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Pressure Ulcers

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Abstract

Pressure ulcers or pressure injuries occur in all health care settings and are considered a quality care indicator. Individuals in every health care setting must routinely be assessed for factors that place them at risk for development of pressure ulcers and have routine skin assessments to assess for the presence of pressure ulcers. If risks for pressure ulcer development or actual pressure ulcers are identified, it is crucial that a prevention and treatment plan be developed and implemented to address the risks and treat the wounds. For a prevention and treatment plan to be comprehensive and effective, it must be evidence based and multidisciplinary. The plan needs to address the risk factors or wound concerns specific to the individual and include education for the providers, caregivers and individuals at risk for pressure ulcer development and/or with pressure ulcers. Expert consensus panels concur that despite evidence-based multidisciplinary comprehensive pressure ulcer prevention plans, there are clinical situations in which pressure ulcers are deemed unavoidable.

Keywords: Pressure, Ulcer, pressure injury, decubitus, bed sore, prevention, treatment

1. Introduction

Pressure ulcers, also referred to as decubitus ulcers, pressure sores or bed sores and recently referred to as pressure injuries by the National Pressure Ulcer Advisory Panel (NPUAP) [1], are a common occurrence in all health care settings, including acute care hospitals, long-term care facilities, rehabilitation centers and subacute care centers [2]. Pressure ulcers have a significant impact on patients, families and health care facilities. These wounds can cause pain and suffering to individuals, produce emotional distress for families and significant others, increase the length of a hospital stay and increase the costs to facilities. The incidence of a pressure ulcer can also lead health care providers to feel as though they have failed to deliver

quality care to those who have been entrusted to their care [3]. It is important to identify individuals who are at risk for pressure ulcer development or those who have developed a pressure ulcer, in order to implement preventative or treatment measures; these individuals also require close monitoring.

2. Definition

The National Pressure Ulcer Advisory Panel (NPUAP), an organization comprised of leading experts in health care dedicated to the prevention and management of pressure ulcers, during a consensus conference held in the spring of 2016 replaced the term pressure ulcer with pressure injury to more accurately reflect injuries related to pressure in both intact and ulcerated skin [1]. The NPUAP also revised their definition of a pressure injury as *localized damaged to the skin and/or underlying soft tissue usually over a bony prominence or related to a medical or other device. The injury 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 may also be affected by microclimate, nutrition, perfusion, comorbidities and condition of the soft tissue* [1]. The European Pressure Ulcer Advisory Panel (EPUAP), also a leading organization of wound care experts, continues to use the term pressure ulcer as well as the definition originally developed in conjunction with the NPUAP, which states a pressure ulcer is a *localized injury to the skin and/or underlying tissue usually over a bony prominence, as a result of pressure, or pressure in combination with shear* [4].

3. Etiology

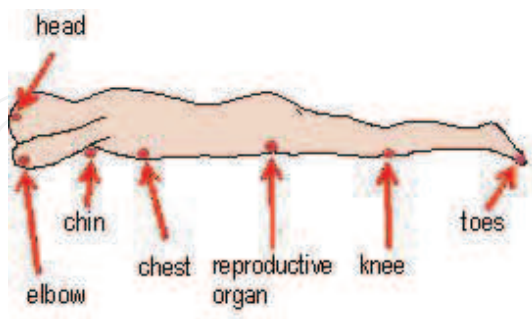
Pressure ulcers, the term that will be used throughout this chapter, occur across all health care settings with the most common setting for the occurrence of pressure ulcers being acute care hospitals followed by long-term care facilities then equally in occurrence in an individual's home and nursing facilities [5]. Pressure ulcers usually occur on the lower half of the body with two-thirds occurring in the pelvic region such as the sacrum, coccyx or hip areas and one-third occurring on the lower extremities. The occurrence of pressure ulcers on the heels is increasing. **Table 1** indicates bony prominences of the body, the location where pressure ulcers occur most often [6].

Approximately 10% of pressure ulcers are device related [7]. Multiple medical devices or pieces of medical equipment can lead to pressure ulcer development. Items such as endotracheal tubes, feeding tubes, cervical collars, tracheostomy tubes and positive pressure airway masks all have the potential for creating pressure ulcers due to pressure points created by the device. Transfer boards or slide boards place an individual at risk for shear injuries due to sliding over the firm surface.

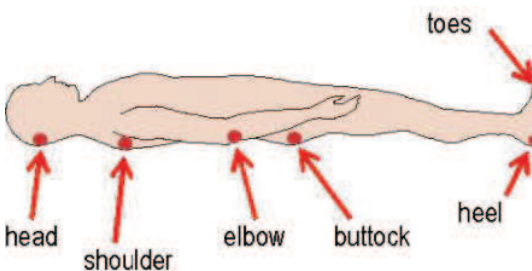
The most common age group for the incidence of pressure ulcers is the elderly, especially those 70 and older. The occurrence of a pressure ulcer in an elderly individual increases their

mortality rate fivefold. In hospital, when a patient has developed a pressure ulcer, mortality increases by 25% in the over 70 age group [5].

