pain, signs of infection, skin breakdown (when the goal may be healing), and whenever the patient's circle of care expresses a significant concern.

There are very definite descriptive terms for skin changes that can be used to facilitate communication between health care professionals (see Statement 7). Until more is known about SCALE, subjective symptoms need to be reported and objective skin changes described. This will allow for identification and characterization of potential end of life skin changes.

An accurate diagnosis can lead to decisions about the area of concern and whether it is related to end of life care and/or other factors. The diagnosis will help determine appropriate treatment and establish realistic outcomes for skin changes. For pressure ulcers, it is important to determine if the ulcer may be (i) healable within an individual's life expectancy, (ii) maintained, or (iii) non-healable or palliative.[17] The treatment plan will depend on an accurate diagnosis, the individual's life expectancy and wishes, family members' expectations, institutional policies,

and the availability of an interprofessional team to optimize care.31 Remember that patient status can change and appropriate reassessments with determination of likely outcomes may be necessary.

It is important to remember that a maintenance or non-healable wound classification does not necessarily equate with withholding treatment. For example, the patient may benefit with improved quality of life from surgical debridement and/or the use of advanced support surfaces.

Statement 9

The probable skin change etiology and goals of care should be determined. Consider the 5 Ps for determining appropriate intervention strategies:

- ***Prevention***
- ***Prescription (may heal with appropriate treatment)***
- ***Preservation (maintenance without deterioration)***
- ***Palliation (provide comfort and care)***
- ***Preference (patient desires)***

Prevention is important for well being, enhanced quality of life, potential reimbursement, and to avoid unplanned medical consequences for end of life care. The skin becomes fragile when stressed with

Table 1: Useful dermatologic terms for describing areas of concern. Additional terms can be found in the Glossary included at end of this document.

Term	*Definition*
Bruise	An injury producing a hematoma or diffuse extravasation of blood without rupture of the skin.[29] Often presents as a reddish, purple, black discoloration of the skin.
Crust	A hard outer layer or covering; cutaneous crusts are often formed by dried serum, pus or blood on the surface of a ruptured blister or pustule.[29]
Erosion (denudation)	A loss of surface skin with an epidermal base.
Eschar	Thick adherent, necrotic tissue that is typically dry and brown, black or gray in color.
Fissure	A thin linear loss of skin with a dermal or deeper base.
Hematoma	A collection of blood in the soft tissues.
Lesion	Any change in the skin that may be a normal or abnormal variant including a wound or injury.[29] It encompasses everything from macular lesions (color changes without elevation or depression of the skin) through total skin breakdown.
Mottling of skin due to vascular stasis	An area of skin composed of macular lesions of varying shades or colors over the smaller or medium sized blood vessels.[29]
Scale	Surface keratin that may be thick or thin, resembling a fish scale, cast off (desquamating) from the skin.[29]
Skin Tear	A traumatic wound occurring principally on the extremities of older adults as a result of friction alone or with shearing and frictional forces, that separate the epidermis from the dermis (partial-thickness wound) or which separate both the epidermis and the dermis from the underlying structures (full-thickness wound).[30]
Slough	Yellow, green, tan, or white putrefied debris often partly separated from the surface of the wound bed.[29]
Ulcer	A loss of surface skin with a dermal or deeper base.

decreased oxygen availability associated with the end of life. The plan of care needs to address excessive pressure, friction, shear, moisture, suboptimal nutrition, and immobilization.

Prescription refers to the interventions for a treatable lesion. Even with the stress of dying, some lesions are healable after appropriate treatment. Interventions must be aimed at treating the cause and at patient centered concerns (pain, quality of life), before addressing the components of local wound care as consistent with the patient's goals and wishes.

Preservation refers to situations where the opportunity for wound healing or improvement is limited, so maintenance of the wound in its present clinical state is the desired outcome. A maintenance wound may have the potential to heal, but there may be other overriding medical factors that could direct the interprofessional team to maintain the status quo. For example there may be limited access to care, or the patient may simply refuse treatment.

Palliation refers to those situations in which the goal of treatment is comfort and care, not healing. A palliative or non-healable wound may deteriorate due to a general decline in the health of the patient as part of the dying process, or due to hypoperfusion associated with non-correctable critical ischemia.[32, 33] In some situations, palliative wounds may also benefit from some treatment interventions such as surgical debridement or support surfaces, even when the goal is not to heal the wound.[34]

Preference includes taking into account the preferences of the patient and the patient's circle of care.

Table 2: Dermatological descriptions of lesions based on characteristics and size.

Lesion Characteristics	*Lesion Size*	
	<1 cm	*>1 cm*
Flat	Macule	Patch
Elevated	Papule	Plaque
Blister	Vesicle	Bulla

The 5P enabler can be used in combination with the SOAPIE mnemonic to help explain the process of translating this recommendation into practice (Figure 1).[35] Realistic outcomes can be derived from appropriate SOAPIE processes with the 5 skin Ps becoming the guide to the realistic outcomes for each individual.

S = Subjective skin & wound assessment: The person at the end of life needs to be assessed by history, including an assessment of the risk for developing a skin change or pressure ulcer (Braden Scale or other valid and reliable risk assessment scale).[36]

O = Objective observation of skin & wound: A physical exam should identify and document skin changes that may be associated with the end of life or other etiologies including any existing pressure ulcers.

