CLINICAL MANAGEMENT

extra

Risk Factors for Pressure Ulcers Including Suspected Deep Tissue Injury in Nursing Home Facility Residents: Analysis of National Minimum Data Set 3.0





Hyochol Ahn, PhD, ARNP, ANP-BC • Assistant Professor • University of Florida College of Nursing • Gainesville **Linda Cowan, PhD, ARNP, FNP-BC, CWS •** Research Health Scientist • North Florida/South Georgia Veterans Health System • Gainesville

Cynthia Garvan, PhD • Research Associate Professor • University of Florida College of Nursing • Gainesville

Debra Lyon, PhD, RN, FAAN • Professor and Executive Associate Dean • University of Florida College of Nursing • Gainesville

Joyce Stechmiller, PhD, ACNP-BC, FAAN • Department Chair and Associate Professor • Department of Biobehavioral Nursing Science, University of Florida College of Nursing • Gainesville

All authors, staff, and planners, including spouses/partners (if any), in any position to control the content of this CME activity have disclosed that they have no financial relationships with, or financial interests in, any commercial companies pertaining to this educational activity.

To earn CME credit, you must read the CME article and complete the quiz and evaluation on the enclosed answer form, answering at least 13 of the 18 questions correctly.

This continuing educational activity will expire for physicians on April 30, 2017, and for nurses on April 30, 2018.

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.

Acknowledgment: The authors disclose that this study was funded by the University of Florida College of Nursing, Gainesville, Florida. The sponsor had no role in the design, methods, data collection, analysis, or preparation of this manuscript. The authors thank Debra McDonald for her work on the project.

PURPOSE:

To provide information on risk factors associated with pressure ulcers (PrUs), including suspected deep tissue injury (sDTI), in nursing home residents in the United States.

TARGET AUDIENCE:

This continuing education activity is intended for physicians and nurses with an interest in skin and wound care.

OBJECTIVES:

After participating in this educational activity, the participant should be better able to:

- 1. Examine the literature related to risk factors for the development of PrUs.
- 2. Compare risk factors associated with the prevalence of PrUs and sDTI from the revised Minimum Data Set 3.0 2012 using a modified Defloor's conceptual model of PrUs as a theoretical framework.

ABSTRACT

OBJECTIVE: This study aims to characterize and compare risk factors associated with pressure ulcers (PrUs), including suspected deep tissue injury (sDTI), in nursing home (NH) residents in the United States.

DESIGN: Secondary analysis of the 2012 Minimum Data Set (MDS 3.0).

SETTING: Medicare- or Medicaid-certified NHs in the United States. **PARTICIPANTS:** Nursing home residents (n = 2,936,146) 18 years or older with complete PrU data, who received comprehensive assessments from January to December 2012.

MEASUREMENTS: Pressure ulcer by stage was the outcome variable. Explanatory variables (age, gender, race and ethnicity, body mass index, skin integrity, system failure, disease, infection, mobility, and cognition) from the MDS 3.0 were aligned with the 4 elements of Defloor's conceptual model: compressive forces, shearing forces, tissue tolerance for pressure, and tissue tolerance for oxygen.

RESULTS: Of 2,936,146 NH residents who had complete data for PrU, 89.9% had no PrU; 8.4% had a Stage 2, 3, or 4 or unstagable PrU; and 1.7% had an sDTI. The MDS variables corresponding to the 4 elements of Defloor's model were significantly predictive of both PrU and sDTI. Black residents had the highest risk of any-stage PrU, and Hispanic residents had the highest risk of sDTI. Skin integrity, system failure, infection, and disease risk factors had larger effect sizes for sDTI than for other PrU stages.

conclusions: The MDS data support Defloor's model and inform clinicians, educators, researchers, and policymakers on risk factors associated with PrUs and sDTI in NH residents in the United States participating in Medicare and Medicaid.

KEYWORDS: pressure ulcer, deep tissue injury, nursing home, Minimum Data Set, Defloor model

ADV SKIN WOUND CARE 2016;29:178-90.

INTRODUCTION

Each year in the United States, more than 2.5 million people experience pressure ulcers (PrUs), which occur across all healthcare settings: 0.4% to 38% in acute care, 0% to 17% in home care, and 2% to 24% in long-term-care facilities. The prevalence of PrUs is a major threat to public health and the US healthcare system. Because of the increasing occurrence

and severity of PrUs, in 2007 the National Pressure Ulcer Advisory Panel revised the classifications of the 4 stages of PrU (I, II, III, and IV) and added 2 more classifications: "unstageable" and "suspected deep tissue injury" (sDTI). The first type is termed "unstageable" because eschar or slough obscures the depth of the pressure-related injury, which is characterized by full-thickness tissue loss where the full depth (Stage III vs IV) is unable to be determined.^{4,5} The second type, sDTI, is due to a pressure-related injury under intact skin. 4,5 This internal injury originates in the muscular tissue that overlies bony prominences as a result of soft tissue (skeletal muscle and fat tissues) deformations and progresses outward⁶ until it appears on the skin surface as a purple or maroon localized area of discolored intact skin or blood-filled blister due to damage of underlying soft tissue from pressure and/or shear.⁵ The area may be preceded by tissue that is painful, firm, mushy, boggy, or warmer or cooler as compared with adjacent tissue. Suspected DTI may be difficult to detect in individuals with dark skin tones. Evolution may include a thin blister over a dark wound bed. These serious wounds may further evolve and be covered by thin eschar. Progression may be rapid, exposing additional layers of tissue even with optimal treatment.4,5,7 Suspected DTI may precede the development of an open Stage III-IV PrU,5 which commonly occurs in points of high pressure, such as the sacrum, buttocks, and heels. 1,4,5,8-10

Considerable research is ongoing in understanding the mechanisms underlying the onset and progression of sDTI. ^{6,11} Prior research has focused on ischemic/hypoxic damage, ischemia-reperfusion injury, structural damage to cells, and interference with lymphatic and interstitial fluid drainage. ⁶ However, recent studies by Slomka et al ⁶ have shown that the involvement of sustained deformation-related events at the cellular level is directly responsible for the distortion of cells, the deformation of plasma membranes leading to increased membrane permeability, impairment of cellular control mechanisms, and eventual cell death caused by loss of cell homeostasis leading to sDTI.

Skin Changes at Life's End (SCALE) is a term introduced in 2009 in reference to unusual wounds that occur at the end of life. The wound types include PrUs, DTI, unavoidable pressure injury, and mottling that appear in individuals who are in the process of dying. ^{12,13} Edsberg et al ¹³ identify some of

the SCALE wounds as examples of unavoidable pressure injuries. In persons who are actively dying, SCALE pressure injuries may occur because of nonmodifiable intrinsic and extrinsic factors that make the individual at risk for pressure, shear, friction, deformation, hemodynamic instability, ischemia, and or reperfusion injury.

