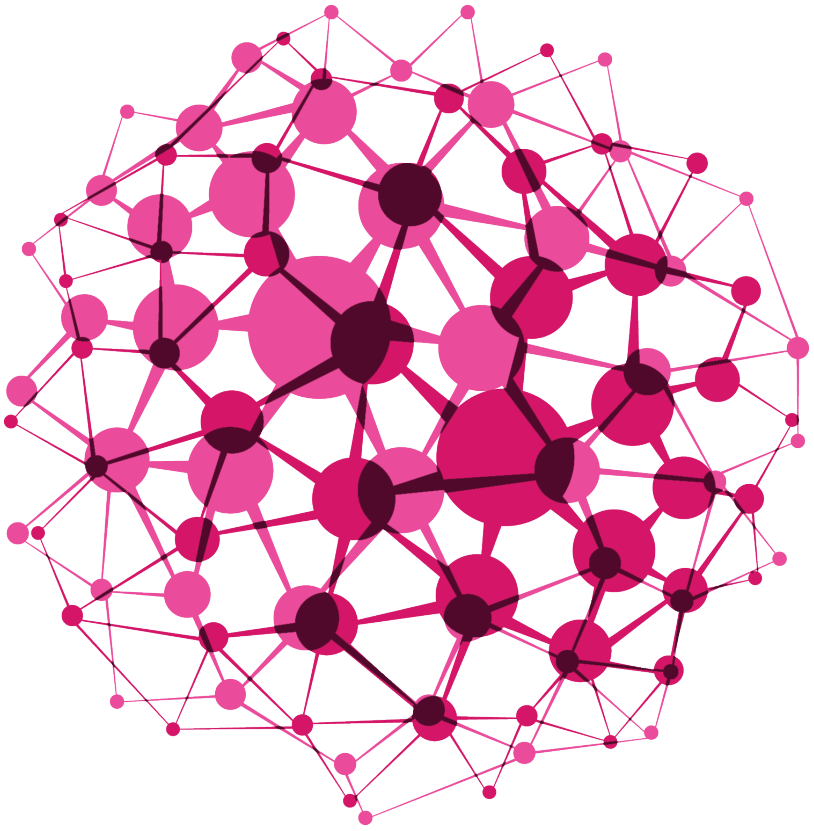

CHRONIC WOUNDS

Providing Efficient and Effective Treatment

Barbara Acello, MS, RN

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HCPro

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Introduction

Many lives have been saved and the human lifespan has been extended markedly as a result of antibiotics. Penicillin, streptomycin, chloramphenicol, and tetracycline were all introduced between 1945 and 1955. Before that, we had no truly effective means of fighting infection. The situation today is not quite as abysmal but is rapidly marching in that direction. The pathogens have effectively changed themselves and become "superbugs" that are drug resistant. A microorganism considered multidrug resistant (MDR) is resistant to one or more classes of antimicrobial agents.[1]

Bacteria began developing resistance to antibiotics immediately. Scientists recognized the problem and developed many new generations of antibiotics, but some have been removed from the market for a variety of reasons. By the early 1960s, 80% of all *Staphylococcus aureus* infections were resistant to penicillin.

Methicillin was originally licensed in the United Kingdom in 1959. One strain of the *S. aureus* pathogen (MRSA-I) developed resistance to methicillin very soon thereafter, in about 1961. Two additional strains (MRSA-II and MRSA-III) were identified in the late 1970s. A fourth type of MRSA (MRSA-IV) was identified in the 1990s.

Community-acquired MRSA (CA-MRSA) appeared in the early 2000s. Until that point, the spread of MRSA was limited to healthcare facilities and was primarily confined to high-risk, immunocompromised persons. CA-MRSA occurs outside of facility settings and is being contracted by healthy persons of all ages.

By 2002, approximately 57% to 71% of all staph infections were methicillin resistant. (Published sources vary on the number.) The first vancomycin-resistant staph strains (VRSA) were also identified in 2002.

Terminology

You may also hear MRSA called oxacillin-resistant *S. aureus* (ORSA), although this is less common. *S. aureus* may be referred to as methicillin sensitive (MSSA). In 2005, the name was officially changed to *meticillin* in accordance with the International Pharmacopoeia guidelines.

Drug Research and Development

The new drug discovery pipeline has slowed to a trickle and has almost stopped. New superbugs, such as carbapenem-resistant *Enterobacteriaceae* (CRE), have been identified. Almost half of those contracting this pathogen die, even with treatment. In addition to *Enterobacter*, some strains of *Pseudomonas aeruginosa*, *Klebsiella*, and *Acinetobacter* have shown resistance to carbapenems.

