Committed to Wound Care Education for the Nurse

Smith & Nephew is committed to advancing the field of chronic wound care through science and education.

Launched in 2005, The Wound Institute® is the largest online wound care education site serving nearly 50,000 members. The Wound Institute® is dedicated to providing online wound care education programs for all healthcare professionals and their patients. Here you’ll find comprehensive education and training programs specifically designed for your educational needs, whether they are patient care, accredited education, staff training, resource tools or simply learning to care for someone with a difficult-to-heal wound.

This resource will provide you with an overview of wound care and an introduction to the courses on The Wound Institute®. To begin taking a course today, please visit www.thewoundinstitute.com.

Note: All accredited educational activities available through The Wound Institute® are offered by independent continuing education providers. Those programs may also be accessed directly through the continuing education provider’s website.
# Contents

Evolution of Wound Care ............................................... 3
The Skin ................................................................. 4
The Layers of the Skin and Their Functions .......... 4
The Skin and Aging ................................................. 5
Chronic Wound Care and Aging .......................... 6
The Healing Process of an Acute Wound .............. 7
Acute Healing Process ......................................... 9
The Biology and Healing Process of a Chronic Wound .......................... 10
Types of Chronic Wounds ...................................... 11
Pressure Ulcers ..................................................... 11
Venous Ulcers ....................................................... 13
Arterial Ulcers ....................................................... 14
Diabetic Ulcers ...................................................... 15
Atypical Wounds ................................................... 17
Associated Conditions .......................................... 17
Lymphedema/Phlebolymphedema ....................... 17
Overall Wound Management .............................. 18
Patient Assessment .............................................. 19
Properly Assessing a Chronic Wound ................. 20
Wound Classification Systems ......................... 21
Partial and Full-Thickness Wounds .................. 21
Managing Chronic Wounds ................................. 22
Wound Bed Preparation ........................................ 22
Components of Wound Bed Preparation ............ 24
Debridement ......................................................... 24
Infection ............................................................ 24
Moisture Imbalance ........................................... 25
Edge of a Wound ................................................ 25
Treatment Algorithm .......................................... 26
Debridement Options .......................................... 27
Autolytic ............................................................ 27
Enzymatic .......................................................... 27
Mechanical ........................................................ 27
Surgical or Sharp Debridement ....................... 28
Biotherapy .......................................................... 28
Wound Infections ............................................... 29
Understanding How to Identify a Critically Colonized versus an Infected Wound ................. 30
Biofilms ............................................................. 31
Infection Control ................................................. 31
Interventions ....................................................... 32
Antimicrobial and Antibacterial Dressings ....... 32
Cadexomer Iodine ................................................. 32
Antibiotics .......................................................... 32
Methylene Blue/Gentian Violet ......................... 32
Polyhexamethylene Biguanide (PHMB) ............... 33
Moisture Balance Options ................................. 34
Negative-Pressure Wound Therapy ................. 35
Advanced Technology Therapies .................... 35
Topical Collagen Matrices ................................. 35
Natural Extracellular Matrices ......................... 35
Living Skin Substitutes ...................................... 35
Growth Factors .................................................... 35
Skin Grafting ....................................................... 35
End-of-Life Care for Wounds ......................... 36
Wound Care and Patient Education ............... 39
The Future .......................................................... 39
The Wound Institute® Course Guide User Chart .. 40
Glossary ............................................................... 41
Accreditations ...................................................... 42

Appendix 1

Lower Extremity Ulcer Characteristics ............... 43
Appendix 2

 Braden Scale for Predicting Pressure
 Sore Risk ......................................................... 44
Appendix 3 .......................................................... 45
 Tissue Destruction Classification Systems ........ 45
 All Wounds ....................................................... 45
Appendix 4

Skin Tears .......................................................... 45
Appendix 5 .......................................................... 46
Appendix 6

 Venous Ulcers .................................................... 47
Appendix 7 .......................................................... 48
Appendix 8

Diabetic Foot Ulcers ......................................... 48
Appendix 9

Peripheral Arterial Disease ............................ 49
Bibliography ....................................................... 50
Evolution of Wound Care

The importance of wound care has been realized since the dawn of recorded human history. A 4,000-year-old Sumerian clay tablet describes early wound care that included washing the wound in beer and hot water, using poultices from substances such as wine dregs and lizard dung, and bandaging the wound. For many thousands of years, a number of plants were commonly used in wound care. Many were astringent and some 2,500 had antimicrobial effects. Hippocrates (c400 BC) described the importance of draining pus, and Galen (c130 – 200 AD) wrote about first and second intention healing. Many common agents were discovered by primitive peoples in various parts of the world despite differences in climate and availability. Herbs were often applied to the wound in a balsam or given as a draught, and leaves or grasses were often used as bandages. In Mesopotamia, wounds were washed with water or milk and dressed with honey or resin.

Only in the 19th century was the importance of infection control, hemostasis and necrotic tissue realized, thus leading to a more rapid advance in wound care in our modern age. During World War I, chemist Henry Drysdale Dakin, on consultation, invented Dakin’s solution, a sodium hypochlorite and boric acid solution, to wash out traumatic wounds of British soldiers fighting in France. A dressing consisting of paraffin-impregnated gauze (tulle gras) was introduced by French physician Auguste Lumiere.

When the petrolatum gauze was introduced in 1942 by Drs. Allen and Koch, wound care moved towards moist dressings for the first time. In 1958, when George Odland observed that a blister healed faster if left unbroken, researchers began further investigation to discover the benefits of moist wound healing. In particular, the work of Dr. George Winter has formed the basis for moist wound healing theory. Dr. Winter demonstrated in pigs, in the late 1960s, that the rate of epithelialization of wounds covered in polyethylene films was double that of wounds healing under a dry scab. This, in turn, led to the use of urethane dressings in the 1970s. Urethane dressings offer the benefits of a moist environment plus management of wound exudates.

Today many adjunctive therapies have been introduced into the realm of wound care, such as enzymatic debrid- ing agents; biologically active dressings; extracellular matrix (ECM) replacement products; living skin equivalents; grafts; and biophysical agents, such as hyperbaric oxygen, electrical stimulation, negative pressure wound ther- apy, light and sound. These products facilitate wound closure and healing, taking therapeutic options to a whole new level.
The Skin

The skin is the largest organ of the body, weighing an average of 4 kg (8 pounds) and covering an area of 2m² (20 square feet). Its major function is to act as a barrier against an inhospitable environment – to protect the body from the influences of the outside world. By its very nature, the skin provides its own defense. The dry, dead, keratinized cells on the surface prevent most microorganisms from infiltrating into deeper layers. In addition, cells from the stratum corneum are shed every day, and attached microorganisms slough off with them. The importance of the skin is well illustrated by the high mortality rate associated with extensive loss of skin from burns. It has different layers which fulfill a number of important functions.

The Layers of the Skin and Their Functions

The skin is made up of three layers, each having its own function, but all working together to provide nutrients and protection. The three layers are:

**Epidermis:** The outermost layer of the skin, the epidermis is composed primarily of keratinocyte cells. It acts as the body’s major barrier against pathogens, preventing infections, in addition to regulating the amount of water released from the body. It contains no blood vessels but receives oxygen and nutrients by diffusion from the dermis.

**Dermis:** A mid-layer between the epidermis and subcutaneous layers, the dermis is the thickest layer of the skin and contains capillaries that feed the cells with nutrient-rich blood. One of the primary cells in this layer is the fibroblast. The dermis consists of connective tissue and contains hair follicles, as well as lymphatic and blood vessels, in addition to mechanoreceptors that provide the sense of touch and heat. The dermis consists of three types of tissues: collagen, elastic tissue and reticular fibers.

**Subcutaneous:** Also known as the hypodermis, this layer of fat and connective tissue houses larger blood vessels and nerves. One of the primary cells is the adipose cell. The subcutaneous layer attaches the dermis to the muscles and bones, and protects the underlying structures. This layer is important in the regulation of the temperature of the skin itself and the body in addition to the storage of fat serving as a ready source of energy.

Overall Functions of the Skin

- Protection against the elements
- Prevention of excessive water loss
- Excretion of waste
- Protection from pathogens
- Temperature regulation
- Sensation
- Production of Vitamin D folates
- Metabolism-Vitamin D synthesis
The Skin and Aging

Skin aging is a complex process in which most of the major changes occur in the dermal region. Aging skin is at an increased risk for injury because the skin is thinner and more fragile due to the declining height of the rete pegs or ridges which occurs throughout life, allowing for easier separation of the epidermis and dermis with minimal trauma. In addition, the ability of aging skin to sense touch, pressure, vibration, heat and cold may be reduced, thus putting older skin at higher risk for injury.58

Older adults often have an increase in the number of chronic conditions which impair wound closure and healing such as diabetes, heart disease, and peripheral arterial disease, to name a few. It is important to be aware of other intrinsic impediments to wound healing which are age-related. These include alterations in the body’s inflammatory response, decreased levels of growth factors necessary to facilitate the wound healing cascade, and delayed epithelialization.79

Frequently skin changes in older adults are seen on the surface of the skin, especially the forearms and hands, as they are more often exposed, but can occur anywhere on the body.19 Skin changes and loss of subcutaneous fat, combined with inactivity and immobility, nutritional deficiencies, decreased blood flow, and other illnesses have been shown to put older adults at higher risk for pressure ulcers.19, 53

Aging skin repairs itself up to four times slower than younger skin. Furthermore older adults have been shown to have a 25% lower proportion of closed wounds compared to younger patients, which contributes to potential infection in these wounds that stay open longer.19

<table>
<thead>
<tr>
<th>Common Conditions Contributing to Skin Disorders in Older Individuals19</th>
</tr>
</thead>
<tbody>
<tr>
<td>Blood vessel diseases such as arteriosclerosis/atherosclerosis</td>
</tr>
<tr>
<td>Diabetes</td>
</tr>
<tr>
<td>Heart disease</td>
</tr>
<tr>
<td>Liver disease</td>
</tr>
<tr>
<td>Nutritional and hydration deficiencies</td>
</tr>
<tr>
<td>Obesity</td>
</tr>
<tr>
<td>Reactions to medications</td>
</tr>
<tr>
<td>Stress</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Structural and Function Changes to Aging Skin18</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Structural Changes</strong></td>
</tr>
<tr>
<td>Dryness, roughness, wrinkling, increased laxity, decreased skin elasticity, and reduction in height of rete pegs/ridges</td>
</tr>
<tr>
<td><strong>Functional Changes</strong></td>
</tr>
<tr>
<td>Decreases in the following areas: barrier function, mechanical protection, sensory perception, wound healing, and immunologic responsiveness</td>
</tr>
</tbody>
</table>
Provision of high quality skin and wound care to older adults requires that caregivers have specific knowledge and skills. Table 1 below lists some recommendations for consideration when caring for older adults with chronic wounds.\textsuperscript{58}

**Table 1**

<table>
<thead>
<tr>
<th>Considerations for Chronic Wound Care in Older Adults</th>
</tr>
</thead>
</table>
| **Discuss goals of care with patient (family or caregiver)** | - Is healing a reasonable goal? This depends on the patient’s underlying medical conditions, life expectancy, functional and cognitive status, and ability to adhere to therapy.  
- Will the patient and wound benefit more from a maintenance plan of care? Is palliative or hospice care appropriate considering the patient’s overall medical condition?  
- If the wound is considered non-healable the focus should be:  
  - Educating the patient and/or family that healing may not be possible  
  - Explain what interventions will be provided (eg. dressings, systemic antibiotics, turning, support surfaces) |

| **Focus on:** | - Preserving quality of life  
- Pain management  
- Preserving the patient’s dignity  
- Controlling odor from wound  
- Preventing new wounds  
- Preventing wounds from worsening  
- Minimizing risk of sepsis from wounds (eg. using antiseptic interventions such as slow-releasing iodophors or silver dressings)  
- Minimizing frequency of dressing changes |

**Clinical Tip**

Rubbing or pulling on the skin can cause skin tears.\textsuperscript{19} Fragile blood vessels are easily broken.\textsuperscript{19} Bruises or hematomas, flat collections of blood (purpura), may form after even a minor injury.\textsuperscript{19, 58}

Gentle handling of older skin is a basic clinical practice that should be observed at all times.
The Healing Process of an Acute Wound

The most current understanding of wound management has been derived from studies of the healing process in acute wounds. Wounds caused by trauma or through surgery generally follow a well-defined wound healing process that involves four main stages. These are:

Coagulation – From initial injury to approximately three hours post injury, coagulation occurs, which is characterized by clotting and a vascular response. During the coagulation phase after injury, platelets initiate the wound healing process by releasing a number of soluble mediators, including various growth factors, which rapidly diffuse from the wound and attract inflammatory cells to the area of injury. Coagulation is the body’s process to quickly control and stop bleeding or blood flow through a blood vessel or organ.

