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Sub-epidermal moisture measurement: an evidence-based approach to the assessment for early evidence of pressure ulcer presence

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Abstract

This paper aims to discuss the literature pertaining to early pressure-shear induced tissue damage detection, with emphasis on sub-epidermal moisture measurement (SEM). The current method for pressure detection is visual skin assessment (VSA); however, this method is fraught with challenges. Advances in early detection of pressure ulcers are reported in the literature and mainly involve measuring inflammation markers on weight-bearing anatomical areas in order to capture the first signs of tissue damage. One novel technique currently in use is SEM measurement. This biophysical marker is the product of plasma that leaks as a response to local inflammation arising due to pressure-shear induced damage over bony prominences. The early detection of tissue damage is beneficial in two different ways. First, it enables early intervention when the damage is still microscopic and reversible and, therefore, has the potential to prevent further aggravation of healthy surrounding tissue. This arises by avoiding the causation of the problem and stopping the knock-on effect of inflammation, especially when the rapid pressure ulceration pathway of deformation is in place. Second, when the slow ischaemic-reperfusion related mechanism is undergoing, cell death can be avoided when the problem is identified before the cell reaches the "death threshold," completely averting a pressure ulcer.

KEYWORDS

pressure ulcer, risk assessment, screening tools, sub-epidermal moisture, wound care

1 | INTRODUCTION

A pressure ulcer (PU) is defined as "localized injury to the skin and/or underlying tissue usually over a bony prominence, as a result of pressure, or pressure in combination with shear."[1] It is believed that pressure and shear are the primary cause of the tissue damage and some risk factors, such as activity, mobility, age, continence, and nutritional status increase the probability of pressure ulcer development.[1] Patients with activity/mobility impairment are those at highest risk of developing a pressure ulcer, due to their inability to safely reposition themselves, leaving them exposed to prolonged unrelieved pressure/shear.[2,3]

O'Brien, Moore[4] studied the urban point prevalence of wounds over 1 week in Ireland and found a 10% prevalence of pressure ulcers within the acute care setting. The authors reported that stage II pressure ulcers

were the most common category (36%; N = 27), while the sacrum was the most affected anatomical location (45%; N = 35). In the United States, VanGilder, Lachenbruch[5] measured pressure ulcer prevalence, between 2006 and 2015 across all American types of facilities. Figures for long-term care spanned from 9.5% (2012) to 12.0% (2010). In Australia, the Clinical Excellence Commission[6] reported an overall prevalence for 2017 of 7.9%, varying from 7.7% to 9.5%, and, in the residential aged care setting, they noted an overall prevalence of 7.7%, which was very similar to figures from acute care.

From the epidemiological data, it is clear that PUs are still challenging clinicians throughout the world and within many different clinical settings. For this reason, a comprehensive skin assessment is a fundamental part of pressure ulcer diagnosis and prevention, as the information gathered can better assist healthcare professionals in the clinical decision-making process.[7] This evaluation process should be holistically performed on admission/patient encounter and should also be integrated into the daily patient assessment.[8]

The skin inspection process is still widely being performed using visual skin assessment (VSA). However, it is very challenging to visually diagnose a pressure ulcer in its early stages, as the problem usually starts in the sub-epidermal layers, making any visual identification of tissue damage is very difficult.[9,10] Thus, when the problem is discovered visually, it may be already too late to prevent its further development.

PU classification and differentiation, in general, are also challenging and the reliability of classification of pressure ulcers by clinicians is inconsistent.[11,12] For example, Defloor, Schoonhoven[13] undertook a two-phase study, where, in phase 1 the authors included 473 nurses who classified photographs of 56 skin lesions into the categories of normal skin, incontinence lesion, blanchable erythema, and pressure ulcers category I to IV.[14] The agreement in classification among the nurses was κ 0.37 (of note a $\kappa > 0.70$ indicates good agreement). In phase 2, 86 different nurses undertook an assessment on two different occasions with results of $\kappa = 0.38$ on the first assessment and $\kappa = 0.43$ on the second assessment.[13] The overall conclusion was that the interrater reliability of the classification of lesions by nurses was very low. Further, there was a major problem in the differentiation between pressure ulcers and incontinence lesions.