Since 2009, only two new antibiotics have been approved in the United States. Only seven new antibiotics were in development in the United States as this book was being written in mid-2013. Some will not make it to market. One company filed Chapter 7 bankruptcy in April 2012. At about the same time, the manufacturer of two of the new drugs said it was reducing spending on antibiotic research. The dearth of research overall is likely due to the lack of profitability of antibiotics compared with other drugs, such as those used to treat chronic disease.

For example, persons with diabetes and chronic obstructive pulmonary disease (COPD) take their medications one or more times daily. Antibiotics are used as infrequently as possible for a brief, time-limited period. To offset the reduced profits, manufacturers are charging prices that are prohibitively expensive for many people.

Recent surveillance data suggest that *Pseudomonas aeruginosa* may become the next antibiotic-resistant superbug.

Hirsch EB and Tam VH. Impact of multidrug-resistant *Pseudomonas aeruginosa* infection on patient outcomes. Expert Rev Pharmacoeconomics Outcomes Res 2010;10(4):441–451.

Gram-negative bacteria cause the most serious infections and are a major concern because the incidence of Gram-negative drug resistance is high and increasing. The cell wall of Gram-negative microbes is difficult for antibiotics to cross. There are other barriers inside the cells. A single bacterium can establish a biofilm

colony very quickly. It is no wonder that the incidence of drug-resistant Gram-negative infections is on the rise. As of today, they are winning the war. This is a frightening proposition.

The Infectious Disease Society of America (IDSA) issued a statement in April 2013, expressing concern over the lack of research and the increasing inability to eradicate bacteria, especially Gram-negative bacilli, with the drugs we have. This report clearly notes that of all the compounds in development for the treatment of MDR infections, none had an entirely Gram-negative spectrum. Clearly, there is a need for information on managing localized wound infection without using the few systemic antibiotics we have left.

> "For Gram positives, we need better drugs. For Gram negatives, we need any drugs."
>
> —Dr. Brad Spellburg, Infectious Disease Specialist at Harbor UCLA Medical Center *http://www.battlingsuperbugs.com/*

The Need for Localized Wound Care

In addition to increasing the risk of drug resistance, systemic antibiotics cannot reach therapeutic levels in chronic wound granulation tissue. Treating nonhealing wounds with topical antimicrobials solves both problems. We don't know what tomorrow's knowledge will bring, but today this is the best approach we have. The information in this book helps the clinician target the appropriate product to the characteristics of the wound, eliminating a great deal of guesswork and trial and error in prescribing and product selection. Eliminating infection is a high priority in nonhealing wounds.

The role of systemic versus topical antimicrobials is evolving. Just when we think we know it all, something changes. We still have much to learn about pathogens, and much more research needs to be done. Learning about biofilms threw much of what we thought we knew out the window. "Additional research and evidence-based guidelines are needed" could be the theme of every chapter of this book. What we know for now is that we must use antibiotics sparingly and judiciously before there are none left. This applies to all antibiotics, both topical and systemic. Never treat a localized infection with a systemic antibiotic unless extenuating circumstances exist.

For example, a culture reveals the presence of a very virulent pathogen, such as *Streptococcus A*. In this case, a systemic antibiotic should be considered in addition to aggressive localized wound care measures. However, most wounds can be treated with a topical product, if at all possible. A topical antiseptic may do the job, making the antibiotic unnecessary. To that end, use the information in this book to identify signs

of localized infection and critical colonization and eliminate them by using one or more topical antimicrobial products before systemic products become necessary.

One important goal of this book is to help healthcare professionals quickly locate appropriate topical products to manage infected wounds. The objective is to provide one-stop shopping rather than using time and multiple sources to identify an appropriate treatment product. We have spent literally hundreds of hours researching this information. The product information here was compiled from print and online information.

Sadly, some products are missing from the list in the appendix because manufacturers are not disclosing target organisms. Some lead the end user to believe that their product eliminates everything, which is obviously not true. The wounds pictured in this book were treated blindly. The wounds worsened. The treatment was obviously ineffective. Quality antimicrobials were used, but there were no cultures or guiding information on bacterial load. If you blindly try several antimicrobials and are unsuccessful, you are wasting time and increasing the risk that the person will develop a systemic infection. If the wound is not improving, obtain a culture and sensitivity (C&S). The best results will be obtained by targeting care to the wound characteristics and the causative agents.

However, do not make assumptions about a single type of planktonic bacteria in your culture results. Look at the whole picture, and measure the response (or lack of response) objectively. There may be a whole village of different organisms living in the basement.

This is not a full text or comprehensive book for healthcare professionals involved in the care of chronic wounds. The book covers care of chronic, nonhealing wounds only and not other dermatological conditions. Although some information may be applicable to children, this is incidental. The book was written for the care of adults. Follow physician orders and the indications for use and all precautions, contraindications, and instructions recommended by the manufacturer of the product being used. The book is a source of information on wound care products. No endorsement is made or implied, and we have no financial or other type of relationship with product manufacturers.