Inflammation – Occurring within a few hours of injury and overlapping with coagulation, acute inflammation involves a complex series of biological activities that seek to remove any bacteria or debris present in the wound bed and to stabilize the wound bed (vs. neutralize or normalize). This allows the wound to progress to the proliferative phase. The inflammatory phase is initiated by the blood clotting and platelet degranulation pro-

Coagulation Phase

- 0-3-hours
- Main focus: Stop bleeding
- Vasoconstriction & clotting to stop bleeding
- Main cells: Platelets aggregate to form clot, and release the contents of their alpha granules including inflammatory mediators and growth factors
- Inflammatory cells attracted to wound site
cess. During this phase there is significant vasodilation, increased capillary permeability, complement activation, and migration of leukocytes and macrophages to the site of the wound. The neutrophils and macrophages engulf and destroy bacteria and release proteases, including collagenase, gelatinase and elastase, which degrade damaged ECM components, and secrete additional growth factors for wound healing. Inflammation is largely regulated by a class of molecules called cytokines, which have powerful stimulatory and inhibitory actions on inflammatory cells to stimulate production of proteases. Macrophages also stimulate the migration of fibroblasts, epithelial cells and endothelial cells to form granulation tissue around day five.

**Cell proliferation and repair of the matrix** – The proliferative phase of healing begins between 3 to 21 days post injury. This phase is characterized by granulation tissue formation, angiogenesis, re-epithelialization of the wound surface by keratinocytes, and contraction of the wound margins by myofibroblasts.

**Epithelialization and remodeling of scar tissue** – From day 21 to 2 or more years post injury, the wound enters the remodeling phase. During this phase, collagen fibers reorganize and remodel to gain tensile strength. A full-thickness wound scar regains about 80% of the former tensile strength.

---

**Inflammatory Phase**
- Immediately - 3-days
- Main focus: Clean wound
- Vasodilation
- Main cells: Leukocytes (neutrophils) & macrophages clean up cellular debris & bacteria to prevent infection
- More growth factors released
- Fibroblast and other cells attracted to wound site

**Proliferative Phase**
- 3 - 21 days
- Main focus: Angiogenesis, granulation tissue formation, & contraction of wound
- Main cells: Fibroblasts, myofibroblasts, keratinocytes

**Maturation/Remodeling Phase**
- 21-days - 1 1/2 - 2 years
- Main focus: Collagen fibers of scar tissue reorganized (collagen synthesis)
- Wound becomes stronger over time
- Scar tissue fragile in beginning

---

**The Wound Institute® Course**

Anatomy and Physiology of Skin and Underlying Tissues

In this course module, students will identify the major layers of skin and underlying tissue and discuss the primary functions of each.

**Acute Healing Process**

Table 2 provides a summary of the phases of healing and the main cells and their function in each phase of healing.59

| Table 2 |
|-----|-----|-----|-----|
| Time | Phases | Main Cell Types | Specific Events |
| Hours | Coagulation | Platelets | Platelet aggregation and release of fibrinogen fragments and other proinflammatory mediators |
| | Platelet aggregation and release of fibrinogen fragments and other proinflammatory mediators | Neutrophils, Monocytes | Endothelial selectins slow down blood cells. Binding to leukocyte integrins → diapedesis |
| | Hemidesmosome breakdown → keratinocyte migration | Macrophages | |
| | | Keratinocytes, Fibroblasts, Endothelial Cells | Signaling from MMPs, integrins, cytokines → cell migration, and ECM production |
| | | Myofibroblasts | MMPs break down disorganized collagen, growth factors stimulate fibroblasts to re-synthesize more organized collagen to improve strength of tissue |
| Days | Inflammation | | |
| | Cell recruitment and chemotaxis, wound debridement | | |
| | | | |
| Weeks to Months | Migration/Proliferation | | |
| | Epidermal resurfacing, fibroplasia, angiogenesis, ECM deposition contraction | | |
| | | | |
| | Remodeling | | |
| | Scar formation and revision, ECM degradation, further contraction and tensile strength | | |
The Biology and Healing Process of a Chronic Wound

A normal acute wound progresses towards healing because it has balanced inflammatory cytokines, rapid cell migration, cells that respond to growth factors, and low protease levels found in the wound fluid. In contrast, a chronic wound tends to have high inflammatory cytokines, high levels of degrading protease, and, cells are often senescent which makes them slow to respond to growth factors and slow to replicate and migrate, thus delaying wound healing. Chronic wounds or non-healing ulcers are therefore characterized by defective ECM, a failure to re-epithelialize, and prolonged inflammation.

---

The Wound Institute® Course

Guide to Chronic Wound Care

In this course, the student will review the types of wounds that are associated with chronic non-healing, discuss the normal healing process and pathophysiology of non-healing wounds, and apply evidence-based methods of wound bed preparation and dressings based on clinical assessment. The course also describes recent scientific and technological advances in chronic wound care, and outlines relative indications for considering patient referral to a specialized wound care clinic.

Types of Chronic Wounds

It is important to note that any wound is considered chronic, if it fails to close within three months. Chronic wounds are the result of complications that delay the normal wound healing process. All wound types have the potential to become chronic and, as such, chronic wounds are traditionally divided etiologically. Identifying and treating the underlying etiology of a chronic wound, such as venous insufficiency, arterial perfusion, diabetes, or unrelieved pressure, as well as systemic factors that may contribute to poor wound healing, such as nutritional status, immunosuppression and infection, are key to successful wound treatment.

Common types of chronic wounds include:

• Pressure Ulcers
• Venous Ulcers
• Arterial Ulcers
• Diabetic Ulcers
• Lymphedema

Pressure Ulcers

Definition: A pressure ulcer is a localized injury to the skin and/or underlying tissue, usually over a bony prominence, resulting from pressure, or pressure in combination with shear. A number of contributing or confounding factors are also associated with pressure ulcers, the primary of which is impaired mobility.

Etiology: The exact mechanism of pressure ulcer development is poorly understood. There are four theories that describe how these wounds develop:

1. Ischemia caused by occlusion of the capillaries leads to vascular insufficiency, tissue anoxia, and cell death.
2. Reperfusion injury occurs, which is a cellular injury resulting from the reperfusion of blood to previously ischemic tissue.
3. Impairment of lymphatic function leads to a buildup of metabolic waste products.
4. Mechanical stress deforms cells in tissue.

Characteristics: These wounds can develop on any area of the body where there is unrelieved pressure, but usually over boney prominences, such as the sacrum, ischium, heel, and trochanter, where there is less tissue to compress. They can have intact skin or be shallow to deep depending on the amount of tissue destruction. Pressure ulcers are staged to classify the degree of tissue damage that is present. See pressure ulcer staging (Appendix 5) for more details on the characteristics and staging of these wounds.

Pressure Ulcer Prevention: The initial primary goal related to pressure ulcers is prevention. Prevention of pressure ulcers requires health care providers to first determine the level of risk for developing pressure ulcers for each individual patient, utilizing a validated risk assessment tool such as the Braden Scale or Norton Scale (see Appendix 2 for Braden Scale). Once the level of risk for acquiring a pressure ulcer has been assessed, a plan of care is developed with interventions for each risk factor identified. There are intrinsic and extrinsic risk factors for pressure ulcer development (see Table 3 for a list of these Factors). The Centers for Medicaid and Medicare Services (CMS) require health care providers to prevent those pressure ulcers that are preventable. However, it is well-recognized by the healthcare community that not all pressure ulcers are preventable. There are times when the body is failing across systems, such as multi-organ failure, which often includes the skin. This is known as the unavoidable pressure ulcer, and is not directly associated with pressure, but rather a person’s co-morbidities at the end stage of life. (See page 36 for End-of-Life Care for Wounds).
The classification of pressure ulcers is documented according to the level of tissue involvement. [See Appendix 5 for the pressure ulcer stages definitions and pictures as defined and provided by the National Pressure Ulcer Advisory Panel (NPUAP)].

**Treatment:** Treatment of pressure ulcers first requires a comprehensive patient and wound assessment, then an individualized plan of care can be developed for each patient. Treatment consists of combinations of interventions including:

- Nutrition/hydration improvement
- Use of support surfaces (beds, overlays, and chair cushions)
- Turning/repositioning schedules for patients unable to readily reposition themselves
- Off-loading of heels
- Wound bed preparation which includes debridement of necrotic tissue, infection control, moisture/exudate control and ensuring wound edges can migrate to closure. (See pages 24-26 for a review of Wound Bed Preparation).
- Standard dressings
- Advanced wound care dressings
- Cellular and/or tissue-based products for wounds (CTPs)
- Various growth factors
- Biophysical technologies to facilitate closure and healing of chronic wounds such as: negative pressure wound therapy, electrical stimulation, electromagnetic agents, photo therapy (infrared, laser, ultraviolet light,) ultrasound and hyperbaric oxygen therapy.

In severe cases surgical interventions such as surgical flaps or grafts may be required, including bone resection in the presence of osteomyelitis. Pain should be assessed and treated as a standard practice for people with pressure ulcers.

**Clinical Tip**

Skin tensile strength is never as strong as before wounding in full-thickness wounds. Extra care should be taken to ensure the wound does not reoccur at same site before fully remodeled which can take up to two years.

---

**Table 3**

<table>
<thead>
<tr>
<th>Intrinsic Risk Factors</th>
<th>Extrinsic Risk Factors</th>
</tr>
</thead>
<tbody>
<tr>
<td>• History of previous pressure ulcer</td>
<td>• Inappropriate treatment protocols</td>
</tr>
<tr>
<td>• Age &gt;70 yrs</td>
<td>• Failure to recognize risk</td>
</tr>
<tr>
<td>• Immobility</td>
<td>• Inappropriate patient handling techniques</td>
</tr>
<tr>
<td>• Malnutrition/dehydration</td>
<td>• Use of restraints</td>
</tr>
<tr>
<td>• Urinary/fecal incontinence</td>
<td>• Poor hygiene</td>
</tr>
<tr>
<td>• Altered sensory perception</td>
<td>• Medications that impair the skin (i.e. steroids)</td>
</tr>
<tr>
<td>• Altered mental status</td>
<td></td>
</tr>
<tr>
<td>• Impaired oxygenation/perfusion</td>
<td></td>
</tr>
<tr>
<td>• Co-morbid conditions</td>
<td></td>
</tr>
<tr>
<td>• Altered blood pressure</td>
<td></td>
</tr>
</tbody>
</table>
Venous Ulcers

Etiology: Venous ulcers, the most common type of leg ulceration, are found in the lower extremities usually on the medial and pre-tibial aspect of the lower leg between the mid-calf and the medial malleolus. This region may often be referred to as the “gaiter area.” These wounds develop due to venous hypertension. Venous hypertension is thought to be caused by vein dysfunction and/or calf muscle pump failure. Normally, as the calf muscles contract during ambulation, blood is forced from the lower leg upward towards the heart by the opening and closing of one way valves. However, with venous hypertension, these valves are not able to approximate and close, creating backflow of the blood into the lower leg. This causes edema which may ultimately result in wounds. Edema is thought to contribute to damaging tissue due to three different theories; the fibrin cuff, leukocyte trapping and ischemia/reperfusion. Ultimately, venous disease and ulcerations result from a complex interaction of anatomy and hemodynamic failure.