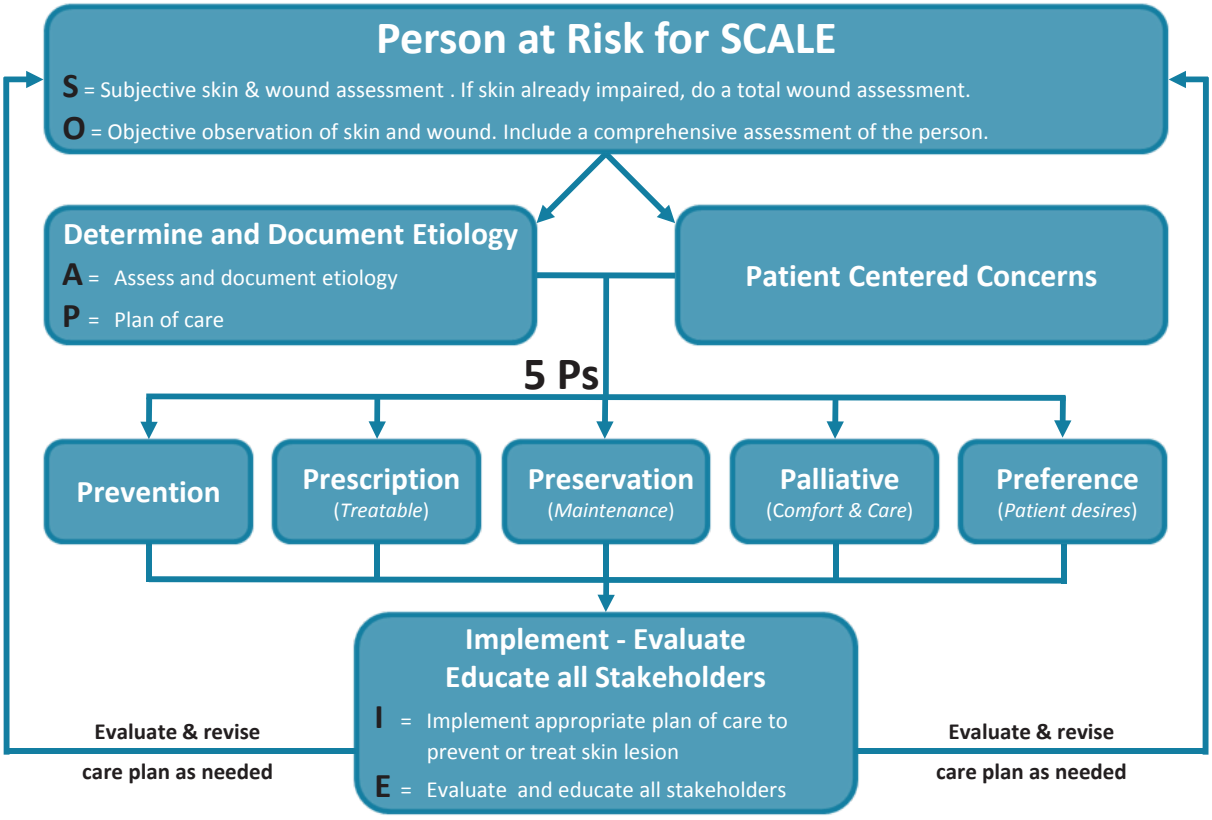
A = Assess and document etiology: An assessment should then be made of the general condition of the patient and a care plan.

P = Plan of care: A care plan should be developed that includes a decision on skin care considering the 5P's as outlined in the Figure 1. This plan of care should also consider input and wishes from the patient and the patient's circle of care.

I = Implement appropriate plan of care: For successful implementation, the plan of care must be matched with the healthcare system resources (availability of equipment and personnel) along with appropriate education and feedback from the patient's circle of care and as consistent with the patient's goals and wishes.

E = Evaluate and educate all stakeholders: The interprofessional team also needs to facilitate appropriate education, management, and periodic reevaluation of the care plan as the patient's health status changes.

Figure 1: The SOAPIE mnemonic with the 5P enabler.



Statement 10

Patients and concerned individuals should be educated regarding SCALE and the plan of care.

Education needs to be directed not only to the patient but also the patient's circle of care. Within the confines allowed by local protected health information regulations (e.g. HIPAA, 1996, USA),[28] the patient's circle of care needs to be included in decision making processes regarding goals of care and the communication of the meaning and method of accomplishing those decisions. Collaboration and communication should be ongoing with designated representatives from the patient's circle of care and the clinical team connecting at regular intervals. Documentation of decision making, educational efforts, and the patient's circle of care perspective is recommended. If adherence to the plan of care cannot be achieved, this should be documented in the medical record (including the reasons), and alternative plans proposed if available and feasible.

Education also extends beyond the patient's circle of care, to other involved healthcare professionals, healthcare administrators, policy makers, and to the payers. Healthcare professionals need to facilitate communication and collaboration across care settings and disciplines; organizations need to prepare staff to identify and manage SCALE. Ongoing discussions with key stakeholders will additionally provide a stimulus for additional evidence based research and education regarding all aspects of SCALE.

Healthcare professionals need to facilitate communication and collaboration across care settings and disciplines; organizations need to prepare staff to identify and manage SCALE.

Recommendations for Future Research

Conduct and disseminate through publications and presentations:

- A thorough review of the literature concerning all aspects of SCALE.
- Research to identify the mechanisms for the proposed decreased hypoperfusion and oxygenation of the skin and soft tissues involved with SCALE and resulting outcomes.
- Research to determine the mechanisms for the proposed, tissue, cellular, and molecular dysfunctions that occur during SCALE.
- Research that helps to clarify and distinguish skin and soft tissue damage associated with SCALE from pressure ulcers and other skin disorders not associated with skin organ compromise or the end of life.
- Research into predictive tools for the onset and measurement of SCALE and the timing of life's end (possibly adaptive use of the Palliative Performance Scale (http://palliative.info/resource_material/PPSv2.pdf)
- Qualitative research to explore the impact of SCALE on the patient, the patient's circle of care, and professional caregivers with regard to healthcare-related quality of life.
- Development of a database of patients (with histories) suspected of exhibiting SCALE to analyze them retrospectively for skin and soft tissue changes and risk factors that occurred just prior to death. Isolate the skin changes and risk factors involved and determine how important each individual variable is to the occurrence of SCALE.
- Research cataloging patients who do not exhibit SCALE to identify factors that may help prevent the occurrence of SCALE.
- Develop a registry of Kennedy Terminal Ulcers to better categorize this phenomenon, including location, clinical description, patient and ulcer outcomes, and the presence of other end of life skin changes including lesions in other locations.
- Both prospective and retrospective prevalence research of individuals suspected of exhibiting SCALE, particularly among hospice patients.
- Research on specific medical and physiologic conditions that may contribute to SCALE. These include but may not be limited to malignancy, hypotension and hemodynamic instability, administration of potent vasoconstrictors, peripheral arterial and vascular disease, hypoxia, malnutrition, and severe anemia.

Conclusions

SCALE Panel members are in agreement that there are observable changes in the skin at the end of life. Our current understanding of this complex phenomenon is limited and the panel concludes that additional research is necessary to assess the etiology of SCALE, to clinically describe and diagnose the related skin changes, and to recommend appropriate pathways of care. The panel recommends that clinicians, laypeople, and policy makers need to be better educated in the medical, social, legal and financial ramifications of SCALE.

Health care organizations need to ensure the provision of resources that enable health care professionals to identify and care for SCALE while maintaining the dignity of the patient, family and circle of care to the end of life.