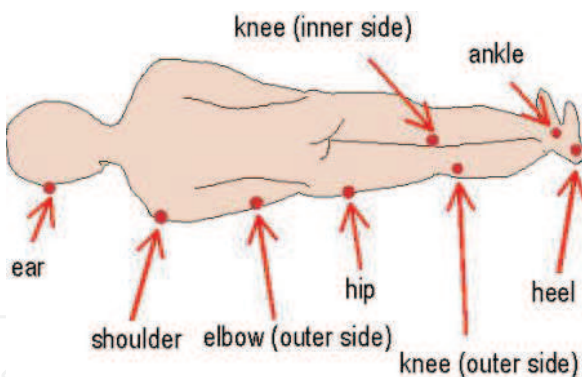
Prone position (lying on stomach)



Supine position (lying on back)



Lateral position (lying on side)



Sitting position

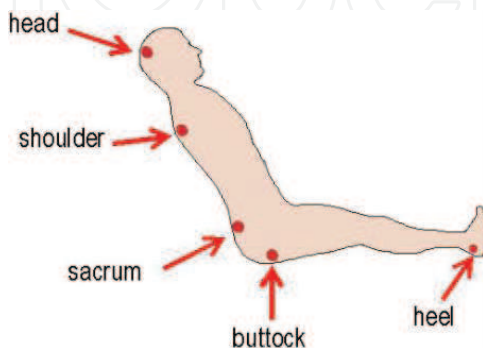


Table 1. Pressure ulcer pressure points.

4. Pathogenesis

The development of a pressure ulcer is not solely dependent upon pressure [8]. Multiple factors that modify the effect of pressure on tissues play a role in the development of pressure ulcers. The tolerance tissues have to external load depends on the duration of the exerted load. High loads can be tolerated for short periods of time, while relatively low loads can be tolerated for longer periods of time. The internal load in the tissues, as a result of the external load, causes cell deformation, occlusion of blood and lymphatic vessels and ischemia. If the internal load could be measured a risk for pressure ulcer, development could potentially be quantified [8].

A pressure ulcer results from sustained compression of soft tissues [5]. This compression occurs most often between a bony prominence and an external surface. Blood flow supplies oxygen and nutrients to the tissues. If pressure is sustained, the blood supply to the tissues is interrupted. When the blood flow is interrupted, oxygen and nutrients are not delivered to the tissues. Without oxygen and nutrients, the tissue will be damaged and eventually die [5].

Not all types of pressure are equally damaging to tissues [9]. Hydrostatic pressure, the pressure exerted by a liquid, as is endured by divers for long periods of time does not result in pressure ulcer formation. Yet localized pressure, as is exerted on the sacrum of a bedbound patient in the supine position for an extended period of time, often causes tissue distortion and blockage of the blood vessels resulting in much more damage. Studies related to localized pressure have found that pressure applied over a bony prominence resulted in more damage to the muscle than to the skin causing the study team to conclude that the muscle is more sensitive to pressure than is the skin or subcutaneous tissue [9].

Further studies identified specific factors associated with the development of a pressure ulcer including; interface pressure, shear, moisture and friction [4]. The NPUAP, after investigating shear and friction which have long been associated with pressure ulcer development, has eliminated friction from its definition of a pressure ulcer the explanation of which will be discussed below [10].

Interface pressure contributes to pressure ulcer development, as it is the pressure that develops between the skin and a surface upon which an individual is sitting or lying. Interface pressure is a measure commonly used to evaluate the effectiveness of a support surface [11]. Pressure mapping measures interface pressures and helps to determine appropriate positioning [8].

Not all localized pressure results in a pressure ulcer. When the pressure of short duration is relieved, blood flow returns to the area. This occurrence is known as reactive hyperemia, blood vessels in the area of pressure dilate in an attempt to overcome the ischemia that occurs with the pressure. Reactive hyperemia is transient and is also described as blanchable erythema—an area that becomes white when pressed with a finger and returns to erythema when the compression is removed [11].

Pressure that is not relieved and is of longer duration leads to further decreased capillary blood flow, occlusion of lymphatic vessels and tissue ischemia. Over a bony prominence, pressure of 20 mmHg can increase to as much as 300 mmHg. If this pressure is sustained, destruction

of deep tissues can occur including destruction of muscle, subcutaneous tissue, dermis and epidermis [11].

When capillaries are occluded metabolic waste begins to accumulate in the surrounding tissues due to the lack of oxygen and nutrients. Capillaries that are damaged become more permeable and leak fluid into the interstitial space-causing edema. Perfusion is slowed through the edematous tissue; therefore, hypoxia worsens. Hypoxia increases cell death that results in an increased metabolic waste released into the surrounding tissues [11]. The ensuing edema further compresses small vessels causing increased edema and ischemia. Local tissue death occurs, which results in a pressure ulcer [7].

Shear is an applied force that causes an opposite yet parallel sliding motion such as when an individual slides in a bed or chair. The individual's skeletal structure slides in one direction yet the skin layer is restrained in the original position secondary to friction forces. In these situations, when shear is involved, multiple studies have found the pressure needed to occlude the blood vessels is much less than in an area where shear force is not involved [5, 8]. Elderly individuals are at higher risk for the effects of shear due to the decreased amount of elastin in their skin which is a normal consequence of aging [5].

Moisture, another factor associated with the development of pressure ulcers, alters the resiliency of the epidermis to external forces [11]. The effects of friction and shear are increased in the presence of moisture. Increased moisture is often associated with incontinence, perspiration or wound exudate [5].

