The SEM Scanner for early pressure ulcer detection: a 360-degree review of the technology

Author:
Amit Gefen

This article reviews the innovative SEM Scanner (Bruin Biometrics) technology for early detection of a forming pressure ulcer (PU) from a 360-degree perspective, considering the physiological, biophysical, medical-clinical and cost-effectiveness points of view, altogether. The SEM Scanner is designed to help healthcare professionals address a major medical need: pressure ulcer prevention (PUP). Currently, there is no technology other than the SEM Scanner for supporting clinicians in their decision-making with regards to the PU risk at specific anatomical sites of their patients. Based upon well-established physiological and biophysical principles underlying the aetiology of PUs, the SEM Scanner is targeting a specific stage in the PU cascade, in which there is a window of opportunity for detection of a localised change in the biocapacitance property of a tissue region at risk. Such change would indicate inflammatory micro-damage that may still be reversible. This is in stark contrast with the conventional clinical thinking of documenting an already existing structural tissue damage, which occurs much later in the injury spiral, typically days after the onset of the micro-damage. In other words, only when the damage becomes macroscopic and wide-spread, it can be spotted by the traditional visual skin assessment (VSA) practice or by an ultrasound examination. The benefits of a quantitative, standardised and objective early-detection of PUs, using the SEM Scanner as an adjunct to the currently subjective process of PU identification, make this device a disruptive innovation, particularly considering that the risks in using this device, if any, are negligible. The SEM Scanner technology has both proven clinical successfulness and cost-effectiveness. Risk assessment and early-detection are the two essential foundations for effective PUP.

***Amit Gefen** is Professor of Biomedical Engineering, Department of Biomedical Engineering, Faculty of Engineering, Tel Aviv University, Israel*

***Disclosure:** The author acts as a scientific advisor to multiple companies in the field of pressure ulcer/injury prevention, including to Bruin Biometrics LLC (USA) whose SEM Scanner technology is referred to in this paper. This had no influence on the conclusions from the analysis of literature which is presented here.*

This article reviews the innovative SEM Scanner (Bruin Biometrics) technology for early detection of a forming pressure ulcer (PU) from a 360-degree perspective, considering all of the physiological, biophysical, medical-clinical and cost-effectiveness points of view. The SEM Scanner is a unique technology-aid, specifically designed to help healthcare professionals address a major medical need, namely, pressure ulcer prevention (PUP) in patients who are immobile or insensate. Currently, there is no diagnostic or risk as-sessment technology other than the SEM Scanner for supporting clinicians in their subjective decision-making with regards to the PU risk or the early diagnosis of a forming injury at specific anatomical sites of their patients. In this work, the SEM Scanner technology is explained in a non-technical language and the benefits of a quantitative, standardised and objective early detection of PUs using the device (as an adjunct to

the currently subjective process of PU identification) are reviewed. As will be described in this article, the SEM Scanner is clearly a disruptive innovation in the practice of PUP, which should be deployed (based a site-specific cost-benefit evaluation) wherever at-risk patients may be admitted, particularly considering that the risks in using this device, if any, are negligible. As reviewed here, the SEM Scanner technology has both proven clinical successfulness and cost-effectiveness, allowing PUP to finally modernise and become technology-aided.

The contemporary and mainstream published knowledge on pressure ulcer (PU) aetiology

During 2018-2019, the author chaired the Aetiology Working Group responsible for writing the Aetiology Chapter of the 2019 International Guideline for Pressure Ulcer/Injury Prevention and Treatment and have led this panel of experts to publication of the most comprehensive, rigorous and up-to-date work thus far on the aetiology of pressure ulcers (PUs), analysing over than a 100 recent research papers in the field. That contemporary, mainstream published knowledge on PU aetiology, which is detailed in the above 2019 version of the International Guideline is summarised below.

Pressure ulcers are injuries that may develop over a timescale of minutes to hours under sustained tissue deformations. Tissue damage in PUs does not appear instantaneously, but rather, develops from the cell scale to the mesoscale and grows to the tissue level and finally, presents itself on the skin surface and often causes skin and underlying tissue breakdown. This implies that in PUs, the damage spiral onsets and progresses from the micro to the macro. Our current fundamental understanding described in the above 2019 guideline is that this damage spiral ultimately leading to PUs is triggered and then driven by cell and tissue exposure to sustained mechanical deformations, or, in bioengineering terms, to mechanical stress concentrations in soft tissues.

