How to keep patients un-HAPI: Cardiac surgery and sacral pressure injuries: Invited expert opinion: Hospital-acquired pressure injuries

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“A superior doctor prevents sickness; A mediocre doctor attends to impending sickness; An inferior doctor treats sickness.”

—Chinese proverb

Hospital-acquired pressure injuries (HAPIs) are debilitating complications with profound consequences. Wounds resulting from the effects of prolonged mechanical tissue loading were described early on by Hippocrates¹ and in the Ayurvedic system of healing.²

Cardiac surgery patients are at particular risk for the development of sacral HAPIs both during and after surgery, with a reported incidence as high as 16.7% to 29.5%.³⁻⁵ Patients who develop HAPIs suffer from associated consequences such as pain, infection, disability, and emotional distress. Direct costs related to HAPI treatment are substantial.⁶ It was estimated in 2007 that each HAPI adds \$43,180 in costs to a single hospital stay.⁷ Of US patients who develop HAPIs, 23% are acquired intraoperatively during surgeries that last longer than 3 hours, and the average estimated cost of treatment is \$750 million to \$1.5 billion per year.⁸ The Centers for Medicare and Medicaid Services have listed stage III and IV HAPI as “never events,” adversely impacting reimbursements for the treating facility.⁹ Due to the significant burden HAPI places on both patients and health care organizations, prevention is critical.

HAPI DEFINITION AND STAGING

Created in 2007, the National Pressure Ulcer Advisory Panel (NPUAP) established a system for categorizing pressure injuries. It defines a pressure injury as “localized damage to the skin and underlying soft tissue usually over a bony prominence or related to a medical or other device.”

Prophylactic placement of sacral silicone foam dressing to prevent pressure injury.

CENTRAL MESSAGE

Placement of a sacral silicone foam dressing before cardiac surgery is a cost-effective tool that prevents HAPI, thereby maximizing patient outcomes.

See Commentary on page 164.

In 2016, the NPUAP and European Pressure Ulcer Advisory Panel expanded the definition of pressure ulcers to 6 different stages^{10,11} (Figure 1):

- Category/stage 1 pressure ulcer: non-blanchable erythema

Intact skin with non-blanchable erythema in an area usually over a bony prominence. Individuals with darkly pigmented skin may not have visible blanching.

- Category/stage 2 pressure ulcer: partial-thickness skin loss

Partial-thickness skin loss with exposed dermis that may present as a shallow open ulcer with a red or pink wound bed or as a blister.

- Category/stage 3 pressure ulcer: full-thickness skin loss

Full-thickness skin loss with exposed subcutaneous tissue. No evidence of bone, tendon, or muscle visualization.

- Category/stage 4 pressure ulcer: full-thickness tissue loss

Full-thickness tissue loss with exposed bone, tendon, or muscle directly visualized or palpable. These can extend into muscle or supporting structures, making osteomyelitis possible.

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Received for publication Nov 1, 2019; revisions received Nov 24, 2019; accepted for publication Dec 4, 2019; available ahead of print Feb 19, 2020.

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J Thorac Cardiovasc Surg 2020;160:158-63

0022-5223/\$36.00

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<https://doi.org/10.1016/j.jtcvs.2019.12.108>

FIGURE 1. National Pressure Ulcer Advisory Panel/European Pressure Ulcer Advisory Panel staging of pressure ulcers. *SDTI*, Suspected deep tissue pressure injury.

• Unstageable pressure ulcer: depth unknown

Obscured, full-thickness tissue loss in which the base of the wound is covered by slough and/or eschar.

• Suspected deep tissue pressure injury (SDTI): depth unknown

Persistent, non-blanchable deep red, maroon, purple discoloration or blood-filled blister.

Operating room–acquired SDTIs may not appear until 48 hours after surgery.¹² They may be confused with electrical injury from energy-generating devices or chemical burns from antiseptic prep solutions. Other types of pressure injuries include medical device–related pressure injury¹³ and mucosal membrane pressure injury, which describes injuries that often conform to the shape of the medical device that is present or was previously in place.¹⁴ Among patients in acute-care hospitals, HAPI prevalence ranges from 10% to 17%.¹⁵ In 2009, 3.3% of patients in intensive care units in the United States developed a severe facility-acquired pressure ulcer defined as stage III, stage IV, unstageable, or deep tissue injury.¹⁶ Using the US Nationwide Inpatient Sample Database, a 5-year analysis between 2008 and 2012 identified an average of >670,000 patients per year who developed HAPI. Of this group, 16% were stage 1, 38% were stage 2, 20% were stage 3, 19% were stage 4, and 7% were unstageable.¹⁷ As high as 23% of hospital-acquired pressure ulcers have been shown to have originated in the operating room.¹⁸ To date, no information regarding prevalence of HAPI by stage in cardiac surgery patients has been published. A HAPI rate as high as 65% has been reported in patients receiving extracorporeal membrane oxygenation.¹⁹

HAPI RISK FACTORS

HAPI pathogenesis is multifactorial and mediated by both pressures of varying intensity and duration composed of compressive and shearing forces, which reduces blood supply and tissue tolerance composed of an individuals'

tolerance for pressure based on factors such as age and nutritional status plus oxygen supply and requirements.^{3,20,21} The interplay between the 2 factors mediates tissue damage (Figure 2).