Glossary of Terms

Arterial Ulcer: An ulcer that occurs almost exclusively in the distal lower extremity due to inadequate perfusion/ischemia.[37]

Avoidable (pressure ulcer): The resident (individual) developed a pressure ulcer and the facility did not do one or more of the following: evaluate the resident's clinical condition and pressure ulcer risk factors; define and implement interventions that are consistent with resident needs, resident goals, and recognized standards of practice; monitor and evaluate the impact of the interventions; or revise the interventions as appropriate (CMS definition).[38]

Charting by Exception (CBE): Charting by exception is premised on an assumption that the patient has manifested a normal response to all interventions unless an abnormal response is charted.39 This type of charting is often performed with flow sheets that are based on preestablished guidelines, protocols, and procedures that identify and document the standard patient management and care delivery. Clinicians need to make additional documentation when the patient's condition deviates from the standard or what's expected.[40]

Crust: A hard outer layer or covering; cutaneous crusts are often formed by dried serum, pus or blood (one or more components may co-exist) on the surface of a ruptured blister or pustule.[29]

Decubitus Ominosus: Medical term first used by Jean-Martin Charcot in the 19th century to signify a sacral ulcer that presages death.

Delphi Method: A systematic, interactive forecasting method which relies on a panel of independent experts. The carefully selected experts answer questionnaires in two or more rounds. After each round, a facilitator provides an anonymous summary of the experts' forecasts from the previous round as well as the reasons they provided for their judgments. Thus, experts are encouraged to revise their earlier answers in light of the replies of other members of their panel. It is believed that during this process the range of the answers will decrease and the group will converge towards the "correct" answer. Finally, the process is stopped after a pre-defined stop criterion (e.g. number of rounds, achievement of consensus, stability of results) and the mean or median scores of the final rounds determine the results.[19]

Denudation: See erosion.

Diabetic Ulcer: A wound occurring most often in the feet of people with diabetes due most commonly to neuropathy and/or peripheral vascular disease.[41]

End of Life: End of life is defined as a phase of life when a person is living with an illness that will worsen and eventually cause death. It is not limited to the short period of time when the person is moribund.[1]

Erosion: A loss of surface skin with an epidermal base.

Fissure: A thin linear loss of skin with a dermal or deeper base.

Healable (wound): A wound occurring on an individual whose body can support the phases of wound healing within the individuals expected lifetime.

Healed (wound): A wound that has attained closure of the epidermal surface. A recently closed wound may only have 20% tensile strength of skin that has never been wounded and may be susceptible to recurrent ulceration.

Kennedy Terminal Ulcer: A pressure ulcer that some individuals develop as they are dying. It is usually shaped like a pear, butterfly, or horseshoe, usually on the coccyx or sacrum (but has been reported on other anatomical areas), has colors of red, yellow or black, is sudden in onset, and usually is associated with imminent death.[2, 42-49]

Lesion: Any change in the skin that may be a normal or abnormal variant including a wound

or injury.[29] It encompasses everything from macular lesions (color changes without elevation or depression of the skin) through total skin breakdown.

Maintenance (wound): An attempt to keep an ulcer from deteriorating by providing good wound care. The wound may not heal due to patient choice or a lack of the health care system to provide optimal resources to promote healing.

Non-healable (wound): A wound that often deteriorates and occurs on an individual whose body cannot support the phases of wound healing within the individuals expected lifetime. There may be inadequate vascular supply to support healing or the cause of the wound cannot be corrected.

Palliative skin care: Providing comfort and support for the bodies cutaneous surface (part of the practice of palliative medicine) is not a time-confined but rather a goal-oriented and patient-centered care delivery model.[50] Palliative wound care is the evolving body of knowledge and skills that take a holistic approach to relieving suffering and improving quality of life for patients (individuals) and families living with chronic wounds, whether the wound is healable, can be maintained or may deteriorate.[32]

Patient circle of care: This is not a legal term, but rather a social term that includes all of the stakeholders in the patient's health and well being. The term includes, but is not limited to, the patient, a legal guardian or responsible party, a spouse or significant other, interested friends or family members, caregivers, and any other individual(s) who may have an interest in the patient's care and well being.

Pressure Ulcer: A pressure ulcer is localized injury to the skin and/or underlying tissue usually over a bony prominence, as a result of pressure, or pressure in combination with shear and/or friction. A number of contributing or confounding factors are also associated with pressure ulcers; the significance of these factors is yet to be elucidated.[51]

Scale (skin): Surface keratin that may be thick or thin, resembling a fish scale, cast off (desquamating) from the skin.[29]

SCALE: The acronym for Skin Changes at Life's End.

Skin breakdown: An interruption in the integrity of the skin surface leading to defect in the epidermal covering with an epidermal, dermal or deeper base.

Skin compromise: A state in which skin's protective function is at risk of breaking down.

Skin failure: An acute episode where the skin and subcutaneous tissues die (become necrotic) due to hypoperfusion that occurs concurrent with severe dysfunction or failure of other organ systems.[8]

Skin tear: A traumatic wound occurring principally on the extremities of older adults as a result of friction alone or with shearing and frictional forces that separate the epidermis from the dermis (partial-thickness wound) or a deeper split that separates both the epidermis and the dermis from the underlying structures (full-thickness wound).[30]

Stakeholders: An individual, facility, or organization with an interest in Skin Changes at Life's End (SCALE).

Stage I Pressure Ulcer: Intact skin with non-blanchable redness of a localized area usually over a bony prominence. Darkly pigmented skin may not have visible blanching; its color may differ from the surrounding area.[51]

Stage II Pressure Ulcer: Partial thickness loss of dermis presenting as a shallow open ulcer with a red pink wound bed, without slough. May also present as an intact or open/ruptured serum-filled blister.[51]

Stage III Pressure Ulcer: Full thickness tissue loss. Subcutaneous fat may be visible but bone, tendon or muscle are not exposed. Slough may be present but does not obscure the depth of tissue loss. May include undermining and tunneling.[51]

Stage IV Pressure Ulcer: Full thickness tissue loss

with exposed bone, tendon or muscle. Slough or eschar may be present on some parts of the wound bed. Often include undermining and tunneling.[51]

Suspected Deep Tissue Injury: Purple or maroon localized area of discolored intact skin or blood-filled blister due to damage of underlying soft tissue from pressure and/or shear. The area may be preceded by tissue that is painful, firm, mushy, boggy, warmer or cooler as compared to adjacent tissue.[51]

Terminal tissue trauma: Damage to the integumentary system that has occurred at the end of life.

Ulcer: A loss of surface skin with a dermal or deeper base.

