

The pressure injury predictive model: A framework for hospital-acquired pressure injuries

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Abstract

Background: Despite decades of research, pressure injuries continue to be a source of significant pain and delayed recovery for patients and substantial quality and cost issues for hospitals. Consideration of the current thinking around pressure injury risk must be evaluated to improve risk assessments and subsequent nursing interventions aimed at reducing hospital-acquired pressure injuries.

Design: This is a discursive paper using Walker and Avant's (2005) theory synthesis framework to examine the relevance of existing pressure injury models as they align with the current literature.

Methods: PubMed and CINAHL indexes were searched, first for conceptual models and then for pressure injury research conducted on hospitalised patients for the years 2006–2016. A synthesis of the searches culminated into a new pressure injury risk model.

Conclusions: Gaps in previous models include lack of attention to the environment, contributing episode-of-care factors and the dynamic nature of injury risk for patients. Through theory synthesis, the need for a new model representing the full risk for pressure injury was identified. The Pressure Injury Predictive Model is a representation of the complex and dynamic nature of pressure injury risk that builds on previous models and addresses new patient, contextual and episode-of-care process influences. The Pressure Injury Predictive Model (PIPM) provides a more accurate picture of the complexity of contextual and process factors associated with pressure injury development.

Relevance to Clinical Practice: Using the PIPM to determine risk can result in improved risk identification. This information can be used to implement targeted, evidence-based pressure injury prevention interventions specific to the patient risk profile, thus limiting unwarranted and unnecessary care.

KEYWORDS

hospitalisation, nursing theory, predictive model, pressure injury, pressure ulcer, theory synthesis

1 | AIM

The aim of this paper is to describe the process of theory synthesis and the development of a framework, the Pressure Injury Predictive Model (PIPM), indicating the complex and dynamic nature of PI risk for hospitalised adult patients.

2 | BACKGROUND

Patients who develop a hospital-acquired pressure injury (HAPI) are 2.8 times more likely to die during their hospital stay ($p < .001$) and 1.69 times more likely to die within 30 days after discharge ($p < .001$) and are subject to significant amounts of pain and suffering, delayed functional recovery and increased length of hospital stay (Lyder et al., 2012; Thomas, Goode, Tarquine, & Allman, 1996). According to the Agency for Healthcare Research and Quality (AHRQ) National Scorecard Data on hospital-acquired conditions, over 1 million patients in the USA develop a pressure injury while in the hospital. Although there was an encouraging 23% decline in the number of patients affected between 2010–2014, rates increased 10% between 2014–2016 (AHRQ, 2018). In addition to the adverse impact on patients (Lyder et al., 2012), recent estimates of care-related costs associated with HAPIs amount to approximately \$25,990 USD per stay (Spector, Limcangco, Owens, & Steiner, 2016). Aggregate estimates are reported to be a staggering \$11 billion (USD) annually (Russo, Steiner, & Spector, 2006).

A pressure injury (PI) is defined as any area of skin or underlying tissue that has been damaged because of intense or prolonged pressure in combination with shear (National Pressure Ulcer Advisory Panel, 2016). The idea that PI is a nursing-sensitive indicator dates back to Florence Nightingale, who described bedsores as “*generally the fault not of the disease, but of the nursing*” (Nightingale, 1859, p. 6). PIs were considered preventable, primarily the result of unrelieved pressure upon bony prominences. Some of the earliest research on PI was done with canine models. For example, Kosiak, Kubicek, Olson, Danz, and Kottke (1958) found an inverse relationship between pressure applied and length of time, such that there was a high degree of tissue susceptibility at low pressure over prolonged periods and higher pressures for shorter timeframes. Microscopic examination of tissue post-pressure application of 60mmHg for one hour showed cellular infiltration, extravasation and hyaline degeneration, with muscular necrosis and venous thrombosis occurring with higher pressure for longer periods of time (Kosiak et al., 1958). This early work gave foundational support for many of the conceptual frameworks developed for identifying a patient's risk for PI, since pressure was identified as the primary cause of what were then called “bedsores” or decubitus ulcers.

Decades of research have resulted in the identification of PI risk factors, interventions to reduce such risk, and evidence-based treatment plans for those who unfortunately develop PIs. These studies illustrate some of the complex systems characteristics of PIs. Specifically, these include the heterogeneity of individual and

What does this paper contribute to the wider global clinical community?

- This paper critically reflects on our understanding of risk for PI development in hospitalised patients, including how risk is represented in current conceptual frameworks.
- Evidence suggests the need for a more accurate, encompassing model that reflects risk for PI.
- The Pressure Injury Predictive Model (PIPM) is the first model to fully capture the complexity and dynamic nature of many PI risk factors, including environmental and episode-of-care factors.
- Use of the PIPM can result in improved identification of risk that can lead to more targeted evidence-based interventions.

environmental factors, the dynamics of the processes and resulting feedback loops involved, and the nonlinear contributing factors at multiple levels. Simply put, PI development is complex and dynamic and involves the interaction of patient, environmental and care process-related factors. In this discursive paper, we examine this complexity as it is reflected in PI conceptual frameworks and current literature.

3 | DESIGN

The original intention of this work was to outline the most recent evidence suggesting factors predictive of hospital-acquired PIs and their alignment with PI risk frameworks. A discursive paper provides a formal mechanism for examining a particular issue or problem, using a for/against, opinion or solution-focused framework. Using Walker and Avant's (2005) theory synthesis method, the need for a new model more accurately representing the complexity in PI risk was made clear. Thus, what follows is a discursive paper suggesting solutions to the identified problem (e.g. failure of current models to reflect factors predictive of hospital-acquired PI).

4 | METHOD

According to Walker and Avant (2005), a primary purpose of theory synthesis is “to represent factors that proceed or influence a particular event (pg. 138).” The theory synthesis process was used to develop the PIPM and includes the following steps:

1. Specifying focal concepts to serve as anchors for the synthesised theory
2. Reviewing the literature to identify factors related to the focal concepts and to specify the nature of relationships;

3. Organising concepts and statements into an integrated and efficient representation of the phenomena of interest (Walker & Avant, 2005).

The first step in the process involved the specification of focal concepts associated with PI, which subsequently provided the foundation for the synthesised theory. For this step, a literature search using PubMed and CINAHL was conducted to identify conceptual models for predicting pressure injury. To be included, the models needed to provide a framework specific for PI development; thus, models focused more broadly on skin alterations were not included.

The second step in the theory synthesis process involved a more in-depth review of the literature to determine the empirical evidence for relationships among the focal constructs and concepts identified in step 1. For this reason, a narrative review was conducted using MeSH and keywords including pressure ulcers/injuries, risk factor and surgical/hospitalised patients in two search engines (PubMed and CINAHL). The primary inclusion criteria included the following: (a) research studies conducted between 2006–2016 and English language; and (b) sample contained adult (>18 years of age) patients. Studies sampling paediatric patients were excluded from the review due to the uniqueness of risk for PI among the two patient populations. The outcome of interest was HAPIs, and thus, studies with the primary purpose of predicting the development of HAPIs were included.

The final step in theory synthesis, organisation of concepts and statements into an integrated representation, included a review of each of the concepts identified in the conceptual model synthesis and empirical evidence. All of the concepts were listed and categorised into clusters. Relational statements were developed among the concepts/clusters to create the graphical representation of the new integrated PIPM.

5 | CONCLUSION

5.1 | Step 1: Specifying focal concepts to serve as anchors for the synthesised theory

Five conceptual models met the inclusion criteria. Concepts and constructs in each of the models were identified and mapped to determine the similarities and differences among the respective models (Table 1).

5.1.1 | Model overview

One of the earliest conceptual models developed for PI research was developed by Braden and Bergstrom (1987). According to the model, pressure—as determined by mobility, activity, sensory perception—in combination with both extrinsic (e.g. moisture, friction, shear) and intrinsic factors (e.g. nutrition, age, arteriolar pressure and other factors) related to tissue tolerance impact PI development.

The intensity and duration of pressure and susceptibility of the tissue determined the level of risk for PI formation. Defloor (1999) expounded upon Braden and Bergstrom's work, arguing that tissue tolerance cannot independently cause a PI. Rather, Defloor (1999) considered tissue tolerance as an intermediate variable, which was further delineated as tissue tolerance for pressure and tissue tolerance for oxygen. According to Defloor (1999), tissue tolerance for pressure refers to factors that “change the capacity of the tissue to redistribute pressure (p. 211).” Factors that further impact tissue tolerance are those affecting oxygen distribution and tissue oxygen needs, which Defloor terms tissue tolerance for oxygen. He further subcategorised pressure as (a) compression force (e.g. perpendicular pressure to the tissue) and (b) shearing force (e.g. parallel pressure to the tissue). The model represented a bidirectional relationship between both types of pressure, mediated by tissue tolerance, as the determinant of PI risk.

In alignment with earlier PI conceptual models, Benoit and Mion (2012) created a conceptual model for PI aetiology in critically ill patients. Like Braden and Bergstrom (1987) and Defloor (1999), they acknowledged pressure and tissue tolerance as the primary constructs associated with PI development. Specifically, the authors posited that tissue tolerance, including intrinsic and extrinsic factors, moderated the effect of pressure on PI development. Recently, Coleman et al. (2014) and García-Fernández, Agreda, Verdú, and Pancorbo-Hidalgo (2014) have considered other factors as contributory to PI development. Coleman et al. (2013) conducted a systematic review and consensus study to identify the critical determinants of PI development. Based on the results of this work, a new conceptual model was developed which recognised PI development as directly related to mechanical boundary conditions and the susceptibility and tolerance of the individual (Coleman et al., 2014). Likewise, García-Fernández et al. (2014) diverged from earlier PI models by identifying only pressure and shear forces as the primary factors associated with PI development. Forces of pressure, according to the model, include dimensions that reduce repositioning and sensory perception. They considered shear force an independent predictor, indicating that the presence of shearing, even with lower pressure, may cause a PI. Tissue tolerance was deemed a predisposing factor but not aetiologic.