Friction was originally determined to be a causative factor in the development of pressure ulcers after a study by Sidney Dinsdale was published in 1974 [10]. The results of this study showed that significantly less pressure was needed to stimulate the development of a full or partial thickness wound when the pressure was applied in conjunction with friction.

There are several forms of friction as they relate to the development of pressure ulcers. Friction, as a general term, is the rubbing of two body parts together. It is also a force that resists the motion of two bodies and/or material elements sliding against each other. In relation to skin breakdown, the type of friction, that is of concern is dry friction, of which there are two types, namely static and kinetic. Static friction is the force that resists the motion between two bodies when there is no sliding. There are multiple aspects that impact the amount of static friction at the skin surface including an individual's hydration level and what the individual is in contact with, for example bed linen. Moisture is an important factor relative to static friction as humidity and liquid moisture increases the friction and may cause an individual to adhere to a surface. Dynamic friction, also known as kinetic friction, is the force between two bodies relative to one another as they are sliding. Dynamic friction occurs when an individual slides downward in bed or rubs a foot in a shoe causing a blister. Such a blister may be misdiagnosed as a pressure ulcer.

In relation to the Dinsdale study, the type of friction applied during the study was not noted. The results of this study showed that the blood flow to the epidermis in a given area was not significantly different when pressure and friction were applied together and when pressure was applied alone. Investigators concluded that increased susceptibility of lesions with friction

was not due to ischemia in the epidermis. Three decades later, it has been hypothesized that the friction used in Dinsdale's study was creating shear strain or deformation in deeper layers of tissue. Current hypothesis is that friction causes mechanically damaging shear strain of superficial tissue cells and tissue damage results directly from excessive deformation not ischemia as previously thought.

Friction is an important factor as it leads to shear stress and strain yet does not alone lead to the development of a pressure ulcer. Friction contributes to the development of a pressure ulcer due to the shear forces it can create. In other words, friction causes the shear forces in the tissue, which can increase the risk of tissue breakdown and lead to the development of a pressure ulcer. Therefore, shear remains in the current NPUAP definition of a pressure ulcer yet friction is eliminated. Including friction would be redundant as friction is now thought to be a cause of shear. Also, eliminating friction may decrease the number of wounds that are misdiagnosed as pressure ulcers when they are caused solely by friction [10].

5. Pressure ulcer stages

Pressure ulcers are classified by the amount of visible tissue loss [4]. Depth of tissue loss is important, as it determines a treatment plan of care and can impact payment. Once a wound is determined to be a pressure ulcer, it is assigned a pressure ulcer-specific stage or category. No other wound utilizes this same staging/categorizing system. A stage or category is assigned after careful and thorough assessment of the pressure ulcer to determine the extent of tissue destruction. To complete this assessment, one must have a competent understanding of the anatomy of the tissue layers involved and of the physiology of pressure ulcer development.

The NPUAP has defined the stages or categories of pressure ulcers as follows (Table 2):

EPUAP staging guideline	
Stage I	Nonblanchable erythema—Intact skin with nonblanchable redness of a localized area usually over a bony prominence. Darkly pigmented skin may not have visible blanching; its color may differ from the surrounding area. The area may be painful, firm, soft, warmer or cooler as compared to adjacent tissue. Category I may be difficult to detect in individuals with dark skin tones. May indicate “at-risk” persons.
Stage II	Partial thickness skin loss—partial thickness loss of dermis presenting as a shallow open ulcer with a red pink wound bed, without slough. May also present as an intact or open/ruptured serum-filled or sero-sanguinous-filled blister. Presents as a shiny or dry shallow ulcer without slough or bruising. This category should not be used to describe skin tears, tape burns, incontinence-associated dermatitis, maceration or excoriation
Stage III	Full-thickness skin loss—Full-thickness tissue loss. Subcutaneous fat may be visible but bone, tendon or muscles are not exposed. Slough may be present but does not obscure the depth of tissue loss. May

EPUAP staging
guideline

	include undermining and tunneling. The depth of a category/stage III pressure ulcer varies by anatomical location. The bridge of the nose, ear, occiput and malleolus do not have (adipose) subcutaneous tissue and category/stage III ulcer can be shallow. In contrast, areas of significant adiposity can develop extremely deep category/stage III pressure ulcers. Bone/tendon is not visible or directly palpable.
Stage IV	Full-thickness tissue loss—Full-thickness tissue loss with exposed bone, tendon or muscle. Slough or eschar may be present. Often includes undermining and tunneling. The depth of a category/stage IV pressure ulcer varies by anatomical location. The bridge of the nose, ear, occiput and malleolus do not have (adipose) subcutaneous tissue and these ulcers can be shallow. Category/stage IV ulcers can extend into muscle and/or supporting structures (e.g., fascia, tendon or joint capsule) making osteomyelitis or osteitis likely to occur. Exposed bone/muscle is visible or directly palpable.
Unstageable	Full-thickness skin or tissue loss—depth unknown—Full-thickness tissue loss in which actual depth of the ulcer is completely obscured by slough (yellow, tan, gray, green or brown) and/or eschar (tan, brown or black) in the wound bed. Until enough slough and/or eschar are removed to expose the base of the wound, the true depth cannot be determined, but it will be either a category/stage III or IV. Stable (dry, adherent, intact without erythema or fluctuance) eschar on the heels service as “the body's natural (biological) cover” and should not be removed
Suspected deep tissue injury (sDTI)	SDTI depth unknown—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. The area may be preceded by tissue that is painful, firm, mushy, boggy, warmer or cooler as compared to adjacent tissue. Deep tissue injury may be difficult to detect in individuals with dark skin tones. Evolution may include a thin blister over a dark wound bed. The may further evolve and become covered with thin eschar. Evolution may be rapid exposing additional layers of tissue even with optimal treatment.