Besides the challenge faced with different classification systems, the specific classification of depth and severity of pressure ulcers is also difficult.[1] UsingVSA, an attempt is made to take into consideration subjective parameters, such as the colour of the skin (red, purple, and bruised). Furthermore, a "guess" is often made as, to which layers of soft tissue might be involved in the damaged area until a fuller development of the ulcer becomes evident. To improve classification and diagnosis of a problem that usually starts from within the deeper layers, more objective approaches based on the pressure ulcer aetiology would substantially contribute not only to an enhanced classification system but also to early detection of tissue damage present under intact skin.[15-17] Thus, this paper aims to discuss the literature pertaining to the early detection of tissue damage and methods of early assessment for pressure ulcers with a specific focus on SEM assessment.

Key messages

- Disruption of cell homeostasis by pressure and shear can cause cell death, triggering the inflammatory process
- Sub-epidermal moisture is a biophysical marker currently used for early pressure ulcer detection. This innovative assessment showed that clinicians have, on average, the confirmation of tissue damage around 5 days prior to any visual presentation on the skin surface
- There is a need for greater use of biomedical technologies that facilitate early detection of cell death, or pressure ulcer development to improve diagnosis and prevention

2 | AIM

This paper aims to discuss the literature pertaining to the early detection of tissue damage and methods of early assessment for pressure ulcers with a specific focus on SEM measurement.

3 | DESIGN

The present article is a discursive paper.

4 | METHODS

This paper discusses the issues relating to the lack of use of technology in the early detection of pressure-shear induced damage focusing on SEM measurement.

5 | EARLY TISSUE DAMAGE DETECTION

The pathways that lead to cell damage included localised ischaemia, ischaemia-reperfusion injury, impaired lymphatic drainage, and tissue distortion, or deformation.[18-28] All these mechanisms cause changes in cell metabolism, alter the inner scaffold structures of the cell (proteins that build the cytoskeleton), and cause changes in the cell membrane.[29,30] Furthermore, disruption of cell homeostasis can cause cell death, triggering the inflammatory process.[31]

Local inflammation is a normal response to any cell injury and is essential for tissue repair at a microscopic level.[10] Each single damage pathway, or a combination of damage pathways, can set off the inflammation process.[10] When the first cells start to die after unrelieved pressure and/or shear damage, chemokines, cytokines and other cell signalling molecules are released.[10,32,33] These molecules "flag" the damaged cells and the microscopic surrounding area, guiding the migration of local and systemic immune cells.[10,16]

Endothelial cells from the blood vessel walls are also sensitive to certain chemokines, and they respond by detaching from each other to enable the circulating immune cells to reach the affected area.[10,32] During this process, plasma also leaks as a response to the increased blood vessel permeability, which increases the water content around the affected area. This local oedema, or SEM, and the local increase of moisture, changes the electrical capacitance of the tissues, which can be measured using an electrical bioimpedance device.[10,17,34,35]

A decrease in the blood supply caused by sustained unrelieved pressure and/or shear over soft tissues may occlude the lymphatic system and blood vessels triggering ischaemic metabolism changes.[25-28] Thus, the change in the affected cells' metabolism, from aerobic to anaerobic, causes a decrease in the local pH and the process may slowly lead to apoptosis (programmed cell death), causing local tissue damage, which initiates local inflammation.[36-38] In the event of an ischaemia-reperfusion injury, the release of the blood flow carrying elevated concentration of waste products and oxygen-free radicals increases and aggravates the inflammation causing further cell damage.[24,38,39]

Cell damage can also occur by the direct effect of distortion, or deformation by pressure and/or shear, compressing the cells and tissues, causing cell death by breaking down the cell membrane and cytoskeleton.[30,40,41] This event occurs more rapidly than ischaemia and may cause deeper damage as the deformation strains are higher at the bone-muscle interface compared with the upper layers of soft tissue.[42,43] The mechanical loads also compress the lymphatic draining system causing its occlusion, and both the catabolite products and interstitial liquid that accumulated in the area during this process, worsen the damage.[25,27,28]

Although inflammation is vital for tissue repair, if the damaging agent (unrelieved sustained pressure and/or shear) persists, the inflammatory process will act as a new damaging agent also contributing to tissue damage.[10] The longer inflammation occurs, the more tissue degradation will happen, as continuous cell death causes further damage to the surrounding tissues.[10] This knock-on effect, that starts at a microscopic level (which can be detected by bioimpedance measurement), increases to a macroscopic level enabling visualisation of the classic signs of inflammation redness/rubor, heat/calor, swelling/tumour, and pain by imaging examination and then eventually using VSA.[10,17] Therefore, the present article defines early detection of a pressure ulcer as any physical or biochemical indicator detected before the visual presentation of damage on the skin surface.

