REVIEW ARTICLE

The Death of the Kennedy Terminal Ulcer

Michael S. Miller, DO, FACOS, WCC*

CEO and Medical Director – Miller Care Group, Indianapolis, IN, USA

KEYWORDS:

Kennedy Terminal Ulcer;
3:30 syndrome;
Pressure injury;
NPUAP;
Miller pressure equivalent
injuries;
Pressure based tissue
injury

Abstract The concept of the Kennedy Terminal Ulcer (KTU) has been ubiquitous in attempting to explain the development of pressure based tissue injuries in patients with actual or presumed terminal conditions. The concept is problematic in that it uses factors other than pressure to explain the development and progression of pressure based tissue injuries, specifically the presence of a terminal condition. Based on the most current understanding of how pressure based tissue injuries develop and progress, the concept of The Kennedy Terminal Ulcer appears to be without physiologic basis and based solely on observation. Since systemic factors affect all tissues with relative equality, the development of a single locus of injury must logically be based on a single locus of cause and affect. The presumption that a single locus of injury will develop in an arbitrary location based on a systemic set of factors is untenable. A new concept called Miller Pressure Equivalent Injuries is proposed to refute the concept of a single pressure based tissue injury developing based solely on terminal systemic factors and why these previously presumed terminal condition associated pressure based injuries occur. © 2017 Elsevier Inc. All rights reserved.

The Kennedy Terminal Ulcer (KTU) is a term used to describe the development and rapid progression of a pressure based tissue injury in patients identified as being terminal or is thought to be itself, an indicator of terminal status. In 1989, the first description of this phenomenon was described by Mary Lou Kennedy NP in Fort Wayne, Indiana and published.¹ Additional considerations of this perceived phenomenon led to the name 3:30 Syndrome. This descriptive title was based on observations in which the a.m. evaluation of a given patient did not identify any skin issues but at 3:30 PM the skin (usually in the sacral region) showed evidence of injury, which progressed. The key findings were that this scenario presented in those with perceived terminal status and the subsequent life expectancy was found to be 8–24 h.

The development of a pressure based tissue injury (PBTI) in any setting is problematic. In today's enhanced medico-legal climate, the far-to-of knee jerk is that there was an error in either omission or commission of care that lead to development and progression. All factors are usually addressed but the mitigating factor hardest to consider involves simple human error(s). Documentation may not accurately depict the turning schedule, and ongoing observations of the skin integrity may not have occurred. Despite written policy and procedure mandates, required interventions may not have been ordered and implemented in a timely fashion. In those cases where the actions of the care staff and facility are related to the development of a pressure based tissue injury, then an answer must be sought, regardless of the surrounding evidence.

The concepts of the Kennedy Terminal Ulcer and 3:30 Syndrome have been used as explanations of why pressure based tissue injuries develop. Their use as a bulwark against litigation by well-intentioned physicians, nurses,

* Corresponding author.

E-mail address: doc@millercaregroup.com

and others was to argue that the development of a pressure based tissue injury was based solely on the presence of an actual or presumptive terminal condition.

On April 13, 2016 the National Pressure Ulcer Advisory Panel released a statement on their website (www.NPUAP.org) based on a consensus panel regarding pressure injuries occurring from localized damage to the skin and/or underlying soft tissue. These usually occurred over a bony prominence presenting as intact skin or an open PBTI. Additionally these injuries occurred as a result of intense and/or prolonged pressure or pressure in combination with shear.

In his Medscape review article entitled Pressure Injuries (Pressure Ulcers) and Wound Care, Kirman noted that the fundamental injury was related to pressure effects on blood flow at the microvascular level in the tissues.²

Gefen, in his two article series entitled Reswick and Rogers Pressure-Time Curve for Pressure Ulcer Risk. Part 1 and Part 2 noted "Tissues are capable of withstanding enormous pressures for brief periods, but prolonged exposure to pressures just slightly above capillary filling pressure initiates a downward spiral toward tissue necrosis and ulceration."^{3,4}

In addition to the effects of direct pressure over a bony prominence, the translation of pressure forces tangential to and along the planes causing friction (injury to the skin directly) and shear (inter-tissue plane disruption) further exacerbate the progression of injury. When these combine to cause microcirculatory occlusion, then the tissues develop injury progressing to ischemia and ultimately to infarction. This continuum of tissue damage manifests as inflammation, necrosis and then ulceration. However, with position changes after a prolonged time in one position, reperfusion injury occurs. In the 2012 white paper from the NPUAP, the panel identified the causes of reperfusion injury. They felt that long periods of ischemia caused direct damage and shorter periods caused indirect damage via reperfusion injury.⁵ There appears to be sufficient evidence to support a direct link between damage from prolonged pressure and that caused by reperfusion when pressure forces are relieved.

The ultimate question becomes; what is the relationship between presumed terminal status only and the development of a pressure based tissue injury? The concept of Kennedy Terminal Ulcer relates only to presumed terminal status as the cause of the development of a pressure based tissue injury without respect to pressure. The presumption that a terminal condition alone will result in a pressure based tissue injury despite appropriate care (including turning, appropriate support surfaces, management of incontinence, etc.) is simply not a viable consideration based on the current understanding of the pathophysiology of how pressure based tissue injuries develop.

The concept behind the development of a Kennedy Terminal Ulcer cannot exist as such. Conceptually, the human body has two ways that a given stimuli or stressors can affect it. The first is a systemic physiological affect. No

leap of faith is required to understand how the compromise of a given system (Cardiac, Respiratory, Endocrine, Renal, etc.) affects the entire body. While it may have a specific target organ, the effects on that target organ(s) and the effects that target organ manifests are systemic and thus "body-wide". In simplest terms, systemic diseases have an equal effect on all body tissues, although that effect manifests differently depending on the tissue. For example, systemic effects of osteoporosis affect all bony structures equally; systemic effects of emphysema affect oxygenation and respiration, which affects all tissues equally, etc.

