Preventing pressure injuries in the emergency department: Current evidence and practice considerations

Nick Santamaria1 | Sue Creehan2 | Jacqui Fletcher3 | Paulo Alves4 | Amit Gefen5

1Department of Nursing, University of Melbourne, Melbourne, Victoria, Australia
2Department of Nursing, Virginia Commonwealth University, Richmond, Virginia
3Wound Healing, Welsh Wound Innovation Centre, Pontyclun, UK
4Department of Nursing, Catholic University of Portugal, Lisbon, Portugal
5Department of Biomedical Engineering, Tel Aviv University, Tel Aviv, Israel

Correspondence
Nick Santamaria, Department of Nursing, Alan Gilbert Building, Level 7, 161 Barry Street, University of Melbourne, Victoria 3010, Australia. Email: n.santamaria@unimelb.edu.au

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The emergency department (ED) is at the front line of hospital pressure injury (PI) prevention, yet ED clinicians must balance many competing clinical priorities in the care of seriously ill patients. This paper presents the current biomechanical and clinical evidence and management considerations to assist EDs to continue to develop and implement evidence-based PI prevention protocols for the high-risk emergency-trauma patient. The prevention of hospital-acquired pressure injuries has received significant focus internationally over many years because of the additional burden that these injuries place on the patient, the additional costs and impact to the efficiency of the hospital, and the potential for litigation. The development of a PI is the result of a complex number of biomechanical, physiological, and environmental interactions. Our understanding of the interaction of these factors has improved significantly over the past 10 years. We have demonstrated that large reductions in PI incidence rates can be achieved in critical care and general hospital wards through the application of advanced evidence-based prevention protocols and believe that further improvement can be achieved through the application of these approaches in the ED.

KEYWORDS
emergency department, pressure injury, pressure ulcer, prevention

1 | INTRODUCTION AND BACKGROUND

The emergency department (ED) is often the interface between the community and the hospital for many patients and frequently functions as the first point of care in what has been described as a complex, and at times potentially dangerous, journey through the acute health care system, particularly for the older individual.1

The aim of this paper is to provide an overview of our current understanding of pressure injury (PI) pathogenesis and prevention to assist ED staff to continue the development and implementation of evidence-based PI prevention processes based on the latest clinical and scientific research evidence.

The prevention of hospital-acquired PIs (HAPIs) remains a significant clinical challenge internationally. These largely preventable injuries can lead to decreased quality of life, pain, suffering and increased morbidity, and in some cases mortality.2 In addition, HAPIs can increase patient length of stay3 and health care costs and may expose the hospital to litigation.4

Prevalence rates for HAPI have been reported to range from 0% to 46%,5,6 with an incidence of 4.8%, rising to 15.7% in patients older than 75 years of age.7 In the Unites States, over a 10-year time span (2006-2015), including all care settings, the overall PI prevalence rate dropped from 13.5% to 9.3%, and the facility-acquired rate reduced from 6.2% to 3.1%.8 Acute care data published by the National Database of Nursing Quality Indictors9 demonstrated a decline in HAPI rates from 7.25% in 2004 to 3% in 2011 for all stages; excluding Stage 1, this rate dropped to 1.75%. Similarly, Padula et al10 reported a 2008 to 2012 HAPI rate decline from 11.8/1000 to 0.8/1000 cases from the discharge data of 55 US academic medical centres. However, there are limited data on ED HAPI rates generally in the literature. This lack of data is exacerbated by hospital adverse event
patients are admitted through the ED,\textsuperscript{12,13} it is critical that prevention begin at this point of entry to the acute health system. As 40\% of hospitalised patients are admitted through the ED,\textsuperscript{12,13} it is critical that prevention begin at this point of entry to the acute health system.