Risk Factors: Risk factors for venous ulcers include age, family history, ligamentous laxity, central adiposity, long periods of time standing or sitting, cigarette smoking, pregnancy, trauma to lower leg, and intravenous drug use.

Characteristics: Venous ulcers are wounds of irregular shapes often with red granulating base and frequently with fibrotic tissue in the wound bed. They are usually located on edematous legs and have moderate to copious drainage. The periwound area often has hemosiderin staining (reddish/brown stain from the breakdown of red blood cells in the skin).

Treatment: Compression of the edematous lower leg to treat venous hypertension is the cornerstone for management of these wounds. Compression bandages are used and seek to relieve ambulatory venous pressure, edema and to increase venous return back to the central system. Compression forces of 30-40 mmHg are recommended; however, in the elderly, frail individual, or those with mixed etiology (venous and arterial insufficiency) lower levels of compression are recommended (20-30 mmHg). In some instances compression is contraindicated as when there is severe arterial insufficiency.

In addition to compression, debridement of necrotic tissue, moist wound healing practices, intermittent elevation above the heart, and exercise of the calf pump are core treatment considerations.

Surgical Approaches: Surgical approaches to correct venous insufficiency due to vein or valve impairments produce inconsistent results. NOTE: It is challenging to restore skin integrity permanently in the presence of chronic venous hypertension, because the underlying pathophysiology is not correctable. There is a need for ongoing control of venous hypertension and edema using life-long compression. The donning and doffing of garments are often difficult due to obesity, poor hand dexterity and strength. New strapped types of devices are helpful but may still be impossible to use for some. The use of pumps with sequential, gradient sleeves have been shown to be beneficial for some patients for long term management in addition to lower compression garments. Adherence to wearing compression garments for the rest of one’s life is difficult for most people to tolerate. Therefore, recurrence of these wounds is often as high as 40%.
Arterial Ulcers

Etiology: Arterial ulcers are wounds that will not heal due to inadequate blood flow to the lower extremities. This decreased blood flow, also known as arterial insufficiency (AI) or peripheral arterial disease (PAD), is primarily due to the atherosclerotic process with concurrent arterial hypertension which causes intimal layer damage to the arteries. As a result, the arterial lumen becomes occluded. Although it is possible to have spontaneous skin breakdown and ulceration due to PAD, it is more common for ulcers to result from some sort of trauma to an already ischemic limb.

Risk Factors: Risk factors for PAD ulcers include hyperlipidemia, smoking, diabetes, hypertension, trauma and advanced age.

Characteristics: Often patients with these wounds have intermittent claudication or rest pain, due to the PAD. The location of these wounds varies depending on the cause of trauma, however they are commonly found on the foot including on the top of the toes, and the anterior tibial and lateral aspect of the lower leg. They often have what is termed a “punched out” effect with well demarcated edges and a pale, non-granulating, often necrotic wound base. The surrounding skin may have a dusky erythema, be cool to touch, hairless, thin and shiny. Often the toenails are thickened. In addition, it is not uncommon to see gangrene on areas of the toes and foot. Usually there is a decreased or absent pulse in the posterior tibialis and dorsalis pedis arteries. Operative intervention is aimed at re-establishing blood flow through bypass surgery or lesser invasive procedures such as angioplasty or stenting. Other measures are to control diabetes or hyperlipidemia, to encourage patients to stop smoking, and to walk to tolerance.

Treatment: Increasing peripheral blood flow may be necessary using reconstructive surgery or angioplasty. There is little opportunity for healing without blood flow to the wounded area. In some cases revascularization is not possible, leaving amputation as the best course of action. Debridement of dry eschar vs. non-viable tissue should not be done in the absence of blood flow as there is insufficient vascularity to close the wounds, and they would likely re-necrose. Ulcers with adequate blood flow, as determined by vascular testing, should be treated with dressings which support moist wound healing practices. Protecting the limb from further trauma should be a focus of the care plan.
Diabetic Ulcers

Etiology: Diabetes and its associated complications have become pandemic, affecting 346 million people worldwide. Of this number approximately 26 million individuals have diabetes in the United States. Diabetic ulcers of the lower extremity, especially the foot, (DFU), are the single biggest risk factor for nontraumatic foot amputation in people with diabetes. A person with diabetes has a 15% to 25% lifetime chance of developing a DFU and a 50% to 70% recurrence rate over the following 5 years. A DFU precedes limb amputations in 85% of cases. Diabetic peripheral neuropathy (DPN), a well-recognized risk factor for ulceration, affects approximately 30% of people with diabetes.

The etiology of DPN in humans is not completely understood, however, in general, there appears to be a small-vessel vascular component (microangiopathy) and a metabolic component that damages nerves, demonstrating that both ischemic and metabolic mechanisms have a role in diabetic neuropathies. These neuropathic changes affect sensation, strength, balance and gait of the person with DPN. DFUs and amputations, consequences of diabetic neuropathy and/or peripheral arterial disease, are common and major causes of morbidity, disability, and mortality in people with diabetes.

Risk Factors: Risk factors for DFUs and for amputations from diabetes are similar. The DFU is a leading cause of amputation of digits, feet and/or legs especially in the presence of other amputation risk factors. (See Table 4 below for a list of Risk Factors).

Table 4

<table>
<thead>
<tr>
<th>Risk Factors for Lower Extremity Amputation in Patients with Diabetes</th>
</tr>
</thead>
<tbody>
<tr>
<td>- Absence of protective sensation</td>
</tr>
<tr>
<td>- Arterial insufficiency</td>
</tr>
<tr>
<td>- Foot deformity and callus formation from high pressure during gait</td>
</tr>
<tr>
<td>- Limited joint mobility</td>
</tr>
<tr>
<td>- Obesity</td>
</tr>
<tr>
<td>- Impaired vision</td>
</tr>
<tr>
<td>- Poor glucose control</td>
</tr>
<tr>
<td>- Cigarette smoking</td>
</tr>
<tr>
<td>- Poor footwear causing skin breakdown or providing inadequate protection for feet</td>
</tr>
</tbody>
</table>

Prevention of Diabetic Ulcers: Prevention of DFUs is considered one of the most important components of care for people with diabetes. Meticulous attention to foot care and proper management of minor foot injuries are key to the prevention of ulcer formation. The American Diabetes Association...
provides guidance in the Diabetes position statement, in their Standard of Medical Care, and recommends annual foot examinations for diabetic foot care which include:

- Assessment of foot pulses
- Testing for loss of protective sensation, loss of protective sensation (LOPS)
- General foot self-care education
- A multi-disciplinary approach for foot ulcers and high-risk feet
- On-going preventive care and life-time surveillance of feet for patients who smoke, have LOPS and structural deformities
- Initial screening for peripheral arterial disease which includes a history for claudication, assessment of pedal pulses and the ankle-brachial index
- Referral of patients with significant claudication or a positive ABI for further vascular assessment and consideration of exercise, medications, and surgical options

Characteristics:
Diabetic ulcers usually occur on the plantar surface of the foot due to the patient walking on a foot without sensation. There may be ulcers that form from blisters due to poorly fitted shoes. The wounds can be full or partial-thickness and infections are common with these wounds.

There are two classification systems used to describe a diabetic foot ulcer: the Wagner Scale, which focuses on the depth of tissue destruction, and the University of Texas San Antonio system, which includes depth, infection, and presence of ischemia. (See Appendix 8).

Treatment: Treatment of diabetic ulcers includes frequent debridement, moist wound care, treatment of infection, off-loading of the ulcer through appropriate techniques such as total-contact casting, and topical dressings. Concensus guidelines suggest that in the absence of reasonable healing that advanced modalities be considered. Growth factors, cellular and tissue based products and other advanced interventions may be appropriate for select patients and wounds. Primary prevention, through general practitioner examinations and foot hygiene, is essential to avoiding ulcer advancement. Patient education to prevent the initial ulcer and recurrence through self-inspection is a key intervention.

The Wound Institute® Course
Diabetic Foot Ulcers

This course covers learning to describe the changes in insulin/glucose regulation in diabetic patients, as well as, explaining the neuropathic and vascular changes that may contribute to the formation of foot ulcers. In this class, students discuss important physical, neurological, and vascular assessment tests commonly used in the assessment of diabetic foot ulcers and learn to identify and describe distinguishing characteristics of foot ulcers.

Atypical Wounds

In addition to the most common types of wounds previously mentioned, there are many other disease states and conditions that cause skin ulcers which are often referred to as atypical wounds. Causes of atypical wounds include infections, metabolic disorders, genetic diseases, neoplasms, and inflammatory processes. Atypical wound etiology should be considered when (1) the wound is in an unusual location, (2) the appearance is different from a common chronic wound, and (3) the wound does not respond to conventional interventions.

It is common for atypical wounds to appear similar to other types of chronic wounds, therefore a definitive diagnosis based on visualization alone is not sufficient. To determine if a wound is atypical, a tissue sample (biopsy) should be taken for histologic evaluation with special stains, tissue cultures (to determine infections), and immunofluorescence testing (to determine inflammatory or immune-based causes). A thorough patient history and physical examination is important to assist in accurately diagnosing the cause of the atypical wound and to determine appropriate treatment interventions. Treatment will be directly correlated to the etiology of the atypical wound.

Associated Conditions

Lymphedema & Phlebolymphedema

**Etiology:** Lymphedema is a chronic, incurable condition characterized by an abnormal collection of fluid due to anatomical alterations in the lymphatic system. Lymphedema can cause significant impairments in function, integumentary disorders (including open wounds), pain, and psychological issues. Lymphedema is classified into two main groups: primary and secondary. Primary lymphedema may be hereditary or congenital, whereas secondary lymphedema is usually due to some identifiable insult to the lymphatic system.

**Risk Factors:** Risk factors for lymphedema include family history, cancer treatment (lymph node dissection and radiation therapy) trauma, infections, various surgeries including knee, hip and pelvic surgery, post-phlebitis, and filariasis (parasitic infections). The most common cause worldwide is lymphatic filariasis. Certain mosquitoes carry larvae and when they bite a person, inject infectious larvae into the skin. Once the larvae mature into adult worms they excrete waste products which include symbiotic bacteria which progressively destroy the human lymphatic system. It is important to note that untreated or poorly treated venous insufficiency, can progress into a combined venous/lymphatic disorder, known as phlebolymphedema. Phlebolymphedema is treated the same as lymphedema.

**Characteristics:** Edema is a classic sign of lymphedema. Lymphedema edema is different from that of venous insufficiency which is mostly water, whereas lymphedema edema has high volumes of protein-rich materials causing the edema to be viscous. Patients may present with varying degrees of swelling severity from mild to grotesque enlargement, called elephantiasis.

**Treatment:** Lymphedema is technically irreversible end-stage lymphatic failure. However, with appropriate treatment, the quality of life for people with this condition can be dramatically improved. Treatment includes manual lymphatic drainage (MLD) by a therapist skilled in this technique, short-stretch compression bandaging, exercise to enhance muscle pump functions, and skin and nail care. Once the extremity is returned to as close to normal size as possible, the patient is ready for custom compression garments which will need to be worn life-long.
Overall Wound Management

Wound healing is a complex and interrelated physiological occurrence that is aided by effective wound management. It is important to consider that an interdisciplinary team approach best serves patients with chronic wounds. Each team member brings specialized skills and approaches in an attempt to reach the best wound care outcomes during this frequently difficult and complicated process. Essential to preparing a wound bed for healing is an accurate diagnosis of the underlying cause, usually determined by an in-depth patient and wound assessment.

The Wound Institute® Course

Principles of Wound Healing

Students will learn to identify the four major principles of wound healing (reduce/eliminate the cause, nutrition/patient support, prepare wound for healing, optimize wound environment) and will discuss appropriate care necessary to achieve healing.

Patient Assessment

Wound healing is determined not only by the etiology, but also by the general health of the patient, so a comprehensive patient assessment is crucial when creating the plan of care. The patient assessment will often include a questionnaire completed by the patient with or without the aid of a nurse and/or family member, depending on the relevant circumstances.