Unavoidable (pressure ulcer): The resident developed a pressure ulcer even though the facility had evaluated the resident's clinical condition and pressure ulcer risk factors; defined and implemented interventions that are consistent with resident needs, goals, and recognized standards of practice; monitored and evaluated the impact of the interventions; and revised the approaches as appropriate (CMS definition).[38]

Unstageable Pressure Ulcer: Full thickness tissue loss in which the base of the ulcer is covered by slough (yellow, tan, gray, green or brown) and/or eschar (tan, brown or black) in the wound bed.[51]

Venous Ulcer: A ulceration that occurs on the lower limb secondary to underlying venous disease; formerly called stasis ulcers.[52]

References

1. Qaseem A, Snow V, Shekelle P, et al. Evidence-based interventions to improve the palliative care of pain, dyspnea, and depression at the end of life: a clinical practice guideline from the American College of Physicians. Ann Intern Med 2008;148(2):141-6.
2. Kennedy KL. The prevalence of pressure ulcers in an intermediate care facility. Decubitus 1989;2(2):44-5.
3. Hanson D, Langemo DK, Olson B, et al. The prevalence and incidence of pressure ulcers in the hospice setting: analysis of two methodologies. Am J Hosp Palliat Care 1991;8(5):18-22.
4. Levine JM. Historical perspective on pressure ulcers: the decubitus ominosus of Jean-Martin Charcot. J Am Geriatr Soc 2005;53(7):1248-51.
5. Charcot JM. Lectures on the Diseases of the Nervous System. Translated by G. Sigerson. London: The New Sydenham Society; 1877.
6. Shenk D. The Forgetting: Alzheimer's: Portrait of an Epidemic. Chapter 1, I Have Lost Myself: Anchor; 2003.
7. Langemo DK. The hot seat: the reality of skin failure. 18th Annual Clinical Symposium on Advances in Skin & Wound Care. Chicago, IL 2003.
8. Langemo DK, Brown G. Skin fails too: acute, chronic, and end-stage skin failure. Adv Skin Wound Care 2006;19(4):206-11.
9. Goode PS, Allman RM. The prevention and management of pressure ulcers. Med Clin North Am 1989;73(6):1511-24.
10. La Puma J. The ethics of pressure ulcers. Decubitus 1991;4(2):43-4.
11. Witkowski JA, Parish LC. Skin failure and the pressure ulcer. Decubitus 1993;6(5):4.
12. Leijten FS, De Weerd AW, Poortvliet DC, De Ridder VA, Ulrich C, Harink-De Weerd JE. Critical illness polyneuropathy in multiple organ dysfunction syndrome and weaning from the ventilator. Intensive Care Med 1996;22(9):856-61.
13. Hobbs L, Spahn JG, Duncan C. Skin failure: what happens when this organ system fails. Poster presented at the 32nd Annual Wound, Ostomy, and Continence Nurses Society, Toronto, Canada June 2000.
14. Witkowski JA, Parish LC. The decubitus ulcer: skin failure and destructive behavior. Int J Dermatol 2000;39(12):894-6.
15. Brown G. Long-term outcomes of full-thickness pressure ulcers: healing and mortality. Ostomy Wound Manage 2003;49(10):42-50.
16. Parish LC, Witkowski JA. Chronic wounds: myths about decubitus ulcers. Int J Dermatol 1994;33(9):623-4.
17. Alvarez OM, Kalinski C, Nusbaum J, et al. Incorporating wound healing strategies to improve palliation (symptom management) in patients with chronic wounds. J Palliat Med 2007;10(5):1161-89.
18. Thomas DR. Are all pressure ulcers avoidable? J Am Med Dir Assoc 2003;4(2 Suppl):S43-8.
19. Wikipedia The Free Encyclopedia: Delphi Method. Available at URL: http://en.wikipedia.org/wiki/Delphi_method. Last Accessed July 20,2009.
20. Sibbald RG, Krasner DL, Lutz JB, et al. Skin changes at life's end (SCALE): a preliminary consensus statement. World Council of Enterostomal Therapists Journal 2008;28(4):15-22.
21. Sibbald RG, Krasner DL, Lutz JB, et al. Skin Changes At life's End (SCALE) Preliminary Consensus Statement. Available at URL: www.Gaymar.com >Medical Research and Education>SCALE Consensus Documents. 2008;Last accessed July 20, 2009.
22. Ayello EA, Capitulo KL, Fife CE, et al. Legal Issues in the

Care of Pressure Ulcer Patients: Key Concepts for Healthcare Providers. White Paper Available from URL: WWW.Medline.com 2009.