Table 2. Pressure ulcer staging [4].

An illustration of the pressure ulcer stages/categories is seen in **Table 3** [1].

As pressure ulcers heal, the lost muscle, subcutaneous fat or dermis are not replaced with like tissue before they re-epithelialize [12]. A pressure ulcer fills in with scar tissue, which is composed primarily of endothelial cells, fibroblasts, collagen and extracellular matrix. Therefore, a stage-III pressure ulcer, for example, cannot, as the wound heals, become a stage-II pressure ulcer and progress on to a stage-I pressure ulcer because the term stage I would not accurately reflect the structures that are now present under the newly re-epithelialized tissue. Referring to a healing stage-III pressure ulcer as a stage II, then a stage-I pressure ulcer is known as reverse staging or down staging and is not acceptable. The stage needs to reflect the scar tissue that has developed. Therefore, the stage for this healing pressure ulcer is “healing stage-III pressure ulcer” and when the pressure ulcer has healed, the stage is a “healed stage-III pressure ulcer,” indicating the pressure ulcer is now filled with granulation or scar tissue and resurfaced with epithelium [12].

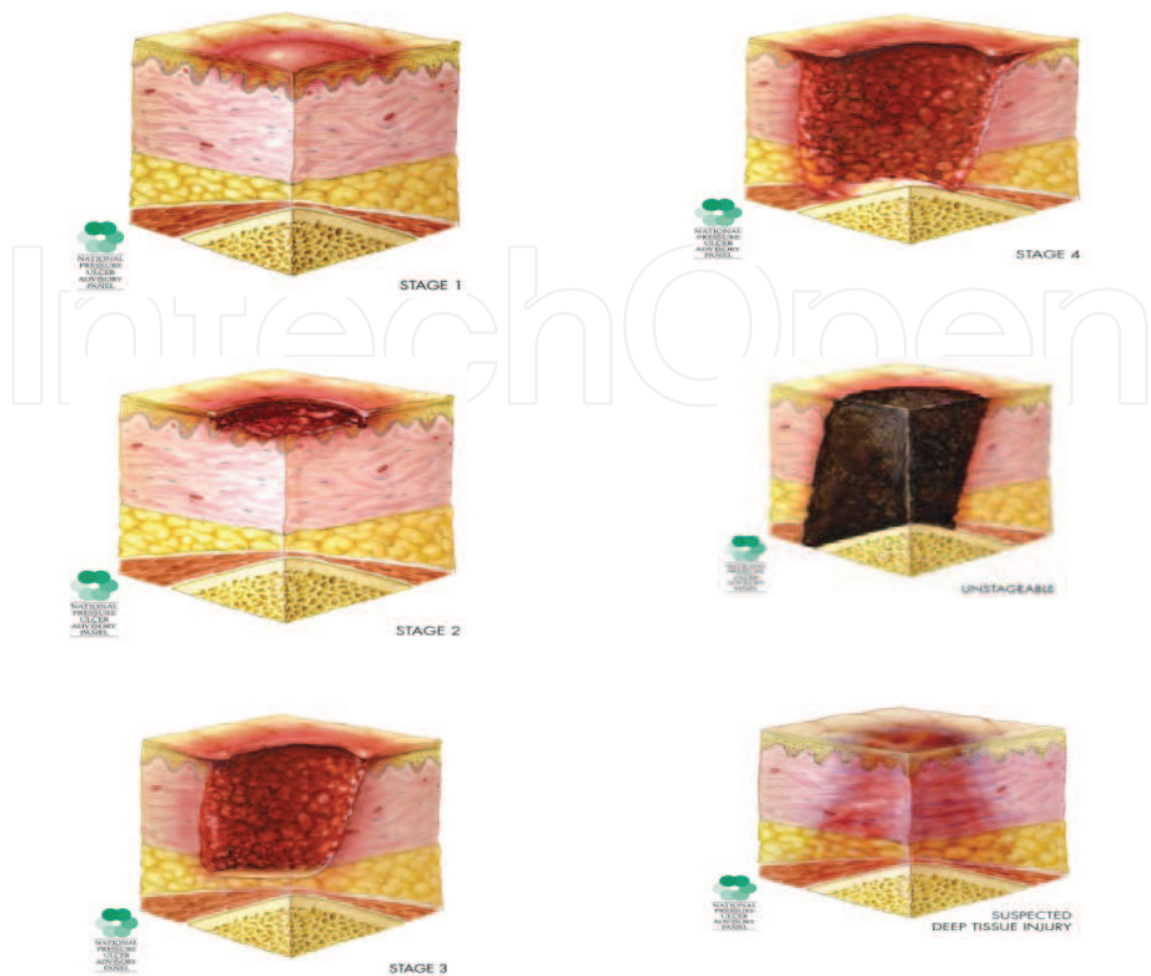


Table 3. Pressure ulcer injury/ulcer stages/categories.