When evaluating a patient’s health status, the following, as well as other relevant topics, should be included in the patient report:

<table>
<thead>
<tr>
<th>Patient Report</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Full Medical History</strong></td>
</tr>
<tr>
<td><strong>Medication</strong></td>
</tr>
<tr>
<td><strong>Nutritional Status</strong></td>
</tr>
<tr>
<td><strong>Lifestyle</strong></td>
</tr>
<tr>
<td><strong>Quality of Life</strong></td>
</tr>
</tbody>
</table>
Properly Assessing a Chronic Wound

Diagnosing the underlying cause of a wound is an essential part of wound assessment. Not only can an inaccurate diagnosis of a wound delay wound healing, it can worsen the condition of the wound, causing unnecessary pain or, in some cases, leading to amputations, sepsis and/or death. In addition, it is important to consider that a chronic wound should have 30% smaller surface area at 4 weeks of wound care with closure in 12 weeks. If this goal is not met, then the patient, the wound, and the wound interventions should be reassessed thoroughly. The wound assessment is generally documented in a wound report. The following is an example of such a report. Other topics relevant to a particular patient and his or her wound(s) should also be included in the report.

<table>
<thead>
<tr>
<th>Wound Report</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Wound Location, Size and Type</strong></td>
</tr>
<tr>
<td><strong>Characteristics of the Wound Bed</strong></td>
</tr>
<tr>
<td><strong>Odor and Exudate</strong></td>
</tr>
<tr>
<td><strong>Condition of the Surrounding Skin</strong></td>
</tr>
<tr>
<td><strong>Clinical Signs of Colonization or Localized Infection</strong></td>
</tr>
<tr>
<td><strong>Pain</strong></td>
</tr>
</tbody>
</table>

The Wound Institute® Course

Wound Assessment and Documentation

This course describes critical elements associated with proper wound assessment and stresses the importance of documentation during the wound assessment process.

Wound Classification Systems

Wounds are classified using several different approaches. They are usually described by etiology, depth of tissue destruction, and types and/or color of tissues in the wound bed. (See Appendix 3 for tissue destruction classification systems).

Partial and Full-Thickness Wounds

Partial-thickness wounds have damage to the epidermis and dermis. However, tissue destruction does not extend below the dermal region. Skin tears, abrasions, serous blisters, and skin-graft donor sites are examples of partial-thickness wounds. Within the pressure ulcer staging system, Stage II pressure ulcers are partial-thickness wounds. Full-thickness wounds extend through the epidermis and dermis and may penetrate into the subcutaneous tissue, fascia and muscle down to bone.

Partial and full-thickness wounds heal differently. Partial-thickness wounds heal by resurfacing or re-epithelialization. Full-thickness wound heal by secondary intention with the formation of granulation tissue, contraction of the wound bed, and finally re-epithelialization. It is important to remember that full-thickness wounds do not ever regain their full tensile strength. In addition, these wounds continue to remodel for up to two years, before they gain their maximum strength, which is at most 80% of their pre-wounded strength. Therefore, full-thickness wounds are at risk for future recurrence, especially pressure ulcers and diabetic ulcers.

Pressure ulcers (see Appendix 5) and diabetic ulcers (see Appendix 8) each have well-known staging and classification systems to designate the depth of tissue involvement and healing methods. Venous (see Appendices 6 and 7) and arterial ulcers (see Appendix 9) also have less commonly used wound classification systems as do skin-tears (see Appendix 4). Burn injuries are described by the extent of the body burned in addition to depth of tissue destruction.
Managing Chronic Wounds

Wound Bed Preparation

Regardless of the specific wound type, local wound management principles exist for a wide variety of chronic wounds. Since 2000, the concept of Wound Bed Preparation (WBP) has been developed as a strategy that allows various aspects of wound care to be separated into individual components. This concept has had a dramatic impact on how we approach chronic wounds and how we view new and established therapies. WBP focuses on endogenous healing or on facilitating the effectiveness of other therapies. In addition, WBP allows healthcare professionals to define the steps involved in the management of chronic wounds through an understanding of the basic science, underlying the wound pathology. It is an approach that should be considered for all wounds that are not progressing through normal healing.
The WBP model is not a static framework, but can be adapted to suit all types of chronic wounds and can incorporate advances in wound management as new approaches emerge. The DIME\textsuperscript{65} acronym, is a simple, yet comprehensive method for defining, communicating, and addressing principal elements associated with impaired wound healing.\textsuperscript{65}

**Figure 1**

Wound bed preparation is employed to eliminate the barriers that can hamper the healing process. The basic goal is to achieve a stable wound that has healthy granulation tissue, and is characterized, by a well vascularized wound bed.\textsuperscript{24} Chronic wounds are commonly managed differently than acute wounds. This helps to optimize the scientific understanding and clinical management of non-healing wounds. Therefore WBP provides a model that is useful for the assessment and treatment of chronic wounds, as it is based on the clinical knowledge and functioning of non-healing ulcers.\textsuperscript{24} After eliminating barriers to healing including necrotic tissue, healthy granulation tissue is left to continue the healing process. Thorough understanding of the microenvironment is important because normal cellular and biochemical processes are inhibited in chronic wounds.\textsuperscript{24}
Components of Wound Bed Preparation

Debridement

“D” refers to debridement of devitalized or necrotic tissue (eschar and slough) that must be removed for wound healing to commence and to avoid infection. Firm eschar perpetuates the inflammatory stimulus which inhibits healing while slough acts as a medium for bacteria growth.65 Non-viable tissue can impede wound healing by blocking cellular migration, obstructing wound contraction, and making the process of wound evaluation more difficult.24 As a result, all non-viable tissue must be removed to promote the healing of the wound, usually through the process of debridement. Depending on the physiology of the wound, provider judgment, debridement skills of treating clinicians, available equipment and supplies, and patient choice, debridement can be achieved in five different methods: autolytic, enzymatic, mechanical, surgical/sharp, and biological (see pages 27-28).60, 63

There may be certain instances where debridement may not be needed. For example, sometimes it is preferable to leave a hardened eschar of dead tissue intact, than to remove it and create an open wound, in particular if the eschar is stable and there are no signs of infection. Other wounds may occur in places where blood flow is impaired and deciding not to debride the wound is a better option because blood flow may be insufficient for proper healing.

Infection

“I” characterizes inflammation or infection within and surrounding the wound site. Inflammation and infection may become apparent when wound margins fail to modify, or through increased exudate. Infection will generally cause discomfort for the patient, affect wound healing and have related negative effects.24 As presence of bacteria does not always indicate infection, infection is presumed when >10⁵ colony-forming organisms of bacteria per gram of tissue are found.24 Treatment of infection may include debridement, topical or systemic antibiotics, wound cleaning, and other topical antimicrobial agents.24

Moisture Imbalance

“M” reflects the state of moisture imbalance. Moisture imbalance slows wound healing, because epithelial migration is slowed by desiccation (drying out), and excess fluid causes maceration of the wound edge and the periwound skin.24 Examples of moisture balancing products are represented by hydrogels, absorbent dressings, moisture management dressings such as hydrogels, foams, alginates and hydrofibers.24 Managing moisture is essential to wound healing.

The Wound Institute® Course

Advances in Wound Healing: Exploring Treatment Modalities

This module defined autolytic debridement as the body’s natural ability to heal itself, compares and contrasts various debridement modalities mechanism of action by way of stimulating autolytic debridement, describe the science of enzymatic debridement and reviews challenging cases of difficult-to-heal wounds where enzymatic debridement agents were used.

Edge of Wound

“E” describes the quality of the wound edge which may be rolled (termed epiboly), non-advancing, hyperkeratotic (calloused) or undermined in the chronic wound, while also describing the extent of re-epithelialization. The clinical appearance of the edge of the wound is a key indicator of wound healing. A wound edge that fails to migrate needs reassessment by the clinician. The patient’s medical history may point to causes for the non-advancing wound edge. Of particular importance are pre-existing conditions such as diabetes or lifestyle factors such as stress and smoking.

Essential to wound healing is a well-vascularized wound bed. If a wound is not 30% smaller at week 4, despite optimal wound bed preparation, then it is unlikely to heal by week 12. At this point, consideration of more advanced treatment such as skin grafts, replacement of extracellular matrices, and artificial skin substitutes may be helpful (see page 36).
Treatment Algorithm*

The following is an example of a treatment algorithm from assessment to wound healing.77
Debridement Options

Efficient debridement is often an essential step in wound management. Chronic wounds are likely to require ongoing maintenance debridement rather than a single intervention. The underlying pathogenic abnormalities in chronic wounds cause a continual build-up of necrotic tissue, and regular debridement is necessary to reduce the necrotic burden and achieve healthy granulation tissue formation. Debridement also reduces wound contamination and therefore assists in reducing tissue destruction via infection/inflammation. The five methods of debridement each have their own advantages and limitations. Those methods that are most efficient at removal of debris may, at the same time, be the most detrimental to fragile new growth, and more than one method may be appropriate.

Autolytic
Autolytic debridement seeks to liquefy necrotic tissue using the body’s own endogenous enzymes. Dressings are used to retain the wound fluid which contains proteolytic enzymes that liquefy necrotic tissue. It occurs naturally in all wounds in healthy people. Autolytic debridement can be limited by the patient’s underlying conditions which affect immune status.

Enzymatic
Enzymatic debridement breaks down necrotic tissue with proteolytic agents such as collagenase by digesting necrotic strands of collagen where they attach to healthy collagen at the wound base. It provides faster elimination of necrotic tissue than autolysis and is generally safe, but may cause local irritation.

Mechanical
Mechanical debridement is a non-selective clinical intervention that involves manually removing a dressing that has proceeded from moist to dry (wet-to-dry dressing), hydrotherapy, ultrasound, and forceful irrigation of necrotic debris. This type of debridement is best used for wounds with a moderate amount of necrotic debris, however, mechanical debridement can be painful for the patient and may remove some healthy granulation tissue as it is not a selective method of debridement.

Surgical or Sharp Debridement
Surgical/sharp debridement is selective, efficient and fast, and may be performed with topical analgesia, pain medications, or in severe cases, under anesthesia in the operating room. Surgical debridement can be performed only by physicians as both non-viable and viable tissues may be removed. Sharp debridement (a more conservative form of debridement) may be performed by non-physicians (registered nurses and physical therapists) in states where their practice allows this intervention, and where they have the training and have demonstrated competency.
Biotherapy

Maggot debridement therapy (MDT) also known as larval therapy, biotherapy, or biosurgery, has deliberately been used in wound care dating as far back as the 16th century.1 MDT has three known effects: debriding necrotic tissue, ingesting bacteria and stimulating wound healing.1 There has been a resurgence in both research and practice as clinical evidence has shown maggots to effectively ingest both necrotic tissue and antibiotic resistant strains of bacteria in the wound bed.1

The Wound Institute® Course

Chronic Wounds: A Look at Causal Factors and the Role of Enzymatic Debridement

This monograph will help you identify the pathophysiologic characteristics of chronic wounds, including specific cellular factors that contribute to a wound’s chronicity, analyze the scientific data regarding the role of enzymatic debridement in the treatment of wounds and apply this understanding to patients with chronic wounds. Review of practical management strategies will take place to assist patients with chronic lower-limb wounds, including assessing for adequate vascular supply and debridement, infection control, appropriate use of off-loading measures, and edema control.

Wound Infections

Wound infections have been mentioned in the oldest discovered medical texts. Increased bacterial burden in ulcers has been shown to delay wound healing. Bacterial load has a progressive continuum from normal levels to abnormal levels which can damage tissues and/or the patient. (See Figure 2). Normally the skin has a healthy microflora consisting of bacteria and fungi which is essential for protecting the body against pathogenic organisms. This normal microflora is on the surface of the wound and does not delay or impair healing. The term for this level of microorganisms on the skin or in the wound is contamination.

The next level of bacterial presence in a wound is termed colonization, where the microorganisms replicate and are imbedded in the wound tissue, but do not harm the wound, or delay healing.

A critically colonized wound, sometimes described as a local wound infection, is where healing is no longer progressing at the expected rate, meaning the wound size is not decreasing and may actually increase in size. The patient may experience pain with increasing exudate and odor from the wound bed in the presence of either a critically colonized or infected wound.

