CLINICAL MANAGEMENT extra

Pressure Injuries in the Pediatric Population: A National Pressure Ulcer Advisory Panel White Paper

CME
1 AMA PRA
Category 1 Credit™

CE
ANCC
1.5 Contact Hours

Barbara Delmore, PhD, RN, CWCN, MAPWCA, IIWCC-NYU • Senior Nurse Scientist • Center for Innovations in the Advancement of Care • Clinical Assistant Professor • Hansjörg Wyss Department of Plastic Surgery • NYU Langone Health • New York, New York
Michelle Deppisch, PT, CWS, FACCWS • Wound Care Regional Clinical Specialist • Mölnlycke Health Care • Norcross, Georgia
Cynthia Sylvia, DNurs, MSc, MA, RN, CWCN • Managing Member • Cynthia Sylvia LLC • Charles Town, West Virginia
Crystal Luna-Anderson, BSN, RN, CWON, CFCN • Wound Care Director, Hospital Division • Kindred Healthcare • Louisville, Kentucky
Ann Marie Nie, MSN, RN, FNP-BC, CWOCN • Wound, Ostomy Care Nurse Practitioner • Children's Minnesota • Minneapolis, Minnesota

Acknowledgments: The authors thank Caroline Portoghese and Sharon Sonenblum for their significant contributions to this article. The authors, faculty, staff, and planners, including spouses/partners (if any), in any position to control the content of this CME/CNE activity have disclosed that they have no financial relationships with, or financial interests in, any commercial companies relevant to this educational activity.

To earn CME credit, you must read the CME article and complete the quiz online, answering at least 13 of the 18 questions correctly.

This continuing educational activity will expire for physicians on August 31, 2021, and for nurses September 3, 2021.

All tests are now online only; take the test at http://cme.lww.com for physicians and www.nursingcenter.com for nurses. Complete CE/CME information is on the last page of this article.

GENERAL PURPOSE:

To review what is known about pediatric pressure injuries (PIs) and the specific factors that make neonates and children vulnerable.

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 should be better able to:

1. Identify the scope of the problem and recall pediatric anatomy and physiology as it relates to PI formation.
2. Differentiate currently available PI risk assessment instruments.
3. Outline current recommendations for pediatric PI prevention and treatment.

ABSTRACT

Pediatric patients, especially neonates and infants, are vulnerable to pressure injury formation. Clinicians are steadily realizing that, compared with adults and other specific populations, pediatric patients require special consideration, protocols, guidelines, and standardized approaches to pressure injury prevention. This National Pressure Advisory Panel white paper reviews this history and the science of why pediatric patients are vulnerable to pressure injury formation. Successful pediatric pressure injury prevention and treatment can be achieved through the standardized and concentrated efforts of interprofessional teams.

KEYWORDS: medical device-related pressure injury, pediatrics, pediatric risk assessment, pressure injuries, pressure injury prevention, pressure ulcers, white paper

ADV SKIN WOUND CARE 2019;32:394–408.

INTRODUCTION

Pediatric pressure injuries (PIs) are increasingly recognized as a source of possible iatrogenic harm, morbidity, suffering, and increased costs.[1,2] The National Pressure Ulcer Advisory Panel (NPUAP) initially drew attention to this problem in a 2001 monograph on PI prevalence and incidence.[3] This was followed by additional data in the 2012 NPUAP incidence and prevalence monograph.[4] The 2014 NPUAP, European Pressure Ulcer Advisory Panel (EPUAP), and Pan Pacific Pressure Injury Alliance (PPPIA) International Guideline devoted an entire chapter to the prevention of PIs in pediatric patients.[5] Much has been learned since those initial efforts to elucidate this critical problem.

The purpose of this article is to review what is known about pediatric PI and the specific factors that make children so vulnerable. The authors describe the scope of the problem; pediatric anatomy and physiology as it relates to PI formation; and the current recommendations for pediatric PI prevention and treatment, including 18 selected skin and PI risk assessment instruments.

Scope of the Problem

Goudie and colleagues[6] looked at the costs of stages 3 and 4 PIs and found that the average PI costs were nearly $20,000 per injury. In patients between 1 and 4 years of age, the average cost was $85,853;[6] when multiplied by available prevalence and incidence data, the cumulative cost is astounding. In general, PIs are reported as prevalence and incidence data with varying formulas.[5]

Early pediatric studies (2010 or earlier), including multicenter studies, reported pediatric PI prevalence estimates ranging from 0.47% to 35%.[7–16] Pediatric ICUs (PICUs) have higher prevalence estimates, ranging from 7.1% to 44%.[16–18] Schlüer and colleagues[16] reported an estimate of 43% in their neonatal ICU (NICU). Facility-acquired pediatric PIs often comprise a substantial portion of these injuries.[11] In one study of nine ICU units, authors reported an 8.7% prevalence estimate; 3.4% of the PIs were facility-acquired.[11]

Earlier incidence data (2010 or earlier), which may be reported in the literature as a percentage or as incidence density, demonstrated higher incidence in ICUs. For example, incidence may range from 0.29% to 7.2%.[7,19] In PICUs, however, the reported incidence ranged from 7% to 27%.[13,20–24] In NICUs, incidence percentage was reported between 8% and 16%.[25,26] Expressed as incidence density, Visscher and colleagues[20] reported 14.3/1,000 patient-days in the PICU (preimprovement effort; postimprovement effort, 3.7/1,000 patient-days) and 0.9/1,000 patient-days in the NICU regardless of improvement effort.

In general, there is little change between the prevalence and incidence data collected before or in 2010 as compared with data collected 2011 and later. Prevalence estimates after 2010 have ranged from 1.4% to 8.2%,[27,28] whereas critical care areas report prevalence as high as 43.1%.[28] In a more recent study of hospital-acquired PI (HAPI), Razmus and Bergquist-Beringer[28] found that prevalence was highest among children aged 9 to 18 years (1.6%) and then those aged 5 to 8 years (1.4%). Critical care (3.74%) and rehabilitation units (4.6%) had the highest HAPI prevalence. The lowest HAPI prevalence estimate was in general pediatric units (0.57%).

Incidence data reported after 2010 tend to be reported as incidence density in relation to quality improvement efforts. For example, Peterson and colleagues[29] reported data both pre- and postimprovement (2010 to 2014). Before their improvement effort, incidence was 3.3/1,000 patient-days (second quarter 2010), which was reduced to 1.7/1,000 patient-days postimprovement (second quarter 2014). Frank and colleagues[1] saw a reduction in stages 3 (0.06–0.03/1,000 patient-days) and 4 PIs (0.01–0.004/1,000 patient-days) after an improvement effort.

Devices are a leading cause of PIs in the younger pediatric population (neonates and children).[2,15,30] Medical devices may account for 38.5% to 90% of PIs,[15,16,20,25] and patients with this type of PI tend to be younger.[25,26] For example, Visscher and Taylor[25] evaluated NICU patients between 2007 and 2009. They found that nearly 80% of the PIs were associated with devices, and more than 90% of device-related PIs occurred in the premature infants. The authors also suspected that the higher estimates in the NICU indicated a "susceptibility to iatrogenic injury in pediatrics."[25] Early studies (published before 2010) did not always separate out device-related PIs or specify whether device-related PIs were included or excluded.[31]

Respiratory devices are particularly problematic. For example, in their progressive care unit, Miske and colleagues[32] found 35

PIs; approximately half were caused by tracheostomy securement devices (fiscal year 2015). Four months after practice change (performing neck assessments every shift), there was a decrease in securement device-related PIs to zero. However, the authors found it challenging to sustain these efforts and maintain this result.[32]

Although it is prudent to use prevalence and/or incidence data to determine if a problem exists and successfully address it, this appears to be easier said than done. Challenges include misunderstandings regarding terms and definitions of prevalence and/or incidence, failure to clearly define the study population, lack of resources for data collection, inability to distinguish between wounds and PIs, failure to acknowledge that devices can cause PIs, and errors in data analysis and interpretation.[33,34] Further, providers should exercise caution when comparing data points from earlier years with those from more recent years because there are differences in the parameters included or excluded (eg, PIs only or PIs and other wound types), methodologies employed, single versus multiple sites, terminology, classification, and study quality.[13,31,33] There have also been revisions to the NPUAP staging system during the years that these studies were conducted.