Mucosal pressure injuries are *pressure injuries found on mucous membranes with a history of a medical device in use at the location of the ulcer* [1]. A mucous membrane is the moist lining of a body cavity, such as the gastrointestinal tract, nasal passages, urinary tract and vaginal canal, that communicates with the exterior. When pressure is applied to a mucous membrane, ischemia can result that can lead to a pressure ulcer. Mucous membranes are vulnerable to pressure especially related to medical devices such as oxygen tubing, feeding tubes, urinary catheters and fecal containment devices [13].

The anatomy of mucous membranes impacts the staging or categorizing of a mucous membrane pressure injury [13]. There are two types of mucous membrane tissue; nonkeratinized stratified squamous epithelium and an underlying connective tissue layer, the lamina propria. These layers are similar to the epidermis and dermis and are connected via rete pegs. At the interface of the two layers is a basal laminal layer. The epithelial layer is continuously renewed through migration of lower layers of epithelium to the surface. The epithelium of the mucosa, although is not keratinized like the epithelium of the skin. The lamina propria generally contains blood vessels, elastin and collagen fibers [13].

Injured mucosa heals similarly as skin with the exception of scar formation [13]. There is an increasing evidence that the fibroblasts in mucosa resembles fetal fibroblasts. Most mucosal injuries heal without scar formation [13].

The staging or categorizing of pressure ulcers that is used for the skin cannot be used to stage mucosal pressure injuries [13]. Nonblanchable erythema cannot be seen in mucous membranes, as superficial tissue losses of the nonkeratinized epithelium are so shallow they cannot be differentiated from deeper, full thickness injuries. The coagulum seen on a mucous membrane pressure injury resembles slough yet it is actually soft blood clot. Muscle is seldom seen in a mucous membrane pressure injury and bone is not present in these tissues. Therefore, pressure injuries located on a mucous membrane are referred to as mucous membrane pressure injuries [13].

6. Pressure ulcer prevention

There are several factors that have been associated with the development of pressure ulcers. Many of these factors affect an individual's ability to withstand episodes of pressure and shear as well as decrease the length of time or amount of pressure necessary to cause tissue damage. Risk factors that can lead to pressure ulcer development include age, immobility, nutritional deficiencies, skin moisture and incontinence, vasopressor use, chronic diseases such as diabetes or stroke, smoking, behavioral issues leading to noncompliance, poor general health and sensory loss [14, 15]. No single factor can explain all pressure ulcers rather it is a complex interaction among factors which increases the probability of pressure ulcer development

Braden scale	Norton scale	Waterlow scale	Jackson Cubbin scale
Sensory perception	Physical condition	Sex	Age
Moisture	Mental status	Age	Weight
Activity	Activity	Appetite	Skin condition
Mobility	Mobility	Nurses' visual assessment of skin	Mental status
Nutrition status	Continence	condition	Mobility
Friction/shear		Mobility	Nutrition
		Continence	Respiration
		Factors contributing to tissue Malnutrition	Continence
		Neurologic deficits	Hygiene
		Major surgery or trauma	Hemodynamic status
		Medication	

Table 4. Pressure ulcer risk assessment tools.

Prevention begins with identifying those individuals at risk for pressure ulcer development. A pressure ulcer risk assessment instrument that has been validated for use in the specific age group should be utilized. In the Unites States, the most common adult risk assessment

instruments are The Braden and Norton scales that have been tested for validity in predicting pressure ulcer development risk [7, 16]. In Britain, the most common scales are the Braden and the Waterlow. The Jackson Cubbin Scale is specific to European critical care (**Table 4**).

These scales will identify specific factors related to assessment categories that place an individual at risk for pressure ulcer development. Once specific factors are identified, a prevention plan to address those factors can be implemented to reduce or eliminate the risk of pressure ulcer development [3, 16]. With the implementation of an evidence-based pressure ulcer prevention plan, pressure reduction can occur which will preserve the microcirculation and prevent the development of pressure ulcers [17]. A pressure ulcer prevention plan is multifaceted. Factors related to prevention and discussed further in treatment, as these factors are also included in a treatment plan, include; mobility, moisture and continence care, nutrition and hydration, support surfaces, documentation and education. No single intervention has been found that will consistently, reliably and completely reduce pressure ulcer development. Pressure ulcer prevention involves multiple interventions and a multidisciplinary team to affect the identified factors and reduce the risk of pressure ulcer development.

7. Pressure ulcer treatment

The treatment of pressure ulcers is based on the physiology of wound healing. Wound healing is a complex process that changes with the health status of the individual [17]. A health care provider needs to have a basic knowledge of the phases of wound healing including; hemostasis, inflammation, proliferation and maturation. Once a provider understands wound healing, one has a significant piece of the knowledge necessary to develop a pressure ulcer treatment plan [18].

7.1. Phases of wound healing

The first phase of wound healing is hemostasis. Briefly, in this phase, damaged blood vessels are sealed when platelets form a stable clot to seal the blood vessel. The platelets also stimulate the clotting cascade through the production of thrombin that initiates the production of fibrin. The fibrin mesh ultimately strengthens the platelet aggregate into a hemostatic plug. Hemostasis occurs within minutes of injury unless the injured individual has underlying clotting disorders [18].