Developed health care systems, such as those in the United States, Europe, United Kingdom, and Australia, have seen significant changes in the demographic and illness profiles of patients presenting to the ED over the past 10 years.\textsuperscript{11,13,14} Patients are generally becoming older and have more comorbid conditions and consequently require more complex care and assessment on admission to the ED.\textsuperscript{15,16} Concurrently, we have seen the demand for ED care increase within an environment of limited increases in available bed stock in hospitals. A consequence of these and other factors is that a greater number of older and sicker patients are unable to be transferred to a ward in a timely manner, often resulting in a longer length of stay in the ED.\textsuperscript{17} As a consequence of these demographic and structural changes, we believe that it is vital for ED clinicians to understand the current evidence as it relates to both PI pathogenesis and PI prevention in an ED context.

### 2 | THE ROLE OF TISSUE DEFORMATION IN THE PATHOGENESIS OF PRESSURE INJURIES

It is essential for clinicians to understand the evolving evidence relating to the pathogenesis of PIs because this evidence forms the basis for all preventative actions in the clinical setting and has implications on PI treatments as well.

For decades, PIs have been 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.\textsuperscript{18} This view of PI causation can be traced back to work published by Landis in 1930\textsuperscript{19} who proposed an absolute generic capillary closing pressure of 32 mm Hg caused by direct pressure on tissues. This paper and subsequent research has dominated our understanding of PI pathogenesis and has influenced clinical practice as well as the development of pressure-reducing surfaces and devices for almost 80 years.

More recently, the advent of sophisticated imaging and computer modelling techniques combined with a greater involvement of bioengineers/scientists who conduct mechanobiological research using state-of-the-art cell and tissue engineering approaches has resulted in fundamental changes to our understanding of PI development. During the past 10 years, the concept of tissue deformation and direct cellular damage driven by the deformations has been demonstrated as a more rapid and powerful factor than ischaemia in PI formation.

To exemplify this process, it is useful to consider the development of a sacral PI. In a supine patient, the forces originating from the weight of the trunk are transferred through the nearly rigid triangular-shaped sacral bone into a relatively thin and deformable layer of skeletal muscle (if not atrophied), subcutaneous fat, and skin. Bodyweight forces continuously distort and deform this delicate layered tissue structure and the living cells within. Moreover, as the sacrum is a highly curved, and perhaps the sharpest, bony element in the body, it tends to heavily distort the inferior soft tissues in a supine position so that cells embedded in these tissues (and the cell organelles) are simultaneously compressed, stretched, and sheared.\textsuperscript{20} Patients who have the head of their bed elevated, for example, to ease respiratory effort or as part of a mechanical ventilation intervention such as the use of continuous positive airway pressure (CPAP) (which is common in the ED), tend to slide down the bed or
trolley because of gravity. The patient's instinctive reaction is to then anchor themselves to the mattress by pushing themselves back up, which considerably adds to the distorting forces that are deforming cells and tissues near the bone-soft tissue interfaces, including under the sacrum and the heel. Another principal biomechanical reason for the sacral region to be particularly susceptible to PIs during supine lying is the direct interface between the rigid sacral bone and substantially more compliant muscle, fat, and skin tissues at this anatomical site. The tissue stiffness gradient is adding to the forceful internal cell and tissue distortions and is especially causing internal tissue shearing. In the elderly, capillary density is generally reduced, and so, the fewer capillaries are more susceptible to the effect of shear, which thus has a more pronounced impact on perfusion quality (therefore adding biochemical stress to the distorted cells and tissues). Pneumonia, which is also common in ED patients, particularly the elderly, may impair tissue oxygenation levels even further if present and add greater biochemical stress as cells are pushed into an anaerobic metabolic state that lowers tissue pH. In addition, 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. These factors, taken together, make the sacral region of the elderly highly vulnerable to PIs.