In summary, the early detection of tissue damage is beneficial in two different ways. First, it enables early intervention when the damage is still microscopic and reversible and, therefore, has the potential to prevent further aggravation of healthy surrounding tissue. This arises by avoiding the causation of the problem and stopping the knock-on effect of inflammation, especially when the rapid pressure ulceration pathway of deformation is in place.[44] Second, when the slow ischaemic-reperfusion related mechanism is undergoing cell death, can be avoided when the problem is identified before the cell reaches the "death threshold,"[43] completely averting a pressure ulcer.[10]

5.1 | Biomarkers

A biomarker is a measurable molecule (such as chemokine), a specific gene, a cell, or a characteristic (such as increased SEM, change of tissue capacitance, epidermal lipid deficiency) that is correlated with the presence of a condition and/or can indicate a risk factor, severity, prognosis or its early detection.[10,16] In a pressure ulcer context, different biomarkers are being extensively studied, and some are already in use in clinical practice, such as SEM. Most of the biomarkers studied for pressure ulcers are linked to the fact that inflammation is one of the earliest responses after an event of cell disturbance.[10,16,17,31] Based on that, studies are identifying potential biomarkers that would be correlated to pressure ulcer development.

Bronneberg, Bouten[45] studied biomarkers, using an in vitro model, on the effects of different loading regimes

and the release of interleukin-1 alpha (IL-1alpha), which is a biomarker for inflammation. The authors found that IL-1alpha is correlated to the magnitude of the loading and concludes that this biomarker can be used to identify epidermal damage due to prolonged mechanical loading and might be an effective tool in pressure ulcer prevention. Similar studies also successfully evaluated other biomarkers correlated with pressure ulceration, such as IL-1RA and IL-8, which can also be collected using non-invasive methods, such as using tapes applied to collect sebum on the skin surface over weight-bearing areas.[16,46] Urine and blood are also an alternative method to collect biomarker.[47] Techniques involving sebum and sweat primarily indicate skin irritation and it is not necessarily correlated to deeper damage whereas urine, blood biomarkers, and SEM are.[48] There is also literature exploring the use of plasminogen activator inhibitor 1 (PAI1), 12 heat shock protein 90α (HSP90α) and vascular endothelial growth factor C (VEGF-C) as a promising molecular tool to early detection[48] although this is still in early stages.

Borzdynski, McGuiness[49] investigated a combination of biophysical markers using sensor technology to measure epidermal hydration/skin wetness, melanin, and erythema using reflectance and a sebumetry to measure markers on lipids from the skin surface. The study included 38 aged participants and the measures were performed twice a day over 7 days in nine common pressure areas. The results compared the biophysical markers to VSA and structured risk assessments and found significant correlations between the Norton scale, VSA, and epidermal hydration over the sacrum and ischium. Erythema was also significantly correlated with pressure ulcer risk at the sacrum ($r = -0.322$; $P < .05$). Those findings suggested that biophysical markers such as epidermal hydration and erythema of the aged skin may be more effective than traditional VSA, whereas lipid readings using the lipid measurement technique scored 0 in all assessments and could not be conducted.

A new approach by Yang, Han[50] studied the roles of matrix metalloproteinase (MMP) 9 genes in the occurrence of pressure ulcers after hip fracture. The authors looked at both healthy controls and pressure ulcer groups, searching for different polymorphisms of genes that had an effect on MMP 9 regulation. They found a polymorphic gene in the pressure ulcer group that could be a novel biomarker in predicting pressure ulceration after a hip fracture. This type of genetic study of pressure ulceration shows that individual intrinsic variabilities also play a role in pressure ulcer development, and there might be some unique risk factors for developing tissue damage, which cannot be captured by subjective assessment.[16]

Although using biomarkers for pressure ulcer detection is a promising field, current limitations do not make them widely available in clinical practice yet. Some reasons are that the studies have limited sample volumes, they are experimental studies, the techniques are very costly, and most of them have a lack of standardisation for clinical practice.[16]

5.2 | Imaging approaches

Imaging, such as magnetic resonance imaging (MRI) and ultrasound, have been used in pressure ulcer diagnostics and attempts to enable early damage detection. A systematic review performed by Oliveira and Moore[51] investigated the clinical significance of ultrasound, thermography, photography, and SEM in detecting tissue damage. The authors included 10 articles and found SEM and ultrasound to be promising techniques. However, they highlighted a significant potential risk of bias within all studies included. Thus, they recommend further robust studies to clarify the potential of these technologies for use as a prevention strategy in clinical practice.