In the second phase of wound healing, the inflammation phase, the erythema, swelling and warmth that occur are often associated with pain. This phase of wound healing usually lasts up to 4 days after injury. During this phase, neutrophils or PMN's (polymorphonucleocytes) and plasma are leaked from the blood vessels into the surrounding tissue. These factors clean debris from the surrounding tissue and provide the first line of defence against infection. Macrophages are also active in the second phase of wound healing acting to destroy bacteria and secreting growth factors which direct the third phase of wound healing.

Chronic wounds, wounds that take longer than 12 weeks to heal, often remain in the inflammatory stage longer than occurs with acute wounds. Cellular and molecular abnormalities

within a wound bed prevent progression through the stages of healing [19]. Chronic wounds contain elevated inflammatory cytokines and proteases. Chronic wounds do not respond to growth factors in the same manner in which acute wounds do. Specifically related to pressure ulcers, the volume of exudate is often times increased in chronic wounds. Secondary to infection, the exudate may be more purulent. If protein levels are low, the exudate may appear thinner [19]. Chronic wounds often have inadequate blood supply that also contributes to delayed healing and the formation of unhealthy granulation tissue.

The third phase of wound healing, the proliferation phase, begins approximately 4 days after injury and usually lasts until day 21 postinjury. The activity during this phase is replacement of dermal tissue and possibly subdermal tissue and contraction of the wound. Fibroblasts secrete collagen, which is the framework upon which new dermal regeneration, can occur. Angiogenesis, development of new capillaries, also occurs during this phase of wound healing. Keratinocytes differentiate to form the protective outer layer.

In the final phase of wound healing, maturation, remodeling of the dermal layer occurs to produce greater tensile strength. The cells that are involved in this process are fibroblasts. This process can take up to 2 years to complete [18].

7.2. Principles of wound healing

In addition to knowledge of wound healing, a provider must also be aware of the principles of wound treatment and intervention. For a wound to progress to healing, the wound bed must be well vascularized, free of devitalized tissue, free of infection and moist. Continual evaluation of a wound is necessary as the wound progresses through the stages of wound healing.

7.3. Dressings

No one dressing is appropriate for all wounds. There are multiple factors that will affect the dressing selected for a particular wound **Table 5**.

Knowledge of the properties of available wound dressings and an understanding that a treatment plan may need to change as the wound progresses through the stages of healing is vital. Wounds that do not advance through the process of healing in a reasonable or expected time frame must be assessed for issues that have not been previously identified or wound changes that have occurred and the treatment plan re-evaluated **Table 6**.

The principles in selecting dressings for pressure ulcer treatment include eliminate dead space, control exudate, prevent bacterial overgrowth, ensure proper moisture balance, cost-efficiency, and manageability for the individual, caregiver and providers.

Several adjuvant therapies/advanced dressings have been used to treat pressure ulcers. These therapies include (a) platelet-derived growth factor (PDGF) applied to the wound bed, which will stimulate the growth of cells involved in wound healing and granulation tissue formation; (b) negative pressure wound therapy (NPWT), which utilizes subatmospheric pressure applied to a wound via a sealed dressing to promote wound healing, the applied suction removes drainage and increases blood flow to the wound; and (c) hyperbaric oxygen therapy,

delivered in multiple modes—total body, body part or mask—exposes the body to 100% oxygen at a higher pressure than normally experienced, this therapy provides oxygen necessary to stimulate wound healing and combats infection by enhancing leukocyte and macrophage activity [20]. PDGR and hyperbaric oxygen, although supported for use, have less support than does NPWT in studies conducted on individuals with pressure ulcers [20].

Wound depth	Partial thickness Full thickness
Wound description	Necrotic Slough Granulating Epithelializing
Wound characteristics	Dry Moist Heavily exudating Malodorous Excessively painful Difficult to dress
Bacterial description	Colonized Infected

Table 5. Wound description.

Alginate	Highly absorbent, useful for wounds with copious exudate. Alginate rope is particularly useful to pack exudate cavities or tracts
Hydrofiber	Absorbent dressing used for exudative wounds
Debriding agents	Useful for necrotic wounds, often used as an adjunct to surgical debridement
Foam	Useful for clean granulating wounds with minimal exudate
Hydrocolloid	Useful for dry necrotic wounds, wounds with minimal exudate or clean granulating wounds
Hydrogel	Useful for dry, sloughy, necrotic wounds
Transparent film	Useful for clean, dry wounds with minimal exudate, protect high friction areas
Negative pressure wound therapy	Conforms to the wound bed by suction and stimulates wound contraction while removing exudate

Table 6. Dressings [17].

There is limited evidence, although moderate strength, indicating support for the use of radiant heat dressings to improve pressure ulcer healing. Radiant heat dressings are noncontact dressings attached to a heating element. These dressings provide warmth to the wound and have been found to increase capillary blood flow to the area and thus increase wound healing. Also with limited yet moderate strength of evidence is the use of electrical stimulation, which provides a direct electrical current through the wound bed using electrodes on the surface of the wound. One hour daily session has been shown to be most effective. Caution has been noted not to use electrical stimulation on individuals with cancer as the treatment could stimulate the cancerous cells. The American College of Physicians specifically notes electrical stimulation in its guidelines [15].

7.4. Treatment plan

Prior to the development of a comprehensive pressure ulcer treatment plan, consideration should be given to an individual's psychological, behavioral and cognitive status. The individual's goals and prognosis need to be determined as well as the resources an individual has available, both financially and as caregivers.

