The subepidermal moisture scanner: the technology explained

Abstract: The objective of this article is to explain the biophysical principles underlying the design of the subepidermal moisture (SEM) scanner, commercially known as the 'SEM scanner'. We also describe the mode of operation of the SEM scanner in monitoring tissue health and detecting subtle abnormal changes in tissue physiology in patients and anatomical sites at a risk of a pressure ulcer (PU: also known as a pressure injury). The technology of the SEM scanner was approved last year for sales in the US by the Food and Drug Administration (FDA). The SEM scanner detects changes in fluid contents of human skin and subdermal tissues, to a tissue depth of several millimetres, by measuring 'capacitance', an electrical property of the locally examined tissue site to store electric charge. The capacitance of tissues, called 'biocapacitance', is strongly affected by the amount of fluid (water) in the tissue. When the first cells die in a forming PU, inflammatory signalling causes the permeability of blood vessel walls to increase and oedema to develop. Simply, the scanner detects the early appearance of oedema, which is called 'micro-oedema.' Calculation of a 'SEM-delta' value, which compares biocapacitance measurements, acquired across several tissue sites, some of which are healthy and others where the PU may evolve, eliminates potential effects of systemic changes in tissue fluid contents and provides a consistent quantitative measure of the tissue health conditions at the monitored anatomical site. Here, we describe SEM scanner technology, how it operates and has been laboratory tested (in computer simulations, *in silico*) before commercial launch. We explain why targeting the physical biomarker of oedema leads to the documented success of the SEM scanner in the multiple published clinical trials, proving its ability to early detect PUs that form under intact skin.
Declaration of interest: GR is an employee of BBI LLC (Bruin Biometrics) and holds equity in the company. AG is consulting to BBI LLC from which he received support for his research in pressure ulcer/injury prevention. These relationships had no influence on the compilation and analysis of published data, interpretation of results and the conclusions made here.

laboratory testing and modelling • localised oedema • pressure injury • pressure ulcer • pressure ulcer prevention • SEM scanner

Pressure ulcers (PU; also known as a pressure injuries) are a common hospital-acquired condition. Despite massive efforts and vast resources invested in mitigating the problem of hospital-acquired PUs (HAPU), it is the only condition on the rise in the US.[1] In the US, PUs consume healthcare costs of at least US$ 26.8 billion per year.[2] Aetiological research of PUs has made seminal breakthroughs in the last decade, describing a detailed picture of how deformation-inflicted damage is formed at the cell level and revealing the damage pathways that were previously unknown, such as gradual deterioration of the distorted cell membranes.[3]

The mechano-biological understanding of PU development is that cell and tissue damage may begin under intact skin through the death of cells exposed to sustained mechanical deformation.[3] There may be clinically significant tissue damage before visual or tactile symptoms, such as discolouration or changes in firmness or temperature, are seen on the skin surface. With the deformation-inflicted death of the first cells, inflammation is triggered via signalling molecules called chemokines and neurotransmitters (for example, histamine) that, among other activities, dilate and increase the permeability of blood vessels, allowing crossing (extravasation) and migration of the immune cells recruited from the blood to the damaged site.[4] This results in leakage of plasma fluids from the diluted and permeable blood vessels in the inflamed site into interstitial spaces in tissues, thereby forming oedema, first microscopically and then macroscopically.[3] Similarly, the lymphatic vessels at the site of inflammation undergo pronounced enlargement and display increased leakiness.[5] Given that the rise in interstitial fluid contents occurs gradually (the clinically familiar swollen, firm and warm appearance of an inflamed tissue is reached at the fully-developed phase of the inflammatory response), biomedical engineers have been able to use this progressive build-up of fluid mass in tissues as a 'biophysical marker' for detecting initial tissue damage.[3,4]

Localised oedema, also called subepidermal moisture (SEM), is the underlying physiological phenomenon upon which the operation of the SEM scanner (Model 200, Bruin Biometrics, US) is based.[6,7]

Our objective is to explain the basic physical principles underlying the design of the SEM scanner and its mode of operation in monitoring tissue health

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Fig 1. The subepidermal moisture (SEM) scanner 200 model. Front side showing the display (**a**) and back side showing the sensor (electrode) (**b**). Coins of one quarter USD and one Euro were placed near the sensor as 'intuitive' scale bars to depict the SEM sensor size. The sensor region has further been magnified for clarity (**c**)

and detecting subtle abnormal changes in tissue physiology in patients and anatomical sites at risk of PU. The technology of the SEM Scanner, which was given marketing authorisation by the US Food and Drug Administration (FDA) last year, has never before been explained in a way accessible to clinicians. Here we elucidate how the SEM scanner operates in a non-technical language for practicing health professionals.