Any bodyweight or device-related forces that cause sustained soft tissue distortions generate large deformations of the cells contained within the affected tissues, with the greatest tissue and cell deformations occurring where these forces are concentrated. With respect to sustained bodyweight forces, the most influenced soft tissue sites are typically found in deep tissue layers under bony prominences, where the highly curved and 'sharp' bone surfaces come into contact with easily deformable soft tissues. The bodyweight forces, which are transferred through the sharp and rigid bony elements, cause large distortions in the soft tissue structures that they encounter, such as under the sacral or calcaneal (heel) bones, with the highest distortions occurring near the sharpest bony surfaces. This is the reason for the tissue damage to typically occur first in the deeper tissues and only then progress towards more superficial layers, until eventually presenting itself on the skin. At the cell scale, the continuous exposure to such mechanical forces that deform soft tissues would gradually damage the integrity of the cytoskeleton — the complex protein scaffold that makes the structural framework of cells. The exterior walls of the cell, called the plasma membrane, are structurally supported by the cytoskeleton. When the cytoskeleton becomes unable to continue providing the sufficient mechanical support to the plasma membrane, pores will form on the membrane. Poration of the plasma membrane will rapidly lead to abnormal transport of ions and molecules from within cell bodies extracellularly, and from the extracellular space inwards into the cell bodies. The inability of multiple cells to control their mass transport yields loss of homeostasis, which results in *en masse* apoptosis within a timeframe of just minutes.

When these multiple cells have been damaged or have died as a direct result of the sustained tissue deformations as described above, the damaged cells and nearby immune cells release pro-inflammatory cytokines, which are signaling proteins that function to attract additional immune cells. This signalling is a programmed normal response which is essential for healing. Recruitment of a large number of immune cells is primarily aimed at counteracting pathogens, clearing dead cell debris and preparing the ground for tissue regeneration. However, in the specific context of PU aetiology, the inflammatory singling itself is a potential contributor to the injury spiral, considering the effects of the pro-inflammatory cytokines on the endothelium in the vasculature adjacent to the initial damage site. Specifically, the secreted pro-inflammatory cytokines act to dilate capillaries and increase the permeability of capillary walls near the initial damage site, by relaxing endothelial cell tight-junctions. This endothelium relaxation facilitates leukocyte extravasation — the migration of immune cells from the blood circulation to the initial damage site. However, the endothelium relaxation also results in leakiness of the vasculature near the damage site and so, plasma fluids build-up in the interstitial tissue spaces, which forms localised oedema. Of note is that this localised oedema, which results from the mechanical insult is fundamentally different from a systemic oedema. Systemic oedematous conditions are typically caused by sodium retention in tissues,

which is associated with heart, liver or kidney dysfunction, or due to a lymphatic disease resulting in lymphoedema, whereas a localised oedema is a characteristic result of a normal immune response trigged by localised tissue damage to allow leukocyte extravasation, as explained above.

Often in a developing PU, soft tissue expansion due to a forming localised oedema is mechanically limited, for example, because the soft tissues are constrained between a bony element and a support surface (e.g. between the sacrum and a mattress). If the affected soft tissues cannot sufficiently expand in volume, the interstitial pressures would increase sharply, causing further cell deformation and thereby, additional deformation-induced cell death. Under such conditions, the inflammatory process would then cause release of reactive oxygen and nitrogen species to degrade the extracellular matrix in an effort to relieve the rising interstitial pressures, which will cause further tissue damage, now to the extracellular structures.

At a certain stage, the growing interstitial pressures may reach a level that would cause obstruction of the vasculature itself, which will impair blood perfusion into the affected tissue site and, thereby, trigger ischaemic damage. These synergistic interactions between sustained cell and tissue deformations, inflammation and ischaemia form the vicious cycle of the development and progression of PUs as we currently understand it. The description of the aforementioned vicious cycle *[Figure 1]*, is the core of the Aetiology Chapter of the 2019 Guideline. The contents of the 2019 Aetiology Chapter visualised in the Figure represent the contemporary understanding from the past decade — a vast change and progress with respect to earlier knowledge.

Of note, inflammation is a critical juncture where the post-injury cascade of events is determined, i.e., whether an early-stage, developing PU will heal normally (without leaving clinically significant tissue damage) or otherwise, would shift to a chronicity state (Cutting and Gefen, 2019). Specifically, the nature of the inflammatory signaling and the associated localised oedema *[Figure 1]* are central factors in any healing process and will ultimately determine the 'fate' of the wound, that is, a good healing and closure outcome, or alternatively, chronicity (Cutting and Gefen, 2019). Conditions of uncontrolled inflammation such as those reported in COVID-19 augment the tissue swelling or the increase in interstitial pressure levels, which then causes a wider spread of the secondary cell death and tissue damage, due to the resulting high cell distortions *[Figure 1]*. Inflammatory signalling further impacts the lymphatic system and as commonly known, typically causes swelling of lymphatic nodes, which adds to the mechanical loading on adjacent cells and, therefore, to the potential for cell damage.