Pressure ulcer risk factors have been mentioned in the scientific literature for hundreds of years. 1-45 Lyder 9 reported more than 100 PrU risk factors identified from the scientific literature in 2003. Berlowitz et al³⁶ validated a risk adjustment model for the development of PrUs in nursing home (NH) residents using the Minimum Data Set (MDS) from 1998. Results of their logistic regression model related NH resident characteristics and the development of PrUs. The highest risk factors identified by Berlowitz et al¹⁹ include age, male gender, nonwhite ethnicity, nonroutine assessment, bed mobility selfperformance, transfer self-performance, bedfast, bladder incontinence, deterioration in cognitive status, diabetes, peripheral vascular disease, hip fracture within 180 days, body mass index (BMI), end-stage disease, Stage I PrU, history of resolved PrU, and edema. In comparison and more recently, Fogerty et al³⁴ identified the highest 45 risk factors associated with PrU prevalence among a sample of 6,610,787 patients hospitalized in acute care settings. Common risk factors include advanced age, immobility, friction, shear, poor nutrition, excessive moisture and incontinence, altered level of consciousness, poor perfusion, certain skin infections, and comorbid conditions. Furthermore, Fogerty et al³⁴ found that persons who were identified as black were more than twice as likely (odds ratio [OR], 2.3) to develop PrUs than those identified as being white, and age was an interacting variable, so that as black persons increase in age, their risk of PrUs increased more than a white person's risk of PrUs as they aged.

Defloor³⁸ created a theoretical model of PrU that contained 4 main elements: (1) compressive forces, (2) shearing forces, (3) tissue tolerance for pressure, and (4) tissue tolerance for oxygen. The first element, compressive forces, relates to when mechanical loads exert pressure on a patient's tissue that is compressed between a surface, such as a chair, and the bony prominences of the human body, such as the sacrum or buttocks. These mechanical loads increase the risk of PrUs, especially when they exist for an extended period on patients who are immobile and in sitting positions. The second element of Defloor's model, shearing forces, relates to sliding forces that occur when a human body is moved along a surface. In the clinical setting, shearing forces are best exemplified when a nurse slides a patient up in bed or transfers a patient from the bed to the chair—actions that entail the patients' continual contact with a surface throughout the motion and result in separation of skin

layers. Defloor³⁸ included friction along with shearing forces, although friction is related to more superficial abrasive or rubbing forces and may not result in separation between skin layers. The third element, tissue tolerance for pressure, relates to the fact that external factors, such as mechanical loads and/or moisture, interact with internal factors for the fourth element, tissue tolerance for oxygen, such as low arteriolar pressure or poor oxygenation, leading to an increased risk of PrUs.³⁸ For example, if tissue tolerance is low, a shorter duration of pressure can damage tissues, and likewise, if tissue tolerance is high, tissue damage may occur only with longer duration of sustained pressures.³⁸ Although no data exist that have precisely timed PrU development, Slomka et al⁶ and Gefen¹¹ have shown that PrUs may develop in high-risk individuals in less than 1 hour of sustained pressure to vulnerable body tissue.

In Defloor's³⁸ conceptual model for PrU, several factors affect tissue tolerance for pressure, and these are age, stress, cognition, sensory awareness to pain and discomfort (acuity), dehydration, and tissue mass, as well as protein and vitamin C deficiency. Similarly, factors that affect tissue tolerance for oxygen are temperature, medication, protein deficiency, smoking, blood pressure, and presence of certain diseases (ie, those that affect oxygen supply, reactive hyperemia, and vascular occlusion). Studies suggest that the development of PrUs may result from major alterations in the normal functioning of these human mechanisms. Pressure ulcers may also develop from the cumulative effects of minor changes in several of these mechanisms/factors, particularly in combination with sustained external pressure (forces perpendicular to the skin), and/or the presence of external moisture (fecal or urinary incontinence, excess perspiration), and/or friction and shear forces (slipping, sliding, or rubbing forces parallel to the skin). 11,38

In general, studies suggest PrUs will develop because of major or continual minor alterations in the normal functioning of human mechanisms in combination with sustained external pressure, external moisture, and/or friction/shear forces. 11,38 With respect to frequency, an analysis of the International Pressure Ulcer Prevalence survey 2006-2009 showed that the overall and nosocomial PrU prevalence decreased by approximately 1% in 2009 after remaining constant in earlier years⁴¹; however, the proportion of ulcers identified as sDTI had increased 3 times, to 9%, in 2009 and was more prevalent than either a Stage III or Stage IV PrU. Patients with sDTIs tended to be older than patients with Stage III or IV ulcers, and unstageable ulcers tended to occur in people with a slightly lower BMI than the surveyed population. In attempting an explanation of these data, the researchers suggested that sDTIs were being identified more frequently because of staff education of staging definitions and that actual prevalence had not increased.⁴¹

The MDS, a federally mandated tool, guides the regular assessment of all residents in NHs certified to participate in US Medicare and Medicaid programs. The revised Section M: Skin Conditions of MDS 3.0 now includes at-risk status and ulcer characteristics, including stage, size, history, tissue type, number of ulcers, and ulcer identification. For the first time, the revised Nursing Home MDS 3.0 added a section related to the presence/absence and number of sDTIs. It is anticipated that the revised MDS 3.0 will change PrU tracking and recording in long-term care and impact the generation of quality indicators for skin conditions, including PrUs. More important, the MDS data may provide further understanding of whether PrUs are actually a reflection of quality care in long-term-care facilities in the United States.

Many variables aligned with the elements of the Defloor model were also available in the MDS 3.0. Thus, the objectives of this study were to characterize and compare risk factors associated with the prevalence of PrUs and sDTI in NH residents in a secondary analysis of the revised MDS 3.0 2012 national data set using a modified Defloor's³⁸ conceptual model (Figure) of PrUs as a theoretical framework.

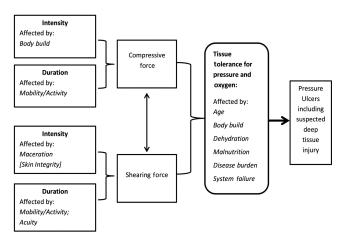
METHODS

Data Source

Data were drawn from the national MDS 3.0 assessment data from January to December 2012. The MDS data are mandatory in all NHs certified to participate in Medicaid and Medicare.

Figure.

MODIFIED DEFLOOR'S CONCEPTUAL MODEL OF PRESSURE ULCER DEVELOPMENT INCLUDING SUSPECTED DEEP TISSUE INJURY



Details about the methods of the MDS assessment data have been published previously. 42,43 This study used the admission assessment (66.34%) or first comprehensive assessment (27.35%) or significant change in status assessment (6.31%) for residents in each NH. The exclusion criteria were age younger than 18 years, missing age data, missing data on PrU status, and having a Stage I PrU. Stage I PrUs were excluded because this stage of pressure-related injury is considered to be reversible, and literature suggests Stage I PrUs are frequently misidentified. The MDS 3.0 national data set included 2,936,146 residents who met the study criteria. Approval for this study was obtained from the University of Florida Health Science Center Institutional Review Board prior to commencement.