ANATOMY AND PHYSIOLOGY

Much of the vulnerability to PIs can be appreciated given an understanding of pediatric skin development and maturity. Many skin issues, including PIs, are attributable to skin immaturity and body size and further compounded by the need for ventilation and invasive technologies to improve survival rates in young patients.[26,35]

Pediatric skin maturity certainly differs from adults, yet differences also exist among pediatric populations. Neonates and infants younger than 2 years are considered especially vulnerable.[21] Makimoto and colleagues[36] noted PI damage as early as day 12 on a patient born at 24 weeks of gestation and weighing 653 g. A recent case series highlighted three infants (two premature, one full-term) believed to have developed PIs in utero, termed "congenital PIs."[37]

The pediatric population can be categorized by age, birth-weight classification, and a combination of birth-weight classification and gestational age (Table 1).[38–42] There may be some overlap or different terminology used to categorize the pediatric population, but it is important to understand that skin anatomy differences and a variety of factors can contribute to pediatric propensity to skin injuries. According to the 2014 NPUAP, EPUAP, PPPIA International Guideline, impairments in skin condition are associated with higher incidence of PIs.[5] Therefore, it is critically important to maintain optimal skin conditions to prevent PIs as pediatric skin matures. For example, as seen in seminal and more recent work, the neonate's vulnerability to skin injuries can be attributed to certain key structures that are underdeveloped, diminished, or unstable, and these may be interrelated.[43] Daily care such as bathing, moisturizing, or removing adhesives can disrupt normal barrier function, putting neonates at risk of PI formation.[43,44]

In neonates, the stratum corneum is thinner and has only two or three layers.[43,45] To put this into perspective, the adult and full-term infant have 10 to 20 layers of stratum corneum. The ruddy appearance common in neonates is because of these few layers of stratum corneum. Therefore, assessing oxygenation using skin color in neonates is a poor method.[43] In neonates younger than 24 weeks' gestational age, there may be virtually no stratum corneum.[43] This underdeveloped and thin skin structure may be a challenge for clinicians as they attempt to stage a PI. Stratum corneum maturity depends on the gestational age of the neonate, but it is believed to be mature by 30 to 32 weeks' gestational age.[45,46] Based on their research, Kalia and colleagues[45] found that the state and rate of barrier function development of the stratum corneum are dependent on a combination of gestational and postnatal age.

The stratum corneum controls the loss of evaporative heat and transepidermal water loss (TEWL), which is an important consideration when caring for preterm infants because it is an indicator for skin barrier function.[47] Normal adult TEWL levels are less than 10 g/m^2 per hour;[48,49] this is the same for a more mature infant (≥37 weeks old).[46,49] However, in younger infants (≤32 weeks old), this value is higher than 10 g/m^2 per hour until maturity is reached.[45,46,50] For clinicians, a higher TEWL presents challenges for fluid balance and temperature control because of high insensible water loss; this may translate to dehydration and heat loss.[46,47,51] In particular, dehydrated skin, which is dry and lacks the protective moisture that makes skin more resistant to trauma, is more predisposed to skin injuries.

Further, Lund and colleagues[50] found that adhesive materials, particularly the removal of adhesive materials, can increase TEWL in neonates. Great care should be taken by clinicians when removing these products or applying products with strong adhesive properties (eg, cloth tape, transparent films). In addition, products that promote adhesion (eg, tincture of benzoin) may result in increased epidermal stripping because of the very strong bond created between a product and the skin.[50]

Other conditions predisposing premature skin to injury include the diminished cohesion between the epidermis and dermis. In premature infants, the fibrils (rete ridges) that connect the epidermis to the dermis at the dermoepidermal junction are widely spaced and fewer. Although this bond becomes stronger with advancing gestational and postnatal age, until maturity is reached, the neonate is susceptible to injury from adhesive removal, blistering from friction damage, and/or thermal injury.[43]

Toxicity may occur in the form of topical agents applied to the skin, which may be too readily absorbed. Certain agents have

Table 1.
PEDIATRIC AGE CATEGORIZATIONS AND BIRTH WEIGHT CLASSIFICATION TERMINOLOGY

Terminology	Additional Terminology	Age/Birth Weight
Birth weight classifications	Micropreemie	<800 g or 1.8 lb
	Extremely low birth weight	<1,000 g or 2.2 lb
	Very low birth weight	<1,500 g or 3.3 lb
	Low birth weight	<2,500 g or 5.5 lb
	Normal birth weight	2,500 g (5.5 lb) – 4,000 g (8.8 lb)
	High birth weight	4,000 g (8.8 lb) – 4,500 g (9.9 lb)
	Very high birth weight	>4,500 g (9.9 lb)
Birth weight and gestational age combined	Appropriate for gestational age	Birth weight between the 10th and 90th percentile for infant's gestational age
	Small for gestational age	Birth weight 2 SDs below mean weight for gestational age or below the 10th percentile for gestational age
	Large for gestational age	Birth weight 2 SDs above mean weight for gestational age or above the 90th percentile for gestational age
Preterm, term late-preterm, and postterm terminology	Extremely preterm	<28 wk
	Very preterm	28–32 wk
	Preterm	<37 wk
	Late preterm	34 0/7 to 36 6/7 wk
	Moderate-late preterm	32–37 wk
	Term	37 0/7 to 41 6/7 wk
	Postterm	42 0/7 wk or more
Neonatal (1st mo of life) terminology	Premature	Born before 37th gestational wk
	Full-term	Born between 37 and 42 wk of gestation
	Postmature	Born after 42 wk of gestation
	Newborn	Birth to 29 d
Infant		Includes the neonatal period (birth) and is up to 12 mo
Child (2–12 y)	Toddler	1–3 y
	Preschool	3–5 y
	Grade school	5–12 y
Adolescent (11–21 y)	Early adolescent	11–14 y
	Teen	12–18 y
	Middle adolescent	15–17 y
	Late adolescent (young adult)	18–21 y

been known to cause issues, including iodine;[44,51,52] isopropyl, ethyl, and methyl alcohol;[44,53] chlorhexidine;[44] and hydrocortisone.[54] If antiseptics are used, they should be in an aqueous form rather than a spirit form.[44,46] In addition, the use of invasive procedures (eg, intravenous infusions) and adhesives that can cause skin trauma (eg, skin stripping) may further compound toxicity or exacerbate skin damage and increase dryness because they interfere with skin barrier function.[44,46,48,55] Preferred barrier products generally are those with a petrolatum base.[44] The message for clinicians is protect neonates against toxicity, infection, injuries, and pressure until the stratum corneum has reached maturity.

Edema may be seen in the neonate because there is less collagen and fewer elastic fibers in the dermis.[43,44] Edema affects the skin's turgor or elasticity and reduces blood flow, which can result in ischemic injury.[43] For clinicians, edema is a signal to protect premature infants from pressure damage using appropriate preventive strategies.

Another important feature of the skin is the acid mantle, a slightly acidic film on the skin's surface. The acid mantle provides protection against some microorganisms and is ideal when the skin surface pH is between 4 and 6.5.[43,44,48,49,56] Skin barrier function is altered when the pH shifts from acidic to neutral, which can increase the total number of bacteria or conditions, such as inflammatory dermatoses ("diaper dermatitis").[43,57] A more alkaline environment can also increase the TEWL. Full-term infants at birth have a more alkaline skin pH, which drops to a more acidic level like that of adults within days of birth,[39,43,49] but this continues to change during the first few weeks of life.[57] Fluhr and colleagues[57] found the greatest mean skin surface pH on newborns was 6. Earlier work conducted by Fox and colleagues[58] on very low birth weight (VLBW) infants found the development of the mantle over time was influenced by birth weight, skin area, and postnatal age. Like term infants, VLBW infants are born with a higher pH that rapidly decreases over the first week and then more gradually over the following 3 weeks.

As children age, issues can occur because of an overall developmental growth issue, such as a larger occipital size versus older children, who are likely to develop a PI in the same areas as an adult (sacrum, heels, etc). The anatomical structure of a young child's head is proportionally larger and heavier, with a lack of adipose tissue. As a result, the occiput is a frequently reported location for PI among younger patients (infants to age 5 years).[12,19,30,59,60] Along with anatomic issues, children younger than 5 years cannot properly differentiate pressure sensation from other sensory perceptions such as devices because of their developmental status.[15]

In summary, because the epidermal layer is thinner and functionally immature and young skin carries a high risk for excess water loss and a higher permeability to chemicals, the prevention of pressure damage and the need for optimal skin care in this group are paramount.[21] If the skin's main function is to serve as a barrier to the outside environment, then prevention strategies are important to protect the skin from damage when it is still developing.

ASSESSING PRESSURE INJURY RISK IN PEDIATRIC POPULATIONS

The shortcomings of PI risk assessment by bedside nurses specifically may be attributable to multiple factors. These include the lack of validated risk assessment instruments or instruments that can capture all risk factors,[20,21,25,26,29,30,61–65] minimal nursing knowledge of neonatal and pediatric integumentary structure and susceptibility to pressure,[29,66–68] inadequate nursing knowledge in PI identification and staging, and the dearth of skin bundles specific to pediatric patients.[1] However, there are several system and facility barriers to adequate pediatric PI risk assessments as well.

Clinical practice guidelines, to this point, have primarily focused on adult PI prevention practice and treatment. The NPUAP, EPUAP, and PPPIA acknowledged this gap and included a chapter on pediatrics in the 2014 Clinical Practice Guideline.[5] Many adult PI research findings cannot be applied to the pediatric population directly because of the anatomic and physiologic differences previously described. Yet there are far fewer PI studies conducted in pediatric populations. This research dilemma is compounded further by the ethical and legal issues involved in carrying out research on a pediatric/neonatal population. The end result has been that clinicians find themselves without evidence from which to determine risk or develop evidence-based interventions. Numerous articles note that it is not unusual for skin care regimens to be based on individual or institutional preference and routine.[30,69] This general lack of a unified pediatric protocol structure for PI risk assessments on admission and at regular intervals during hospitalization has led to a varied range of reported pediatric PI data.

