Pressure Injuries in Critical Care Patients: A Conceptual Schema

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GENERAL PURPOSE: To outline a conceptual schema describing the relationships among the empirically supported risk factors, the etiologic factors, and the mitigating measures that influence pressure injury (PI) development in the critical care population.
TARGET AUDIENCE: This continuing education activity is intended for physicians, physician assistants, nurse practitioners, and nurses with an interest in skin and wound care.
LEARNING OBJECTIVES/OUTCOMES: After participating in this educational activity, the participant will:
1. Choose a static intrinsic factor that increases the risk for the development of PI.
2. List several dynamic intrinsic risk factors for developing a PI.
3. Identify dynamic extrinsic risk factors that may predispose a patient to developing a PI.
4. Explain the pathophysiology of PI development.

ABSTRACT

BACKGROUND: The first step in successful pressure injury (PI) prevention is to determine appropriate risk factors. In patients who are critically ill, PI risk is multietiologic, including the pathophysiologic impacts associated with a critical illness, concomitant preexisting comorbid conditions, and treatment-related factors that are essential in the ongoing management of a critical illness.
OBJECTIVE: To outline a conceptual schema describing the relationships among the empirically supported risk factors, the etiologic factors, and the mitigating measures that influence PI development in the critical care population.
METHODS: Risk factors for PI included in the conceptual schema were identified after a comprehensive review of the literature. Risk factors were categorized as static intrinsic factors, dynamic intrinsic factors, or dynamic extrinsic factors.
RESULTS: The schema illustrates the complex relationships between risk factor duration and intensity and the underlying etiology of PI development. The relationships among cumulative risk factors, etiologic factors, and mitigating measures for PI prevention are also outlined in the schema within the context of potentially unavoidable PI development.
CONCLUSION: Examining PI development in patients who are critically ill through the lens of a conceptual schema may guide future research endeavors focusing on the etiologic bases for PI development. It may also provide a framework to explore alternatives to current formal PI risk assessment in this unique subset of hospitalized patients.
KEYWORDS: conceptual schema, critical care, etiology, hospitalized patients, intensive care, pressure injury, risk factors, wound healing
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INTRODUCTION

Intensive care units provide care to the sickest patients in our healthcare system. Even with an overall increase in the severity of illness among patients who are critically ill, ICU survival has improved, which can be attributed to advances in medical technology and enhanced knowledge and expertise in the treatment of critical illness.[1] However, survival may be fraught with unintended consequences such as the development of a pressure injury (PI). Intensive care units report the highest PI rates among hospitalized populations, with rates varying between 12% and 25% globally.[2] Although overall hospital-acquired PI rates have declined in the past decade, severe PIs (stage 3, stage 4, unstageable, and deep-tissue PIs), especially among ICU patients, have not followed this trend.[3] In fact, a 24.6% increase in more severe hospital-acquired PIs (stage 3, stage 4, unstageable) was reported nationally in 2015 and 2016.[4] Accordingly, healthcare experts want to know why these PIs continue to occur despite evidence-based PI prevention programs, especially in patients who are critically ill.

The first step in successful PI prevention is to determine the relevant risk factors. In critically ill populations, PI risk is multietiologic and rooted in many sources. These include the pathophysiologic impacts associated with a critical illness, concomitant preexisting comorbid conditions, and treatment-related factors. In recent systematic reviews of the literature in this population, significant predictors of PI included age,[5–7] impaired mobility/activity,[6,7] vasopressor infusion,[5–7] prolonged ICU admission,[5] comorbid conditions (eg, diabetes mellitus),[5,7] cardiovascular disease,[5] hypotension,[5–7] prolonged mechanical ventilation,[5–7] hemodialysis,[7] and sedation.[7]

Understanding the interactions among these risk factors within the context of the known etiologic underpinnings for PI is important and will enhance the determination of avoidable versus unavoidable PIs in this population. The purpose of this continuing education article is to outline a conceptual schema to describe the relationships between the empirically supported risk factors and the etiologic factors that influence PI development in the critical care population.

METHODS

Pressure injury risk factors included in the conceptual schema were derived from a comprehensive review of the critical care literature using the CINAHL and PUBMED databases and the following search terms: "pressure ulcer," "pressure injury," "critical care," "intensive care," and "risk factors," yielding 559 potential research reports.

The review inclusion criteria were (1) English-language, peer-reviewed quantitative studies with a focus on PI risk factors in adult ICU patients published between 2010 and 2020; and (2) multivariate analysis with PI development as the outcome variable. Studies in which the primary focus was on PI prevention, treatment, or on the use of PI risk assessment scales were excluded from review.

Pressure injury risk factors were categorized as static intrinsic risk factor, dynamic intrinsic risk factor, or dynamic extrinsic risk factor for the conceptual schema. Static intrinsic risk factors describe risk factors present at the time of admission to the ICU. Although these risk factors may potentiate PI risk, the impact of these risk factors remains constant during a critical illness. For example, the pathophysiologic effects of advancing age or underlying comorbidities such as diabetes mellitus, end-stage renal disease (ESRD), or cardiac disease will impact PI risk and cannot be eliminated during a critical illness.

Dynamic intrinsic risk factors are internal factors that arise as sequelae of critical illness. For example, conditions such as hypoxia, hypotension, and hemodynamic instability are all dynamic physiologic parameters that have the potential to worsen or be corrected through treatment or physiologic stabilization. The body's ability to adapt physiologically positively or negatively is a result of one's intrinsic homeostatic mechanisms.