Variables

Outcome Variables

The outcome variables of interest were (1) the presence of sDTI; (2) a Stage II, III, or IV or unstageable PrU (excluding sDTI); and (3) a Stage II, III, or IV or unstageable PrU or the presence of sDTI (all PrUs, including sDTI). In the validation study of the MDS 3.0, the average for interrater agreement in identifying sDTI was 0.94.⁴⁴

Risk Factors (Explanatory Variables)

The risk factors used in the MDS analysis were classified as resident characteristics (age, gender, race, ethnicity, and BMI) and factors that influence skin integrity, including nutritional factors (anemia, malnutrition, dehydration, urinary incontinence, and bowel incontinence), system failure (comatose, heart failure, respiratory failure, end-stage renal disease, cirrhosis), disease burden (diabetes, multiple sclerosis, coronary artery disease [CAD], chronic obstructive pulmonary disease, peripheral vascular disease [venous and arterial]), infection (septicemia, pneumonia, urinary tract infection [UTI], multidrug resistant organism), mobility (hemiplegia, paraplegia, quadriplegia, hip fracture, activities of daily living [ADLs] impairment), sensory alterations, and cognition. These risk factors were selected a priori based on the hypothesized association with PrU and sDTI supported by extant literature and Defloor's model. Table 1 shows ORs found in previous literature and shows how selected MDS variables correspond with Defloor's model.

Analysis

To examine group differences on study variables, the groups "no PrU" and "any PrU" (including Stages II-IV, unstageable, and sDTI) were compared using descriptive statistics. Descriptive statistics for the "sDTI only group" were also calculated. Descriptive statistics were presented as mean or percentages. Logistic regression was used to compute the unadjusted OR and

Table 1. RISK FACTORS FOR PRUs WITH CORRESPONDENCE TO DEFLOOR'S MODEL AND ODDS RATIOS FOUND IN PREVIOUS LITERATURE $^{32-36}$

Variables	Odds Ratios Found in Previous Literature ^{32–34}	Corresponds to Defloor's Model
Age	Age >75 y = OR, 12.63 ³² Age (per additional 10 y) ³³ = OR, 1.1 ³⁴	Tissue tolerance for pressure: "the pressure distribution capacity of tissue correlates negatively with age," (aging leads to decrease in muscle and fat mass, altered cell senescence, altered nutrient transport, and oxygenation) 10,11,33-36
Gender	Female gender = OR, 0.84 ³² Male gender = OR, 1.4 ³⁴	Tissue changes relating to age and relationship to compressive and shearing forces
Race and ethnicity	Black = OR, 2.3; Asian = OR, 1.01; Hispanic = OR, 1.37; other = OR, 1.11 ³² ; nonwhite ethnicity = OR, 1.3 ³⁴	This is an additional risk factor not specifically listed in the Defloor model, but impacting early identification of tissue changes relating to both compressive and shearing forces
ВМІ	OR, 0.95 ³⁴	Compressive forces: Intensity affected by body weight/build; "with cachectic persons, higher peak pressures are measured at the skin surface than those of normal weight For obese persons, greater areas of increased pressure but lower peak pressures are recorded." Tissue tolerance for pressure affected by body build (amount of subcutaneous tissue mass for "padding") 11,33,35,36
Malnutrition	OR, 9.18 for poor nutrition ³²	Tissue tolerance for pressure and oxygen (malnutrition alters cellular senescence, nutrient and waste transport, and cellular repair). 11,33 "Long-term protein deficiency causes ar edema as a result of hypoalbuminemia. This edema decreases oxygen supply to tissue "34,36"
Dehydration	No OR listed in Fogerty et al ³² article	Shearing forces and tissue tolerance for pressure and oxygen: dehydration shrinks cells and alters cellular senescence, nutrient and waste transport, cellular repair, and tolerance to tissue deformation and shearing forces. "Dehydration decreases skin elasticity and increases the capacity for deformation of the tissue, increasing the risk of damage."
Anemia	OR, 2.62 for anemia ³²	Tissue tolerance for pressure and oxygen: anemia reduces oxygen levels in the blood and oxygen tissue transport, which may result in cellular hypoxia and alter cellular senescence, nutrient and waste transport, cellular repair, and tolerance to tissue deformation 11,33,36
Urinary or fecal incontinence	Bladder incontinence = OR, 1.4 ³⁴	Shearing forces—intensity: maceration and friction (incontinence alters skin integrity, making tissue more susceptible to damage by pressure and friction or shearing forces) ³⁶
Neuro (coma)	Deterioration in cognitive status = OR, 1.3 ³⁴	Coma impacts duration of compressive and shearing forces by impacting sensory-motor activity and mobility 11,36
		"A comatose state ablates the ability of the individual to move independently, making them vulnerable to PrUs unless they are repositioned" 11
		(continues

Table 1. RISK FACTORS FOR PRUS WITH CORRESPONDENCE TO DEFLOOR'S MODEL AND ODDS RATIOS FOUND IN PREVIOUS LITERATURE, 32-36 CONTINUED

Variables	Odds Ratios Found in Previous Literature 32-34	Corresponds to Defloor's Model
Heart failure/CHF respiratory failure Renal failure Cirrhosis Edema ³⁴	CHF = OR, 2.63 ³² Respiratory failure = OR, 4. ³² Acute renal failure = OR, 4.16 ^{32,34} OR, 1.3 ³⁴	All of these biological system failures impact tissue oxygenation and tissue tolerance to pressure and shear forces, as well as contribute to skin failure. ¹⁰ Cardiac failure and respiratory failure play a part in reduced oxygen supply, delayed reactive hyperemia, and accelerated vascular occlusion. ³⁶ Renal and liver failure results in alterations in cellular nutrient and waste transport. Both result in loss of tissue protection during pressure and shearing forces and greater risk of tissue ischemia ³⁶
Diabetes Coronary artery disease Vascular disease Chronic obstructive pulmonary disease	Diabetes with complication = OR, 2.63^{32} ; OR, 1.3^{34} Other circulatory disease = OR, 1.68^{32} Peripheral atherosclerosis = OR, 2.09^{32} ; OR, 1.0^{34} COPD = OR, 1.62^{32}	All of these biological system failures impact tissue oxygenation and tissue tolerance to pressure and shear forces as well as contribute to skin failure. Persons with diabetes display a delayed reactive hyperemia, reduced sympathetic nervous system function, increased blood viscosity, and thickening of basement membrane in capillaries. CAD, vascular disease, and COPD directly impact tissue oxygenation by reduced oxygen supply, delayed reactive hyperemia, and accelerated vascular occlusion ³⁶
Pneumonia UTI Septicemia Multidrug-resistant organism	Pneumonia = OR, 3.47^{32} UTI = OR, 7.17^{32} Septicemia = OR, 9.78^{32} Sepsis = OR, 11.3^{11}	Tissue tolerance to pressure and oxygenation, and tolerance to friction and shear forces ¹⁰ "Infection is the invasion and multiplication of microorganisms within the body, causing cellular injury due to competitive metabolism, toxins, intracellular replication, or antigen-antibody response" ¹¹
MS		Decreased mobility results in increases in duration and intensity of compressive, as well as shearing forces and friction. MS results in decreased mobility, as well as "intermittent or sustained involuntary muscle activation that may be painful and deforming. Involuntary spastic movements can create shear and contribute to the development of PrU".11
Hemiplegia Paraplegia Quadriplegia	Paralysis = OR, 10.30 ³²	Paralysis results in increased compressive forces (both intensity and duration) and shearing forces (and friction) due to sensory motor deficits, as well as decreased tissue tolerance to pressure due to tissue changes that occur below the level of paralysis 11,36
Hip fracture	Hip fracture within 180 d ³⁴	Hip fracture results in decreased mobility, which results in increased compressive forces (both intensity and duration) and shearing forces (and friction) due to sensory-motor changes, as well as pain ^{34,36}
ADL impairment	Activity was strongly associated with risk of PrU in previous literature, such that as activity increases, PrU risk decreases ^{32,34} Bed mobility self-performance = OR, 1.3 ³⁴ Bedridden = OR, 1.4 ³⁴	Decreased mobility/activity results in increased compressive forces (both intensity and duration) and shearing forces (and friction) ³⁶
Cognition	Senility = OR, 4.84 ³²	Senility is associated with decreased mobility (or increased repetitive motions)