5.1.2 | Model synthesis

The conceptual models reviewed were remarkably similar in terms of major constructs and concepts associated with PI development, although the relational statements varied between the models (Table 1). Pressure, although being defined through various concepts within the respective models, played a primary role in PI development among the conceptual models. Pressure can be impacted by a variety of factors, including mobility, activity, shear, nutrition and perfusion. Individuals who had reduced activity/mobility, inadequate nutrition or poor perfusion were at greater risk for PIs. The specific intensity and duration of pressure needed for PI development was

TABLE 1 Mapping of constructs in current pressure injury risk models

Construct	Braden and Bergstrom (1987)	DeFloor (1999)	Garcia-Fernández, Agreda, Verdu, & Pancorbo-Hidalgo (2014)	Benoit and Mion (2012)	Coleman et al. (2013)
Pressure	Mobility Activity Sensory perception	<u>Compression force</u> : intensity (support, posture, body build, medical/nursing interventions); duration (pain reaction, pain sensitivity, mobility/activity, medical/nursing interventions) <u>Shearing force</u> : intensity (support, posture, maceration, friction); duration (pain reaction, pain sensitivity, mobility/activity, medical/nursing interventions)	Capacity for repositioning Sensory perception	Mobility Activity Sensory perception	<i>*deemed Mechanical Boundary Conditions</i> Magnitude and duration of load; Type of load (pressure, friction, shear) Risk factors: immobility, poor sensory perception and response
Shear	<i>Aligned with tissue tolerance</i>	<i>Aligned as a component of pressure</i>	Shear forces (e.g. opposite/parallel sliding of tissue)	<i>Aligned with tissue tolerance</i>	<i>Aligned as a component of pressure</i>
Tissue tolerance	Extrinsic factors: moisture, friction, shear Intrinsic factors: nutrition, age, arteriolar pressure, other (interstitial fluid flow, emotional stress, smoking, skin temperature)	<u>Tissue tolerance for pressure</u> : tissue mass, age dehydration, protein/Vit C deficiency, corticosteroid, stress <u>Tissue tolerance for oxygen</u> : O2 Needs (temperature); O2 Supply (medication, protein deficiency, smoking, diseases (O2 supply, reactive hyperaemia, vascular occlusion), blood pressure	Considered a coadjuvant (predisposing factor that can influence or induce the development of lesion)	<u>Extrinsic factors</u> : moisture, friction/shear <u>Intrinsic factors</u> : metabolic supply and demand, pressure distribution capacity, threats to skin integrity	<i>*Deemed individual susceptibility and tolerance</i> Physiology and repair; transport and thermal properties; mechanical properties of tissue (stiffness strength); geometry (size/shape of different tissue layers) of the tissue and bones Risk factors: skin/PI status, poor perfusion, poor nutrition, diabetes, moisture, low albumin
Other			Other adjuvant factors include tissue nutrition, oxygen alterations, skin alterations		

dependent on other factors, including the ability of tissue to tolerate the intensity and duration.

Like pressure, tissue tolerance was significant in each of the models reviewed, although interpretation varied. Four of the five models denoted concepts of tissue tolerance, such as moisture, friction, smoking, skin temperature and fluid volume, respectively. Despite inclusion of tissue tolerance as a construct, differences in the relational role varied among models. For example, Benoit and Mion (2012) identified tissue tolerance as a moderator between pressure and PI, whereas Coleman et al. (2014) described a bidirectional relationship between individual susceptibility (e.g. tissue tolerance) and mechanical boundary conditions (e.g. pressure).

The final construct found in all models was shear. Shear was primarily considered in association with pressure (Coleman et al., 2014; DeFloor, 1999) or tissue tolerance (Benoit & Mion, 2012; Braden & Bergstrom, 1987). Only Garcia-Fernández et al. (2014) recognised shear as an independent construct. The relationship between shear and PI development warrants further study.

5.1.3 | Summary

Despite the similarities, it is clear from the review that further consideration of the risks of PI development must be explored.

Conceptual models to date primarily represent only one-directional relationships, including a smattering of risk factors, certainly not all of the factors that are predictive of PI. For example, thus far, the roles that evolving care processes and environmental factors may play in PI development are not included in current conceptual models. Similarly, model developers to date have not considered the impact of varying combinations of risk, which could exponentially influence levels of PI risk. For this reason, the next phase of work included a review of the current evidence about factors that are predictive of PI development.

5.2 | Step 2: Reviewing the literature to identify factors related to the focal concepts and to specify the nature of relationships

A total of 662 distinct publications were found during the initial search. The research team appraised titles and abstracts, eliminating 305 articles after title review and an additional 162 articles after abstract review. Full text articles were obtained and reviewed to assure alignment with the primary purpose of risk prediction for HAPI development. Upon this final review, only 59 studies met all the inclusion criteria (Figure 1). Several studies were excluded because the dependent variable was not HAPI (e.g. community-acquired PIs) or the purpose was on the evaluation of specific nursing interventions

to reduce PI risk (e.g. mattress type, positioning and mobility protocols, nutrition interventions), rather than predicting the development of a HAPI.

For each of the remaining studies, the levels of evidence were examined and categorised according to Melnyk and Fineout-Overholt (2011), one of a number of evidence rating systems used by academics, researchers and clinicians to evaluate the strength of the evidence. Levels range from level 1 (systematic review and meta-analysis of randomised controlled trials or clinical guidelines based on systematic reviews or meta-analyses)–level 7 (e.g. expert opinion); the higher the level of evidence, the greater the strength in the identified results. For the studies included in this review, the majority were case-control or cohort studies (level 4, $n = 21$) and single descriptive or qualitative studies (level 6, $n = 33$). There were two meta-analyses (level 1) and three systematic reviews (level 5).

The focal constructs and concepts for PI risk, as identified in the literature, will be described under the following headings: pressure, tissue tolerance and shear. In an effort to synthesise the studies, the total number of studies including each concept as an independent variable was determined. Subsequently, the findings for each study were reviewed to determine whether the specific concept was deemed a significant predictor of PI, in order to compute a percentage reflecting the frequency of significance (e.g. number of studies with a significant finding for the specified construct divided by the

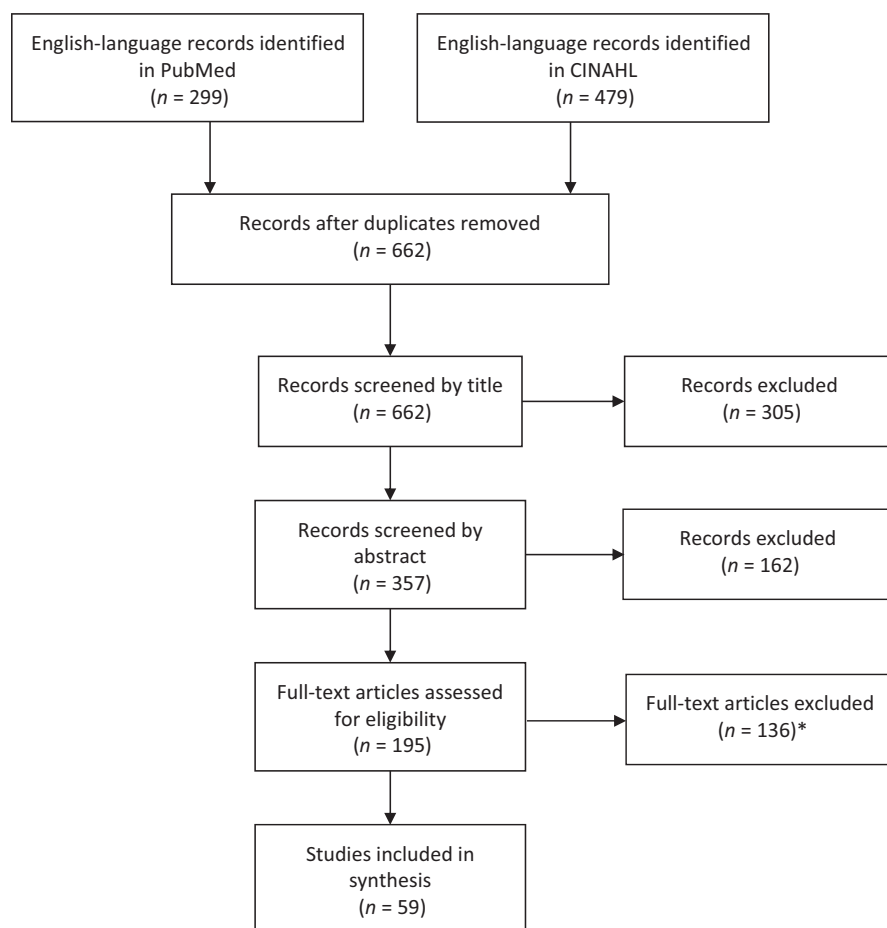


FIGURE 1 Literature flow diagram for years 2006–2016. *Articles excluded when the dependent variable was not hospital acquired pressure injury or the purpose was not the prediction of pressure injury

total number of studies evaluating the specific construct). Details of the constructs, concepts, indicators and relationships to PI development are shown in Table 2.

5.2.1 | Pressure

Factors that affect the duration and intensity of pressure have been the focus of research designed to test the extent to which they contribute to PI development. A total of 24 studies evaluated concepts associated with pressure, including mobility/activity, sensory perception, body posture/stature, pain and equipment type (e.g. mattress type, medical devices) (Table 3). Of the studies reviewed, indicators of mobility (80%, $n = 16$ of 20 studies evaluating mobility found it to be a significant predictor) and activity (62%, $n = 8$ of 13 studies) were significantly associated with PI development, such that limitations in mobility and activity resulted in greater risk for PIs (Table 2). Pain and sensory perception were evaluated less frequently; however, results show that these concepts are related to HAPI development. Specifically, lower sensory perception, which included an inhibited sense of pain in one study, resulted in an increased risk for HAPI. Two studies directly examined the association of pain and PIs, both of which found significant positive relationships with higher pain levels resulting in an increased risk for HAPI (Rao, Preston, Strauss, Stamm, & Zalman, 2016; Skogestad et al., 2017). The evidence did not substantiate body posture and stature, initially identified as a focal concept in the model presented by DeFloor (1999). No significant relationship was identified in any of the reviewed studies in which HAPI was the outcome (Kaitani, Tokunaga, Matsui, & Sanada, 2010; Tsaousi et al., 2014; Yoshimura et al., 2015). Medical devices, such as endotracheal tubes, faecal diversion devices, tracheostomies and face masks, were found to put patients at greater risk for HAPI in two of three studies (Black et al., 2010; Bly, Schallom, Sona, & Klinkenberg, 2016; Slowikowski & Funk, 2010). Black et al. (2010) reported that patients with medical devices were 2.4 times more likely to develop a HAPI than those without such devices. Furthermore, a significant association between hospital mattress type and HAPI development was found in two of three studies reviewed (Bly et al., 2016; Kaitani et al., 2010; Tescher, Branda, Byrne, & Naessens, 2012).

