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Chapter 8

Pressure Injury in the ICU: Major Reconstructive Surgery Required

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Additional information is available at the end of the chapter

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Abstract

Pressure injury (PI) has replaced the former nomenclature pressure ulcer, a change initiated by the National Pressure Ulcer Advisory Panel (NPUAP) however, substitutes such as pressure ulcers, decubitus ulcers, and bedsores will continue to be used by many. Increased knowledge and awareness of PIs has lead to a decline in their overall prevalence. A review of the most common risk factors, including two risk factor assessment tools, the Braden scale and the Cubbin & Jackson are presented. Diagnosing PIs must be a methodical, meticulous process in order to accurately document and monitor their progression and improvement. In 2016 the NPUAP revised the definitions as well as the stages of PIs incorporating the etiology and anatomical features present or absent in each stage of injury. Treatment strategies such as managing co-morbidities, nutrition optimization, and pain management are important aspects to consider in treating PIs in addition to thorough wound care cleansing and debridement. Highlighted are the various effective debridement options such as surgical sharp, mechanical, autolytic, enzymatic and larval debridement. Wound dressing alternatives, their advantages, disadvantages, indications and contraindications are all are mentioned. Concluding the chapter are pressure injury rates of healing, prognosis and surgical indications.

Keywords: pressure injury, pressure ulcers, decubitus ulcers, bedsores, ulcer prevention, ulcer treatment

1. Case introduction

Cynthia is a 70-year-old retired African-American patient with a past medical history significant for anemia and lung cancer—now 10 years in remission. She initially presented to the hospital complaining fatigue and severe cough that she "just couldn't get rid of." She was

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subsequently found to have severe pneumonia requiring intravenous antibiotics. She was transferred to the medical intensive care unit (ICU) due to hypoxia and rapid decompensation on the floor; before she knew it 2 weeks had gone by since her admission. She noticed increasing amounts of lower back pain, presumably from sitting in bed all day. While her family and nurses encouraged her to get out of bed and walk she refused, stating that she was just "too weak to walk." One day while repositioning Cynthia in order to change the bed sheets; her nurse noted a new dime sized, red mark right above her gluteal cleft. She knew that she was supposed to document all wounds but she did not think this small, little mark "counted" as it was pretty minor. The nurse finished her shift and did not think much about the lesion. As the days progressed the dime sized lesion became the size of a nickel, then a quarter and soon the wound was as large as your hand. The patient complained of very severe pain in that region. The once red tissue was now grayish-black and it occasionally it produced a yellow-green exudate. The nurses did their best to pad her sacral region with various dressings however she was so thin and malnourished that you could literally see the vertebrae along her spine; subsequently the tissue continued to break down. Eventually, Cynthia's pressure injury was so deep that a small piece of bone started to protrude from the wound base. The medical ICU team tried various modalities to simulate wound healing, once they had exhausted all options for pressure wound injuries, they felt it was necessary to ask the plastic surgery team to evaluate her. Due to the depth and size of the sacral lesion, the plastic surgeons determined that Cynthia was going to require an operation — more specifically a multistep, rotational flap procedure. The patient and her family cuold not believe the news. How could this have happened? Could this outcome have been prevented?

2. Introduction—what is a pressure injury?

Pressure injury has replaced the former nomenclature pressure ulcer, a recent change initiated by the National Pressure Ulcer Advisory Panel (NPUAP). The change in terminology derives inception from past histopathological work, which indicates that small changes in pressure-related injuries start in the tissue prior to the changes being visible on physical examination [1–3].

A pressure injury is localized damage to the skin and/or underlying soft tissue usually over a bony prominence or related to a medical or other device [6]. An ulcer is defined as a break in skin or mucous membrane with loss of surface tissue, disintegration and necrosis of epithelial tissue, and often, purulent exudate [5]. Thus, a pressure injury can be present without an ulcer however an ulcer cannot be present without a prior injury [6]. While the new terminology, pressure injury is the more technically correct term; many have argued that the name change does nothing to add clarity, improve accuracy or correct patient outcomes [7, 8]. Regardless of the advisory panels' endorsed term substitutes such as pressure ulcers, decubitus ulcers, and bedsores will continue to be used by many.

Pressure injuries can present as intact skin or an open ulcer and may be painful. The injury occurs as a result of intense and/or prolonged pressure or pressure in combination with shear. The tolerance of soft tissue for pressure and shear can be affected by microclimate, nutrition, perfusion, co-morbidities as well as the condition of the soft tissue [4]. The foul odors associated

with many chronic, nonhealing wounds may impair patient's quality of life, lead them toward isolation and can reduce their contact with family, significant others and care givers [9].

3. Patient demographics, incidence and prevalence

Pressure injuries most commonly affect the elderly ages 65–70 years old, those with limited mobility, including hospitalized patients, nursing home occupants, those with neurologic impairment and people with severe illnesses [10]. General facility demographic trends indicate that mean patient age acquiring pressure injuries has decreased, scores for the Braden Scale, a PI risk factor assessment tool have remained constant, and patient weight has increased in most care settings [11].

Increased knowledge and awareness of pressure injuries has lead to a decline in their overall prevalence (OP). A large sample sized study with data collection spanning from 2006 to 2015 showed that the OP of pressure injuries in all facilities declined from 13.5% (2006) to 9.3% (2015) [11]. The annual prevalence of pressure ulcers among patients 65 years and older in general medicine practice has varied from 0.31 to 0.7% [12]. An Australian study found that excluding Stage 1 ulcers, overall hospital-acquired pressure injury prevalence from 2012 to 2014 was 11% for intensive care patients and 3% for non-intensive care patients [13]. Intensive care patients were found to be 3.8 times more likely (RR 2.7–5.4, 95% CI) than non-intensive care patients to develop a pressure injury while in the hospital [13]. Prevalence varies according to the patient's residence or surrounding environment. Facility-acquired prevalence (FAP) declined from 6.2% (2006) to a range of 3.1–3.4% (2013–2015). Acute care OP was 13.3% in 2006 and declined to a range of 8.8–9.3% (2012–2015). Long-term acute care (LTAC) had the highest OP at 32.9% in 2006; it declined to 28.8% in 2015 [11].

The sacrum/coccyx is the most common site of hospital-acquired pressure injury in all patients (intensive care patients 22%; non-intensive care patients 35%) [13]. Stage 2 hospital-acquired pressure injury (HAPI) prevalence is the most common stage reported, 53% for intensive care patients compared to 63% for non-intensive care patients [13]. Mucosal tissue lines the tongue, gastrointestinal (GI) tract, nasal passages, urinary tract and vaginal canal. This type of tissue is vulnerable to pressure from medical devices, such as oxygen tubing, endotracheal tubes, orogastric and nasogastric tubes and urinary catheters. Mucosal pressure injuries have been found to be significantly higher in intensive care patients (22%) than in non-intensive care patients (2%) [13].

Incidence is a commonly reported measure; however it is computed by counting the number of patients with newly acquired pressure ulcers and dividing that number by the number of patients examined for pressure ulcers over a given period of time. Smaller facilities can appear to have a higher percentage of patients with ulcers because there are fewer patients in the denominator. For example, five patients with ulcers in a 100 patient facitlity equals a 5% incidence. The same number of patients with ulcers (5) in a 500 patient facility is only a 1% incidence. Incidence density is the best quality measure of pressure ulcer prevention programs, according to the NPUAP [14]. Pressure ulcer incidence density is a computation based on the number of in-patients who develop a new pressure ulcer(s) divided by 1000

patient days. Using the larger denominator of patient days allows fair comparisons between institutions of all sizes and stabilizes results. Many state reporting systems and hospital-acquired conditions (e.g., CAUTI) currently use incidence density [14].

4. Risk factors

There are a multitude of risk factors that can contribute to PIs. Literature has stated that the elderly, aged >60 years old are the most prone to the development of pressure injuries, due to changes in aging skin such as decreased skin elasticity, insufficient skin hydration, decreased sensitivity, and factors that are associated with chronic comorbid conditions [15, 16]. Conditions that interfere with peripheral circulation and tissue perfusion have also been linked to PIs, such as congestive heart failure (CHF), diabetes mellitus and smoking [16].

Limited mobility is considered a fundamental component of PI formation. A reduction in movement can be related to a spinal cord injury, progressive neurological disorders, stroke, pain, and fractures among other etiologies. Pressure from any hard surface (e.g. bed, wheelchair or stretcher), friction from a patient's inability to move well in bed and shear from involuntary muscle movements are also a major contributors toward pressure injuries. Moisture from bowel or bladder incontinence, excessive perspiration and wound drainage have also been found to be correlated with pressure injuries. Poor nutrition resulting in reduced body mass due to wasting or cachexia typically results in less subcutaneous fat. Less protection over areas of bony prominence can lead to increased risk of pressure injuries, thus optimizing patients' nutritional status is essential for skin protection as well as wound healing.

5. Risk factor assessment tools

The Braden Scale is one of the most common pressure injury risk scales used in the United States. It is valid and effective for assessing the risk of developing pressure ulcers [17]. The Braden and Cubbin & Jackson are valid scales for measuring the pressure injury risk in patients admitted to intensive care units (ICUs) [18]. The health status of ICU patients vastly differs from general hospital patients. ICU patients are more likely to have several comorbid conditions, be hemodynamically unstable, receive vasoactive medications, be ventilator- dependent and are often sedated [19]. Literature states that for every mmHg decrease in diastolic blood pressure, the odds of a deep tissue injury increases by approximately 7.5% (1/0.93 = 1.075) [20]. Some literature has shown that of the two ICU risk assessment tools, the Cubbin & Jackson scale is the most effective in predicting risk of decubitus ulcers in patients admitted in ICU with sensitivity of 99.3% and specificity of 55.5% [17]. The Cubbin and Jackson scale has been used for the ICU population with great results [19].

5.1. Braden scale for predicting pressure sore risk: used on general hospital and ICU patients

The Braden Scale is the most widely used pressure ulcer risk assessment system in the world [21]. It is a clinically validated tool that was developed to help all health professionals, especially

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nurses, assess and reliably score a patient's risk of developing a pressure injury [22]. It consists of six categories: sensory perception, moisture, activity, mobility, nutrition, and friction/shear. Each category is rated on a scale of 1–4, excluding the 'friction and shear' category, which is rated on a 1–3 scale. The total score can range from 6 to 23. The scale is an inverse scoring system thus a lower score indicates a higher risk of developing an injury and vice versa. A score of 23 means there is no risk for developing a pressure ulcer while a score of six points represents the most severe risk for developing a pressure ulcer [22] (**Table 1**).