Figure 2

Contaminated/Colonization
Bacteria on the wound surface (contaminated)
Bacteria in wound tissue, no tissue damage, does not delay healing (colonization).

Critically Colonized/Local Infection
Wound no longer healing. Granulation tissue may be friable and more painful, and exudate may increase.

Infection
Bacteria spread deep into wound and to surrounding tissues.

Systemic Infection
Invasion and multiplication of microorganisms in body tissues, especially that causes local cellular injury due to competitive metabolism, toxins, intracellular replication or antigen-antibody response.

Photos and chart courtesy of Pamela Scarborough, PT, Wimberley, TX
Critically Colonized versus an Infected Wound

In general a wound is considered infected when the bacterial load in the wound is $10^5$. However, this definition may be misleading as it doesn’t consider different bacterial strains that have different virulence and different people who have different levels of resistance to infection. Therefore, host resistance, the ability of the host to resist bacterial invasion and damage with an adequate immune response, is a critical factor in the equation for resisting bacterial damage to the wound or host.

Table 5

<table>
<thead>
<tr>
<th>Condition</th>
<th>Signs</th>
<th>Treatment</th>
</tr>
</thead>
</table>
| **Superficial Infection** | Any 3 criteria:  
  • Non-healing  
  • Exudate  
  • Red + Bleeding  
  • Debris  
  • Smell | Treat Topically (i.e. silver, iodine, honey) |
| **Deep Infection** | Any 3 criteria:  
  • Size bigger  
  • Temperature  
  • Os (probes to bone/exposed)  
  • New breakdown  
  • Exudate  
  • Erythema, Edema  
  • Smell | Treat Systemically |
| **Systemic Infection** | • Fever  
  • Rigors  
  • Hypotension  
  • Multi-organ failure | Treat Parenterally |
Biofilms

Biofilms are complex microbial communities that typically contain multiple species of bacteria and fungi. In marked contrast to single, non-attached (or very weakly attached) planktonic bacteria, the microorganisms in a biofilm community synthesize and secrete a protective matrix that attaches the biofilm firmly to a living or non-living surface.34

Infection Control

Treatment

Treatment According to Bacterial Status: When deciding which treatment to initiate on an infected wound, it is first important to determine the level of bacterial load in the wound. Table 6 gives a brief overview of the types of interventions determined by the bacterial load or status.

Table 6

<table>
<thead>
<tr>
<th>Level of Bacteria</th>
<th>Bacterial Status</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Surface bacteria</td>
<td>Contamination</td>
<td>Appropriate and regular wound cleansing, adequate dressing management</td>
</tr>
<tr>
<td>Superficial tissue involvement</td>
<td>Colonization Critical colonization</td>
<td>Topical antimicrobials</td>
</tr>
<tr>
<td>Surrounding and deep tissue invasion of bacteria</td>
<td>Infection</td>
<td>Topical and systemic agents</td>
</tr>
<tr>
<td>Systemic</td>
<td>Sepsis</td>
<td>Topical and systemic agents; may require parenteral management</td>
</tr>
</tbody>
</table>

Explanation of Table 6:

- **Surface bacteria** and **Contamination**: This category indicates that the bacterial load is low and manageable. The treatment includes appropriate and regular wound cleansing and adequate dressing management.
- **Superficial tissue involvement** and **Colonization/Critical colonization**: Indicates a moderate bacterial load. Topical antimicrobials are recommended.
- **Surrounding and deep tissue invasion of bacteria** and **Infection**: Here, the bacterial load is high, requiring both topical and systemic agents.
- **Systemic** and **Sepsis**: This category signifies the most severe bacterial load. Treatment includes topical and systemic agents, and may require parenteral management.

The Wound Institute® Course

Exploring Biofilms

In this module, students will learn the origin of biofilm theory, including traditional views of bacteria, and be able to define biofilm bacteria and related terms. The course will also cover the problem of biofilms and discuss methods of detection and treatment of biofilms.

Interventions

Products available to combat infection include: antiseptic cleansing solutions, antimicrobial/antibacterial dressings, and systemic antibiotics. Some antiseptics, such as povidone iodine, hydrogen peroxide and Dakin’s solution are known to be cytotoxic to cells critical to wound healing such as fibroblasts and keratinocytes. If properly diluted, use for short periods of time is appropriate. Decision making will be based on presence or absence of necrotic tissue, assumed degree of wound contamination, and goal of use: cleansing or disinfection. If wound cleansing is the desired outcome, normal saline or standard wound cleansers are appropriate.

Antimicrobial and Antibacterial Dressings

The use of silver in medicine has been documented for centuries. A renewed interest in the use of silver in topical antimicrobial dressings has resulted in multiple studies to assess its ability to reduce bacterial growth and the risk of wound infection, to manage active infection, and to reduce the risk of hospital-acquired wound infections. This renewed interest is largely attributed to silver’s bactericidal efficacy at low concentrations and its relatively limited toxicity to human cells. Silver has proven antimicrobial activity, which includes antibiotic-resistant bacteria such as methicillin-resistant S. aureus (MRSA) and vancomycin-resistant enterococci (VRE).78

Cadexomer Iodine

The term cadexomer iodine actually describes a delivery system rather than an antimicrobial agent. In this novel delivery system, the iodine is contained within a cadexomer starch bead. As wound exudate is drawn up, it enters the cadexomer bead, causing its openings to swell and allowing a slow, sustained release of the iodine molecules; it maintains a steady-state 0.9% concentration at the wound bed, providing a nontoxic level for healing wounds. Iodine is brown, and iodide, which is the inactive form of iodine, is colorless. The dressing progresses from brown to colorless, indicating that the iodine has been used and as a signal to change the dressing.41

Antibiotics

Topical antibiotics are the appropriate course of action to decrease the bacterial burden in chronic wounds with active but localized infection, but should be used with caution and for a prescribed length of time. Systemic antibiotics therapy should be used in chronic wounds where there is an active deep infection that extends into deeper tissue spaces. In addition, in the presence of a wound infection showing signs of a full-system host response (systemic infection), parenteral therapy should be administered.59

Methylene Blue / Gentian Violet

Impregnated into either polyvinyl alcohol (PVA) or polyurethane foams, these pigments are antibacterial against most clinically relevant bacteria. Coupled with its absorption, the methylene blue/gentian violet–bound foam traps and inhibits exudate-associated bacterial growth. In clinical use, the PVA foam also has been noted to reduce hypergranulation tissue, as well as flatten out slightly rolled wound edges, particularly in venous leg ulcers, allowing epidermal edge cellular migration.75
Polyhexamethylene Biguanide (PHMB)

PHMB is a biocide that has been used for many years with no known resistance. The gauze sponges have also been used extensively as part of the dressing interface layer with numerous negative-pressure wound therapy devices. The incorporation of PHMB into dressings, including gauze sponges, non-adherent dressings, foams, and a biosynthesized cellulose wound dressings, has been shown to be an effective barrier to protect a wound from outside contamination as well as to have bactericidal activity on relevant bacteria absorbed into the dressing material. By attaching itself to the bacterial cell membrane, PHMB causes structural changes that kill the bacteria.16
Moisture balance is critical to wound healing. There are a range of products available for this category depending on the amount and level of exudate.⁶⁴

### Overview of Product Categories

Clinicians have a wide variety of products from which to choose when treating wounds.

<table>
<thead>
<tr>
<th>Dressing Type</th>
<th>Description</th>
<th>Indications/Contraindications</th>
<th>Wound Types</th>
</tr>
</thead>
<tbody>
<tr>
<td>Calcium alginites</td>
<td>Sheet or fibrous rope which has hemostatic abilities.</td>
<td>Exudate management of full and partial-thickness wounds. / Not for dry wounds. Low tensile strength – avoid packing in deep sinuses.</td>
<td></td>
</tr>
<tr>
<td>Charcoal</td>
<td>Odor-absorbent charcoal within product.</td>
<td>Odor. / Some products are inactivated by moisture. Ensure that edges are sealed.</td>
<td></td>
</tr>
<tr>
<td>Composite dressings</td>
<td>Multi-layered, combination dressings to increase absorbency and autolysis. Primarily used as a secondary covering dressing.</td>
<td>For the management of acute and chronic wounds.</td>
<td></td>
</tr>
<tr>
<td>Films or membranes</td>
<td>Semi-permeable adhesive sheet. Impermeable to moisture and bacteria.</td>
<td>Management of superficial wounds as secondary dressing. Moisture vapor transmission rate varies. Not to be used on draining or infected wounds.</td>
<td></td>
</tr>
<tr>
<td>Foams</td>
<td>Non-adhesive or adhesive polyurethane foam, may have occlusive backing, sheets or cavity packing, and some have fluid lock.</td>
<td>On moderate to heavily draining wounds. / Occlusive foams not for infected or heavily draining wounds.</td>
<td></td>
</tr>
<tr>
<td>Hydrocolloids</td>
<td>May contain sodium, polysaccharides, gelatin, pectin. Sheet dressings are occlusive with polyurethane film outer layer.</td>
<td>For clean, granulating superficial wounds with low to medium exudate. Creates occlusive barrier which protects from outside contamination. / Not for infected wounds or heavily draining wounds.</td>
<td></td>
</tr>
<tr>
<td>Hydrogels</td>
<td>Polymers with high H₂O content. Gels, honey impregnated gauze or solid sheets.</td>
<td>Protects the wound and provides a moist environment for dry wounds. / Not for draining wounds. Solid sheets not for infected wounds.</td>
<td></td>
</tr>
<tr>
<td>Hydrophilic fibers</td>
<td>Sodium carboxymethylcellulose sheets which converts to a solid gel when activated by moisture.</td>
<td>For moderate amount of exudates. / Not for dry wounds. Low tensile strength, not for deep sinuses.</td>
<td></td>
</tr>
<tr>
<td>Hypertonic</td>
<td>Sheet, ribbon or gel impregnated with sodium concentrate.</td>
<td>Exudate management for full and partial-thickness wounds. / Ribbon not on dry wounds, but gel may be used. Could be painful on sensitive tissue.</td>
<td></td>
</tr>
<tr>
<td>Non-adherent layer</td>
<td>Wound contact layer with low adherence to tissue without backing. Non-medicated tulles.</td>
<td>Allows drainage to seep through pores to secondary dressing which facilitates the application of topical ointments.</td>
<td></td>
</tr>
<tr>
<td>Silicone dressings</td>
<td>A dressing coated with soft silicone and includes a variety of different dressing types such as atraumatic wound contact layers, absorbent dressings for exuding wounds and also a dressing for the treatment of hypertrophic scars and keloids.</td>
<td>Soft silicone dressings are suitable for almost all indications where it is important to prevent trauma to the wound and the surrounding skin and pain to the patient. The different types of soft silicone dressings meet different clinical needs.</td>
<td></td>
</tr>
</tbody>
</table>

Photos courtesy of Mobile Wound Solutions, Kansas City, MO.
Negative-Pressure Wound Therapy

Negative-Pressure Wound Therapy (NPWT) is the delivery of intermittent or continuous sub-atmospheric pressure to the wound bed. Foam or gauze dressings are sealed with a semi-occlusive film or hydrocolloid dressing to create an airtight system to maintain negative pressure through a regulated vacuum pump. The negative pressure delivered via the interface dressing causes cellular microdeformation (stretch) and macrodeformation (pulling the wound edges together) stimulating more rapid cell proliferation and formation of granulation tissue, removal of fluid and wastes, reduction of periwound edema, and enhancement of wound contraction.

Advanced Technology Therapies

Topical Collagen Dressings

Collagen solutions involve all types of wound dressings that are derived from porcine, avian or bovine sources. Topical collagen matrices can promote the healing of chronic wounds by absorbing excess saline solution or wound exudates through formation of a biodegradable sheet that balances wound moisture. Collagen is a biologically derived material which is the basic structural material of the body. Sources can vary, but most dressings are of bovine, porcine, or ovine origin. It is bioresorbable and biodegradable, so the primary function is not related to exudate management or moisture retention. Collagen is hemostatic by nature, and can enhance wound healing by attracting cells into the wound site, provide a temporary scaffolding to guide tissue in-growth, as well as modulate the effects of excessive matrix metalloproteases by acting as a sacrificial substrate.