Risk Assessment Scales and Psychometric Testing

In 2013, Kottner and colleagues[70] published their systematic review of articles addressing pediatric PI risk assessment scales and reported that, of 1,141 articles, only 12 described standardized PI scales for children. They determined that none of the scales that had undergone psychometric testing were superior to another, albeit based on "sparse results." Although there have been many assessments of pediatric skin, skin condition, wound, PI risk assessment instruments, or structured protocols created over the years (Table 2), only a limited number have undergone more serious psychometric testing (Table 3), even since Kottner and colleagues' review. The following paragraphs highlight the pediatric PI risk assessment instruments that have undergone initial psychometric testing.

The Neonatal Skin Risk Assessment Scale, first published in 1997, was developed by Huffines and Logsdon[35] specifically for the neonatal population (gestational age 26–40 weeks) at risk of injury and is based off of the Braden scale. It has six subscales: general physical condition, mental status, mobility, activity, nutrition, and moisture. Interrater reliability for the subscales general physical condition, activity, and nutrition was 97%.[35] The three subscales that were not included for the pilot study because of low reliability coefficients were mental status, mobility, and moisture. Evidence for predictive validity was present using a cutoff score of 5 with a sensitivity of 83% and specificity of 81%. However, despite the low reliability of the three subscales Huffines and Lodgson[35] suggested using all six subscales of the instrument because all are considered important in determining neonate risk.

The Braden Q scale was adapted from the adult Braden Risk Assessment scale in 1996. In 2003, Curley and colleagues[71] published

Table 2.
SUMMARY OF PEDIATRIC RISK AND SKIN ASSESSMENT INSTRUMENTS/GUIDELINES

Instrument or Guideline	Rating Scale	Author	Population
Assessing Patients at Risk for Development of Pressure-Related Breakdown	4–5 = No risk 6–7 = Level I 8–12 = Level II 13–16 = Level III	Garvin[104]	PICU
Neonatal Skin Condition Score (NSCS)	Perfect score = 3 Worst score = 9	AWHONN/Lund and Osborne[105]	Neonatal
Braden Q	25 = ↓Risk 21 = Med risk 16 = ↑Risk	Curley et al[71]	21 d to 8 y
Braden Q+P Pressure Ulcer Risk Assessment Tool	Yes/no[a]	Galvin and Curley[106]	Pediatric perioperative
Braden QD	≥13 At risk	Curley et al[2]	Preterm to 21 y
Derbyshire Children's Hospital Paediatric Risk Assessment Score	0–5 = ↓Risk 6–10 = Med risk >11 = ↑Risk	Pickersgill[107]	Pediatric
Neonatal Skin Risk Assessment Scale	>13 At risk	Huffines and Logsdon[35]	Neonatal
Paediatric Pressure Area Risk Assessment	Yes/no[a]	Barnes[77]	Pediatric
Paediatric Pressure Sore Risk Assessment	↓Score = ↓risk ↑Score = ↑risk	Cockett[108]	PICU
Paediatric Risk Assessment Chart	10+ = At risk 15+ = ↑Risk 20+ = Very ↑risk	Bedi[109]	Pediatric
Pediatric Pressure Ulcer Prediction and Evaluation Tool	18–26 = ↑Risk[b]	Sterken et al[74]	Pediatric
Pressure Sore Risk Assessment	Yes/no[a]	Waterlow[110]	Pediatric
Pressure Ulcer Skin Risk Assessment Scale	↑Score = ↑risk	Gordon[111]	Pediatric burn
Seton Infant Skin Risk Assessment Scale	(Not finalized)	Vance et al[61]	Neonatal
Skin Injury Risk Assessment + Prevention	No total scoring[c]	Foster et al[75]	Preterm-adult
Starkid Skin Scale	Lower scores = ↑risk	Suddaby et al[60]	Pediatric
The Glamorgan Paediatric Pressure Ulcer Risk Assessment Scale	10+ = At risk 15+ = ↑Risk 20+ = Very ↑risk	Willock et al[73]	Pediatric
The Pattoid Pressure Scoring System	17–14 = ↓Risk 15–20 = Med risk 20+ = ↑Risk[d]	Olding and Patterson[112]	PICU

Abbreviations: AWHONN, Association of Women's Health, Obstetric, and Neonatal Nurses; PICU, pediatric ICU.
[a]Scale with nursing interventional prompts.
[b]Score of 3 in any category requires nursing intervention; score of 2 in nutrition requires nursing intervention.
[c]Patient considered at risk if 1 point is assigned in any of the eight categories.
[d]Scaling system as reported in publication.

their reexamination of the scale's predictive validity and critical cutoff point for classifying risk. The Braden Q scale has the same six subscales as the Braden scale, with an added seventh subscale: tissue perfusion and oxygenation. The seventh subscale was added to reflect unique pediatric developmental characteristics and optimize the benefits of data that are commonly available in PICUs.[72] The Braden Q scale is intended for use on pediatric patients from 21 days to 8 years old. Curley and colleagues[71] reported

Table 3.

PRESSURE INJURY RISK ASSESSMENT INSTRUMENTS THAT HAVE UNDERGONE PSYCHOMETRIC TESTING AND THE RISK FACTORS THEY MEASURE

	Instruments					
Subscale Factors	**Braden Q**	**Braden QD**	**Glamorgan**	**NSRAS**	**PPUPET**	**SIRA+P**
Age		✓	✓			✓
Appetite/diet/nutrition	✓	✓	✓	✓	✓	
Cast/splint		✓			✓	
Continence/elimination			✓			
Fever >4 h			✓			
Friction/shear	✓	✓			✓	✓
Hemoglobin/anemia			✓			
Hypoxia/oxygenation	✓	✓			✓	
Infection				✓		
Infusion/drain/nasogastric tube		✓			✓	
Medical devices		✓	✓		✓	✓
Mental status				✓		
Mobility/activity	✓	✓	✓	✓	✓	✓
Sensory perception	✓	✓			✓	✓
Low serum albumin			✓			
Skin moisture	✓			✓	✓	✓
Skin condition					✓	
Skin tolerance				✓		
Tissue perfusion	✓	✓	✓		✓	✓
Weight			✓			✓

Abbreviations: NSRAS, Neonatal Skin Risk Assessment Scale; PPUPET, Pediatric Pressure Ulcer Prediction and Evaluation Tool; SIRA+P, Skin Injury Risk Assessment + Prevention.

a sensitivity of 88% and specificity of 58%; with an at-risk score of 16, high-risk patients will not be missed, nor will preventive therapies be applied on those who do not develop PIs.

The Glamorgan scale[73] was the first widely used risk assessment scale to include devices. Willock and colleagues[73] published their examination of the scale in 2009. They found that at a risk score of 10, sensitivity was 100% and specificity was 50.2%; at a risk score of 15, sensitivity was 98.4% and specificity was 67.4%; and at a risk score of 20, sensitivity was 93.4% and specificity was 71.5%.[73]

In 2015, Sterken and colleagues[74] published their Pediatric Pressure Ulcer Prediction & Evaluation Tool (PPUPET). On admission, the PPUPET, which also has a subscale for external devices, was found to have a sensitivity of 74.58% and a specificity of 57.94%, retrospectively. On discharge, the PPUPET had a sensitivity of 76.27% and a specificity of 75.70%. However, sensitivity could not be calculated prospectively because of lack of patients with PIs; further, the specificity was very low. The authors believe the latter may be caused by the PPUPET's definition of risk.[74]

The electronic Skin Injury Risk Assessment + Prevention instrument was published in 2017 by Foster and colleagues[75] and also includes external devices. The authors used the Braden and Braden Q as a framework, but modified the eight subscales to have two responses that were "scored" either at risk or not at risk. The foundational concept of this instrument is that if a patient is at risk for any subscale, then the patient is at risk for all subscales. When "at risk" is selected, a list of evidence-based interventions is provided. The authors have compared the reliability and validity to the Braden and Braden Q; the Skin Injury Risk Assessment + Prevention has a correlative reliability of 0.556 and correlative validity range of −0.778 to −0.634.

The newest of these instruments, the Braden QD, was introduced by Curley and colleagues[2] in their 2018 publication and was founded on the Braden Q scale. Intended for those patients born preterm to 21 years, the Braden QD scale examines the five subscales of the Braden Q with the addition of two others: number

of medical devices and repositionability/skin protection. A total score of 13 or higher indicates that the patient is at risk, with a sensitivity of 0.86 and a specificity of 0.59.

Performing a PI risk assessment is still a valuable method for PI prevention. The 2014 NPUAP, EPUAP, PPPIA International Guideline[5] recommended that a structured risk assessment be conducted as soon as possible upon admission (up to a maximum of 8 hours after admission), as often as required by the patient's acuity, and with any significant change to the patient's condition. The admission assessment should include both a risk assessment (to evaluate risk for developing a PI) and a skin assessment (to detect existing PIs). These two assessments should be thought of as a single process step: a PI admission assessment.[76]

Risk Assessment Instrument Factors and Subscales

The authors' review of literature identified 18 skin and PI risk assessment instruments, some of which have undergone psychometric testing. In these 18 instruments, 58 different factors were identified. This variation and the sheer number of factors support the historic practice of each facility and/or nurse using clinical judgment to prevent pressure or skin injuries at a point in time. Although this may have served many patients successfully, it does leave a potential gap in practice. Any tenured nurse has a wealth of knowledge that cannot be immediately conveyed to newly graduated nurses during preceptorship. However, a risk assessment instrument has the sole purpose of presenting evidence-based practice in an algorithm that results in PI prevention through subscale intervention.