The SEM Scanner is designed to function based upon this contemporary aetiological understanding of PUs and targets the inflammatory phase in the formation of PUs which is characterised by localised accumulation of plasma in the interstitial compartments. Noteworthy is that the localised nature of plasma fluid accumulation in soft tissues due to a forming PU is inherently different from a systemic oedema mechanism, in both the pathophysiology and clinical outcomes, as described earlier.

As mentioned in multiple places in the 2019 guideline, there are a number of physical and chemical biomarkers that characterise the inflammatory phase in PU formation and among these biomarkers, biocapacitance is a very robust biophysical measure of the localisation and extent of the tissue damage. While systemic oedema may develop due to a variety of causes e.g. heart failure, low protein levels, liver or kidney diseases, a localised oedema in a person who is at-risk for PUs will very likely indicate a forming PU. The SEM Scanner is specifically detecting a localised oedema (as opposed to a systemic oedema) by comparing the biocapacitance marker, which correlates with the interstitial fluid content across different tissue locations, e.g. in multiple tissue sites around the sacrum.

The difference between the biocapacitance readings acquired at multiple different tissue locations, which is quantified by the SEM-delta measure, represents the inhomogeneity in interstitial fluid distribution, which only increases if one specific site — a PU formation site — starts accumulating plasma due to a locally inflamed, leaky vasculature *[Figure 1]*. Currently, there is no other feasible technological alternative to use of biocapacitance as the biophysical measure of the build-up of this local inflammatory cell and tissue damage that points to an early-stage, but still likely reversible damage.

The inextricable links between COVID-19 and the pathophysiology of PUs

Based on recent Italian data reported in the literature, a rate of 12% of all positive coronavirus disease 2019 (COVID-19) cases required admission in an intensive care unit (ICU) and the ICU length of stay with this diagnosis is relatively long. At the time of writing this article, there are already nearly 10m positive COVID-19 cases (*www.worldometers.info* accessed on June 25, 2020), which is indicative of approximately 1.2m ICU patients who have already been added or will be added to the healthcare

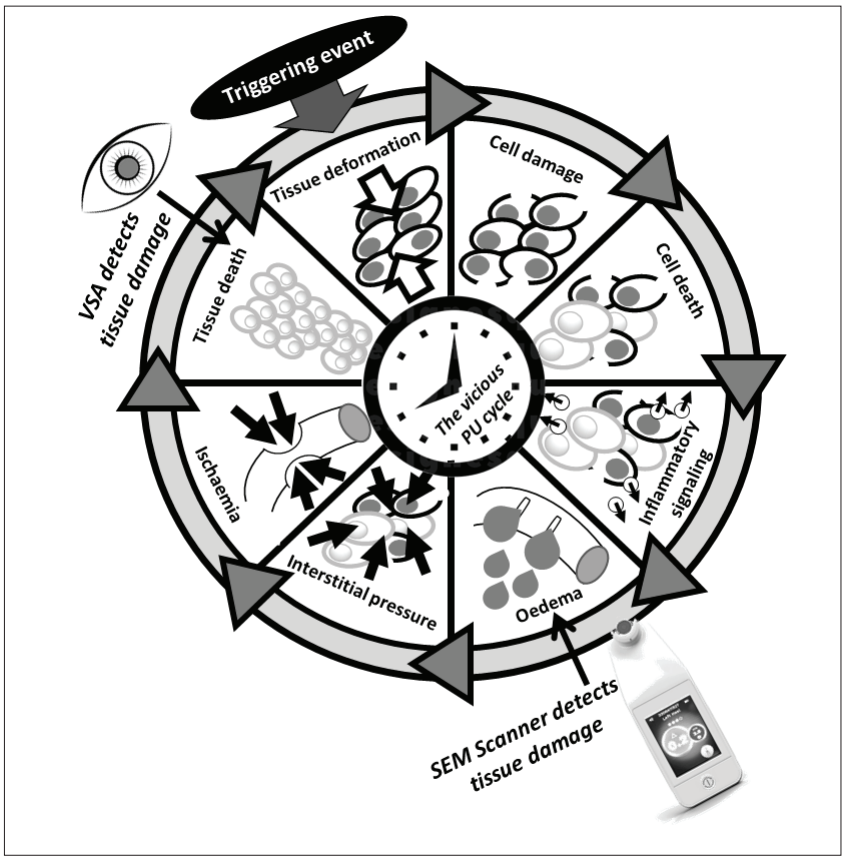

Figure 1. The vicious cycle of pressure ulcer formation and its progress with time. Based on the changes in interstitial fluid contents resulting from the build-up of the oedema The SEM Scanner is able to detect the forming cell and tissue damage early in the cascade, where damage is still at a micro-scale and is highly likely to be reversible. A visual skin assessment, in contrast, will detects an already-existing, macroscopic tissue damage which is unlikely to be reversible.