Unique preoperative, intraoperative, and postoperative factors may account for the extraordinarily high risk of HAPI in cardiac surgery patients. Preoperative causes include advanced age, low or high body mass indices, sub-optimal nutritional status, anemia, renal dysfunction with creatinine >3 mg/dL or hemodialysis, previous or current tobacco use, diabetes, vascular disease, incontinence, decreased mobility or gait disturbances, impaired skin integrity in the area at risk, and previous HAPI.²²⁻²⁵ Relevant intraoperative events include skin shearing during transfers, pressure on bony prominences due to inadequate protection, friction due to frequent operating table adjustments, prolonged anesthesia preparation time, use of cardiopulmonary bypass, procedure duration >3 hours,^{26,27} hypothermia, hypotension, blood loss, and

FIGURE 2. A schema of HAPI-causative factors. *HAPI*, Hospital-acquired pressure injury.

hypoxia.²²⁻²⁵ Postoperative elements include intensive care unit length of stay >3 days, use of vasoactive agents, transfusion, mechanical ventilation with elevated head of bed, prolonged sedation, corticosteroid use, need for intra-aortic balloon pump or mechanical circulatory support, and lack of timely/adequate repositioning²⁸ (Table 1).

Although further investigation is required to better assess the inter-relationship of these various factors, it is most likely that when a cardiac surgery patient develops a HAPI within days after procedure, it first developed during the operation.²⁹

HAPI MEASUREMENT

Developed in 1987, the Braden score is a clinically validated tool³⁰ that health care providers can use in conjunction with clinical judgment to assess a patient's risk of developing HAPI.³¹ Ranging from a total score of 6 to 23, the 6 subscales measured include sensory perception, moisture, activity, mobility, nutrition, and friction/shear. Patients are then classified into 1 of 4 risk categories for HAPI development: mild (15-18), moderate (13-14), high (10-12), or severe risk (≤ 9).³²

The Braden scale has been found to have mixed results in acute-care settings.³⁰ In critical care patients, the subscales of sensory perception, mobility, moisture, fracture, and shear were found to be associated with an increased likelihood for HAPI development, whereas the subscales of activity and nutrition were found to not be significantly predictive.³³ Thus, modified Braden score scales have been proposed, some of which now include "body build for height" and "skin type," with exclusion of the nutrition subscale.³⁴ It has been reported to have a low predictive validity with an estimated sensitivity of 0.42 and a specificity of 0.84.³⁵ Therefore, multiple other scales were investigated for surgical patients. The commonly used Norton scale³⁶ included subscale measurements such as physical condition, mental status, activity, mobility, and incontinence. It has been found to have comparable sensitivity to the Braden scale but lower specificity. Within a cardiac surgery population, no scale was satisfactory.³⁷

HAPI TREATMENT

Once formed, a myriad of treatments for HAPI have been proposed. Interventions such as pressure relief, infection control, debridement, and dressings (eg, gauze, alginate, foam, hydrocolloid, hydrogel, silver-containing dressings, honey-coating dressings, transparent film dressings, negative pressure wound therapy) may be used. Other treatments include recombinant platelet-derived growth factor, electrical stimulation, electromagnetic agents, pulsed radiofrequency energy, phototherapy, acoustic energy, hydrotherapy, or oxygen therapy.³⁸ Current thinking places a greater emphasis on prevention. A multimodality approach including use of an assessment tool with

TABLE 1. HAPI risk factors for cardiac surgery patients

Preoperative:	Intraoperative:
<ul style="list-style-type: none"> • Advanced age • Low of high body mass indices • Suboptimal nutritional status • Anemia • Renal dysfunction with creatinine >3 mg/dL or hemodialysis • Previous or current tobacco use • Diabetes • Vascular disease • Incontinence • Decreased mobility or gait disturbances • Impaired skin integrity in the area at risk • Previous HAPI 	<ul style="list-style-type: none"> • Skin shearing during transfers • Pressure on bony prominences due to inadequate protection • Friction due to frequent operating table adjustments • Prolonged anesthesia preparation time • Use of cardiopulmonary bypass • Procedure duration >3 h • Hypothermia • Hypotension • Blood loss • Hypoxia
Postoperative:	
<ul style="list-style-type: none"> • Intensive care unit length of stay >3 d • Use of vasoactive agents • Transfusion • Mechanical ventilation with elevated head of bed • Prolonged sedation • Corticosteroid use • Need for IABP or mechanical circulatory support • Lack of timely or adequate repositioning 	