23. Victoria Hospice Society, Palliative Performance Scale version 2 (PPSv2). Available at URL: http://palliative.info/resource_material/PPSv2.pdf 2001.
24. International Association for Hospice & Palliative Care: Assessment and Research Tools: Assessment and Research Tools. 2009;Available at URL: http://www.hospicecare.com/resources/pain-research.htm.
25. Price P. Health-Related Quality of Life. In: Krasner D, Rodeheaver GT, Sibbald RG, editors. Chronic wound care: a clinical source book for healthcare professionals. 4th ed. Malvern, PA: HMP Communications; 2007. p. 79-83.
26. Krasner DL, Papen J, Sibbald RG. Helping Patients Out of the SWAMP©: Skin and Wound Assessment and Management of Pain. In: Krasner D, Rodeheaver GT, Sibbald RG, editors. Chronic wound care: a clinical source book for healthcare professionals. 4th ed. Malvern, PA: HMP Communications; 2007. p. 85-97.
27. Langemo DK. When the goal is palliative care. Adv Skin Wound Care 2006;19(3):148-54.
28. U.S. Department of Health and Human Services. Standards for Privacy of Individually Identifiable Health Information; Final Rule, 45 C.F.R. Parts 160, and 164. Code of Federal Regulations Available at: http://wedi.org/snip/public/articles/45CFR160&164.pdf. Accessed May 13, 2009.
29. Stedman's Medical Dictionary, 27th Edition. Lippincott Williams & Wilkins. 2008 Available from URL: http://www.stedmans.com.
30. Payne RL, Martin ML. Defining and classifying skin tears: need for a common language. Ostomy Wound Manage 1993;39(5):16-20, 22-4, 26.
31. Krasner D, Rodeheaver GT, Sibbald RG. Interprofessional Wound Caring. In: Krasner D, Rodeheaver GT, Sibbald RG, editors. Chronic wound care: a clinical source book for healthcare professionals. 4th ed. Malvern, P.A.: HMP Communications; 2007. p. 3-9.
32. Ferris FD, Al Khateib AA, Fromantin I, et al. Palliative wound care: managing chronic wounds across life's continuum: a consensus statement from the International Palliative Wound Care Initiative. J Palliat Med 2007;10(1):37-9.
33. Romanelli M, Dini V, Williamson D, Paterson D, Pope M, Sibbald R. Measurement: Lower Leg Ulcer Vascular and Wound Assessment. In: Krasner D, Rodeheaver GT, Sibbald RG, editors. Chronic wound care: a clinical source book for healthcare professionals. 4th ed. Malvern, PA: HMP Communications; 2007. p. 463-80.
34. Lee KF, Ennis WJ, Dunn GP. Surgical palliative care of advanced wounds. Am J Hosp Palliat Care 2007;24(2):154-60.
35. Weed LJ. The problem oriented record as a basic tool in medical education, patient care and clinical research. Ann Clin Res 1971;3(3):131-4.
36. Bergstrom N, Braden BJ, Laguzza A, Holman V. The Braden Scale for Predicting Pressure Sore Risk. Nurs Res 1987;36(4):205-10.
37. Holloway GA. Arterial Ulcers: Assessment, Classification, and Management. In: Krasner D, Rodeheaver GT, Sibbald RG, editors. Chronic wound care: a clinical source book for healthcare professionals. 4th ed. Malvern, PA: HMP Communications; 2007. p. 443-449.
38. CMS Manual System Department of Health & Human Services (DHHS). Pub. 100-07 State Operations. Provider Certification. Centers for Medicare & Medicaid Services (CMS).Transmittal 4. Date: November 12, 2004.
39. Murphy EK. Charting by exception - OR Nursing Law. AORN J 2003 (NOV).
40. Smith LM. How to chart by exception. Nursing 2002 (SEPT).
41. Steed DL. Wounds in People with Diabetes: Assessment, Classification, and Management. In: Krasner D, Rodeheaver GT, Sibbald RG, editors. Chronic wound care: a clinical source book for healthcare professionals. 4th ed. Malvern, PA: HMP Communications; 2007. p. 537-542.
42. Kozier B, Erb G, Olivieri R. Fundamentals of nursing: concepts, process and practice. 4th ed. Redwood City, Calif.: Addison-Wesley Nursing; 1991.
43. Milne CT, Corbett LQ. The Kennedy Terminal Ulcer. In: Wound, Ostomy, and Continence Secrets. Philadelphia, PA: Hanley & Belfus; 2003. p. 198-199.
44. Kennedy KL. Gaymar Pictoral Guide to Pressure Ulcer Assessment. Second ed. Orchard Park, NY: Gaymar Industries, Inc.; 2006.
45. Weir D. Pressure ulcers: assessment, classification, and management. In: Krasner DL, Rodeheaver GT, Sibbald RG, editors. Chronic Wound Chare: A Clinical Source Book for Healthcare Professionals. 4th ed., edited by Diane L. Krasner, George T. Rodeheaver, R. Gary Sibbald. ed. Malvern, Pennsylvania: HMP Communications; 2007. p. 577-578.
46. The different types of wounds. In: The Wound Care Handbook. Mundelein, IL: Medline Industries, Inc.; 2007. p. 110-11.
47. Ayello EA, Shank JE. Ulcerative lesions. In: Kuebler KK, Heidrich DE, Esper P, editors. Palliative & end-of-life care: clinical practice guidelines. 2nd ed. ed. Philadelphia, Pa. ; Edinburgh: Elsevier Saunders; 2007. p. 523.
48. Turnbull GB. Year's end notables. Ostomy Wound Manage 2001;47(12):10.
49. Hogue EE. Key legal issues for wound care practitioners in 2005. The Remmington Report May/June 2005;13(3):14-16.
50. Davis MP, Walsh D, LeGrand SB, Lagman R. End-of-Life care: the death of palliative medicine? J Palliat Med 2002;5(6):813-4.
51. National Pressure Ulcer advisory Panel: Updated Staging System. Available at URL: http://www.npuap.org/pr2.htm. 2007.
52. Sibbald RG, Williamson D, Contreras-Ruiz J, et al. Venous Leg Ulcers. In: Krasner D, Rodeheaver GT, Sibbald RG, editors. Chronic wound care: a clinical source book for healthcare professionals. 4th ed. Malvern, PA: HMP Communications; 2007. p. 429-442. © 2008 SCALE Expert Panel.

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Definition and Characteristics of Chronic Tissue Injury

A Unique Form of Skin Damage

Mary F. Mahoney ◆ Barbara J. Rozenboom

ABSTRACT

BACKGROUND: The purpose of this article is to examine the evidence related to a unique phenomenon of purple-maroon discoloration of the buttocks found in homecare patients and to recommend a label for this phenomenon.

CASES: Initially, we searched the literature to identify and retrieve any evidence related to this unique form of purple-maroon discoloration of the buttocks. No evidence was found. To illustrate the condition, we compared 4 cases of what we have labeled chronic tissue injury to 6 patients with purple-maroon discoloration of the buttocks from different causes.

CONCLUSION: Chronic tissue injury is characterized by a persistent purple-maroon discoloration located on the fleshy portion of the buttocks that does not improve or deteriorate. Unlike other causes of purple discoloration such as deep tissue pressure injury, there is minimal change in the discoloration over time. Additional research is needed to further our understanding of the histopathology of this phenomenon.

KEY WORDS: Chronic tissue injury, Chronic wound, Deep tissue pressure injury, Moisture-associated skin damage, Skin failure, Venous ulcers.

INTRODUCTION

Understanding the etiology of various forms of skin damage is necessary for accurate assessment and classification. For example, pressure injury categories have clinically relevant regulatory and cost implications.[1,2] Misidentification can lead to inconsistent and inaccurate benchmarking, ineffective treatment, and inaccurate use of resources.

Through our combined 35 years of homecare nursing experience, we have reviewed thousands of homecare patients' medical records with a form of skin injury of the buttocks that did not resemble any known skin injury category. The injury was noted to be a purple-maroon discoloration of the fleshy buttocks present for a long period. The area of damaged skin sometimes included superficial abraded skin or small open lesions. Anecdotal conversations with expert WOC nurses practicing in the homecare setting corroborated our observations. Given the absence of a recognized label for this phenomenon, WOC nurses must arbitrarily choose a wound type, resulting in inconsistent classification, even among wound care experts. In addition, acute care and homecare clinicians, who are required to classify wounds due to regulatory requirements, are also confused as to how to categorize this tissue injury.[3]

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Procedures were reviewed by UnityPoint at Home and UnityPoint Health. The article was determined to be exempt from individual informed consent.

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All clinicians must accurately identify wound type to guide management, comply with federal regulations, and achieve reimbursement.[2] Choosing the wound type may be limited by the terminology available in the organization's electronic medical record system. For example, home health clinicians must classify wounds using the Outcomes and Assessment Information Set (OASIS). We have found that clinicians typically label these unique purple-maroon lesions as Stage 1 or Stage 2 pressure injury (PI), deep tissue pressure injury (DTPI), skin failure, moisture-associated skin damage, trauma, or inflammatory lesions. The purpose of this article is to examine evidence related to chronic tissue injury, present a case series of individuals with chronic tissue injury compared to patients with similar characteristics of the buttocks, and recommend a new label for this unique phenomenon.