What is being assessed by the SEM scanner?

The SEM scanner (Fig 1) measures the biocapacitance of the local skin and subdermal tissues under its sensor (Fig 1b). Biocapacitance is a bioelectrical property that is the ratio of the change in an electric charge in a tissue region to the corresponding change in its electric potential. A large self-biocapacitance of a tissue region indicates that this region is able to hold more electric charge at a given voltage than a different region with a low self-biocapacitance. The biocapacitance is a function of the geometry and structure, which for the SEM scanner measurement is the area of the device sensor and the composition of the examined soft tissues in the immediate vicinity of the sensor, especially the dielectric properties (which allows electrostatic fields to pass through) of these tissues. For tissues, as with many dielectric materials, the biocapacitance is independent of the electrical potential applied by the SEM sensor. However, the biocapacitance of tissues is variable and highly sensitive to the interstitial water content of the tissue. The dielectric constant of water (which is approximately 80) is 10–20 times greater than that of all solid tissue components, for example, collagen and elastin.[8,9] In a certain anatomical region, with a given anatomical configuration, the SEM scanner reading of biocapacitance will be mainlt affected by the dielectric tissue properties, which are highly sensitive to the amount of water in the examined tissues. Accordingly, any inflammation-related increase in the permeability of the vascular and/or lymphatic walls will almost immediately be measureable due to its impact on the effective dielectric property of the affected tissues. Hence, the tissue biocapacitance will increase rapidly and dramatically, even if the inflammatory response has just been initiated and visible (clinical) signs have not yet developed.

The SEM scanner reports the level of biocapacitance of a tissue site as an 'SEM value.' A comparison of the SEM values at the inflamed tissue site with those from adjacent, healthy tissue sites will identify the maximum difference between the SEM values, which is called the 'SEM-delta.' The greater the SEM-delta, the greater the oedema and tissue damage expected at the scanned site. Therefore, the SEM-delta is an objective and quantitative reading of the tissue health conditions, wherein a low SEM-delta indicates healthy tissue and a high SEM-delta points to inflammation as a result of cell and tissue death. In particular, a trend of increasing SEM-delta values acquired at a common body site over time (i.e. from one day to another) may indicate an increasing, spreading inflammation that is the response to an ongoing tissue degradation process.

Features of the SEM scanner

The SEM Scanner is a handheld, wireless device (Fig 1a) that displays the SEM values on its front panel. The circular SEM sensor (diameter 20mm) for measuring the biocapacitance of the monitored tissues is located on the back of the device (Fig 1b and 1c) and facilitates measurements at anatomically-curved or narrow surfaces, such as the posterior heel regions. This SEM sensor is also sensitive to the force a user applies to achieve contact between the sensor and the skin of a patient. It will assess the tissue biocapacitance only when the device is adequately (but not too forcefully) in contact with the skin, ensuring valid measurements. When the applied user-force is removed, the SEM scanner automatically resets and becomes ready to acquire a new reading.

The physical units of tissue biocapacitance are picofarads but, for simplicity, standardisation and clinical utility, device readings are displayed in a non-dimensional scale of SEM values, which may theoretically range between 0.3 and 3.9. The aforementioned range is considered theoretical because a value as low as 0.3 is characteristic of a SEM measurement taken in open air, whereas the maximal value of 3.9 is expected when the sensor is completely

submerged in pure water. The practical SEM data range, i.e. readings acquired from soft tissues of patients, should be narrower as living tissues are biphasic (contain both water and a solid phase). Nevertheless, if the soft tissue mass is extremely small, for example, due to severe atrophy or cachexia, SEM values may be as low as 0.4.[10] On the other end of the spectrum, if oedema is already fully-developed and widespread, or in cases of progressive lymphoedema, SEM values may be as high as 3.8.[10]

