

Pressure Care References - June 2008

Nixon J, Cranny G, Iglesias C et al. Randomised, controlled trial of alternating pressure mattresses compared with alternating pressure overlays for the prevention of pressure ulcers: PRESSURE (pressure relieving support surfaces) trial. *BMJ* 2006, 332 (7555): 1413.

Pressure Ulcer References.

This month's pressure ulcer references focus upon the emerging issue of deep tissue injury as a form of pressure damage. From MEDLINE these publications cover clinical insights through to elegant laboratory experiments to establish whether deeper tissue layers may be vulnerable to pressure induced tissue damage.

Agam, L. and A. Gefen (2007). "Pressure ulcers and deep tissue injury: a bioengineering perspective." *Journal of Wound Care* 16(8): 336-42.

Wheelchair users are highly susceptible to deep tissue injury. Interface pressures are unlikely to predict this, and an alternative assessment approach is needed that can easily monitor internal mechanical stresses and deformations. [References: 108]

Ankrom, M. A., R. G. Bennett, et al. (2005). "Pressure-related deep tissue injury under intact skin and the current pressure ulcer staging systems." *Advances in Skin & Wound Care* 18(1): 35-42.

OBJECTIVE: To identify how current pressure ulcer staging systems and experts describe pressure-related deep tissue injury under intact skin in the published research literature. **DESIGN:** A systematic review of published English-language literature as of November 2002 with the words decubitus or pressure ulcer(s) in the title. Additional relevant articles were identified by National Pressure Ulcer Advisory Panel members and were included in the analysis. An expert commentary was developed by iterative review by the National Pressure Ulcer Advisory Panel members. **MAIN OUTCOME MEASURES:** Manuscripts were reviewed for staging systems cited or described, definitions of Stage I pressure ulcers, and descriptions or definitions of pressure-related deep tissue injury under intact skin. **MAIN RESULTS:** Ninety-four relevant articles were identified. Seventy-three articles (78%) described a staging system, and 55 of 73 (75%) cited the staging definitions from Shea, the National Pressure Ulcer Advisory Panel, or the Agency for Health Care Policy and Research. The National Pressure Ulcer Advisory Panel's staging definitions were the most frequently cited overall. Twenty-three articles (25%) included some discussion that could be interpreted as relevant to the topic of pressure-related deep tissue injury under intact skin; however, no consistency in definitions of Stage I pressure ulcers or terminology for pressure-related deep tissue injury under intact skin was found. **CONCLUSIONS:** Several

pressure ulcer staging systems are frequently cited, but none define pressure-related deep tissue injury under intact skin. The National Pressure Ulcer Advisory Panel recommends using the terms "pressure-related deep tissue injury under intact skin" or "deep tissue injury under intact skin" for describing these lesions and encourages investigators to establish the epidemiology and natural history of these lesions. [References: 44]

Baugh, N., H. Zuelzer, et al. (2007). "Wound wise: wounds in surgical patients who are obese." *American Journal of Nursing* 107(6): 40-50; quiz 51.

The number of surgical patients who are obese in the United States is rising, a trend that's likely to continue. Such patients are at higher risk than nonobese patients are for surgical site infections and other complications such as dehiscence, pressure ulcers, deep tissue injury, and rhabdomyolysis. This article details the factors that can contribute to such complications, including a high number of comorbidities, and offers practical suggestions for preventing them. Nurses should understand that special equipment, precautions, and protocols may be needed at every stage of care, and that obese patients aren't anomalies but rather a part of a growing population with particular needs. [References: 54]

Berlowitz, D. R. and D. M. Brienza (2007). "Are all pressure ulcers the result of deep tissue injury? A review of the literature." *Ostomy Wound Management* 53(10): 34-8.

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. [References: 27]

Black, J., M. Baharestani, et al. (2007). "National Pressure Ulcer Advisory Panel's updated pressure ulcer staging system." *Dermatology Nursing* 19(4): 343-9; quiz 350.