The most common factors in each instrument were the same as items normally found in adult risk assessment tools (Table 4). Nutrition was assessed in 12 (67%) of the 18 instruments, and weight was assessed in 6 (33%). These are important factors in PI prevention, but not all instruments assessed either or both of these factors. Tissue perfusion and skin moisture were each assessed in eight instruments (44%). One would expect that all 18 instruments would have looked at these important subscale factors. More surprisingly, only seven instruments (39%) assessed sensory perception, medical device/cast/splints, or friction/shear factors. However, as expected, mobility/activity was included in 14 (78%) of the 18 instruments. Again, with an effective and comprehensive risk assessment instrument, factors leading to pediatric PI are better understood and identified so that interventions can be put in place for prevention.

In addition, some instruments[60,71,74,77] have subscales that measure the concept of shear, which can be an underlying causal factor in PI development. However, this concept has many definitions; risk assessment instruments that claim to measure the concept of shear may in fact be measuring friction, shear force, shear strain, shear stress, or other indirect factors that may not specifically measure all or any components of shear.[78] A dedicated exploration of the complex concept of shear is beyond the scope of this article. The authors suggest that the evolution of science regarding shear should be taken into account in the development of future risk assessment scales, and researchers should clearly identify which aspect applies to a given risk assessment score. The forthcoming 2019 NPUAP, EPUAP, PPPIA International Guideline will discuss shear and tissue deformation in detail.

Clinical knowledge and practice have advanced since the first risk assessment instrument was developed. It is imperative that instruments be regularly reviewed against the current science of PI development and validated by reporting reliability, sensitivity, specificity, and predictive values. These instruments are a means for common communication and practice among direct care providers to protect pediatric patients. Initiating PI prevention strategies for at-risk patients, rather than all patients, will optimize the appropriate use of resources.[72]

VULNERABILITY TO PRESSURE INJURIES: CHALLENGES AND OPPORTUNITIES

Given the complexity of pediatric medicine, there are noted challenges in preventing PIs in this population. Most pediatric prevention protocols have been extrapolated from adult practice because the empiric data on which to base clinical practice guidelines are scarce, particularly for infants.[79] Providers should not treat children simply as scaled-down adults;[80] as previously discussed, pediatric PIs can be inherently different in etiology. Despite the many challenges, there are many opportunities to implement evidence-based recommendations.

Prevention Strategies for Optimal Skin Health

As previously discussed, maintaining skin integrity, especially in younger pediatric populations, is important in preventing PIs. Avoiding excess moisture is critical, because skin is susceptible to injury not only from moisture, but also the chemicals found in moisture sources such as stool, urine, respiratory devices, and caustic gastrointestinal effluent (eg, tube leakage). The fragility of the skin and the increased PI risk when the skin is damaged require products that can prevent, absorb, and/or diminish further damage. Routine use of petroleum-based products or products with zinc oxide is recommended for dermatitis, as well as "crusting" techniques using stoma powder in combination with a skin ointment barrier. Caustic effluent from a percutaneous endoscopic gastrostomy tube may require the use of a foam dressing for protection and absorption. The most common dressings in pediatric PI management include hydrocolloids, hydrogels (available as amorphous gel and sheets), polyurethane foams, and transparent films.[81] Exercise caution with dressings that can trap moisture and cause epidermal stripping, such as hydrocolloids

Table 4.

AN OVERVIEW OF PRESSURE INJURY RISK AND SKIN ASSESSMENTS/PROTOCOLS AND THE RISK FACTORS THEY MEASURE

Category	Factors	Risk/skin assessments/protocol
Physical and patient assessment	Body mass index, weight, age, gender, medication, mobility/activity, appetite/diet/nutrition, mean blood pressure x 24 h	Derbyshire, Pressure Sore, Paediatric Risk Assessment Chart, Paediatric Pressure Sore Risk Assessment, Pattoid, Paediatric Pressure Area Risk Assessment, SISRA, Assessing Patients at Risk, Starkid, PrUSRAS, Braden Q+P
Conditions	Continence/elimination, hypothermia/thermoregulation, increased bony prominences, infection, intensive care, physical disabilities, percent of total body surface area burned, sedation, sensory perception, severe illness, severe physical disability, trauma, neurological deficit, prior/current pressure injury, underlying conditions	Paediatric Risk Assessment Chart, Derbyshire, Paediatric Pressure Sore Risk Assessment, Pattoid, Paediatric Pressure Area Risk Assessment, PrUSRAS, SISRA, Pressure Sore, Assessing Patients at Risk, Starkid, Braden Q+P
Disease and diagnoses	Bone marrow transplant, head injury, malignancy, diabetes, respiratory, congenital heart disease	Pressure Sore, Paediatric Risk Assessment Chart, Paediatric Pressure Sore Risk Assessment, Pattoid, Braden Q+P
Forces	Friction/shear	Paediatric Pressure Area Risk Assessment, Starkid, Braden Q+P
Medical devices	Friction/shear/pressure	Paediatric Pressure Sore Risk Assessment, Pressure Sore, Paediatric Pressure Area Risk Assessment, PrUSRAS, SISRA, Braden Q+P
Oxygenation and circulation	Cardiac hemodynamic support/inotropic support, circulatory/vascular, cyanosis, tissue perfusion, tissue malnutrition, hypoxia/oxygenation	Paediatric Risk Assessment Chart, Paediatric Pressure Sore Risk Assessment, Pattoid, Starkid, SISRA, Braden Q+P
Risk scores	Glasgow Coma Scale, American Society of Anesthesiologists score, Braden Q score < 16	Paediatric Pressure Sore Risk Assessment, Braden Q+P
Skin integrity	Breakdown/excoriation, dryness, erythema, skin moisture, skin type, skin condition, skin tolerance	Neonatal Skin Condition Score, SISRA, Assessing Patients at Risk, Starkid, PrUSRAS, Paediatric Risk Assessment Chart, Derbyshire, Pattoid, Pressure Sore, Paediatric Pressure Area Risk Assessment, Paediatric Pressure Sore Risk Assessment, Braden Q+P
Surgical	Surgery intensity, duration, position; major surgery	Paediatric Pressure Sore Risk Assessment, Pressure Sore, Paediatric Pressure Area Risk Assessment, Paediatric Risk Assessment Chart, Braden Q+P

Abbreviations: Assessing Patients at Risk, Assessing Patients at Risk for Development Pressure Related Breakdown; Derbyshire, Derbyshire Children's Hospital Paediatric Risk Assessment Score; Pattoid, Pattoid Pressure Scoring System; Pressure Sore, Pressure Sore Risk Assessment; PrUSRAS, Pressure Ulcer Skin Risk Assessment Scale; SISRA, Seton Infant Skin Risk Assessment Scale; Starkid, Starkid Skin Scale.

and transparent films. Gentle dressings or adhesives (eg, tapes) with silicone are generally recommended.

Preventing Medical Device-Related Pressure Injuries

With the recent acknowledgement of increasing medical device-related PIs (MDRPIs) among pediatric patients,[82,83] more attention has been paid to ill-fitting medical devices or equipment that were not designed for pediatric patients, especially respiratory devices such as tracheostomies, endotracheal tubing, and continuous positive airway pressure (CPAP) machines.[25,30,35,36,46,83–85] Interestingly, it appears that various terms other than PI, including "injuries," "necrosis," breakdown," or "trauma," have been used over the years to describe the damage created by respiratory devices that may now be called MDRPI.

For example, Robertson and colleagues[86] reported nasal deformities along with "snubbing," "flaring," and columella nasi necrosis from CPAP prongs. Buettiker et al[82] performed a randomized study among three different CPAP systems: one nasopharyngeal tube and two types of prong systems. They found that the prong systems caused more "nasal injuries." Yong and colleagues[87] randomized different CPAP systems on 89 VLBW infants. One system was a nasal CPAP (N-CPAP) mask, and the other was a nasal prong device. A higher incidence of nasal "trauma" was seen in the nasal prong group. The most common "traumatized" sites were at the nasal septum/philtrum junction for the mask group and the nasal septum walls in the prong group. Further, an integrative review of skin "breakdown" in preterm infants performed by Newnam and colleagues[64] identified factors associated with "skin injury" during N-CPAP. A total of 46 studies were selected for data extraction on frequency of occurrence, severity, location, and type of "skin injuries" associated with nasal device interface.[64]

Because devices are a risk factor for pediatric PI, prevention strategies should aim to mitigate their effect.[34] The 2014 NPUAP, EPUAP, PPPIA International Guideline[5] made recommendations for reducing MDRPIs in conjunction with clinical judgment based on the patient's clinical situation and goals of care. While there are 19 recommendations for avoiding MDPRIs, the essence of these recommendations is to select and properly fit the medical device; assess the area around and under the device at least twice daily or as needed; and rotate, remove, relieve, or replace the device as necessary. The 2014 NPUAP, EPUAP, PPPIA International Guideline[5] recommendations have been upheld as the national standard and adopted by facilities and governing organizations. Adaptation of these recommendations can be seen in the literature; for example, using a moisture- and pressure-redistribution dressing such as a foam dressing[83] at the device interface[81] to reduce tracheostomy-related PIs. The forthcoming 2019 NPUAP, EPUAP, PPPIA International Guideline will incorporate new evidence regarding the prevention of MDPRI for all populations including children.