HAPI, Hospital-acquired pressure injury; IABP, intra-aortic balloon pump.

appropriate documentation, proper surface support, routine turning and repositioning, as well as adequate nutrition are now standard.

HAPI PREVENTION

Due to the significant burden HAPI places on both patients and health care organizations, prevention is critical. The use of silicone foam dressings when used prophylactically has been found to be superior to standard HAPI-prevention protocols.³⁹

For patients admitted via an emergency department, it has been recommended to begin pressure ulcer prevention early if they were likely to undergo surgery or stay in a critical care unit. A 5-layer silicone-bordered dressing was shown to reduce sacral pressure ulcers in conjunction with routine prevention practices.⁴⁰ A large retrospective observational cohort study of US acute-care academic medical centers showed that the use of 5-layer silicone-bordered dressings resulted in statistically significant and clinically meaningful decreases in HAPI.⁴¹ This was also shown in surgical intensive care patients.⁴² Further studies have confirmed clinical efficacy and cost-effectiveness.⁴³⁻⁴⁶ In addition, it was also demonstrated in a randomized trial⁴⁷ and in a review of nonexperimental prospective studies.⁴⁸

Prophylactic silicone foam dressings have 4 potential inter-related mechanisms of action⁴⁹:

1. Pressure redistribution: The presence of a dressing with adequate thickness distributes forces over a larger area, thus accomplishing pressure redistribution by reducing the percentage of magnitude of forces applied to the skin.
2. Shear redistribution: The dressing translates shear force to the skin outside the area of concern. Within the dressing itself, the interface of multiple layers aids in the absorption of shear. The elastic nature of the silicone adhesive also absorbs shear.
3. Friction reduction: The texture and material of construction of the dressing's outer layer is "slippery." Friction is the result of 2 surfaces moving relative to each other and decreasing friction reduces shear forces.
4. Microclimate management: The dressing maintains an optimal level of humidity at the skin surface, which maximizes resilience by avoiding either excessive dryness or maceration predisposing to skin breakdown.

HAPI IN CARDIAC SURGERY PATIENTS

The use of wound dressings as a potential additional protective means for the prevention of pressure injuries has been investigated over the past 20 years.⁵⁰ The protective mechanisms underlying the use of such dressings is through the reduction of tissue loading and internal deformations by creating an interface between the patient and support surface.⁵¹ Its first use in cardiac surgery patients was described in 2012.⁵² In this small, single-center nonrandomized study, silicone foam dressings were placed perioperatively and patients monitored during their time in a cardiac surgery intensive care unit. Of those receiving "standard" care, 4 of 35 developed HAPI (11%) versus 1 of 50 (2%) of those receiving a sacral dressing. A subsequent larger study of elective cardiac surgery procedures found no occurrence of SDTI in 224 patients measured at 5 days postoperatively in whom silicone foam dressings were prophylactically applied to the sacrum preoperatively versus a 2.33% incidence in 300 historic controls.⁵³ Our own work,⁵⁴ which began as a quality-improvement initiative, now totals more than 300 consecutive patients and included all major cardiac surgery procedures (none requiring or involving mechanical circulatory support). The only exclusion criterion was reported allergy to the dressing material, of which there were none. After a skin assessment, a silicone foam dressing was applied preoperatively either on a patient's hospital unit for urgent or emergent procedures or in the holding area for elective cases. Proper placement was confirmed and documented by the operating room nursing team. The dressing remained in place throughout the patient's stay in the intensive care unit and until the time of their first shower, which was typically on the morning of postoperative day 3. Total average length of stay postoperatively was 4 days. Sacral skin integrity was formally

assessed preoperatively, following dressing removal, before discharge and 30 days postoperatively. For virtually all patients, only a single dressing was required during hospitalization. During the study period, only 1 patient developed an SDTI, which was completely resolved by postoperative day 30. Overall, a HAPI incidence of <0.30% was achieved. As HAPI were only reported anecdotally and not formally tracked before the initiation of the study, a baseline incidence could not be accurately established.