system worldwide since the outbreak of the pandemic in the western hemisphere, in February 2020. In the context of this current widespread of the first wave of COVID-19, where many of the newly admitted ICU patients are anaesthetised for mechanical ventilation and are, therefore, by definition, at-risk for PUs, it is important and relevant to discuss how COVID-19 interacts with the known aetiological factors described above (please see a comprehensive review of this topic in Gefen and Ousey, 2020 and the monthly updates to this paper).

First, COVID-19 activates the immune system promptly and sharply, which positions COVID-19 patients with a cytokine release syndrome (also known as 'cytokine storm') at a high risk for developing PU-related inflammatory tissue damage. This is because their inflammatory response is unleashed and their cytokine sensitivity thresholds are, therefore, disrupted. In addition, COVID-19 patients are also at a high risk for PU-related ischaemic tissue damage as their oxygen saturation levels are typically low and their cardiac output may be abnormal, e.g. due to myocarditis, acute myocardial infarction or heart failure, all of which are reported cardiovascular complications of COVID-19.

Another potential contributor to tissue ischaemia in COVID-19 is the hypercoagulability leading to a tendency for thrombosis in these patients. These timely examples illustrate how COVID-19 interacts directly with two of the three primary aetiological factors in the vicious cycle of PUs, inflammation and ischaemia and further suggest that COVID-19 may be a confounder of PUs. Indeed, the prevalence rate of PUs in ICUs among COVID-19 patients could be 10-times or more the respective PU rates at the same ICUs prior to the COVID-19 outbreak (Gefen and Soppi, 2020). Considering that already before the COVID-19 outbreak, PUs were a well-recognised independent prognosticator of death among ICU patients, the interaction of the cytokine storm in COVID-19 with the inflammatory damage factor in the PU spiral underpins the importance of PUP for this particular patient population (Gefen and Soppi, 2020). Based on its underlying physical and physiological principles described above, the SEM Scanner as an adjunct to clinical judgment can be a very effective tool for this task.

Visual skin assessments, palpation examinations and pain complaints as limited indications for PU diagnosis

The process by which serious, hospital-acquired deep PUs form under intact skin, spread in deep tissues and eventually present themselves as full-thickness wounds has been rigorously described in the medical literature in the last decade, from a basic science and aetiological perspectives. The mechanobiology of such PUs is that soft tissue damage initiates near bony prominences — typically the sacrum and heels. The force of concentrated bodyweight under these bony prominences causes intensified and sustained cell and tissue deformations which compromise cell integrity, transport function, leading to cell death and eventually, to massive tissue death *[Figure 1]*. Since these PUs may not form initially on skin, even the best nursing skills and diligence relating to tissue care will be ineffective in achieving timely detection of sub-epidermal injuries. In other words, without an insight into deep-tissue health status and viability, there is no feasible way for a nurse relying on current risk assessment scales, visual skin assessments (VSAs) or physical palpation examinations (including where the nurse is attempting to probe skin surface temperature changes) to detect the developing injury in a timely

way (Takahashi et al, 2017; Gefen, 2018; Gefen and Ross, 2020; Gefen et al, 2020). It is not surprising therefore that these deep PUs, which emerge at the skin surface only after considerable deeper tissue damage has already been caused, are the ones associated with the majority of the global expenditure on treating PUs (Gefen et al, 2020).

In terms of nursing time, VSAs cost approximately £6 per patient, per skin check session (Gefen et al, 2020). Accordingly, conducting routine VSAs for each and every hospitalised patient is financially implausible and hence, regular VSAs are only conducted for patients who are determined to be at-risk for PUs based on the outcome of a risk assessment tool upon admission. If VSAs would have been hypothetically implemented for all patients routinely during their hospitalisation period, the result will be spending of many billions of pounds sterling on patients who will never be at a meaningful risk, as only a small fraction are at a true risk for PUs. Indeed, current risk assessments typically classify up to two of five of all hospitalised patients as being at a high risk for developing PUs, but the sensitivity and specificity of risk assessments is often criticised, given the unacceptable extent and rate of deaths from PUs and the total expenditure on PUs (Oliveira et al, 2017; Padula and Delarmente, 2019; Gefen et al, 2020).