Support Surfaces

Based on the patient's identified risk, it is important that prevention strategies such as turning and repositioning are implemented. To augment prevention strategies, support surfaces may be employed to alleviate poor tissue tolerance and shear, improve microclimate, and/or address pain but should not take the place of turning and repositioning.[5] Choosing support surfaces for the pediatric population requires critical thinking and understanding of the anatomy and physiology. Pediatric muscle and fat tissue structures are softer than those of adults, making newborns and young children more susceptible to deformation-related injuries.[79] A surface needs to envelop the patient to redistribute the pressure, provide a low-friction interface to reduce shear, accommodate the patient's mobility status, and be appropriate for the developmental age of the patient.[5,88] Providers should take into consideration the frequent movements and growth requirements of pediatric patients. Further, a support surface should conform to the misplacement of tubes and lines and decrease PI susceptibility.

Computer simulations indicate that air cell mattresses provide superior protection against increased soft tissue deformation around a misplaced tube in NICU and PICU populations, compared with foam mattresses.[79,88] Other manufacturers produce low air-loss options such as mattress replacements, overlays, or pads. Unfortunately, the most common products for pressure redistribution, and the research behind them, are geared toward the adult population. The 2014 NPUAP, EPUAP, PPPIA International Guideline[5] not only provided general recommendations for selecting a support surface based on mobility level; controlling microclimate; shear reduction; risk of developing PIs; and the number, severity, and location of existing PIs for the adult population, but also recommendations for pediatric patients (under *Special Populations: Pediatrics*) when selecting support surfaces and repositioning while on a surface.

Prevention by Age Range

Extant literature[15] recommends that PI prevention in the pediatric population be conceptualized according to age. Some research has tried to demonstrate the effectiveness of interventions specifically based on age. There appears to be a natural division for care pathways and overall approach to care when guided by a framework based on selection of some defined age range according to characteristics such as integumentary development.

One such example is the neonate's skin immaturity and propensity to PIs and other skin injuries. Based on their findings of extremely low birth weight as a risk factor for nasal trauma, Chen and colleagues[89] advocated for guideline development around gestational age and birth weight because of physiologic differences. A multisite prospective study in Japan on PIs in a neonatal population (N = 211) demonstrated that skin texture was a predictor of PI risk.[26] Early work by Harpin and Rutter[46] demonstrated differences in the effectiveness of skin barrier properties explained by physiologic development of skin structure according to age. Infants who were 32 gestational weeks or less had marked drug absorption and water loss until 2 weeks of age compared with infants born at 37 or more gestational weeks. Newnam and colleagues[64] found that neonates of smaller birth weights and younger gestational ages were at risk during N-CPAP use and recommended prevention strategies including frequent skin assessments, focused examinations, correct prong size, adequate humidification, strategies for positioning, and the use of skin barriers to protect them from direct pressure. The authors noted that many of the

"injuries" were preventable but that a lack of standardization made prevention difficult, especially in preterm infants.[64]

In their retrospective study, Schlüer and colleagues[15] identified factors associated with the development of occipital PIs in pediatric populations. They concluded that their data support early prediction and intervention to prevent PIs according to age.[15] In Switzerland, Schlüer and colleagues[16] conducted a descriptive, multicenter point prevalence study on 412 patients from birth to 17 years. They found that the age and the department/unit were the two single characteristics that influenced PI occurrence, stages 2 to 4. Patients with PI stages 2, 3, and 4 were older than 8 years and had chronic conditions or surgical procedures, especially orthopedics. One case-control study (N = 59) in a 30-bed PICU found that the majority of PIs developed in infants younger than 3 years.[90] Distribution of PIs in patients younger than 1 year was 36%, whereas patients between 1 and 3 years had 30% of the PIs.

Based on literature like this, it seems evident that age-appropriate ranges are one way to categorize prevention strategies. However, part of the problem in addressing PI prevention by age may be that extant prevalence data may not include all pediatric-age groups or individuals, and thus epidemiologic data may not be reflective of pediatric populations at large. Further, to date, the literature demonstrates differences among pediatric populations regarding risk factors and PI locations.[91] More research is required to identify whether these differences truly lead to an increase in PIs and thus require prevention strategies according to age group. Regardless, an argument may be made for the allocation of intervention bundles according to specific age ranges to allow for individualized care.

Prevention Bundles and Programs

Empiric evidence has shown that multicomponent interventions may be more effective than individual actions in the prevention of PIs.[92] Pre- and postintervention studies, framed as quality improvement, describe interprofessional and multifaceted intervention bundles that have successfully decreased PI prevalence and incidence in specific populations.[20]

Solutions for Patient Safety, an initiative designed to reduce occurrences of harm in pediatric hospitals, examined active participation in a collaborative to implement PI prevention bundles of nursing interventions and found that PI occurrence decreased by 57%.[93] Implemented from 2009 to 2016, the bundles included five risk factors: medical devices, moisture, immobility, skin integrity, and support surface selection. No particular risk factor exerted a greater effect on PI occurrence, supporting the need for intervention bundles.

Frank and colleagues[1] also studied the pediatric population in 33 hospitals that were members of the *Solutions for Patient Safety* initiative. Their objectives were twofold: to increase the detection of PIs through active surveillance and to reduce the number of serious PIs (stage 3, stage 4, unstageable, deep tissue PI). This project used a three-pronged approach consisting of active surveillance, a prevention bundle, and the deployment of a wound ostomy continence nurse. After the implementation of the bundle, they found a decrease in PIs: 0.06 to 0.03 per 1,000 patient-days in participating institutions. They also found that hospitals reporting greater than 80% bundle adherence reported fewer PIs. Using a patient/day formula, Visscher and colleagues[20] found that PICU PIs decreased from 14.3 to 3.7/1000 patient-days after implementing a quality improvement bundle. In the NICU, however, they found that PIs did not significantly change after bundle implementation.

In a children's hospital, Boesch and colleagues[94] noted a high number of tracheostomy-related PIs in their ventilator unit and implemented a bundled intervention model that included reducing moisture and pressure at the device interface. They implemented the model over a 30-month period and found a decrease in the number of patients who developed a tracheostomy-related PI, from 8.1% to 2.6%. With the intent to improve quality of care and decrease incidence of nasal trauma, Chen and colleagues[89] initiated a standardized process and protocol with prepackaged kits. The kits and standardized nursing protocol decreased the incidence of nasal trauma from 42.2% to 19.6%, except for infants with extremely low birth weight (less than 1,000 g). The authors concluded that preventing nasal trauma by implementation of standard nursing protocol during N-CPAP is potentially one of the greatest opportunities for preventing of skin injury in this patient population.

In a PICU of a large tertiary care center, a prospective, quasi-experimental study was conducted to determine if a PI prevention program reduced PI development.[21] The program included a skin care bundle that incorporated five components: appropriate support surface, frequent turning and repositioning, moisture and incontinence management, appropriate nutrition, and nursing staff education. The staff in the control group received education, and the patients received standard care. The intervention group also received education and standard care, but incorporated skin care champions (staff nurses) who helped to facilitate adherence to the bundle and unit-based advanced practice nurses who performed root-cause analyses. A χ^2 analysis revealed that PI development in the control group was significantly higher than in the experimental group. This study demonstrates the importance of a systems approach to bundled interventions, along with dedicated skin care champions who reinforce and implement change into daily practice.

Prevention Through Industry Partnerships

Taking into consideration pediatric vulnerability to PIs and the current standard of care, several implications become apparent for the future of informed clinical practice. For example, given the

high occurrence of MDRPIs, collaboration with industry partners to develop innovative solutions is one way to attack the root cause of PIs. The recommendations of Robertson and colleagues[86] on pediatric CPAP use included providing rest time and ensuring an appropriate fit, but more importantly, they reported working with the manufacturer to address design flaws. The study led to a collaborative effort with the manufacturer to develop a new curved design with tapering nasal prongs that would help to eliminate the issues. In addition, the manufacturer worked with staff on modifications and learning activities that would support care until the new design was available. This is an example of a positive and productive resolution arising from a collaborative effort between clinicians and a manufacturer.

In their integrative literature review, Newnam and colleagues[64] found that researchers had recommended device design changes that would ultimately reduce tissue injury. A retrospective chart analysis of occipital PI incidence found that the majority of patients younger than 1 year were critically ill and using multiple medical devices,[59] including endotracheal tubes, mechanical ventilation, extracorporeal membrane oxygenation, central venous catheters, nasogastric tubes, saturation probes, and electroencephalography leads. This finding demonstrates the imperative to collaborate with industry partners to minimize the risks associated with these medical devices.

In their secondary analysis of the National Database of Nursing Quality Indicators, Razmus and Bergquist-Beringer [95] found that pressure redistribution support surface use as an intervention in the pediatric population was lower than in a previously reported adult population. Based on this finding, they recommended further investigation to better understand the effectiveness of support surface use in decreasing pediatric HAPIs. What was not clear to them was whether the pressure redistribution support surfaces were designed for pediatric or adult patients.

Clearly, end users of a product are in a position to offer recommendations and suggestions for innovative designs based on daily clinical experience. All clinical disciplines offer value when consulting with industry members involved in the research and design of equipment and devices that address the unique aspects and needs of a special population, such as anatomical or care needs.