Dynamic extrinsic factors are external or treatment factors either used to treat critical illness or that occur as a result of treatment modalities. These factors and their duration of impact are variable from patient to patient. For example, mechanical ventilation (an iatrogenic PI risk factor) is used to treat respiratory failure. Ventilator settings and the amount of time the patient requires mechanical ventilation are based on the patient's physiologic response. Similarly, treatments such as continuous venovenous hemodialysis, extracorporeal membrane oxygenation and sedation, or the use of neuromuscular-blocking agents to induce chemical paralysis create clinical circumstances that can lead to impaired mobility. These extrinsic reasons for immobility are therefore different from impaired mobility at baseline.

RESULTS

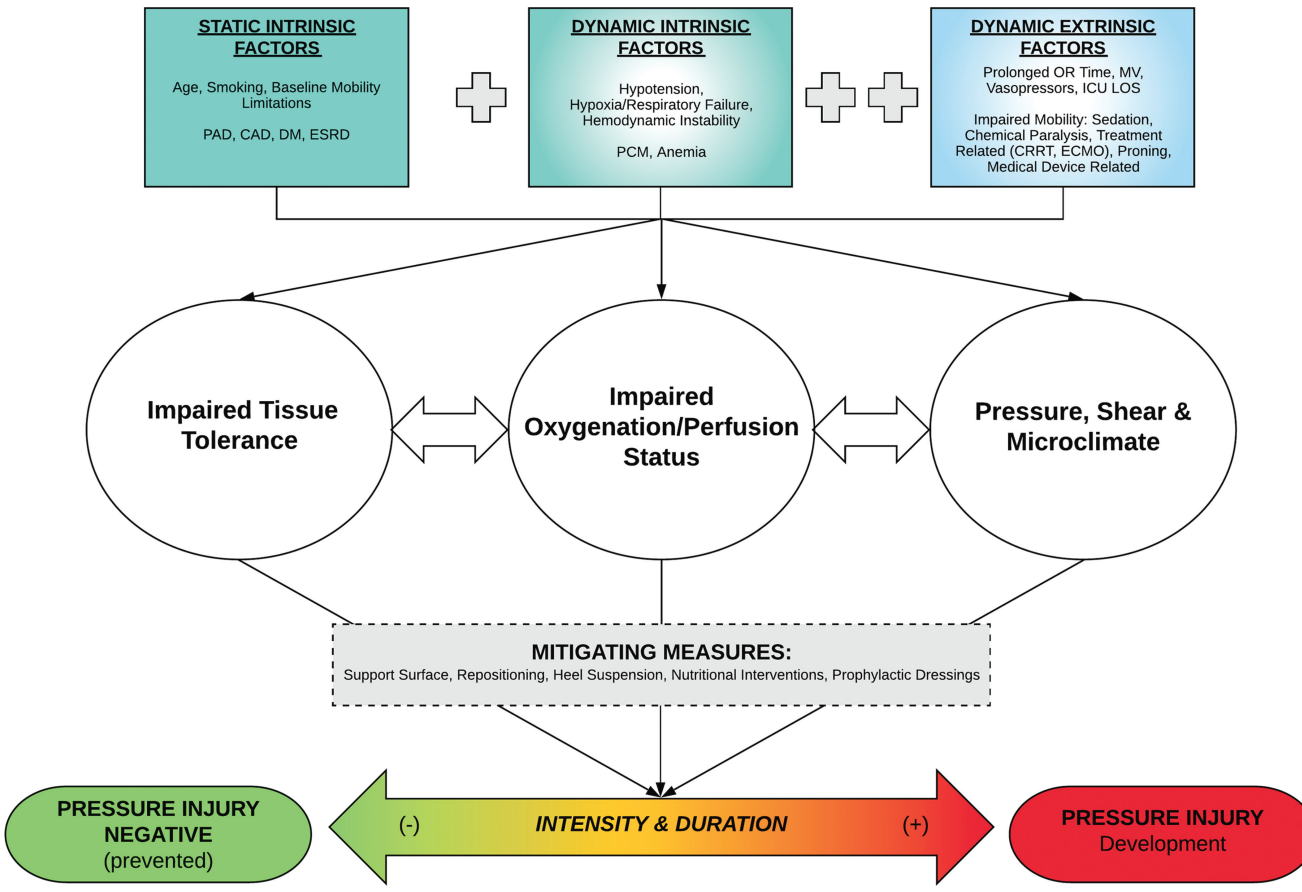
After applying the inclusion/exclusion criteria, 54 reports were examined for possible inclusion, and 28 studies were included in the final review (Supplemental Table, http://links.lww.com/NSW/A55).

Static Intrinsic Factors

The conceptual schema (Figure) identifies the following conditions as static intrinsic risk factors in patients who are critically ill: age, baseline impaired mobility, history of smoking, peripheral arterial disease, coronary artery disease, diabetes mellitus, and ESRD.

