It's humbling to have to comment on one’s own paper 25 years after it was written, particularly when the subject matter includes the discussion of what we now refer to as deep tissue injury (DTI). Twenty-five years ago, there was the excitement of publishing in the first issue of a new journal, then known as Deciduous; founding a new organization in the National Pressure Ulcer Advisory Panel (NPUAP); and working with the NPUAP to add pressure ulcer (PrU) taxonomy to the Omnibus Budget Reconciliation Act of 1987. All of these seminal accomplishments were memorialized through the journal now known as Advances in Skin & Wound Care.

Looking back 25 years ago, we didn’t yet fully appreciate the relative importance of DTI as a co-contributor of PrUs. In 1988, I was compelled to write an article about the concept of pressure-induced injuries to the deep tissues of the body that occurred during long surgical procedures. Until this time, the term DTI had not been proposed in relationship to PrUs, nor was the term in use by the NPUAP or the Wound Ostomy and Continence Nurses Society, and the European Pressure Ulcer Advisory Panel didn’t exist yet!

I wish that I could tell you that, at that time, as I wrote about wounds or ‘lesions’ that were being described as ‘burns’ postoperatively, I knew of the relative importance of what is now known as DTI. But back then, I didn’t realize the importance of DTI in the PrU puzzle. More important, I didn’t realize how advances in research would contribute to our understanding of the development of PrUs and thus finally conquering them. And by conquering them, I mean first diagnosing them correctly and then preventing the vast majority of them. As others have observed, in more than 2 decades, the point prevalence and incidence of PrUs have not changed dramatically. This leads us to reconsider the pathogenesis of PrUs; I think this would be wise. The current literature suggests that our understanding of causation may be faulty. As we move into a new area of healthcare, and given significant gaps in our DTI acumen, I propose we reconsider what we think we know about DTI and vigorously pursue new translational research and clinical care models.

In my initial article 25 years ago, I posited that the postoperative “closed PrU” was a special case of the “closed pressure sore” described by Shea, with damage caused primarily at the level of the subcutaneous tissue and/or muscle of the surgical patient under anesthesia. It hadn’t dawned on me that this may be the mechanism for the formation of most Stage III and IV PrUs, but it was certainly where I was first introduced to the thought.

The wound care community may find it interesting that in 1988 and earlier the surgical, anesthesia, critical care, and intensive care unit staff often tended to mistake what we now refer to as DTIs for burns. However, if you follow the progression of a DTI, it is not uncommon for it to go through a process that would resemble that of a burn. And after the fact, particularly if there are blisters or eschar present, it is often impossible to distinguish one wound from the other. What the wound care community may find surprising, however, is that today, there are still a number of healthcare professionals who classify DTIs as burns.

In a quarter of a century, relative to DTI, what has changed and what new perspectives are there? A further understanding of DTI is developing through the combined efforts of serious investigators, clinicians, industry leaders, and wound care organizations.

Advancing DTIs from scholarly discussions to scientific debate and then to clinical practice keeps the DTI agenda in the forefront. In 1994, Salcido et al were the first to establish an oxygen-free radical pathophysiological basis for DTI. Their team demonstrated that, in response to sustained pressure, the oxygen-free radical destruction of the muscle is primary. They confirmed that the muscle tissue (deep tissues) is more sensitive to ischemia and reperfusion injury than the skin. In 1995, Salcido et al also analyzed sequential histopathological changes that occurred in the development of PrUs experimentally induced in the fuzzy rat model. Computer-controlled pressure was applied for 6 hours at a maximum of 5 sessions to skin over the greater trochanter of the surgical patient under anesthesia. It hadn’t dawned on me that this may be the mechanism for the formation of most Stage III and IV PrUs, but it was certainly where I was first introduced to the thought.

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recurrant pressure results in increasingly severe damage to the vascular system and parenchyma, consistent with an ischemia/reperfusion insult, initiated through a free-radical mechanism. More recently, Salcido et al. described the phenomena as a myosubcutaneous-dermal infarct. In other words, the deeper structures are the most sensitive to injury, and the lesion at the skin is just the tip of the iceberg.

We now need to continue to push the understanding of DTI into clinical practice. Certainly, a more profound understanding of DTI and reperfusion injury is key to understanding the true development, progression, and clinical course of PrUs. This knowledge may ultimately lead us to their prevention and eradication. However, new insight is needed if we are to make any progress. We must be willing to challenge some of our present beliefs and dogmas on the subject of staging and causation.

The understanding of DTI and moving beyond the standard clinical model of PrU staging are appropriate. Refocusing on the model of DTI—there exists a deep zone of infarction or inverted cone of injury that has yet to manifest visually as a “Stage I.” This DTI is certainly beneath or subserosally under the skin—this DTI is a serious ulcer emanating from DTI or what we now call Stage III or Stage IV. The DTI-focused model puts the focus on the early detection and prevention of DTI, not the prevention of Stage I and II PrUs. This may sound counterintuitive; however, I’m not advocating neglecting Stage I and II PrUs. Stage I and II PrUs need to be treated and prevented. I’m just opening my mind to the thought that Stage I and II PrUs are most likely not the precursors of most Stage III and IV PrUs. And that we have been diverting our attention away from DTI and that our surveillance and early intervention systems should be focused not on superficial insults to the skin but to insults that can cause harm to the more susceptible deeper tissues. This should be the first line of defense.

The prevention and treatment of PrUs by pressure relief should not necessarily match the particular stage. Variable pressure relief and distribution for a DTI should be the same as for all other stages, including a Stage IV.

Twenty-five years of work in this field have caused me (like many of you) to reflect on some of the basic “truths” or clinical practices passed on uncritically from one generation to the next. In terms of the pathogenesis and the clinical presentation of PrU development, we should challenge the “truths” through disruptive innovation. We need to take advantage of all the technological advances to detect and treat the DTI before it manifests to the clinical eye and is relegated to a staging system, for the sake of the patient. I challenge you, the new generation of clinical wound specialists, to remember the past but embrace the future.

References