associated 95% confidence interval (CI) of risk of any PrU (comparison is no PrU) due to age, sex, race and ethnicity, BMI, skin integrity, biological system failure, disease burden, infection, mobility, and cognition. Odds ratios and associated 95% CIs were then calculated for the risk of PrU excluding sDTI (comparison is no PrU). Odds ratios and associated 95% CIs were also calculated for the risk of sDTI only (comparison is no PrU). The SAS version 9.3 software (SAS Institute Inc, Cary, North Carolina) was used for all analyses. To examine whether resident characteristics were differentially associated with PrU excluding sDTI or sDTI only, those residents with any PrUs were analyzed. In the subset of NH residents with any PrU, 2 groups were compared: PrU excluding sDTI and sDTI only. χ^2 analyses were used to compare these 2 groups on resident characteristics. Multiple logistic stepwise regression was used to determine the risk factors most associated with the prevalence of PrUs.

RESULTS

Of the 2,936,146 NH residents included in the analysis, 89.9% did not have a PrU, whereas 10.1% had a PrU (4.9% had a Stage II PrU, 1.0% had a Stage III PrU, 1.8% had an unstageable PrU, 0.7% had a Stage IV PrU, and 1.7% had sDTI). Table 2 describes the overall sample, the subgroup who had no PrU, the subgroup who had a PrU including sDTI, and the subgroup that had only sDTIs. Table 2 also shows the percentage of missing data for each variable. The majority of residents were older than 75 years (65%), female (65%), and white (81%) and had normal, overweight, or obese BMI (94%). Among factors influencing the skin integrity variables, urinary and bowel incontinence (42% and 36%, respectively) and anemia (29%) were conditions with the highest prevalence in the sample population. Among the biological system failure variables, heart failure (20%) and end-stage renal disease (12%) were the most prevalent, whereas among the disease variables, diabetes (33%), CAD (23%), and chronic obstructive pulmonary disease (22%) were the most prevalent. Almost 13% of residents had a UTI, and 13% had severe mobility limitations due to paralysis or hip fracture. Sensory alterations included deficits of hearing (8%), speech (13%), and vision (10%) and inability to understand others (14%). Approximately 30% of residents were reported to have severe cognitive impairment, and 21% were reported to have moderate impairment. Forty-one percent of residents were on insulin, and 26% were on an anticoagulant.

Table 3 presents the magnitude of risk of the prevalence of all PrUs (including sDTI), only PrU (excluding sDTI), and only sDTIs. The youngest group of NH residents (aged 18–39 years) had the highest risk of PrU (13%). Males had a higher risk of both PrU and sDTI. Black residents and those who were very severely underweight had the highest risk of any-stage PrU.

Among the skin integrity variables, ORs for both PrU and sDTI were greater than 2 for residents with malnutrition and dehydration. Among the biological system failure variables, ORs for both PrUs and sDTIs were greater than 2 for comatose residents and residents with respiratory failure. Among disease factors, the ORs for all PrUs were greater than 2.0 for multiple sclerosis. The ORs for any PrUs were close to or greater than 2.0 for all of the measured infection variables. Among the mobility factors, the OR for any PrUs for paraplegia was 6.3, and the OR for quadriplegia was 3.4. Mean ADL impairment was higher in both the PrU and sDTI groups.

In comparing the magnitude of risk of only PrU (excluding sDTI) and only sDTI, the risk of sDTI (OR, 1.17) was larger for NH residents older than 75 years than for all other PrU stages (OR, 0.70). The sDTI risk effect size was larger for Asian (OR, 1.11) and Hispanic (OR, 1.53) residents than it was for white residents. The sDTI ORs were larger than the ORs for all other stages of PrUs and for all the skin integrity variables. Odds ratios were larger for all of the system failure variables in the sDTI group compared with all other stages of PrU. Among the disease variables, ORs were larger for CAD and vascular disease in the sDTI group compared with all other stages of PrUs. Among the infection variables, ORs were larger for septicemia, pneumonia, and UTI in the sDTI group compared with all other PrUs. Among other conditions, OR was higher for sDTI than all other PrUs if hip fracture was present (OR for sDTI 2.88 vs 1.73 for all other PrUs).

In a multiple logistic stepwise regression to model presence/ absence of PrU, the following risk factors were retained in all 3 models (ie, any PrU, any PrU except sDTI, sDTI only): race, sex, age, BMI, anemia, malnutrition, dehydration, heart failure, respiratory failure, end-stage renal disease, diabetes, multiple sclerosis, peripheral vascular disease, cirrhosis, septicemia, pneumonia, multidrug resistant organism, UTI, bowel continence, hip fracture, mobility (ADL impairment), and impaired cognition. In addition, the "any PrU" and "sDTI only" models retained the multiple sclerosis and CAD variables. The nature of association of risk was the same in the adjusted and unadjusted models.