There are various opportunities to partner with industry and manufacturers to improve the design, safety, and efficacy of devices, especially in the pediatric population.[96] Clinicians can partake in these opportunities through product evaluation committees in their own institutions or participate for advisory boards that engage clinicians and researchers for new product development; in fact, the American Nurses Association works to promote nurses who serve on various types of advisory boards through their Nurses on Boards Coalition.[97] The NPUAP, which is composed of medical, nursing, physical therapy, nutrition, and industry stakeholders, forms a cohesive partnership with a mission to serve as the authoritative voice for improved patient outcomes in PI prevention and treatment through public policy, education, and research.[98] This includes the Support Surface Standards Initiative (overseen by the NPUAP Research Committee), which is an interprofessional mix of industry, researchers, academics, and practicing clinicians who work together to standardize support surface performance evaluations.[99]

To be clinically relevant and meet the needs of the pediatric population, collaboration is required between professional caregivers and those involved in the design and development of equipment and medical devices. Developing devices and equipment that achieve highly reliable and quality care through the partnership of industry, researchers, academics, and providers should be the norm. Such interprofessional teams achieve the synergy required to enhance products and ultimately care.

Opportunities for PI Treatment

Unfortunately, like prevention strategies, pediatric PI treatment protocols are extrapolated from adult practice because of the paucity of relevant empiric data on which to base guidelines for clinical practice, particularly in infants.[79] Pediatric wound management lacks consensus because research is geared toward adults, which can pose risks to the neonatal and pediatric populations. Baharestani and Ratliff[30] stated that the rapid, uncomplicated wound healing of pediatric patients gives rise to a limited need for intervention, making it the "normative expectation" in this population. Some providers believe that healing occurs more expeditiously in younger patients, which is one of several factors that have resulted in\ the lack of consideration for wound care protocols with these populations.[30]

Wound care practices are currently based on a combination of provider experience and preference as well as a small number of published clinical guidelines based on expert opinion. This includes the choice of specific dressings or other wound care products for pediatric populations as evidenced by the following examples. Transparent films and hydrocolloids were favored at one point, especially in the younger pediatric populations. However, their increased propensity to cause skin stripping and/or moisture-associated skin damage has caused them to fall out of favor.[44,48,55,81,100] Medical-grade honey has been used more recently because it is seen as a "natural" product and parents and children respond positively to natural and gentle dressings that are effective and easy to use.[81,101] There is also documentation regarding its efficacy in healing wounds in pediatric populations, particularly oncology patients.[81,101–103]

However, rigorous evidence-based criteria and clinical guidelines for wound management for these populations are limited at best.[81] Unfortunately, because of the lack of clear-cut treatment

guidance, several problems arise. Importantly, clinicians may have to balance manufacturer-recommended products (which may not have been created with the pediatric patient in mind) with clinical concerns such as skin immaturity and absorption issues.[30] Given these issues, clinicians often find it difficult to determine the appropriate treatment.

The future of wound management for neonates and other pediatric populations will depend on continued research and guidelines created to assist clinicians in treating PIs. Currently, there are only a limited number of published clinical guidelines for the evaluation and management of wounds in neonatal and pediatric populations. None of these have undergone the rigorous assessment required for the generation of evidence-based guidelines.[81]

CONCLUSIONS

This white paper reviewed the history of and continued journey to pediatric PI prevention to reveal the scope of the problem, how pediatric anatomy and physiology can lead to PI formation, and the current recommendations for pediatric PI prevention and treatment. More recent published literature and trends show that clinicians are paying attention to the specific issues that make this population vulnerable to PI formation. This population requires special consideration, protocols, and approaches compared with adult or other specific populations. The slow but steady realization of this fact has significantly advanced provider thought processes, approaches, and care provisions particularly when addressing PI prevention and treatment. The wound care discipline must construct standardized approaches that involve targeted risk assessment, evidence-based guidelines, prevention strategies, medical equipment and device design, and wound treatments specific to this special and vulnerable population. This can only be accomplished by working in interprofessional teams that integrate all stakeholders, including industry partners. ●

PRACTICE PEARLS

- Pediatric patients, especially neonates and infants, are vulnerable to PI formation.
- Pediatric patients should have a risk assessment using a validated risk assessment instrument for successful PI prevention.
- The known PI prevention principles are appropriate for the pediatric population, but the implementation is slightly different for each developmental age.
- There are limited evidence-based guidelines for treatment and management of pediatric PI.
- Providers must work in interprofessional teams that integrate all stakeholders, including industry partners, to effectively prevent and treat pediatric PI. ●

REFERENCES

1. Frank G, Walsh KE, Wooton S, et al. Impact of a pressure injury prevention bundle in the Solutions for Patient Safety Network. Pediatr Qual Saf 2017;2(2):e013.
2. Curley MAQ, Hasbani NR, Quigley SM, et al. Predicting pressure injury risk in pediatric patients: the Braden QD scale. J Pediatr 2018;192:189-95.e2.
3. Cuddigan J, Holinger K, Brown C, Horslen SP. Pressure ulcers in infants and children. In: Cuddigan J, Ayello EA, Sussman C, Baranoski S, eds. Pressure Ulcer in America: Prevalence, Incidence, and Implications for the Future. Reston, VA: National Pressure Ulcer Advisory Panel; 2001:163-6.
4. National Pressure Ulcer Advisory Panel. Pressure Ulcers: Prevalence, Incidence, and Implications for the Future. 2nd ed. Pieper B, ed. Washington, DC: National Pressure Ulcer Advisory Panel; 2012.
5. National Pressure Ulcer Advisory Panel, European Pressure Ulcer Advisory Panel, Pan Pacific Pressure Injury Alliance. Prevention and Treatment of Pressure Ulcers: Clinical Practice Guideline. Haesler E, ed. Osborne Park, Western Australia: Cambridge Media; 2014.
6. Goudie A, Dynan L, Brady PW, Fieldston E, Brilli RJ, Walsh KE. Costs of venous thromboembolism, catheter-associated urinary tract infection, and pressure ulcer. Pediatrics 2015;136(3):432-9.
7. Baldwin KM. Incidence and prevalence of pressure ulcers in children. Adv Skin Wound Care 2002;15(3):121-4.
8. Barczak CA, Barnett R, Jarczynski E, Bosley LM. Fourth national pressure ulcer prevalence survey. Adv Wound Care 1997;10(4):18-26.
9. Dixon M, Ratliff C. Pediatric pressure ulcer prevalence—one hospital's experience. Ostomy Wound Manage 2005;51(6):44-50.
10. Groeneveld A, Anderson M, Allen S, et al. The prevalence of pressure ulcers in a tertiary care pediatric and adult hospital. J Wound Ostomy Continence Nurs 2004;31(3):108-20.
11. McLane KM, Bookout K, McCord S, McCain J, Jefferson LS. The 2003 National Pediatric Pressure Ulcer and Skin Breakdown Prevalence Survey: a multisite study. J Wound Ostomy Continence Nurs 2004;31(4):168-78.
12. Noonan C, Quigley S, Curley MA. Skin integrity in hospitalized infants and children. A prevalence survey. J Pediatr Nurs 2006;21(6):445-53.
13. Schindler CA, Mikhailov TA, Kuhn EM, et al. Protecting fragile skin: nursing interventions to decrease development of pressure ulcers in pediatric intensive care. Am J Crit Care 2011; 20(1):26-35.
14. Schlüer AB, Cignacco E, Muller M, Halfens RJ. The prevalence of pressure ulcers in four paediatric institutions. J Clin Nurs 2009;18:3244-52.
15. Schlüer AB, Schols JMGA, Halfens RJG. Risk and associated factors of pressure ulcers in hospitalized children over 1 year of age. J Spec Pediatr Nurs 2014;1:80-9.
16. Schlüer AB, Halfens RJ, Schols JGA. Pediatric pressure ulcer prevalence: a multicenter, cross-sectional, point prevalence study in Switzerland. Ostomy Wound Manage 2012;58(7):18-31.
17. Vangilder C, Amlung S, Harrison P, Meyer S. Results of the 2008-2009 International Pressure Ulcer Prevalence Survey and a 3-year, acute care, unit-specific analysis. Ostomy Wound Manage 2009;(November):39-46.
18. Neidig J, Kleiber C, Oppliger RA. Risk factors associated with pressure ulcers in the pediatric patient following open-heart surgery. Prog Cardiovasc Nurs 1989;4(July-September):99-106.
19. Willock J, Hughes J, Tickle S, Rossiter G, Johnson C, Pye H. Pressure sores in children—the acute hospital perspective. J Tissue Viability 2000;10(2):59-62.
20. Visscher M, King A, Nie AM, et al. A quality-improvement collaborative project to reduce pressure ulcers in PICUs. Pediatrics 2013;131(6):e1950-60.
21. Schindler CA, Mikhailov TA, Cashin SE, Malin S, Christensen M, Winters JM. Under pressure: preventing pressure ulcers in critically ill infants. J Spec Pediatr Nurs 2013;18(4):329-41.
22. Schmidt J, Berens R, Zollo M, Weisner M, Weigle C. Skin breakdown in children and high-frequency oscillatory ventilation. Arch Phys Med Rehabil 1998;79(12):1565-9.
23. Zollo MB, Gostisha ML, Berens R, Schmidt J, Weigle C. Altered skin integrity in children admitted to a pediatric intensive care unit. J Nurs Care Qual 1996;(December):62-7.
24. Curley MAQ, Quigley SM, Lin M. Pressure ulcers in pediatric intensive care: incidence and associated factors. Pediatr Crit Care Med 2003;4(3):284-90.
25. Visscher M, Taylor T. Pressure ulcers in the hospitalized neonate: rates and risk factors. Sci Rep 2014;4(1):7429.
26. Fujii K, Sugama J, Okuwa M, Sanada H, Mizokami Y. Incidence and risk factors of pressure ulcers in seven neonatal intensive care units in Japan: a multisite prospective cohort study. Int Wound J 2010;7(5):323-8.
27. Al-Ashhab S, Saleh MYN, Nabolsi M, Al-Horani E. Pressure ulcer prevalence among hospitalized children in Jordan. Jordan Med J 2013;47(3):241-52.

