Skin Failure at End of Life

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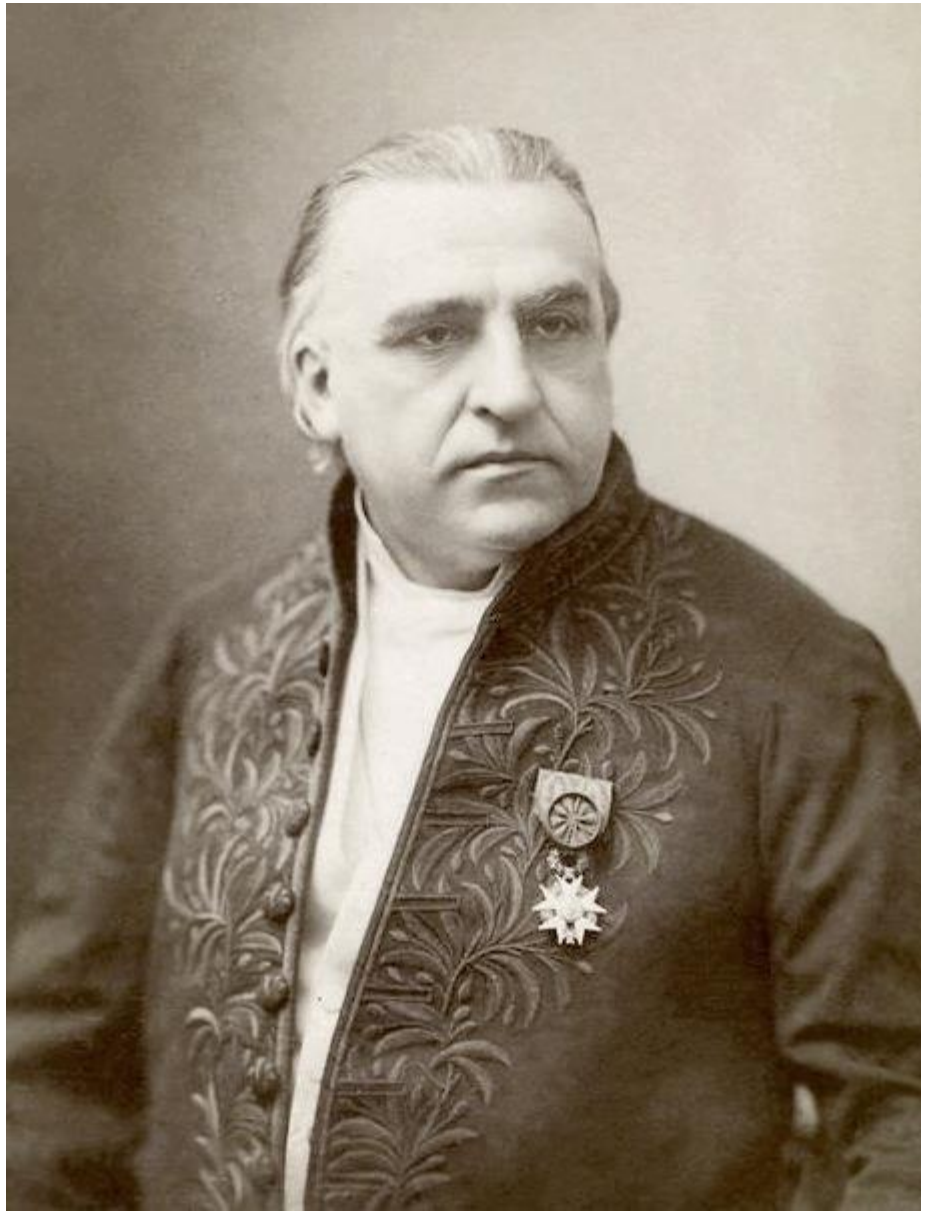
Skin: The Unique Body Suit

Reflects the:

- Ethnicity
- Physiological changes related to ageing & health
- Pathophysiological changes related to trauma or comorbid conditions


Egyptians 5,000BC



“Decubitus ominos”

“We have now…the principal circumstance under which acute bedsore is produced, under influence of lesions of the brain and spinal cord…”

A pear shaped lesion.

Neurotrophic ulcer

Jean-Martin Charcot 1825-1893

Pressure Injuries

Localised damage to the skin and/or underlying tissue, as a result of pressure or pressure in combination with shear. PIs usually occur over a bony prominence but may also be related to a medial device or object.

(NPIAP, EPUAP, PPPIA, 2019)

Pressure Injuries in Palliative Care

Pressure injuries 11.7%[1] to 40%[2] prevalence reported internationally

1. Ferris, A. (2019)
2. Degerli, MS. (2023)

When is a Pressure Injury Not a Pressure Injury?