DISCUSSION

This prevalence study is the first to provide a detailed summary of the relationship between NH resident characteristics and PrUs including sDTI using the national MDS 3.0. Results demonstrated that just over 10% of NH residents had a Stage II–IV PrU, unstageable PrU, or sDTI. This number is slightly lower than that of other studies and may reflect clinical improvements in prevention and treatment strategies. In reviewing the prevalence of PrUs in long-term care using the 2004 National Nursing Home Survey, approximately 11% of

Table 2.
RESIDENT CHARACTERISTICS, OVERALL, AND BY PRU STATUS

Resident Characteristics	Missing	Overall	No PrU	Any PrU	Only sDTI
Age, y	0.00				
18–39		1.00	0.97	1.24	0.87
40–58		8.03	7.97	8.49	6.36
59–75		26.47	26.32	27.80	25.23
>75		64.50	64.74	62.47	67.54
Gender	0.02				
Male		35.35	34.59	42.00	42.21
Female		64.65	65.41	58.00	57.79
Race	2.39				
Asian		1.61	1.62	1.54	1.68
Black		12.23	11.81	15.93	14.91
Hispanic		4.71	4.62	5.43	6.61
Other		0.55	0.54	0.62	0.50
White		80.90	81.41	76.47	76.30
Body mass index (BMI), kg/m ²	4.94				
Very severely underweight (BMI < 15)		0.39	0.32	0.98	0.87
Severely underweight (15 ≤ BMI < 16.0)		0.75	0.66	1.54	1.41
Underweight (16.0 ≤ BMI < 18.5)		5.15	4.81	8.29	8.08
Normal (18.5 ≤ BMI < 25.0)		37.63	37.21	41.37	42.96
Overweight (25.0 ≤ BMI < 30.0)		28.16	28.57	24.48	25.37
Obese class I (30.0 ≤ BMI < 35.0)		15.15	15.47	12.28	11.72
Obese class II (35.0 ≤ BMI < 40.0)		7.10	7.23	5.90	5.39
Obese class III (BMI > 40.0)		5.67	5.73	5.16	4.20
Skin integrity					
Anemia	0.02	29.02	28.17	36.51	37.85
Malnutrition	0.01	2.97	2.60	6.25	6.56
Dehydration	0.02	0.31	0.28	0.60	0.71
Urinary incontinence	7.57	42.44	41.05	57.34	60.56
Bowel incontinence	1.94	35.81	33.45	57.14	60.56
System failure					
Comatose	0.02	0.21	0.17	0.62	0.70
Heart failure	0.01	19.56	19.05	24.05	24.57
Respiratory failure	0.01	2.37	2.03	5.31	5.53
ESRD	0.01	12.44	11.73	18.71	20.28
Disease					
Diabetes	0.01	32.77	31.89	40.51	40.70
Multiple sclerosis	0.01	0.95	0.86	1.72	1.19
Coronary artery disease	0.01	22.58	22.24	25.57	27.95
COPD	0.02	21.89	21.79	22.76	22.12
Vascular disease	0.01	8.24	7.78	12.27	13.23
Cirrhosis	0.01	0.83	0.80	1.07	0.89 (continues

Table 2.
RESIDENT CHARACTERISTICS, OVERALL, AND BY PRU STATUS, CONTINUED

Resident Characteristics	Missing	Overall	No PrU	Any PrU	Only sDTI
Infection					
Septicemia	0.01	1.31	1.09	3.24	3.62
Pneumonia	0.01	6.54	6.08	10.54	11.73
Urinary tract infection	0.03	12.70	11.94	19.39	21.05
Multidrug-resistant organism	0.01	1.63	1.37	3.98	3.59
Mobility					
Hemiplegia	0.01	6.31	6.31	6.36	6.64
Paraplegia	0.01	0.62	0.40	2.49	1.68
Quadriplegia	0.01	0.47	0.38	1.27	0.85
Hip fracture	0.01	5.38	5.03	8.41	13.22
ADLs impairment, mean (SD)	0.01	17.26 (6.49)	16.83 (6.46)	21.04 (5.51)	21.60 (5.18)
Cognitive impairment	2.17				
Intact		1.91	1.88	2.13	2.10
Mild impairment		47.84	48.20	44.61	41.56
Moderate impairment		20.65	20.65	20.63	20.87
Severe impairment		29.61	29.27	32.63	35.46

Abbreviations: ADLs, activities of daily living; COPD, chronic obstructive pulmonary disease; ESRD, end-stage renal disease; MDS, Minimum Data Set; PrU, pressure ulcer; sDTI, suspected deep tissue injury.

Note: Values are presented in percentages. Overall n = 2,936,146 no PrU (n = 2,639,104 89.9%), and any PrU, including sDTI (n = 297,042; 10.1%) and only sDTI (n = 50,073; 1.7%). The level of activities of daily living impairment was measured using the MDS ADL-Long form. ³⁶ Cognition was measured using the MDS 3.0 Brief Interview for Mental Status for residents who could be interviewed and the MDS-Cognitive Performance Scale for those who could not be interviewed.

NH residents had a PrU of any stage; 50% were Stage II, and the other 50% were Stages I, III, or IV. These PrUs were more likely to present in those 64 years or younger (14%) versus those older than 64 years. Men (10%) were less likely to have PrUs than women (13%). Persons who were residents of NHs for less than 1 year duration were less likely to have PrUs than those who were residents for more than 1 year. Other risk variables identified in the 2004 National Nursing Home Survey, included weight loss, high immobility, and polypharmacy.²

Notably, this study found that risk factors of Stage II–IV and unstageable PrUs identified in this analysis were also risk factors of sDTI. In addition, several risk factors were strongly associated with all stages of PrUs, including anemia, malnutrition, dehydration, infection, urinary incontinence, and bowel incontinence. Many of these risk factors are potentially modifiable and may facilitate the development of strategies to help prevent PrUs in this population in the future.

This study also had 2 findings that represent promising areas for future research. For example, data revealed that the youngest group of NH residents had a higher risk of PrUs. This finding may indicate greater impairment or medical complexity in this age group and/or the fact that preventive

interventions are focused on older residents. In addition, the MDS data demonstrated significant racial and ethnic disparities in the prevalence of PrUs in NH residents. Data for this study showed that black residents had the highest risk of any-stage PrU, and Hispanic residents had the highest risk of sDTI. Considering these findings, it is clear that more research is needed that examines young, racial/ethnic minority NH residents, who are at high risk of PrUs, in order to reduce PrU prevalence in these populations.

Consistent with Defloor's³⁸ conceptual model, this study found that a very high risk of PrU and sDTI was evidenced in the mobility-impaired groups, which included patients with multiple sclerosis and other neurodegenerative diseases. This study also determined that NH residents older than 75 years had the greatest risk of sDTI. Finally, the data revealed potential relationships between PrU risk and level of paralysis (paraplegia vs quadriplegia), as well as PrU risk and specific neurodegenerative disorders (multiple sclerosis), both of which warrant further investigation. Other contributing factors related to younger patients and those with spinal cord injury or paraplegia (such as biobehavioral and psychological aspects of care and compliance) also warrant further investigation.¹³

Table 3.