Importantly, even for patients correctly identified to be at-risk by risk assessments, who receive a high-specification support surface, as well as other best-practice prophylactic interventions and repositioning, nursing staff will never be able to detect a deep tissue injury (DTI) evolving under intact skin by means of the VSAs. The VSAs currently used in practice are only able to detect the DTI once the damage has reached the skin, which is clearly too late. This simple logical flaw in classic PUP strategies points to the true barrier to effective PUP and to the associated cost reductions: the lack of a reliable technology, based on solid physical and physiological foundations, to evaluate the tissue health of patients under an apparently normal skin at specific anatomies.

Another common misconception hindering the timely clinical diagnosis of PUs is that patients who develop PUs will complain about discomfort or pain. Pain is not a good predictor of PUs, particularly where there is an impaired sensation due to central or peripheral neural damage caused by injury or disease or anaesthetics, sedation or any medications which affect sensation. Pain only becomes relevant where a person is able to sense (but not necessarily move), which is not the case for the majority of the at-risk patients. For example, one (relatively rare) condition where discomfort or pain may predict a later onset of a PU would be a locked-in syndrome (pseudocoma) where a person loses their ability to move, but can still sense discomfort (Gefen and Soppi, 2020).

The SEM Scanner's mode of action

The SEM Scanner *[Figure 1]* measures the biocapacitance of the local skin and subdermal tissues under its sensor. As mentioned above, the biocapacitance is a temporal and spatial physical property of the tested tissue region, and more specifically, a bioelectrical property that is the ratio of the change in an electric charge in the scanned tissue region to the corresponding change in its electric potential (Gefen, 2018; Peko Cohen and Gefen, 2019; Ross and Gefen, 2019; Gefen and Ross, 2020).

A large self-biocapacitance of a tissue region indicates that this tissue 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 architecture, which in the context of a SEM Scanner measurement is the area of the sensor of the device and the composition of the examined soft tissues in the immediate vicinity of the sensor, especially the dielectric properties of these tissues. For tissues, as with many dielectric materials, the biocapacitance is independent of the electrical potential applied by the SEM sensor. The biocapacitance of tissues is, however, variable and highly sensitive to the interstitial water content of the tissue (Gefen, 2018; Peko Cohen and Gefen, 2019; Ross and Gefen, 2019; Gefen and Ross, 2020).

The dielectric constant of water (which is approximately 80) is 10 to 20-times greater than that of all solid tissue components, e.g. collagen and elastin. In a certain anatomical region, with a given anatomical configuration, the SEM Scanner reading of biocapacitance will be predominantly affected by the dielectric tissue properties, which are, in turn, 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 despite visible (clinical) signs of it have not developed yet (Gefen, 2018; Peko Cohen and Gefen, 2019; Ross and Gefen, 2019; Gefen and Ross, 2020).

The SEM Scanner reports the level of biocapacitance of a tissue site as a non-

dimensional '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 extent of the developing inflammatory oedema and, therefore, the potential tissue damage to be expected at the scanned site. Indeed, in our published work, we could identify the formation of a heel PU in a patient under their intact skin (i.e. a heel DTI) through a consistent rise in the SEM-delta readings at the examined heel, 2 days before VSA indicated tissue damage and importantly, 3 days before the appearance of a hypoechoic lesion demonstrating the fully-developed macroscopic oedema in an ultrasound examination of that same heel (Gefen and Gershon, 2018). This is strong evidence of the detective power of the SEM Scanner in identifying the forming oedema under a spotless skin, already at the initial, microscopic phase of the oedematous development.

The SEM-delta is an objective, quantitative and standardised reading of the tissue health conditions, wherein a low SEM-delta indicates healthy tissue and a high SEM delta points to a local inflammation as a result of localised cell and tissue death. In particular, a trend of increase in 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. What is noteworthy is that if there is a condition of systemic oedema, e.g. lymphoedema or heart or kidney dysfunction, the SEM values acquired at adjacent points will be similar and hence, the SEM-delta would be low. Accordingly, selection of the SEM-delta measure (rather than the individual SEM values) allows to distinguish a localized inflammatory process which most likely indicates a forming PU from any systemic increase in interstitial fluid contents, either normal or abnormal (Ross and Gefen, 2019; Gefen and Ross, 2020).