Internal shearing deformations near the weight-bearing sacrum are caused as the soft tissue layers attempt to slide upon each other and over the sacrum but cannot as they are constrained by connective tissue fibres at the interfaces. These internal mechanical constraints are causing the tissues themselves and the cells within to severely distort and change shape. Over time, these shape changes in tissues and cells cause tissue breakdown. The first event in the onset of tissue damage occurs at the micro scale: death of the first individual cells, leading to multiple necrotic and apoptotic cell death events and triggering an inflammatory response. As growing masses of cells are destroyed, the injury progresses macroscopically to the tissue scale and becomes detectable, first by medical imaging examinations if such are conducted (eg, bioimpedance, ultrasound, or magnetic resonance imaging [MRI]) and eventually through visual skin assessment—when damage has spread, this may often be irreversible. Current aetiological research points to sustained tissue deformations as the primary cause of PIs both at the skin and in deeper tissues. The sustained exposure to tissue deformations has a multifactorial influence on tissue health and cell viability, including direct damage to the distorted cells through the failure of cytoskeletal structures and pore formation in plasma membranes of cells as well as compromised perfusion and lymphatic function. Multiple model systems, including MRI of human subjects, animal models, tissue-engineered constructs, and cell culture models facilitating the application of sustained, controlled deformations to tissues and cells, have highlighted the role of direct deformation damage to cells. Specifically, recent published experimental evidence demonstrates that poration of the plasma membrane caused by the sustained deformations interferes with transport processes that normally occur through the plasma membrane and, thereby, causes loss of homeostasis (biological equilibrium). Moreover, these model systems altogether identified the short time frames at which damage to cells is inflicted, which is in the order of tens of minutes and much faster than the previously assumed ischemic damage cascade, which takes several hours to develop.

At the microscopic scale, the chronic distortion of cells causes disruption of the cytoskeleton, which, especially in the lack of available energy, gradually loses its capacity to structurally supporting the plasma membrane. As indicated above, this leads to the formation of nanometre-wide openings (pores), which form in the plasma membrane of the distorted cells. These mechanically (and sometimes biochemically) stressed cells are typically unable to repair the poration. Hence, fluxes of biomolecules and ions penetrate the cell bodies and/or escape cells uncontrollably, eventually causing the loss of homeostasis in the cells and resulting in apoptotic cell death in growing cell numbers. Inflammation develops consequently; however, older ED patients with background diseases such as diabetes or immunodeficiency syndrome that affect the inflammatory response may, depending on the individual patient conditions and severity, develop an abnormal, hypo-, or hyper-inflammatory response, and either state can contribute to the damage spiral within tissues.

Another important type of PI that is highly relevant to EDs is device-related PIs as the typical ED stretcher/bed may often contain catheters and tubing, electrocardiography electrodes, blood pressure monitor tubing, a pulse oximeter, wiring, etc. In addition, potentially, there could also be small objects, including plastics (eg, needle covers), consumables, or pieces of packaging of sterile equipment, that may have fallen or have been forgotten on the bed and may come into contact with the patient. Rigid equipment, devices, and any objects that are misplaced between the body and support surface act like an “external bony prominence” and can inflict localised, aggressive soft tissue distortions in addition to those caused near the bony prominences because of bodyweight. Procedures to thoroughly check the correct placement of catheter tubing and wiring, as well as careful collection of all packaging and plastics/consumables so that they do not become trapped under the weight-bearing body, is critically important for the prevention of PIs in the ED. One should consider that the ED, unlike other clinical settings, is particularly rich with such equipment and is also typically busy and hectic, which can make the staff more prone to human errors. Device-related PIs can also be caused...
by devices that have been specifically designed to be in continuous contact with the body tissues, particularly CPAP masks that are, again, commonly used in the ED. Fragile patients and unconscious or cognitively impaired patients who are administered oxygen via a nose mask or a full-face mask should be routinely checked for signs of skin irritation or redness, and appropriate padding or dressings should be used where applicable to protect the bridge of the nose, chin, and cheeks in such cases.31

The above-described cell-scale destructive processes are strongly affected by the mechanical state of tissues, one important factor being tissue stiffness. Connective tissues and specifically skin tend to stiffen with old age, and likewise in individuals with type-2 diabetes, because of localised fusion of collagen fibres and pathologically increased fibre thickness, which reinforces the skin and makes it less able to relieve mechanical stress.30,32 These age-related and/or diabetes-related changes exacerbate the mechanical state in tissues subjected to bodyweight forces, particularly around the sacrum, and may contribute to the susceptibility to sacral PIs in older individuals compared with younger (and non-diabetic) individuals.30,32

The above-described changes to our understanding of the mechanism of injury underlying PIs presents ED clinicians with specific challenges and considerations in adopting or modifying clinical care to minimise exposure to tissue distortion and deformation whilst the patient is in the ED.