PERCENTAGES AND UNADJUSTED ORs OF ALL PRUS (INCLUDING SDTI), ONLY PRU (EXCLUDING SDTI), AND ONLY SDTI BY RESIDENT CHARACTERISTICS, CONTINUED

Resident Characteristics	All Prus, %	OR	95% CI	Only PrU, %	OR	95% CI	Only sDTI, %	OR	95% CI
Age									
18-39 (ref)	12.62			11.31			1.66		
40–58	10.80	0.84	0.81-0.87	9.58	0.83	0.80-0.86	1.49	0.90	0.81-0.99
59–75	10.72	0.83	0.80-0.86	9.25	0.80	0.77-0.83	1.79	1.08	0.98-1.18
>75	9.89	0.76	0.73-0.79	8.25	0.70	0.68-0.73	1.94	1.17	1.06-1.29
Gender									
Male	12.13	1.37	1.36-1.38	10.30	1.37	1.36-1.38	2.26	1.38	1.36-1.41
Female (ref)	9.16			7.75			1.65		
Race									
Asian	9.76	1.01	0.98-1.05	8.13	1.00	0.96-1.03	1.92	1.11	1.03-1.19
Black	13.28	1.44	1.42-1.45	11.44	1.45	1.44-1.47	2.33	1.35	1.31-1.38
Hispanic	11.76	1.25	1.23-1.27	9.60	1.20	1.17-1.22	2.63	1.53	1.47-1.58
Other	11.52	1.22	1.16-1.28	10.13	1.27	1.20-1.34	1.71	0.98	0.87-1.12
White (ref)	9.63			8.16			1.74		
Body mass index									
Very severely underweight	25.33	2.76	2.64-2.88	22.37	2.84	2.71-2.97	4.86	2.37	2.14-2.62
Severely underweight	20.45	2.09	2.02-2.16	17.86	2.14	2.06-2.22	3.81	1.84	1.70-1.99
Underweight	16.05	1.55	1.53-1.58	13.78	1.57	1.55-1.60	3.04	1.46	1.41-1.51
Normal (ref)	10.97			9.23			2.11		
Overweight	8.67	0.77	0.76-0.78	7.27	0.77	0.76-0.78	1.63	0.77	0.75-0.79
Obese class I	8.09	0.71	0.71-0.72	6.88	0.73	0.72-0.74	1.40	0.66	0.64-0.68
Obese class II	8.29	0.73	0.72-0.75	7.11	0.75	0.74-0.77	1.37	0.65	0.62-0.67
Obese class III	9.07	0.81	0.80-0.82	7.93	0.85	0.83-0.86	1.35	0.63	0.61-0.66
Skin integrity									
Anemia	12.84	1.47	1.46-1.48	10.86	1.45	1.44-1.46	2.49	1.55	1.53-1.58
Malnutrition	21.47	2.50	2.46-2.54	18.40	2.47	2.43-2.52	4.57	2.63	2.54-2.73
Dehydration	19.66	2.16	2.05-2.27	16.42	2.08	1.97-2.20	4.59	2.54	2.29-2.83
Urinary continence	13.33	1.95	1.93-1.97	9.69	1.88	1.86-1.90	2.22	2.21	2.16-2.25
Bowel continence	18.11	2.59	2.57-2.61	13.46	2.58	2.56-2.6	3.27	3.05	3.00-3.11
System failure									
Comatose	29.73	3.74	3.54-3.95	25.55	3.65	3.44-3.87	7.38	4.23	3.79-4.71
Heart failure	12.55	1.35	1.33-1.36	10.64	1.34	1.33-1.35	2.39	1.38	1.36-1.41
Respiratory failure	22.92	2.71	2.66-2.76	19.72	2.69	2.63-2.74	4.91	2.82	2.71-2.93
ESRD	15.36	1.73	1.72-1.75	12.94	1.70	1.68-1.72	3.18	1.92	1.87-1.96
Disease									
Diabetes	12.62	1.46	1.44-1.47	10.73	1.45	1.44-1.47	2.36	1.47	1.44–1.49
Multiple sclerosis	18.48	2.01	1.95-2.07	16.71	2.14	2.07-2.21	2.55	1.39	1.28-1.50
Coronary artery disease	11.56	1.20	1.19-1.21	9.65	1.17	1.16–1.18	2.33	1.36	1.33–1.38
COPD	10.61	1.06	1.05-1.07	9.05	1.07	1.06-1.08	1.89	1.02	1.00-1.04
Vascular disease	15.20	1.66	1.64-1.68	12.81	1.63	1.61-1.65	3.12	1.81	1.76–1.85
Cirrhosis	13.20	1.34	1.29-1.39	11.57	1.39	1.33-1.44	2.07	1.12	1.02-1.23
									(continues

Table 3.

PERCENTAGES AND UNADJUSTED ORs OF ALL PRUS (INCLUDING SDTI), ONLY PRU (EXCLUDING SDTI), AND ONLY SDTI BY RESIDENT CHARACTERISTICS, CONTINUED

Resident Characteristics	All PrUs, %	OR	95% CI	Only PrU, %	OR	95% CI	Only sDTI, %	OR	95% CI
Infection									
Septicemia	25.20	3.03	2.96-3.10	21.50	2.96	2.88-3.03	5.92	3.40	3.24-3.57
Pneumonia	16.45	1.82	1.80-1.84	13.81	1.77	1.75-1.80	3.53	2.05	2.00-2.11
Urinary tract infection	15.58	1.77	1.76-1.79	13.13	1.74	1.72-1.76	3.24	1.97	1.93-2.01
Multidrug-resistant organism	24.84	2.99	2.93-3.05	21.91	3.05	2.98-3.12	4.74	2.68	2.56-2.82
Mobility									
Hemiplegia	10.29	1.01	0.99-1.03	8.65	1	0.98-1.02	1.96	1.06	1.02-1.09
Paraplegia	41.23	6.30	6.12-6.50	38.36	6.72	6.52-6.94	7.32	4.22	3.93-4.53
Quadriplegia	27.49	3.37	3.24-3.50	25.19	3.60	3.46-3.74	4.07	2.25	2.04-2.48
Hip fracture	15.97	1.73	1.71–1.76	12.29	1.52	1.49-1.54	4.75	2.88	2.8-2.95
ADLs impairment	N/A	1.12	1.12-1.12	N/A	1.13	1.13–1.13	N/A	1.13	1.13-1.14
Cognitive impairment									
Intact (ref)	11.36			9.68			2.05		
Mild impairment	9.47	0.82	0.79-0.84	8.12	0.83	0.80-0.85	1.60	0.77	0.73-0.82
Moderate impairment	10.14	0.88	0.86-0.91	8.58	0.88	0.85-0.90	1.87	0.91	0.85-0.97
Severe impairment	11.19	0.98	0.96–1.01	9.35	0.96	0.94-0.99	2.23	1.09	1.02–1.16

Abbreviations: ADLs, activities of daily living; CI, confidence interval; COPD, chronic obstructive pulmonary disease; ESRD, end-stage renal disease; N/A, not applicable; OR, odds ratio; PrU, pressure ulcer.

ORs greater than 1.00 indicate PrU risk.