The National Pressure Ulcer Advisory Panel has updated the definition of a pressure ulcer and the stages of pressure ulcers based on current research and expert opinion solicited

from hundreds of clinicians, educators, and researchers across the country. The amount of anatomical tissue loss described with each stage has not changed. New definitions were drafted to achieve accuracy, clarity, succinctness, clinical utility, and discrimination between and among the definitions of other pressure ulcer stages and other types of wounds. Deep tissue injury was also added as a distinct pressure ulcer in this updated system. [References: 28]

Donnelly, J. and P. National Pressure Ulcer Advisory (2005). "Should we include deep tissue injury in pressure ulcer staging systems? The NPUAP debate." *Journal of Wound Care* 14(5): 207-10.

This year's annual conference of the US National Pressure Ulcer Advisory Panel (NPUAP) included a consensus meeting to evaluate the current NPUAP pressure ulcer staging system. Jeannie Donnelly recounts the lively debate that ensued. [References: 8]

Doughty, D., J. Ramundo, et al. (2006). "Issues and challenges in staging of pressure ulcers." *Journal of Wound, Ostomy, & Continence Nursing* 33(2): 125-30; quiz 131-2.

Wound assessment is a key element of effective wound care, and assessment of pressure ulcers includes accurate determination of wound stage. Although the original staging system established by Shea was based on his understanding of the pathology involved in pressure ulcer development, subsequent staging systems (and the one currently in use) were intended simply to establish the level of tissue damage. Recently, clinicians have drawn attention to numerous limitations associated with the current staging system, including the inability to differentiate between an inflammatory response involving intact skin and a deep tissue injury (deep bruising) underneath intact skin. This is a clinically significant difference because clinicians have noted that most inflammatory responses resolve with intervention, whereas most areas of deep tissue injury progress to full-thickness ulcers even when appropriate intervention is provided. A second area of controversy involves partial-thickness (Stage 2) lesions; because many of these lesions are caused by maceration and/or friction (as opposed to pressure) clinicians are frequently unclear regarding which of these lesions should be staged. In response to these concerns, the National Pressure Ulcer Advisory Panel convened a consensus forum and published white papers to clearly outline the issues; they solicited clinician feedback on the white papers and the Wound, Ostomy, Continence Nurses Society provided a written response. This article summarizes the key points of the white papers, WOCN Society response, and consensus forum discussion. [References: 10]

Fleck, C. A. (2007). "Suspected deep tissue injury." *Advances in Skin & Wound Care* 20(7): 413-5.

Gawlitta, D., C. W. J. Oomens, et al. (2007). "Temporal differences in the influence of ischemic factors and deformation on the metabolism of engineered skeletal muscle." *Journal of Applied Physiology* 103(2): 464-73.

Prolonged periods of tissue compression may lead to the development of pressure ulcers, some of which may originate in, for example, skeletal muscle tissue and progress underneath intact skin, representing deep tissue injury. Their etiology is multifactorial and the interaction between individual causal factors and their relative importance remain unknown. The present study addressed the relative contributions of deformation and ischemic factors to altered metabolism and viability. Engineered muscle tissue was prepared as previously detailed (14) and subjected to a combination of factors including 0% oxygen, lactic acid concentrations resulting in pH from 5.3 to 7.4, 34% compression, and low glucose levels. Deformation had an immediate effect on tissue viability {[3-(4,5-dimethylthiazol-2-yl)-2,5-diphenyltetrazolium bromide] (MTT) assay}, which increased with time. By contrast, hypoxia evoked metabolic responses (glucose and lactate levels) within 24 h, but viability was only reduced after 48 h. In addition, lactic acidification downregulated tissue metabolism up to an acid concentration (approximately 23 mM) where metabolism was arrested and cell death enhanced. A similar tissue response was observed during glucose deprivation, which, at negligible concentration, resulted in both a cessation of metabolic activity and a reduction in cell viability. The combination of results suggests that in a short-term (<24 h) deformation, extreme acidification and glucose deprivation increased the level of cell death. By contrast, nonextreme acidification and hypoxia influenced tissue metabolism, but not the development of cell death. These data provide more insight into how compression-induced factors can lead to the onset of deep tissue injury.

Gefen, A. (2007). "The biomechanics of sitting-acquired pressure ulcers in patients with spinal cord injury or lesions." *International Wound Journal* 4(3): 222-31.