The SEM-delta parameter

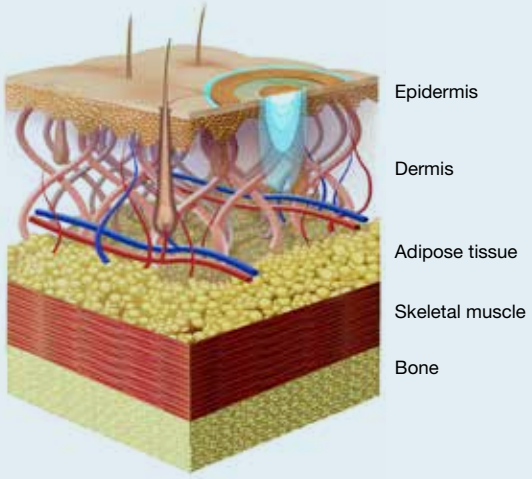
After acquisition of a set of SEM values from an at-risk region, for example, the sacrum or posterior heel, the scanner automatically compares the values in this set and identifies the maximal and minimal values within the set. The difference between the maximal and minimal values in the set is then calculated and displayed as the SEM-delta, which is the parameter of clinical interest for the examined anatomical region. The SEM-delta quantifies a difference between the biocapacitance at a tissue site that may contain subsurface damage (indicated by detection of elevated local tissue fluid contents at any stage of evolution of the oedema from the microscale to the macroscale), with respect to other, healthy (reference) tissue sites.

For example, a set of six measurements may be taken from adjacent sites in the sacral region. If the tissue under one of these sites has been affected by cell and tissue damage, which promoted an inflammatory response, then the resulting localised oedema will elevate the biocapacitance in that site. In the absence of a clinically visible PU in the examined sacral region, it is highly likely that at least one of the other tissue sites within the same region will be undamaged tissue and therefore serve as the healthy reference tissue site. In other words, the difference between the highest and lowest SEM data in the set will almost certainly increase if at least one of the SEM measurement sites has developed localised oedema resulting from a subdermal tissue injury. It is this non-uniformity of the SEM value dataset that indicates the likelihood of forming tissue damage. A healthy patient will have low variability and a low SEM-delta value, in their values acquired from sites within the same anatomical region as all measurement points should reflect the biocapacitance of healthy tissues. If any measurement point captured an increase in biocapacitance due to localised oedema, then the variability in the SEM dataset will rise and be represented by a greater SEM-delta. In clinical measurements, a low SEM-delta in the range of 0.0–0.2 is indicative of a lower risk of PU, whereas SEM-delta readings of ≥0.6 indicate a higher risk of PU.[10]

Characterisation of tissue damage development using the SEM-delta

The field of detection of the SEM scanner sensor penetrates at its deepest directly under the centre of the sensor, to a typical depth of 3–4mm, through the epidermis and the dermis, reaching the superficial subcutaneous fat (Fig 2). Hence, the SEM scanner assesses tissue that cannot be examined by traditional visual skin assessments, which only become effective when subdermal tissue damage has already presented itself on the skin surface. Elevated SEM-delta values have not only been clinically associated with visible signs of existing tissue damage but also with damage that visually appears several days to approximately one week later, including deep tissue injuries (DTI).[10–18] These clinical findings, consistent in multiple large acute-and long-term-care patient cohorts as well as in nursing-home residents, are further supported by reports of excellent inter-operator and inter-device agreements.[19]

Fig 2. Illustration of the electric field used in the process of measurement of the local biocapacitance property of tissues, showing the shape and depth of penetration of the electric field of the subepidermal moisture (SEM) scanner into the epidermis and dermal layers



Given its use to identifying increased risk of PU earlier than visual skin inspections, the SEM scanner is particularly useful for implementation of PU prevention strategies, as demonstrated, for example, by Raizman et al.[18] Their paper, as well as the published work of the Bates-Jensen group,[11–15] and the Gefen and Gershon study,[10] demonstrated the effectiveness of the SEM scanner technology in minimising the occurrence of DTIs—the most dangerous form of PUs which develop invisibly under intact skin and is common at the sacral and heel locations of supine patients.[20] With that in mind, it is crucial, from a scientific standpoint, to understand how deep the SEM scanner can assess body tissues and which tissue layers are adequately assessed by the device. A powerful scientific method to study the depth of penetration of the SEM scanner signal for revealing information about potential subdermal damage is computer modelling and bioengineering simulations. Such bioengineering work can consider a geometry of a layered tissue structure (as depicted in Fig 2) and reconstruct it in a computer (virtual) simulation environment that captures the shape and size of the SEM sensor and the voltage applied

by the SEM scanner during SEM data acquisition. Based on this information, it can then be determined how that voltage translates to an intra-tissue electric field.

