Are All Pressure Ulcers the Result of Deep Tissue Injury?
A Review of the Literature

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Pressure ulcers are a common problem that significantly contributes to morbidity and mortality. To elucidate the confusion surrounding the origin of pressure ulcers, the question of whether pressure ulcers are caused exclusively by deep tissue injury is addressed. A review of the literature relevant to the pathophysiology and pathogenesis of pressure ulcers is presented and focuses on studies that examine the relationship between mechanical stresses and deep and superficial tissue injury. The studies suggest that deep tissue is more susceptible than superficial tissue to injury caused by externally applied pressure; clinically superficial skin injuries induced by pressure tend to be associated with deep tissue damage; and superficial injuries appear to be caused by factors other than pressure. Based on these observations, pressure ulcers are believed to be the result of deep tissue damage, implying that prevention and treatment of superficial lesions need not necessarily conform to pressure ulcer management that makes eliminating pressure the highest priority. Conversely, the treatment of pressure ulcers should account for the likelihood, even if not visually noted, that deep tissue is involved.

KEYWORDS: pressure ulcers, spinal cord injuries, deep tissue injury


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Pressure ulcers are a common problem that significantly contributes to morbidity and mortality among patients with spinal cord injury and other chronic conditions. Pressure ulcer occurrence is frequently viewed as a medical error; efforts to improve pressure ulcer preventive care are a national priority. However, for such efforts to succeed, a comprehensive understanding of the pathogenesis of pressure ulcers is necessary. As emphasized in one editorial on pressure ulcers, “a strategy designed to limit or solve a problem requires, first and foremost, a clear understanding of the nature and underlying causes of the problem.”

Although understanding of pressure ulcer pathogenesis has advanced considerably, confusion still exists among many clinicians. Organizations such as the National Pressure Ulcer Advisory Panel (NPUAP) are examining new data as they grapple with issues such as what defines a superficial pressure ulcer, what constitutes deep tissue injury, and whether the currently used staging system adequately reflects current knowledge.
Traditionally, pressure, shear, friction, and moisture were the major external factors considered to have a causative role in pressure ulcer development. However, these factors need to be re-examined and factors such as heat, stress, strain, and reperfusion injury considered. Data on pressure ulcer pathogenesis are reviewed herein with regard to an important question in the management of patients with spinal cord injury: Are all pressure ulcers the result of deep tissue injury?

In order to comprehensively address this question, three corollary questions need to be discussed: 1) what is the nature of pressure-induced tissue injury? 2) are clinically superficial skin injuries induced by pressure associated with deep tissue damage? and 3) if not pressure, what causes most superficial ulcers? A review of the relevant literature was conducted to determine whether all pressure ulcers result from deep tissue injury.

**Pressure and Tissue Injury**

How does pressure damage tissue and which tissues are most susceptible to pressure-induced damage? To address this, it is necessary to discuss the fundamental pathophysiological cause(s) of pressure ulcers. The four most commonly hypothesized pathophysiological explanations for the development of pressure ulcers include: 1) ischemia caused by capillary occlusion; 2) reperfusion injury — ie, injury resulting from the accumulation of substances associated with the inflammatory response to ischemia as blood is reintroduced into an ischemic region; 3) impaired lymphatic function that causes metabolic waste products, proteins, and enzymes to accumulate; and 4) prolonged mechanical deformation of tissue cells. Most likely, all four are contributing factors. Regardless of their relative importance, each of the aforementioned factors is fundamentally caused by stress (pressure) and strain (deformation) in soft tissue. The question regarding how force exerted on the skin affects the underlying tissues and subsequently increases risk of pressure ulcer development can be logically simplified to a contemplation of the external forces that cause stress and strain in underlying soft tissue. It is useful to consider that the forces acting on the skin are reactive, countering the gravitational forces that pull the body toward Earth. These gravitational forces are concentrated at various posture-dependent, weight-bearing bony prominences such as the heel, sacrum, and pelvis. The tissue at risk for pressure ulcer development is soft tissue (muscle, fat, connective tissue, and skin) between these bony prominences and any external cushioning device (eg, a seat cushion, mattress, or shoe).