5.2.2 | Tissue tolerance

According to current models, tissue tolerance, conceptually defined by Braden and Bergstrom (1987) as the ability of the skin and supporting structures to endure pressure without complications, is a primary construct in determining a patient's risk for PIs. Of the studies included in the narrative review, 41 studies examined a concept associated with tissue tolerance: presence of certain comorbidities, age, body mass index (BMI), moisture, nutrition, oxygenation and perfusion (Table 3). Higher rates of HAPIs have

been found in patients with several chronic conditions including diabetes mellitus (Delmore, Lebovits, Suggs, Rolnitzky, & Ayello, 2015; Liu, He, & Chen, 2012; Nassaji, Askari, & Ghorbani, 2014), pulmonary diseases (Bly et al., 2016), cardiovascular diseases (Cox & Roche, 2015), malignant tumours (Rao et al., 2016) and renal/liver disease (O'Brien, Shanks, Talsma, Brenner, & Ramachandran, 2014). Of the studies including chronic conditions as a predictor of HAPIs, 74% (23 of 31 studies) noted a significant increase in risk with the presence of the chronic condition (Table 2). Increase in age was consistently shown to be a significant predictor of HAPI (66%, $n = 29$ of 44 studies); however, BMI was only a significant predictor in 45% of the studies included in the literature review ($n = 10$ of 22). Specifically, those with extreme BMIs (e.g. morbid obesity or underweight) were more likely associated with HAPI development. Weight was only related to HAPIs in 21% of the studies that included it as an independent variable ($n = 3$ of 14). Evidence for including nutrition as predictors of HAPI is stronger, with 78% ($n = 18$ of 23) of studies noting a significant relationship. Albumin levels, an indicator of nutrition status, were found to have a significant negative relationship with HAPI development in 69% ($n = 9$ of 13) of the studies reviewed, and glucose levels were a significant predictor in both studies examining the relationship (e.g. higher glucose levels associated with increased risk). Patients who required support for oxygenation via mechanical ventilation, facemask and nasal cannula were consistently found to be at higher risk for HAPIs (70%, $n = 7$ of 10 studies). Any form of oxygenation support during a patient stay or use of bronchodilators increased the risk for PI development, with some studies finding a greater than fivefold increase in risk among patients with oxygen support. Similarly, studies evaluating the impact of perfusion on PIs found significant relationships (75%, $n = 12$ of 16). For example, the use of vasopressors during the patient's stay resulted in up to an 8 times increase in risk for PI (66%, $n = 6$ of 9 studies). Laboratory values used as proxy measures for perfusion were found to be mixed in terms of their usefulness for predicting HAPI development. Lower haemoglobin levels were associated with an increased risk for HAPI in 55% ($n = 6$ of 11) of the reviewed studies, while haematocrit values were less predictive (20%, $n = 1$ of 5). Temperature, also used as a measure for perfusion, was shown to be a significant predictor for HAPI in 71% ($n = 5$ of 7) of studies reviewed. Like BMI, extremes in temperature (low or high) increase HAPI risk. Finally, moisture, measured as incontinence, excessive moisture or oedema or via the moisture Braden subscore, was found to be a significant predictor of HAPIs in 56% of the studies reviewed ($n = 13$ of 23) (Table 2). Patients exhibiting excessive moisture, via diaphoresis, incontinence or weeping oedema, were shown to be at an increased risk for HAPI.

5.2.3 | Shear

García-Fernández et al. (2014) identified shear as an independent focal concept. According to the National Pressure Ulcer Advisory

TABLE 2 Focal constructs and concepts identified in the literature for PI risk

Construct	Concept	Indicators and types of variable measurement within the studies	# studies finding significance/# studies with variable	Type of relationship (summary)	References
Patient	Comorbidities	<ul style="list-style-type: none"> - Presence of comorbidities (e.g. cardiac, pulmonary, vascular, infection, paralysis, trauma, presence of PI, neurological, cancer) - Number of chronic diseases - Presence of multiple-organ failure - Self-reported health status 	23/31	<ul style="list-style-type: none"> - Type of comorbidities does impact the development of PI - Greater numbers of comorbidities increases risk for PI development - Smokers at greater risk for PI - Those reporting poor health status at greater risk for PI (1 study) 	<p>Aljezawi, Al Qadire, & Tubaishat, 2014; Bly et al., 2016; Corniello et al., 2014; Cowan et al., 2012; Cox, 2011; Cox & Roche, 2015; Delmore et al., 2015; Demarre et al., 2015; Liu et al., 2012; Man & Au-Yeung, 2013; Manzano et al., 2010; Nassaji et al., 2014; O'Brien et al., 2014; Raff, Waller, Griffin, Kerby, & Bosarge, 2016; Rao et al., 2016; Sardo et al., 2016; Saunders et al., 2012; Scheel-Sailer, Wyss, Boldt, Post, & Lay, 2013; Schoonhoven et al., 2006; Serra et al., 2014; Shahin et al., 2010; Skogestad et al., 2017; Slowikowski & Funk, 2010; Smit, Harrison, Letzkus, & Quatrara, 2016; Tescher et al., 2012; Thomas, Vinodkumar, Mathew, & Setia, 2015; Tsaousi et al., 2014; Tschannen, Bates, Talsma, & Guo, 2012; Webster et al., 2015; Yoshimura et al., 2015; Shaw, Chang, Lee, Kung, & Tung, 2014</p>
Patient	Age	<ul style="list-style-type: none"> - Age in years - Categorized by age 	29/44	<ul style="list-style-type: none"> - Increase in age results in a greater risk for PI development 	<p>Schoonhoven et al., 2006; Raff et al., 2016; Tescher et al., 2012; Slowikowski & Funk, 2010; Aljezawi et al., 2014; Connor, Sledge, Bryant-Wiersma, Stamm, & Potter, 2010; Corniello et al., 2014; Bredesen, Bjoro, Gunningberg, & Hofoss, 2015; Brito, Vasconcelos Generoso, & Correia, 2013; Chen, Shen, Xu, Zhang, & Wu, 2015; Skogestad et al., 2017; Sardo et al., 2016; Delmore et al., 2015; Cowan et al., 2012; Rao et al., 2016; Cox & Roche, 2015; Fred, Ford, Wagner, & VanBrackle, 2012; Hyun et al., 2014; Man & Au-Yeung, 2013; Saunders et al., 2012; Smit et al., 2016; Yoshimura et al., 2015; Demarre et al., 2015; Baumgarten et al., 2006; Cox, 2011; Hayes et al., 2015; Manzano et al., 2010; Nassaji et al., 2014; O'Brien et al., 2014; Raju, Su, Patrician, Loan, & McCarthy, 2015; Scheel-Sailer et al., 2013; Shahin et al., 2010; Shen, Chen, Xu, Zhang, & Wu, 2015; Stifter et al., 2015; Tsaousi et al., 2014; Tschannen et al., 2012; Webster et al., 2015; Coleman et al., 2013; Cremasco, Wenzel, Zanei, & Whitaker, 2013; Serra et al., 2014; Baldi, Ferrando, Foltran, Ciccone, & Gregori, 2010; Kaitani et al., 2010; Shaw et al., 2014</p>

(Continues)

TABLE 2 (Continued)

Construct	Concept	Indicators and types of variable measurement within the studies	# studies finding significance/# studies with variable	Type of relationship (summary)	References
Patient	Body mass index (BMI)	- Categorized according to the World Health Organization	10/22	- Those with extremes of BMI (e.g. underweight, morbid obesity) are at greater risk for PI development	Shahin et al., 2010; Tescher et al., 2012; Skogestad et al., 2017; Tsaousi et al., 2014; Baumgarten et al., 2006; Bly et al., 2016; Corniello et al., 2014; Hyun et al., 2014; Cowan et al., 2012; Delmore et al., 2015; Kaitani et al., 2010; Raff et al., 2016; Rao et al., 2016; Cox & Roche, 2015; Miller, Frankenfield, Lehman, Maguire, & Schirm, 2016; Slowikowski & Funk, 2010; Yoshimura et al., 2015; Connor et al., 2010; O'Brien et al., 2014; Tschannen et al., 2012; Shaw et al., 2014; Gardiner, Reed, Bonner, Haggerty, & Hale, 2016
Patient	Weight	- Pounds/kilograms - Percentage of weight loss in specified timeframe - Categorized as heavy/light via Kg	3/14	- Those with weight loss or thinner deemed at greater risk	Schoonhoven et al., 2006; Corniello et al., 2014; Miller et al., 2016; Chen et al., 2015; Webster et al., 2015; Shahin et al., 2010; Shen et al., 2015; Cox & Roche, 2015; Demarre et al., 2015; Hayes et al., 2015; Smit et al., 2016; Fred et al., 2012; Manzano et al., 2010; Tsaousi et al., 2014
Patient	Gender	- Male/female	6/31	- Male to be of greater risk at developing PI in those with significant findings	Aljezawi et al., 2014; Aydin, Donaldson, Stotts, Fridman, & Brown, 2015; Baldi et al., 2010; Shaw et al., 2014; Baumgarten et al., 2006; Bredesen et al., 2015; Brito et al., 2013; Chen et al., 2015; Coleman et al., 2013; Corniello et al., 2014; Cremasco et al., 2013; Demarre et al., 2015; Fred et al., 2012; Gardiner et al., 2016; Hyun et al., 2014; Kaitani et al., 2010; Man & Au-Yeung, 2013; Manzano et al., 2010; O'Brien et al., 2014; Raff et al., 2016; sardo et al., 2016; Saunders et al., 2012; Scheel-Sailer et al., 2013; Shen et al., 2015; Skogestad et al., 2017; Slowikowski & Funk, 2010; Smit et al., 2016; Tescher et al., 2012; Tsaousi et al., 2014; Tschannen et al., 2012; Yoshimura et al., 2015
Patient	Ethnicity	- Categorized by ethnicity (e.g. Caucasian, African American, other)	3/8	- African American and "other race" noted to be at greater risk for PI development	Baumgarten et al., 2006; Coleman et al., 2013; Corniello et al., 2014; Cowan et al., 2012; Gardiner et al., 2016; Hyun et al., 2014; Raff et al., 2016; Saunders et al., 2012
Patient	Height	- Actual measurement	0/2	- No relationship identified	Demarre et al., 2015; Fred et al., 2012
Patient	Marital status	- Categorized as married/nonmarried	1/1	Those nonmarried were at greater risk for PI development	Corniello et al., 2014

