Bruin Biometrics, LLC (BBI) is pleased to provide this series of white papers to help lay readers understand commonly debated topics in pressure ulcer research and clinical practice. These papers are the distillation of a comprehensive literation search and review, rather than the result of primary research.

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White Paper

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Subepidermal Moisture: An Early Indicator of Tissue Damage and Pressure Ulcer Development

Education	Evidence	Evaluation

Subepidermal Moisture: An Early Indicator of Tissue Damage and Pressure Ulcer Development

Introduction

A pressure ulcer, or bedsore, is localized injury to the skin and/or underlying tissue usually over a bony prominence, such as a heel or an elbow.[i] This injury is caused by the inability of the skin and the supporting structures to redistribute the pressure that is exerted by forces of mechanical loading, friction, and shear (parallel force)[ii]. This excessive force threatens dermal tissue health by causing the collapse of small blood vessels, the impediment of arteriolar circulation, and the reduction of lymphatic flow to the affected area, all of which result in tissue edema (swelling) and subsequent tissue death. Once a pressure ulcer develops, very little can be done to prevent additional tissue loss or heal the ulcer. Pressure ulcers are also difficult to heal. Although more than 70% of Stage II ulcers heal after six months of appropriate treatment, only 50% of Stage III ulcers and 30% of Stage IV ulcers heal within this period.[iii] For that reason, prevention is imperative. However, current detection techniques that could facilitate prevention are inadequate.

Pressure Ulcer Formation: Pathophysiology

The skin is the largest organ in the human body. Together, the skin's three layers (see Figure 1)[iv] – the epidermis (uppermost layer), dermis, and hypodermis (innermost layer) – play a vital role in acting as a barrier that protects the body from the invasion of surface microorganisms that can cause systemic infection. The stratum corneum, or outermost layer of the epidermis, maintains and allows the barrier function of the skin, limiting the body's loss of water.[v]

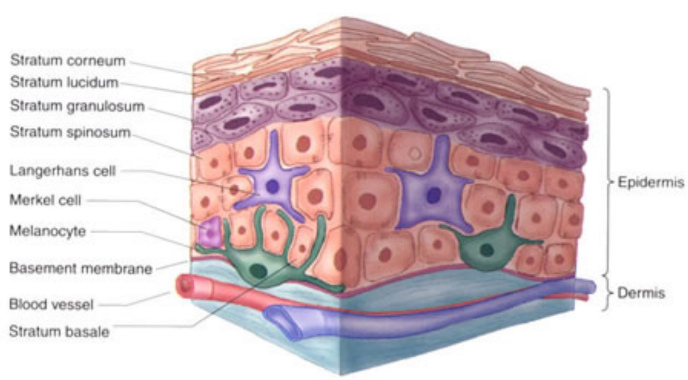

Figure 1: Stratum Corneum, Epidermis, Dermis[vi]

Mechanical forces resulting from a patient's bodyweight applied to tissue and bony prominences induce a series of physiological changes beneath the skin's surface. A pressure ulcer injury ultimately forms when the skin and the supporting tissues are unable to redistribute this external pressure.[ii] This pressure causes the collapse of small blood vessels, the impediment of arteriolar circulation, and the reduction of lymphatic flow to the affected area. The traditionally quoted pressure threshold value that will induce capillary closure and tissue damage is 32mmHg (4.27kPa) or equivalently half a pound per square inch.[vii,viii] This capillary occlusion causes ischemia, restriction of blood supply to the tissue. According to the "Ischemia Hypothesis", the tissue is then deprived of oxygen, nutrients, and heat normally delivered by the blood. Consequently, the cells become hypoxic and subsequently necrotic.[ix] The impeded circulation then leads to an accumulation of potentially deleterious metabolic waste products that are not cleared by the circulatory system.

In response, inflammation, the body's first response to tissue damage (see Figure 2), presents in the form of localized tissue edema. Its purpose is to localize and eliminate the injurious bodies and to remove damaged tissue components so that the body can begin to heal by increasing the permeability of blood vessels, as well as the migration of fluid, proteins, and white blood cells (leukocytes) from the circulatory system to the site of tissue damage. Therefore, as the level of skin damage increases so does the level of moisture present within the subepidermal tissue.[x]