BRADEN SCALE FOR PREDICTING PRESSURE SORE RISK

Patient's Name _____ Evaluator's Name _____ Date of Assessment

SENSORY PERCEPTION ability to respond meaningfully to pressure-related discomfort	**1. Completely Limited** Unresponsive (does not moan, flinch, or grasp) to painful stimuli, due to diminished level of consciousness or sedation. OR limited ability to feel pain over most of body	**2. Very Limited** Responds only to painful stimuli. Cannot communicate discomfort except by moaning or restlessness OR has a sensory impairment which limits the ability to feel pain or discomfort over ½ of body.	**3. Slightly Limited** Responds to verbal commands, but cannot always communicate discomfort or the need to be turned. OR has some sensory impairment which limits ability to feel pain or discomfort in 1 or 2 extremities.	**4. No Impairment** Responds to verbal commands. Has no sensory deficit which would limit ability to feel or voice pain or discomfort.				
MOISTURE degree to which skin is exposed to moisture	**1. Constantly Moist** Skin is kept moist almost constantly by perspiration, urine, etc. Dampness is detected every time patient is moved or turned.	**2. Very Moist** Skin is often, but not always moist. Linen must be changed at least once a shift.	**3. Occasionally Moist:** Skin is occasionally moist, requiring an extra linen change approximately once a day.	**4. Rarely Moist** Skin is usually dry, linen only requires changing at routine intervals.				
ACTIVITY degree of physical activity	**1. Bedfast** Confined to bed.	**2. Chairfast** Ability to walk severely limited or non-existent. Cannot bear own weight and/or must be assisted into chair or wheelchair.	**3. Walks Occasionally** Walks occasionally during day, but for very short distances, with or without assistance. Spends majority of each shift in bed or chair	**4. Walks Frequently** Walks outside room at least twice a day and inside room at least once every two hours during waking hours				
MOBILITY ability to change and control body position	**1. Completely Immobile** Does not make even slight changes in body or extremity position without assistance	**2. Very Limited** Makes occasional slight changes in body or extremity position but unable to make frequent or significant changes independently.	**3. Slightly Limited** Makes frequent though slight changes in body or extremity position independently.	**4. No Limitation** Makes major and frequent changes in position without assistance.				
NUTRITION usual food intake pattern	**1. Very Poor** Never eats a complete meal. Rarely eats more than ⅓ of any food offered. Eats 2 servings or less of protein (meat or dairy products) per day. Takes fluids poorly. Does not take a liquid dietary supplement OR is NPO and/or maintained on clear liquids or IV's for more than 5 days.	**2. Probably Inadequate** Rarely eats a complete meal and generally eats only about ½ of any food offered. Protein intake includes only 3 servings of meat or dairy products per day. Occasionally will take a dietary supplement. OR receives less than optimum amount of liquid diet or tube feeding	**3. Adequate** Eats over half of most meals. Eats a total of 4 servings of protein (meat, dairy products per day. Occasionally will refuse a meal, but will usually take a supplement when offered OR is on a tube feeding or TPN regimen which probably meets most of nutritional needs	**4. Excellent** Eats most of every meal. Never refuses a meal. Usually eats a total of 4 or more servings of meat and dairy products. Occasionally eats between meals. Does not require supplementation.				
FRICTION & SHEAR	**1. Problem** Requires moderate to maximum assistance in moving. Complete lifting without sliding against sheets is impossible. Frequently slides down in bed or chair, requiring frequent repositioning with maximum assistance. Spasticity, contractures or agitation leads to almost constant friction	**2. Potential Problem** Moves feebly or requires minimum assistance. During a move skin probably slides to some extent against sheets, chair, restraints or other devices. Maintains relatively good position in chair or bed most of the time but occasionally slides down.	**3. No Apparent Problem** Moves in bed and in chair independently and has sufficient muscle strength to lift up completely during move. Maintains good position in bed or chair.					
					Total Score			

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Risk Scale: Low risk: 23-19; Mild risk: 18-15; Moderate risk: 14-13; High risk: 12-10; severe risk: 9-6.

Table 1. Braden Scale [22].

5.2. The Cubbin & Jackson scale for predicting pressure sore risk in ICU patients

The Cubbin & Jackson scale was specifically designed in 1991 for intensive care patients and is a modification of another assessment scale, the Norton scale. The scale consists of 10 items: age, weight, general skin condition, mental condition, mobility, hemodynamics, respiration, nutrition, incontinence, and hygiene. Each item has a 4-point scale; thus, a maximum of 40 points total. This is another inverse scale in that the lower the point total is, the higher the likelihood of pressure ulcer development is [23] **Table 2**.

Categories of scale	Score	Operational definition
Age (yr)		
<40	4	
40–54	3	Age at the time of admission to the ICU
55–69	2	
>70	1	
Weight		
Average weight	4	
Obese	3	BMI on the medical record at the time of admission to the ICU
Cachectic	2	
Any of above and edema	1	Edema on the assessment form
General skin condition		
Intact	4	No pressure ulcer, no sore or sore none on the nursing record
Red skin	3	Redness on the nursing record
Grazed/excoriated skin	2	Abrasion, or bullae on the nursing record
Necrosis/exuding	1	Necrosis on the nursing record
Mental condition		
Awake and alert	4	
Agitated/restless/confused	3	Consciousness on the assessment form at the time of admission to the ICU
Apathetic/sedated but responsive	2	
Coma/unresponsive	1	
Mobility		
Fully ambulant	4	Not applicable
Walks with slight help	3	Not applicable
Very limited/chairbound	2	3 or 4 on position change score of Braden scale
Immobile/bedrest	1	1 or 2 on position change score of Braden scale
Hemodynamics		
Stable without inotropic support	4	MBP ≥ 65 mmHg without inotropic support

Categories of scale	Score	Operational definition
Stable with inotropic support	3	MBP ≥ 65 mmHg with inotropic support
Unstable with inotropic support	2	55 mmHg < MBP < 65 mmHg with inotropic support
Critical with inotropic support	1	MBP ≤ 55 mmHg with inotropic support
Respiration		
Spontaneous	4	
CPAP/T-piece	3	Airway and oxygen supply on the assessment form at the time of admission to the ICU
Mechanical ventilation	2	
Breathless at rest/on exertion	1	
Nutrition		
Full diet, fluids	4	Prescription of regular diets or soft diets
Light diet/oral fluids/enteral feeding	3	Prescription of enteral nutrition, or full liquid diets without regular or soft diets
Parenteral feeding	2	Prescription of TPN only without diets or enteral nutrition
Clear intravenous fluid only	1	No prescription of TPN, enteral nutrition or diets
Incontinence[a]		
None/anuric/catheterized	4	None/anuric/catheterized on clinical assessment form
Urine	3	Not applicable
Feces	2	More than two bowel movements per day
Urine, feces	1	Not applicable
Hygiene		
Competent in maintaining own hygiene	4	Not applicable
Maintaining own hygiene with slight help	3	4 on position change score of Braden scale
Requiring much assistance	2	3 on position change score of Braden scale
Fully dependent	1	1 or 2 on position change score of Braden scale

Table 2. Cubbin & Jackson Scale [23]. Used with Permission.

6. Diagnosing pressure injuries

Diagnosing pressure injuries must be a methodical and meticulous process in order to accurately document and monitor progression as well as improvement. Keen documentation of the physical examination including pictures should be established. Initially, a complete head to toe physical exam should be performed to assess the patient and identify all of his/her wounds and lesions. Look for signs of systemic infection including, fever, chills, fatigue, diaphoresis, hypotension and tachycardia.

6.1. Skin assessment

When diagnosing a pressure injury, it is essential to confirm the presence of pressure and/or shear as a causative factor [6]. In order to perform an accurate visual assessment, pressure injury staging should take place only after the wound bed has been cleansed [6]. Have the patient in a neutral position when assessing the wound and use a consistent method when measuring the length, width and depth of the lesion. Note and document the number, location and size of the lesion(s) at each assessment, ideally with picture documentaiton. Look for accompanying edema, erythema, blanching response, warmth, tenderness, induration, exudate, purulence, odor, sinus tracts, necrosis, eschar formation, tunneling, undermining, wound margins, and possible exposed or palpable bone. Since the NPUAP pressure injury staging system is based on the extent of tissue damage, an understanding of anatomy is essential when evaluating the type of tissue present in the wound [4, 24] (**Figure 1**).

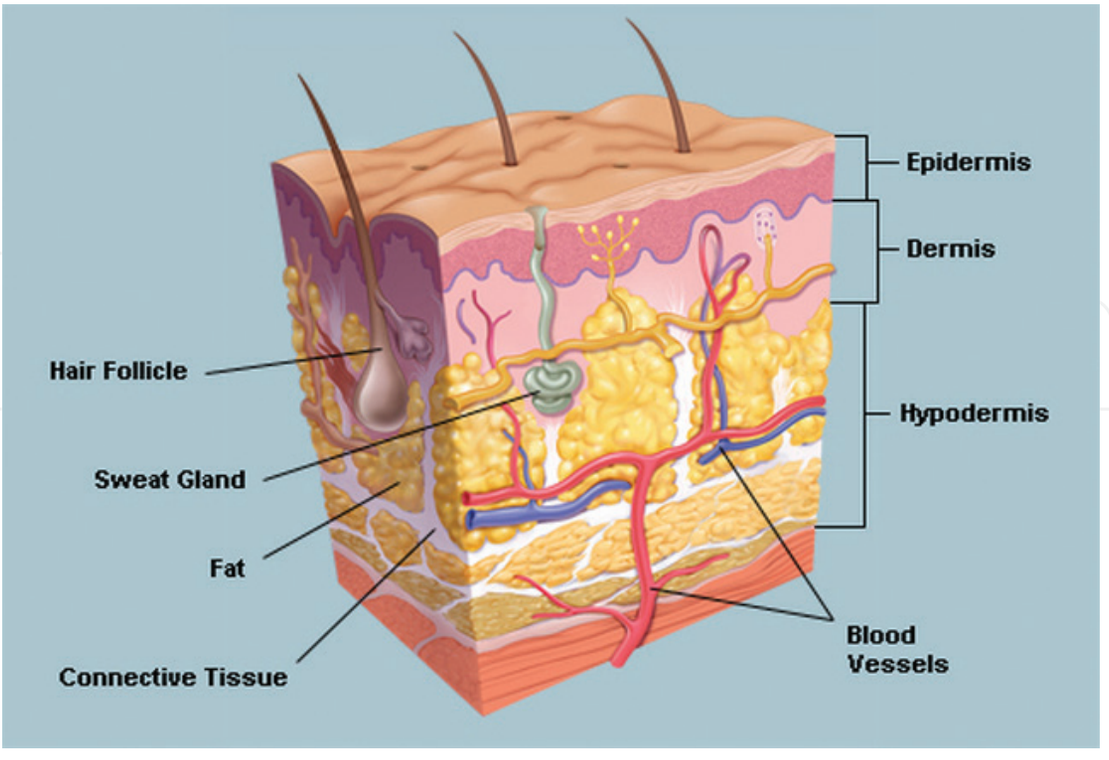


Figure 1. Skin Anatomy. [24] Used with permission. 2014 WebMD, LLC. All rights reserved.

6.2. Assess for blanching

Finger pressure method: press on erythema for 3 seconds remove finger, then assess for blanching

Transparent disc method: apply pressure equally on all areas of erythema with a transparent disc; access for blanching during the application

Of note, assessing for blanching in patients with dark skin may be challenging; in this patient population focus on skin temperature, skin tenderness, tissue consistency and pain levels. It is wise to rule out neuropathy in all patients by testing the skin's sensation at the level of the lesion. Do this by performing the Semmes-Weinstein monofilament exam. Once a thorough physical exam has been performed and documented, one can make the clinical diagnosis and stage the lesion [25].

7. Staging pressure injuries

Attempts to classify pressure injuries date back to the 1975 staging system developed by J.D. Shea [26]. Since then, other staging systems have been proposed from the International Association of Enterostomal Therapy (now the Wound, Ostomy and Continence Nurses Society), [27] as well as the National Pressure Ulcer Advisory Panel (NPUAP). The NPUAP's initial 1989 pressure injury staging system was based on the International Association of Enterostomal Therapy's system. The NPUAP made revisions by incorporating deep tissue injury and also collaborated with the European Pressure Ulcer Advisory Panel to publish guidelines with category/stage differentiation [28]. NPUAP's staging system has been widely adopted internationally [6].

Most recently in 2016, the NPUAP revised the definition as well as stages of pressure injury. The revision was undertaken to incorporate the current understanding of the etiology of pressure injuries, as well as to clarify the anatomical features present or absent in each stage of injury [6]. Each definition now describes the extent of tissue loss present and the anatomical features that may or may not be present in the stage of injury [6]. The nomenclature and staging changes have caused some uproar. Opponents stress concern that the existing staging system continues to perpetuate the fallacy that pressure induced skin damage presents as a top (epidermis) down (bone) sequence of evolution in severity, that can be accurately classified by simple visual assessment of the skin layers, which is not entirely true [8]. Other changes made by the NPUAP such as denoting the stages using Arabic numerals rather than Roman numerals have caused less of an issue.

Pressure injury staging is important as it has become the basis for treatment, comparison of outcomes, and, if applicable, reimbursement [6]. Regardless of the stage assigned by visual examination, the examiner must take into account all of the available information and incorporate their interdisciplinary clinical expertise into defining the pressure injury etiology and development; he/she should then classifying the lesion [6] (**Figures 2** and **3**).