Non-pressure related skin failure in the critically ill is defined as skin injury that occurs despite standard preventive interventions and for which no other etiology has been identified (NPIAP, August 2024)

Kennedy Terminal Ulcer

- Karen Lou Kennedy reported 55.7% of patients with a PI died within 6 weeks of onset
- Progresses to stage 3 or 4 within days to hours of death

Bilateral presentation:

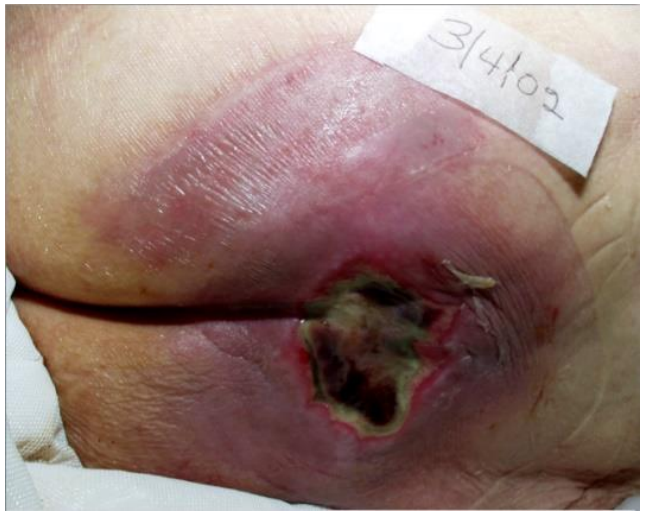
- Pear, butterfly or horseshoe shaped necrotic lesion on sacrum progresses to stage 3 or 4 within days to hours of death
- Usually on the sacrum
- Red, yellow, black or purple, irregular margins
- Sudden onset, occurs 2 weeks to several months prior to death

Unilateral presentation:

- 24-48 hours prior to death
- Black, purple, irregular margins
- Skin usually stays intact

'3.30 Lesion'

- 8-24 hours to death



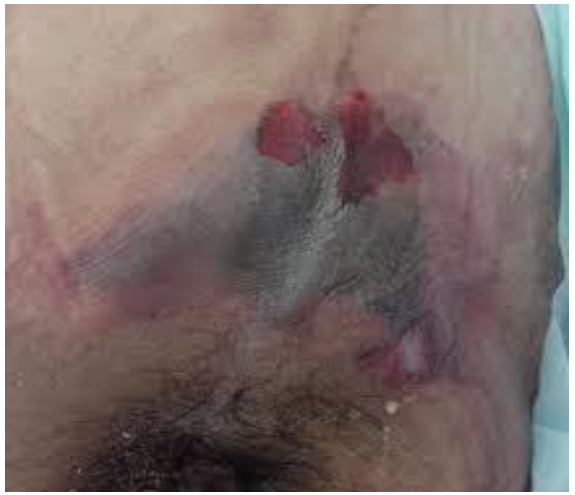


Photo courtesy D. Weir

Skin Failure

Langemo & Brown[1] hypothesized in 2006 that skin failure was attributable to hypoperfusion associated with severe organ dysfunction in critically or chronically ill or those near death.

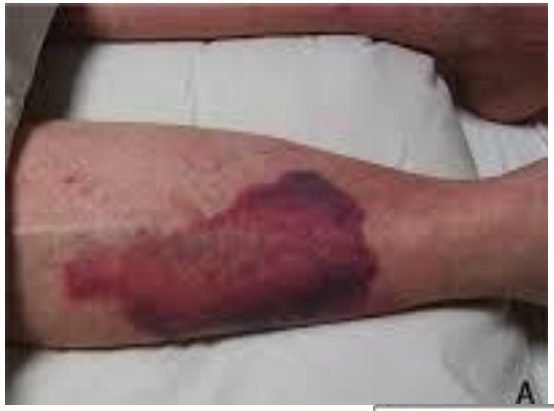
Tippett[2] postulated in 2005 that is the development of wounds as a person nears the end of life is an indicator of overall frailty.

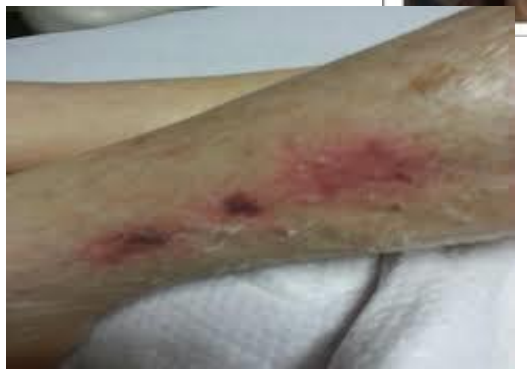
Trombley et al.[3] theorized in 2012 that skin discoloration was caused by death of the underlying tissue at end of life.

1. Langemo & Brown, (2006). Advances in Skin & Wound Care, 19(4), 206-211.
2. Tippett. (2005). Wounds, 17(4), 91-98.
3. Trombley et al. (2012). American J Palliative Care, 29(7), 541-545.