Using laboratory bioengineering phantoms of soft tissues in organs (the head and heels), the author and his research group have demonstrated in their published work (Peko and Gefen, 2019; 2020) that indeed, the SEM Scanner is able to detect intra-tissue fluid content changes that are as small as 1 millilitre and that the SEM-delta reading is sensitive to these changes. The latter findings were shown to be robust and reproducible for both the SEM-200 (first generation) model and the new SEM-250 (second generation) Scanner model (Peko and Gefen, 2019; 2020).

The clinical efficacy of the SEM Scanner

It is a striking fact that the SEM Scanner technology has been tested in clinical trials more than any other emerging preventative/diagnostic technology in the PU arena, which is known to the author. Specifically, there are multiple clinical studies published in the peer-reviewed wound care literature, which reported a significant diagnostic value of the SEM Scanner, leading to improved health care and reduction in treatment costs post implementation of the device in the care practice. This published literature is reviewed in the work of the author, which is cited here and in particular, in Gefen and Gershon (2018). One example from the latter paper is given below, to demonstrate the clinical importance, applicability and usefulness of the SEM Scanner in different clinical settings.

A clinical study was conducted to evaluate consistency between the SEM Scanner and ultrasound examinations of suspected deep PUs under intact skin, known as DTIs. Specifically, using an observational, prospective cohort study design, patients >55 years of age were recruited (Gefen and Gershon, 2018). In addition to SEM Scanner measurements, conventional VSAs, as well as ultrasound assessments were further performed. These examinations were conducted daily for a minimum of 3 and maximum of 10 consecutive days following patient enrollments. The ultrasound results were considered indicative of a DTI if hypoechoic lesions were present in the acquired images. The SEM Scanner readings were considered abnormal when the SEM-delta at a specific body region (sacrum or each of the heels) was equal or greater than 0.6 for at least 2 consecutive days.

Boolean analysis was utilised to systematically determine the consistency between the ultrasound and SEM Scanner readings where DTI was the clinical judgment. Among the 15 participants (10 of whom were women, mean age 74 ± 10.9 years), which were, in general, a nursing home population at a high risk for PUs, there was consistent agreement between the SEM Scanner readings and ultrasound when DTIs existed. Noteworthy is a case of a patient, which has been reported in another paper (Gefen and Gershon, 2018), where the patient developed a heel DTI during the study. Their SEM Scanner readings in that case were abnormal 2 days before VSA indicated tissue damage and 3 days before the appearance of a hypoechoic lesion in the ultrasound.

Given our current aetiological knowledge, the ability of the SEM Scanner to detect the injury at such an early stage, prior to it being visible on the skin or even detectable under the skin by means of ultrasound, is due to the fact that the SEM Scanner targets early, microscopic damage associated with

inflammation, whereas both ultrasound and VSAs document existing, macroscopic structural damage to tissues *[Figure 1]*. It is not surprising therefore that with respect to subdermal tissue damage or DTIs, ultrasound and SEM Scanner results in Gefen and Gershon (2018) were similar. Moreover, in the evolving DTI case monitored during the aforementioned study, the SEM Scanner detected a lesion earlier than the ultrasound.

As per the medical claims made by the manufacturer, the SEM Scanner is currently being suggested as an adjunct to VSAs, not as a replacement of these conservative manual examinations. Despite the common conception, there is no point in validating the SEM Scanner measurements against the VSAs conducted in a medical facility where the SEM Scanner is considered for use, since VSAs document existing macroscopic structural tissue damage, whereas the SEM Scanner detects early, microscopic-scale damage. The latter event occurs at an earlier time point on the timeline of the PU damage cascade and so, the technology-aided SEM-delta readings should always be abnormally elevated prior to a positive (and subjective) VSA diagnosis, as in the above example Gefen and Gershon (2018) study. Indeed, a large volume of other, independent clinical studies have been reported in the literature and are reviewed in the published work of the author; all of these consistently demonstrated the early-detection feature of the SEM Scanner, which is not surprising based on the known PU aetiology as reported in the 2019 International Clinical Guideline.

Cost-benefit analyses of the SEM Scanner

In collaboration with the manufacturer and a panel of external expert health economists, the author has published a comprehensive cost-benefit analysis focusing on the financial savings associated with implementation of the SEM Scanner technology in hospital settings (Gefen et al, 2020). The latter paper is, in fact, the first ever to report the predicted savings that a diagnostic PUP technology may achieve. Specifically, in the above study, implementation of the SEM Scanner technology as an adjunct to the current VSA standard of care practice has been tested using probabilistic cost-benefit modelling. The author developed a decision-tree model type and Monte Carlo simulations representing the various pathways of care that 10,000 patients, admitted to NHS hospitals in the United Kingdom, may experience.