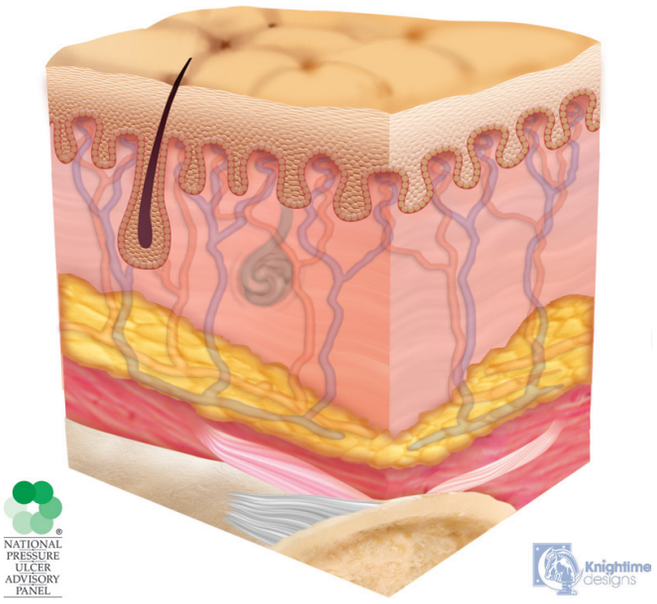


Figure 2. Heathy skin lightly pigemented. Illustrations for figure is used with permission from Ref. [4].

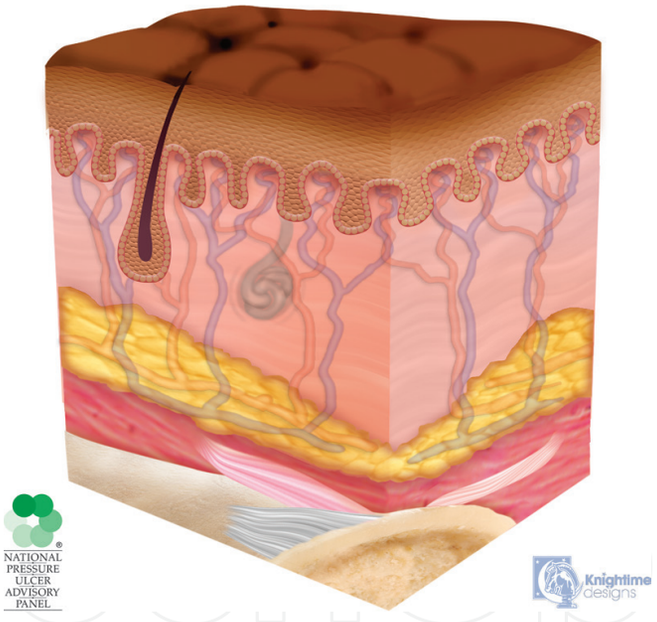


Figure 3. Heathy skin darkly pigemented. Illustrations for figure is used with permission from Ref. [4].

7.1. Newly defined stages of pressure injury as defined by the National Pressure Ulcer Advisory Panel (NPUAP)

7.1.1. Stage 1 pressure injury

Intact skin with a localized area of non-blanchable erythema. The presence of blanchable erythema or changes in sensation, temperature, or firmness may precede visual changes [6].

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Beware: Nonblanchable erythema may appear differently in darkly pigmented skin. Also, color changes do not include purple or maroon discoloration; these colors may indicate deep tissue pressure injury (DTPI) (**Figures 4** and **5**).

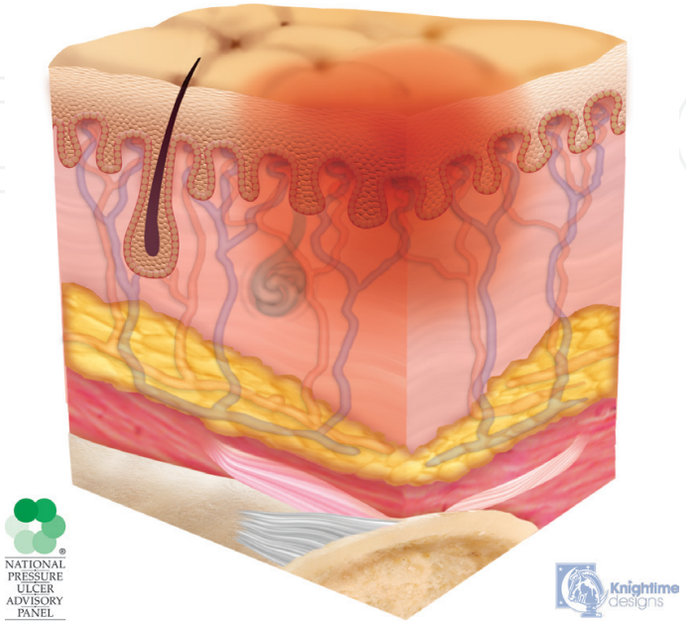


Figure 4. Stage 1 pressure injury lightly pigmented. Illustrations for figure is used with permission from Ref. [4].

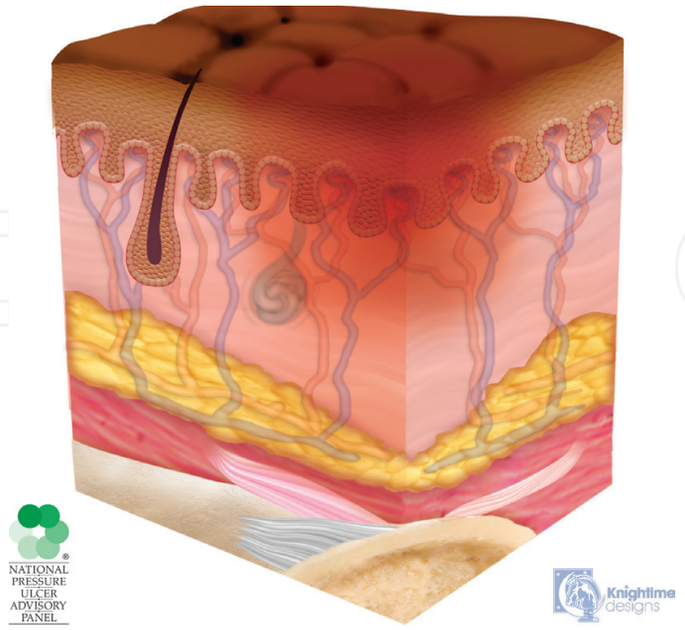


Figure 5. Stage 1 pressure injury darkly pigmented. Illustrations for figure is used with permission from Ref. [4].

7.1.2. Stage 2 pressure injury

Partial-thickness skin loss of skin with exposed dermis. The wound bed is viable, pink or red, moist, and may present as an intact or ruptured serum-filled blister. Fat is not visible and deeper tissues are not visible. Granulation tissue, slough and eschar are not present. These injuries commonly result from adverse microclimate and shear in the skin over the pelvis and shear in the heel [4] (**Figure 6**).

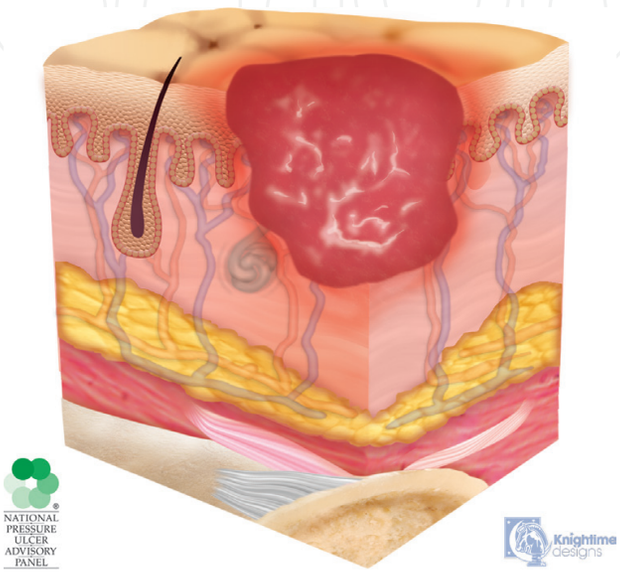


Figure 6. Stage 2 pressure injury. Illustrations for figure is used with permission from Ref. [4].

The revised definition of a Stage 2 pressure injury seeks to clarify the difference between moisture-associated skin damage and injury caused by pressure and/or shear [6]. Stage 2 injuries should not be used to describe moisture associated skin damage (MASD), incontinence associated dermatitis (IAD), intertriginous dermatitis (ITD), medical adhesive related skin injury (MARSI), or traumatic wounds such as skin tears, burns or abrasions [4].

7.1.3. Stage 3 pressure injury

Full-thickness loss of skin, in which fat is visible within the ulcer; granulation tissue and epibole (rolled wound edges) are often present. Slough and/or eschar may be visible. The depth of tissue damage varies by anatomical location; areas of significant adiposity can develop deep wounds. Undermining and tunneling may occur. Fascia, muscle, tendon, ligament, cartilage and/or bone are not exposed. If slough or eschar obscures the extent of tissue loss then it should be classified as an unstageable pressure injury [4] (**Figures 7** and **8**).

7.1.4. Stage 4 pressure injury

Full-thickness skin and tissue loss with exposed or directly palpable fascia, muscle, tendon, ligament, cartilage or bone within the ulcer. Like in Stage 3 wounds, slough and/or eschar

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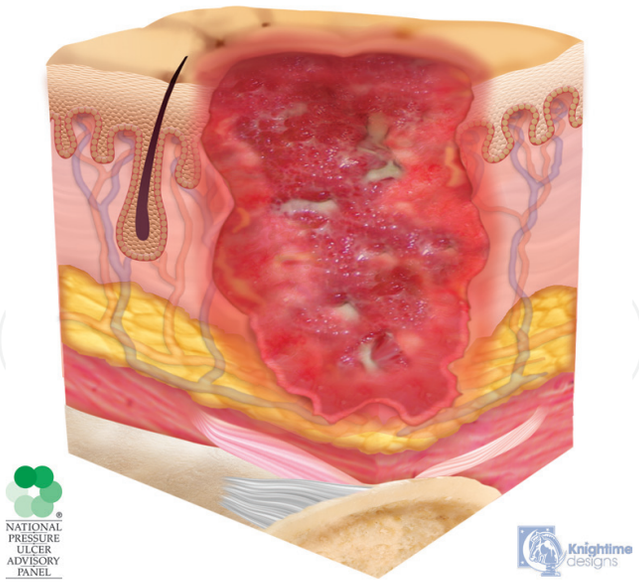


Figure 7. Stage 3 pressure injury. Illustrations for figure is used with permission from Ref. [4].

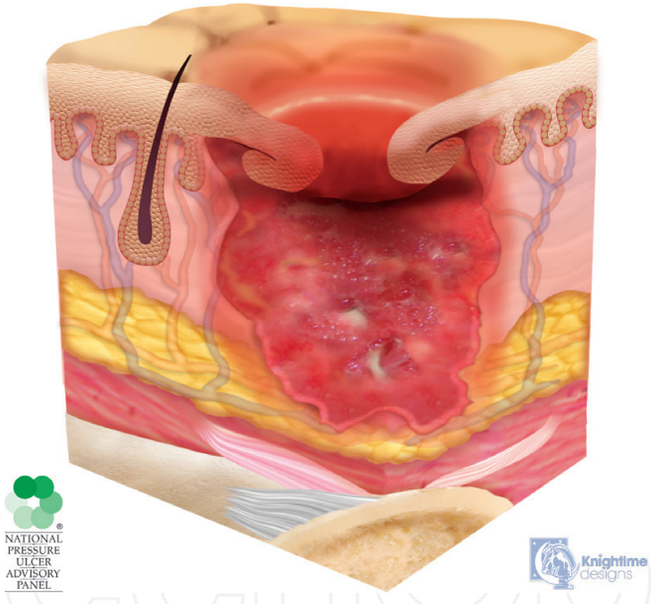


Figure 8. Stage 3 pressure injury with epibole. Illustrations for figure is used with permission from Ref. [4].

may be visible as well as epibole (rolled edges). Undermining and/or tunneling often occur in Stage 4 wounds. Depth varies by anatomical location. If slough or eschar obscures the extent of tissue loss then it should be classified as an unstageable pressure injury [4] (**Figure 9**).