(Continues)

TABLE 2 (Continued)

Construct	Concept	Indicators and types of variable measurement within the studies	# studies finding significance/# studies with variable	Type of relationship (summary)	References
Patient	Socio-economic status	<ul style="list-style-type: none"> - Education level - Income level - Healthcare coverage (e.g. self-report: insurance, seen provider in last 12 months, unable to see provider due to cost) 	1/1	- Those with less than a bachelor's degree at greater risk for PI development	Saunders et al., 2012
Pressure	Mobility	<ul style="list-style-type: none"> - Braden subscale: mobility - Chemical or mechanical paralysis - RAS mobility subscale - Immobility (e.g. plegia, debility, CVA, joint disease and orthopaedic surgeries) - Required assistance with mobility - Modified Barthel Index - Mobility categorisation (e.g. slightly limited/full mobility; or immobile/very limited) - Number of daily turns 	16/20	<ul style="list-style-type: none"> - Reduced mobility increases risk for PI development - Those with paralysis or bedridden increases the risk for PI 	Aljezawi et al., 2014; Baumgarten et al., 2006; Bly et al., 2016; Brito et al., 2013; Coleman et al., 2013; Cowan et al., 2012; Cox, 2011; Delmore et al., 2015; Demarre et al., 2015; Kaitani et al., 2010; Lahmann & Kottner, 2011; Man & Au-Yeung, 2013; Michel et al., 2012; Raju et al., 2015; Rao et al., 2016; Schoonhoven et al., 2006; Slowikowski & Funk, 2010; Stifter et al., 2015; Tescher et al., 2012; Webster et al., 2015
Pressure	Medical devices	<ul style="list-style-type: none"> - Presence of a medical device (e.g. endotracheal tube, faecal diversion device, tracheostomy and face masks) 	2/3	- Patients with a medical device at a greater risk for PI (the majority of studies focused on a specific device).	Bly et al., 2016; Black et al., 2010; Slowikowski & Funk, 2010
Pressure	Body posture/stature	<ul style="list-style-type: none"> - Bony protrusion near sacral region (yes/no) - Bony prominence (yes/no) - Arm and calf perimeter 	0/3	- No relationship identified	Kaitani et al., 2010; Tsaousi et al., 2014; Yoshimura et al., 2015
Pressure	Pain	<ul style="list-style-type: none"> - Presence of pain with activity - Presence of pain (general) - Presence of severe pain at rest 	2/2	- Higher levels of pain increase the risk for PI development	Skogestad et al., 2017; Rao et al., 2016
Pressure	Sensory perception	<ul style="list-style-type: none"> - Braden subscale: sensory perception - Inhibited sense of pain 	6/10	<ul style="list-style-type: none"> - A lower sensory perception score increases risk for PI development in studies reporting significance - Inhibited sense of pain increased risk for PI development ($n = 1$) 	Aljezawi et al., 2014; Coleman et al., 2013; Cowan et al., 2012; Cox, 2011; Demarre et al., 2015; Kaitani et al., 2010; Lahmann & Kottner, 2011; Raju et al., 2015; Rao et al., 2016; Tescher et al., 2012
Pressure	Mattress type	<ul style="list-style-type: none"> - Final surface of bed (e.g. standard, pressure relieving, specialty) - Specialty bed (yes/no) 	2/3	- Type of mattress and time of which patient placed on mattress impact PI development	Tescher et al., 2012; Bly et al., 2016; Kaitani et al., 2010

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TABLE 2 (Continued)

Construct	Concept	Indicators and types of variable measurement within the studies	# studies finding significance/# studies with variable	Type of relationship (summary)	References
Pressure	Activity	<ul style="list-style-type: none"> - Braden subscale: Activity - Bedrest (yes/no) - Activity types (e.g. no limitation, walks occasionally, chair or bedfast) - Activities of Daily Living - RAS mobility subscale 	8/13	- Limited activity increases risk for PI development	Cox, 2011; Demarre et al., 2015; Lahmann & Kottner, 2011; Raju et al., 2015; Tescher et al., 2012; Shahin et al., 2010; Slowikowski & Funk, 2010; Aljezawi et al., 2014; Cowan et al., 2012; Tsaousi et al., 2006; Kaitani et al., 2010; Coleman et al., 2013
Shear	Friction and shear	<ul style="list-style-type: none"> - Braden subscale: Friction & Shear - Shear assessment (e.g. no problem or potential/actual problem) 	8/12	- Greater friction and shear results in increased risk for PI development	Aljezawi et al., 2014; Cowan et al., 2012; Cox, 2011; Demarre et al., 2015; Kaitani et al., 2010; Lahmann & Kottner, 2011; Raju et al., 2015; Rao et al., 2016; Schoonhoven et al., 2006; Shahin et al., 2010; Tescher et al., 2012; Shaw et al., 2014
Tissue tolerance	Nutrition	<ul style="list-style-type: none"> - Appetite level - Diet type (e.g. oral, enteral, parenteral) - Basal metabolic index - Braden subscale: nutrition - Levels of malnourishment - Subjective Global Assessment (SGA) - Presence of feeding tube 	18/23	<ul style="list-style-type: none"> - Lower Braden nutrition scores increased risk for PI development - Malnourished patients at greater risk for PI development - Those with abnormal appetite at greater risk 	Slowikowski & Funk, 2010; Tsaousi et al., 2014; Fred et al., 2012; Miller et al., 2016; Shahin et al., 2010; Aljezawi et al., 2014; Cowan et al., 2012; Rao et al., 2016; Michel et al., 2012; Serra et al., 2014; Stifter et al., 2015; Kaitani et al., 2010; Coleman et al., 2013; Demarre et al., 2015; Lahmann & Kottner, 2011; Cox, 2011; Raju et al., 2015; Tescher et al., 2012; Brito et al., 2013; Bly et al., 2016; Banks, Graves, Bauer, & Ash, 2010; Baumgarten et al., 2006; Man & Au-Yeung, 2013
Tissue tolerance	Perfusion	<ul style="list-style-type: none"> - Blood pressure (e.g. hours with hypotension, low MAP) - Presence of perfusion related comorbidities (e.g. CV disease, ankle brachial index; oedema) - Hydration levels - Use of cardiopulmonary bypass - Hydration and perfusion: NNN labels 	12/16	<ul style="list-style-type: none"> - Hypotension associated with increased risk for PI - Hydration issue may increase risk for PI development 	Aljezawi et al., 2014; Bly et al., 2016; Coleman et al., 2013; Connor et al., 2010; Corniello et al., 2014; Cox, 2011; Cox & Roche, 2015; Delmore et al., 2015; Demarre et al., 2015; Man & Au-Yeung, 2013; O'Brien et al., 2014; Raff et al., 2016; Rao et al., 2016; Shahin et al., 2010; Slowikowski & Funk, 2010; Stifter et al., 2015

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TABLE 2 (Continued)

