

Clinical innovations: can dressings help to prevent pressure ulcers in high-risk nursing home residents?



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The aim of this paper is to present current and emerging clinical and scientific evidence for the prevention of pressure ulcers in highly dependent aged care residents. The authors discuss recent developments in the use of multilayer silicone foam dressings used prophylactically in both the acute and nursing home settings and explain this preventative approach in the light of our current understanding of the role of cell and tissue deformations in the pathogenesis of these wounds. The authors also discuss how certain dressings can reduce the exposure to tissue deformations resulting from the mechanical loads of pressure, friction and shear in these highly vulnerable individuals.

Aged nursing home residents who are immobile (Wong, 2011; Moore and Cowman, 2012), poorly nourished (Horn et al, 2004; Banks et al, 2010), incontinent (Wong, 2011; Long et al 2012; Moore and Cowman, 2012), have ageing skin-related changes (Foreman et al, 1993), are cognitively impaired (Capon et al, 2007), and have multiple comorbidities (Santamaria et al, 2005; Kwong et al, 2009; Lyman, 2009) are highly vulnerable to the development of facility-acquired pressure ulcers (PUs). It has been clearly established that many of these vulnerable residents who develop a facility-acquired PU will experience additional pain, morbidity and, in some cases, the wound will lead to amputation or the person's death (Capon et al, 2007; Kwong et al, 2009; Liao et al, 2010).

Prevalence and incidence rates for aged care facility-acquired PUs have been reported to range from 4.3% to 35.1% (Kottner et al, 2010, Long et al, 2012) and 2.5% to 25.1% (Kwong et al, 2009; 2011) respectively, although we urge caution in interpreting these figures due to the potential for differing methodologies used in conducting the prevalence and incidence surveys. The most commonly reported anatomical sites for the development of PUs are the sacrum (Kwong et al, 2010) and heels (Moore and Cowman, 2012).

New insights into the mechanisms of injury in pressure ulceration

Our understanding of the underlying mechanisms of PUs (also called pressure injuries)

has for the past 80 years been based on work by Landis (1930) who proposed an absolute generic capillary closing pressure of 32 mmHg caused by direct pressure on tissues. This paper and subsequent work has, unfortunately, resulted in PUs being mistakenly thought to be principally an ischaemic event where soft tissues are compressed, for prolonged periods of time, between a bony prominence, such as the sacrum or the calcaneus and a surface resulting in capillary occlusion, hypoxia and subsequent tissue necrosis.

More recently, the involvement of bioengineers/scientists has resulted in fundamental changes to how we understand PU development based on the concept of cell and tissue deformation and direct cellular damage driven by the deformations. This is a more rapid and powerful factor than ischaemia in PU formation. In a supine patient, the forces originating from the weight of the trunk are transferred through triangular-shaped sacral bone into thin and deformable layer of skeletal muscle, subcutaneous fat and skin. Forces considerably distort and deform this layered tissue structure.

Due to the highly curved shape of the sacrum and its sharp topography, it tends to heavily distort the soft tissues between the bone and the surface, so that the cells that reside in these tissues are compressed, stretched and sheared simultaneously (Gefen et al, 2005). These forces are exacerbated if the head of the bed is elevated, which adds additional frictional forces on the skin and internal shearing sub-dermally

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(Gefen, 2017). In older people, capillary density is generally reduced, and so the reduced number of capillaries are more susceptible to the effect of shear, which have a more pronounced impact on perfusion quality (Linder-Ganz and Gefen, 2007). Respiratory disease, which is relatively common in older patients, may further impair tissue oxygenation levels. The overall mass of soft tissues surrounding the sacrum may be diminished in older individuals, and the anchoring between the skin layers (specifically the interlocking at the epidermal-dermal junctions) is typically compromised (Gefen, 2014).

Additionally, the inflammatory response, including molecular signalling for recruiting immune system cells to the damage sites and adequate cell migration — both being essential for tissue repair — are often partial or impaired, which further tilts the physiological balance towards a faster accumulation of damage (Laflamme et al, 2017). These factors, taken together, make the sacral region of older people that are highly vulnerable to PUs. In addition, there is recent evidence indicating that tissue deformation results in damage to the cellular cytoskeleton and plasma membrane, causing the uncontrolled movement of ions through the cell membrane, which leads to apoptotic cell death (Slomka and Gefen 2012; Leopold and Gefen 2013; Gefen and Weihs 2016). These destructive processes are also affected by local tissue stiffness properties. Connective tissues and specifically skin tend to stiffen with old age, and, likewise, in individuals with type-2 diabetes, due to localised fusion of collagen fibres and increased fibre thickness, resulting in a decreased capacity to relieve mechanical stress (Gefen et al, 2016; Levy and Gefen, 2016).