7.1.5. Unstageable pressure injury

Full-thickness skin and tissue loss in which the extent of the tissue damage within the ulcer cannot be confirmed because it is obscured by slough or eschar. Once the slough or eschar is removed, a Stage 3 or Stage 4 pressure injury will be revealed. Stable eschar is often dry,

adherent, and intact without erythema or it is commonly on the heel or on an ischemic limb and should not be softened or removed [4] (**Figures 10** and **11**).

7.1.6. Deep tissue pressure injury (DTPI)

Intact or non-intact skin with a localized area of persistent non-blanchable deep red, maroon, purple discoloration or an epidermal separation revealing a dark wound bed or a blood filled blister. Discoloration may appear differently in darkly pigmented skin. Pain and temperature changes often precede the skin color changes. This injury results from intense and/or

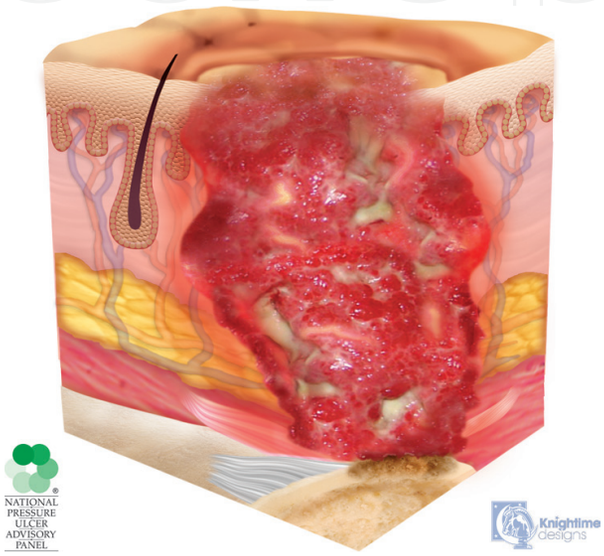


Figure 9. Stage 4 pressure injury. Illustrations for figure is used with permission from Ref. [4].

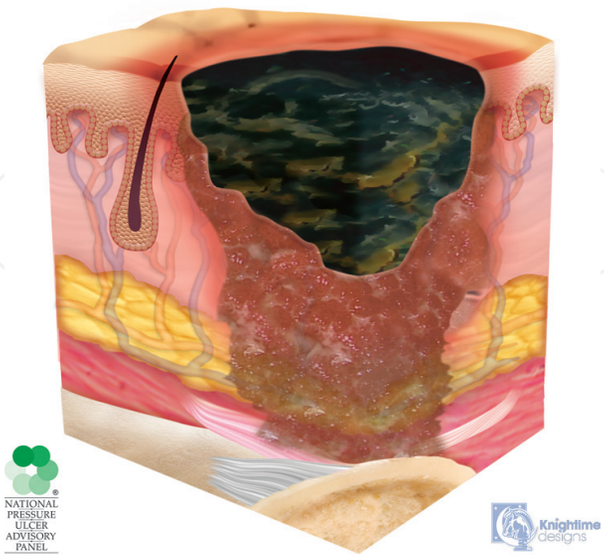


Figure 10. Unstageable pressure injury—Dark Eschar. Illustrations for figure is used with permission from Ref. [4].

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prolonged pressure and shear forces at the bone-muscle interface. The wound may evolve rapidly to reveal the actual extent of tissue injury, or may resolve without tissue loss. If there is necrotic tissue, subcutaneous tissue, granulation tissue, fascia, muscle or other underlying structures visible, this indicates a full thickness pressure injury—Unstageable, Stage 3 or Stage 4. Do not use DTPI to describe vascular, traumatic, neuropathic, or dermatologic conditions [4] (**Figure 12**).

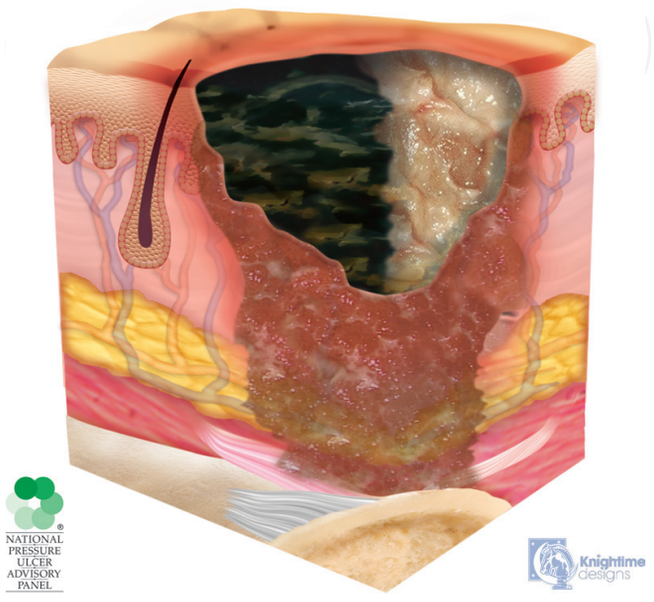


Figure 11. Unstageable pressure injury—Slough and Eschar. Illustrations for figure is used with permission from Ref. [4].

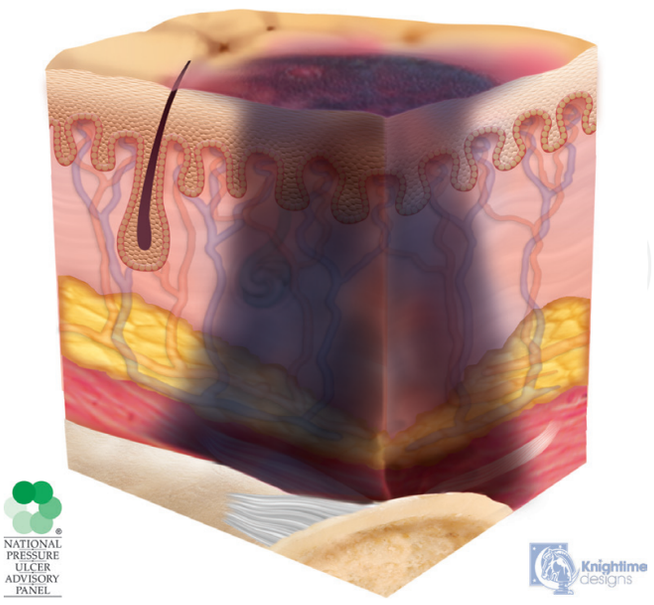


Figure 12. Deep tissue pressure injury. Illustrations for figure is used with permission from Ref. [4].

7.1.7. Additional terms of importance as defined by the NPUAP

7.1.7.1. Medical device related pressure injury

Medical device related pressure injuries result from the use of devices designed and applied for diagnostic or therapeutic purposes. The resultant pressure injury generally conforms to the pattern or shape of the device. The injury should be staged using the staging system [4].

7.1.7.2. Mucosal membrane pressure injury

Mucosal membrane pressure injury is found on mucous membranes with a history of a medical device in use at the location of the injury. Due to the anatomy of the tissue, these injuries cannot be staged [4].

7.1.7.3. Important points

The deterioration of a pressure injury does not predictably follow a linear evolution from Stage 1 to Stage 4 [6]. In addition, only pressure injuries should be staged with the NPUAP Pressure Injury Staging System. Many non-pressure-related ulcers and wounds are subject to unique staging or classification systems based upon the wound type for example: diabetic foot ulcers (Wagner Classification System), venous leg ulcers (Clinical Etiology Anatomy Pathophysiology), skin tears (International Skin Tear Advisory Panel), adhesive or tape injuries (medical adhesive related skin injury categories (MARSI)), and burn classification (total body surface area). It is essential that the intended staging or classification system be used for each type of injury to ensure appropriate treatment [6].

8. Treatment of pressure injuries

Managing and treating pressure injuries is only effective when a multidisiplinary team approach is utilized in the management of these patients. One must incorporate teams across several specialties and address the patient's issues as a whole in order for him/her to heal quickly and successfully. Below, we will briefly address some aspects in patient care to be mindful of when constructing treatment strategies; addressing these topics early in the management process will aid you in achieving positive outcomes.

8.1. Manage co-morbidities

Patients may have many comorbidities in addition to their pressure injury all of which need to be addressed and managed appropriately; for example stabilize glycemic control in diabetics and assess peripheral artery disease with a screening ABI ankle-brachial index (ABI) in vascular patients. An in depth discussion of the management of comorbid conditions will not be included in this chapter but be mindful to review all of the patient's comorbid conditions and assess whether or not there are correlations to wound care that need to be addressed.

8.2. Optimize nutritional status

Poor nutritional status can be a contributing risk factor toward the formation of a pressure injury and can also contribute toward nonhealing. No matter what stage of pressure injury your patient is diagnosed with, if the patient's nutritional status is not optimized he/she will have a delayed healing evolution or even worse—never heal due to lack of energy. Serologic markers such as hemoglobin, serum albumin, prealbumin, transferrin and total lymphocyte count may be beneficial in assessing a patient's nutritional status. You should also use a valid and reliable nutritional screening tool to determine the patient's nutritional risk.

One should determine the patient's daily caloric requirements to ensure they are obtaining enough fuel to support all of the patient's energy expenditures, known as the total energy expenditure (TEE) [29]. During illness, injury, or times of stress, the body may become hypermetabolic. Typically a patient with a wound requires additional calories to offset the hypermetabolic response triggered by the wound [29].

1) A simple way to calculate an estimate of caloric needs is by using a standard number of kcals/kg of body weight per day. Typical standards are listed below:

a. Daily Total Energy Expenditure Estimation

Normal Maintenance: 25–28 kcal/kg
Mild-Moderate stress/illness, injury or malnutrition: 30–35 kcal/kg
Severe major stress, critical illness or injury: 35–40 kcal/kg [25, 29].

Guidelines from the Agency for Health Care Policy and Research (now the Agency for Healthcare Research and Quality) state that approximately 30–35 kcals/kg/day are required for most patients with Stage 2 wounds or pressure injuries in risk of malnutrition. Increase to 35–40 kcal/kg/day if the patient is underweight or is losing weight; these estimates will likely achieve the desirable positive nitrogen balance [25, 29, 30].

2) A more precise formula to determine caloric requirements is to use the three-component equation:

Caloric Requirements = Harris Benedict Equation × Injury Factor × Activity Factor [29]

a. Daily Basal Energy Expenditure Estimate (aka Harris Benedict Equation)

Males: 66.5 + (13.7 × Wt. in kg) + (5.0 × Ht. in cm) − (6.8 × age in Yrs.)

Females: 655 + (9.6 × Wt in kg) + (1.8 × Ht in cm) − (4.7 × age in Yrs.) [29]

b. Injury Factor (IF)

IFs are variable and subjectively based according to the practitioner's judgment. Use the numbers below as a guide:

Normal, minor Surgery, burn post graft = 1.0–1.2

Long Bone Fracture = 1.2

COPD, Malnourished = 1.3

Severe Head Injury = 1.4

Cancer = 1.0–1.5

<50% Burns = 1.5

Ventilator = 1.6

Major Surgery, Multiple Traumas, 0–20% Burns Pre Grafting = 1.2–1.6

Acute Sepsis = 1.2–1.7

Severe infection = 1.4–1.8

20–40% Burn Pre Grafting 1.5–2.0

50% Burn = 2.0 [29, 31]

c. Activity Factor (AF)

1.2—Confined to bed

1.3—Out of bed

1.5—Normal, healthy activity [29, 31]

8.3. High protein diet

Provide a high protein diet (1.25–1.5 g/kg/day) to maintain a positive nitrogen balance for adults assessed to be at risk or with an existing pressure injury. A high protein diet in combination with arginine and micronutrient supplements is ideal. Arginine enriched mixed nutritional supplements have been shown to improve mean pressure ulcer healing time [32]. Literature has also shown that an arginine, zinc and antioxidant enriched nutritional formula increased the rate of healing in malnourished adults, [33] yet zinc supplementation alone appeared ineffective and was associated with adverse effects [34]. Thus, possibly it's the combination of both arginine and zinc that improves wound healing. Nutritional shakes in addition to vitamin and mineral supplements are strongly advised. If patients are unable to tolerate oral intake consider enteral or parenteral nutritional substitutions.