A multidisciplinary team is needed to develop a comprehensive pressure ulcer prevention and treatment plan, as numerous factors are addressed. The team may include the individual's primary care provider, a wound care specialist, nurses or medical assistants who will provide wound care or education, social workers who will assist the individual and family members with resources and emotional concerns, a physical therapist who will provide assistance with mobility therapy and any other necessary consultants.

Within the plan, the following needs may need to be addressed.

Debridement of necrotic tissue within an acute wound may be necessary to be able to completely assess the wound. Necrotic tissue may obscure underlying fluid collections that need to be identified. Necrotic tissue also promotes bacterial growth that impairs wound healing and therefore should be debrided [17]. However, debridement is not recommended for stable dry eschar on heel wounds with no edema, erythema or fluctuance. Debridement can be achieved by multiple methods including sharp debridement, mechanical, enzymatic or autolytic debridement. Most sharp debridement can be completed at bedside, yet if more extensive sharp debridement is needed it may need to be performed in an operating room [17].

Mobilization of an individual is an important component to a pressure ulcer treatment plan. Since, by definition, a pressure ulcer is caused, in part, by pressure, if an individual begins to mobilize pressure will be relieved individuals who cannot ambulate redistributing pressure on a support surface needs to be investigated.

Moisture management, controlling incontinence and excess perspiration by wicking moisture away from the skin, will impact the effect of moisture. Managing moisture will increase the ability of the epidermis to return to its original state after being exposed to pressure. Shear and friction also will not be as detrimental to the skin when moisture is not allowed to be in contact with the skin for prolonged periods of time.

Nutrition studies indicated weak evidence that nutritional interventions provide benefits in the prevention or treatment of pressure ulcers [15]. A guideline presented by the American College of Physicians in 2015 cited moderate quality evidence supporting protein supplements in treating pressure ulcers. A Cochrane review in 2014 concluded that there is no evidence to support nutritional interventions, including protein, provide any benefits in preventing or treating pressure ulcers. A study regarding vitamin C supplements concluded that there was no change in wound healing. No results were noted related to zinc due to insufficient evidence. Although, evidence supports that providing adequate nutrition is important. Oral nutrition is preferred, yet if not possible; provide nutrition by the most appropriate route.

Oxygenation and perfusion must be ensured. A primary reason for inadequate tissue oxygenation is vasoconstriction as a result of sympathetic over activity. Blood volume deficit, pain

and hypothermia are common causes of sympathetic overactivity for which the end result could be increased risk for pressure ulcer development.

Infection is usually determined clinically [15]. All open wounds contain some degree of bacteria. Healing is most often not impaired until bacteria reach a high colony count. If a wound culture needed, evidence indicates the Levine technique should be used. This technique involves rotating a swab over a 1 cm² patch of wound with enough pressure to express fluid from the wound for 5 s. A tissue or bone biopsy is the preferred method of identification of osteomyelitis, although biopsies of this nature are not always feasible. Magnetic resonance imaging (MRI) and nuclear medicine tests are more sensitive and specific than conventional plain radiography in identifying osteomyelitis. When bone is exposed in a pressure ulcer, osteomyelitis is often presumed.

An individual with increasing pain may be exhibiting a sign of a wound infection. Other signs of an acute wound infection include erythema around the ulcer's edges, induration, warmth and purulent drainage, no progression toward healing for 2 weeks, friable granulation tissue, foul odor, new necrotic tissue or lack of even spread of granulation tissue across the base of the wound. An individual may also exhibit systemic symptoms of a wound infection including fever, delirium and confusion [15].

Repositioning is replacing the term turning. The aim of repositioning individuals at risk for pressure ulcer development is to relieve pressure and/or redistribute pressure. It has been found that a slight change in position can be adequate to aid in relieving pressure. A turn of 30°, as previously encouraged for an individual in bed, for pressure relief, is not always needed to relieve pressure from bony prominences.

There is no research to support repositioning individuals every 2 h will aid in preventing the development of pressure ulcers; this recommendation is based on expert opinion [15]. The frequency of repositioning will, in part, be determined by an individual's tissue tolerance or the ability of both the individual's skin and its underlying structures to withstand pressure without an adverse effect.

As a provider or caregiver, when an individual at risk for pressure ulcer development is in bed, avoid positions with the head of the bed elevated to the point in which excess pressure and shear are applied to the sacrum and coccyx. This is most often any point beyond 30°.

In the seated position, the greatest exposure to pressure is to the ischial tuberosities. The area of the ischial tuberosities is relatively small; therefore, the pressure will be high. Without pressure relief, a pressure ulcer will develop quickly.

If a patient has a reddened area as a result of a previous episode of pressure loading, it is not advisable to position the individual on the same body surface. The reddened area indicates the body has not recovered from the previous position on the body surface and continues to require relief from the pressure load.

If heels are left in contact with a surface for a prolonged period of time, it is not unusual for heel pressure ulcers to develop due to the significant volume of bony structure in relation to the soft tissue in the heel. For the protection of the heels or treatment of heel, pressure ulcers

assure that heels are elevated off any surface. Heels should be elevated so as to distribute the weight of the leg along the calf without putting pressure on the Achilles tendon. To avoid obstruction of the popliteal vein, which begins behind the knee, which may lead to a deep vein thrombosis, care must be taken to not hyperextend the knee.