LITERATURE REVIEW

We completed a focused literature review using the CINAHL and MEDLINE databases with no language, time, or date of publication limiters. The key search words included suspected deep tissue pressure injury, pressure, moisture-associated skin damage, skin failure, friction injury, purple, venous, venous pooling, venous engorgement, recliner butt, and chronic tissue injury. We limited our search to articles published in the English language. Based on these inclusion/exclusion criteria, we retrieved and reviewed 41 abstracts and selected 9 articles that were read in full. We also reviewed current wound textbooks, National Pressure Ulcer Advisory Panel clinical resources, and the Federal Guidance Manual for homecare.

In addition to the literature search, 2 expert WOC nurses (M.F.M. and B.J.R.) reviewed medical records of patients from our home healthcare agency with evidence of this phenomenon. This review was approved by UnityPoint at Home nursing

administration, UnityPoint at Home compliance officers, and UnityPoint Health Director of Ethics; study procedures were determined to be exempt from individual informed consent.

Our literature review identified no evidence of a unique form of skin damage presenting as chronic purple-maroon discoloration of the buttocks. Although this discoloration frequently coexists with other skin injuries, we assert this injury is unique and requires further investigation.

CASE SELECTION

Based on the lack of published evidence, we monitored our practice for patients who presented with purple-maroon discoloration of the buttocks that was inconsistent with other typical skin injuries located in this area of the body. The skin damage is characterized by a typically bilateral, purple-maroon discoloration of the fleshy buttocks that does not improve or deteriorate over time. We selected 4 cases that exemplify this unique phenomenon and compared them to other skin injuries (Table 1). Cases were typically chair-bound adults whose prime seating choice was a recliner chair. Our experience indicates that many homecare patients sit and sleep in a favored recliner chair for long periods. Thus, the term "recliner butt" is often used by homecare WOC nurses to describe this form of skin damage. We acknowledge that we need a term that better characterizes the cause of this unique skin damage.

Chronic Tissue Injury

Chronic tissue injury (CTI) is a distinct form of skin damage affecting the buttocks. It presents as purple-maroon discoloration with thinning of the epidermis, with or without open skin injury. Chronic tissue injury is located on the fleshy portion of the buttocks and not necessarily over a bony prominence. The distinguishing factor is that CTIs do not innately improve or deteriorate into partial or full-thickness injury.

The etiology of CTI is unknown, rendering differential diagnosis difficult. It may occur with other skin injuries. Table 2 summarizes 4 patients with CTI, and Table 3 describes 7 cases of patients with other forms of unique skin damage that may be confused with CTI.

Chronic Tissue Injury Versus Friction Skin Injury

Our understanding of friction and pressure injuries has advanced in recent years.[4-7] Friction is defined as the force that resists the motion created when surfaces (such as the skin and recliner surface) slide against each other.[7] In contrast, the term "pressure" is a measure of force that exists when the skin is compressed between a bony prominence and a surface such as a chair or bed, whereas shear is the force created when layers of skin are laterally shifted against one another.[6] Berke[4] observed that friction skin injuries (FSIs) are located on the rounded portion of the buttocks but not over a bony prominence. She postulated that because aging skin is thinner and may have less sensation, it may be more susceptible to friction injury.[4] Friction skin injuries are characterized by lichenification and skin ridging; they may be full thickness, and are amenable to treatment. Descriptions of FSI do not include skin discoloration.[4,6]

In summary, while both CTI and FSI occur on the fleshy buttocks, CTI has a distinct purple-maroon discoloration visible over time. In addition, while the thinned epidermal tissue of the CTI is easily damaged with friction forces, CTI lacks the lichenification and ridging of FSI (Table 3, Case 5).

Chronic Tissue Injury Versus Moisture-Associated Skin Damage

Moisture-associated skin damage (MASD) is characterized by inflammation and erosion of the epidermis resulting from prolonged exposure to various sources of bodily secretions or effluent.[8-12] As its name implies, MASD is characterized by excessive exposure to moisture such as perspiration, incontinence, or wound exudate that may not be present with CTI. In addition, CTI has a characteristic purple-maroon discoloration whereas the inflammation of MASD tends to be bright red or a subtler red in persons with darker skin tones. Unlike MASD, CTI is not frequently associated with secondary cutaneous infections (Table 3, Case 6).

Chronic Tissue Injury Versus Venous Discoloration

Venous pooling or venous engorgement has been suggested as a possible etiologic factor in the development of CTI.[13] Nevertheless, the pathophysiology and clinical relevance of venous pooling and engorgement are not well understood. Given this gap in evidence, it is not possible to conclusively link the purple-maroon discoloration of CTI to venous pooling. Similarly, hemosiderosis, defined as a reddish-brown black pigmentation discoloration of the skin as a result of extravasated red blood cells and subsequent release of iron into the tissue in patients with venous disease, has also been suggested as a possible contributor to the discoloration of CTI (Table 3, Cases 7 and 8).[14,15]

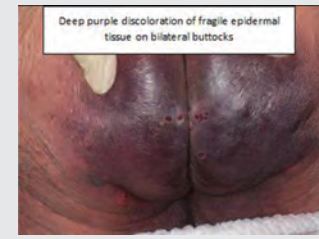
Both CTI and venous engorgement are characterized by a purplish discoloration of the skin. Patients with CTI are typically chair-bound, possibly creating venous pooling in the trunk owing to prolonged time in a seated position with the

TABLE 1.
Differential Assessment of Various Forms of Skin Damage

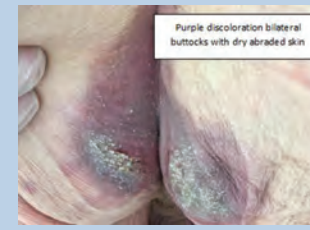
Type of Injury	Distinguishing Differences
Friction skin injury	No purple or purple-maroon discoloration, presents with lichenification and ridges
Moisture-associated skin damage	Presents with erythema and not purple-maroon discoloration
Stage 1 pressure injury	Nonblanchable erythema, no purple-maroon discoloration
Stage 2 pressure injury	No purple-maroon discoloration, partial-thickness skin loss over bony prominence
Deep tissue pressure injury	Similar skin discoloration, occurs over a bony prominence; variable trajectory of deterioration or healing
Venous pooling/engorgement	Purple-maroon discoloration of chronic tissue injury is located on fleshy buttocks
Venous disease/hemosiderin	Reddish brown discoloration of skin over lower extremities
Skin failure	Tissue necrosis associated with multisystem organ failure seen at end of life
Trauma	Variable discoloration of skin due to blunt force trauma

TABLE 2.
Chronic Tissue Injury: Cases 1 to 4

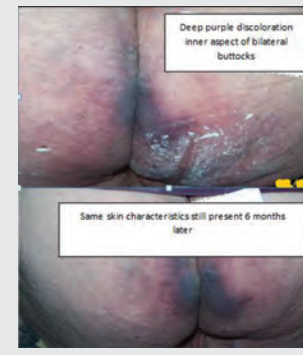
Case 1
A 72-y-old man with chronic kidney disease, diabetes, and back pain, and urinary incontinence. He had an unsteady gait and used a walker but sits and sleeps in a recliner chair. Assessment revealed deep purple-maroon discoloration and thinned epidermal tissue of bilateral fleshy buttocks with small open lesions. These small lesions were scattered, and intermittently opened. The purple-maroon discoloration did not worsen or improve during his 6 months in homecare.