8.4. Pain management

While some pressure injuries lead to insensate tissue others injuries can be extremely painful. Pain management strategies should be addressed especially prior to any cleansing or debridement. An in depth discussion of pain management options will not be included in this chapter. However, the application of topical opioids such as benzydamine 3% or diamorphine gel (not available in the US) as well as topical anesthetics such as lidocaine-prilocaine should be considered [25].

9. Wound care: cleansing

Clean all pressure injuries at each dressing change to help facilitate removal of debris and bacteria. Water or normal saline are appropriate to use as cleansers and irrigation agents for most pressure ulcers. For compromised patients, wounds, or wound healing environments consider aseptic techniques. If there is debris, infection or high bacterial colonization, consider using a cleanser with surfactants and/or antimicrobials. Apply your choice of cleansing solution with ample pressure to clean the wound of debris without damaging the tissue, which may be friable. For each dressing change, a new, unopened container of cleansing solution should be used. Used irrigation solution should be appropriately discarded [25].

10. Debridement

If you are concerned about whether or not a wound needs to be debrided, a surgery consult should be placed. Surgeons have been exposed to a wide variety of wounds and are experts on assessing whether or not tissue debridement is indicated. All necrotic and nonviable tissue needs to be removed so that new granulation tissue may form; this includes the wound bed and edges. Maintenance debridement should occur frequently until all devitalized tissue is removed and the wound is covered with granulation tissue. There are no surgical indications to debride stable, hard, dry eschar in ischemic limbs because there is inadequate perfusion and it will not revitalize [25].

10.1. Infection

One should always have a high index of suspicion for infection in pressure injuries especially if there is necrotic tissue present or if the wound is deep or large in size. Anatomic location also plays a large role in infection control particularly when the area is prone to exposure of contaminants (e.g., near the anus). Ulcers with signs of infection are in need of urgent surgical debridement—emergent surgical debridement if necrotizing fasciitis is suspected. Some signs of necrotizing fasciitis include rapidly spreading erythema, tenderness out of proportion to the physical attributes of the wound and crepitus. Beware of foreign bodies within wounds; they are commonly a nidus for infection. Patients at high risk of infection include those with diabetes mellitus, malnutrition, autoimmune diseases and immunosuppression [25].

If there are clinical signs of spreading infection, such as extension of erythema beyond the wound, induration, purulence, increase in erythema, warmth or pain, obtain a quantitative culture via tissue biopsy or less preferably quantitate swab technique. An initial trial of antibiotics it is reasonable to administer. If you are dealing with a nonhealing pressure injury, needle aspiration or an ulcer biopsy for culture should be performed to determine bio-burden/microbial load. In the absence of clinical signs of infection, the quantity of the organisms or microbial load is believed to be the best indicator of wound infection. Superficial wound cultures are not generally recommended due to the fact that all lesions are colonized with bacteria

and superficial swab cultures are unreliable. The gold standard for determining microbial load is a quantitative culture of viable biopsied wound tissue [25].

The ulcer bed should be debrided if a biofilm is suspected. Be suspicious of biofilm formation if the wound has been present for >4 weeks, if healing still appears delayed after 2 weeks of attention and treatment, or if there is no response to antibiotics. Confirmation of a biofilm can be obtained once biopsy culture shows ≥ 10^5 colony forming units (CFU)/gram tissue. Beta hemolytic streptococci is a common culprit [25].

11. Debridement options

The most common debridement options include: surgical sharp, mechanical, autolytic, enzymatic and larval. The method of debridement chosen depends upon the condition of the wound, the health status of the patient as well as the capabilities and confidence of the healthcare provider. More than one debridement method may be deemed appropriate for a given pressure injury and multiple modalities can be combined as well.

11.1. Surgical-sharp debridement

This type of debridement involves the use of instruments such as scissors, scalpels and forceps to remove necrotic, nonviable tissue from the wound [35]. Surgical debridement can be performed so that it extends into healthy tissue or conservative-sharp debridement can be performed which does not extend into or excise healthy tissue. Surgical debridement is an appropriate debridement option when a large amount of necrotic tissue needs to be removed from the wound bed. Surgical sharp debridement is indicated for extensive necrosis, advancing cellulitis, crepitus, fluctuance and/or when sepsis is related to the pressure injury. Perform urgent surgical debridement for ulcers with erythema, tenderness, edema, purulence, fluctuance, crepitus and malodor suggesting an infection around the injury. Most of the presentations listed above require operating room equipment, however, moderate surgical debridement may be performed by a competent medical professional at the bedside using a sterile scalpel or scissors. Extensive debridement should be executed in the operating room [25].

11.2. Mechanical debridement

Mechanical debridement physically removes nonviable, necrotic tissue with techniques such as wet-to-dry dressings, dry gauze and hydro-surgery such as wound irrigation and whirlpool techniques [35]. The wet-to-dry dressing system is one of the oldest forms of wound debridement. This method typically consists of applying moist to wet dressings to the wound, which are manually removed later once the dressings have dried. This causes non-selective debridement of necrotic tissue, eschar and slough; unfortunately sometimes viable, newly formed tissue is damaged and/or removed as well. This form of debridement can also be very painful to the patient if the dressings are very dry and hardened.

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Pulsed lavage as well as high- or low-pressure streams can be quite effective in removing loose necrotic tissue from the wound bed. A common practice after surgical wound debridement is pulsed lavage treatment for wound irrigation. Wound vacuum cleaning is thought to improve the healing of pressure injuries [36]. Ulcer irrigation pressures range from 4 to 15 psi of pressure; this range typically yields successful debridement yet does not cause trauma to the wound bed. Irrigation pressures below 4 psi may not be effective to cleanse the wound and pressures greater than 15 psi may cause trauma and drive the bacteria into the tissue [35]. Some pulsation devices allow copious amount of irrigation, which can be infused with antibiotics. These medical devices are certainly used in the operating room and portable versions are now available for bedside use in the appropriate setting. Normal saline "flushes" through a 60 cc Toomey syringe is another technique that works well to aid with debridement and a copious amount of irrigation is possible with this technique.

Whirlpools may be used initially to loosen and remove debris, bacteria, exudates, and necrotic tissue however prolonged use and periods of wetness may macerate the tissue or may be associated with bacterial contamination. NPUAP guidelines advise against whirlpool use for routine use in treating pressure ulcers due to the potential for contamination and the emergence of newer hydrotherapies [25]. In addition, individuals with dependent lower extremity edema or peripheral vascular disease, [37] immunocompromised individuals, those who are mechanically ventilated and lethargic, and incontinent individuals should never be immersed in water [25, 38].

11.3. Autolytic debridement

Autolytic debridement uses the body's own endogenous enzymes and moisture to break down tough eschar and slough. This form of debridement is accomplished by moist interactive dressings, which allow the patient's natural wound fluid and endogenous enzymes to be kept in constant contact with the wound. The self produced fluid and endogenous enzymes soften and liquefy slough and promote granulation tissue formation [35]. The dressings used can be occlusive or semi-occlusive and consist of various materials such transparent films hydrogels and hydrocolloids. These dressings will be discussed more detail in the Wound Dressings section. Endogenous enzymes do not damage healthy skin however the wound will need to be cleaned after autolytic debridement in order to remove the necrotic debris. The time to break down dead and devitalized tissue can be slow and is quite variable. If tissue autolysis is not apparent in 1–2 weeks, another debridement method should be used [35]. Autolytic debridement is not recommended for infected wounds or very deep wounds that require packing [35, 38].

11.4. Enzymatic debridement

Enzymatic debridement is achieved by topical application of exogenous enzymes to the wound surface to remove necrotic tissue [35]. The chemical agents are useful for debriding wounds with a large amount of necrosis and/or eschar. While enzymatic debridement effectively breaks down nonviable tissue, it also may damage nearby healthy tissue as well. In addition, these agents must be prescribed and a small tube can be quite costly. Enzymatic

debridement is thought to be faster than autolytic debridement however it may cause increased wound pain and /or an uncomfortable burning sensation to the patient. Enzymatic debridement is most useful in patients who cannot tolerate sharp debridement and should not be used if an infection is present. Preparations available in the US include collagenase and papain/urea with or without chlorophyll [38].

11.5. Larval therapy/Medicinal maggot therapy

Maggot debridement therapy is the intentional application of live, "medical grade" fly larvae to wounds. Medicinal maggots have been sited to perform major actions such as debridement, disinfection, stimulation of wound healing as well as biofilm inhibition and eradication [39]. Larval debridement is unique in that maggots selectively only engulf necrotic tissue—no healthy tissue is affected. This method is gaining in popularity due to its low cost, incredible effectiveness and expedited time for successful debridement. Some patients however find the method somewhat painful and their perception of maggots may stand in the way of using this highly effective method of debridement [38].

12. Topical agents

12.1. Topical antiseptics

When it comes to topical agents for pressure injuries the options are vast. Indications for specific topical agents can vary as the pressure stage progresses or the wound starts to heal. Antiseptic agents commonly used in wounds include: iodine (povidone, iodine or slow release cadexomer iodine), silver (silver sulfadiazine), polyhexanide and betaine (PHMB), chlorhexidine, sodium hypochlorite and acetic acid [25]. Topical antiseptics should be used for pressure ulcers that are not expected to heal, are critically colonized or topically infected. They should be used in conjunction with maintenance debridement. In managing biofilms, consider topical antiseptics to control bacterial bio-burden in wounds with delayed healing [25].

Be aware that some skin cleaners or antiseptics may destroy granulation tissue [10]. Avoid hydrogen peroxide as it is toxic to tissues even at low concentrations [40, 41]and should not be used as a preferred topical antiseptic. Its use should be completely avoided in cavity wounds due to the risk of surgical emphysema and gas embolus [28, 41–43].

Iodine products should be avoided in patients with impaired renal failure, history of thyroid disorders or known iodine sensitivity [44, 45]. There is a risk of acidosis when acetic acid is used for extended periods over large wound surface areas [46]. Silver may have toxic properties, especially to keratinocytes and fibroblasts; the extent of the toxicities however, is not fully described. Topical silver products should not be used on individuals with silver sensitivities, and silver sulfadiazine products are not recommended for people with sulfur sensitivities [47]. It is however appropriate to consider using silver sulfadiazine in heavily contaminated or infected pressure ulcers until definitive debridement is accomplished. Sodium hypochlorite

(Dakin's solution) is cytotoxic at all concentrations and should be used with caution, at concentrations no greater than 0.025%, for short periods only when no other appropriate option is available [25, 48–50].

12.2. Other agents of interest

New, less commonly used agents' effectiveness appear promising. Atorvastatin powder mixed with petroleum jelly and beeswax forms an ointment, which has been used as a topical agent resulting in a reduction in the size of Stage 1 and 2 pressure ulcers [51]. Recombinant human platelet-derived growth factor (rPDGF) has been shown to accelerate the healing of pressure ulcers [52]. Topical phenytoin may be more effective than DuoDERM dressing, saline dressings and topical antibiotics for treating pressure ulcers [53].

13. Antimicrobials: systemic & topical

13.1. Systemic

Consider a systemic antibiotic only if clinical signs of systemic infection are present or there is a positive blood culture, cellulitis, fasciitis, osteomyelitis, systemic inflammatory response syndrome (SIRS) or sepsis. If systemic antibiotics are given, empiric antibiotics should cover methicillin-resistant *Staphylococcus aureus*, *anaerobes*, *enterococci* and Gram-negatives such as *Pseudomonas*, *Proteus* and *Providencia* species [25].