Age is the most common risk factor that emerges in multivariate analysis; 12 studies report age as a significant predictor.[8–19] Factors such as frailty, disability, and

Figure. CONCEPTUAL SCHEMA FOR PRESSURE INJURY DEVELOPMENT IN CRITICALLY ILL PATIENTS



+, increased risk; ++, cumulative increased risk.
Abbreviations: CAD, coronary artery disease; CRRT, continuous renal replacement therapy; DM, diabetes mellitus; ECMO, extracorporeal membrane oxygenation; ESRD, end-stage renal disease; LOS, length of stay; MV, mechanical ventilation; PAD, peripheral artery disease; PCM, protein-K calcium malnutrition.

multimorbidity are more prevalent with age and increase the risk of adverse outcomes.[20] The comorbidities of diabetes mellitus,[10,13,21,22] coronary artery disease,[11,14,21,23] ESRD,[23] peripheral arterial disease,[14,18] and smoking[10] have been empirically supported as PI risk factors. In recent clinical practice guidelines, diabetes mellitus has been highlighted with regard to PI risk (A-level evidence).[24] On closer examination of these comorbidities as well as smoking status, the underlying pathophysiologic precept is consistent with impaired oxygenation and perfusion, which impairs cellular function and leads to tissue ischemia.

Mobility impairments in critical illness may be attributable to preexisting limitations and can create a situation predating facility admission in which the patient requires complete assistance of caregivers to change position. In one study,[11] lower scores on the Braden mobility subscale at ICU admission predicted PI development.

Dynamic Intrinsic Risk Factors

Factors in the schema identified as dynamic intrinsic risk factors in this population include hypotension, hypoxia/respiratory failure, hemodynamic instability, protein-calorie malnutrition, and anemia. These factors have similarities in their underlying pathophysiologic precepts, that is, impaired oxygenation and perfusion. Hemodynamic instability is one of the primary clinical presentations of patients admitted to the ICU manifested by impairments in BP, heart rate, and oxygenation. Absolute hypotension is a systolic BP <90 mm Hg or mean arterial pressure (MAP) <65 mm Hg.[25] Nine studies reported hypotension as a significant predictor, but with varying definitions (systolic BP <90 mm Hg,[26,27] MAP <70 mm Hg,[28] abnormal MAP,[19] diastolic BP <60 mm Hg,[17] MAP <60 mm Hg on admission,[18] MAP <60 mm Hg on vasopressors,[29] lower diastolic BP [value not reported],[30] lower MAP [value not reported],[31] and hypotension [*International Classification of*

Diseases, Ninth Revision coding]).[14] These variations in the measurement of BP as a variable preclude one consistent measure to objectively define hypotension in relation to PI risk; however, collectively, it does illustrate the level of empirical support for this risk factor.

Although a definitive value to define oxygen impairment in relation to PI risk is also lacking, the need for mechanical ventilation as a modality can be considered a proxy. Respiratory failure requiring ventilator support is one of the top five most common admitting diagnoses reported in CCUs in the US.[32] Eight studies identified the use of mechanical ventilation as a significant predictor in this population, supporting hypoxia and respiratory failure as a significant threat to PI prevention.[12,15,17,18,22,29,31,33]

In patients who are critically ill, severe protein-calorie malnutrition is a common finding resulting from impaired intake and as a result of the hypercatabolic/hypermetabolic response to injury or severe illness.[34,35] In two studies,[14,21] protein-calorie malnutrition was found to be a significant predictor. Four studies[17,21,31,36] identified lower albumin levels as significant in multivariate analysis. However, albumin and prealbumin are not considered reliable indicators of protein stores and nutrition status during a critical illness as a result of fluid shifts and hydration status.[37,38]

Anasarca is a clinical finding related to protein-calorie malnutrition and hypoalbuminemia and manifests as generalized fluid distribution in the interstitial spaces leading to impaired nutrient delivery and waste removal from the tissues and skin that impacts perfusion, leading to impaired tissue tolerance.[39] Last, anemia was found to be a significant predictor in one study.[10] Anemia results in impaired oxygen-carrying capacity of the blood and contributes to tissue hypoxia, impaired tissue perfusion, and tissue tolerance.[40,41]

Dynamic Extrinsic Factors

Factors in the schema termed "dynamic extrinsic factors" include ICU length of stay (LOS), prolonged OR time, and treatment-related factors such as vasopressor administration and mechanical ventilation. Interventions that reduce mobility including sedation and neuromuscular-blocking agents causing chemical paralysis and renal replacement therapies are also considered dynamic extrinsic factors in the schema. Likewise, medical device-related PIs can occur as a result of a multitude of medical devices, and this may also be a dynamic extrinsic factor.

Researchers frequently identify ICU LOS in risk models as a dynamic extrinsic factor contributing to PI development.[8,9,11,14–17,27,41,42] Sustained oxygenation and perfusion deficits and efforts to treat these deficits can have a cumulative effect, with prolonged requirements of ICU levels of care reflecting sustained instability, immobility, and need for therapy. Length of OR time, similar to ICU LOS, may reflect prolonged exposure to dynamic intrinsic factors and impaired mobility over time. Longer time in surgery[30,43] and frequency of surgery[43] have both been identified as predictors of PI. Major procedures such as cardiovascular surgery increase risk of PI,[14,31] whereas in an analysis of intraoperative risk factors in ICU patients, researchers found that noncardiac surgery was predictive.[23]

Therapies such as mechanical ventilation,[12,15,17,18,22,29,31,33] vasopressor administration[26,27,29] (in particular norepinephrine[11,14] or vasopressin[29]), and renal replacement therapies[14,18,30] contribute to PI risk. This may be related to impaired mobility associated with the treatments, the severity of perfusion and oxygen deficits necessitating these therapies, or device-related injury. With regard to the impact of repositioning, Kaitani and colleagues[44] found that infrequent repositioning (measured as the mean daily number of turns) was predictive of PI, and Tayyib and colleagues[8] identified lower mean hours per day of patient repositioning was predictive.