Sitting-acquired pressure ulcers (SAPU) in permanent wheelchair users with traumatic or non traumatic disorders of the central nervous system (CNS) are a great medical challenge. The purpose of this review is to summarise what is currently known concerning the aetiology of SAPU, particularly in its severe form, which may now be classified as a 'deep tissue injury' according to the US National Pressure Ulcer Advisory Panel. Specifically, this review focuses on biomechanical aspects of deep SAPU and describes the relevant bioengineering methodologies and research evidence. It discusses the unique biomechanical conditions in deep tissues, which are caused by chronic sitting associated with CNS disorders, and overall, the present review indicates that avoiding interface pressures above 32 mmHg in such patients is not necessarily a 'pressure relief.' [References: 57]

Gefen, A. (2007). "Risk factors for a pressure-related deep tissue injury: a theoretical model." *Medical & Biological Engineering & Computing* 45(6): 563-73.

Pressure-related deep tissue injury is the term recommended by the United States National Pressure Ulcer Advisory Panel to describe a potentially life-threatening form of pressure ulcers, characterized by the presence of necrotic tissue under intact skin, and associated with prolonged compression of muscle tissue under bony prominences. In this study, a theoretical model was used to determine the relative contributions of the backrest inclination angle during prolonged wheelchair sitting, the muscle tissue stiffness and curvature of the ischial tuberosities (ITs) to the risk for injury in the gluteus muscles that pad the IT bones during sitting. The model is based on Hertz's theory for analysis of contact pressures between a rigid half-sphere (bone) and an elastic half-space (muscle). Hertz's theory is coupled with an injury threshold and damage law for muscle-both obtained in previous studies in rats. The simulation outputs the time-dependent bone-muscle contact pressures and the injured area in the gluteus. We calculated the full-size (asymptotic) injured area in the gluteus and the time for injury onset for different sitting angles α (90-150 degrees), muscle tissue long-term shear moduli G (250-1,200 Pa) and bone diameters D (8-18 mm). We then evaluated the sensitivity of model results to variations in these parameters, in order to determine how injury predictions are affected. In reclined sitting ($\alpha=150$ degrees) the full-size injured area was approximately 2.1-fold smaller and the time for injury onset was approximately 1.3-fold longer compared with erect sitting ($\alpha=90$ degrees). For greater G the full-size injured area was smaller but the time for injury onset was shorter, e.g., increasing G from 250 to 1200 Pa decreased the full-size injured area approximately 2.5-fold, but shortened the time for injury onset 6.2-fold. For smaller D the time for injury onset dropped, e.g., decreased approximately 1.5-fold when D decreased from 18 to 8 mm. Interestingly, the full-size injured area maximized at D of about 12 mm but decreased for smaller or larger D . The susceptibility to sitting-acquired deep tissue injury strongly depends on the geometrical and biomechanical characteristics of the bone-muscle interface, and, particularly, on the radius of curvature of the IT which mostly influenced the size of the wound, and on the muscle stiffness which dominantly affected the time for injury onset.

Gefen, A. (2008). "Bioengineering models of deep tissue injury." *Advances in Skin & Wound Care* 21(1): 30-6.

Gefen, A. and E. Haberman (2007). "Viscoelastic properties of ovine adipose tissue covering the gluteus muscles." *Journal of Biomechanical Engineering* 129(6): 924-30.

Pressure-related deep tissue injury (DTI) is a life-risking form of pressure ulcers threatening immobilized and neurologically impaired patients. In DTI, necrosis of muscle and enveloping adipose tissues occurs under intact skin, owing to prolonged compression by bony