28. Razmus I, Bergquist-Beringer S. Pressure injury prevalence and the rate of hospital-acquired pressure injury among pediatric patients in acute care. J Wound Ostomy Continence Nurs 2017;44(2):110-7.
29. Peterson J, Adlard K, Walti BI, Hayakawa J, Mcclean E, Feidner SC. Clinical nurse specialist collaboration to recognize, prevent, and treat pediatric pressure ulcers. Clin Nurse Spec 2015;29(5):276-82.
30. Baharestani MM, Ratliff CR. Pressure ulcers in neonates and children: an NPUAP white paper. Adv Skin Wound Care 2007;20(4):208-20.
31. Kottner J, Wilborn D, Dassen T. Frequency of pressure ulcers in the paediatric population: a literature review and new empirical data. Int J Nurs Stud 2010;47(10):1330-40.
32. Miske LJ, Stetzer M, Garcia M, Stellar JJ. Airways and injuries: protecting our pediatric patients from respiratory device-related pressure injuries. Crit Care Nurs Clin N Am 2017; 29:187-204.
33. Baharestani MM, Black JM, Carville K, et al. Dilemmas in measuring and using pressure ulcer prevalence and incidence: an international consensus. Int Wound J 2009;6(2):97-104.
34. Delmore BA, Ayello EA. Pressure injuries caused by medical devices and other objects: a clinical update. Am J Nurs 2017;117(12):36-45.
35. Huffines B, Logsdon MC. The Neonatal Skin Risk Assessment Scale for predicting skin breakdown in neonates. Issues Compr Pediatr Nurs 1997;20(2):103-14.
36. Makimoto M, Kawasaki Y, Inomata S, Tamura K, Yoshida T. Early upper lip pressure ulcer in a preterm neonate. Pediatr Int 2017:1-2.
37. Johnson DE. Recognizing congenital pressure injuries. J Wound Ostomy Continence Nurs 2018;46(February):65-8.
38. Food and Drug Administration. Pediatric Expertise for Advisory Panels. 2003. www.fda.gov/MedicalDevices/DeviceRegulationandGuidance/GuidanceDocuments/ucm082185.htm. Last accessed May 22, 2019.
39. Fluhr JW, Darlenski R, Taieb A, et al. Functional skin adaptation in infancy—almost complete but not fully competent. Exp Dermatol 2010;19(6):483-92.
40. Hardin AP, Hackell JM. Age limit of pediatrics. Pediatrics 2017;140(3):e20172151.
41. World Health Organization. Preterm Birth. Fact Sheets. www.who.int/news-room/fact-sheets/detail/preterm-birth. 2018. Last accessed February 15, 2019.
42. Gomella TL, Cunningham MD, Eya FG, Tuttle DJ, eds. Gestational age and birthweight classification. In: Neonatology: Management, Procedures, On-Call Problems, Diseases, and Drugs. 7th ed. New York: McGraw-Hill; 2013.
43. Lund C, Kuller J, Lane A, Wright Lott J, Raines DA. Neonatal skin care: the scientific basis for practice. Neonatal Netw 1999;18(4):15-27.
44. Puttgen KB, Cohen BA. Neonatal dermatology. In: Cohen BA, ed. Pediatric Dermatology. 4th ed. Atlanta, GA: Elsevier Limited; 2013:14-67.
45. Kalia YN, Nonato LB, Lund CH, Guy RH. Development of skin barrier function in premature infants. J Invest Dermatol 1998;111(2):320-6.
46. Harpin VA, Rutter N. Barrier properties of the newborn infant's skin. J Pediatr 1983;102(3): 419-25.
47. Evans NJ, Rutter N. Development of the epidermis in the newborn. Biol Neonate 1986; 49(2):74-80.
48. Lo JS, Oriba HA, Maibach HI, Bailin PL. Transepidermal potassium ion, chloride ion, and water flux across delipidized and cellophane tape-stripped skin. Dermatology 1990;180(2):66-8.
49. Fluhr JW, Pfisterer S, Gloor M. Direct comparison of skin physiology in children and adults with bioengineering methods. Pediatr Dermatol 2000;17(6):436-9.
50. Lund CH, Nonato LB, Kuller JM, Franck LS, Cullander C, Durand DK. Disruption of barrier function in neonatal skin associated with adhesive removal. J Pediatr 1997;131(3):367-72.
51. Eichenfield LF, Hardaway CA. Neonatal dermatology. Curr Opin Pediatr 1999;11:471-4.
52. Chabrolle JP. Danger of iodine skin absorption in the neonate. J Pediatr 1978;93(1):158-9.
53. Schick JB, Milstein JM. Burn hazard of isopropyl alcohol in the neonate. Pediatrics 1981; 98(4):587-8.
54. Feinblatt BI, Aceto T, Beckhorn G, Bruck E. Percutaneous absorption of hydrocortisone in children. Am J Dis Child 1966;112:218-24.
55. Visscher M, DeCastro M, Combs L, et al. Effect of chlorhexidine gluconate on the skin integrity at PICC line sites. J Perinatol 2009;29(12):802-7.
56. Baranoski S, Ayello EA, Levine JM, LeBlanc K, Tomic-Canic M. Skin: an essential organ. In: Baranoski S, Ayello EA, eds. Wound Care Essentials: Practice Principles. 4th ed. Philadelphia, PA: Wolters Kluwer; 2016:52-81.
57. Fluhr JW, Darlenski R, Lachmann N, et al. Infant epidermal skin physiology: adaptation after birth. Br J Dermatol 2012;166(3):483-90.
58. Fox C, Nelson D, Wareham J. The timing of skin acidification in very low birth weight infants. J Perinatol 1998;18(4):272-5.
59. Manning MJ, Gauvreau K, Curley MA. Factors associated with occipital pressure ulcers in hospitalized infants and children. Am J Crit Care 2015;24(4):342-8.
60. Suddaby EC, Barnett S, Facteau L. Skin breakdown in acute care pediatrics. Pediatr Nurs 2005;31(2):132-48.
61. Vance DA, Demel S, Kirksey K, Moynihan M, Hollis K. A Delphi study for the development of an infant skin breakdown risk assessment tool. Adv Neonatal Care 2015;15(2):150-7.
62. August DL, Edmonds L, Brown DK, Murphy M, Kandasamy Y. Pressure injuries to the skin in a neonatal unit: fact or fiction. J Neonatal Nurs 2014;20(3):129-37.
63. Bonfim S De FSF, de Vasconcelos MGL, de Sousa NFC, da Silva DVC, Leal LP. Nasal septum injury in preterm infants using nasal prongs. Rev Lat Am Enfermagem 2014;22(5):826-33.
64. Newnam KM, McGrath JM, Estes T, Jallo N, Salyer J, Bass WT. An integrative review of skin breakdown in the preterm infant associated with nasal continuous positive airway pressure. J Obstet Gynecol Neonatal Nurs 2013;42(5):508-16.
65. Van der Kuip D, Opperbeck A. Current Risk Factors for Iatrogenic Pressure Injuries in Neonates: A Literature Review [bachelor's thesis]. Jyväskylä, Finland: JAMK University of Applied Sciences; 2016.
66. Nikolovski J, Stamatas GN, Kollias N, Wiegand BC. Barrier function and water-holding and transport properties of infant stratum corneum are different from adult and continue to develop through the first year of life. J Invest Dermatol 2008;128(7):1728-36.
67. Visscher MO, Chatterjee R, Ebel JP, LaRuffa AA, Hoath SB. Biomedical assessment and instrumental evaluation of healthy infant skin. Pediatr Dermatol 2002;19(6):473-81.
68. Visscher MO, Chatterjee R, Munson KAN, Pickens WL, Hoath SB. Changes in diapered and nondiapered infant skin over the first month of life. Pediatr Dermatol 2000;17(1):45-51.
69. Malloy-McDonald MB. Skin care for high-risk neonates. J Wound Ostomy Continence Nurs 1995;22(4):177-82.
70. Kottner J, Hauss A, Schlüer A-B, Dassen T. Validation and clinical impact of paediatric pressure ulcer risk assessment scales: a systematic review. Int J Nurs Stud 2013;50:807-18.
71. Curley MAQ, Razmus IS, Roberts KE, Wypij D. Predicting pressure ulcer risk in pediatric patients: the Braden Q scale. Nurs Res 2003;52(1):22-33.
72. Noonan C, Quigley S, Curley M. Using the Braden Q scale to predict pressure ulcer risk in pediatric patients. J Pediatr Nurs 2011;26(6):566-75.
73. Willock J, Baharestani MM, Anthony D. The development of the Glamorgan Paediatric Pressure Ulcer Risk Assessment Scale. J Wound Care 2009;18(1):17-21.
74. Sterken DJ, Mooney J, Ropele D, Kett A, Vander Laan KJ. Become PPUPET master: mastering pressure ulcer risk assessment with the Pediatric Pressure Ulcer Prediction and Evaluation Tool (PPUPET). J Pediatr Nurs 2015;30(5):598-610.
75. Foster KL, Bergerhofer LD, Smith JB, Fix MM, Olney A, Sherman A. SIRA+P: development and testing. J Pediatr Nurs 2017;34:65-71.
76. Ratliff CR, Droste LR, Bonham P, et al. WOCN 2016 Guideline for Prevention and Management of Pressure Injuries (Ulcers): an executive summary. J Wound Ostomy Continence Nurs 2017;44(3):241-6.
77. Barnes S. The use of a pressure ulcer risk assessment tool for children. Nurs Times 2004; 100(14):56-8.
78. McGraw-Hill. Dictionary of Engineering. 2nd ed. New York: McGraw-Hill Companies, Inc; 2003.
79. Levy A, Kopplin K, Gefen A. Adjustability and adaptability are critical characteristics of pediatric support surfaces. Adv Wound Care 2015;4(10):615-22.
80. Levy A, Kopplin K, Gefen A. Device-related pressure ulcers from a biomechanical perspective. J Tissue Viability 2016;26(1):57-68.
81. King A, Stellar K, Blevins A, Shah K. Dressings and products in pediatric wound care. Adv Wound Care (New Rochelle) 2014;3(4):324-34.
82. Buettiker V, Hug MI, Baenziger O, Meyer C, Frey B. Advantages and disadvantages of different nasal CPAP systems in newborns. Intensive Care Med 2004;30(5):926-30.
83. Kuo CY, Wootten CT, Tylor DA, Werkhaven JA, Huffman KF, Goudy SL. Prevention of pressure ulcers after pediatric tracheotomy using a Mepilex Ag dressing. Laryngoscope 2013;123(12):3201-5.
84. Visscher M, Narendran V. The ontogeny of skin. Adv Wound Care 2014;3(4):291-303.
85. Jaryszak EM, Shah RK, Amling J, Peña MT. Pediatric tracheotomy wound complications. Arch Otolaryngol Neck Surg 2011;137(4):363.
86. Robertson NJ, McCarthy LS, Hamilton PA, Moss AL. Nasal deformities resulting from flow driver continuous positive airway pressure. Arch Dis Child Fetal Neonatal Ed 1996;75(3): F209-12.
87. Yong SC, Chen SJ, Boo NY. Incidence of nasal trauma associated with nasal prong versus nasal mask during continuous positive airway pressure treatment in very low birthweight infants: a randomised control study. Arch Dis Child Fetal Neonatal Ed 2005;90(6): F480-3.