Pressure ulcer prevention for the nursing home resident

There is clear guidance on the preventative assessment and interventions that are recommended through the International Clinical Practice Guideline for the Prevention and Treatment of Pressure Ulcers (European Pressure Ulcer Advisory Panel, National Pressure Ulcer Advisory Panel and Pan Pacific Pressure Injury Alliance, 2014) and the authors do not intend to review this document in detail in relation to the nursing home resident. Suffice to say it is essential that the individual is comprehensively and systematically assessed for their risk of developing a PU. This assessment needs to be conducted as soon as possible and comprise determination of risk based on age, mobility

status, skin condition, nutritional status, continence status, cognitive status, history of previous PUs, health status and presence of comorbid conditions that may impact PU risk.

The authors note that the use of a validated PU risk assessment scale, such as Braden, Waterlow or Norton scales, provides clinicians with a useful structured approach, however, these scales provide only part of the required assessment to accurately determine PU risk. One major drawback of all existing PU risk assessment tools is that they include subjective components and overall, there is lack of technology embedded in the risk assessment procedure to evaluate the risk and, specifically, to determine the health status of subdermal (invisible) tissues. This stands in stark contrast to other fields of medicine, such as cardiology, where medical technology (electrocardiography, blood pressure monitoring) provides key inputs to the risk assessment. There are seeds for changes in this regard, and industry has identified the above technological gap, which is expected to be narrowed over time. For now, the risk assessment procedure must be individualised and conducted by a suitably educated and experienced clinician. Additionally, residents need to be reassessed should their physical condition changes due to acute illness or other (patho)physiological changes.

Preventative interventions are based on the initial and ongoing assessment of the individual and include measures to minimise exposure to localised elevated pressures, to frictional forces, to shear in tissues and to moisture at the skin surface/interface. This is achieved through ensuring that surfaces such as mattresses, cushions and chairs have the required physical/mechanical characteristics that are appropriate to the detected risk level. This may include the use of high specification, visco-elastic foam or alternating air mattresses as required.

Care needs be focused on the prevention of PUs to the heels using appropriate elevation of the heels from the bed surface if possible. If this is not a viable intervention due to cognitive status or other factors, then the use of high-quality boots or prophylactic multi-layered foam dressings should be considered. Additionally, regular repositioning must be included to minimise tissue loading for prolonged periods of time. Skin care is an essential component of pressure ulcer prevention, particularly for the incontinent individual. This will require continual vigilance of the skin and the use of appropriate pH balanced cleansers where

necessary. Management of nutritional deficits has been shown to reduce the risk of pressure ulceration in older people and the clinician needs to maximise the individuals' general health status and prevent acute exacerbations of existing comorbidities.

The evidence for the addition of prophylactic to existing pressure ulcer prevention protocols

Over the past 8 years, there has been an increased research interest in the clinical use of multi-layer silicone foam dressings to prevent PUs. Brindle (2010) published an important pioneering cohort study suggesting that the prophylactic use of the Mepilex® Border Sacrum (Mölnlycke Health Care) significantly decreased the incidence of sacral PUs in surgical ICU patients. Santamaria et al (2013) conducted the first randomised controlled trial (RCT) of the use of the Mepilex Border Sacrum and Mepilex® Heel dressings (Mölnlycke Health Care) to prevent PUs in 440 ICU patients. The dressings were applied in the emergency department, changed every 3 days and maintained for the duration of the ICU stay. Results indicated an 80% reduction in PU incidence for patients with dressings as opposed to controls.