Construct	Concept	Indicators and types of variable measurement within the studies	# studies finding significance/# studies with variable	Type of relationship (summary)	References
Tissue tolerance	Oxygenation	<ul style="list-style-type: none"> - Mechanical ventilation - Oxygen requirements (e.g. ventilator, nasal cannula, face mask) - SvO₂ - P/F < 200 - Use of inhaled dilators - Shortness of breath 	7/10	<ul style="list-style-type: none"> - Use of mechanical ventilation increased risk for PI - Poor oxygenation increased risk for PI - Use of dilators increased risk for PI 	Bly et al., 2016; Cox & Roche, 2015; Delmore et al., 2015; Manzano et al., 2010; O'Brien et al., 2014; Raff et al., 2016; Rao et al., 2016; Skogestad et al., 2017; Slowikowski & Funk, 2010; Smit et al., 2016
Tissue tolerance	Medication: vasopressor use (perfusion)	<ul style="list-style-type: none"> - Intraoperative use of vasopressor (yes/no) - Postoperative use of vasopressor (yes/no) - Total hours of administration of vasopressors - Highest/lowest dose - count of vasopressors 	6/9	<ul style="list-style-type: none"> - Administration of vasopressors (either intra- or postoperative) increases the risk of PI development 	Rao et al., 2016; Chen et al., 2015; Cox, 2011; Bly et al., 2016; Cox & Roche, 2015; Tschannen et al., 2012; O'Brien et al., 2014; Shen et al., 2015; Smit et al., 2016
Tissue tolerance	Moisture	<ul style="list-style-type: none"> - Incontinence (e.g. urinary, faecal, dual) - Excessive moisture (e.g. weeping, drainage) - Braden subscale: moisture - Perspiration - Contenance: NNN labels - Oedema - Wet (yes/no) 	13/23	<ul style="list-style-type: none"> - Those with increased moisture results in greater PI development - Incontinence may increase the risk for PI development 	Beeckman, Van Lancker, Van Hecke, & Verhaeghe, 2014; Rao et al., 2016; Corniello et al., 2014; Cowan et al., 2012; Slowikowski & Funk, 2010; Nassaji et al., 2014; Demairre et al., 2015; Schoonhoven et al., 2006; Coleman et al., 2013; Cox, 2011; Lahmann & Kottner, 2011; Raju et al., 2015; Tescher et al., 2012; Kaitani et al., 2010; Man & Au-Yeung, 2013; Baumgarten et al., 2006; Webster et al., 2015; Yoshimura et al., 2015; Shahin et al., 2010; Aljezawi et al., 2014; Stifter et al., 2015; Skogestad et al., 2017; Shaw et al., 2014
Tissue tolerance	Level of consciousness	<ul style="list-style-type: none"> - Senility/dementia - Mental status scale - Cognition (based on NNN labels) 	1/3	<ul style="list-style-type: none"> - Senility/dementia placed patient at greater risk for developing a PI (1 study) 	Stifter et al., 2015; Coleman et al., 2013; Cowan et al., 2012
Tissue tolerance	Nutrition (laboratories: albumin)	<ul style="list-style-type: none"> - Albumin level (actual, average, binary) - Prealbumin (actual, average) 	9/13	<ul style="list-style-type: none"> - Lower albumin levels increase the risk for PI development 	Aljezawi et al., 2014; Bly et al., 2016; Coleman et al., 2013; Corniello et al., 2014; Cowan et al., 2012; Michel et al., 2012; Raju et al., 2015; Serra et al., 2014; Skogestad et al., 2017; Smit et al., 2016; Kaitani et al., 2010; Man & Au-Yeung, 2013; Cox & Roche, 2015
Tissue tolerance	Nutrition (laboratories: BUN)	<ul style="list-style-type: none"> - BUN (actual, average, binary) 	2/3	<ul style="list-style-type: none"> - Higher BUN increases the risk for PI development 	Raju et al., 2015; Corniello et al., 2014; Aljezawi et al., 2014

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TABLE 2 (Continued)

Construct	Concept	Indicators and types of variable measurement within the studies	# studies finding significance/# studies with variable	Type of relationship (summary)	References
Tissue tolerance	Nutrition (laboratories: creatinine)	- Creatinine (actual, average, binary)	3/4	- Higher levels of creatinine associated with increased risk of PI development	Aljezawi et al., 2014; Rao et al., 2016; Corniello et al., 2014; Raju et al., 2015
Tissue tolerance	Perfusion (laboratories: HgB)	- HgB levels (average, binary, actual) - anaemia at admission	6/11	- Lower HgB levels associated with increased risk of PI development	Coleman et al., 2013; Corniello et al., 2014; Cowan et al., 2012; Kaitani et al., 2010; Man & Au-Yeung, 2013; Shahin et al., 2010; Aljezawi et al., 2014; Bly et al., 2016; Skogestad et al., 2017; Rao et al., 2016; Shaw et al., 2014
Tissue tolerance	Nutrition (laboratories: glucose)	- Glucose levels (average, categorised as high/low/normal)	2/2	- High glucose levels associated with increased risk of PI development	Bly et al., 2016; Corniello et al., 2014
Tissue tolerance	Perfusion (laboratories: Hct)	- Hct levels (average, categorised as low/high)	1/5	- Lower Hct associated with increased risk (1 study)	Corniello et al., 2014; Cowan et al., 2012; Shahin et al., 2010; Slowikowski & Funk, 2010; Skogestad et al., 2017; Shaw et al., 2014
Tissue tolerance	Laboratories: others	- WBC (actual, binary) - Total protein (average) - Potassium - Lymphocyte - Sodium	3/6	- Higher levels of total protein may increase risk for PI Bivariate association only*	Man & Au-Yeung, 2013; Coleman et al., 2013; Aljezawi et al., 2014; Skogestad et al., 2017; Coleman et al., 2013; Kaitani et al., 2010
Tissue tolerance	Temperature (perfusion)	- Intraoperative temperature (actual, categorised) - Body temperature during hospital stay (average, categorised) - Temperature drop postsurgery	5/7	- Increased temperatures can increase risk for PI development - Extremes in temperature (low or high) can increase risk for PI development	Demarre et al., 2015; Coleman et al., 2013; Bly et al., 2016; Yoshimura et al., 2015; Fred et al., 2012; Shahin et al., 2010; Shaw et al., 2014
Environment	Hospital environment	- Bed size (categorised) - Hospital setting (rural/urban) - Hospital type (university/public or philanthropic/private) - hospital teaching status - Hospital Magnet certification (yes/no)	4/6	- Magnet certification not associated with PI development (controlling for unit/hospital covariates) - Private hospitals may put patients at greater risk for PI development (1 study) - larger hospitals may put patients at greater risk - no relationship with hospital setting and PI	Aydin et al., 2015; Bredeesen et al., 2015; Brito et al., 2013; He, Staggs, Bergquist-Beringer, & Duntton, 2013; Ma & Park, 2015; Manojlovich, Antonakos, & Ronis, 2010

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Construct	Concept	Indicators and types of variable measurement within the studies	# studies finding significance/# studies with variable	Type of relationship (summary)	References
Environment	Nursing workload	- Nursing Activities Score (NAS); activities included positioning, laboratories, medications and mobilisation	1/1	- More nursing activity (e.g. higher workload for a patient) reduced the risk for PI	Cremaresco et al., 2013
Environment	Nurse staffing	- HPPD - Number of patients cared for on last shift (self-report) - Nurse/patient ratio - Per cent contract hours: RN turnover - Nursing skill mix - Nursing expertise (% with certification)	5/7	- Higher HPPD may result in lower PI risk - Nursing expertise not associated with PI (1 study) - More contract hours associated with increased PI risk - Higher number of patients increase risk for PI - Higher skill mix may result in lower PI	Aydin et al., 2015; Cho, Chin, Kim, & Hong, 2016; Choi, Choi, & Kim, 2014; He et al., 2013; Ma & Park, 2015; Manojlovich et al., 2010; Stifter et al., 2015
Environment	Environment: census	- Bed turnover - Average daily census per unit	1/1	- Higher bed turnover results in fewer developed PIs	Aydin et al., 2015
Environment	Work environment	- Culture (categorised as developmental, hierarchical, rational, group) - Team climate - Practice Environment Scale of Nursing Working Index (PES-NWI)	2/3	- Better work environment (PES-NWI) results in fewer developed PIs	Bosch et al., 2011; Cho et al., 2016; Ma & Park, 2015
Environment	Unit type	- Surgical versus Medical - ICU (yes/no) - Categorised units (e.g. general care, ICUs, step-down, cardiac) - Ward type (as it relates to prevention strategies)	12/13	- Patients with ICU stays at greater risk for PI development - Overall, surgical patients at greater risk for PI development than medical patients	Gardiner et al., 2016; Manzano et al., 2010; Rao et al., 2016; Corniello et al., 2014; Tescher et al., 2012; Delmore et al., 2015; Baldi et al., 2010; Demarre et al., 2015; Sardo et al., 2016; Schoonhoven et al., 2006; Thomas et al., 2015; He et al., 2013; Bredesen et al., 2015
Environment	Seasonal variations	- Fall, winter, spring, summer	2/2	- Seasonal variations can increase risk for PI development	He et al., 2013; Manzano et al., 2010
Environment	Risk assessment for PI	- Percentage of patients at risk for PI - Per cent risk assessed within 24 hr of admission	1/1	- Higher % of patients at risk for PI development in a system results in greater PIs	Aydin et al., 2015

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TABLE 2 (Continued)

Construct	Concept	Indicators and types of variable measurement within the studies	# studies finding significance/# studies with variable	Type of relationship (summary)	References
Episode of care	Admission type	<ul style="list-style-type: none"> - Categorised as scheduled, nonscheduled/emergent, transferred - Identification as to patient residence prior to admission 	4/6	<ul style="list-style-type: none"> - Those admitted to home less likely to develop PI - Emergency and transfer admission patients more likely to develop PI 	Bly et al., 2016; Webster et al., 2015; Sardo et al., 2016; Gardiner et al., 2016; Kaitani et al., 2010; Raff et al., 2016
Episode of care	Braden score (total)	<ul style="list-style-type: none"> - Total score of Braden - Categorised as "at risk" or "no risk" - Admission total Braden score 	21/24	<ul style="list-style-type: none"> - Lower total Braden scores increase the risk of PI development - Admission Braden score predictive of PI development 	Tschannen et al., 2012; Cox & Roche, 2015; Cox, 2011; Cremasco et al., 2013; Rao et al., 2016; Hayes et al., 2015; Delmore et al., 2015; Baldi et al., 2010; Hyun et al., 2014; Shahin et al., 2010; Cowan et al., 2012; Demarre et al., 2015; Miller et al., 2016; Raju et al., 2015; Slowikowski & Funk, 2010; Tescher et al., 2012; Corniello et al., 2014; Smit et al., 2016; Bredesen et al., 2015; Sardo et al., 2016; Connor et al., 2010; Coleman et al., 2013; Fred et al., 2012; Shaw et al., 2014
Episode of care	Admission/primary diagnosis	<ul style="list-style-type: none"> - Admitting diagnosis for hospital - Admitting diagnosis for ICU - Categorised as (1) rehabilitation or (2) other admission - Categorised by body system or disease process (e.g. infection, cardiovascular, trauma/pain, respiratory, neurological) 	5/5	- Mixed findings	Scheel-Sailer et al., 2013; Shen et al., 2015; Cox & Roche, 2015 Miller et al., 2016; Chen et al., 2015; Demarre et al., 2015
Episode of care	Medication	<ul style="list-style-type: none"> - Sedatives - Steroids - Other medications 	5/9	<ul style="list-style-type: none"> - Patients receiving steroids are at greater risk for PI - Patients receiving sedatives at no greater risk for PI (exception of 1 study) - Other medications (e.g. calcium channel blockers, nitrates) not associated with PI development 	Corniello et al., 2014; Kaitani et al., 2010; Man & Au-Yeung, 2013; O'Brien et al., 2014; Rao et al., 2016; Shen et al., 2015; Slowikowski & Funk, 2010