13.2. Topical

There is insufficient evidence to support whether topical antimicrobials improve wound healing or reduce infection in patients with ≥Stage II pressure ulcers. Generally topical antibiotics are not recommended and should be limited or avoided if possible. Use is indicated in specific situations where the topical antibiotics' benefits outweigh the risks of antibiotic side effects and resistance. Consider topical Metronidazole for control of odor due to anaerobic bacteria and protozoal infections [25].

13.3. Osteomyelitis & abscesses

Osteomyelitis can be a detrimental finding in any patient thus rapid identification and treatment is necessary. If osteomyelitis is suspected in a patient's wound bed, obtain blood cultures and sensitivities, leukocyte count, erythrocyte sedimentation rate and a C-reactive protein level. Imaging should also be obtained. A plain X-ray is the initial imaging study of choice. Once osteomyelitis is confirmed, a CT scan is useful for needle guided biopsies, identifying necrotic bone and soft tissue extension. The gold standard test of choice for detection of early osteomyelitis and associated soft tissue disease is an MRI with gadolinium [25].

Cultures at the time of presentation or first debridement may not be useful. Swabbing cultures from draining wounds and sinus tracts may not be reliable for predicting organisms

that will be isolated from infected bone, but might be helpful for infection control measures. Obtaining cultures of bone biopsies after an initial debridement is performed can be helpful in guiding therapy. Both aerobic and anaerobic cultures should be sent for culture; if within a few days there is no growth then fungal and mycobacterial cultures should be processed. Drain all local abscesses [25].

14. Types of wound dressings

There are many factors to consider in choosing a wound dressing such as the ulcer size, depth and location, the ability to keep the wound bed moist, the need to address bacterial bio-burden, the nature and volume of wound exudate, the condition of the tissue in the ulcer bed, the condition of periulcer skin, the presence of tunneling and/or undermining as well as the goals of the individual with the pressure injury [25]. Ideally, one should choose a dressing that maintains a moist wound environment and controls exudate without desiccating the ulcer bed all while promoting wound healing. A moist wound environment physiologically favors migration and matrix formation while accelerating healing of wounds by promoting autolytic debridement [35]. It is also thought that moist wound healing reduces wound pain [35]. Keeping the wound environment clean may be a constant challenge especially when the ulcers are in unfavorable, not easily accessible locations however, any contaminated environment will make wound healing extremely difficult.

14.1. Hydrocolloid dressings

Hydrocolloid Dressings are a wafer-type dressing that contains hydro-active particles which, when in contact with wound exudate, forms a fluid/gel environment over the wound bed. Both occlusive and semi-occlusive dressings have been shown to provide and maintain a moist, hypoxic wound environment, provide protection and insulation to a healing wound and facilitate autolytic debridement [54, 55].

Hydrocolloid dressings can be self-adhering with a surface that repels water, bacteria and other outside contaminants. Various specific types of hydrocolloid dressings are on the market in sheet, paste, gel, and powder form. They may differ in size, shape, exudate absorption and intended use [55, 56]. Hydrocolloid Dressings are indicated for light to moderate exuding wounds, typically Stage 2 and shallow Stage 3 pressure injuries. They should be used in areas where they will not roll or melt. Filler dressings beneath hydrocolloid dressings can be used in deep ulcers in order to fill in the dead space. They can often be left on a wound for several days and can be used as a primary or secondary dressing [25, 57].

Contraindications to hydrocolloid dressing use include infected wounds, wounds with heavy exudate, deep cavities, sinus tracts (unless used as a secondary dressing over the packing), burns and grafts.

Examples: DuoDERM, Tegaderm Thin, Reliamed border sacral, Reliamed beveled, Flexicol bordered, Medihoney sheet (hydrocolloid with honey), Medihoney tube [57] (**Figure 13**).

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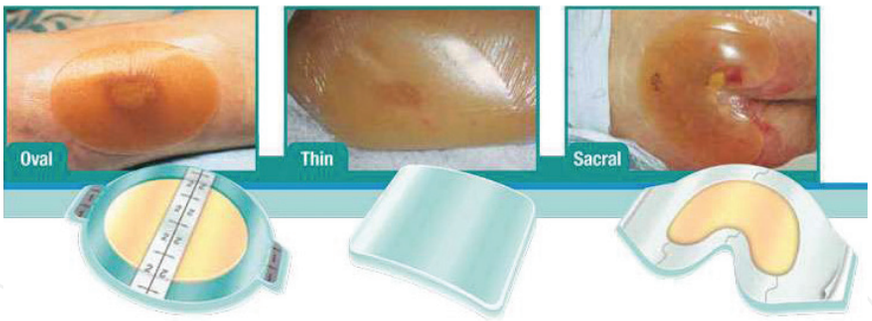


Figure 13. Hydrocolloid dressings.

14.2. Transparent film dressings

Transparent film dressings are semipermeable membranes, generally polyurethane, which are self-adhering, thin and waterproof. They allow gaseous exchange between the wound bed and the environment, but water, bacteria and other contaminants cannot penetrate the dressing. These dressings have a wide variety of uses because they are available in multiple shapes and sizes to conform to many different wounds. The transparency of the dressing allows for wound visualization, assessment of its healing progress and any drainage [25, 57]. Transparent film dressings are indicated for partial thickness wounds, Stage 2 ulcers and dry, necrotic wounds requiring debridement but should not be used in moderate to heavy exuding ulcers. These dressings help to maintain a moist wound surface and facilitate autolytic debridement. They should be avoided in immunocompromised individuals. They can be used as a secondary dressing with alginates or other wound fillers that will likely remain in the ulcer bed for 3 days or more however should not be used over enzymatic debriding agents, gels, or ointments [25, 57].

Contraindications to transparent film dressing use include heavy exudating wounds, deep cavities, sinus tracts, undermining (unless used as a secondary dressing) and wounds with friable skin in the periwound area.

Examples: 3 M Tegaderm, Mefilm [56] (**Figure 14**).

14.3. Hydrogel dressings

Hydrogel dressings vary in composition but most are water or glycerin based, depending on the manufacturer and have a gel consistency. The non-occlusive dressing comes in sheets, beads, impregnated in gauze and in gel form. These dressings are indicated for shallow, minimally exuding pressure ulcers that are not clinically infected and are granulating. They may also be used on painful, dry wounds needing additional moisture such as burns and grafts because they will add additional moisture. These dressings work to provide moisture to a wound site while eliminating or preventing infection however their use is somewhat limited to dry and low exuding wounds because they can cause maceration to surrounding tissues when high volumes of the wound exudate is present [55, 58]. Hydrogel dressings work well on pressure injuries that have contours and/or on body areas that are at risk for wound

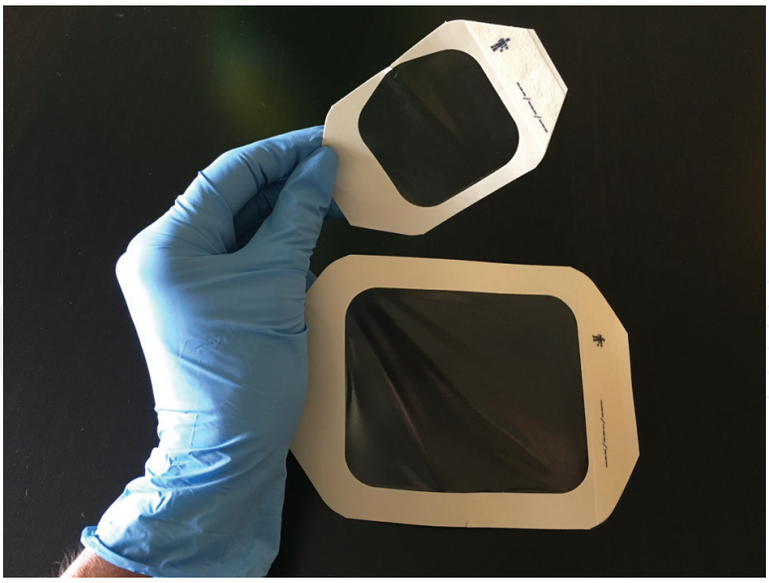


Figure 14. Transparent film dressings.

dressing migration. The sheet form of hydrogel can be used for hastier healing of cosmetic procedures and/or scar reduction. These dressings typically have to be changed daily but are biocompatible with other dressings [57].

There are no specific contraindications to hydrogel dressings, however there is a risk that when hydrogel dressings are used on exuding ischemic ulcers a resultant shift from dry to wet gangrene within the wound can take place [55, 59]. Also, because of the moist nature of these dressings, care must be taken to observe the wound edges and protect the skin from maceration.

Examples: Skintegrity, SpanGel, Normlgel [57].

Of note, Hydrogel dressings are also available with additives such as silver for antimicrobial benefits. However, Hydrogel Silver dressings should not be used in conjunction with Tegaderm Matrix as the deactivation of both dressings will result [25, 57].

14.4. Alginate dressings

Alginate dressings are hydrophilic, non-woven fiber dressings that are derived from brown seaweed [55]. When the dressing contacts a wound's exudate it forms a gel mass in the wound. Alginate comes in a sheet or rope form and is absorbent. The dressing will absorb excess exudate while maintaining a moist wound environment. It facilitates autolytic debridement of loose, necrotic tissue and is indicated for partial thickness and full thickness wounds having moderate to heavy exudate. It is effective in filling cavities, tracts and undermining. A secondary dressing should be used to cover the alginate dressing. Irrigation will aid in its removal from the wound bed. Consider lengthening the interval between wound dressing changes or changing the type of wound dressing if the alginate dressing is still dry at the scheduled time for a dressing change. Alginate dressings are contraindicated in dry wounds [25, 57].

Examples: Maxorb, Algicel, Melgisorb, Reliamed, Sorbion Sachet S [57].

Of note, this dressing also comes in a silver added form for antimicrobial benefits. The silver form is not to be used with hydrogels and should not be used with products that create an ionic exchange in the wound site. Alginate dressings cannot be used with enzymatic products. (ex Santyl Collagenase)

Examples: Maxorb Extra AG, Silverlon CA, Algicel AG, Reliamed AG-CMC (hydrofiber) [57] (**Figure 15**).



Figure 15. Sheet & strip alginate wound dressings.

14.5. Gauze dressings/"Wet-to-Dry Gauze"

The NPUAP's guidelines recommends avoiding use of gauze dressings for open pressure ulcers that have been cleansed and debrided because they are labor-intensive, cause pain when removed if dry, and can lead to desiccation of viable tissue. The association also advocates against the use of wet-to-dry gauze dressings. Gauze dressing is an economical means of debridement that can be effective when preformed often and appropriately. While the initial cost of moist gauze is less expensive than more advanced wound care products, the notion that moist gauze is more cost effective than all other wound care options may not be entirely true. When determining cost efficacy, one must look at all factors involved in the wound healing process. In addition to the product cost, one must take into account health care provider time, patient care goals and resources, ease of use and healing rate [35]. Once all of these aspects are accurately accounted for, gauze may not be the most economical wound care supply. When other forms of moisture retentive dressings are not available, continually applying moist gauze is preferable to dry gauze. Dry gauze dressings should only be used as the cover dressing to reduce evaporation when the tissue interface layer is moist. Consider using impregnated forms of gauze to prevent evaporation of moisture from continuously moist gauze dressings [25, 57].

Example: Kerlix [57].

14.6. Recommended gauze application technique

If gauze is to be applied as your wound dressing it is recommended that loosely woven gauze be used to treat highly exuding ulcers so that a high volume of the exudate may be absorbed.