There is no consensus in the literature on exactly how prematurely the diagnosis of a pressure ulcer needs to be undertaken to be considered "early." However, Gefen[10] argues that imaging techniques detect damage, generally referred to as pockets of oedema, when they are large enough to be visualised by the examiner. Conversely, when measuring SEM, for example, the sensitivity of the assessment technique enables the examiner to detect the damage when it is still microscopic. Furthermore, imaging approaches require expertise, significant training and they may not be easily included in daily practice.[10,31] Another consideration is that imaging techniques, such as ultrasound, are not objective because of the potential for disagreements between examiners in interpreting results, which can be influenced by their expertise, the method used, and the type of equipment.[10]

Gefen and Gershon[52] evaluated the consistency between ultrasound and SEM when examining suspected deep tissue injury (DTI). This pilot study included 15 participants at risk of developing a pressure ulcer. The authors included the presence of hypoechoic lesions considered as indicative of a DTI when using ultrasound assessment, and two consecutive days of abnormal SEM measures as indicative of a DTI when using SEM assessment. Results showed that SEM assessment was abnormal 2 days before nurses identified abnormalities using VSA and, interestingly, 3 days before the appearance of a hypoechoic lesion in the ultrasound. The authors concluded that, in patients with existing DTI, SEM detected tissue damage at least 2 days earlier than ultrasound.

When analysing other approaches, such as MRI, Nuutinen, Ikaheimo[53] identified that this is an expensive technique with limited availability in clinical practice and, therefore, MRI is not practical for routine evaluation of oedema. Although there are other indirect methods to measure macroscopic oedema, for example, directly measuring the limb's volume by immersion, or recording the limb circumference, these methods can only be applied to the limb and thus is not possible for use in other body sites. Furthermore, when the oedema is already macroscopic, it may be too late to prevent further damage.[53]

5.3 | Sub-epidermal moisture

Sub-epidermal moisture is a biophysical marker and is a product of the leak of plasma after the inflammation process increases local vasculature permeability.[10,34,35] Of note, SEM is different from epidermal hydration, another biophysical marker for superficial damage, that expresses the water content of the epidermis and it is known to be influenced by microclimate parameters, such as temperature and moisture (faeces, urine, and sweat).[31,54,55] As SEM is related to deeper layers it is not influenced by environmental changes, yet is directly related to inflammation.[10,17,31] When tissue damage progresses to a greater number of cells, the inflammation markers increase along with the plasma leakage through the blood vessels. SEM that started as microscopic oedema grows to a macroscopic scale and becomes detectable on imaging examinations and later with the naked eye.[10]

When SEM increases after tissue damage, it changes the electrical capacitance of the tissues even when oedema is still at microscopic levels, and can be measured using surface electrical bioimpedance devices (with a suitable sub-epidermal depth penetration capacity which varies depending on the device manufacturer).[34,35,56] Surface electrical capacitance is the capacity of the tissue to hold electrical charges.[57] Bioimpedance is the measure of how well the tissue impedes electrical forces, and the SEM (water content) makes tissues less resistant to electrical fields because water improves the electric flow.[57]

Goretsky, Supp[34] observed the potential of SEM in wound care, mostly based on a study performed by Leveque and DeRigal,[56] using a dermal phase metre called NOVA Petite (NOVA Technology Corporation, 75 Congress St., Portsmouth, New Hampshire). The device is an electronic laboratory instrument that measures skin impedance. The authors performed measures on cultured skin substitutes and split-thickness skin autografts of five patients hospitalised in a burn unit after their surgery. They found a decrease in SEM in all grafts during the 28 days follow up. Further, high SEM values of injured skin that were identified in the first days after surgery, reverted to uninjured area SEM values after an average of 12 days, showing SEM to be a marker for tissue changes (inflammation and healing process).