EXTENSION OF HAPI PROPHYLAXIS

As a result of the dramatic results in cardiac surgery patients, the wound care team at our 4-hospital health care system has undertaken an initiative to extend the use of prophylactic sacral silicone foam dressings to other patients. High-risk inclusion criteria will include any patient who meets 1 of the following: recent cardiac arrest, vasopressor medications for >48 hours, mechanical ventilation >48 hours, septic/hypovolemic/cardiogenic shock, history of sacral/coccygeal pressure ulcers, current redness in the sacral/coccygeal area, ejection fraction <25%, anticipated operative procedure lasting more than 4 hours in the supine position, quadriplegic/paraplegic/hemiplegic status, cerebral vascular accident, traction, or inability to reposition oneself. Patients will be additionally included should they have 3 or more of the following criteria: body mass index >35 or <18, Braden score <18, weeping edema or anasarca, left ventricular assist device, right ventricular assist device, or intra-aortic balloon pump drive lines in place, age >70 years, diabetes, renal failure, liver failure, malnutrition (albumin <2.5 g/dL), and bed rest for a minimum of 4 hours. However, as a single-center study with indeterminate historical controls, the applicability and external validity of our results is uncertain. Findings should be validated in a large, multicenter study involving a matched control group with appropriate randomization processes and long-term follow-up. The fundamental issue that hinders evidence-based progress in this area is that prevention, as opposed to therapeutic efficacy, is difficult to prove. Assuming a HAPI incidence of only 10% over a 1-year period, this would require examining 1000 patients in a treatment group and 1000 patients in a control group to compare and determine statistically significant differences. Such a large trial is not anticipated in the near future. Additionally, there are currently 4 major marketed silicone foam dressings: Alle-vyn (Smith & Nephew, London, United Kingdom), Aquacel (ConvaTex, Inc, Deeside, United Kingdom), Mepilex (Mölnlycke Health Care, Gothenburg, Sweden), and Optifoam (Medline, Northfield, Ill). All have somewhat-different construction and intrinsic properties. Few published head-to-head data regarding clinical superiority exist. A critical analysis of current testing of silicone foam dressings highlights the benefits and faults of current biomechanical and clinical research in a call for higher

standards in the field.⁵⁰ The use of steel ball testing to obtain interface pressure measurements using a threshold of 32 mm Hg as a supposed baseline for capillary closure resulting in tissue hypoxia is disputed. Presumably this testing method does not adequately or accurately simulate the anatomy, physiology, or biomechanics of the human body. Alternative approaches with these considerations in mind need to be developed to properly determine superiority amongst the various brands.

ADVANTAGE OF HAPI REDUCTION

Although most commonly attributed to Benjamin Franklin, the aphorism, “An ounce of prevention is worth a pound of cure. It’s more prudent to head off a disaster actually beforehand than to deal with it after it occurs,” can actually be traced to Henry de Bracton’s *De Legibus et Consuetudinibus Angliae*. Whether referring to 18th-century Philadelphia firefighting readiness, 13th-century English jurisprudence, or 21st-century medical care, it is axiomatic that both the direct and indirect costs of inadequate deterrence are significant. Patients undergoing cardiac surgery are at particularly increased risk for HAPI. Preoperative prophylactic placement of a silicone foam dressing on the sacrum is a fiscally prudent and transformative method of preventing a profoundly debilitating condition. It can result not only in potentially significant cost savings through reduced use of hospital resources and shortened length of stay, but also in improved patient, family, and staff satisfaction. The NPUAP/European Pressure Ulcer Advisory Panel 2014 guidelines include a level of evidence B recommendation to “consider applying a polyurethane foam dressing to bony prominences (eg, heels, sacrum) for the prevention of pressure ulcers in anatomical areas frequently subjected to friction and shear.”⁵⁵ Updated guidelines are presently pending. Based on size, shape, and manufacturer, the average cost of such a dressing in 2011 was just over \$7.⁵⁶ Currently, the most expensive such dressing marketed typically costs less than \$11. Given that it costs more than \$40,000 to treat a single HAPI, the decision is clear. Multidisciplinary approaches have been shown to reduce the incidence of HAPI development in cardiac surgery patients. Implementation of institutional protocols such as formal mandatory staff education, development of turning guidelines, use of skin care bundles, and clinical coaching have been shown to decrease the rate of development of HAPI by 44% in patients with ECMO.¹⁹ In conjunction with additional standard prevention methods such as skin risk assessment, appropriate surface support, and adequate nutrition, preoperative prophylactic sacral silicone foam dressing application should be considered as a new standard of care and included as a component in the next iteration of the Enhanced Recovery After Surgery guidelines for perioperative care in cardiac surgery.⁵⁷

Conflict of Interest Statement

Authors have nothing to disclose with regard to commercial support.

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