3 | WHY IS THE SERIOUSLY ILL ED PATIENT AT HIGH RISK OF DEVELOPING A PI?

Seriously ill patients are defined, for the purposes of this paper, as patients with presenting illness and/or injury and trauma that is potentially life threatening and may require intensive care unit admission. These patients have a significantly increased risk of developing a PI because of a complex process of interaction between intrinsic and extrinsic factors related to their illness or injury. The most important interaction to consider is that of tissue tolerance to mechanical loads and cellular deformations caused by bodyweight forces (or medical devices as explained above) and the duration and intensity of those forces. Tissues can tolerate relatively long exposure to low-intensity mechanical loads or short exposure to high-intensity loads; however, long exposure to high intensity loads triggers the process of tissue damage, so the ED clinician is faced with the situation of needing to consider not only the intensity of the force but its duration whilst at the same time balancing competing clinical priorities related to the patients’ presenting problems.

The degree of tissue tolerance to mechanical loads is mediated by many intrinsic physiological factors such as age, elevated body temperature, presence of hypotension, haemorrhage, conscious state, nutritional status, skin integrity, ability to reposition, moisture at the skin-surface interface, comorbidities such as diabetes, and the current acute illness and medications.

The emergency/trauma patient can be exposed to many extrinsic factors in the ED that may contribute to exceeding tissue tolerance levels to mechanical loads. These factors include prolonged positioning on less-than-ideal surfaces for pressure redistribution such as backboards, ED stretchers, and MRI or computed tomography scan tables as well as the process of being transferred between a stretcher and other surfaces.33,34 To highlight these points, Berg et al35 describe how, in healthy adults, lying on an unpadded rigid backboard for 30 minutes can result in sacral tissue hypoxia. A further important consideration for PI prevention in the ED is the time prior to the patient’s admission. For some patients, they may have been exposed to prolonged immobility and exposure to significant tissue deformation secondary to mechanical force, such as patients who may have been trapped in a motor vehicle following accidents or the stroke patient who may have been on the floor for many hours or even days prior to admission. This exposure needs to be documented on admission and a determination made of the potential contribution to PI development.

The use of medical devices such as cervical collars and backboards in cases where there is suspected spinal injury is also a significant risk for the development of PIs because of their hard and unyielding surfaces. As explained above, relatively innocuous devices such as oxygen tubing, CPAP masks, nasogastric tubes, and urinary catheters can also pose a PI risk to patients if not safely positioned and managed.

4 | SPECIFIC PATIENT GROUPS WHO ARE AT HIGH RISK OF DEVELOPING PI IN ED

Current evidence clearly identifies specific patient characteristics that increase the risk of PI development in the ED.36 Broadly, any patient who is unable to effectively reposition is at risk; similarly, patients exposed to prolonged moisture at the skin-surface interface because of incontinence are also at risk.37 An important issue for the ED clinician to consider is that of the time prior to the patient’s admission to the ED. Patients may have been exposed to significant mechanical forces sufficient to commence tissue damage because of entrapment in vehicles or extended periods of immobility on hard surfaces. In addition, for the high-risk PI patient, even the time spent in the ambulance may contribute to PI development because of the nature of ambulance surfaces and the inability to reposition the patient. This factor has had some prominence in the United Kingdom because of ED overcrowding and bed shortages (https://www.theguardian.com/society/2018/jan/04/16900-people-in-a-week-kept-in-nhs-ambulances-waiting-for-hospital-care). Generally, the greater acuity and complexity of the patient’s condition combined with longer length of stay in the ED results in a
significant and continuously growing risk of developing a HAPI.\textsuperscript{7,38}

Patient position on an ED stretcher is also a risk factor whether it is from prolonged lying in the supine position or having the head of the stretcher elevated at 30° to maintain respiratory integrity. Elevation of the head of the stretcher increases the pressure and shear forces exerted on the sacrum and significantly increases the risk of a sacral PI. As previously mentioned, direct forces (compressive and shear) on the sacrum and ischial tuberosities are increased with the head of the stretcher elevated, and additional shear forces are generated in cases where the patient’s body slides down because of bed elevation. There is also potential for tissue shear secondary to friction between the patient’s skin and the surface to be exerted at the heels and sacrum as the patient tries to maintain his or her position by anchoring his or her body or pushing him- or herself back up if he or she are sliding down on the stretcher.\textsuperscript{39}