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TABLE 2 (Continued)

Construct	Concept	Indicators and types of variable measurement within the studies	# studies finding significance/# studies with variable	Type of relationship (summary)	References
Episode of care	Length of stay	<ul style="list-style-type: none"> - Hospital LOS - ICU LOS - LOS prior to PI development 	21/23	<ul style="list-style-type: none"> - Higher LOS in the hospital and/or ICU increases the risk for PI development 	<p>Cornielo et al., 2014; Cox & Roche, 2015; Cowan et al., 2012; Man & Au-Yeung, 2013; Tsaousti et al., 2014; Cox, 2011; Cremasco et al., 2013; Rao et al., 2016; Raju et al., 2015; Brito et al., 2013; Nassaji et al., 2014; Skogestad et al., 2017; Bredesen et al., 2015; Thomas et al., 2015; Sardo et al., 2016; Miller et al., 2016; Bly et al., 2016; Delmore et al., 2015; Baldi et al., 2010; Aydin et al., 2015; Hyun et al., 2014; Smit et al., 2016; Manzano et al., 2010</p>
Episode of care	Nursing interventions	<ul style="list-style-type: none"> - Overall measure of PI prevention protocol implementation - Restraint use or in bed, chair or adequate - Specific interventions/protocols implemented at the time of the study 	2/5	<ul style="list-style-type: none"> - Prevention measures play a significant role, although direction not clearly delineated - Use of restraints increases risk of PI development 	<p>Manzano et al., 2010; Bosch et al., 2011; Baldi et al., 2010; Man & Au-Yeung, 2013; Aydin et al., 2015</p>
Episode of care	Skin status	<ul style="list-style-type: none"> - General skin status (e.g. dry skin, abnormal appearances) - Presence of nonblanchable erythema - Skin condition healthy (yes/no) - Skin (NNN labels) 	7/8	<ul style="list-style-type: none"> - Poor general skin status associated with increased risk for PI development - Presence of nonblanchable erythema increased risk for PI development 	<p>Baumgarten et al., 2006; Coleman et al., 2013; Demarre et al., 2015; Rao et al., 2016; Schoonhoven et al., 2006; Stifter et al., 2015; Webster et al., 2015; Yoshimura et al., 2015</p>
Episode of care	Severity of illness	<ul style="list-style-type: none"> - ASA score - APACHE - Charleston comorbidity index (CCI) - Present of CRRT or haemodialysis - RAAS score - SOFA score - Simplified Acute Physiology Score (SAPS II) 	9/13	<ul style="list-style-type: none"> - ASA classes 4 and 5 at greater risk for PI - Mixed findings for CRRT - Higher APACHE score increase risk for PI development - Lower RAAS score increase risk for PI - Mixed findings for CCI 	<p>Bly et al., 2016; Cremasco et al., 2013; Coleman et al., 2013; Cox, 2011; Fred et al., 2012; Gardiner et al., 2016; Kaitani et al., 2010; Man & Au-Yeung, 2013; Manzano et al., 2010; O'Brien et al., 2014; Rao et al., 2016; Slowikowski & Funk, 2010; Yoshimura et al., 2015</p>
Episode of care	Surgical procedure: or time (episode of care)	<ul style="list-style-type: none"> - Total case length - Total time in the operating room - OR time categorised (less than/greater than four to six hours) 	8/12	<ul style="list-style-type: none"> - Greater time in the operating room results in increased risk for PI development 	<p>Bly et al., 2016; Chen et al., 2015; Cowan et al., 2012; Hayes et al., 2015; O'Brien et al., 2014; Rao et al., 2016; Shahin et al., 2010; Shen et al., 2015; Slowikowski & Funk, 2010; Shaw et al., 2014; Tschannen et al., 2012; Yoshimura et al., 2015</p>

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TABLE 2 (Continued)

Construct	Concept	Indicators and types of variable measurement within the studies	# studies finding significance/# studies with variable	Type of relationship (summary)	References
Episode of care	Surgical procedure: anaesthesia (episode of care)	<ul style="list-style-type: none"> - Anaesthesia duration - Type of anaesthesia (general vs. other) 	3/4	<ul style="list-style-type: none"> - Longer duration of anaesthesia increased the risk for PI development - General anaesthesia type (as opposed to others) may increase risk for PI 	Connor et al., 2010; O'Brien et al., 2014; Shahin et al., 2010; Shaw et al., 2014
Episode of care	Surgical procedure: type (episode of care)	<ul style="list-style-type: none"> - Surgical procedure (yes/no) - Total number of surgical procedures - Specific types of procedures (e.g. cardiac, general, orthopaedics) - Surgical procedure at specified points in hospital stay (e.g. <5 days from admission, >5 days from admission) 	8/9	<ul style="list-style-type: none"> - Patients having surgery at greater risk for PIs - Higher numbers of surgeries increase the risk of PIs - Those with emergent surgeries (vs. scheduled) at greater risk for PI development - Type of surgery can increase risk for PIs 	Bly et al., 2016; Cowan et al., 2012; O'Brien et al., 2014; Rao et al., 2016; Schoonhoven et al., 2006; Shahin et al., 2010; Tescher et al., 2012; Tschannen et al., 2012; Shaw et al., 2014
Episode of care	Surgical procedure: nursing interventions (episode of care)	<ul style="list-style-type: none"> - Use of polyurethane film (yes/no) - Total number of nursing interventions completed - Use of surgical warming unit (yes/no) 	2/4	<ul style="list-style-type: none"> - Use of polyurethane film or surgical warmer not associated with PI development - More nursing interventions (although not clearly defined) increased risk of PI development 	Fred et al., 2012; Shahin et al., 2010; Yoshimura et al., 2015; Shaw et al., 2014
Episode of care	Surgical procedure: perfusion (episode of care)	<ul style="list-style-type: none"> - Time on the cardiopulmonary bypass - Amount of intraoperative bleeding - Hypotension (e.g. DBP < 60mmHg and total time with low BP) 	2/4	<ul style="list-style-type: none"> - Increased intraoperative bleeding increases risk for PI development - Mixed findings of impact of bypass on PI development 	Fred et al., 2012; Shahin et al., 2010; Yoshimura et al., 2015; Shaw et al., 2014
Episode of care	Surgical procedure: position (episode of care)	<ul style="list-style-type: none"> - Use of rotation (yes/no) - Type of position (e.g. supine, prone, lithotomy, lateral, others) - Type of position (supine vs. non-supine) - Pillow under knee during surgery 	2/6	<ul style="list-style-type: none"> - Two studies found position (non-supine) to increase risk for PI development 	Webster et al., 2015; Shahin et al., 2010; O'Brien et al., 2014; Yoshimura et al., 2015; Connor et al., 2010; Shaw et al., 2014

*Column 4 represents the number of studies that found a significant relationship between the variable and PI development, which is subsequently divided by the total number of studies evaluating the variable.

TABLE 3 Primary constructs and associated evidence evaluating prediction of concepts for hospital-acquired pressure injuries

Construct	# studies	References
Patient	49	Aljezawi et al., 2014; Aydin et al., 2015; Baldi et al., 2010; Baumgarten et al., 2006; Bly et al., 2016; Bredesen et al., 2015; Brito et al., 2013; Chen et al., 2015; Coleman et al., 2013; Connor et al., 2010; Corniello et al., 2014; Cowan et al., 2012; Cox, 2011; Cox & Roche, 2015; Cremasco et al., 2013; Delmore et al., 2015; Demarre et al., 2015; Fred et al., 2012; Gardiner et al., 2016; Hyun et al., 2014; Hayes et al., 2015; Kaitani et al., 2010; Liu et al., 2012; Man & Au-Yeung, 2013; Manzano et al., 2010; Miller et al., 2016; Nassaji et al., 2014; O'Brien et al., 2014; Raff et al., 2016; Rao et al., 2016; Raju et al., 2015; Shen et al., 2015; Stifter et al., 2015; Sardo et al., 2016; Saunders et al., 2012; Scheel-Sailer et al., 2013; Schoonhoven et al., 2006; Serra et al., 2014; Shahin et al., 2010; Shaw et al., 2014; Skogestad, et al., 2017; Slowikowski & Funk, 2010; Smit et al., 2016; Tescher et al., 2012; Thomas et al., 2015; Tsaousi et al., 2014; Tschannen et al., 2012; Webster et al., 2015; Yoshimura et al., 2015;
Pressure	24	Aljezawi et al., 2014; Baumgarten et al., 2006; Bly et al., 2016; Brito et al., 2013; Coleman et al., 2013; Cowan et al., 2012; Cox, 2011; Delmore et al., 2015; Demarre et al., 2015; Kaitani et al., 2010; Lahmann & Kottner, 2011; Man & Au-Yeung, 2013; Michel et al., 2012; Raju et al., 2015; Rao et al., 2016; Schoonhoven et al., 2006; Slowikowski & Funk, 2010; Stifter et al., 2015; Tescher et al., 2012; Webster et al., 2015; Black et al., 2010; Tsaousi et al., 2014; Yoshimura et al., 2015; Skogestad et al., 2017
Shear	12	Aljezawi et al., 2014; Cowan et al., 2012; Cox, 2011; Demarre et al., 2015; Kaitani et al., 2010; Lahmann & Kottner, 2011; Raju et al., 2015; Rao et al., 2016; Schoonhoven et al., 2006; Shahin et al., 2010; Tescher et al., 2012; Shaw et al., 2014
Tissue tolerance	41	Aljezawi et al., 2014; Beeckman et al., 2014; Brito et al., 2013; Bly et al., 2016; Banks et al., 2010; Baumgarten et al., 2006; Chen et al., 2015; Coleman et al., 2013; Connor et al., 2010; Corniello et al., 2014; Cowan et al., 2012; Cox, 2011; Cox & Roche, 2015; Delmore et al., 2015; Demarre et al., 2015; Fred et al., 2012; Kaitani et al., 2010; Lahmann & Kottner, 2011; Manzano et al., 2010; Michel et al., 2012; Miller et al., 2016; Man & Au-Yeung, 2013; Nassaji et al., 2014; O'Brien et al., 2014;; Raff et al., 2016; Raju et al., 2015; Rao et al., 2016; Serra et al., 2014; Shahin et al., 2010; Stifter et al., 2015; Tescher et al., 2012; Skogestad et al., 2017; Smit et al., 2016; Shen et al., 2015; Shaw et al., 2014; Slowikowski & Funk, 2010; Tsaousi et al., 2014; Schoonhoven et al., 2006; Tschannen et al., 2012; Webster et al., 2015; Yoshimura et al., 2015; Coleman et al., 2013
Environment	22	Aydin et al., 2015; Bredesen et al., 2015; Bosch et al., 2011; Brito et al., 2013; Corniello et al., 2014; Cremasco et al., 2013; Cho et al., 2016; Choi et al., 2014; Delmore et al., 2015; Baldi et al., 2010; Demarre et al., 2015; He et al., 2013; Gardiner et al., 2016; Manzano et al., 2010; Ma & Park, 2015; Manojlovich et al., 2010; Stifter et al., 2015; Rao et al., 2016; Sardo et al., 2016; Schoonhoven et al., 2006; Tescher et al., 2012; Thomas et al., 2015