Tombley-Brennan Terminal Tissue Injury

- Lesions differed to KTU, but can be confused with SDTI
- Rapid onset, near end of life
- Purple-maroon-red skin changes, increasing in surface area
- Skin remains intact
- Appear on bony and non-bony prominences
- Commonly on sacrum but all anatomical locations
- Linear and mirror images may appear on lower extremities
- Not associated with pain or discomfort
- When the center of the wound blanched death was within 2 hours





(Trombley-Brennan, 2012)

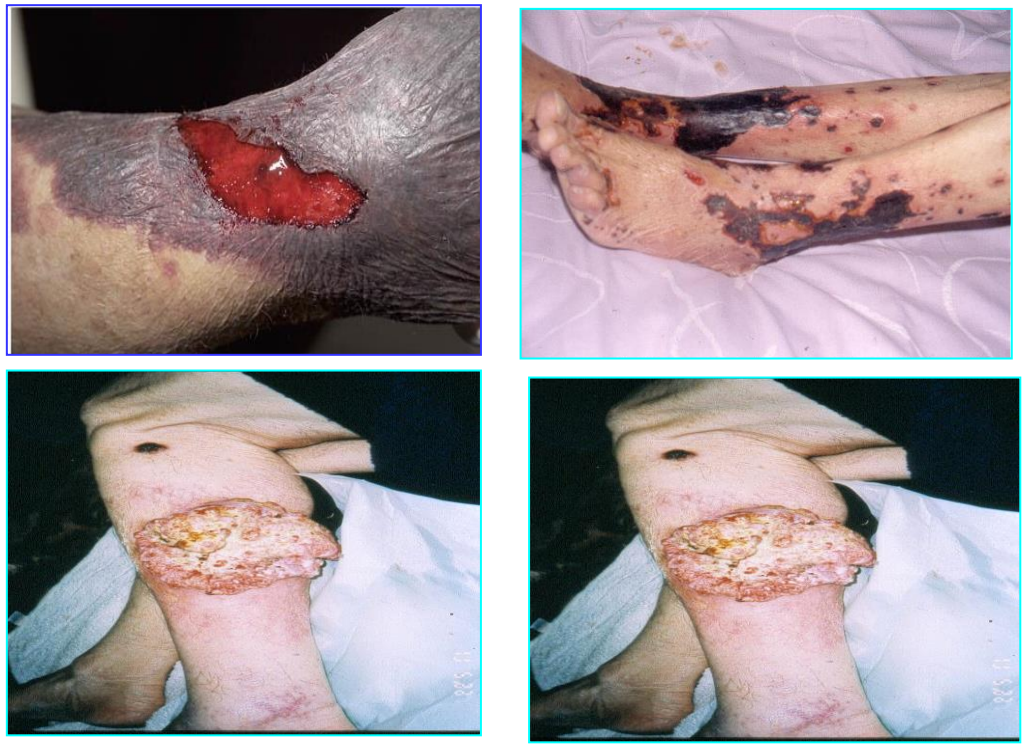
Skin Changes at End of Life (SCALE)

An international Delphi process. Physiological skin changes that may occur as a result of the dying process can be unavoidable regardless of standard of care interventions

Various wounds can result:

Cancer wounds
SDTI
Gangrene
Haemorrhagic wounds
Ischaemic wounds
Skin tears
Pressure injuries, KTU
Vasculitic ulcers
Wounds of unknown aetiology

(Sibbald, 2009)



PI or Skin Failure

Pressure Injury	KTU	TB-TTI
Pressure & shear	Hypoperfusion, organ failure	Hypoperfusion, organ failure
Over bony prominences	Sacrum, unilateral	Commonly sacrum, leg
Demarcated edges	Butterfly, horseshoe shape	Skin intact, lesion extends in size
Superficial to deep	Intact or S3 or S4	Red, maroon, purple
Gradual to sudden onset	Hours, weeks, months prior to death	Rapid onset near death

Contemporary Constructs

Melnychuk & Servetnyk (2024) proposed that skin failure occurs due to anatomic arterial aberrancies of the median sacral artery and lateral sacral artery leading to hypoperfusion and skin alterations.

Conclusion

A distinct pathophysiology for non-pressure related skin failure in the critically ill is not clear.

Based on limited evidence, hypoperfusion has been proposed to contribute to the pathophysiology of non-pressure related skin failure.

There are no formal diagnostic criteria at this point.

Research is needed to establish a reproducible description of the characteristic morphology and natural history of non-pressure related skin failure.

(Black, J. 2024)

References

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11. Trombley K , Brennan MR , Thomas L , Kline M. Prelude to death or practice failure? Trombley-Brennan terminal tissue injuries. Am J Palliat Care. 2012; 29(7):541-545 .
12. Sibbald RG , Krasner DL , Lutz J . SCALE: Skin changes at life's end: Final consensus statement: October 1, 2009. Adv Skin Wound Care. 2010; 23(5):225-236 .