The author tested two alternate acute hospital scenarios, of lower (1.6%, categories 1–4) and higher (6.3%, categories 1–4) PU incidence rates. Under a conservative range of assumptions and input parameters, we found that implementation of the SEM Scanner technology as an adjunct to the current standard of care is highly likely to lead to significant financial benefits and cost savings. For example, our modelling demonstrated that the expected saving per patient, by routine implementation of the SEM Scanner in care facilities with the above low and high incidence rates, is £15.23 and £80.68 per admission, respectively. For an average UK Trust with 40,802 admissions (excluding day cases) per annum, the estimated total financial savings from implementing the SEM Scanner, using the assumptions and inputs set out here, would range between £0.6m to £3.3m per annum. These cost reductions, even under our conservative modelling assumptions, reflect the above explained (i) detection and treatment of anatomy-specific, non-visible tissue damage which is not possible without the SEM Scanner, (ii) higher rates of detection of category 1 PUs than possible without the technological aid of the SEM Scanner, and (iii) avoidance of some unnecessary treatments of patients without PUs, due to higher confidence by clinicians to rule out PUs with the SEM Scanner readings than without.

The fundamental basis of the above cost-benefit analyses is that patients are in a given PU-state (no damage, sub-clinical damage, Stage 1 or later damage) and, accordingly, the author modelled changes in the probability of correct detection of that state with and without the SEM Scanner. Savings from the aforementioned factors (i) and (ii) arise from earlier and more sensitive diagnostic accuracy of skin and tissue deterioration in the earliest phases of damage, as indicated by the SEM Scanner as an adjunct to VSA, however, the author and colleagues assumed that the efficacy of treatments remains the same as without the SEM Scanner in place. In other words, these considerable savings are from properly including patients with developing but invisible PUs into the care pathway and properly excluding patients without developing PUs from the care pathway who would otherwise have been deemed at risk (which is saving point no. iii above). Accordingly, the work reported in Gefen et al (2020) clearly demonstrates that wide implementation of the SEM Scanner technology in the UK, as well as in other countries, is well justified from a financial perspective and will lead to cost savings. While more research is in need to further establish the cost-benefits of the SEM Scanner, in particular in specific clinical settings, e.g. geriatric or rehabilitation centers,

no other diagnostic PUP technology has ever been investigated so rigorously in breadth and depth as the SEM Scanner was (Gefen et al, 2020) for its financial justification.

Bioengineering evaluation of the second generation SEM Scanner

In the second-half of 2019, Bruin Biometrics LLC introduced a 2nd-generation SEM Scanner model called Provizio™ SEM Scanner. This new version of the SEM Scanner is elegantly designed to include an improved user interface and better wireless connectivity. Peko and Gefen (2020) have conducted a bioengineering study to evaluate the sensitivity of Provizio™ SEM Scanner in identifying fluid content changes in laboratory phantoms of a human heel and skull/face, relatively to their 1st-generation SEM measurement device (also known as the SEM 200 model). They performed SEM measurements on the aforementioned physical phantoms simulating the head and heels of an examined patient, as described in their previously published work (Peko and Gefen, 2019).

Following the experimental protocol detailed in the latter publication, they injected 1ml ('reference'), 2, 3 and 4ml of water to the 'soft tissue' substitutes in each phantom and location. Next, Peko and Gefen (2019) calculated the corresponding SEM-delta, which quantifies the dimensionless difference in these experiments between the biocapacitance properties of the 'soft tissues' at the reference (1ml) site versus each of the 2, 3 and 4ml sites simulating inflammatory oedema. Finally, they conducted Bland-Altman (B&A) statistical analyses to determine the levels of statistical agreement between the Provizio™ SEM Scanner and previous (200 model SEM Scanner) device readings, for each phantom type and location.

Consistent with their published work concerning the 200 model of the SEM Scanner (Peko and Gefen, 2019), the Peko and Gefen (2020) studies of the Provizio™ SEM Scanner demonstrated that this device is sensitive enough to detect water content variations that were as small as 1 ml. Furthermore, the above B&A analyses established that any differences in readings between the Provizio™ and 200 model of the SEM Scanner were clinically negligible. In addition, these differences did not tend to become larger as the mean of the two device readings increased, which indicates stability and precision of both devices. Hence, the Provizio SEM Scanner was shown to perform identically to the 200 model SEM Scanner in laboratory experiments evaluating its sensitivity to small water content variations within physical phantoms of human body tissues (Peko and Gefen, 2020).