In engineering terms, this complex problem is analysed by solving Maxwell's equations of electromagnetism in the aforementioned sensor-tissue geometry, by means of an iterative (numerical) computer method called finite element analysis. Maxwell's equations are a set of fundamental equations in classical physics which describe how electric and magnetic fields are generated by electric charges, voltages or currents, and on the properties of the medium (space) in which these charges, voltages or currents act.

In the context of the SEM scanner technology, the shape and depth of the electric field generated by the SEM sensor, as defined by Maxwell's equations, will depend on the interaction of the field with the different tissue layers, each with its specific thickness and dielectric property. The shape and depth of the electric field used by the SEM scanner sensor for measuring the tissue biocapacitance is demonstrated in Fig 3. This figure uses colour to indicate the strength of the electric field of detection of the SEM scanner for realistic tissue layer thicknesses and dielectric properties. The specific computer simulation that produced the field map depicted in Fig 3 included a surface stratum corneum layer as well as epidermal and dermal tissue layers, with thicknesses representing a healthy skin structure in the young-adult population. The overall thickness of the tissue mass analysed by this modelling was chosen so that it far exceeded any possible depth of the electric field to ensure the field penetration shape (Fig 3; lower left frame) was not be truncated or disturbed due to an overly restricted simulation domain size.

This *in silico* work, which is commonly accepted in the contemporary design of medical devices at large, has supported the verification that the SEM scanner is indeed able to detect biocapacitance changes throughout the dermis and down to the superficial subcutaneous fat (Fig 3). The SEM scanner is unlikely to identify the presence of oedema that is deeper than this field of detection of the SEM sensor (3–4mm), as the biocapacitance of the tissue volume examined by the device would not be affected if deeper localised oedema develops. Nevertheless, in clinical 'real-world' scenarios, and in the absence of an effective intervention to stop or reverse the progression of damage, the 'wave' of the evolving deeper localised oedema will approach the skin surface and the SEM sensor. This will increase the biocapacitance and the associated SEM value of tissue within the sensor's depth of field and yield an increased SEM-delta value, indicating to the clinician that there may be tissue damage at this location.

As with any other clinical biomarkers used for detecting tissue damage or disease, the trend of change in the SEM-delta over time is at least as important as the absolute SEM-delta values, particularly since individual tissue tolerances vary.[4] Accordingly, a consistent trend of increasing SEM-delta values recorded over time in a specific patient and body region should warrant medical attention even if these SEM-delta values did not yet exceed the 0.6 threshold.[10] Such day-to-day escalation in SEM-deltas may possibly indicate a very early stage (potentially reversible) PU, a tendency to develop a PU under the current patient conditions, an increasing risk for occurrence of a PU, or a combination, which justifies implementation of one or more preventive interventions.[4,10]

In the context of the expected time-behaviour of SEM-delta values acquired from a specific at-risk body region, the recently published theoretical modelling work of Peko Cohen and Gefen,[21] established that there is strong non-linearity in the relationship between fluid contents in a tissue volume and the biocapacitance of that tissue volume. That is, small increments in fluid contents (related to the development of the inflammatory oedema) will progressively accelerate the change in biocapacitance and the rise in the SEM-delta over time. Their physical theory and findings, supported by laboratory test-bench experiments, explained the sensitivity of the SEM scanner to even mild tissue fluid content changes that occur in the early stage of cell and tissue damage in the PU cascade. Moreover, the work of Peko Cohen and Gefen was able to elucidate the 'exploding' nature of the SEM-delta versus the inflammatory stage, based on the theory of capacitance.[21] The pathophysiological implication of

Fig 3. An effective engineering method to evaluate the sensitivity of the SEM (subepidermal moisture) scanner in detecting subsurface tissue damage is to calculate the depth of penetration of the electric field into the tissue layers using computer modeling and simulations. A computer model simulating the bioelectrical interactions in tissues when the SEM scanner operates allows the evaluation of both the shape and depth of the electric field produced by the SEM scanner during a measurement session. The resulting field shape and depth depend on parameters of the engineering specification of the device such as the size and materials of the SEM sensor and the voltage applied to the sensor, as well as on the structural and electrical properties of the examined tissues, i.e. their thicknesses and dielectric constants. Furthermore, each of the different tissue layers may absorb and retain plasma fluids in its interstitial spaces (the layered tissue structure is illustrated at the bottom right frame), thereby changing its dielectric properties and therefore the effective biocapacitance which is measured by the SEM scanner