Natural Extracellular Matrices

A natural extracellular matrix (ECM) is a bio-based complex network of macromolecules which forms a lattice-type structure that both binds cells and tissues together, and allows for them to communicate and interact efficiently. Product classes available in this area include: cadaveric human skin with epidermis and dermal cells removed, naturally occurring bio-scaffold derived from porcine tissue, and intact three-dimensional matrix naturally derived from porcine small intestine (SIS).

Cellular and Tissue Based Products

For almost 20 years, medical products have been developed that use biologically generated skin cells that can be used to stimulate wound healing through the use of cell therapy. The following classes of products are available: bi-layer bovine collagen matrix containing allogeneic human fibroblasts and keratinocytes isolated from human infant foreskin, human fibroblasts and extracellular matrix in a polyglactin mesh, and a bi-layer matrix of epidermal keratinocyte and dermal fibroblasts embedded into a bovine collagen sponge.

Growth Factors

Growth factors are key proteins involved in mediating cellular activity in wound repair. Platelet derived growth factor (PDGF) plays a key role in all phases of wound healing, and a recombinant form is the only topical growth factor approved by the FDA for the treatment of diabetic foot ulcers.

Skin Grafting

Skin grafting is a transplantation of human skin to sites where the existing skin is so damaged, that it can no longer function properly. It can also be used for non-healing wounds or 3rd degree burns. Skin grafting can be either an autograft which uses a thin layer of intact skin from another area of the patient’s body to close the wound, or the more risky allograft, which is donor skin, often a full-thickness layer, obtained from another human, usually from cadavers, that is frozen and stored until needed. A skin graft should improve the quality of the wound site, and may help to prevent the serious complications associated with burns or non-healing wounds.
End-of-Life Care for Wounds

End-of-life is defined as the phase of life when a person is living with an illness that will often worsen and eventually cause death.\textsuperscript{57} It is well accepted that during the end stages of life, any number of vital systems can falter and have devastating effects that may contribute to further deterioration and eventual death. The skin, being the largest organ of the body, is no different, and can become dysfunctional with varying degrees of physiological compromise. Skin organ compromise at life’s end is not a new concept in literature and was first described in early 19th century French medical literature by Jean-Martin Charcot.\textsuperscript{48}

The first clinical description in modern medical literature of an end-of-life ulcer appeared in 1989 with the Kennedy Terminal Ulcer (KTU).\textsuperscript{38} Based on a retrospective chart review, Kennedy described the KTU as a specific subgroup of pressure ulcers that some individuals develop as they are dying, which are usually shaped like a pear, butterfly or horseshoe, and are located predominantly on the coccyx or sacrum. Upon further evaluation of these individuals, it appears 55.7\% died within six weeks of discovery of their pressure ulcer(s).\textsuperscript{39} The observations were further supported by Hanson and colleagues who reported that 62.5\% of pressure ulcers in hospice patients occurred in the 2 weeks prior to death.\textsuperscript{35} Consequently, this observation has been noted by several studies in the early 2000s.
When this compromised state occurs, the manifestations are termed, “Skin Changes at Life’s End (SCALE)”. This term applies to all individuals across the continuum of care settings. In 2007, an international panel of experts met, and over a two year period developed the SCALE document, using a modified Delphi approach with input from multiple audiences and 69 international reviewers. The following 10 statements were proposed in the SCALE Final Consensus Document in 2009, for consideration when treating end of life patients with skin failure:

1. Physiologic changes that occur as a result of the dying process (days or weeks), may affect the skin and soft tissues and may manifest as observable (objective) changes in skin color, turgor, or integrity, or as subjective systems such as localized pain. These changes can be unavoidable and may occur with the application of appropriate interventions that meet or exceed the standard of care.

2. The plan of care and patient response should be clearly documented and reflected in the entire medical record. Charting by exception is an appropriate method of documentation.

3. Patient-centered concerns should be addressed including pain and activities of daily living.

4. Skin changes at life’s end are a reflection of compromised skin (reduced soft tissue perfusion, decreased tolerance to external insults, and impaired removal of metabolic wastes).

5. Expectations around the patient’s end-of-life goals and concerns should be communicated among the members of the inter-professional team, and the patient’s circle of care. The discussion should include the potential for SCALE including other skin changes, skin breakdown and pressure ulcers.

6. Risk factor symptoms and signs associated with SCALE have not been fully elucidated, but many include:
   a. Weakness and progressive limitation of mobility.
   b. Suboptimal nutrition including loss of appetite, weight loss, cachexia and wasting, low cerium albumin/pre-albumin, and low hemoglobin, as well as dehydration.
   c. Diminished tissue perfusion, impaired skin oxygenation, decreased local skin temperature, mottled discoloration and skin necrosis.
   d. Loss of skin integrity from any of a number of factors including equipment or devices, incontinence, chemical irritants, chronic exposure to body fluids, skin tears, pressure, shear, friction, and infections.
   e. Impaired immune functions.

7. A total skin assessment should be performed regularly and document all areas of concern consistent with the wishes and condition of the patient. Pay special attention to bony prominences and skin areas with underlying cartilage. Areas of special concern include the sacrum, coccyx, ischial tuberosities, trochanters, scapulae, occiput, heels, digits, nose and ears. Describe the skin or wound abnormality exactly as assessed.

8. Consultation with a qualified health care professional is recommended for any skin changes associated with increased pain, signs of infection, skin breakdown (when the goal may be healing) and whenever the patient’s circle of care expresses a significant concern.

9. The probable skin change etiology and goals of care should be determined. Consider the five Ps for determining appropriate intervention strategies.
   a. Prevention
   b. Prescription (may heal with appropriate treatment)
   c. Preservation (maintenance without deterioration)
   d. Palliation (provide comfort and care)
   e. Preference (patient desires)

10. Patients and concerned individuals should be educated regarding SCALE and the plan of care.
Patient education is a critical element to ensure wounds heal. In studies to evaluate the effects of a structured nurse-led education program aiming to improve patient concordance and prevent venous leg ulcer recurrence, it was found that a significant improvement occurred in the patient’s condition with a decreased recurrence of venous ulcers. Studies have also shown that persons with limited health literacy skills have higher utilization of hospital and emergency services and lower preventative care.

It is also essential that patient education is offered at the appropriate learning level for the patient. A JAMA published study found that:

- Only 42% of patients are able to understand directions for taking medicine on an empty stomach.
- 60% of patients do not understand an informed consent form.
- 33% of patients do not understand instructions for an upper GI tract x-ray written at a 4th grade level.

For patient education to be effective, it must be simple. This translates to plain language and simple graphics that are easy to use and understand.

The Wound Institute® offers patients and their caregivers useful information on preventing and caring for wounds. Twelve patient modules are available in English and Spanish:

- Debridement of Sores
- Diabetic Foot Care
- Diabetes and Meal Planning
- Eating Healthy
- Exercising for a Healthy Lifestyle
- Hand Washing
- Introduction to Diabetes
- Keeping your Skin Healthy
- SENSES: Sensing the Signs of Pressure Ulcers
- SCALE (End of Life Skin Care)
- Sores that Will Not Heal
- Treatment of Sores
The Future

Wound healing is a complex and dynamic process of restoring the cellular structures and tissue layers. Future advances in wound healing, will focus on the agents that influence the repair of damaged tissue. These may include laser techniques, stem cells, fetal tissue and other modalities that enhance the proliferation and migration of cells, and the acceleration of the healing process.

To help stay in touch with current information, please visit www.thewoundinstitute.com.
<table>
<thead>
<tr>
<th>Courses Offered</th>
<th>Course Completed (Check box for “yes”)</th>
<th>Date Completed</th>
<th>CNE Credits Earned</th>
<th>CNE Contact Hrs. Earned</th>
</tr>
</thead>
<tbody>
<tr>
<td>Advances in Wound Healing: Exploring Treatment Modalities</td>
<td>☐</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Anatomy and Physiology of Skin and Underlying Tissues</td>
<td>☐</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Chronic Wounds: A Look at Causal Factors and the Healing Role of Enzymatic Debridement</td>
<td>☐</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Chronic Wounds: Cellular Biology and Management</td>
<td>☐</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Diabetic Foot Ulcers</td>
<td>☐</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Exploring Biofilms</td>
<td>☐</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Guide to Chronic Wound Care</td>
<td>☐</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Principles of Wound Healing</td>
<td>☐</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Wound Assessment and Documentation</td>
<td>☐</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Angiogenesis: Formation of new blood vessels.

Antigen Presentation: Activity in which antigen-presenting cells (APCs), typically dendritic cells and macrophages, ingest and/or partially digest antigens and then present the antigen on their surfaces to T cells.

Apoptosis: Programmed cell death initiated when activating molecules bind to their specific receptors on target cells; mechanism to delete unwanted cells from the body.

Biofilm: Polysaccharide matrix that microorganisms produce and live in. Bacteria within biofilm respond to signals from other bacteria in the community to change their phenotype. Highly resistant to and poorly penetrated by antimicrobials. Must be removed by debridement. Reformation is prevented with antimicrobials and maintenance debridement (including autolysis).

Capillaries: Tiny blood vessels that pass blood from the arterioles into the venules. The capillaries perfuse the tissues of the body with needed oxygen and important nutrients supplied by blood.

Cytokine: A small protein released by cells that has a specific effect on the interactions between cells, on communications between cells or on the behavior of cells. The cytokines include the interleukins, lymphokines and cell signal molecules, such as tumor necrosis factor and the interferons, which trigger inflammation and respond to infections.

Epiboly: The growth of a rapidly dividing group of cells around a more slowly dividing group of cells, as in the formation of a gastrula.

Epithelialization: Regeneration of the epidermis across a wound surface.

Fibrin Cuff Theory: When the veins in the lower limbs are hypertensive, the condition can cause the walls of the capillaries to stretch. The pores are then more open, and proteins called fibrinogen as well as red blood cells leak into the interstitial areas. The fibrinogen becomes fibrin, which gathers around the capillary walls in bands called cuffs.

Fibroblast: Cell or corpuscle from which connective tissue is developed.

Full-Thickness: Tissue damage involving total loss of epidermis and dermis and extending into the subcutaneous tissue and possibly into the muscle or bone.

Granulation: Formation or growth of small blood vessels and connective tissue in a full-thickness wound.

Granulation Tissue: Pink/red, moist tissue composed of new blood vessels, connective tissue, fibroblasts, and inflammatory cells, which fills an open wound when it starts to heal. Typically appears deep pink or red with an irregular, “berry-like” surface.

Hemostasis: The body’s process to quickly control and stop bleeding or blood flow through a blood vessel or organ.

Hyperkeratosis: Hard, white/gray tissue surrounding a wound.

Inflammation: A complex series of biological activities that occur to remove any bacteria or debris present in the wound bed and allow the wound to progress to the proliferative phase.

Keratinocyte: The predominant cell type in the epidermis, the outermost layer of the human skin, constituting 95% of the cells found there.

Ligamentous laxity: a term given to describe “loose ligaments.”

Lymphocytes: A type of white blood cell, formed in the bone marrow, which is an important part of the immune system. Lymphocytes can make antibodies or kill infected cells.

Maturation and Remodeling: When the levels of collagen production and degradation equalize, the maturation phase of tissue repair is said to have begun.

Macrophage: Type of white blood cell that regulates wound repair through the ability to destroy bacteria and devitalized tissue and produce a variety of growth factors.

Necrosis: General term for death of cells, but the term is also used to describe death due to extreme variations in physiologic conditions (such as hypoxia or hypothermia).

Neuropathy: Neuropathic progression from functional to structural to nerve death.

Neutrophils: The most common type of white blood cells, comprising 50-70% of all white blood cells. Neutrophils are present in the bloodstream until signaled to a site of infection by chemical cues in the body (a process known as chemotaxis) which indicate that bacteria, necrotic tissue or a foreign body is present in a wound. They are the first immune cells to arrive at a site of infection and are phagocytic. The main role of neutrophils is to ingest invasive microbes and release proteins that kill bacteria.