The other consideration for a stressor is local. The concept of pressure is the main consideration. Unless there is complete envelopment of a body surface in a contacting medium, there is pressure placed on specific areas, a phenomenon I call Point Pressure. Where there are bony prominences, pressure will be greater over those areas due to less area over which the pressure can be spread. A given area of the body may have thicker or thinner skin, more or less subcutaneous fat, and thus be more or less prone to the effects of point pressure, friction and shear. The presentation and timing of pressure at that pressure point causes an effect specific to that area and not whole body/systemic wide.

The next consideration mitigating the concept of the Kennedy Terminal Ulcer has to do with the developmental scenario itself. If one understands the concepts of systemic conditions in contrast to specific point pressure, then the development of a "Terminal Ulcer" becomes even more illogical. At its most basic, the KTU develops in an area (usually the sacrum) that has the same systemic stressors as every other tissue. If indeed a turning schedule is utilized in which pressure, friction and shear are equally distributed over all available surfaces, then the expectation is that all tissues will have essentially the same pressure effects exerted for the same amount of time and thus the effects of that pressure are distributed uniformly over all potentially affected tissues. And yet, according to the concept of the Kennedy Terminal Ulcer, only a single given area develops an injury, which then progresses over an approximate 8-h period.

Regardless of any factors other than terminal status, there must be some undefined and yet purposeful mechanism that decides which area will break down without respect to any other factors. It is as though the human brain arbitrarily and with conscious thought decides that a given area will lose its integrity and become injured. Further, this occurs without consideration of any other factors other than the presence of an actual or presumed terminal condition.

The more logical phenomenon is that a specific area affected by systemic conditions, become stressed by specific point pressure (as well as localized friction and shear forces). Regardless of the existence of a terminal systemic condition, that specific area now has received a stress not affecting any other area. That area responds to this insult and a pressure based tissue injury develops and progresses.

This scenario now makes it problematic for medico-legal and standard of care situations in which the development of a pressure based tissue injury is defended as due to a terminal condition (The Kennedy Terminal Ulcer) and not from any other care factors or lack thereof.

We propose a more scientific, logical scenario based on the current understanding of how pressure based tissue injuries develop including in those with a terminal condition. If systemic diseases have systemic affects and pressure is assumed to be applied equally and for approximately equal times, then there must be manifestations of these affects identified on all tissues and surfaces so affected. If a patient is positioned approximately every 2 h onto all available body surfaces, the same pressure forces and timing are equal on these areas. Thus, factors causing pressure based tissue injury should be equal over all these same areas. If a pressure based tissue injury is identified at any given area, then there must be pressure based tissue injuries identified at all these areas. This does not propose that what is currently termed a Stage 4 pressure based tissue injury at a given area will occur on all other areas but rather, there must be some evidence of the effects of this pressure on all other areas. Finding a deep tissue injury on the Sacrum associated with a Stage 2 pressure based tissue injury of the right hip, a stage 1 PBTI 1 of the left buttock with a Stage 3 PBTI of the right buttock, etc. unquestionably resolves the incongruence of the concept of The Kennedy Terminal Ulcer. The presence of multiple areas of pressure based tissue injury logically connects systemic affects, local effects (point pressure, friction, and shear) and any interventions provided to all areas uniformly. Regardless of the severity of the systemic stressors, interventions that are timely, consistent and commenced early at areas prone to pressure based tissue injury should either mitigate injury or in a worse case scenario, result in essentially uniform injuries at all involved surfaces. This *Miller Pressure Equivalent Injuries (MPEI)* scenario explains how pressure based tissues injuries must be present if an argument for uniformity of care for the entire patients skin and body is to be accepted, regardless of any terminal or systemic conditions. Terminal status becomes another systemic stressor instead of a definitive cause for pressure based tissue injuries.

A single article from 1989 suggested the relationship between presumed terminal status and development of tissue injury and death. The issues identified in this paper demonstrate incongruence between the current scientific thought on causality of pressure based tissue injuries and the suggested relationship to the presence of a terminal condition without respect to any other factors. More, the use of the concept to definitively state that there are patients who will go on to develop a single pressure based tissue injury based solely on the systemic effect of a terminal condition is no longer a viable consideration.

No longer can we blindly believe and accept that there are factors beyond our control with respect to the development of pressure based tissue injuries. Despite purported adherence to accepted risk reducing interventions, the most logical conclusion when a solitary pressure based tissue injury is identified is that uniformity of care did not occur. The Kennedy Terminal Ulcer represents concepts not based on current, objective scientific principles. It is not scientifically valid and while it provides a unique historical perspective, it must be discarded as a scenario under which pressure based tissue injuries occur for reasons other than related to pressure.

There must be further evaluation of the interrelationship proposed between the 1989 observation of pressure based tissue injuries, terminal status and timing of ultimate demise. Retrospective studies in which all three parameters are noted and correlation with Pressure based Tissue Injuries will go a long way towards connecting these dots. With the current nationwide attention on hospice and palliative care and use of electronic medical documentation by entities engaged in these types of care, the data further refuting or supporting the existence of a relationship may yet be encountered.

References

1. *Adv Skin Wound Care* (Formerly *Decubitus*). May 1989;2(2):44–45.
2. <http://emedicine.medscape.com/article/190115-overview>.
3. *Nurs Stand*. 2009 Jul 15–21;23(45). 64, 66, 68.
4. *Nurs Stand*. 2009 Jul 22–28;23(46):40–44.
5. <http://www.npuap.org/wp-content/uploads/2012/01/DTI-White-Paper.pdf>.