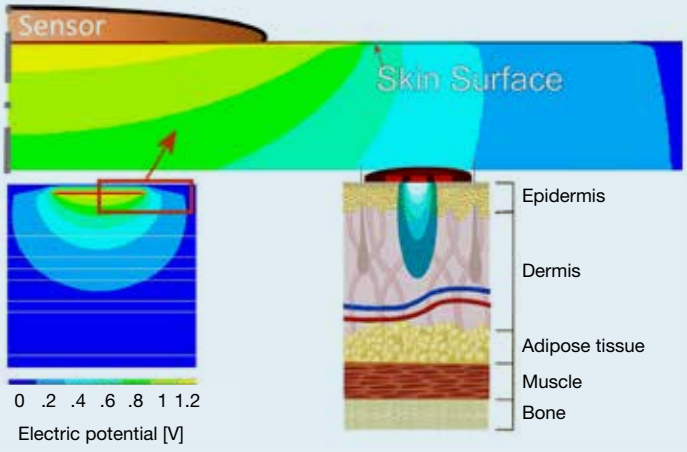
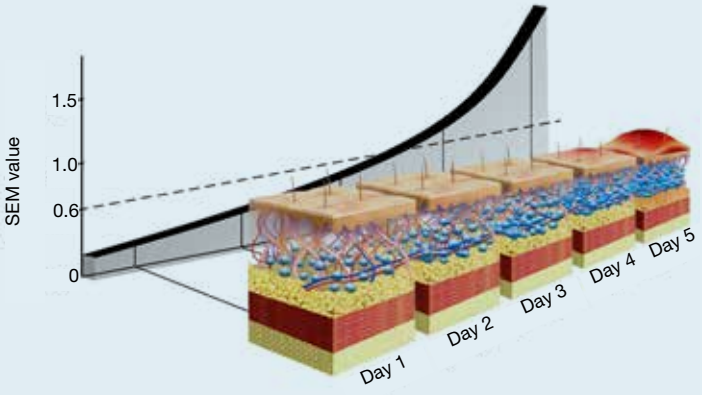


Fig 4. A schematic diagram demonstrating how subepidermal moisture (SEM)-delta values will most likely change with time as local tissue damage evolves and spreads in a forming pressure ulcer. The relationship between the increase in plasma fluid volume resulted by the tissue damage is not linear with the rise in SEM-delta levels: the increase in SEM-deltas will rapidly accelerate with the damage progression



their modelling work for a forming PU is presented in Fig 4. As the 'wave' of the localised oedema progresses upwards, as part of the development of a PU—from subdermal tissues toward the skin surface—the slope of the SEM-delta curve increases and the day-to-day changes of SEM-delta become larger (Fig 4).

Discussion

The described SEM scanner technology for identifying sub-epidermal localised (micro-) oedema that forms at tissue depths of several millimetres is an innovation in the PU prevention arena. It provides the first quantitative and clinically established technological means of assessing the health status of sacral and heel tissues (and likely in the future, at additional body regions) in patients who are at risk for PUs. This paper addresses the question of why targeting the specific physical biomarker of localised oedema, which the SEM scanner identifies as the SEM-delta, leads to the reported success in multiple large-scale clinical trials, demonstrating the ability of the SEM scanner to provide awareness of increased risk of PUs, while the injury is still invisible under intact skin.[10–18]

Fluid contents in soft tissues and the associated dielectric properties of tissues are bounded between relatively narrow physical ranges which are constituted by the tissue components, for example, cells, collagen, elastin, other proteins and the basal (healthy) interstitial water level. The electric field generated by the sensor of the SEM scanner crosses through several tissue layers, typically including the stratum corneum, epidermis and dermis and, in some cases, the superficial subcutaneous fat (Fig 2). Each of these tissue layers differs in the type of cells, proportion of extracellular matrix (ECM) and its specific composition and the local layer thickness. Accordingly, the effective dielectric property depends on the local anatomical site, health status of the individual (which globally impacts tissue layer morphology and composition) and the status of tissue and body hydration, which is further affected by temporal and long-term nutrition as well as by the ambient conditions and perspiration levels. Moreover, inflammatory conditions, either acute or chronic (for example, obesity related, neural injury related, or diabetes related inflammation), are known to alter tissue dielectric properties as does lymphoedema.[8,22–25]