Phagocytosis: The process in which phagocytes including neutrophils and macrophages, engulf and digest microorganisms and cellular debris.

Platelets: The smallest and lightest cells found in blood. Platelets play a fundamental role in hemostasis and are a natural source of growth factors. The principle function of platelets is to prevent bleeding.

Proliferative Phase: About two or three days after the wound occurs, fibroblasts begin to enter the wound site, marking the onset of the proliferative phase.

Protease: Enzyme capable of breaking down a protein.

Protease Inhibitor: Substance that blocks the activity of a protease.

Reactive Oxygen Species (ROS): Molecule containing an oxygen atom with an unpaired electron; highly reactive and damaging to other molecules.

Senescent Cells: Age-related decrease in proliferation potential of dermal fibroblasts; occurrence observed in chronic wounds in which fibroblasts have impaired responsiveness to growth hormone; response that may be due to increased number of senescent cells.

Slough: Soft moist avascular (devitalized) tissue; may be white, yellow, tan, or gray, may be loose or firmly adherent.

Vasodilation: Dilation of blood vessels, especially small arteries and arterioles; also called vasodilatation.

Wound Bed Preparation: The global management of the wound to accelerate endogenous healing or to facilitate the effectiveness of other therapeutic options.

Wound Margin: Rim or border of wound.
Accreditations

These educational activities are knowledge based activities that are made possible by the support of Smith & Nephew. There are no fees associated with these activities.

Target Audience: These activities are designed for physicians, podiatrists, nurses, and pharmacists who treat patients with wounds.

CME: NACCME designates this enduring material for a maximum of 1 AMA PRA Category 1 Credit™. Physicians should claim only the credit commensurate with the extent of their participation in the activity.

The Postgraduate Institute for Medicine (PIM) is accredited by the ACCME to provide continuing medical education for physicians.

CPME: North American Center for Continuing Medical Education, LLC (NACCME) is approved by the Council on Podiatric Medical Education as a provider of continuing education in podiatric medicine. NACCME has approved this activity for a maximum of 1 continuing education contact hour.

CNE: This continuing nursing education activity awards 1.0 contact hour. Provider approved by the California Board of Registered Nursing, Provider #13255 for 1.0 contact hour.

The Postgraduate Institute for Medicine (PIM) is an approved provider of continuing education by the Colorado Nurses Association, an accredited approver by the American Nurses Credentialing Center’s Commission on Accreditation, and approved provider by the California Board of Registered Nursing, Provider #13485.

CPE: This activity is approved for 1.0 contact hour (0.1 CEUs) of continuing pharmacy education (UAN 0275-0000-14-084-H01-P).

NACCME

Hardware/Software Requirements: All educational activities are accessible via a PC (Windows 2000/XP/Vista/7) or Mac (Mac OS 10.x or later) computer with current versions of the following browsers: Internet Explorer, Mozilla Firefox, Google Chrome, or Safari. Windows Media Player or compatible alternative, sound card, and speakers are required for streamed audio. The latest version of the Adobe Flash Player is suggested for video programs. A PDF reader is required for print publications. Please direct technical questions to webmaster@naccme.com.

Activity Overview: These on-demand webcast are available with synchronized slides and audio.

To be eligible for documentation of credit, participants must complete the educational activity, complete the 10-question online post-test with a score of 70% or better, and complete the evaluation form. After successful completion of the post-test and evaluation form online at www.naccme.com, participants may immediately print their documentation of credit.

There is no fee associated with these activities. These educational activities are knowledge-based activities. For questions regarding these educational activities, please call 609-371-1137.

PIM

PIM protects the privacy of personal and other information regarding participants, educational partners, and joint sponsors. PIM and our joint sponsors will not release personally identifiable information to a third party without the individual’s consent, except such information as is required for reporting purposes to the appropriate accrediting agency.

PIM maintains physical, electronic, and procedural safeguards that comply with federal regulations to guard your non-public personal information.
## Appendix 1
### Lower Extremity Ulcer Characteristics

<table>
<thead>
<tr>
<th>Wound Characteristics</th>
<th>Venous Ulcers</th>
<th>Arterial Ulcers</th>
<th>Diabetic Neuropathic Ulcers</th>
</tr>
</thead>
<tbody>
<tr>
<td>Photos</td>
<td><img src="image1" alt="Venous Ulcer" /></td>
<td><img src="image2" alt="Arterial Ulcer" /></td>
<td><img src="image3" alt="Diabetic Ulcer" /></td>
</tr>
<tr>
<td><strong>Most Common Locations</strong></td>
<td>Medial lower leg (“gaiter area”)</td>
<td>Anterior/Lateral lower leg and digits</td>
<td>Bony prominence of foot; Metatarsal head; Plantar; Pressure bearing surfaces; Heels</td>
</tr>
<tr>
<td><strong>Wound Bed</strong></td>
<td>Red granulation tissue; may contain fibrin/slough, moist to highly exudating</td>
<td>No or pale granulation tissue; Necrotic tissue/eschar; Dry</td>
<td>Varies, can be pale if arterial involvement; Commonly dry</td>
</tr>
<tr>
<td><strong>Depth/Edges</strong></td>
<td>Shallow, diffuse, irregular edges</td>
<td>Deep, punched out appearance</td>
<td>Shallow to deep, callused edges</td>
</tr>
<tr>
<td><strong>Pulses</strong></td>
<td>Present if no PAD</td>
<td>Faint or absent</td>
<td>Can be absent or faint, or may be normal or bounding.</td>
</tr>
<tr>
<td><strong>Edema</strong></td>
<td>Significant edema</td>
<td>Periwound edema</td>
<td>Not common</td>
</tr>
<tr>
<td><strong>Staining (Hemosiderin deposits)</strong></td>
<td>Periwound and lower leg hemosiderin staining common</td>
<td>None unless mixed disease present</td>
<td>None</td>
</tr>
<tr>
<td><strong>Wound Pain</strong></td>
<td>Typically yes, especially at site of ulceration, and often treatment related.</td>
<td>Typically yes</td>
<td>Depends on level of neuropathy; May see increased pain w/ infection</td>
</tr>
<tr>
<td><strong>Limb Pain</strong></td>
<td>Typically yes, when leg is dependent. Aching of leg is a common early sign.</td>
<td>Yes, when leg is at work (intermittent claudication) or when elevated</td>
<td>Not typically</td>
</tr>
<tr>
<td><strong>Ankle Brachial Index=ABI</strong></td>
<td>Adequate blood flow; ABI&gt;0.8 unless mixed disease with PAD</td>
<td>Blood flow impaired; ABI&lt;0.8</td>
<td>Varies; ABI may be falsely elevated due to calcification of the vessels with diabetes; Toe pressures are more reliable</td>
</tr>
</tbody>
</table>
# Appendix 2
Braden Scale for Predicting Pressure Sore Risk

<table>
<thead>
<tr>
<th>Patient’s Name</th>
<th>Evaluator’s Name</th>
</tr>
</thead>
</table>

## Sensory Perception

**Ability to respond fully to pressure-related discomfort**

<table>
<thead>
<tr>
<th>Level</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Completely Limited</td>
<td>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.</td>
</tr>
<tr>
<td>2. Very Limited</td>
<td>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 ¼ of body.</td>
</tr>
<tr>
<td>3. Slightly Limited</td>
<td>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 one or two extremities.</td>
</tr>
<tr>
<td>4. No Impairment</td>
<td>Responds to verbal commands. Has no sensory deficit which would limit ability to feel or voice pain or discomfort.</td>
</tr>
</tbody>
</table>

## Moisture

**Degree to which skin is exposed to moisture**

<table>
<thead>
<tr>
<th>Level</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Constantly Moist</td>
<td>Skin is kept moist almost constantly by perspiration, urine, etc. Dampness is detected every time patient is moved or turned.</td>
</tr>
<tr>
<td>2. Very Moist</td>
<td>Skin is often, but not always, moist. Linen must be changed at least once a shift.</td>
</tr>
<tr>
<td>3. Occasionally Moist</td>
<td>Skin is occasionally moist, requiring an extra linen change approximately once a day.</td>
</tr>
<tr>
<td>4. Rarely Moist</td>
<td>Skin is usually dry, linen only requires changing at routine intervals.</td>
</tr>
</tbody>
</table>

## Activity

**Degree of physical activity**

<table>
<thead>
<tr>
<th>Level</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Bedfast</td>
<td>Confined to bed.</td>
</tr>
<tr>
<td>2. Chairfast</td>
<td>Ability to walk severely limited or non-existent. Cannot bear own weight and/or must be assisted into chair or wheelchair.</td>
</tr>
<tr>
<td>3. Walks Occasionally</td>
<td>Walks occasionally during the day, but for very short distances, with or without assistance. Spends majority of each shift in bed or chair.</td>
</tr>
<tr>
<td>4. Walks Frequently</td>
<td>Walks outside room at least twice a day and inside room at least once every two hours during waking hours.</td>
</tr>
</tbody>
</table>

## Mobility

**Ability to change and control body position**

<table>
<thead>
<tr>
<th>Level</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Completely Immobile</td>
<td>Does not make even slight changes in body or extremity position without assistance.</td>
</tr>
<tr>
<td>2. Very Limited</td>
<td>Makes occasional slight changes in body or extremity position but unable to make frequent or significant changes independently.</td>
</tr>
<tr>
<td>3. Slightly Limited</td>
<td>Makes frequent though slight changes in body or extremity position independently.</td>
</tr>
<tr>
<td>4. No Limitation</td>
<td>Makes major and frequent changes in position without assistance.</td>
</tr>
</tbody>
</table>

## Nutrition

**Usual food intake pattern**

<table>
<thead>
<tr>
<th>Level</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Very Poor</td>
<td>Never eats a complete meal. Rarely eats more than 1/2 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 IVs for more than 5 days.</td>
</tr>
<tr>
<td>2. Probably Inadequate</td>
<td>Rarely eats a complete meal and generally eats only about 1/2 of any food offered. Protein intake includes only 3 servings of meat or dairy products per day. Occasionally will take a dietary supplement. OR received less than optimum amount of liquid diet or tube feeding.</td>
</tr>
<tr>
<td>3. Adequate</td>
<td>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.</td>
</tr>
</tbody>
</table>

## Friction & Shear

<table>
<thead>
<tr>
<th>Level</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Problem</td>
<td>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.</td>
</tr>
<tr>
<td>2. Potential Problem</td>
<td>Moves freely 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.</td>
</tr>
<tr>
<td>3. No Apparent Problem</td>
<td>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.</td>
</tr>
</tbody>
</table>

Total Score
Appendix 3
Tissue Destruction Classification Systems

Tissue destruction classification systems have been developed to assist describing the depth of penetration of wounds. Some systems are specific to the wound etiology such as the pressure ulcer staging system. Documenting the levels of tissue destruction correctly is critical for communication between clinicians and in some instances, required for reporting to CMS. For instance, CMS regulatory mandates require accurate staging for reporting, reimbursement, and tracking of pressure ulcers in different health care settings.

All Wounds

All wounds, regardless of etiology, can be assessed as either partial or full-thickness.