One intuitive but overly simplistic description of how forces are transmitted through soft tissue is that the gravitational force pulling the body down through the skeletal bone is first concentrated in a relatively small volume at the interface between bone and soft tissue immediately surrounding the bone, which is then subsequently diffused (spread out) over larger and larger volumes as the force is transmitted through soft tissue down toward an external supporting device. The more widely the forces are diffused, the lower the stress on tissues involved. The foregoing description is overly simplistic because the exact nature of how the forces are distributed depends on the combined mechanical properties and geometry of soft tissues and the external devices. Small stiff elements tend to cause areas of high stress (ie, stress concentrations). Soft compliant elements tend to have large deformations (ie, high strain). To complicate the task of predicting stress and strain distributions, soft tissue has been shown to have time- and loading history-dependent mechanical characteristics and can change based on host physiological responses. Both stress and strain are believed to increase the risk of soft tissue injury.

Mathematical models can predict the distribution of stress and strain in at-risk soft tissue; thus, valid models should be able to predict where stress and strain are greatest and subsequently where pressure ulcers are most

**KEY POINTS**

- A review of currently available evidence suggests that, unlike visible and invisible areas of deep tissue injury, superficial injuries are not caused by pressure.
- The implications of this observation for research, patient care, and costs of care are substantial.
- Clinicians must take current evidence into consideration. Research to confirm its potential clinical significance is urgently needed.
likely to develop. Over the past 30 years, analytical modeling techniques and techniques for measuring anatomical geometries have improved along with knowledge of soft tissue mechanical properties. These advancements have allowed researchers to construct more and more accurate models describing how stress and strain are distributed through soft tissue. Hence, several research groups have developed tissue-loading models to study pressure ulcer development. The analytical studies discussed show that stress and strain are critically severe in deep tissue before or in the absence of critically severe loads in the superficial tissue.

Chow and Odell constructed a linear finite element (FE) model derived from geometric primitives to study the effect of different cushions on stress and strain distribution. They found that stress and strain were highest on the interior of the buttock model and not on the surface. Todd and Thacker developed a linear FE model using geometry obtained from MRI scans of supine humans and confirmed Chow's results. Oomens et al created a non-linear FE buttock model with separate skin, fat, muscle, and bone layers and showed that stress and strain were highest in the muscle layer near the bone and high stress was created in the fat layer between the bone and skin. A potentially important result of this study was that interface stress (pressure) could be reduced by using a soft external cushioning material but stresses and strains in deep muscle tissue remained at high levels. Recently, Linder-Ganz et al created “subject-specific” models of buttock tissue in its naturally loaded state in a sitting human using an open-frame MRI and also confirmed that stresses and strains were highest in the deep muscle layers near the bony prominence.

The results of these mathematical model-based studies are consistent with other types of studies that have shown that externally applied pressure results in high stress primarily in deep tissue and secondarily in superficial tissue. Furthermore, animal model and clinical studies have shown that deep muscle tissue appears to be more susceptible to pressure damage compared to superficial skin and fat. Nola and Vistnes developed an experimental rat model and protocol to study the relative sensitivity to pressure-induced tissue damage of sites consisting of skin and bone versus sites consisting of skin, muscle, and bone. Although the introduction of a transposed muscle flap or the pre-existence of muscle tissue reduced the incidence of cutaneous ulcers, histological examination of non-cutaneous ulcer cases revealed muscle damage in the absence of cutaneous damage “in almost every case.”

Although the focus has been on pressure, it is important to recognize that pressure interacts with other factors to contribute to tissue injury. Porcine models have demonstrated that heat accumulation resulting in abnormally high skin temperature may be an important factor leading to pressure ulcer development. However, the adverse affect of higher temperatures is fundamentally ischemia-related because elevated temperatures increase metabolic rates and subsequently increase O\textsubscript{2} consumption rates. In other words, elevated skin temperatures exacerbate the effects of ischemia by increasing the need for O\textsubscript{2}. Interestingly, these studies of the effects of heat on pressure ulcer development in swine support the hypothesis that deep tissue is more sensitive to external pressure. As skin temperature increased with pressure held constant, histological analysis of the resulting wounds showed that deep tissue damage more commonly appeared for lower skin temperatures than for superficial tissues. Experts also suggest that circulation may be an important contributor to tissue injury, particularly in critically ill patients. Where perfusion is decreased (such as from hypotension, shock, or dehydration), blood flow to skin is likely to be compromised, increasing ischemia. Deep tissues, with their extensive vascular supply, may be particularly vulnerable.