prominences. Modeling the process of DTI in the buttocks requires knowledge on viscoelastic mechanical properties of the white adipose tissue covering the gluteus muscles. However, this information is missing in the literature. Our major objectives in this study were therefore to (i) measure short-term (H(S)) and long-term (H(L)) aggregate moduli of adipose tissue covering the glutei of sheep, (ii) determine the effects of preconditioning on H(S) and H(L), and (iii) determine the time course of stress relaxation in terms of the transient aggregate modulus H(t) in nonpreconditioned (NPC) and preconditioned (PC) tissues. We tested 20 fresh tissue specimens (from 20 mature animals) in vitro: 10 specimens in confined compression for obtaining the complete H(t) response to a ramp-and-hold protocol (ramp rate of 300 mms), and 10 other specimens in swift indentations for obtaining comparable short-term elastic moduli at higher ramp rates (2000 mms). We found that H(S) in confined compression were 28.9 \pm 14.9 kPa and 18.1 \pm 6.9 kPa for the NPC and PC specimens, respectively. The H(L) property, 10.3 \pm 4.2 kPa, was not affected by preconditioning. The transient aggregate modulus H(t) always reached the plateau phase (less than 10% difference between H(t) and H(L)) within 2 min, which is substantially shorter than the times for DTI onset reported in previous animal studies. The short-term elastic moduli at high indentation rates were 22.6 \pm 10 kPa and 15.8 \pm 9.4 kPa for the NPC and PC test conditions, respectively. Given a Poisson's ratio of 0.495, comparison of short-term elastic moduli between the high and slow rate tests indicated a strong deformation-rate dependency. The most relevant property for modeling adipose tissue as related to DTI is found to be H(L), which is conveniently unaffected by preconditioning. The mechanical characteristics of white adipose tissue provided herein are useful for analytical as well as numerical models of DTI, which are essential for understanding this serious malady.

Gefen, A. and J. Levine (2007). "The false premise in measuring body-support interface pressures for preventing serious pressure ulcers." *Journal of Medical Engineering & Technology* 31(5): 375-80.

Presently, commercial cushioning products for pressure ulcer prevention are being evaluated for their protective effect exclusively based on interfacial pressures between the cushion/mattress and the patient. However, interface pressures cannot predict elevated mechanical stresses in deep tissues adjacent to bony prominences. Such deep tissue stress concentrations are associated with local ischaemia and hypoxia, which over time result in deep tissue necrosis, particularly of muscle tissue. In order to demonstrate this phenomenon, a physical phantom of the mechanical interaction between the ischial tuberosities (IT) and gluteus muscles of the buttocks was built, incorporating geometric replica of the human IT and real (bovine) muscle tissue. Internal muscle stresses directly under the IT were five to 11-fold greater than stresses at more distal locations, and a Pearson correlation test showed that they could not have been predicted from the interface pressures in the phantom. Accordingly, though pressure

ulcer prevention clinics which utilize routine sitting pressure measurements report effective outcomes, the present results highlight a problem in using body-support pressure measurements to predict the risk for pressure-related deep tissue injury.

Linder-Ganz, E. and A. Gefen (2007). "The effects of pressure and shear on capillary closure in the microstructure of skeletal muscles." *Annals of Biomedical Engineering* 35(12): 2095-107.

Deep tissue injury (DTI) is a severe pressure ulcer, which initiates in muscle tissue under a bony prominence, and progresses outwards. It is associated with mechanical pressure and shear that may cause capillaries to collapse and thus, induce ischemic conditions. Recently, some investigators stipulated that ischemia alone cannot explain the etiology of DTI, and other mechanisms, particularly excessive cellular deformations may be involved. The goal of this study was to evaluate the functioning of capillaries in loaded muscle tissue, using animal and finite element (FE) models. Pressures of 12, 37, and 78 kPa were applied directly to one gracilis muscle of 11 rats for 2 h. Temperatures of the loaded and contralateral muscles were recorded with time using infrared thermography (IRT) as a measure of the ischemic level. In addition, a non-linear large deformation muscle-fascicle-level FE model was developed and subjected to pressures of 12-120 kPa without and with simultaneous shear strain of up to 8%. For each simulation case, the accumulative percentage of open capillary cross-sectional area and the number of completely closed capillaries were determined. After 2 h, temperature of the loaded muscles was 2.4 +/- 0.3 degrees C (mean +/- standard deviation) lower than that of the unloaded contralateral limbs (mean of plateau temperature values across all pressure groups). Temperature of the loaded muscles dropped within 10 min but then remained stable and significantly higher than room temperature for at least 30 additional minutes in all pressure groups, indicating that limbs were not completely ischemic within the first 40 min of the trials. Our FE model showed that in response to pressures of 12-120 kPa and no shear, the accumulative percentage of open capillary cross-sectional area decreased by up to 71%. When shear strains were added, the open capillary cross-sectional area decreased more rapidly, but even for maximal loading, only 46% of the capillaries were completely closed. Taken together, the animal and FE model results suggest that acute ischemia does not develop in skeletal muscles under physiological load levels within a timeframe of 40 min. Since there is evidence that DTI develops within a shorter time, ischemia is unlikely to be the only factor causing DTI.