Age, illness severity, and comorbidity have also been clearly associated with PI risk.\textsuperscript{40} Dugaret et al\textsuperscript{7} demonstrated a tripling in PI incidence for ED patients older than 75 years of age (4.9% vs 17.5%) compared with younger patients. Length of stay in the ED is also associated with greater risk,\textsuperscript{7,38} as is inflammation measured by C-reactive protein (CRP) levels.\textsuperscript{7} Raised body temperature increases metabolic demands and has been associated with decreased tissue tolerance to mechanical loads. Poor nutritional status has been linked to increased PI risk because of decreased tissue tolerance and decreased fat and muscle mass over bony prominences such as the sacrum, which intensifies soft tissue deformations.\textsuperscript{37}

5 | EVIDENCE-BASED INTERVENTIONS TO MINIMISE THE DEVELOPMENT OF PIs IN ED

Risk assessment is the essential first step in the process of minimising the development of ED PIs.\textsuperscript{38,41} The ED clinician can often be confronted with many concurrent and competing priorities in the care of the patient, and the prevention of a PI is obviously of lesser priority in life-threatening situations. However, in situations where the patient's condition is relatively stable, the use of a validated PI risk assessment tool is an essential first step in the process of PI prevention. International guidelines recommend that risk assessment be conducted within the first 8 hours of admission;\textsuperscript{36} however, as we have seen from emerging evidence of the pathogenesis of PIs, this timeframe is probably inadequate in the high-risk, severely ill individual;\textsuperscript{32,42} therefore, we propose that an initial PI assessment be conducted within 4 hours of admission to ED. Whilst the Braden scale is widely used to determine PI risk, other scales such as the Norton scale have been modified for use in the ED,\textsuperscript{11} and specific shorter screening tools such as the PURPOSE T are gaining popularity because of their ease of use in some settings.\textsuperscript{43} Dugaret recommends that rapid assessment be carried out in the ED by focussing on age and presence of elevated CRP levels and the potential for the patient to remain in the ED for a protracted time as essential assessment elements for PI prevention.

Following rapid assessment of PI risk, there is a need to minimise exposure to unrelieved pressure, shear, and moisture through the implementation of repositioning schedules and ensuring the skin is protected from urine, faeces, and other fluids. The removal of medical devices such as backboards and cervical collars should be performed as soon as the absence of spinal injury has been confirmed.\textsuperscript{44} Heel elevation using longitudinally positioned pillows or the use of specialty off-loading devices have been shown to significantly reduce heel PI development.\textsuperscript{34}

In situations where it is not possible to reposition the patient frequently or the patient is agitated, clinicians should consider the application of prophylactic sacral and heel dressings. Santamaria et al\textsuperscript{42,45} and Cubit et al\textsuperscript{13} demonstrated that the use of the multi-layer silicone foam Mepilex Border Sacrum and Mepilex Heel dressings (Molnlycke Health Care AB, Gothenburg, Sweden) significantly decreased PI incidence in critically ill patients when applied in the ED.\textsuperscript{45} The prophylactic use of these dressings has also been shown to decrease overall wound care costs when applied in the ED for critically ill patients.\textsuperscript{46–48}

6 | LEADERSHIP, LOCAL CULTURE, AND MANAGEMENT

All levels of senior and unit-based clinical and hospital management have a vital role in the prevention of PIs in the ED. It is the role of senior-level executives to set expectations and support staff to accomplish PI prevention goals, remove barriers, encourage collaboration, and provide the required human and financial resources. As with any clinical problem, the first step in solving it is to understand the scope and severity of the problem. It is important for management to ensure that valid and reliable data collection processes are in place to assist in understanding the problem of ED-acquired PI.\textsuperscript{41} These systems must provide clinicians with accurate and timely data on the incidence rates of PI developed in the ED and on which patients are at the greatest risk. Similarly, clinicians in the ED need rapid access to appropriate equipment and training to minimise pressure and shear forces in the highly vulnerable emergency patient.