Changes in dielectric properties have been proposed as a potential biophysical marker for early detection of lymphoedema—given the relation between fluid contents resulting from the impaired lymph fluid drainage and the development of the clinical condition—which affects functions of the legs or arms.[23] Tissue dielectric property changes resulting from systemic inflammation or lymphoedema would be associated with an increase in all SEM values but, importantly, they are unlikely to affect the SEM-delta. This is because the SEM-delta parameter has been specifically designed to target more localised fluid content changes, targeting the early indication of damage that may lead to PUs, by calculating the difference between SEM values from adjacent sites. In other words, any systemic disease effects will cause a similar rise of SEM readings from all tissue sites and the variability in that dataset would still be low since all the sampled tissue sites have been affected by the systemic condition. The use of the SEM-delta parameter therefore improves the specificity of the SEM scanner regardless of systemic or environmental conditions, which are assumed to have systemic impact on tissues at neighbouring locations.

The SEM scanner also promotes a cultural change in wound care. Many medical professions rely heavily on technology and cannot deliver the outcomes expected in a modern medical setting without the medical device technologies that have become the standard of care. Cardiologists, for example, are unlikely to diagnose without use of electrocardiograms, Doppler ultrasound and blood pressure monitoring, all of which provide clinical information beyond what human skills can capture with the five senses. Orthopaedists diagnose by means of radiology and similar practice exists in oncology, pulmonary medicine, neurology etc. In fact, it is difficult to think of a medical discipline other than wound care that is so deprived of technology and in which health professionals are still dependent on vision, palpation and even their sense of smell, much like in the historical roots of medicine. In that aspect, the SEM scanner paves the way for technologies to identify increased risk of PUs where the evolving PU has not revealed itself but, if identified in a timely manner, is still reversible.[3,4]

Conclusion

The SEM scanner is novel and its measurements are valuable from a clinical perspective. It is therefore likely that the same approach and technology will be extended in the future to indications other than the sacrum and heels of adult patients. Such future indications may include additional anatomical sites that are known to be

at risk of PUs (for example, the ischial tuberosities, the scapula prominences and trochanters, the occiput etc.), other wound types, for instance diabetic foot ulcers and venous leg ulcers. We may also witness future development of this technology into imaging modalities involving arrays of SEM sensors. Such SEM images would provide continuous spatial diagrams of SEM and SEM-delta values (which could be formulated as SEM spatial gradients) as previously predicted by one of the developers of the original SEM scanner technology.[24] JWC

Reflective questions

- Which electrical property of soft tissues is measured by the subepidermal moisture (SEM) scanner and why?
- What is the SEM-delta parameter and how is it useful for detecting localised tissue damage?
- Why are SEM measurements highly valuable from a clinical perspective?