Tightly woven gauze should be used for minimally exuding ulcers. Loosely fill ulcers with large tissue defects and dead space with salinemoistened gauze when other form of moisture retentive dressings are not available; this technique is preferred over tightly packing the wound which can lead to excessive pressure on the ulcer bed. Change gauze packing often enough to manage the exudate. It is preferable to use a single gauze strip or roll to fill deep ulcers rather than using multiple gauze dressings; as this will reduce the risk of retained gauze in the ulcer bed, which can serve as a nidus for infection [25].

14.7. Collagen matrix dressings

A collagen matrix dressing is a naturally derived scaffold dressing. Scaffolds facilitate infiltration of cells such as fibroblasts and keratinocytes through pores of a controllable size while maintaining optimal healing conditions [55]. Ideally, a dermal scaffold mimics the tissue's natural extracellular matrix, allowing bioactive molecules to be incorporated while also being biocompatible [55]. Collagen matrix dressings are used for nonhealing Stage 3 and 4 pressure wounds. These dressings come in pads, gels or particles and promote the deposit of newly formed collagen into the wound bed. They can be used on any type of wound with minimal, moderate or heavy drainage. Some specific brands require the wound to be free of necrotic tissue but not all. The collagen provides a moist healing environment and promotes tissue granulation and epithelialization. Some brands reduce destructive elements within wound fluid to trigger healing while allowing the patient's growth factors to effectively heal the wound. No specific contraindications exist however, collagen may enable the transmission of infectious agents and thus it requires vigorous disinfection protocols [55, 57].

Examples: Pads—Prisma, Promogran (collagen with silver), BioPad, Fibracol Plus (alginate/collagen combination); Gel—Stimulen Gel; Powder—Stimulen [57].

14.8. Silicone dressings

Silicone dressings can be considered as a wound contact layer to promote atraumatic dressing changes or as the contact layer within a dressing, for example, Mepilex, a polyurethane foam membrane is coated with a soft silicone layer [55, 60]. Consider silicone dressings to prevent periwound tissue injury when the surrounding tissue is fragile or friable. Silicone can be used on a range of acute and chronic wounds as it is incorporated in many different bandaging strategies [55, 57].

Examples: Mepitel, Mepliex Ag [57].

14.9. Foam dressings

Polyurethane foam dressings have an absorbent wound contact surface and typically also a moisture-repellant outer surface, which is non-occlusive. Polyurethane foam dressings are easy to use and customize as they can be cut to shape and come in a range of absorbencies [55]. Foam's ability to absorb exudate is dependent upon the thickness and density of the dressing. Foam dressings are designed to maintain a moist wound environment but absorb excess exudate thereby preventing maceration of the surrounding healthy tissue. Foam also

has thermal insulation and protection properties. Removal of the foam often facilitates the removal of slough. Foam may be used as a primary or secondary dressing and is available in self-adhering or non-adherent forms although some may have adhesive border. Foam dressings are indicated for partial and full thickness wounds with moderate to heavy exudate. They also can be used to cover wounds containing packing material. Contraindications to its use are wounds with dry eschar as well as wounds with minimal to no exudate [25, 57].

Examples: Mepilex (with Safetac technology), Mepilex Border (water resistant with Safetac technology), Tegaderm foam, Polymem, Polymem Dot (with silver) [57].

15. Special dressings

15.1. Silver-impregnated dressings

One can consider using silver-impregnated dressings for pressure ulcers that are clinically infected, heavily colonized or at high risk of infection. Prolonged use of silver-impregnated dressings should be avoided and silver dressings should be discontinued once the wound infection is controlled. As mentioned earlier, silver may have toxic properties but it is unknown to what extent [25].

15.2. Honey impregnated dressings

Use of medical-grade honey as well honey impregnated dressings has been used in heavily contaminated or infected pressure ulcers. Honey impregnated dressings have been used on Stage 2 and 3 pressure ulcers until definitive debridement is accomplished. Before applying a honey dressing, ensure the individual is not allergic to honey. Individuals who have bee or bee stings allergies are usually able to use properly irradiated honey products [25].

15.3. Cadexomer iodine dressings

Consider using cadexomer iodine dressings in moderate to highly exuding pressure ulcers. Iodine products should be avoided in individuals with impaired renal failure, history of thyroid disorders or known iodine sensitivity [44, 45]. Iodine dressings are not recommended for individuals taking lithium or for pregnant or breast-feeding women. The risk of systemic absorption increases when iodine products are used on larger, deeper wounds or for prolonged periods of time. Iodine toxicity has been reported in a few case studies, especially in those individuals with large wounds, in whom dressings were changed often [25].

16. Biologic dressings

16.1. Recombinant platelet-derived growth factor

Consider using platelet-derived growth factors for treatment of Stage 3 and 4 pressure ulcers that have delayed healing. (Refer to topical agents section for more information) [25].

16.2. Other growth factors

Due to insufficient evidence to support or refute the use of growth factors (other than recombinant platelet-derived growth factor) in the treatment of pressure ulcers, they are not recommended for routine use at this time by the NPUAP guidelines [25].

17. Biophysical agents

A number of biophysical agents have been studied in the management of pressure ulcers. All provide some form of biophysical energy with the goal of promoting healing. Common forms of biophysical agents include energy from the electromagnetic spectrum (e.g., electrical stimulation, electromagnetic fields, pulsed radio frequency energy and phototherapy), acoustic energy (high and low frequency ultrasound) and mechanical energy (e.g., heric energy [hyperbaric and topical oxygen]) [25]. Many of the above mentioned biophysical agents are uncommon and are out of the chapter's scope of coverage. Only commonly used biophysical agents will be addressed.

17.1. Mechanical energy—negative pressure wound therapy (NWPT)

Consider negative pressure wound therapy (NWPT) as an early adjuvant for the treatment of deep, Stage 3 and 4 pressure ulcers. NWPT is not recommended in inadequately debrided, necrotic or malignant wounds; where vital organs are exposed; in wounds with no exudate, or in individuals with untreated coagulopathy, osteomyelitis or local or systemic clinical infection. Cautious use by an experienced health professional is recommended for individuals on anticoagulant therapy; in actively bleeding wounds; or when the wound is in close proximity to major blood vessels [25, 61]. Debride the pressure ulcer of necrotic tissue prior to the use of NPWT. Follow a safe regimen in applying and removing the NPWT system and evaluate the pressure ulcer with each dressing change. The optimal dressing change interval has not been well established, and should be based on characteristics of the individual and the wound. If pain is anticipated or reported consider tactics such as placing a nonadherent interface dressing on the wound bed, underneath the foam, lowering the level of pressure, changing the type of pressure (continuous or intermittent), and/or using a moist gauze filler instead of foam [25].

17.2. Kinetic energy—hydrotherapy—pulsatile lavage with/without suction

Refer to Debridement section 11 for more information

17.3. Kinetic energy—hydrotherapy—whirlpool

Refer to Debridement section 11 for more information

17.4. Atmospheric energy—oxygen

Hyperbaric oxygen therapy (HBOT) and topical oxygen therapy can both be used in the treatment of chronic wounds. Due to an insufficient amount of evidence to support or refute the

use of HBOT as well as topical oxygen therapy in the treatment of pressure ulcers, these forms of oxygen therapy are not recommended for routine use according to the NPUAP guidelines at this time [25].

18. Physical treatment tactics

18.1. Repositioning & offloading techniques

Repositioning & offloading techniques as well as early mobilization are extremely important in the prevention and treatment of pressure ulcers. Repositioning so that pressure is relieved or redistributed will reduce the duration and magnitude of pressure over vulnerable areas of the body. All individuals at risk of or with existing pressure ulcers, should be repositioned often unless contraindicated; this will contribute to comfort, hygiene, dignity, and functional ability. Inspect the skin for changes and any new or developing transformations at every position. Frequent assessment will allow for early identification of signs of pressure damage such as non-blanchable erythema [25].

If skin changes occur or the individual is not responding as expected to the repositioning regime then the frequency, method and technique of repositioning should be reassessed. Controlling the patients positioning contributes to maintenance of capillary circulation [62]. One should avoid positioning the patient on bony prominences with existing non-blanchable erythema as the pressure and/or shearing forces sustained will further occlude the blood supply to the skin, worsen the damage and result in more severe pressure ulceration [25].

Safe manual handling techniques should be utilized to ensure the safety of both the patient and the health professional. Use simple, manual handling aids such as lift sheets to help reduce friction and shear. Lift—do not drag the patient when repositioning. Do not leave moving equipment under the patient after use, unless the equipment is specifically designed for that purpose. Avoid positioning patients directly onto medical devices, such as tubes, drainage systems or other foreign objects. Patients should not be left on a bedpan longer than necessary [25].

18.2. Repositioning frequency

The frequency of repositioning will vary from patient to patient. Pay close attention and consider the patient's tissue tolerance, level of activity and mobility, general medical condition, overall treatment objectives, skin condition, and comfort when determining a patient's frequency of off loading. While medical professionals primarily aid in repositioning, the patient and his/her family should be educated and taught pressure-relieving maneuvers to do themselves throughout the day [25].

18.3. Repositioning individuals in bed

Position patients in the 30–40° tilted side-lying position (alternating, right side, back, left side) or the prone position if tolerable. Encourage patients who can reposition themselves to sleep

in a 30–40° side-lying position. It is best to place the hip joint in the neutral position when the legs are in contact with the bed in order to distribute the pressure over the greater trochanter in the 30–40° laterally inclined positions. The 40° laterally inclined positioned has been shown to have lower average sacral peak pressure index (PPI) than the 30° (15 mmHg versus 20 mmHg) and should be strived for in patients with sacral pressure ulcers [63] (**Figures 16** and **17**).

Individuals should be positioned and supported to prevent sliding down in bed. If sitting in bed is necessary; avoid head-of-bed elevation or a slouched position that places pressure and shear on the sacrum and coccyx. Avoid lying postures that increase pressure, such as the 90° side-lying position, or the semi- recumbent position. Limit the head-of-bed elevation to 30° for an individual on bed rest unless it is contraindicated by medical condition or feeding and digestive considerations. Elevating the head of the bed may be medically necessary to facilitate breathing and/or prevent aspiration and ventilator associated pneumonia; in these cases the semi-Fowler's position is preferred [25, 64].

18.4. Prone

Use a pressure redistribution surface to off load pressure points on the face and body while in the prone position. At each rotation, assess other body areas (e.g., breasts, knees, toes, penis, clavicles, iliac crest, pubic symphysis) that may be at risk of bearing excessive pressure [25].