Linder-Ganz, E., N. Shabshin, et al. (2007). "Assessment of mechanical conditions in sub-dermal tissues during sitting: a combined experimental-MRI and finite element approach." *Journal of Biomechanics* 40(7): 1443-54.

A common but potentially severe malady afflicting permanent wheelchair users is pressure sores caused by elevated soft tissue strains and stresses over a critical prolonged

period of time. Presently, there is paucity of information regarding deep soft tissue strains and stresses in the buttocks of humans during sitting. Strain and stress distributions in deep muscle and fat tissues were therefore calculated in six healthy subjects during sitting, in a double-donut Open-MR system, using a "reverse engineering" approach. Specifically, finite element (FE) models of the undeformed buttock were built for each subject using MR images taken at the coronal plane in a non-weight-bearing sitting posture. Using a second MR image taken from each subject during weight-bearing sitting we characterized the ischial tuberosity sagging toward the sitting surface in weight-bearing, and used these data as displacement boundary conditions for the FE models. These subject-specific FE analyses showed that maximal tissue strains and stresses occur in the gluteal muscles, not in fat or at the skin near the body-seat interface. Peak principal compressive strain and stress in the gluteus muscle were 74+/-7% and 32+/-9 kPa (mean+/-standard deviation), respectively. Peak principal compressive strain and stress in enveloping fat tissue were 46+/-7% and 18+/-4 kPa, respectively. Models were validated by comparing measured peak interface pressures under the ischial tuberosities (17+/-4 kPa) with those calculated by means of FE (18+/-3 kPa), for each subject. This is the first study to quantify sub-dermal tissue strain and stress distributions in sitting humans, in vivo. These data are essential for understanding the aetiology of pressure sores, particularly those that were recently termed "deep tissue injury" at the US National Pressure Ulcer Advisory Panel (NPUAP) 2005 Consensus Conference.

Nagase, T., I. Koshima, et al. (2007). "Ultrasonographic evaluation of an unusual peri-anal induration: a possible case of deep tissue injury." *Journal of Wound Care* 16(8): 365-7.

High-resolution ultrasound produces good quality echo images of skin and subcutaneous lesions. Here, it was used to characterise an atypical induration, which the authors speculate might have been a deep tissue injury. [References: 14]

Salcido, R. S. (2007). "Myosubcutaneous infarct: deep tissue injury." *Advances in Skin & Wound Care* 20(5): 248-50.

Solis, L. R., D. P. Hallihan, et al. (2007). "Prevention of pressure-induced deep tissue injury using intermittent electrical stimulation." *Journal of Applied Physiology* 102(5): 1992-2001.

Pressure ulcers develop due to morphological and biochemical changes triggered by the combined effects of mechanical deformation, ischemia, and reperfusion that occur during extended periods of immobility. The goal of this study was to test the effectiveness of a novel electrical stimulation technique in the prevention of deep tissue injury (DTI). We propose that contractions elicited by intermittent electrical stimulation (IES) in muscles subjected to constant pressure would induce periodic relief in internal pressure; additionally, each contraction would

also restore blood flow to the tissue. The application of constant pressure to the quadriceps muscles of rats generated a DTI that affected 60 +/- 15% of the compressed muscle as assessed by magnetic resonance imaging. In contrast, in the groups of rats that received IES at 10- and 5-min intervals, DTI of the muscle was limited to 16 +/- 16 and 25 +/- 13%, respectively. Injury to the muscle was corroborated by histology. In an experiment with a human volunteer, compression of the buttocks reduced the oxygenation level of the muscles by approximately 4%; after IES, oxygenation levels increased by approximately 6% beyond baseline. Concurrently, the surface pressure profiles of the loaded muscles were redistributed and the high-pressure points were reduced during each IES-induced contraction. The results of this study indicate that IES significantly reduces the amount of DTI by increasing the oxygen available to the tissue and by modifying the pressure profiles of the loaded muscles. This presents a promising technique for the prevention of pressure ulcers in immobilized and/or insensate individuals.