Proning, used to improve oxygenation in patients with severe lung injury and acute respiratory distress syndrome,[45] is a therapy generally not examined in PI risk factor analysis heretofore. With the widespread adoption of proning practices in ICUs during the novel coronavirus 2019 pandemic, medical device-related PIs are an increasing concern and can be related to the artificial airway or pressure to the face, chest, and other dependent areas such as the knees and iliac crests.[46–49]

Other therapies for the critically ill population such as extracorporeal membrane oxygenation[50,51] or ventricular assist devices are likely additional dynamic extrinsic factors; however, most risk factor analyses have not considered these factors in their models.

Pathogenesis for Pressure Injury Development

The pathogenesis of PI centers on the relationships among various key precepts. These include the major load forces of pressure and shear, the influence of microclimate, and the ability of the tissues to tolerate pressure and/or shear. Associated systemic deficits in oxygenation and perfusion also play an important role in PI development. The interactions among these elements, influenced by the individual patient's static and dynamic risk factors, drive PI development.

Major Load Forces. The etiologic basis for PI development has been studied for decades.[52–54] Coleman and colleagues[55] and the 2019 International Guideline for Pressure Injury Prevention and Treatment[24] describe the complex interplay of the major mechanical forces of pressure and shear coupled with the individual's ability to tolerate these forces. The forces' intensity and duration impact the degree of internal stress and strain

transferred. Stress and strain cause damage to the internal cell structure and impede cellular function including perfusion, lymphatic drainage, and fluid transport. When cells are damaged irreparably, cell death results in an inflammatory response that increases permeability in the vessel, resulting in inflammatory edema and an increase in the mechanical loads as a result of rising pressure in the interstitium.[56,57] Tissue ischemia, as a result of sustained deformation from strain forces, leads to cellular hypoxia, decreased nutrient supply, and impaired removal of metabolic wastes. This induces an acidic environment that will lead to cell death and tissue destruction.[58]

Gefen[59] proposed that the interaction of tissue deformation, the inflammatory response, and tissue ischemia explains the damage that occurs at the cellular level and subsequent tissue damage. Direct deformation of the cell as a result of sustained tissue deformation ignites a cascade of pathophysiologic events that begins with the inflammatory response that leads to ischemic damage.[60] Pressure injury ensues when the rate of cell and tissue death exceeds the potential rate of cellular repair.

Microclimate. Microclimate is an emerging concept in the pathogenesis of PI development and comprises the temperature, humidity, and airflow next to the skin or between the skin and a support surface. Microclimate conditions affect the skin's response to mechanical forces and thus may potentiate PI development. When skin is warm and moist, it is weaker and more vulnerable to mechanical forces, and if skin is dry, it is more susceptible to fissure formation.[24] Likewise, moist skin from perspiration or humidity can influence tissue deformation as a result of friction and shear.[24,61] Although friction is no longer considered a mechanical force that contributes to PI development,[62] it is a force that when combined with changes in microclimate can impact both surface and internal tissue loads.[24]

Impaired Tissue Tolerance. Tissue tolerance describes the individual's unique tolerance to mechanical forces. Specific biomechanical tolerance of the tissues can be related to age, tissue morphology, and underlying health conditions, such as micro- and macrovasculature disease, as well tissue repair capacity.[24] The unique characteristics of the individual contribute to the magnitude of mechanical loads, the distribution of load in the tissues, duration of exposure to mechanical load, and the tolerance of the tissues to this load. Once either the individual's threshold for tissue tolerance or the internal stress produced from mechanical load has been exceeded, the patient is primed for PI.

Impaired Oxygenation and Perfusion. These are the most important pathophysiologic precepts to consider when determining PI risk in this population.[24] Hypoxia is the most common reason for cellular injury and results from many sources such as hemoglobin deficits, decreases in arterial oxygen levels, and respiratory and cardiovascular disease.[58] Hypoxia is also associated with inflammation and ischemia, with ischemia being the most common cause of hypoxia.[58] At the cellular level, hypoxia results in mitochondrial damage that decreases adenosine triphosphate production and leads to anaerobic metabolism and acidosis and loss of the sodium-potassium and calcium transport systems and (if not reversed quickly) cell death. Persistent ischemia is associated with irreversible injury and cellular necrosis.[58]

In the pathogenesis of PI evolution, perfusion, oxygenation, and circulation all affect the susceptibility and tissue tolerance of the skin, impacting the reparative threshold of tissue as well as the transport of cellular nutrients that maintain cellular function. In critical illness, conditions that impact tissue oxygenation and perfusion are pervasive: risk factors such as advanced age, diabetes, hypotension, cardiovascular disease, mechanical ventilation, and vasopressor agents all impact tissue oxygenation and perfusion.

Prevention

Mitigating measures to halt PI development are germane to PI prevention and included in the conceptual schema. Over the past 30 years, evidence-based PI prevention guidelines have been established and incorporated into clinical practice. In 1992, the Agency for Healthcare Research and Quality published the first comprehensive national guideline.[63] Other resources and best practice initiatives from organizations including the Agency for Healthcare Research and Quality,[64] the Institute for Healthcare Improvement,[65] and the National Pressure Injury Advisory Panel[24] have also been made widely available to guide clinical practice.