The formation of a PrU is multifaceted, and the goal of care is prevention. Nevertheless, even with excellent interprofessional prevention and treatment, unavoidable PrUs do occur. Based on an extensive review of the literature on PrU risk factors and prevalence, the National Pressure Ulcer Advisory Panel 2014 Consensus Conference established consensus on irremediable risk factors that in some circumstances have been shown to predict the likelihood that an unavoidable PrU can develop. Recognizing that every individual is unique in complexity, consensus on certain risk areas includes cardiopulmonary status, hemodynamic instability, elevation of the head of the bed, septic shock, body edema, burns, immobility, medical devices, terminal illness, spinal cord injury, and nutrition. Despite doing all that is possible in the care of an individual to prevent PrUs, there are nonmodifiable risk factors that may contribute to an unavoidable skin injury. Long-term-care residents who are immobile with malnutrition in combination with multiple comorbidities are at increased risk of the prevalence of unavoidable PrUs. Individuals with cachexia or individuals with terminal-stage illness who become immobile are at increased risk of unavoidable PrUs. 45

Several study limitations should be noted. First, this study design was cross-sectional, and as a result, a causal relationship

could not be established. Second, this study is a secondary analysis of a national data set, and the clustering effect within the facility was not controlled. Finally, the number of variables was limited to those available in the MDS 3.0 data set, and other unmeasured or uncontrolled variables associated with sDTI, such as those in the environment, were not included in the analysis. The authors included all NH residents and did not distinguish between PrUs that residents had upon admission or NH-acquired PrUs. Investigating the source of PrU is important for future research.

Despite these limitations, results suggest that the prevalence of sDTI among NH residents is almost 2% and associated with several potentially modifiable factors, such as malnutrition, dehydration, anemia, infection, and urinary and bowel incontinence. These findings indicate that the MDS 3.0 can function as a predictive tool for characterizing risk factors for PrUs and sDTI in NH residents, providing preventive protocols for specific patient aggregates. It is important to note that some risk factors may contribute to unpreventable PrUs. It will be up to the NHs to create the documentation needed to satisfy monitoring agencies that they were also "unavoidable." Clinical education programs should focus on early recognition

of NH residents at risk of the development of PrUs and sDTI, as well as specific risk characteristics and preventive strategies. Pressure ulcer "prevention protocols" in most facilities do not include interventions that alter some of the important potentially modifiable risk factors noted here. It remains an area of deep concern that these protocols continue to focus on turning and moisture rather than on interventions that impact the other factors clinicians identify (eg, anemia, infection, other variables that affect tissue oxygen levels). Similarly, clinical translational researchers should test preventive strategies in future studies and develop tools to determine sDTI risk in order to reduce sDTI prevalence in high-risk populations.

CONCLUSIONS

In summary, this study characterized the risk factors associated with PrUs and sDTI. The strongest risk factors associated with all stages of PrU prevalence, including sDTI, were anemia, malnutrition, dehydration, infection, urinary incontinence, and bowel incontinence. These risk factors are modifiable and may aid in the development of strategies to prevent PrUs in NH residents in the future. The magnitude of risk of sDTI is higher for factors that include age, race, skin integrity, system failure, disease, and infection variables. Individuals with these factors should be targeted with frequent skin assessments and implementation of off-loading pressure strategies. Modifiable risk factors for sDTI in NHs that should be targeted by interventions include malnutrition, dehydration, anemia, UTI, urinary and bowel incontinence, reduction of friction and shearing, and effective pressure off-loading/redistribution in mobility-impaired individuals.

PRACTICE PEARLS

- The revised Nursing Home MDS 3.0 added a section related to the presence/absence and number of sDTI.
- Suspected DTI may precede the development of an open Stage III-IV PrU, which commonly occurs in points of high pressure, such as the sacrum, buttocks, and heels.
- Study results suggest that the prevalence of sDTI among NH residents is almost 2% and associated with several potentially modifiable factors, such as malnutrition, dehydration, anemia, infection, and urinary and bowel incontinence.
- Studies suggest PrUs will develop because of major or continual minor alterations in the normal functioning of human mechanisms in combination with sustained external pressure, external moisture, and/or friction/shear forces.
- Defloor's model of PrUs contained 4 main elements: (1) compressive forces, (2) shearing forces, (3) tissue tolerance for pressure, and (4) tissue tolerance for oxygen.

REFERENCES

- Agency for Healthcare Research and Quality. Preventing pressure ulcers in hospitals. http://www.ahrq.gov/professionals/systems/long-term-care/resources/pressure-ulcers/ pressureulcertoolkit/putoolkit.pdf. Last accessed January 27, 2016.
- National Pressure Ulcer Advisory Panel (NPUAP) Monograph. In: Pieper B, ed. Pressure Ulcers: Prevalence, Incidence, and Implications for the Future. Washington, DC: NPUAP; 2012.
- Sen CK, Gordillo GM, Roy S, et al. Human skin wounds: a major and snowballing threat of public health and the economy. Wound Repair Regen 2009;17:763-71.
- National Pressure Ulcer Advisory Panel (NPUAP). NPUAP pressure ulcer stages/categories. http://www.npuap.org/resources/educational-and-clinical-resources/npuap-pressure-ulcer-stagescategories/. Last accessed January 27, 2016.
- Black J, Baharestani MM, Cuddigan J, et al. National Pressure Ulcer Advisory Panel's updated pressure ulcer staging system. Adv Skin Wound Care 2007;20:269-74.
- Slomka N, Or-Tzadikario S, Gefen A. Cellular Deformations under Compression in Cells Involved in Deep Tissue Injury. In: Gefen A, ed. Bioengineering Research of Chronic Wounds: A Multidisciplinary Study Approach. Berlin Heidelberg, Germany: Springer-Verlag Berlin Heidelberg; 2009.
- Niezgoda JA, Mendez-Eastman S. The effective management of pressure ulcers. Adv Skin Wound Care 2006;19(Suppl 1):3-15.
- Perneger TV, Héliot C, Rae AC, Borst F, Gaspoz JM. Hospital-acquired pressure ulcers: risk factors and use of preventive devices. Arch Intern Med 1998;158:1940-5.
- 9. Lyder CH. Pressure ulcer prevention and management. JAMA 2003;289:223-6.
- Vangilder C, Macfarlane GD, Meyer S. Results of nine international pressure ulcer prevalence surveys: 1989 to 2005. Ostomy Wound Manage 2008;54(2):40-54.
- Gefen A. How much time does it take to get a pressure ulcer? Integrated evidence from human, animal, and in vitro studies. Ostomy Wound Manage 2008;54(10):26-8.
- 12. Sibbald RG, Krasner DL, Lutz J. SCALE: Skin Changes at Life's End: final consensus statement. October 1, 2009. Adv Skin Wound Care 2010;23:225-38.
- Edsberg LE, Langemo D, Baharestani MM, Posthauer ME, Goldberg M. Unavoidable pressure injury: state of the science and consensus outcomes. J Wound Ostomy Continence Nurs 2014;41:313-34.
- Allman RM, Goode PS, Patrick MM, Burst N, Bartolucci AA. Pressure ulcer risk factors among hospitalized patients with activity limitation. JAMA 1995;273:865-70.
- Baldwin KM, Ziegler SM. Pressure ulcer risk following critical traumatic injury. Adv Wound Care 1998;11:168-73.
- Bergquist S, Frantz R. Pressure ulcers in community-based older adults receiving home health care. Prevalence, incidence, and associated risk factors. Adv Wound Care 1999; 12:330-51
- Bergstrom N, Braden B. A prospective study of pressure sore risk among institutionalized elderly. J Am Geriatr Soc 1992;40:747-58.
- Bergstrom N, Braden B, Kemp M, Champagne M, Ruby E. Multi-site study of incidence of pressure ulcers and the relationship between risk level, demographic characteristics, diagnoses, and prescription of preventive interventions. J Am Geriatr Soc 1996;44(1):22-30.
- Berlowitz DR, Wilking SV. Risk factors for pressure sores. A comparison of cross-sectional and cohort-derived data. J Am Geriatr Soc 1989;37:1043-50.
- Brandeis GH, Ooi WL, Hossain M, Morris JN, Lipsitz LA. A longitudinal study of risk factors associated with the formation of pressure ulcers in nursing homes. J Am Geriatr Soc 1994;42:388-93.
- Chan WS, Pang SM, Kwong EW. Assessing predictive validity of the modified Braden scale for prediction of pressure ulcer risk of orthopaedic patients in an acute care setting. J Clin Nurs 2009;18:1565-73.
- Fife C, Otto G, Capsuto EG, et al. Incidence of pressure ulcers in a neurologic intensive care unit. Crit Care Med;29:283-90.
- Goodridge DM, Sloan JA, LeDoyen YM, McKenzie JA, Knight WE, Gayari M. Risk-assessment scores, prevention strategies, and the incidence of pressure ulcers among the elderly in four Canadian health-care facilities. Can J Nurs Res 1998;30(2):23-44.
- Hatanaka N, Yamamoto Y, Ichihara K, et al. A new predictive indicator for development of pressure ulcers in bedridden patients based on common laboratory tests results. J Clin Pathol 2008;61:514-8.
- Okuwa M, Sanada H, Sugama J, et al. A prospective cohort study of lower-extremity pressure ulcer risk among bedfast older adults. Adv Skin Wound Care 2006;19:391-7.
- Schultz A, Bien M, Dumond K, Brown K, Myers A. Etiology and incidence of pressure ulcers in surgical patients. AORN J 1999;70:434, 437-440, 443-39.