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TABLE 3 (Continued)

Construct	# studies	References
Episode of care	47	Aydin et al., 2015; Baldi et al., 2010; Bosch et al., 2011; Baumgarten et al., 2006; Bly et al., 2016; Bredesen et al., 2015; Brito et al., 2013; Chen et al., 2015; Connor et al., 2010; Coleman et al., 2013; Connor et al., 2010; Corniello et al., 2014; Cox & Roche, 2015; Cox, 2011; Cremasco et al., 2013; Cowan et al., 2012; Demarre et al., 2015; Delmore et al., 2015; Fred et al., 2012; Gardiner et al., 2016; Hayes et al., 2015; Hyun et al., 2014; Kaitani et al., 2010; Man & Au-Yeung, 2013; Manzano et al., 2010; Miller et al., 2016; Nassaji et al., 2014; O'Brien et al., 2014; Raff et al., 2016; Rao et al., 2016; Shahin et al., 2010; Raju et al., 2015; Sardo et al., 2016; Slowikowski & Funk, 2010; Smit et al., 2016; Shaw et al., 2014; Scheel-Sailer et al., 2013; Shen et al., 2015; Schoonhoven et al., 2006; Stifter et al., 2015; Skogestad et al., 2017; Thomas et al., 2015; Tescher et al., 2012; Tsaousi et al., 2014; Tschannen et al., 2012; Webster et al., 2015; Yoshimura et al., 2015

Panel (NPUAP), shear involves two adjoining internal body parts (e.g. bone, muscle, fat) that distort in the horizontal plane of the body (NPUAP, n.d.). Measurement of shearing force and its relationship to PI development has not been fully established. Friction, which includes the rubbing of a body part with another or against another material element, is commonly included when considering the impact of shear on PI (Antokal et al., 2012). Only twelve studies considered concepts associated with friction and shear (Table 3). Despite limited measurement, studies consistently identified both friction and shear as having a significant positive relationship with risk for HAPI (66%, $n = 8$ of 12) (Table 2).

5.2.4 | Other factors

Findings from this literature review include several additional factors that were not aligned with constructs or focal concepts in the existing PI models. These factors were primarily associated with patient demographics ($n = 49$), environmental context ($n = 22$ studies) or episode of care ($n = 47$ studies) (Table 3). Although several patient factors were evaluated as predictors of HAPIs including ethnicity, gender, socio-economic status, height, level of consciousness and marital status, there was little support for these aspects. For example, there was only limited evidence for the predictive ability of ethnicity (38%, $n = 3$ of 8) and no relationship among the studies evaluating height and HAPI development ($n = 0$ of 2) (Table 2). Although marital status and socio-economic status were deemed significant predictors of HAPI development, the generalisability of these findings is limited as each factor was only evaluated in a single study, respectively (Corniello et al., 2014; Saunders, Krause, & Acuna, 2012). Cognitive impairment, as measured by level of consciousness, presence of dementia or standardised documentation related to mental status which included various mental status scales, was believed to be a predictor in 1 of 3 studies (33%) reviewed (Coleman et al., 2013; Cowan, Stechmiller, Rowe, & Kairalla, 2012; Stifter et al., 2015). Although the other demographic concepts

did not substantially add predictive value, studies involving environmental context and episode-of-care-related concepts show encouraging results.

Environmental context factors evaluated for HAPI risk included hospital environment, census, work environment, unit type, nurse staffing and nursing workload. Hospital environment, measured as bed size, hospital setting (rural/urban), hospital type (public/private), Magnet designation and teaching status, was typically predictive of HAPI development (66%, $n = 4$ of 6). Specific findings revealed an increased risk of HAPI development for patients admitted to larger, private hospitals. There was no relationship found between HAPI development and hospital setting or Magnet designation (e.g. recognition obtained for excellence in nursing care). Census, or bed turnover, was recognised as a significant predictor in the only study evaluating the relationship. Specifically, higher bed turnover was associated with fewer HAPIs, which may be related to patient length of stay. Work environment, measured by the unit culture or team climate, was a significant predictor in two of the three studies reviewed (66%). Unit type was a strong predictor of HAPI ($n = 12$ of 13). Specifically, patients with an ICU stay or admitted to a surgical unit were at greater risk for HAPI. Nurse staffing, measured as hours per patient day (HPPD), skill mix and expertise, were also significant predictors of HAPI development (71%, $n = 5$ of 7). Higher HPPD was negatively associated with HAPI, while higher skill mix was associated with a lower risk for HAPIs. Nursing workload was measured in one study, reporting more nursing activity resulted in a reduced risk for HAPI.

Factors directly related to the hospital episode of care were also identified in the literature review, including admission source/type, admission diagnosis, patient severity of illness, surgical experience and length of stay. Of the studies evaluating the link between admission type (e.g. scheduled, nonscheduled or transferred) and source (e.g. home, other), four (66%, $n = 6$) found a significant relationship. Specifically, patients admitted emergently or transferred to the hospital were at greater risk for HAPI, whereas patients who resided at home prior to admission were at

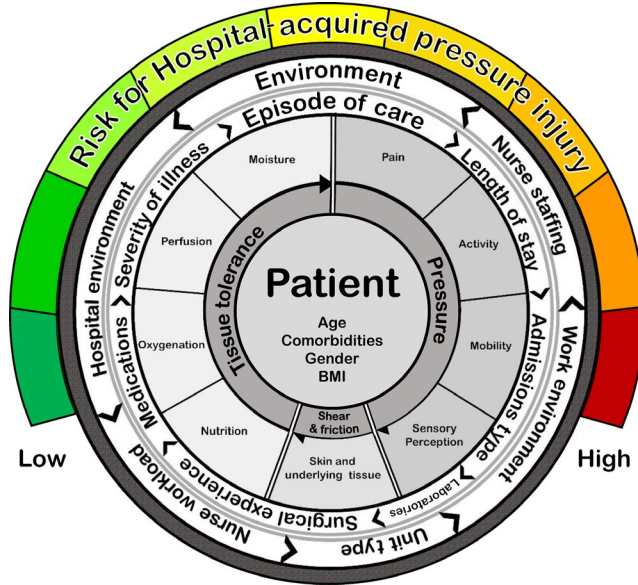


FIGURE 2 Pressure Injury Predictive Model [Colour figure can be viewed at wileyonlinelibrary.com]

lower risk for HAPI. Admission diagnosis was a significant predictor in all of the studies reviewed (100%, $n = 5$), but the sampling frame within each respective study limits generalisability and interpretation. Patient severity of illness, which has been measured by the American Society of Anesthesiologists (ASA) score, Acute Physiologic Assessment and Chronic Health Evaluation (APACHE), Charlson comorbidity index and Sequential Organ Failure Assessment (SOFA), was found to have a significant association with HAPI (69%, $n = 9$ of 13). Higher APACHE scores and ASA classes 4 and 5 were linked with increased risk, whereas there was a negative association between the Richmond Agitation–Sedation Scale (RAAS) scores and HAPIs. For patients undergoing surgery, factors associated with the operative experience (e.g. number or type of surgery, time on the operating table, type of anaesthesia, operative position, perfusion during the operation) potentially increased risk for HAPI development. Patients with multiple or emergent surgeries were at greater risk for HAPI (89%, $n = 8$ of 9), although the type of surgery and its relevant impact on HAPI development showed mixed results. Longer operative time resulted in an increased risk for HAPI (66%, $n = 8$ of 12), as did patients receiving general anaesthesia or having a longer duration of anaesthesia (75%, $n = 3$ of 4). For patients with increased intraoperative bleeding or hypotensive episodes, greater risk for HAPI was shown (50%, $n = 2$ of 4). A final episode-of-care factor, patient length of stay, is still at the forefront for HAPI risk, substantiated by several reviewed studies. Length of stay, measured as total LOS and/or ICU LOS, was predictive of HAPI development in 90% of the studies reviewed ($n = 21$ of 23). Higher LOS in the hospital and/or ICU increased the risk of HAPI development. Sardo et al. (2016) found patients with a LOS greater than 20 days were at a 7.5 times greater risk for HAPI development.

5.2.5 | Summary

There is empirical evidence to support the inclusion of many of the focal concepts represented in the existing conceptual models used for predicting PI development. Pressure and tissue tolerance—as measured through various concepts and subconcepts—continue to be supported as predictors of PI development. Factors associated with increased pressure for longer durations of time (e.g. immobility, pain, devices) result in greater risk for PI. Factors negatively affecting tissue tolerance, including poor oxygenation, limited perfusion or excessive moisture, also contribute to PI risk. Shear, although less studied, similarly has evidence to support its relationship to PIs. There is also empirical evidence that new constructs not represented in current models are strong predictors for PI development among hospitalised patients, including additional patient demographics, environmental characteristics and episode-of-care factors. Although the discovery of this is not surprising, a model fully representing the complexity and dynamic nature of risk remains nonexistent. A complete understanding of the risk for HAPIs requires that these additional concepts be explored and integrated into PI risk models and assessments.