A subsequent cost/benefit analysis of the findings revealed that the intervention group wound care costs was 3.6 times less than controls due to the reduced incidence of PUs (Santamaria et al 2015a). Kalowes et al (2016) found similar outcomes in the USA when dressings were added to their skin care bundle in a large RCT. In a follow-up study, Santamaria et al (2015b) investigated the protective performance of Mepilex Border Heel dressing in a 300-ICU patient study and found a 0% incidence rate for heel PU in patients treated with the dressings. Yoshimura et al (2016) compared Mepilex Border dressings to film dressings to prevent operating room PUs in spinal surgical patients and found significantly reduced PU incidence with the Mepilex Border dressings compared to film alone.

At the hospital-wide level, Santamaria et al (2015c) reported a reduction of 60% in PU prevalence when hospital policy was changed to require the use of these dressings for all patients with a high risk of PU development. To date, the only RCT that has been conducted in the aged care sector investigating the clinical efficacy of the Mepilex Border and Mepilex Heel dressings found a statistically reduced PU incidence for newly admitted residents with a high risk of PUs (Braden <13) when the dressings were applied on admission, changed three times daily and

maintained for a 4-week period (Santamaria et al, 2018).

The effectiveness of the Mölnlycke dressings is clearly linked to the ability of the dressings to alleviate tissue deformations and hence reduce the subsequent risk for PU development (Levy and Gefen, 2016; 2017; Levy et al, 2015; 2017) at both the sacrum and heel. In the aforementioned bioengineering studies, several unique features have been identified that distinguish the Mölnlycke Mepilex Border technology from other products: (a) The Mepilex dressings are flexible and, hence, deform compression, tension and shear under weight-bearing, which cushions and, thereby, protects underlying tissues; (b) The Mepilex have a sandwich-like alternating stiffness structure of the dressing, composed of a softer layer between each two less soft layers. This internal structure of the dressing effectively absorbs shear deformations within the dressing, and, accordingly, deformations are taken off the tissues; (c) The outer surface of the dressing is relatively smooth i.e., has a low coefficient of friction which contributes substantially to the minimisation of frictional forces at the dressing-support interface, which consequently causes less deep tissue distortion in shear; (d) The sacral dressing model (Mepilex® Border Sacrum) is more flexible in its lateral direction (of the buttocks cheeks) compared to the longitudinal direction (along the line of the spine), a feature known as anisotropy and called 'deep defence' by the manufacturer; (e) The dressing adequately manages moisture and so its protective performances are stable under different microclimate conditions (Call et al, 2015; Levy et al, 2015; Levy and Gefen, 2016; Levy and Gefen, 2017; Levy et al, 2017).

It is, therefore, not surprising that systematic reviews of the published research evidence (Clark et al 2014; Tayyib and Coyer, 2016) further support the clinical efficacy of the Mölnlycke dressings in preventing PUs when used as an adjunct to best practice PU prevention. The use of prophylactic dressings for PU prevention has also been reviewed in a consensus statement released by the World Union of Wound Healing Societies (WUWHS, 2016) and provides clinicians a useful and contemporary overview of the use of the dressings for prophylaxis.

It is essential that clinicians use the best available evidence when choosing which dressings to use for PU prevention. There are many dressings claiming to be effective in PU prevention but few with compelling research evidence proving their performance. Gefen et al (2016) caution

against basing decisions on dressing selection on marketing/advertising or price alone. This important decision must be based on high-quality research evidence as we must avoid the potential to harm these highly vulnerable individuals. The biomechanical properties and function of dressings to prevent PUs varies significantly based on the formulation of the materials used and in the construction of the dressing and the internal structure and architecture of the product (Call et al, 2015; Levy et al, 2015; Levy and Gefen, 2016; Levy and Gefen, 2017; Levy et al, 2017). It is, therefore, crucial to understand that not all multi-layer silicone foam dressings are as effective in PU prevention.

Conclusion

As our understanding of PU development has evolved from a purely ischaemic model to a more sophisticated, science-based one that incorporates exposure to cell and tissue deformation as a fundamental damage pathway, it is important for clinicians and administrators involved in caring for the most vulnerable individuals in nursing homes to use the compelling body of clinical and cost/effectiveness evidence that is now available — supporting the clinical efficacy of multi-layer silicone foam dressings to prevent facility PU development. The authors emphasise that the use of prophylactic dressings is an adjunct to high-quality PU prevention and not an alternative. Clinicians and scientists have an ethical responsibility to only use dressings that have strong product-specific research evidence supporting their prophylactic effectiveness in caring for our most vulnerable nursing home residents.

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