- Stordeur S, Laurent S, D'Hoore W. The importance of repeated risk assessment for pressure sores in cardiovascular surgery. J Cardiovasc Surg (Torino) 1998;39:343-9.
- Tescher AN, Branda ME, Byrne TJ, Naessens JM. All at-risk patients are not created equal: analysis of Braden pressure ulcer risk scores to identify specific risks. J Wound Ostomy Continence Nurs 2012;39:282-91.
- Baumgarten M, Margolis D, van Doorn C, et al. Black/White differences in pressure ulcer incidence in nursing home residents. J Am Geriatr Soc 2004;52:1293-8.
- Levine JM. Historical perspective: impact of plaster of paris splints on pressure ulcer occurrence in World War II. Adv Skin Wound Care 2008;21:526-8.
- Levine JM. Historical notes on pressure ulcers: the decubitus ominosus of Jean-Martin Charcot. J Am Geriatr Soc 2005;53:1248-51.
- Levine JM. Historical notes on pressure ulcers: the cure of Ambrose Paré. Decubitus 1992;5(2):23-4. 26.
- Levine JM. Historical perspective: the neurotrophic theory of skin ulceration. J Am Geriatr Soc 1992;40:1281-3.
- Fogerty MD, Abumrad NN, Nanney L, Arbogast PG, Poulose B, Barbul A. Risk factors for pressure ulcers in acute care hospitals. Wound Repair Regen 2008;16:11-8.
- Stechmiller JK, Cowan L, Whitney JD, et al. Guidelines for the prevention of pressure ulcers. Wound Repair Regen 2008;16:151-68.
- Berlowitz D, Brandeis G, Anderson J, et al. Evaluation of a risk-adjustment model for pressure ulcer development using the Minimum Data Set. J Am Geriatr Soc 2001;49:872-6.

- Cowan LJ, Stechmiller JK, Rowe M, Kairalla JA. Enhancing Braden pressure ulcer risk assessment in acutely ill adult veterans. Wound Repair Regen 2012;20:137-48.
- Defloor T. The risk of pressure sores: a conceptual scheme. J Clin Nurs 1999;8: 206-16
- Braden B, Bergstrom N. A conceptual schema for the study of the etiology of pressure sores. Rehabil Nurs 1987;12:8-12.
- 40. Baranoski S. Pressure ulcers: a renewed awareness. Nursing 2006;36(8):36-41.
- VanGilder C, MacFarlane GD, Harrison P, Lachenbruch C, Meyer S. The demographics of suspected deep tissue injury in the United States: an analysis of the International Pressure Ulcer Prevalence Survey 2006-2009. Adv Skin Wound Care 2010;23:254-61.
- 42. Ahn H, Horgas A. The relationship between pain and disruptive behaviors in nursing home resident with dementia. BMC Geriatr 2013;13:14.
- 43. Ahn H, Stechmiller J, Horgas A. Pressure ulcer-related pain in nursing home residents with cognitive impairment. Adv Skin Wound Care 2013;26:375-80.
- Saliba D, Buchanan J. Development and validation of a revised nursing home assessment tool: MDS 3.0. 2008. http://www.cms.gov/Medicare/Quality-Initiatives-Patient-Assessmentinstruments/NursingHomeQualityInits/downloads/MDS30FinalReport.pdf. Last accessed February 3, 2016.
- National Pressure Ulcer Advisory Panel, European Pressure Ulcer Advisory Panel and Pan Pacific Pressure Injury Alliance. Prevention and Treatment of Pressure Ulcers: Clinical Practice Guideline. Haesler E, ed. Perth, Australia: Cambridge Media; 2014.

For more than 137 additional continuing education articles related to Skin and Wound Care topics, go to NursingCenter.com/CE.

CE COMECTION

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*TM. Physicians should only claim credit commensurate with the extent of their participation in the activity.

PROVIDER ACCREDITATION INFORMATION FOR NURSES

Lippincott Williams & Wilkins, publisher of the Advances in Skin & Wound Care journal, will award 3.0 contact hours for this continuing nursing education activity.

LWW 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 3.0 contact hours. LWW is also an approved provider by the District of Columbia, Georgia, and Florida CE Broker #50-1223. Your certificate is valid in all states.

OTHER HEALTH PROFESSIONALS

This activity provides ANCC credit for nurses and AMA PRA Category 1 $Credit^{TM}$ 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 178. For nurses who wish to take the test for CE contact hours, visit www.nursingcenter.com. For physicians, who wish to take the test for CME credit, visit http://cme.lww.com.
- You will need to register your personal CE Planner account before taking online tests. Your planner will keep track of all your Lippincott Williams & Wilkins 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: April 30, 2018 (nurses); April 30, 2017 (physicians).

PAYMENT AND DISCOUNTS

• The registration fee for this test is \$27.95 for nurses; \$22 for physicians.