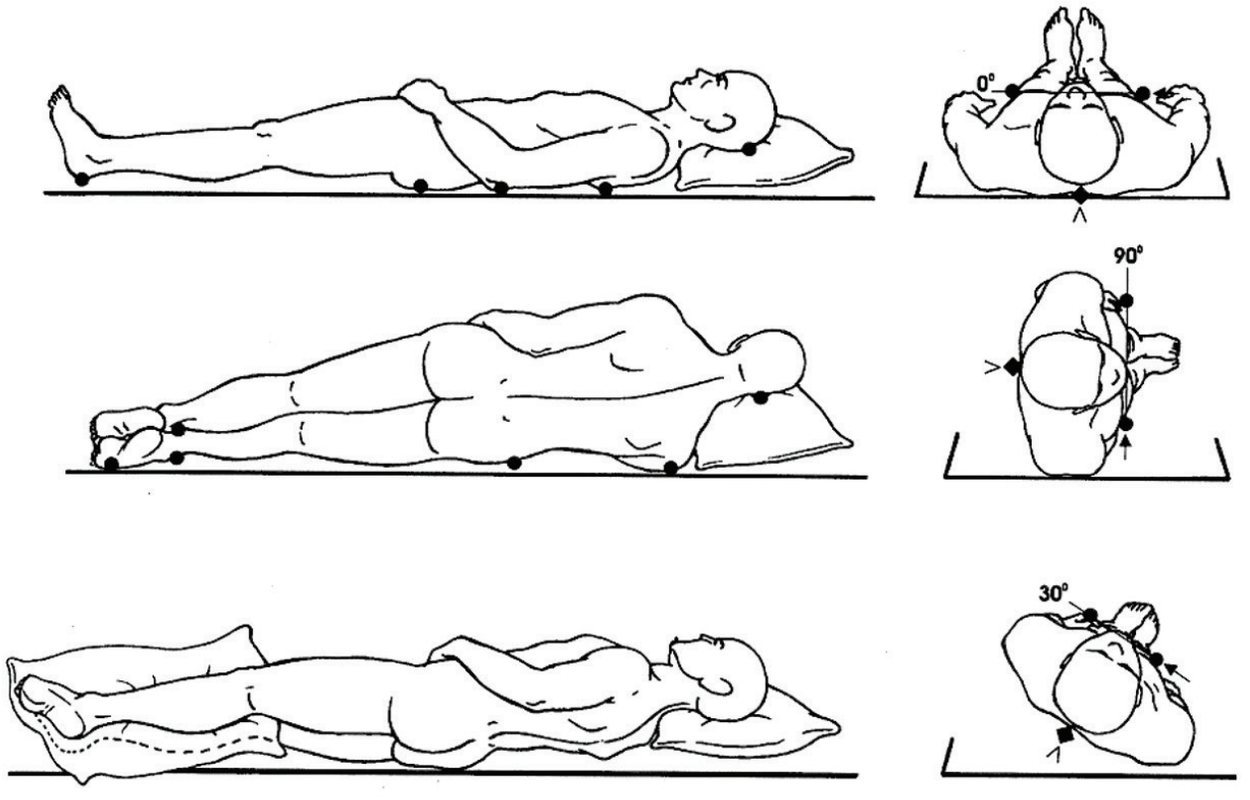


Figure 16. 30°–40° side-lying position.

http://dx.doi.org/10.5772/intechopen.69904

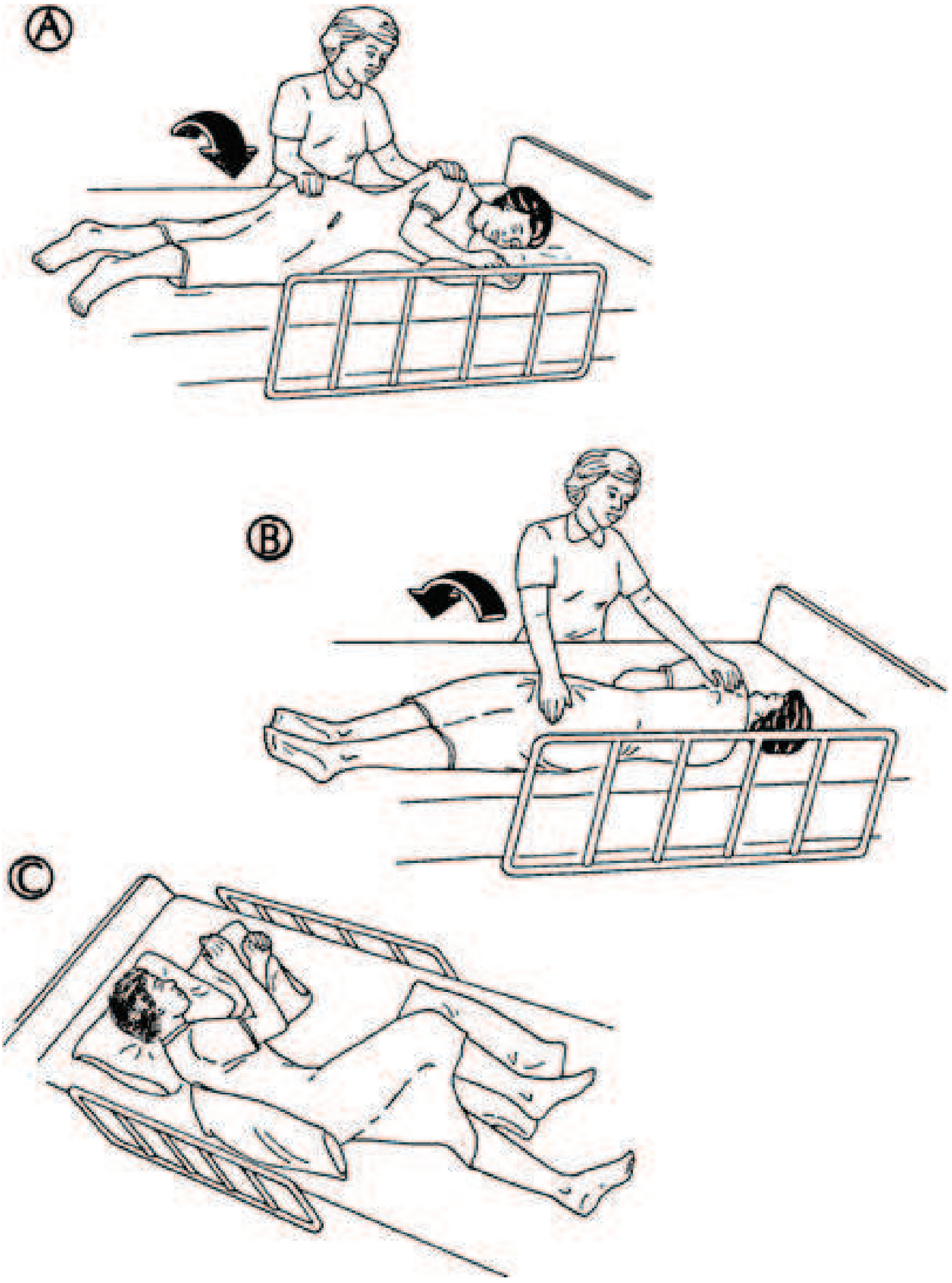


Figure 17. Turning and repositioning.

18.5. Repositioning seated individuals

Select a seated posture with an appropriate seat-to- floor height for the individual to avoid shear and friction. The seat should minimize pressure and shear exerted on the skin and soft tissues. Provide adequate seat tilt to prevent sliding forward in the wheelchair or chair, and adjust the footrests and armrests to maintain proper posture and pressure redistribution. Ensure that the feet are properly supported either directly on the floor, on a footstool, or on footrests when sitting upright in a chair or wheelchair. If the individual's feet cannot be positioned directly on the ground, footrest height should be adjusted so as to slightly tilt the pelvis forward by positioning the thighs slightly lower than horizontally. Limit the time an individual spends seated in a chair without pressure relief. One should limit sitting to ≤3 times a day and for ≤1 h each sitting period [25].

18.6. Recommendations for individuals with existing pressure ulcers

One should not be positioned directly on a pressure ulcer or on area(s) of suspected deep tissue injury with intact skin because pressure reduces perfusion to the injured tissue. Continued pressure on an existing pressure ulcer will delay healing and may cause additional deterioration. If repositioning cannot relieve pressure over the area, select an appropriate support surface and continue to turn and reposition the patient. Inspect the skin for additional damage each time the individual is turned or repositioned. Do not turn the individual onto a body surface that is damaged or still reddened from a previous episode of pressure loading, especially if the area of redness does not blanch. Ongoing assessment of the skin is necessary in order to detect additional skin damage.

19. Relieving devices/support surfaces for pressure injury prevention

Regular positioning is not possible for some individuals because of their medical condition, body habitus, etc. thus alternative prevention strategies such as providing a high-specification mattress or bed may need to be considered. Relieving devices or support surfaces play a major role in reducing compression on dependent tissue & preventing additional breakdown. Certain devices such as mattresses, integrated bed systems, mattress replacements, overlays, seat cushions, and seat cushion overlays have the ability to redistribute pressure for more effective tissue management and can serve other therapeutic functions as well [25].

19.1. General recommendations for mattress and bed support surfaces

Select a support surface that meets the individual's needs. Consider the individual's need for pressure redistribution based on the level of immobility and inactivity, the need for microclimate control, shear reduction, the size and weight of the individual, the risk for development of new pressure ulcers, and the number, severity, and location of existing pressure ulcer(s) [25].

Choose a support surface that is compatible with the care setting. Consider the weight of the bed, the structure of the building, the width of doors, the availability of uninterrupted electrical power,

and a safe location for the pump/motor, including its ventilation. Proper selection and operation of support surfaces is the key to preventing complications. Repositioning is still required for pressure relief and comfort when a support surface is used but typically not as often [25].

19.2. Mattress and bed support surfaces

Pressure redistributing support surfaces are designed to either increase the body surface area that comes in contact with the support surface in order to reduce interface pressure or to sequentially alter the parts of the body that bear load, thus reducing the duration of loading at any given anatomical site. These support surfaces are especially helpful when frequent manual repositioning is not possible such as in morbidly obese patients. Describing the specifications of various mattresses and support surfaces is beyond the scope of this chapter however in general, one should use a high-specification reactive foam mattress rather than a non-high specification reactive foam mattress for all individuals assessed as being at risk for pressure ulcer development [25].

When pressure ulcers deteriorate or fail to heal, the clinician should consider replacing the existing support surface with one that will provide a properly matched support surface environment in terms of pressure, shear, and microclimate for the individual. Changing the support surface is only one of several strategies to consider. More frequent repositioning, preventive interventions and local wound care should also be intensified as needed. For all practical purposes, evolving deep tissue injury should be provided the same level of pressure redistribution as a Stage 3 or 4 pressure ulcer. Off loading and pressure redistribution may allow reperfusion of ischemic and injured tissue, limiting the extent of infarcted or dead tissue. Once the ulcer has fully evolved, the need for a support surface can be re-evaluated [25].

19.3. General recommendations on seating support surfaces

Individualize the selection of a seating support surface for posture and pressure redistribution with consideration to body size and configuration; the effects of posture and deformity on pressure distribution; mobility and lifestyle needs. Select a stretchable/breathable cushion cover that fits loosely on the top surface of the cushion and is capable of conforming to the patient's body contours. A tight, non-stretch cover will adversely affect cushion performance. Assess the cushion and cover for heat dissipation. Select a cushion and cover that permit air exchange to minimize temperature and moisture at the buttock interface. Provide complete and accurate training on use and maintenance of seating support surfaces, including wheelchairs and cushion devices delivered to the individual. If the patient already has an existing pressure injury, select a cushion that effectively redistributes the pressure away from the pressure ulcer [25].

20. Rates of healing, prognosis & surgery

Pressure injuries are difficult to resolve. After 6 months of appropriate treatment the rates of healing are >70% for Stage 2 ulcers, 50% for Stage 3 ulcers and 30% for Stage 4 ulcers [10]. In general, healing time increases with ulcer size. Therefore, it is extremely important to identify

and start treating pressure injuries as soon as they are discovered to avoid problems and complications as time progresses. Overall median time to heal an ulcer has been cited as 46 days, 33 median days to heal small ≤ 1 cm^2 ulcers, 53 days to heal medium, >1 to ≤4 cm^2 ulcers and 73 days for large ulcers >4 cm^2 [65].

The majority of pressure ulcer patients are frail and malnourished; some are too unstable to even be brought to the operating room, thus surgical intervention is only indicated for wounds refractory to less aggressive care or for use when rapid closure is indicated. Surgery is typically saved as a last effort measure once all other non-surgical options in wound management have been exhausted. Surgical procedures can be divided into those that prepare the patient for successful healing, and those that provide definitive closure [35]. Surgical intervention is the final invasive choice because unfortunately, for many large, nonhealing pressure injuries; a reconstructive flap procedure is indicated. Most of the reconstructive flap procedures are long, procedures are long, may have to be performed in several stages and have a high rate of morbidity. The overall complication rate after surgical intervention of pressure injures has been cited as almost 60%. Wound dehiscence is the most common complication seen in >30% of patients and pressure ulcer recurrence rate is slightly <30% [66]. The mortality rate in patients with a pressure ulcer has been found to be significantly higher than in patients without a pressure ulcer (9.1% versus 1.8%, OR = 5.08, CI: 5.03–5.1, P < 0.001) [67]. The importance of early identification cannot be stressed enough. All personnel involved in patient care should seek to minimize the risks of pressure ulcer injuries and address tissue breakdown early on so that ulcer formation can be prevented all together.

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