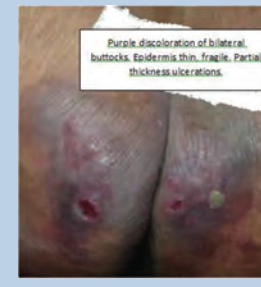
Case 2
An 88-y-old woman with heart failure and osteoarthritis. She was occasionally incontinent of urine. She spent the majority of her day sitting in a recliner with her legs elevated to decrease lower extremity edema. Assessment revealed a purple-maroon discoloration on bilateral inner and fleshy buttocks with thinned epithelium. The discoloration was originally thought to a cutaneous candida infection and friction injury.



Case 3
A 75-y-old man with obesity, diabetes mellitus, and dual urinary and fecal incontinence. He spent the majority of waking hours in a recliner chair due to generalized weakness limiting his mobility. Skin assessment revealed a deep purple-maroon discoloration of the bilateral fleshy buttocks with thinned epidermal tissue. The buttocks had similar appearance 6 months later.



Case 4
A 79-y-old man with chronic pain, obesity, and diabetes mellitus. He was frequently incontinent of urine and occasionally experienced fecal incontinence. He spent the majority of his waking hours in a recliner chair. Skin assessment revealed a purple-maroon discoloration with thin, fragile epidermal tissue initially noted on admission to our homecare service. A similar area of skin damage was noted during several readmissions to homecare. The small partial-thickness lesions intermittently open and heal. This area of skin damage retained a similar appearance over a period of 2 y.



extremities elevated. However, there is insufficient evidence to conclude that the purple discoloration of CTI is similar to the pathophysiologic mechanisms seen in patients with chronic venous disease of the lower extremities.[14,15]

Chronic Tissue Injury Versus Pressure Injury

Pressure injury occurs as a result of "intense and/or prolonged pressure or pressure in combination with shear."[1(p586)] These forces create an ischemia/reperfusion injury that may or may not result in an open wound. Stage 1 PI is a localized area of nonblanchable erythema.[1] The National Pressure Ulcer Advisory Panel states that the color change seen with a stage 1 PI does not include purple or maroon. Stage 2 PIs are characterized by partial-thickness skin loss with exposed dermis.[1] In contrast, CTI is characterized by small areas of skin loss that does not occur over bony prominences. In addition, Stage 2 pressure does not have the purple-maroon discoloration of CTI.

Unlike Stage 1 or 2 pressure injuries, DTPIs are characterized by purple discoloration of affected skin. However, the discoloration of DTPI appears as deeper violet hue, which may reflect the intense the tissue load posited to cause DTPI (Table 3, Case 9).[16,17] In addition, DTPI follows a variable course of deterioration or healing whereas the severity of CTI persists over time.[18] Deep tissue pressure injury is often associated with pain not seen in our patients with CTI.

Chronic Tissue Injury and Skin Failure

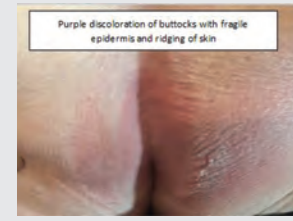
Skin failure is defined as death of the skin and underlying tissues due to poor blood circulation occurring in a context of multisystem organ failure (Table 3, Case 10).[19-21] In contrast, CTI persists over time, is not characterized by tissue necrosis, and occurs in patients with stable chronic health conditions who are not at end of life.

Chronic Tissue Injury and Trauma

Contusion, commonly referred to as bruising, is a common form of skin damage caused by blunt force trauma and leakage of blood leaking into local tissues. The discoloration of a contusion varies from deep purple to light red or even yellow that is located over the area of trauma. Case 11 (Table 3)

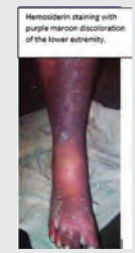
TABLE 3.
Other Forms of Skin Damage Affecting the Buttocks: Cases 5 to 11

Case 5: Friction skin injury
An 86-y-old woman with chronic obstructive pulmonary disease oxygen dependent. Urinary incontinence, fecal continence. She spent the majority of her waking and sleeping hours seated in a lift chair. Skin assessment revealed a mild purple-maroon discoloration of tissue, with abraded area typical of friction skin injury. The friction skin injury and discoloration resolved within several weeks following intervention.

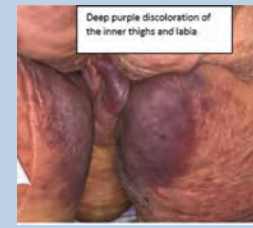


Case 6: Moisture-associated skin damage
A 65-y-old man was admitted to our homecare service following discharge from hospital for treatment for pneumonia. He was incontinent of urine and stool upon admission, had generalized weakness and limited mobility. He spent the majority of waking hours in a chair. Skin assessment revealed partial-thickness skin loss and tissue discoloration of bilateral buttocks and linear gluteal cleft upon admit to homecare. This area of skin damage resolved within a week of treatment for incontinence. Assessment showed no discoloration of tissue following resolution of skin injury.

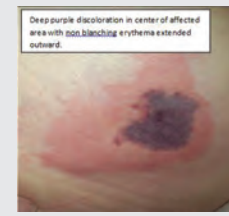
Case 7: Chronic venous disease
A 75-y-old woman with chronic venous disease, diabetes mellitus, and coronary artery disease. Skin assessment revealed discoloration of the lower extremity typical of hemosiderin staining associated with venous disease.



Case 8: Possible venous pooling
A 78-y-old woman with obesity, diabetes mellitus, coronary artery disease, and dual urinary and fecal incontinence. She spent the majority of her waking hours seated in a recliner chair. Skin assessment revealed a deep purple discoloration of the inner thighs and labia present upon admission to our homecare service. This area did not involve the fleshy buttocks. The purple discoloration persisted during several homecare episodes that spanned 2 y.