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References

1 Agency for Healthcare Research & Quality (AHRQ). National scorecard on hospital-acquired conditions updated baseline rates and preliminary results 2014–2017. http://www.ahrq.gov (accessed 20 January 2020)
2 Padula WV, Delarmente BA. The national cost of hospital-acquired pressure injuries in the United States. Int Wound J 2019; 16(3):634–640. https://doi.org/10.1111/iwj.13071
3 Gefen A, Brienza D, Edsberg L et al. The etiology of pressure injuries. In: Haesler E (ed). European Pressure Ulcer Advisory Panel (EPUAP), National Pressure Injury Advisory Panel (NPIAP) and the Pan Pacific Pressure Injury Alliance (PPPIA). Prevention and treatment of pressure ulcers/injuries: clinical practice guideline (3rd edn). Cambridge Media, 2019
4 Gefen A. The future of pressure ulcer prevention is here: detecting and targeting inflammation early. EWMA Journal 2018; 19(2):7–13
5 Schwager S, Detmar M. Inflammation and lymphatic function. Front Immunol 2019; 10:308. https://doi.org/10.3389/fimmu.2019.00308
6 Gefen A. The sub-epidermal moisture scanner: the principles of pressure injury prevention using novel early detection technology. Wounds International 2018; 9(3):30–35
7 Ross, G, Gefen A. Assessment of sub-epidermal moisture by direct measurement of tissue biocapacitance. Med Eng Phys 2019; 73:92–99. https://doi.org/10.1016/j.medengphy.2019.07.011
8 Tomaselli VP, Shamos MH. Electrical properties of hydrated collagen. I. Dielectric properties. Biopolymers 1973; 12(2):353–366. https://doi.org/10.1002/bip.1973.360120212
9 Li L, Li C, Zhang Z, Alexov E. On the dielectric 'constant' of proteins: smooth dielectric function for macromolecular modeling and its implementation in DelPhi. J Chem Theory Comput 2013; 9(4):2126–2136. https://doi.org/10.1021/ct400065j
10 Gefen A, Gershon S. An observational, prospective cohort pilot study to compare the use of subepidermal moisture measurements versus ultrasound and visual skin assessments for early detection of pressure injury. Ostomy Wound Manage 2018; 64(8):12–27. https://doi.org/10.25270/owm.2018.9.1227
11 Bates-Jensen BM, McCreath HE, Kono A et al. Subepidermal moisture predicts erythema and stage 1 pressure ulcers in nursing home residents: a pilot study. J Am Geriatr Soc 2007; 55(8):1199–1205. https://doi.org/10.1111/j.1532-5415.2007.01261.x
12 Bates-Jensen BM, McCreath HE, Pongquan V, Apeles NC. Subepidermal moisture differentiates erythema and stage I pressure ulcers in nursing home residents. Wound Repair Regen 2008; 16(2):189–197. https://doi.org/10.1111/j.1524-475X.2008.00359.x
13 Bates-Jensen BM, McCreath HE, Pongquan V. Subepidermal moisture is associated with early pressure ulcer damage in nursing home residents with dark skin tones: pilot findings. J Wound Ostomy Continence Nurs 2009; 36(3):277–284. https://doi.org/10.1097/WON.0b013e3181a19e53
14 Bates-Jensen BM, McCreath HE, Patlan A. Subepidermal moisture detection of pressure induced tissue damage on the trunk: the pressure ulcer detection study outcomes. Wound Repair Regen 2017; 25(3):502–511. https://doi.org/10.1111/wrr.12548
15 Bates-Jensen BM, McCreath HE, Nakagami G, Patlan A. Subepidermal moisture detection of heel pressure injury: the pressure ulcer detection study outcomes. Int Wound J 2018; 15(2):297–309. https://doi.org/10.1111/iwj.12869
16 Guihan M, Bates-Jenson BM, Chun S et al. Assessing the feasibility of subepidermal moisture to predict erythema and stage 1 pressure ulcers in persons with spinal cord injury: a pilot study. J Spinal Cord Med 2012; 35(1):46–52. https://doi.org/10.1179/204577211X13209212104141
17 Kim CG, Park S, Ko JW, Jo S. The relationship of subepidermal moisture and early stage pressure injury by visual skin assessment. J Tissue Viability 2018; 27(3):130–134. https://doi.org/10.1016/j.jtv.2018.05.002
18 Raizman R, MacNeil M, Rappl L. Utility of a sensor-based technology to assist in the prevention of pressure ulcers: a clinical comparison. Int Wound J 2018; 15(6):1033–1044. https://doi.org/10.1111/iwj.12974
19 Clendenin M, Jaradeh K, Shamirian A, Rhodes SL. Inter-operator and inter-device agreement and reliability of the SEM Scanner. J Tissue Viability 2015; 24(1):17–23. https://doi.org/10.1016/j.jtv.2015.01.003
20 Black J, Santamaria N, Gefen A et al. Prevention and management of pressure injury to the heel. Wounds International 2018; 9(2):43–49
21 Peko Cohen L, Gefen A. Phantom testing of the sensitivity and precision of a sub-epidermal moisture scanner. Int Wound J 2019; 16(4):iwj.13132. https://doi.org/10.1111/iwj.13132
22 Schwan HP, Kay CF. Capacitive properties of body tissues. Circ Res 1957; 5(4):439–443. https://doi.org/10.1161/01.RES.5.4.439
23 Mayrovitz HN. Assessing free and bound water in skin at 300 MHz using tissue dielectric constant measurements with the MoistureMeterD. In: Greene A, Slavin S, Brorson H. (eds) Lymphoedema. Springer, 2015. https://doi.org/10.1007/978-3-319-14493-1_13
24 Wang FT. Biomedical system for monitoring pressure ulcer development. PhD thesis, University of California Los Angeles (UCLA) School of Electrical Engineering 2013. http://escholarship.org/uc/item/2cq3823d (accessed 20 January 2020)
25 Warner RR, Myers MC, Taylor DA. Electron probe analysis of human skin: determination of the water concentration profile. J Invest Dermatol 1988; 90(2):218–224. https://doi.org/10.1111/1523-1747.ep12462252