undermining and tunnelling.

Unstageable

Full thickness tissue loss in which the base of the ulcer is covered by slough (yellow, tan, grey, green, or brown) and / or eschar (tan, brown or black) in the wound bed.

The NPUAP's definitions of pressure ulcers arise from clinical experiences with them and are largely based on the ulcer's appearance after they have formed and do not necessarily reflect the true aetiological factors that lead to these conditions. For example, a significant body of literature scientifically describes deep tissue injuries as being much more insidious than a "localized area of discoloured intact skin" and suggests that many Stage III and IV pressure ulcers are actually advanced deep tissue injuries rather than advanced Stage I or II ulcers [17]. This chasm between the clinically accepted and scientifically observed definitions of deep tissue injuries is likely due to the lack of any clinical detection ability [29]. What is agreed upon is that deep tissue injuries are a major problem and more needs to be done to facilitate preventing and treating them [30], [31]. One of the largest hurdles to preventing and treating DTI is the lack of any substantial early detection ability [32], [33].

2.1.1 Aetiology and Histology

Deep tissue injuries are thought to occur through the combinatory effects of three distinct but related mechanisms: ischemia, insufficient lymph drainage, and cell deformation. Ischemia is a condition where the blood supply to tissue has been cut off, rendering the tissue unable to function appropriately. Insufficient lymph drainage refers to how waste products may accumulate in

tissue when the lymph vessels that normally carry them away become occluded. Cell deformation occurs when mechanical strains are imparted upon the tissue, causing excessive deformation in not only the extracellular matrix, but in the cells as well. Taken together, the presence of these factors has been shown to greatly increase the risk of developing a deep tissue injury [4].

For quite some time, ischemia was regarded as the chief acute risk factor for developing late-stage pressure ulcers [34]–[36]. Although studies have shown that healthy tissue is able to survive complete ischemia for approximately 4 hours before severe necrosis sets in [37], [38], deep tissue injuries are clinically found when loading times are substantially less than this [25], [39]. The model of ischemic damage alone could not account for the rate of late-stage pressure ulcers that we were witnessed.

Once it was realized that ischemia alone could not be the culprit behind deep tissue injury formation, ischemia-induced reperfusion injury became implicated in the formation of DTI [40]–[42]. An ischemia-induced reperfusion injury is caused when blood is allowed to flow back into a region of tissue that was previously ischemic. While seeming somewhat contrary to its expected effect, the restoration of circulation results in a swelling and inflammatory effect which causes extensive microvascular damage [41]. The effect of reperfusion was confirmed when comparing pure ischemic conditions in tissue to a cycle of ischemic-reperfused conditions over the same period of time, where it was found that significantly greater damage was caused by repeated loading and unloading rather than simple constant loading [42], [43]. While ischemia-reperfusion injuries provide a more complete explanation about the formation of deep tissue injuries, they still do not account for those injuries acquired under constant pressure over short time periods.

In order for cells to function in a healthy manner, the waste they produce must be constantly carried off and processed via the lymphatic system and its series of lymph vessels that perfuse tissue. If the magnitude of pressure applied to tissue reaches a threshold level, the pressure occludes the lymph vessels and lymphatic drainage ceases [44]. Once lymphatic drainage ceases, cell waste accumulates in the tissue and is thought to initiate necrosis in the cells [45]–[47].

In order to account for deep tissue injuries that form over short time periods, a model of cell deformation leading to necrosis has more recently been proposed [48]–[50]. It has constantly been observed that tissue regions which eventually form deep tissue injuries exhibit signs of locally increased strains [10], [16], [51]–[53], with greater degrees of deformation correlating to greater degrees of damage. To account for these results, it has been proposed that excessively deforming strains applied to cells over extended periods of time can alter the permeability of the cell's plasma membranes, leading to an overall reduced cell viability [54]. Further, it has been shown both in finite-element models and experimentally that the stiffness of soft tissue and the corresponding strains that are developed within them are closely related [5], [55]–[57]. Not only does the amount of deformation depend on the stiffness of tissue, but the stiffness of tissue was found to correlate to the level of deep tissue injury damage seen in the resulting histology [58] with immediate 1.6-fold to 3.3-fold stiffening of the tissue occurring immediately after injury [5], [15]. Further, the stiffness of tissue severely drops below that of healthy tissue when it begins to decompose [5], [59], leading to a relationship between injury progression and stiffness as shown in Fig. 2.2 (adapted from [17]).

There have been many models of deep tissue injury formation throughout