This book has a limited focus and is very basic for those who need information at this level. The information on use of honey and development of biofilms should be new to many. Terminology has also changed, and terms such as sessile, planktonic, bioburden, and critical colonization are regularly seen in professional journals. These are defined. Consistent, authoritative best practice guidelines are not available, so we have

assembled as much evidence-based information as possible to assist with your practice. If something seems too simple, please understand that someone needs it and do not be offended. Move ahead. You will find something that is applicable to your practice or new to you. Most of the references are free downloads. There is something here for everyone. If you need more complete information on the resistance problem, you may wish to review Cubist Pharmaceuticals' *A Guide to Superbugs and Antibiotic Resistance* from *http://tinyurl.com/superbug-battle* and the CDC's *Antibiotic resistance threats in the United States, 2013* from *http://www.cdc.gov/drugresistance/threat-report-2013/*.

This book presumes some prior knowledge and provides a simple overview of what we currently know about caring for chronic, nonhealing wounds. Persons needing a more comprehensive manual are referred to *Pressure Ulcers: Long-Term Care Clinical Manual* (Acello, B. 2010. Marblehead, MA: HCPro Inc.; *http://tinyurl.com/LTCCMPU-Acello*); *Evidence-Based Pressure Ulcer Prevention: A Study Guide for Nurses* (Clay, KS. 2008. Marblehead, MA: HCPro Inc.; *http://tinyurl.com/EBPUP-Nurses*); and *Evidence-Based Pressure Ulcer Management: Methods to Prevent, Assess, and Treat* (Sale, C. 2007. Marblehead, MA: HCPro Inc.; *http://tinyurl.com/Prevent-Assess-Treat*).

This book was written with a great deal of personal and professional collaboration. I am sincerely grateful for the assistance and cooperation of my colleagues:

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Perhaps we are preaching to the choir, but we believe that all professionals must be aware of the antibiotic resistance problem and have a responsibility to help preserve the antibiotics we have. The author and publisher of this book are committed to doing what we can to reduce the overuse and misuse of systemic antibiotics. We appreciate the confidence you have in our publications and will endeavor to continue to provide current, evidence-based information.

Barbara Acello, HCPro, Inc.
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Reference

1. Siegel JD, Rhinehart E, Healthcare Infection Control Practices Advisory Committee (2006). Management of multidrug-resistant organisms in health care settings, 2006. Am J Infect Control 2007; 35(10 Suppl 2):S165-93.

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The following items and many more are available for download at *www.hcpro.com/downloads/11347*.

- Wound Care Product and Technology Information
- Organism Sensitivity to Topical Agents
- Aerobic Microorganisms
- AHCPR Pressure Ulcer Treatment Guidelines
- When Alcohol-Based Hand Antiseptics Are Contraindicated
- Anaerobic Microorganisms
- Antimicrobial Resistance Patterns for Healthcare-Associated Infections (HAIs) Reported to the National Healthcare Safety Network (NHSN)
- Bacteria That Cannot Be Gram-Stained or Are Difficult to Gram Stain
- Beginning and Ending Procedure Actions
- CDC MRSA Brochure
- CDC Sequence for Donning Personal Protective Equipment (PPE)
- CDC Recommendations for Hand Hygiene
- CDC Recommended Immunizations for Adults
- Debridement Information
- ESKAPE Acronym
- Essential Oils in Wound Care
- Gram Stain Quick Reference
- Hydrogen Peroxide Grades
- Leg Ulcer Comparison
- Information About Medicare Patients Who Develop Pressure Ulcers During Hospitalization

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1 Overview of Wounds

Acute Wounds

Acute wounds are often characterized by the mechanism of injury, such as surgical incisions and traumatic injuries like lacerations, abrasions, avulsions, and burn injuries (see Figure 1.1). Repair of this wound is predictable, orderly, and timely and restores functional integrity to the area. If an acute wound fails to heal in six weeks, it will likely progress to a chronic wound. Classifications for surgical wounds are listed in Figure 1.2.

Chronic Wounds

All chronic wounds begin as acute wounds. They do not heal in an orderly fashion or in a predictable amount of time. Some never heal. **Chronic wounds** seem to be stuck in a phase of the healing process,

FIGURE 1.1

Types of Acute Wounds

Type of Wound	Cause
Contused wound	Blunt force causes tissue damage resulting in bruising and swelling. The skin is not usually broken.
Lacerated wound	Caused by something that tears tissues, producing jagged, irregular edges, such as glass, jagged wire, or a blunt knife.
Incised wound	Made with a clean cut of a sharp instrument, such as a surgical incision with a scalpel.
Avulsion wound	Caused by shearing forces and friction that result in significant tearing and destruction of tissues, such as a degloving injury.
Puncture wound	Caused by a pointed instrument, such as a staple gun, ice pick, bullet, or nail.
Burn wound	Caused by electrical, thermal, or chemical injury.