Stekelenburg, A., C. W. J. Oomens, et al. (2006). "Compression-induced deep tissue injury examined with magnetic resonance imaging and histology." *Journal of Applied Physiology* 100(6): 1946-54.

The underlying mechanisms leading to deep tissue injury after sustained compressive loading are not well understood. It is hypothesized that initial damage to muscle fibers is induced mechanically by local excessive deformation. Therefore, in this study, an animal model was used to study early damage after compressive loading to elucidate on the damage mechanisms leading to deep pressure ulcers. The tibialis anterior of Brown-Norway rats was loaded for 2 h by means of an indenter. Experiments were performed in a magnetic resonance (MR)-compatible loading device. Muscle tissue was evaluated with transverse relaxation time (T₂)-weighted MRI both during loading and up to 20 h after load removal. In addition, a detailed examination of the histopathology was performed at several time points (1, 4, and 20 h) after unloading. Results demonstrated that, immediately after unloading, T₂-weighted MR images showed localized areas with increased signal intensity. Histological examination at 1 and 4 h after unloading showed large necrotic regions with complete disorganization of the internal structure of the muscle fibers. Hypercontraction zones were found bilateral to the necrotic zone. Twenty hours after unloading, an extensive inflammatory response was observed. The proposed relevance of large deformation was demonstrated by the location of damage indicated by T₂-weighted MRI and the histological appearance of the compressed tissues. Differences in damage development distal and proximal to the indenter position suggested a contribution of perfusion status in the measured tissue changes that, however, appeared to be reversible.

Stekelenburg, A., G. J. Strijkers, et al. (2007). "Role of ischemia and deformation in the onset of compression-induced deep tissue injury: MRI-based studies in a rat model." *Journal of Applied Physiology* 102(5): 2002-11.

A rat model was used to distinguish between the different factors that contribute to muscle tissue damage related to deep pressure ulcers that develop after compressive loading. The separate and combined effects of ischemia and deformation were studied. Loading was applied to the hindlimb of rats for 2 h. Muscle tissue was examined using MR imaging (MRI) and histology. An MR-compatible loading device allowed simultaneous loading and measurement of tissue status. Two separate loading protocols incorporated uniaxial loading, resulting in tissue compression and ischemic loading. Uniaxial loading was applied to the tibialis anterior by means of an indenter, and ischemic loading was accomplished with an inflatable tourniquet. Deformation of the muscle tissue during uniaxial loading was measured using MR tagging. Compression of the tissues for 2 h led to increased T2 values, which were correlated to necrotic regions in the tibialis anterior. Perfusion measurements, by means of contrast-enhanced MRI, indicated a large ischemic region during indentation. Pure ischemic loading for 2 h led to reversible tissue changes. From the MR-tagging experiments, local strain fields were calculated. A 4.5-mm deformation, corresponding to a surface pressure of 150 kPa, resulted in maximum shear strain up to 1.0. There was a good correlation between the location of damage and the location of high shear strain. It was concluded that the large deformations, in conjunction with ischemia, provided the main trigger for irreversible muscle damage.

Wang, Q., L. Kong, et al. (2006). "Portable gage for pressure ulcer detection." *Conference Proceedings: ... Annual International Conference of the IEEE Engineering in Medicine & Biology Society* 1: 5997-6000.

Pressure ulcers are widely considered to be a critical problem in rehabilitation since they result in severe discomfort and high healthcare cost. The prevention of pressure ulcers is a constant preoccupation for every nursing team. This paper introduces a novel handheld instrument that can detect subtle changes in the skin biomechanical properties by measuring its biomechanical response. This could be used to detect stage-I pressure ulcers and deep tissue injury. Its high bandwidth makes it possible to load the skin under wide range of conditions. The instrument is portable, inexpensive, and intrinsically precise. Several experiments were conducted to validate the function of the device. Preliminary results show that the device could effectively measure the difference in the viscoelasticity between human skin of different sites, hence paving the way for the development of clinical protocols and trials.

Zulkowski, K., D. Langemo, et al. (2005). "Coming to consensus on deep tissue injury." *Advances in Skin & Wound Care* 18(1): 28-9.