The issue of adequate financial resources for PI prevention in the ED is important, particularly in settings where budgets are devolved to specific departments. The adoption of new evidence-based prevention protocols may increase local clinical costs. An example of this is the introduction of the use of prophylactic dressings to prevent PIs in critically ill ED patients. ED managers need to be prepared to make the case
to the hospital executive for the clinical and cost-effectiveness of such interventions based on the available strong evidence relating to reduction in PI incidence rates, reduction in length of stay, and overall reduction in wound care costs.\textsuperscript{46,48}

A designated executive-level champion for hospital-wide PI prevention can be a valuable link between the boardroom and the bedside of all units, including the ED. In addition, it is important that the ED management team be supportive of an ED-based initiative and practice change to provide PI prevention. Without this mid-level support, unit-based programmes often lack success.\textsuperscript{49} In addition to management support for PI prevention, the hospital's overarching mission, vision, and values typically creates the basis for the local culture that underpins the type and quality of care delivered on individual units. When a change in practice is required, this process is successfully accomplished when the change agent takes into consideration the specific unit's culture. Cultural aspects of the ED should be considered because the assessment of PI risk and the use of systematic evidence-based PI prevention strategies have often not been a standard element of ED practice.

ED PI prevention evidence-based protocols should be developed jointly between ED staff and wound management specialists (often clinical nurse consultants in wound care, wound ostomy continence nurses, or tissue viability nurses) so that the protocols are appropriate to the characteristics, casemix, and staffing of the specific ED. It is the responsibility of the wound management specialist to provide current, evidence-based, validated literature and data supporting the ED practice change to improve PI prevention.

Individual unit-based staff designated to champion a PI prevention programme in the ED are also vital. They directly provide the necessary peer-to-peer influence, holding one another accountable for practice changes. In addition, they can collect and report data, assist with education and competencies, and create ways to celebrate successful outcomes.\textsuperscript{49}

The successful implementation of contemporary PI prevention strategies in the ED may require not only education of all ED staff on the ED incidence rates and pathogenesis of PI but also the evidence of which patients are at greatest risk, recommended preventative interventions, and the need for ongoing monitoring.\textsuperscript{50}

Similarly, the ED staff bears the responsibility of understanding the positive or negative impact their care has on an admitted patients' skin integrity. Collaboration assists engagement from the ED staff and encouragement of “early adopters” who can help drive and sustain practice change. Once new protocols have been developed, implemented, and established in practice, the ED staff can then assume accountability for ED-associated PI prevention and incidence.

\section*{CONCLUSION}

Patients in the ED can be exposed to either sustained tissue deformations because of bodyweight or body interaction with medical devices. The effects of this exposure can be exacerbated by age, critical illness, comorbidity, and environmental factors both prior and during admission to the ED.

The prevention of HAPIs in the ED needs to be improved internationally, and as our understanding of the pathogenesis of these largely preventable injuries evolves, it is essential that EDs continue to develop specific evidence-based preventative protocols against ED-acquired PIs.

The ED is a unique hospital environment with its own strengths and limitations. Clinicians need support from the hospital executive to enable them to more effectively protect the patient who is vulnerable to the development of a HAPI. The enhancement of ED PI prevention strategies will not only help avoid these potentially serious injuries to patients, but they will also improve the efficiency and effectiveness of the hospital by reducing length of stay and helping to reduce costs and the potential for litigation.

We are fortunate to now have much better knowledge, understanding, skills, and equipment underpinned by a large scientific and clinical evidence base on how to prevent PIs developing in the ED. What remains now is to make the prevention of ED-acquired PIs an international priority and to systematically eradicate these injuries.

\section*{ORCID}

Nick Santamaria \(\text{https://orcid.org/0000-0001-5270-6943}\)

Paulo Alves \(\text{https://orcid.org/0000-0002-6348-3316}\)

Amit Gefen \(\text{https://orcid.org/0000-0002-0223-7218}\)

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