The 2019 International Guideline published the most contemporaneous evidence for PI prevention and was developed as a collaborative effort among the National Pressure Injury Advisory Panel, the European Pressure Ulcer Advisory Panel, and the Pan Pacific Pressure Injury Alliance.[24] According to the guideline, the major elements of a comprehensive PI prevention program include risk assessment, skin and tissue assessment, repositioning to offload bony prominences, early mobilization, prophylactic dressing use, heel elevation, support surfaces, and nutrition assessment.

Although the consistent application of PI prevention strategies is important in PI mitigation, in most published studies in this population, PI prevention strategies were rarely considered in the multivariate analysis. Whereas formal PI risk assessment scores were reported in 19 of the studies reviewed, only four studies included PI prevention in multivariate analysis. In two studies,[8,44]

infrequent repositioning was predictive of PI development, whereas Bly and colleagues[26] reported that any more than 2 days' delay in patient placement on a support surface was significantly predictive of PI development. Conversely, Gonzalez-Mendez and colleagues[66] found the days of immobilization to be protective; that is, those patients immobilized the longest had decreased probability for PI development.

Unavoidable Pressure Injury. In terms of PI avoidance versus PI development, the consistent application of PI prevention strategies must be considered. The NPIAP defines unavoidable PI as one that develops despite the consistent and appropriate application of PI prevention strategies or if lifesaving modalities take precedence over PI prevention.[67] Although substantial evidence supports the efficacy of PI prevention programs in reducing PI,[68–73] no studies have found that the application of these strategies has eliminated all PIs, especially in critically ill patients.

An awareness that certain clinical scenarios may stretch beyond the compensatory abilities of skin and underlying tissues or beyond the preventive capacity of caregivers is important to the understanding of why PIs occur in this population despite best practice implementation. In this conceptual schema, the risk factors that have garnered the strongest empirical support are (potentially) nonmodifiable: respiratory instability, arterial insufficiency, vasopressor use, impaired tissue oxygenation, and cardiopulmonary dysfunction resulting from hemodynamic instability, heart failure, hypoxemia, and hypotension.[67,74] Further, static intrinsic factors such as age, diabetes mellitus, peripheral arterial disease, ESRD, and cardiac disease cannot be reversed and can potentiate and complicate a critical illness. Dynamic intrinsic conditions such as hypotension and hypoxia, depending on the underlying cause, may not be quickly reversed and may further exacerbate perfusion and oxygenation derangements. Finally, dynamic extrinsic factors used to treat hypotension or hypoxia such as vasopressors or mechanical ventilation are lifesaving modalities that may need to be prioritized over standard PI prevention practices in those patients who are hemodynamically unstable.

DISCUSSION

Pressure injury risk factors in the conceptual schema can exert a cumulative effect on PI risk such that PI risk escalates when patients possess risk factors from each category. For example, although static intrinsic factors present on admission to the ICU can impact the patient's overall severity of illness, the level of hemodynamic instability can further exacerbate and accelerate PI risk. Dynamic extrinsic factors possess their own inherent dangers and in combination with both static and dynamic intrinsic factors create added risk. Essentially, the more risk factors the patient has combined with the associated treatment needed accelerate PI risk.

Impaired oxygenation and perfusion are at the core of many of the risk factors identified in the schema. These include the patient's preexisting comorbidities (static intrinsic factors) that affect the macro- and microvasculature and dynamic intrinsic factors that impose oxygenation and perfusion abnormalities and form the pathophysiologic basis for many critical illnesses. In addition, dynamic extrinsic factors used to improve and restore tissue oxygenation and perfusion such as with vasopressor administration can contribute to impaired tissue oxygenation and perfusion manifested by circulatory changes to the hands, feet, and splanchnic and renal systems.[75]

Impaired tissue tolerance is a major determinant of PI development and is influenced by the patient's unique response to mechanical loads and microclimate. Tissue tolerance can be further impacted by static and dynamic intrinsic factors including conditions that affect both the micro- and macrocirculation, as well as conditions such as severe protein-calorie malnutrition and anasarca.

The initiation and consistent application of mitigating measures to prevent PIs are the first line of defense. Providers may need to make alterations to certain elements of PI prevention programs (eg, repositioning schedules) in the critical care population as a result of potential iatrogenic factors that impair mobility or hemodynamic instability.

For patients who develop a PI, a comprehensive review is warranted, including the mitigating measures implemented based on the patient's risk factors (intrinsic/extrinsic) and response to treatments associated with critical illness. For example, in older patients with multiple comorbidities on admission, the adverse effects of a superimposed critical illness may be magnified. Hemodynamic instability may be insurmountable in some patients such that derangements in oxygenation and perfusion cannot be corrected despite the use of mechanical ventilation, vasopressor agents, or other supportive treatment modalities. In these cases, providers, patients, and families should consider that PI development may be unavoidable.

CONCLUSIONS

The purpose of this conceptual schema was to create a framework to explain the development of PIs in the critical care population. The interactions of static intrinsic factors, dynamic intrinsic factors, and dynamic extrinsic factors are complex and illustrate the multietiologic basis for PI development in this population. Impaired oxygenation and perfusion are underlying precepts implicated in many of these risk factors. The patient's innate ability to

overcome mechanical forces assisted by PI prevention strategies holds the potential to impede PI development. Conversely, the inability to overcome these forces as a result of comorbidities, illness severity, or the treatments used—even with consistent PI prevention practices—may result in the development of an unavoidable PI. The medical team needs to carefully consider the likelihood of an unavoidable PI within the context of the individual's clinical situation.