88. Turnage-Carrier C, McLane KM, Gregurich MA. Interface pressure comparison of healthy premature infants with various neonatal bed surfaces. Adv Neonatal Care 2008;8(3):176-84.
89. Chen CY, Chou AK, Chen YL, Chou HC, Tsao PN, Hsieh WS. Quality improvement of nasal continuous positive airway pressure therapy in neonatal intensive care unit. Pediatr Neonatol 2017;58(3):229-35.
90. McCord S, McElvain V, Sachdeva R, Schwartz P, Jefferson LS. Risk factors associated with pressure ulcers in the pediatric intensive care unit. J Wound Ostomy Continence Nurs 2004; 31(4):179-83.
91. Schlüer AB, Schols JMGA, Halfens RJG. Pressure ulcer treatment in pediatric patients. Adv Skin Wound Care 2013;26(11):504-10.
92. Loudet CI, Marchena MC, Maradeo MR, et al. Reducing pressure ulcers in patients with prolonged acute mechanical ventilation: a quasi-experimental study. Rev Bras Ter Intensiva 2017;29(1).
93. Singh CD, Anderson C, White E, Shoqirat N. The impact of pediatric pressure injury prevention bundle on pediatric pressure injury rates: a secondary analysis. J Wound Ostomy Continence Nurs 2018;45(3):209-12.
94. Boesch RP, Myers C, Garrett T, et al. Prevention of tracheostomy-related pressure ulcers in children. Pediatrics 2012;129(3):e792-7.
95. Razmus I, Bergquist-Beringer S. Pressure ulcer risk and prevention practices in pediatric patients: a secondary analysis of data from the National Database of Nursing Quality Indicators. Ostomy Wound Manage 2017;63(2):26-36.
96. Murray JS, Noonan C, Quigley S, Curley MAQ. Medical device-related hospital-acquired pressure ulcers in children: an integrative review. J Pediatr Nurs 2013;28(6):585-95.
97. Nurses on Boards Coalition. Improving the health of communities and the nation. www.nursesonboardscoalition.org. 2017. Last accessed October 30, 2017.
98. National Pressure Ulcer Advisory Panel. Mission. www.npuap.org/about-us/mission. Last accessed February 27, 2019.
99. Posthauer ME, Jordan RS, Sylvia CJ. Support surface initiative: terms, definitions and patient care. Adv Skin Wound Care 2006;19(9):487-9.
100. Delmore B, Cohen JM, O'Neill D, Chu A, Pham V, Chiu E. Reducing postsurgical wound complications: a critical review. Adv Skin Wound Care 2017;30(6):272-85.
101. Amaya R. Little patients, big outcomes: the role of MEDIHONEY® in pediatric wound care. Ostomy Wound Manage 2014;60(8):8-10.
102. Amaya R. Safety and efficacy of active Leptospermum honey in neonatal and paediatric wound debridement. J Wound Care 2015;24(3):95-103.
103. Mohr LD, Reyna R, Amaya R. Neonatal case studies using active Leptospermum honey. J Wound Ostomy Continence Nurs 2014;41(3):213-8.
104. Garvin G. Wound and skin care for the PICU. Crit Care Nurs Q 1997;20(1):62-71.
105. Lund CH, Osborne JW. Validity and reliability of the Neonatal Skin Condition score. J Obstet Gynecol Neonatal Nurs 2004;33(3):320-7.
106. Galvin PA, Curley MAQ. The Braden Q+P: a pediatric perioperative pressure ulcer risk assessment and intervention tool. AORN J 2012;96(3):261-70.
107. Pickersgill J. Taking the pressure off. Paediatr Nurs 1997;9(8):25-7.
108. Cockett A. Paediatric pressure sore risk assessment. J Tissue Viability 1998;8(1):30.
109. Bedi A. A tool to fill the gap: developing a wound risk assessment chart for children. Prof Nurse 1993;9(2):112-20.
110. Waterlow J. Pressure sore risk assessment in children. Paediatr Nurs 1997;9(6):21-4.
111. Gordon MD. Psychometric Evaluation of a New Pressure Ulcer Skin Risk Assessment Scale for the Pediatric Burn Patient. Galveston, TX: University of Texas Medical Branch; 2009.
112. Olding L, Patterson J. Growing concern. Nurs Times 1998;94(38):74-9.

For more than 149 additional continuing education articles related to Skin and Wound Care topics, go to NursingCenter.com/CE.

CE CONNECTION

CONTINUING MEDICAL EDUCATION INFORMATION FOR PHYSICIANS

Lippincott Continuing Medical Education Institute, Inc., is accredited by the Accreditation Council for Continuing Medical Education to provide continuing medical education for physicians.

Lippincott Continuing Medical Education Institute, Inc., designates this journal-based CME activity for a maximum of 1 *AMA PRA Category 1 Credit™*. Physicians should claim only the credit commensurate with the extent of their participation in the activity.

PROVIDER ACCREDITATION INFORMATION FOR NURSES

Lippincott Professional Development will award 1.5 contact hours for this continuing nursing education activity.

LPD is accredited as a provider of continuing nursing education by the American Nurses Credentialing Center's Commission on Accreditation.

This activity is also provider approved by the California Board of Registered Nursing, Provider Number CEP 11749 for 1.5 contact hours. LWW is also an approved provider by the District of Columbia, Georgia, and Florida CE Broker #50-1223.

OTHER HEALTH PROFESSIONALS

This activity provides ANCC credit for nurses and *AMA PRA Category 1 Credit™* for MDs and DOs only. All other healthcare professionals participating in this activity will receive a certificate of participation that may be useful to your individual profession's CE requirements.

CONTINUING EDUCATION INSTRUCTIONS

- Read the article beginning on page 394. For nurses who wish to take the test for CNE contact hours, visit http://nursing.ceconnection.com. For physicians who wish to take the test for CME credit, visit http://cme.lww.com. Under the Journal option, select *Advances in Skin and Wound Care* and click on the title of the CE activity.
- You will need to register your personal CE Planner account before taking online tests. Your planner will keep track of all your Lippincott Professional Development online CE activities for you.
- There is only one correct answer for each question. A passing score for this test is 13 correct answers. If you pass, you can print your certificate of earned contact hours or credit and access the answer key. Nurses who fail have the option of taking the test again at no additional cost. Only the first entry sent by physicians will be accepted for credit.

Registration Deadline: August 31, 2021 (physicians); September 3, 2021 (nurses).

PAYMENT

- The registration fee for this CE activity is $17.95 for nurses; $22.00 for physicians.