The idea of measuring SEM for pressure ulcer detection was first used by Bates-Jensen, McCreath.[35] They examined the relationship between SEM and VSA of erythema and stage I pressure ulcers in 35 nursing home residents over a 52 week period. Using the dermal phase meter NOVA Petite, Bates-Jensen et al[35] found that SEM had progressively increased measures for erythema, stage I and ≥ stage II across all seven anatomical sites included in the study ($P < .001$). The authors concluded that high SEM measures were associated with erythema, and stage I pressure ulcers and high SEM measures were also associated with visual erythema and pressure ulcers stage I, which became evident 1 week after the SEM measurement.

Nuutinen, Ikaheimo[53] investigated seven adult patients undergoing haemodialysis treatment, using the dermal phase meter MoistureMeter D (Delfin Technologies Ltd, Kuopio, Finland) with 2.5 mm penetration depth capacity. The authors measured the forearm circumference (to measure subcutaneous oedema) and SEM at baseline, and then two times during treatment, with the last measurement being performed at the end of the haemodialysis session. Results showed a statistically significant correlation between the device measurement and the reduction of the forearm circumference ($r = -0.99$; $P < .05$). Thus, Nuutinen, Ikaheimo[53] concluded that the SEM measurement technique can assess changes in the water content in skin and fat tissues. Later, Guihan, Bates-Jensen[58] used the same dermal phase metre, but with two different probes of 0.5 and 1.5 mm penetration depth. A total of 12 participants were assessed daily, and another 22 participants with spinal cord injury (SCI) were assessed weekly to compare both SEM and VSA across nine anatomical sites over a 6-week period. The authors found lower SEM measures were associated with normal skin, and higher SEM values were associated with erythema, and stage I pressure ulcers, for all anatomical sites. These findings confirmed that the findings of previous studies of older persons cared for in long-term settings were congruent with the findings from the SCI population.[35,59,60]

Another study in the SCI population performed by Harrow and Mayrovitz[61] used the same MoistureMeter D device with a 2.5 mm penetration depth to investigate if SEM was associated with stage III and stage IV pressure ulcers over the sacrum and ischium. They found that SEM was able to differentiate intact skin from pressure ulcers but raised concerns on how the measures should

be performed in future studies (including positions, angles, and anatomical site effects). It is valuable to differentiate intact skin from damaged tissue when a deep pressure ulcer is suspected as, visually, the true extension of the wound under the intact skin is very challenging to correctly assess.[7,62]

A recent study with large sample sizes by Bates-Jensen, McCreath[63] was performed to compare VSA to SEM in older persons cared for in long-term care. A total of 417 residents were included and had their heels assessed heels and trunk anatomical sites (sacrum and ischium)[31] using the MoistureMeter D with a 2.5 mm penetration depth probe. Bates-Jensen, McCreath[31] found an incidence of all stage skin damage of 52%, and high SEM measures were associated with visual damage 1 week later regardless of patient skin colour (SEM predicted 41% of future damage and VSA predicted 27%). Bates-Jensen, McCreath[63] study also added that SEM detected DTI on heels and differentiated the damaged skin over a 16-week period, from cases that resolved to reinforce the ability of SEM to be used as a biophysical marker of early pressure ulceration.

A different device is currently available, known as the SEM Scanner (Bruin Biometrics, LLC, Los Angeles, California), which also assesses local bioimpedance, but at a depth up to 7 mm depending on the anatomical site and examination protocol.[10,64] Although this new device also measures bioimpedance similarly to other aforementioned, it has the ability to take pressure-controlled measurements where the probe is applied on the skin surface (newer version of the MoistureMeter D Compact also equipped). This makes measures more homogeneous in terms of how the examiner compresses the tissue under investigation. This functionality is important as Gonzalez-Correa, Brown[65] demonstrated in their work on early diagnosis of cancer using electrical bioimpedance spectroscopy that bioimpedance measures increased as the pressure in both rat and human tissues also increased. The authors hypothesised that the squeezing of the tissues by the examiner when using the probe may have changed the water content in the underlying tissues, spreading the moisture away from the extracellular space under the site of measurement. In a review performed by Moore, Patton,[17] the authors highlighted this observation and highlighted the need for pressure-controlled measurement when applying a probe for SEM measures in order to collect a reliable SEM assessment.