Examining PI development in the critical care population through the lens of this conceptual schema may be useful to guide future research endeavors focusing on the etiologic bases for PI development. It may also provide a framework to explore alternatives to current formal PI risk assessment in this unique subset of hospitalized patients.

PRACTICE PEARLS

- Pressure injury risk determination in patients who are critically ill is multifactorial but a necessary first step to successful PI prevention.
- A conceptual schema can provide the groundwork to explore the complex relationships among empirically related PI risk factors, underlying etiologic factors, and prevention efforts that contribute to PI development in patients who are critically ill.
- Pressure injury risk factors can be broadly categorized as static intrinsic factors, dynamic intrinsic factors, and dynamic extrinsic factors.
- A conceptual schema provides an empirically supported foundation to explore alternatives to current formal PI risk assessment in patients who are critically ill.
- Future research endeavors should focus on the etiologic bases for PI development and deepen understanding of unavoidable PI. ●

REFERENCES

1. Society for Critical Care Medicine. Critical care statistics. www.sccm.org/Communications/Critical-Care-Statistics. Last accessed December 15, 2020.
2. Chaboyer W, Thalib L, Harbeck E, et al. Incidence and prevalence of pressure injuries in adult intensive care patients: a systematic review. Crit Care Med 2018;46(11):e1074-81.
3. Kayser S, VanGilder K, Lachenbruch C. Predictors of superficial and severe hospital-acquired pressure injuries: a cross-sectional study using the International Pressure Ulcer Prevalence survey. Int J Nurs Stud 2019;89:46-52.
4. Padula W, Black J, Davidson P, Kang S, Pronovost P. Adverse effects of the Medicare PSI-90 hospital penalty system on revenue-neutral hospital-acquired conditions. J Patient Saf 2020;16(2): e97-e102.
5. Cox J. Pressure injury risk factors in adult critical care patients: a review of the literature. Ostomy Wound Manage 2017;63(11):30-43.
6. Alderden J, Rondinelli J. Risk factors for pressure injuries among critical care patients: a systematic review. Int J Nurs Stud 2017;71:97-114.
7. Lima-Serrano M, Gonzalez-Mendez MI, Carrasco-Cebollero FM, Lima Rodriguez JS. Risk factors for pressure ulcer development in intensive care units: a systematic review. Med Intesiva 2017;41(6): 339-46.
8. Tayyib N, Coyer F, Lewis P. Saudi Arabian adult intensive care unit pressure ulcer incidence and risk factors: a prospective cohort study. Int Wound J 2016;13(5):912-9.
9. Campinilli T, Conceicao de Gouveia Santos V, Strazzieri-Pulido K, Thomaz P, Nogueria P. Incidence of pressure ulcers in cardiopulmonary intensive care unit patients. Rev Esc Enferm USP 2015;49:7-13.
10. Nassaji M, Askari Z, Ghorbani R. Cigarette smoking and risk of pressure ulcers in adult intensive care unit patients. Int J Nurs Pract 2014;20:418-23.
11. Cox J. Predictors of pressure ulcers in adult critical care patients. Am J Crit Care 2011;20(5):364-74.
12. Manzano F, Navarro M, Roldan D, et al. Pressure ulcer incidence and risk factors in ventilated intensive care patients. J Crit Care 2010;25(3):469-76.
13. Slowikowski G, Funk M. Factors associated with pressure ulcers in patients in a surgical intensive care unit. J Wound Ostomy Cont Nurs 2010;37(6):619-26.
14. Cox J, Schallom M, Jung C. Risk factors for pressure injuries in adult critical care patients. Am J Crit Care 2020;29(3):204-13.
15. Strazzieri-Pulido KC, Gonzalez CVS, Nogueria PC, Padilha KG, Santos VLCG. Pressure injuries in critical patients: incidence, patient associated factors, and nursing workload. J Nurs Manage 2019; 27:301-10.
16. Ponchio Pacha H, Lemana Faria J, Aparecido de Oliveira K, et al. Pressure ulcers in intensive care units: a case control study. Rev Bras Enferm 2018;71(6):3027-34.
17. Deng X, Yu T, Hu A. Predicting the risk for hospital-acquired pressure ulcers in critical care patients. Crit Care Nurs 2017;37(4):e1-e11.
18. Ranzani O, Simpson E, Japiassu A, et al. The challenge of predicting pressure ulcers in critically ill patients: a multicenter cohort study. Ann Am Thorac Soc 2016;13(10):1775-83.
19. Soodmand M, Moghadamnia T, Aghaei I, et al. Effects of hemodynamic factors and oxygenation on the incidence of pressure ulcers in the ICU. Adv Skin Wound Care 2019;32:359-64.
20. Brummel N, Ferrante L. Integrating geriatric principles into critical care medicine: the time is now. Annals ATS 2018;25(5):518-21.
21. Serra R, Caroleo S, Buffone G, et al. Low serum albumin level as an independent risk factor for the onset of pressure ulcers in intensive care unit patients. Int Wound J 2012;11(5):550-3.