Furthermore, the Provizio SEM Scanner is also substantially more compact and user-friendly, has a smaller sensor which facilitates easier access to small and/or curved body sites, and it features improved connectivity with other medical data systems in hospital settings (Peko and Gefen, 2020).

Summary and conclusions

The BBI LLC (Bruin Biometrics) SEM Scanner technology addresses a major and unmet medical need in prevention of PUs and supports healthcare professionals who are currently not supported by any other technology to aid in their clinical decision-making with regards to the PU risk at specific anatomical sites of individuals. The SEM Scanner is built upon well-established physiological and biophysical principles, which were explained here. The SEM Scanner is targeting a specific stage in the PU injury cascade in which there is a window of opportunity for detection of a localised change in the biocapacitance property of a tissue region at risk. Such change in the local tissue biocapacitance would indicate inflammatory micro-damage that may still be reversible *[Figure 1]*. This is in stark contrast with the conventional clinical thinking of documenting an existing macroscopic, structural tissue damage, which occurs much later in the injury spiral (typically days after the onset of the micro-damage) and only then, that structural damage can be spotted by VSAs or ultrasound examinations.

Published literature by the author and by others clearly shows that the above theoretical basis is well supported by clinical data, including laboratory bioengineering work as well as large clinical trials. There is no current feasible technological alternative to the use of biocapacitance, the biophysical measure used by the SEM Scanner technology, for detecting the inflammatory stage of cell and tissue damage in PUs. The benefits of a quantitative, standardised and objective risk assessment and early detection of PUs using a technological tool — the SEM Scanner — to aid and support the currently subjective process of PU identification are significant, and the risks in using the device, if any, are negligible. The SEM Scanner technology has proven cost-effectiveness, demonstrated in comprehensive published work, which has been summarised above. Risk assessment and early-detection

are the two essential foundations for effective PUP, which can finally be based on modern and relevant medical technology — the SEM Scanner — rather than just the art and subjective clinical skills. WINT

References

Cutting K, Gefen A (2019) *Made Easy: PolyMem and Countering Inflammation*. Wounds International: London. Available at: https://www.woundsinternational.com/resources/details/made-easy-polymem-and-countering-inflammation (accessed 08.09.2020)

Gefen A, Gershon S (2018) 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* 64(9): 12–27

Gefen A (2018) The sub-epidermal moisture scanner: The principles of pressure injury prevention us-ing novel early detection technology. *Wounds International* 9(3): 10–5

Gefen A, Brienza D, Edsberg L et al (2019) *The etiology of pressure injuries. In: Prevention and Treatment of Pressure Ulcers/Injuries*. European Pressure Ulcer Advisory Panel (EPUAP), National Pressure Injury Advisory Panel (NPIAP) and the Pan Pacific Pressure Injury Alliance (PPPIA) (3rd edn) EPUAP-NPIAP-PPPIA: Westford MA, USA.

Gefen A, Kolsi J, King T, Grainger S, Burns M (2020) Modelling the cost-benefits arising from technology-aided early detection of pressure ulcers. *Wounds International* 11(1): 22–9

Gefen A, Ross G (2020) The subepidermal moisture scanner: the technology explained. *J Wound Care* 29(Sup2c): S10–S6

Gefen A, Ousey K (2020) Update to device-related pressure ulcers: SECURE prevention. COVID-19, face masks and skin damage. *J Wound Care* 29(5): 245–59

Gefen A, Soppi E (2020) What is new in our understanding of pressure injuries: the inextricable association between sustained tissue deformations and pain and the role of the support surface. *Wound Practice and Research* 28(2): 84–91

Oliveira AL, Moore Z, O Connor T, Patton D (2017) Accuracy of ultrasound, thermography and subepidermal moisture in predicting pressure ulcers: a systematic review. *J Wound Care* 26(5): 199–215

Padula WV, Delarmente BA (2019) The national cost of hospital-acquired pressure injuries in the United States. *Int Wound J* 16(3): 634–40

Peko Cohen L, Gefen A (2019) Phantom testing of the sensitivity and precision of a sub-epidermal moisture scanner. *Int Wound J* 16(4): 979–88

Peko L, Gefen A (2020) Sensitivity and laboratory performances of a second-generation sub-epidermal moisture measurement device. *Int Wound J* 17(3): 864–7

Ross G, Gefen A (2019) Assessment of sub-epidermal moisture by direct measurement of tissue bio-capacitance. *Med Eng Phys* 73: 92–9

Takahashi Y, Mizokami F, Isogai Z (2017) Palpation for pressure ulcers: examining the bony promi-nence and physical properties of the wound. *Int Wound J* 14(6): 1402–4