Using the SEM Scanner device with pressure-controlled measurement, O'Brien, Moore[66] explored the relationship between VSA and SEM, by collecting data using both assessments on the heels and sacrum of acutely ill patients, daily over a 4 week period. From a total of 47 participants, 40% (N = 19) developed visual pressure ulcers, all stage I and all 19 participants also had abnormal SEM measures. The authors found a medium correlation between VSA and SEM assessment ($r = 0.47$; $P = .001$). SEM assessment detected tissue damage on average 1.5 days (min: 1 day; max: 7 days; SD: 1.4 days) before it appeared visually on the skin surface, whereas the nurses' VSA took on average 5.5 days (min: 2 days; max: 11 days; SD: 2.5 days) to detect damage on the skin surface. The study confirmed the feasibility of SEM measurement as a useful tool for improving the early detection of pressure ulcers.

A recent study[67] in the older population, also using this device among 150 participants cared for in long-term settings, undertook SEM assessment and VSA to identify how activity and mobility lead to pressure ulceration. The authors, using an observational design, followed the participants up for 20 days and assessed mobility using a piezoelectric movement sensor and the activity subscale of the Braden scale. SEM and visual skin assessment were undertaken daily, and the results showed that the odds of PU detection were 25 times greater with SEM versus VSA (OR 25.42; 95% CI: 13.68-47.25) supporting the use of this technology when assessing the at-risk areas. As all patients had preventative measures in place, not all abnormal SEM translated into tissue damage. However, when tissue damage could not be prevented by these measures, the mean number of days to visually detect a PU among the participants was 14.4 days, whereas SEM assessment detected a PU 8.2 days before it appeared visually on the skin surface.

Although SEM is promising in early pressure ulcer detection, this assessment modality is still unable to accurately determine the extent of the damage.[16,60] Bader and Worsley[15] argue that there is not enough evidence regarding the sensitivity and specificity of biophysical techniques, such as epidermal hydration and SEM to distinguish between mechanical, chemical, or environmentally induced skin damage. In addition, SEM assessment is not able to identify the depth of the damage, which would be valuable clinical information. Nevertheless, the studies of SEM measurement in clinical practice, in varied populations, and using different devices, have shown that SEM assessment may be effective in the early detection of pressure ulceration, while providing more objective information when compared with other techniques, such as ultrasound.[10,51,52] Furthermore, the literature shows that SEM is also applicable in wound healing[34] and is promising as a continuous "smart" assessment if small bioimpedance sensors, such as those proposed by Swisher, Lin,[68] can be applied to the skin. The literature suggests that these may have a huge potential in tracking both intact and healing wounds, enabling not only the size and shape of damage identification but also

providing information on different types of wounds and stages of healing. In the future, these smart sensors may give objective real-time guidance to clinicians in the wound care decision-making process optimising and individualising patient care.[16]

6 | CONCLUSION

For pressure ulcer prevention, Gefen[10] posits that if specific technological support is not implemented, society will probably never achieve real advancements in pressure ulcer statistics. As discussed in this paper, studies are pointing to technological-based approaches, such as the use of biomarkers and biophysical markers measurement, as being more effective than relying solely on VSA. SEM may be a technological-based approach that is viable to use in clinical practice for the early detection of tissue damage, basically in two different ways. The benefits could be two-fold; first, it would enable early intervention, thereby improving preventative measures. Second, by implementing prevention correctly and promptly, tissue damage, when identified in a preventable stage, maybe averted from progressing.

6.1 | Relevance to clinical practice

To date, when performing skin assessment visually, the assessment method is comparable to an astronomer studying the planets and galaxies just by looking at the sky with their naked eye. For this reason, the literature presented strongly suggests the use of technologies, which are "telescope-like" such as SEM assessment, to objectively assess the skin and underlying tissues for early pressure-shear damage. In the future, the use of superficial chemical biomarkers, such as those collected using tape technique, will help to understand where the damage started to be this either superficially or in deeper tissues. Such a combination of assessments will enable a more focused assessment and accurate identification of individuals' real-time responses to pressure and shear forces. Furthermore, SEM technology also enables assessment of the patient from the moment he or she enters the healthcare setting and if continued until discharge it allows the clinician to identify where and when tissue deterioration started. This information is relevant not only to guide which specific setting within an organisation needs to improve preventative measures but also in the case of litigation that may provide useful information as to when and where the pressure ulcer first developed. Importantly, SEM assessment showed that clinicians have on average the confirmation of tissue damage around 5 days prior to any visual presentation on the skin surface, fundamentally this will enable early intervention and prevention of further damage.

CONFLICT OF INTEREST

The authors have the following conflicts of interest, which they wish to declare:

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