22. Hyun S, Moffat-Bruce S, Cooper C, et al. Prediction model for hospital-acquired pressure ulcer development: retrospective cohort study. JMIR Med Inform 2019;7(3):e13785.
23. O'Brien D, Shanks A, Talsma A, Brenner P, Ramachandran S. Intraoperative risk factors associated with postoperative pressure ulcers in critically ill patients: a retrospective observational study. Crit Care Med 2013;42(1):40-7.
24. National Pressure Injury Advisory Panel, European Pressure Ulcer Advisory Panel, Pan Pacific Pressure Injury Alliance. Prevention and Treatment of Pressure Injuries/Ulcers: Clinical Practice Guideline. Haesler Emily, ed. Cambridge Media: Osborne Park, Western Australia; 2019.
25. Galeski D, Mikkelsen M. Evaluation of and initial approach to the adult patient with undifferentiated hypotension and shock. 2020. www.uptodate.com/contents/evaluation-of-and-initial-approach-to-the-adult-patient-with-undifferentiated-hypotension-and-shock. Last accessed December 2, 2020.
26. Bly D, Schallom M, Sona C, Klinkenberg D. A model of pressure, oxygenation, and perfusion risk factors for pressure ulcers in the intensive care unit. Am J Crit Care 2016;25(2):156-64.
27. El-Marsi J, Zein-El-Dine S, Zein B, Doumit R, Badr LK. Predictors of pressure injuries in a critical care unit in Lebanon. J Wound Ostomy Continence Nurs 2018;45(2):131-6.
28. Wilczweski P, Grimm D, Gianakis A, Gill B, Sarver W, McNett M. Risk factors associated with pressure ulcer development in critically ill traumatic spinal cord injury patients. J Trauma Nurs 2012; 19(1):5-10.
29. Cox J, Roche S. Vasopressors and development of pressure ulcers in adult critical care patients. Am J Crit Care 2015;24(6):501-10.
30. Kirkland-Kyhn H, Teleten O, Wilson M. A retrospective, descriptive, comparative study to identify patient variables that contribute to the development of deep tissue injury among patients in intensive care units. Ostomy Wound Manage 2017;63(2):42-7.
31. Cramer E, Seneviratne M, Sharif H, Ozturk A, Hernandez-Boussard T. Predicting the incidence of pressure ulcers in the intensive care unit using a machine learning. EGEMS (Wash DC) 2019;7(1):49.
32. Barrett ML, Smith MW, Elixhauser A, Honigman LS, Pines JM, Utilization of Intensive Care Services, 2011. Healthcare Cost and Utilization Project. 2014. http://hcup-us.ahrq.gov/reports/statbriefs/sb185-Hospital-Intensive-Care-Units-2011.jsp. Last accessed December 2, 2020.
33. Apostolopoulou E, Tselebis A, Terzis K, Kamarinou E, Lambropoulous I, Kalliakmanis A. Pressure ulcer incidence and risk factors in ventilated intensive care patients. Health Sci J 2014;8(3):333-42.
34. Mulherin D, Cogle S. Updates in nutrition support for critically ill adult patients. Hosp Pharm 2017; 52(1):17-26.
35. Patel J, Hurt R, McClave S, Martindale R. Critical care nutrition: where's the evidence? Crit Care Clin 2017;33:397-412.
36. Efteli E, Gunes U. A prospective, descriptive study of risk factors related to pressure ulcer development among patients in intensive care units. Ostomy Wound Manage 2013;59(7):22-7.
37. Stotts N. Nutritional assessment and support. In: Acute and Chronic Wounds: Current Management Concepts. 4th ed. Bryant R, Nix D, eds. Elsevier: St Louis, MO; 2017.
38. Rote N. Innate immunity: inflammation and wound healing. In: Understanding Pathophysiology. 6th ed. Heuther S, McCance K, eds. Elsevier: St Louis; 2017.
39. Huether S. Fluids and electrolytes. In: Understanding Pathophysiology. 6th ed. Heuther S, McCance K, eds. Elsevier: St Louis; 2017.
40. Schwartz A, McCance K, Rote N. Alterations in hemotologic function. In: Understanding Pathophysiology. 6th ed. Heuther S, McCance K, eds. Elsevier: St Louis; 2017.
41. Cremasco M, Wenzel F, Zanei S, Whitaker I. Pressure ulcers in the intensive care unit: the relationship between nursing workload, illness severity, and pressure ulcer risk. J Clin Nurs 2012;22:2183-91.
42. de Almeida Medeiros AB, da Conceição Dias Fernandes MI, de Sá Tinôco JD, Cossi MS, de Oliveira Lopes MV, de Carvalho Lira ALB. Predictors of pressure ulcer risk in adult intensive care patients: a retrospective case-control study. Int Crit Care Nurs 2018;45:6-10.
43. Tschannen D, Bates O, Talsma A, Guo Y. Patient-specific and surgical characteristics in the development of pressure ulcers. Am J Crit Care 2012;21(2):116-24.
44. Kaitani T, Tokunaga K, Matsui N, Sanada H. Risk factors related to the development of pressure ulcers in the critical care setting. J Clin Nurs 2010;19:414-21.
45. Guérin C, Reignier J, Richard J-C, et al. Prone positioning in severe acute respiratory distress syndrome. N Engl J Med 2013;368:2159-68.