5.3 | Step 3: Organising concepts and statements into an integrated and efficient representation of the phenomena of interest

The newly developed PIPM integrates previous models for PI risk with the current evidence associated with risk for HAPI (Figure 2). During the classification phase of this step, six primary constructs emerged. Pressure, tissue tolerance and shear and friction align with earlier models. New constructs include patient, environment and episode of care. Revolving concentric circles depict the dynamic and multilevel nature of patient, episodic and environmental factors within the hospital stay that could change the level of HAPI risk for a patient. Arrows within the circles are meant to depict movement not direction, recognising the fluctuations that can occur for many of the constructs and concepts either for the better (e.g. perfusion measures are stable, adequate blood pressure) or worse (e.g. perfusion measures failing, extremely low blood pressure). Various combinations of concepts (within the constructs) can place a patient at greater or lower risk.

5.3.1 | Pressure

Pressure remains a primary construct in predicting risk for HAPI. The PIPM, in contrast to previous models, recognises the dynamic nature of the concepts associated with pressure, including sensory perception, activity, mobility and pain. The PIPM depicts the dynamic nature of these factors via the rotating mechanisms within the concentric circles. For example, mobility and activity levels can vary throughout a patient's hospital stay (e.g. patient fully mobile upon admission can become immobile postsurgery). Furthermore,

interactions among the concepts may also impact risk. Patients with high levels of pain may become immobile and have reduced sensory perception, placing a patient at greater risk for HAPI development. To predict risk of HAPI, changes in these dynamic factors must be considered throughout the patient's stay.

5.3.2 | Tissue tolerance

The construct of tissue tolerance has been depicted in previous models identifying risk for PI development. Concepts such as perfusion, oxygenation, nutrition and moisture have significant impact on HAPI risk, as noted in the evidence. Similar to the construct of pressure, tissue tolerance is represented in the PIPM as a dynamic concept that fluctuates throughout the patient stay. Studies to date have simply looked at factors predictive of PI as fixed during an episode of care. Thus, patients who were mechanically ventilated for one day would have the same "risk" as a patient who had been ventilated for 21 days. Yet, the level of risk for PI may actually be exponentially higher for the patient with prolonged mechanical ventilation. The PIPM reflects this dynamically, as illustrated by the revolving arrows and the interaction effects among the various constructs and concepts. Even within the construct of tissue tolerance, interaction effects are considered, such as in the case of a patient who develops perfusion issues (e.g. haemodynamic instability) which can subsequently influence oxygenation levels. By considering variance in the concepts, a more accurate prediction of HAPI development can be determined.

5.3.3 | Shear and friction

Although the relationship between shear and PI development has varied among models, the limited number of studies continues to support the significant role shear plays on predicting PI. For shear to occur, friction must also be considered to accurately determine risk for HAPI; thus, both constructs are included in the PIPM. The construct of shear/friction is impacted by pressure and tissue tolerance, as depicted in the PIPM by the revolving arrows around the concepts. Patients with skin and/or underlying tissue issues may develop a HAPI with lower levels of pressure or due to low tissue tolerance as compared to someone with healthy skin. Furthermore, interactions among the various concepts can increase risk for HAPI (e.g. underlying skin issues in addition to poor nutrition or underlying disease such as diabetes mellitus).

5.3.4 | Patient

Patient characteristics are relatively stable concepts that play a significant role in PI development. For this reason, the patient construct is represented as central to other risk factors for HAPI. Concepts within the patient construct, including age, gender, BMI and the presence of various comorbidities, have resulted in increased risk for HAPIs. The patient construct directly affects pressure, tissue tolerance, shear/

friction, environment and episodic factors, which subsequently affect risk for HAPI. For example, patients with a history of cardiac disease already have impaired perfusion, which can become exacerbated during hospitalisation, especially if undergoing surgery, for example.

5.3.5 | Episode of care

A significant enhancement to previous PI models accounts for the complex nature of care processes and patient response to care during a hospitalisation. The episode-of-care concentric circle depicts the factors within a hospitalisation that can drastically modify risk for HAPI. Episodic factors include both fixed (e.g. admission type) and dynamic factors (e.g. surgical experiences, patient illness severity, medications, length of stay) associated with the stay. As patients undergo a surgical procedure, for example, changes in many of the concepts—at least for a time—may be evident, including reduced mobility/activity due to anaesthesia and procedure time, poor nutrition due to NPO status, or pain associated with the surgical procedure. These changes can result in increased risk for HAPI and thus may require further nursing interventions aimed at prevention.

5.3.6 | Environment

A further enhancement from current models is the inclusion of environment. Empirical evidence strongly supports the impact of nursing workload, unit type, nurse staffing levels and hospital environment on the development of HAPIs. The new model recognises the complexity associated with care delivery in the hospital setting, such that changes in the environmental characteristics can impact PI development. Changes in some of the concepts—nurse staffing, unit type and workload—can occur frequently throughout a patient stay. According to the PIPM, these factors must be evaluated throughout the hospital stay to determine their impact on HAPI development.

In summary, the PIPM represents the dynamic nature of PI risk for a hospitalised patient. A multitude of combinations of the constructs and concepts throughout the model can drastically increase or decrease risk to a hospitalised patient. This is the first PI risk model to represent the environment and episode-of-care factors associated with HAPI development. Inadequate staffing levels or high workload (environment) combined with an immobile patient (pressure) with poor nutrition (tissue tolerance) recovering from a 10-hr surgery (episode of care) would be at significant risk for HAPI. When the interaction effects of the various combinations of concepts are considered as denoted in the PIPM, those at risk for HAPI can more accurately be determined.

6 | RELEVANCE TO CLINICAL PRACTICE

Studies have identified interventions to reduce PI risk and evidence-based treatment plans for those who develop PIs (Berlowitz et al.,

2011; Institute for Healthcare Improvement, 2011). Accurate risk identification is the first step in prevention. Current models fail to consider the magnitude of factors predictive of PIs that have been empirically identified over the past several years. Thus, allocation of evidence-based PI prevention care to the *right person* at the *right time* remains elusive. This discursive paper reports on the use of Walker and Avant's (2005) theory synthesis model to examine the accuracy of PI frameworks—as it aligns with the evidence—in predicting risk for hospital-acquired PIs. Through a synthesis of current conceptual frameworks and relevant literature, a new model for PI risk emerged.

The PIPM is the first conceptual model representing the complexity and dynamic nature of HAPI risk for hospitalised patients, thus improving the accuracy of HAPI risk identification. Recognition of environment and episode-of-care-related factors affecting HAPI risk, in addition to the consideration for continuous variation in other factors, significantly improves upon current models. Although some environmental and episodic factors cannot be modified, recognition of the role these factors play in risk is important. For example, organisational leadership may want to consider specialty beds for all ICU patients or those of elder years with multiple comorbidities.

Optimisation of prevention among hospitalised patients requires early identification of risk that is accurate and precise, followed by aggressive preventive interventions. The PIPM reflects the synthesis of conceptual frameworks with current evidence, resulting in an all-encompassing depiction of PI risk. Use of the model may assist in a more accurate identification of risk among hospitalised patients. Rather than relying on a risk assessment completed once a shift, data from the medical record can be reviewed more consistently (e.g. every 2–4 hr), identifying combinations of factors that may put a patient at risk. For example, the combination of factors for Patient A may include patient (diabetic, older people, history of hypertension), tissue tolerance (mechanically ventilated, hypotensive), episode-of-care (undergoing extensive surgical procedure, currently receiving vasopressors) and environmental factors (admitted to the ICU) that signify high levels of risk for PI. Another example may be a patient with obesity and diabetes (patient factors), mobility limitations (pressure factors) and the need for supplemental oxygen (tissue tolerance factors), who was an emergent admission resulting in a long length of stay (episode-of-care factors) where low staffing (environment) was identified. Clinicians could use such information to guide their decision-making related to priority interventions.

Understanding the various combinations of factors can aid in the development of risk profiles which can then be aligned with individual intervention. For example, a patient with a risk profile including multiple chronic conditions (e.g. diabetes mellitus, cardiovascular disease) with a recent hypotensive event scheduled for an extensive surgery may need alternative support surfaces or pressure redistribution overlays (higher cost) or more frequent pressure relief during a surgical procedure (AORN Guidelines for Perioperative Practice, 2019), whereas a patient risk profile including normal BMI, haemodynamically sound vitals and no underlying

conditions undergoing the same procedure may only require the standard intraoperative mattress (lower cost) and repositioning protocol. More strategic deployment of critical resources and implementation of nursing interventions tailored to the risk profile may result not only in a reduction of HAPIs, but also improved cost efficiencies.

The new conceptual framework also has implications for future research. For example, empirical testing to assure relationships between the various concepts are adequately represented in the model is required. Interaction effects among the various concepts must be evaluated, which may result in the development of a more accurate, robust model of risk factors, including recognition of variables most predictive of PIs. The analysis of large-scale data sets from electronic health records is one method of achieving this aim. The PIPM is a first step towards representing pressure injury dynamics. Extending the model using complex system methods such as agent-based modelling may further our understanding of the mechanisms involved in the development of pressure injuries over time, leading to improved understanding of risk and subsequent clinical care for hospitalised patients.

ACKNOWLEDGEMENTS

We wish to express our gratitude to Shannon Coon for her significant contribution in editing and formatting the model.

CONFLICT OF INTEREST

The authors have no conflicts of interest to report.

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How to cite this article: Tschannen D, Anderson C. The pressure injury predictive model: A framework for hospital-acquired pressure injuries. *J Clin Nurs*. 2020;29:1398–1421. <https://doi.org/10.1111/jocn.15171>