Physical conditions of certain populations require additional care in positioning. These populations include those with spinal cord injuries, those that are insensate, older adults, individuals that have sustained hip fractures or those that do not maintain a healthy lifestyle.

Support surface use has been validated in studies for the prevention of pressure ulcers in high-risk individuals and for the treatment of individuals with pressure ulcers. A support surface reduces pressure by spreading the tissue load over a larger area, thus decreasing the load over bony prominences. A support surface also manages the microclimate including moisture and temperature.

Support surface selection is based on mobility, comfort and circumstances of care. In a home setting, consideration is given to the structure of the home including width of doors, power supply and available ventilation for heat from the motor as these factors relate to the support surface to be utilized. If a spouse or significant, other will share the bed consideration should be given to his or her comfort also.

Regular foam does not distribute patient weight uniformly and may worsen or cause pressure ulcers. A higher specification foam mattress is more effective in preventing pressure ulcers

When an individual is placed on a low-air loss, surface consideration must be given to the linens and pads used on the surface. Linens and pads should not be of materials that will block the air flow.

An issue that can negatively impact an individual at risk for pressure ulcer development or with a pressure ulcer that is related to any support surface is bottoming out. Bottoming out occurs when an individual's pelvic region or buttocks sink down and the support surface no longer provides adequate redistribution of pressure. An assessment for bottoming out can be performed with a hand check. Place a hand, palm side up under the support surface directly below the individual's buttocks region. If the individual can feel your hand or if less than an inch of support material is evident, the individual has bottomed out and the surface should be replaced [16].

Physician consults are generally part of a comprehensive treatment plan for individuals with pressure ulcers. Specialists may be consulted to debride wounds or with more complex wounds to perform flap procedures. Infectious disease physicians may be consulted to provide input or to monitor infected wounds especially if osteomyelitis is suspected or confirmed.

Education of providers, caregivers and individuals with pressure ulcers is a vital component to any prevention or treatment plan. Without adequate education, failure of a plan is probable. Education of providers should include how to complete a comprehensive skin and wound assessment as well as documentation of the assessments. Providers also require education on the facilities process for wound care treatment, including the principles of wound care and the products available on the wound care formulary.

Caregivers or the individual with a pressure ulcer need to be educated on the cause of the pressure ulcer, the contributing factors, prevention measures, proper nutrition, appropriate wound treatment and appropriate times to contact a provider. Education should include materials in a format understandable for the caregivers and individuals. For individuals with chronic conditions, such as spinal cord injuries, there are often times formal education programs in rehabilitation centers. It is also common to request a caregiver to receive education in the hospital prior to a patient being discharged. Education is crucial to an effective prevention and treatment plan.

8. Unavoidable pressure ulcers

In 2014, the NPUAP hosted a multidisciplinary international conference to explore the issue of unavoidable pressure ulcers. This conference brought experts together to explore, within the context of organ systems, the issue of unavoidable pressure ulcers [14]. At a previous conference in 2010, also hosted by the NPUAP, an unavoidable PU was defined *as one that may occur even though providers have evaluated the individual's clinical condition and PU risk factors have been evaluated and defined and interventions have been implemented that are consistent with individual needs, goals and recognized standards of practice*.

It was agreed upon by those in attendance at the 2014 conference that unavoidable pressure ulcers do occur. This conference also established consensus on risk factors that have in some situations been shown to increase the likelihood of the development of unavoidable pressure ulcers. In summary, the organ systems which were identified that may in some situations contribute to the development of unavoidable pressure ulcers included; (a) impaired tissue oxygenation/cardiopulmonary dysfunction—an individual cannot be repositioned due to the potential for a fatal event related to hemodynamic status, (b) hypovolemia—an individual is hemodynamically unstable which often leads to an inability to reposition an individual, (c) body edema/anasarca—leads to decrease pressure-loading tolerance and increased risk of pressure ulcer development, (d) peripheral vascular disease, lower extremity arterial and venous disease—compromised circulation that contributes to ischemia which leaves tissues more vulnerable to pressure ulcer development. Within this category, other subcategories were identified including chronic kidney disease, whereas the change in tissue tolerance may increase the likelihood of pressure ulcer development, hepatic injury which results in hypoalbuminemia that leads to edema and anasarca, sensory impairment, skin issues related to extremes in age, multiorgan dysfunction syndrome, critical status and burns all which leave patients prone to pressure ulcer development, (e) body habitus—obesity compromises an individual's ability to prevent shear injury during movement, pressure ulcer development related to moisture due to increased diaphoresis and inability to redistribute pressure over bony prominences and (f) immobility—associated with vascular congestion, dependent edema, compromised lung aeration, decreased red blood cell mass, dyspnea and activity tolerance leading to increased risk for unavoidable pressure ulcer development. The consensus panel also agreed that further research is necessary to examine the issue of unavoidable pressure ulcers [21].

9. Summary

A pressure ulcer rate is considered a quality care indicator in most health care settings and being an international health care concern. Most pressure ulcers are preventable. With a thorough assessment, including an assessment of an individual's skin and an assessment of pressure ulcer development risk, a comprehensive prevention and treatment plan can be developed and implemented to enhance positive outcomes.

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