46. Lucchini A, Babmi S, Matiussi E, et al. Prone position in acute respiratory distress syndrome patients a retrospective analysis of complications. Dimens Crit Care Nurse 2020;39(1):39-46.
47. Peko L, Barakat-Johnson M, Gefen A. Protecting prone positioned patients from facial pressure ulcers using prophylactic dressings: a timely biomechanical analysis in the context of the COVID-19 pandemic [published online July 3, 2020]. Int Wound J.
48. Gefen A, Alves P, Ciprandi G, et al. Device-related pressure ulcers: SECURE prevention. J Wound Care 2020;29(Sup2a):S1-52.
49. Amrani G, Gefen A. Which endotracheal tube location minimises the device-related pressure ulcer risk: the centre or a corner of the mouth? Int Wound J 2020;17(2):268-76.
50. Nowicki JL, Mullany D, Spooner A, et al. Are pressure injuries related to skin failure in critically ill patients? Aust Crit Care 2018;31:257-63.
51. Tignanelli CJ, Weinberg A, Napolitano LM. Optimal methods to secure extracorporeal membrane oxygenation bicaval dual-lumen cannulae: what works? ASAIO J 2019;65:628-30.
52. Kosiak M. Etiology and pathology of ischemic ulcers. Arch Phys Med Rehabil 1959;40(2):62-9.
53. Oomens C, Bader D, Loerakker S, et al. Pressure induced deep tissue injury explained. Ann Biomed Eng 2015;43(2):297-305.
54. Gefen A. How much time does it take to get a pressure ulcer? Integrated evidence from human, animal, and in vitro studies. Ostomy Wound Manage 2008;54(10):26-35.
55. Coleman S, Nixon J, Keen J, et al. A new pressure ulcer conceptual framework. J Adv Nurs 2014; 70(10):2222-34.
56. Gefen A, Gershons. An observational, prospective cohort pilot study to compare the use of subepidermal moisture measurements versus ultrasound and visual skin assessments for early detection of pressure injury. Ostomy Wound Manage 2018;64(9):12-27.
57. Slomka N, Gefen A. Relationship between strain levels and permeability of the plasma membrane in statically stretched myoblasts. Ann Biomed Eng 2012;40(3):606-18.
58. McCance K, Grey T. Altered cellular and tissue biology. In: Huether S, Mccance K, eds. Understanding Pathophysiology. 6th ed. Elsevier; St Louis, MO; 2017.
59. Gefen A. The future of pressure ulcer prevention is here: detecting and targeting inflammation early. EWMA J 2018;19(2):7-12.
60. Gefen A. How medical engineering has changed our understanding of chronic wounds and future prospects. Med Eng Phys 2019;72:13-8.
61. Kottner J, Black J, Call E, Gefen A. Microclimate: a critical review in the context of pressure ulcer prevention. Clin Biomech 2018;59:62-70.
62. Antokal S, Brienza D, Bryan N, et al. Friction induced skin injuries—are they pressure ulcers? A National Pressure Ulcer Advisory Panel While Paper. 2012. https://cdn.ymaws.com/npiap.com/resource/resmgr/white_papers/1c._npuap-friction-white-pap.pdf. Last accessed December 15, 2020.
63. Agency for Health Care Policy and Research. Pressure Ulcers in Adults: Prediction and Prevention. Publication No. 92-0047. Rockville, MD: US Department of Health and Human Services; 1992.
64. Agency for Healthcare Quality and Research. Preventing pressure ulcers in hospitals: a toolkit for improving quality of care. www.ahrq.gov/patient-safety/settings/hospital/resource/pressureulcer/tool/index.html. Last accessed December 2, 2020.
65. Institute for Healthcare Improvement. How to guide: prevent pressure ulcers. 2011. www.ihi.org/resources/Pages/Tools/HowtoGuidePreventPressureUlcers.aspx. Last accessed December 2, 2020.
66. Gonzalez-Mendez M, Lima-Serrano M, Martin-Castano C, et al. Incidence and risk factors associated with the development of pressure ulcers in an intensive care unit. J Clin Nurs 2018;27(5-6):1028-37.
67. Black J, Edsberg L, Baharestani M, National Pressure Ulcer Advisory Panel. Pressure ulcers: avoidable or unavoidable. Results of the National Pressure Ulcer Advisory Panel Consensus Conference. Ostomy Wound Manage 2011;57(2):24-37.
68. Sullivan N, Schoelles KM. Preventing in-facility pressure ulcers as a patient safety strategy: a systematic review. Ann Intern Med 2013;158(5 Pt 2):410-6.
69. Ballard N, McCombs A, DeBoor S, et al. How are ICU decreased the rate of hospital acquired pressure ulcers. J Nurs Care Qual 2008;23(1):92-6.
70. Dibsie L. Implementing evidence-based practice to prevent skin breakdown. Crit Care Nurse Q 2008; 31(2):140-9.
71. Kelleher A, Moorer A, Makic M. Peer to peer nursing rounds and hospital-acquired pressure ulcer prevalence in a surgical intensive care unit. J Wound Ostomy Continence Nurs 2012;39(2):152-7.
72. Gray-Siracusa K, Schrier L. Use of an intervention bundle to eliminate pressure ulcers in critical care. J Nurse Care Qual 2011;26(3):216-25.
73. Richardson A, Peart J, Wright S, McCuallaugh I. Reducing the incidence of pressure ulcers in critical care units: a 4-year quality improvement. Inter J Qual Health Care 2017;29(3):433-9.
74. Edsberg LE, Langemo D, Baharestani MM, Posthauer ME, Goldberg M. Unavoidable pressure injury: state of the science and consensus outcome. J Wound Ostomy Continence Nurs 2014;41:313-34.
75. Manaker S. Use of vasopressors and inotropes. 2020. www.uptodate.com/contents/use-of-vasopressors-and-inotropes. Last accessed December 2, 2020.

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