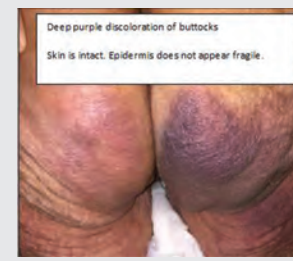


Case 9: Deep tissue pressure injury
A 67-y-old woman with debilitating neurological disease rendering her bedbound. She was managed with an indwelling urinary catheter. She preferred a side-lying position and developed extensive reddened area with intact deep purple discoloration over the trochanter bony prominence. The area subsequently deteriorated into eschar and a full-thickness pressure injury.



Case 10: Skin failure
An 85-y-old woman admitted to hospice for end-of-life care occurring in a context of an advanced state malignancy. She was bedbound at this point in her care and had an indwelling urinary catheter. She was not incontinent of stool. She developed a sudden onset of erythema with central purple-maroon discoloration and skin loss encompassing large area of the buttocks and sacral area. She died within a week following the onset of this lesion.

Case 11: Blunt force trauma
A 78-year-old man with atrial fibrillation managed by anticoagulant therapy, generalized weakness with limited mobility, and urinary incontinence. He spent the majority of his waking hours in a chair. He fell at home and developed a subsequent purple discoloration of unilateral buttock. His skin remained intact and discoloration subsequently resolved over a period of approximately 2 wk.



summarizes a patient with a contusion of the buttocks. We have observed that many of our homecare patients are on anticoagulant drug therapy, which may create bruising even with minimal trauma. In contrast, we have found that patients with CTI do not have a history of trauma and CTI persists over time, while contusions normally heal within 2 weeks.[6]

CONCLUSION

Based on our clinical experience and observations, we recommend using the term chronic tissue injury for the purple-maroon discoloration located on the fleshy portion of the buttocks. These injuries are characterized by thinned epidermal tissue, with or without open skin injury, that persist over time. While CTIs share some similarities with a variety of other skin injuries, we assert that its etiology and pathophysiology are unique. Nevertheless, we further acknowledge that additional research is needed to increase our knowledge of this clinically relevant phenomenon.

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KEY POINTS

- Chronic tissue injury is a purple-maroon discoloration of the fleshy portion of the buttocks that persists over time, with or without open skin injuries.
- Chronic tissue injury is commonly misidentified as friction injury, moisture-associated skin damage, venous engorgement, pressure injury, skin failure, and trauma.
- Further consensus and research are needed to elucidate its etiology, histopathologic characteristics, assessment, prevention, and management.

REFERENCES

1. Edsberg LE, Black JM, Goldberg M, McNichol L, Moore L, Sieggreen M. Revised national pressure ulcer advisory panel pressure injury staging system: revised pressure injury staging system. *J Wound Ostomy Continence Nurs*. 2016;4(6):585-597.
2. Centers for Medicare & Medicaid Services. OASIS-C2 2018 Guidance Manual.https://www.cms.gov/Medicare/Quality-Initiatives-Patient-Assessment-Instruments/HomeHealthQualityInits/HHQIOASISUserManual.html. Accessed October 7, 2018.
3. Savage E, Lebovitz S, Delmore B. 50 Shades of Purple: A Reference Guide for Identifying Purple Discoloration. Paper presented at: WOCN® Society & CAET Joint Conference; June 4-8, 2016; Montreal, Quebec, Canada.
4. Berke CT. Pathology and clinical presentation of friction injuries. *J Wound Ostomy Continence Nurs*. 2015;42(1):47-61.
5. National Pressure Ulcer Advisory Panel. Terms and definitions related to support surfaces. http://www.npuap.org/wp-content/uploads/2012/03/NPUAP_S3I_TD.pdf. Published 2007. Accessed January 20, 2018.
6. Pieper B. Pressure ulcers: impact, etiology, and classification. In: Bryant RA, Nix DP, eds. *Acute & Chronic Wounds: Current Management Concepts.* 4th ed. St Louis, MO: Mosby Inc; 2012.
7. Brienza D, Antokal S, Herbe L, et al. Friction-induced skin injuries—are they pressure ulcers? *J Wound Ostomy Continence Nurs*. 2015;42(1):62-64.
8. Gray M, Black JM, Baharestani MM, et al. Moisture-associated skin damage: overview and pathology. *J Wound Ostomy Continence Nurs*. 2011;38(3):233-241.
9. Woo K, Beeckman D, Chakravarthy D. Management of moisture-associated skin damage: a scoping review. *Adv Skin Wound Care*. 2017;30(11):494-500.
10. Beeckman D. A decade of research on incontinence-associated dermatitis (IAD): evidence, knowledge gaps and next steps. *J Tissue Viability*. 2017;26:47-56.
11. Voegli D. Moisture-associated skin damage: aetiology, prevention and treatment. *B J Nurs*. 2012;21(9);517-521.
12. Gray M, McNichol LL, Nix DP. Incontinence-associated dermatitis: progress, promises, ongoing challenges. *J Wound Ostomy Continence Nurs*. 2016;43(2):188-192.
13. Black JM, Brindle CT, Honaker JS. Differential diagnosis of suspected deep tissue injury. *Intl Wound J*. 2016;13(4):531-539.
14. Johnson J, Yates SS, Burgess J. Venous insufficiency, venous ulcers and lymphedema. In: Doughty D, McNichol L, eds. *WOCN® Core Curriculum: Wound Management*. Philadelphia, PA: Wolters Kluwer; 2016:383-419.
15. Carmel JE, Bryant RA. Venous ulcers. In: Bryant RA, Nix DP, eds. *Acute & Chronic Wounds: Current Management Concepts*. 4th ed. St Louis, MO: Mosby Inc; 2012.
16. Gefen A, Farid K, Shaywitz I. A review of deep tissue injury development, detection, and prevention: shear savvy. *Ostomy Wound Manage*. 2013;59(2):26-35.
17. Wilson M, Simman R. Unusual presentation of deep tissue injuries: a case series. *Wounds*. 2018;30(10):306-309.
18. Zaratkiewicz S, Whitney JD, Baker MW, Lowe JR. Defining unstageable pressure ulcers as full-thickness wounds: are these wounds being misclassified? *J Wound Ostomy Continence Nurs*. 2015;42(6):583-588.
19. Levine JM. Skin failure: an emerging concept. *J Am Med Dir Assoc*. 2016;17(7):666-669.
20. Alvarez OM, Brindle CT, Langemo D, et al. The VCU Pressure Ulcer Summit: The Search for a Clearer Understanding and More Precise Clinical Definition of the Unavoidable Pressure Injury. *J Wound Ostomy Continence Nurs*. 2016;43(5):455-463.
21. Langemo D, Brown G. Skin fails too: acute, chronic, and end-stage skin failure. *Adv Skin Wound Care*. 2016;19(4):206-212.

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