In summary, these studies all suggest that externally applied pressure will increase pressure and damage in the deep tissues near bony prominences before causing damaging superficial tissues near the skin surface. The resulting deep tissue injury, as emphasized by the NPUAP in their 2007 updates to the pressure ulcer staging system, may initially appear as “purple or maroon localized areas of discolored intact skin or blood-filled blister due to damage to underlying soft tissue from pressure and/or shear. The area may be preceded by tissue that is firm, mushy, boggy, warmer or cooler as compared to adjacent tissue.”

**Pressure and Superficial Injury**

How, then, is pressure associated with superficial injury? A recent NPUAP systematic review of clinical and histological studies in human and animal models...
has shown that external pressure may result in deep tissue injury with intact overlying skin. This clinical observation was described by Paget as early as 1873. Animal studies have not convincingly demonstrated an important role for pressure in superficial dermal injuries. Salcido studied histopathological changes occurring in a rat model following repeated application of controlled pressure and found that lesions developed first in muscle then in the skin. These studies involve application of an external load, including pressure and shear, to soft tissues for a specified period of time with follow-up histological examination. Both superficial and deep injury may result depending on the characteristics of the load. However, as emphasized by Bouten et al., both animal and clinical studies suggest that superficial lesions are caused mostly by the application of shear forces. Studies in porcine models cited as demonstrating early pressure-induced changes in the dermis did not evaluate deeper structures, ensuring that questions remain as to the co-existence of deep tissue damage.

Moreover, studies in humans that have involved full-thickness punch biopsies have shown that lesions characterized clinically as Stage I ulcers may already demonstrate damage to deeper layers. Biopsies show a normal epidermis, dermal capillary and venule engorgement and hemorrhage, and necrosis within the subcutaneous fat. More advanced pressure ulcers demonstrate continued trauma to the microcirculation including compression-induced occlusion of blood vessels and shear-induced tearing of blood vessels that causes microvascular hemorrhages.

**Superficial Skin Injury**

Pressure ulcers are defined as 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. This would suggest that superficial lesions caused mainly by friction or maceration that only affect superficial tissue are not pressure ulcers. In fact, a variety of different dermal lesions may occur in patients with spinal cord injury and other chronic conditions. Skin maceration is a common result of urinary or fecal incontinence. Superficial erosions may develop from friction when an immobile patient is dragged in bed. Shear forces also may cause blood vessels to stretch and tear, resulting in non-blanchable erythema. Commonly, these lesions will occur over a bony prominence. Often, the inclination is to call these lesions pressure ulcers because they are occurring in patients at-risk for ulcers and at locations typical for a pressure ulcer. However, because pressure ulcers by definition are pressure-induced lesions, it is believed that these non-prolonged pressure-related dermal injuries should not be called pressure ulcers.

The importance of not labeling these lesions “pressure ulcers” is widely recognized among experts in the field. At the 2005 NPUAP Consensus Conference, the strongest point of agreement among expert respondents was that the definition of a Stage II pressure ulcer should be changed to specifically exclude non-prolonged pressure-related injuries such as from friction or moisture. Such mislabeling only engenders confusion among clinicians. Although no epidemiological research or clinical studies describing the extent of such mislabeling exists, experience suggests that many superficial lesions are inappropriately labeled pressure ulcers.

Collectively, these considerations suggest that caution is required in ascribing superficial injuries to pressure. Most dermal lesions are related to other factors such as friction and moisture, not pressure. Lesions that appear superficial and are caused by pressure are likely to have evidence of damage to deeper tissues.

**Conclusion**

Clinicians need to have a clear understanding of the causes of pressure ulcers to improve care. Considerable confusion remains as to what pressure ulcers are and how they develop. A growing body of evidence suggests that most pressure ulcers are the result of deep tissue injury. Specifically, 1) pressure-induced injury mostly may affect deeper tissues; 2) lesions that appear clinically superficial and induced by pressure are usually associated with deeper injury; and 3) most lesions that are truly superficial are caused by friction and moisture, not pressure. The clinical significance of this conclusion is that pressure need not be the priority in the prevention and treatment of superficial lesions. Conversely, the treatment of pressure ulcers should account for the likelihood that deep tissue is involved even if such involvement is not apparent during visual assessment. It is important for clinicians to be aware of this evidence
so they can implement practices that will have the highest likelihood of success in preventing and treating pressure ulcers. Future clinical practice will need to be guided by additional research, both clinical and basic science, that helps elucidate the roles of multiple factors in causing dermal injury. - OWM

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