<table>
<thead>
<tr>
<th>Classification of Wound by Thickness of Tissue Destruction</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Partial-thickness</strong></td>
</tr>
<tr>
<td>Extends through the epidermis (first layer of skin), but not through dermis (second layer)</td>
</tr>
<tr>
<td><strong>Full-thickness</strong></td>
</tr>
<tr>
<td>Extends through epidermis and dermis; may involve subcutaneous tissue, muscles, joint capsule, bone, etc.</td>
</tr>
</tbody>
</table>

Appendix 4
Skin Tears

<table>
<thead>
<tr>
<th>Star Skin Tear Classification System^{13, 42}</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Category 1a</strong></td>
</tr>
<tr>
<td>A skin tear where the edges can be realigned to the normal anatomical position (without undue stretching) and the skin or flap colour is not pale, dusky or darkened.</td>
</tr>
<tr>
<td><strong>Category 1b</strong></td>
</tr>
<tr>
<td>A skin tear where the edges can be realigned to the normal anatomical position (without undue stretching) and the skin or flap colour is pale, dusky or darkened.</td>
</tr>
<tr>
<td><strong>Category 2a</strong></td>
</tr>
<tr>
<td>A skin tear where the edges cannot be realigned to the normal anatomical position and the skin flap colour is not pale, dusky or darkened.</td>
</tr>
<tr>
<td><strong>Category 2b</strong></td>
</tr>
<tr>
<td>A skin tear where the edges cannot be realigned to the normal anatomical position and the skin flap colour is pale, dusky or darkened.</td>
</tr>
<tr>
<td><strong>Category 3</strong></td>
</tr>
<tr>
<td>A skin tear where the skin flap is completely absent.</td>
</tr>
</tbody>
</table>
Payne-Martin Skin Tear Classification System

<table>
<thead>
<tr>
<th>Category</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Category I</td>
<td>No tissue loss, may be linear (tears without tissue loss, resembling an incision) or flaps (epidermal flap covers the dermis to within 1 mm of the edge).</td>
</tr>
<tr>
<td>Category II</td>
<td>Tissue loss. Scant, partial tissue loss = 25% or less of epidermal flap lost. Moderate to large partial tissue loss = more than 25% of epidermal flap is lost.</td>
</tr>
<tr>
<td>Category III</td>
<td>Complete tissue loss; no epidermal flap.</td>
</tr>
</tbody>
</table>

Appendix 5

International NPUAP-EPUAP Pressure Ulcer Classification System

For more information, visit www.npuap.com

<table>
<thead>
<tr>
<th>Stage</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stage I</td>
<td>Intact skin with non-blanchable 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.</td>
</tr>
<tr>
<td>Stage II</td>
<td>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 blister.</td>
</tr>
<tr>
<td>Stage III</td>
<td>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 include undermining and tunneling.</td>
</tr>
<tr>
<td>Stage IV</td>
<td>Full-thickness tissue loss with exposed bone, tendon or muscle. Slough or eschar may be present on some parts of the wound bed. Often include undermining and tunneling.</td>
</tr>
<tr>
<td>Unstageable</td>
<td>Full-thickness tissue loss in which the base of the ulcer is covered by slough (yellow, tan, gray, green or brown) and/or eschar (tan, brown or black) in the wound bed.</td>
</tr>
<tr>
<td>Suspected Deep Tissue Injury</td>
<td>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.</td>
</tr>
</tbody>
</table>

The depth of a Stage III pressure ulcer varies by anatomical location. The bridge of the nose, ear, occiput and malleolus do not have subcutaneous tissue and Stage III ulcers can be shallow. In contrast, areas of significant adiposity can develop extremely deep Stage III pressure ulcers. Bone/tendon is not visible or directly palpable.

The depth of a Stage IV pressure ulcer varies by anatomical location. The bridge of the nose, ear, occiput and malleolus do not have subcutaneous tissue and these ulcers can be shallow. Stage IV ulcers can extend into muscle and/or supporting structures (e.g., fascia, tendon or joint capsule) making osteomyelitis possible.

Until enough slough and/or eschar is removed to expose the base of the wound, the true depth, and therefore stage, cannot be determined. Stable (dry, adherent, intact without erythema or fluctuance) eschar on the heels serves as “the body’s natural (biological) cover” and should not be removed.

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 wound may further evolve and become covered by thin eschar. Evolution may be rapid exposing additional layers of tissue even with optimal treatment.
# Appendix 6

## Venous Ulcers

<table>
<thead>
<tr>
<th>Attribute</th>
<th>Absent = 0</th>
<th>Mild = 1</th>
<th>Moderate = 2</th>
<th>Severe = 3</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>PAIN</strong></td>
<td>None</td>
<td>Occasional, not restricting activity or requiring pain medication</td>
<td>Daily moderate activity limitation; occasional pain medication</td>
<td>Daily, severe limiting of activities or requiring regular use of pain medications</td>
</tr>
<tr>
<td><strong>VARICOSE VEINS</strong></td>
<td>None</td>
<td>Few scattered</td>
<td>Multiple; great saphenous veins, confined to calf and thigh</td>
<td>Extensive: thigh and calf or great and small saphenous distribution</td>
</tr>
<tr>
<td><strong>VENOUS EDEMA</strong></td>
<td>None</td>
<td>Evening ankle swelling</td>
<td>Afternoon swelling, above ankle</td>
<td>Morning swelling above ankle and requiring activity change, elevation</td>
</tr>
<tr>
<td><strong>SKIN PIGMENTATION</strong></td>
<td>None</td>
<td>Diffuse, but limited in area and old (brown)</td>
<td>Diffuse over most of gaiter distribution (lower third) or recent pigmentation (purple)</td>
<td>Wider distribution (above lower third) plus recent pigmentation</td>
</tr>
<tr>
<td><strong>INFLAMMATION</strong></td>
<td>None</td>
<td>Mild cellulitis, limited to marginal area around ulcer</td>
<td>Moderate cellulitis, involves most of gaiter (lower third)</td>
<td>Severe cellulitis (lower third and above) or significant</td>
</tr>
<tr>
<td><strong>INDURATION</strong></td>
<td>None</td>
<td>Focal, circummalleolar</td>
<td>Medial or lateral, less than lower third of leg</td>
<td>Entire lower third of leg or more</td>
</tr>
<tr>
<td><strong>NUMBER OF ACTIVE ULCERS</strong></td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>&gt;2</td>
</tr>
<tr>
<td><strong>ACTIVE ULCER DURATION</strong></td>
<td>None</td>
<td>&lt;3 months</td>
<td>&gt;3 months, &lt;1 year</td>
<td>Not healed &gt;1 year</td>
</tr>
<tr>
<td><strong>ACTIVE ULCER DIAMETER</strong></td>
<td>None</td>
<td>&lt;2 cm</td>
<td>2-6 cm</td>
<td>&gt;6 cm</td>
</tr>
<tr>
<td><strong>COMPRESSION THERAPY</strong></td>
<td>Not used or patient not compliant</td>
<td>Intermittent use of stockings</td>
<td>Wears elastic stocking most days</td>
<td>Full compliance, stockings + elevation</td>
</tr>
</tbody>
</table>

Attribute Absent = 0  Mild = 1  Moderate = 2  Severe = 3
### Appendix 7

<table>
<thead>
<tr>
<th>Class Descriptors</th>
<th>CEAP Classification</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>C- Clinical signs</strong></td>
<td><strong>Descriptors</strong></td>
</tr>
<tr>
<td>Six Grades, supplemented by (S) for symptomatic or (A) for asymptomatic presentation</td>
<td>C0: No visible or palpable signs of venous disease</td>
</tr>
<tr>
<td></td>
<td>C1: Telangiectasies or reticular veins</td>
</tr>
<tr>
<td></td>
<td>C2: Varicose veins; distinguished from reticular veins by having 3mm or more diameter</td>
</tr>
<tr>
<td></td>
<td>C3: Edema</td>
</tr>
<tr>
<td></td>
<td>C4: Changes in skin &amp; subcutaneous tissue</td>
</tr>
<tr>
<td></td>
<td>C4a: Pigmentation or eczema</td>
</tr>
<tr>
<td></td>
<td>C4b: Lipodermatosclerosis or atrophie blanche</td>
</tr>
<tr>
<td></td>
<td>C5: Healed venous ulcer</td>
</tr>
<tr>
<td></td>
<td>C6: Active venous ulcer</td>
</tr>
<tr>
<td><strong>Symptoms:</strong> pain, aching, tightness, skin irritation, heaviness, muscle cramps or other complaints due to venous dysfunction</td>
<td>Example: C3A</td>
</tr>
<tr>
<td><strong>E- Etiologic factors</strong></td>
<td>Ec: Congenital</td>
</tr>
<tr>
<td></td>
<td>Ep: Primary</td>
</tr>
<tr>
<td></td>
<td>Es: Secondary (postthrombotic)</td>
</tr>
<tr>
<td></td>
<td>En: No venous cause identified</td>
</tr>
<tr>
<td><strong>A- Anatomic distribution</strong></td>
<td>As: Superficial veins</td>
</tr>
<tr>
<td></td>
<td>Ap: Perforator veins</td>
</tr>
<tr>
<td></td>
<td>Ad: Deep veins</td>
</tr>
<tr>
<td></td>
<td>An: No venous location identified</td>
</tr>
<tr>
<td><strong>P- Pathophysiologic dysfunction</strong></td>
<td>Pr: Reflux</td>
</tr>
<tr>
<td></td>
<td>Po: Obstruction</td>
</tr>
<tr>
<td></td>
<td>Pro: Reflux &amp; obstruction</td>
</tr>
<tr>
<td></td>
<td>Pn: No venous pathophysiology identifiable</td>
</tr>
</tbody>
</table>

### Appendix 8

**Diabetic Foot Ulcers**

<table>
<thead>
<tr>
<th>Grade</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Grade 0</td>
<td>Pre-ulcerous lesion, healed ulcer, bony deformity</td>
</tr>
<tr>
<td>Grade 1</td>
<td>Superficial ulcer without subcutaneous tissues involvement</td>
</tr>
<tr>
<td>Grade 2</td>
<td>Deep ulcer, penetration through the subcutaneous tissue; may have exposed bone, tendon or ligament or joint capsule</td>
</tr>
<tr>
<td>Grade 3</td>
<td>Deep ulcer with cellulitis, abscess formation, or osteomyelitis</td>
</tr>
<tr>
<td>Grade 4</td>
<td>Localized gangrene of digit</td>
</tr>
<tr>
<td>Grade 5</td>
<td>Extensive gangrene involving whole foot</td>
</tr>
</tbody>
</table>
### University of Texas Classification System of Diabetic Foot Ulcers

<table>
<thead>
<tr>
<th>0</th>
<th>I</th>
<th>II</th>
<th>III</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>Pre-or post-ulcerative lesion</td>
<td>Superficial wound, not involving tendon, capsule, or bone</td>
<td>Wound penetrating to tendon or capsule</td>
</tr>
<tr>
<td>B</td>
<td>Pre-or post-ulcerative lesion</td>
<td>Pre- or post-ulcerative lesion, completely epithelialized with infection</td>
<td>Superficial wound, not involving tendon, capsule, or bone with infection</td>
</tr>
<tr>
<td>C</td>
<td>Pre- or post-ulcerative lesion, completely epithelialized with ischemia</td>
<td>Superficial wound, not involving tendon, capsule, or bone with ischemia</td>
<td>Wound penetrating to tendon or capsule with ischemia</td>
</tr>
<tr>
<td>D</td>
<td>Pre- or post-ulcerative lesion, completely epithelialized with infection and ischemia</td>
<td>Superficial wound, not involving tendon, capsule, or bone with infection and ischemia</td>
<td>Wound penetrating to tendon or capsule with infection and ischemia</td>
</tr>
</tbody>
</table>

### Rutherford Classification for PAD

<table>
<thead>
<tr>
<th>Stage 0</th>
<th>Asymptomatic</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stage 1</td>
<td>Mild claudication</td>
</tr>
</tbody>
</table>
| Stage 2 | Moderate claudication  
The distance that delineates mild, moderate and severe claudication is not specified in the Rutherford classification, but is mentioned in the Fontaine classification as 200 meters. |
| Stage 3 | Severe claudication |
| Stage 4 | Rest pain |
| Stage 5 | Ischemic ulceration not exceeding ulcer of the digits of the foot |
| Stage 6 | Severe ischemic ulcers or frank gangrene |


68. Weinberg L., Rutherford Classification, Vascular Medicine, Angiologist, Aug, 2010


73. Weinberg L., Rutherford Classification, Vascular Medicine, Angiologist, Aug, 2010


Special Recognition

We would like to thank the following organizations and individuals for their support in creating this piece.

Pamela Scarborough, PT, DPT, MS, CDE, CWS, CEEAA
Dot Weir, RN, CWON, CWS
National Pressure Ulcer Advisory Panel (www.npuap.org)
Mobile Wound Solutions, Kansas City, Mo
American Association for the Advancement of Wound Care (http://aawconline.org)

*The Association for the Advancement of Wound Care (AAWC) is the leader in interdisciplinary wound healing and tissue preservation. For more information, visit www.aawconline.org.