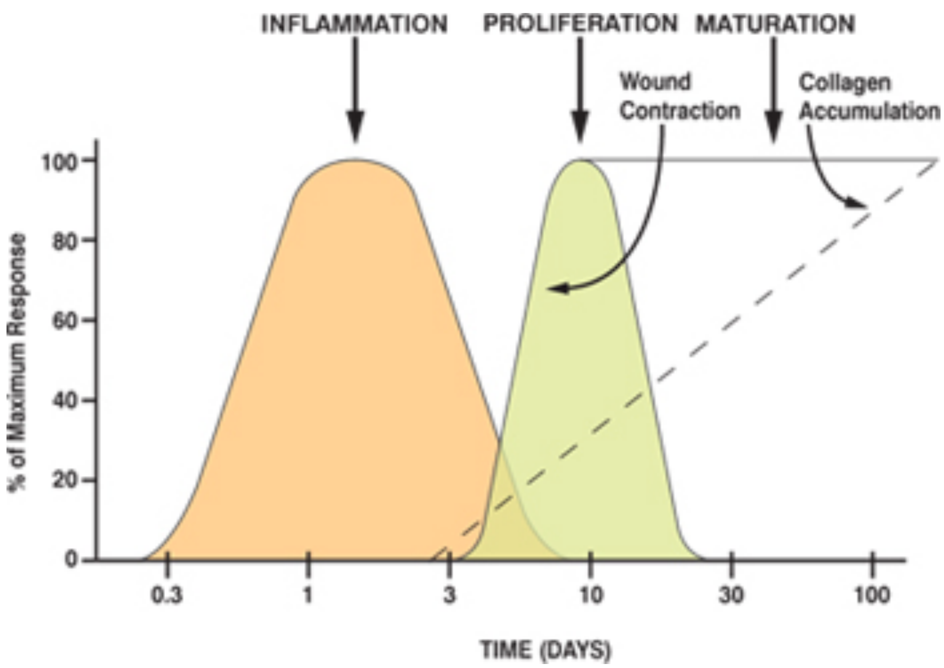

Figure 2: Approximate times of the three phases of wound healing.[xi]

An individual's susceptibility to ulceration is dependent not only upon the type of mechanical force applied to the bony prominence, but also upon the intensity of the mechanical force and length of time it is applied. High-intensity pressure threatens tissue survival, as does low-intensity pressure applied over a long interval of time.[ii] Studies on animal subjects show an inverse relationship between the magnitude of force and duration of loading required to develop irreversible tissue damage.[xii] This finding suggests that higher pressure loads require less time to initiate tissue breakdown. Equally, long periods of low pressure can induce similar damage.

Subepidermal Moisture: A Biomarker of Tissue Health

Inflammation presents an opportunity to detect tissue damage that may not be visible via visual skin assessments. Sub-epidermal moisture (SEM), the water present in tissue beneath the skin's surface, is a biophysical measure which can be used to assess the functional integrity of the epidermal barrier.[xiii] Because SEM levels are directly proportional to capacitance (retention of electrical charge) the impedance of the skin to electrical forces can be used to calculate the skin's surface electrical capacitance, a measure that directly reflects SEM.[xiv]

Measuring SEM through surface electrical capacitance has several applications. Because SEM directly correlates to the water content of the epidermal and subepidermal tissues, it may be used to quantify the severity of localized tissue edema, and thus, the cellular swelling associated with inflammation brought on by deep tissue injury. Increased surface electrical capacitance correlates to higher levels of SEM and thus, greater tissue damage.

The Predictive Capability of SEM

Accordingly, there is predictive value in sub-epidermal moisture measurements. In a sample of 66 nursing home residents, higher levels of SEM were associated with both concurrent and incident (1 week later) skin damage in persons with dark skin tones.[xiv] Pressure ulcers were visible at the sacrum, left buttock, or right buttock a week following higher SEM values at the same anatomical sites without visible pressure ulcer damage, thereby demonstrating the predicative ability of SEM.[xiv] This pattern was more pronounced among darkly pigmented patients. SEM values detected up to 30% of occurrences of damage to the skin the following week representing an up to 25% increase from detection with visual skin assessment by a skin care expert.[xiv] Ultimately, being able to predict a third more pressure ulcers by measuring SEM levels suggests that SEM threshold values can be used to detect tissue damage and accordingly, enable care providers the opportunity to target rapid interventions for early, effective pressure ulcer treatment and prevention.

Bruin Biometrics, LLC (BBI) has developed the SEM Scanner to assist clinicians in these efforts. The SEM Scanner measures relative tissue surface electrical capacitance through the application of low amplitude signals from electrode structures placed on the subject's skin. Additionally, since the SEM Scanner sensor technology penetrates up to 2 mm into the skin, the device can assess moisture in the dermis as well as the epidermis, enabling healthcare practitioners to identify incipient pressure ulcers 3 to 10 days before such damage can be seen at the skin's surface.[xv,xvi] The SEM Scanner has the ability to acquire these measurements regardless of skin pigmentation, therefore addressing the barriers in detecting pressure ulcer formation via visual assessment for dark skin-toned patients.

By targeting detection of the inflammatory stage, particularly at the cellular level, the SEM Scanner enables clinicians to intervene prior to any other detection method currently available. The SEM Scanner gives practitioners insight into the relative health – or damage – present in subepidermal tissue. For the first time, the ability to prevent pressure ulcer formation is possible.

Endnotes

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