FIGURE 1.2

Classification of Surgical Wounds

Classification	Description
Class I-Clean (Rate of infection 1% to 5%)	• An uninfected, aseptic operative wound with no inflammation. Primarily closed and, if necessary, drained with closed drainage. • The alimentary, respiratory, or genitourinary tracts have not been entered. • Primarily closure.
Class II-Clean/Contaminated (Rate of infection 8% to 11%)	• Aseptically created wound that enters the respiratory, alimentary, or genitourinary tracts. • No contamination is present. • Primarily closure. • No evidence of infection or break in technique.
Class III-Contaminated (Rate of infection 15% to 20%)	• Open, fresh, avulsive, traumatic, or accidental wounds. • Contaminated surgical wounds caused by major breaks in technique or gross spillage from the GI tract. • Wounds that have been exposed to excessive amounts of bacteria.
Class IV-Dirty/Infected (Rate of infection 27% to 40%)	• Wounds with devitalized tissue, a foreign body, or a preoperative infection. • Wounds involving perforated viscera. • Class IV wounds are often left open to drain.
Modified from Centers for Disease Control and Prevention. http://www.cdc.gov/hicpac/SSI/table7-8-9-10-SSI.html	

usually the inflammatory phase. As a rule, wounds that do not heal within three to six months are considered chronic, but definitions vary. They are painful, may interfere with care, reduce quality of life, cause severe physical and emotional distress, and often cause a significant financial burden. Additional considerations include the following:

- A chronic wound does not heal in an orderly pattern or predictable amount of time. Wounds that do not heal within three months are often considered chronic.[1]

- As a general rule, wounds that do not progress approximately 15% in two weeks are considered "stalled."[2, 3]

- If a pressure ulcer does not decrease in size by at least 19.5% in one week, it may not heal in a timely manner.[4]

- If a venous ulcer does not show greater than a 30% reduction in size in two weeks, it is probable that it will not be healed at six months.[5]

- If a diabetic ulcer does not show a 30% reduction in size within the first two weeks of treatment, there is only a 9% chance it will heal in three months.[6]

Pressure Ulcers

Most chronic wounds are venous ulcers and pressure ulcers. Refer to the chart in the downloadable materials that accompany this book for information on differentiating leg ulcers. Pressure ulcers are staged by doing an assessment of the ulcer and then determining the classification or stage based on the anatomic depth of soft-tissue damage and underlying structures that are visible or palpable. The National Pressure Ulcer Advisory Panel (NPUAP) is a panel of experts in pressure ulcer prevention, management, and care. Monitor their Web page for updated information and current clinical practice guidelines: *www.npuap.org*.

NPUAP Stages and Definitions

In 2007, the NPUAP published revised definitions of the four pressure ulcer stages that have been used for many years. They added categories for unstageable ulcers, deep-tissue injury, and incontinence-associated dermatitis (IAD). The staging listed below reflects the new definitions. Refer to the NPUAP Web page for the precise terminology.

A **pressure ulcer** is caused by unrelieved pressure that has damaged the underlying tissue. They typically occur over bony prominences and are staged to classify the degree of tissue damage. Wounds are staged according to the identifiable structures and tissue in the wound bed. Depth is one factor but is not the sole determining factor. A pressure ulcer may not progress from stage I to stage IV in an orderly manner. It definitely does not heal from stage IV to stage I.

Pressure ulcers may contain **necrotic tissue**. Necrosis means "tissue death." Slough and eschar are two types of necrotic tissue. Wounds cannot be staged if covered by necrotic tissue and will not heal until slough and eschar have been removed. In fact, removing the devitalized tissue is probably the most important part of localized wound care.

Eschar is usually dark (black, brown, deep red) in color but may be tan. It is typically thick, leathery, and hard. Note whether the area surrounding the eschar is reddened, dry, moist, macerated, inflamed, etc. If the wound is various colors, describe the wound's appearance by using percentages, such as, "10% yellow, 40% red, 50% black."

Slough is usually lighter in color and has a stringy consistency. Slough that is yellow and stringy suggests that subcutaneous fat has died. Slough that is thick and yellow suggests muscular degeneration. Nonadherent slough separates easily from the wound and is mucous-like, thin, and watery. Loosely adherent slough is stringy and thick and may appear as clumps of debris attached to the wound. However, slough can be very difficult to remove and may return immediately after both sharp and chemical debridement.

Fluctuance (see Figure 1.3) typically occurs beneath eschar (especially on the heels) and suggests a bacterial infection and the presence of pus. Upon palpation, the area may be described as "mushy" or "boggy." (This feeling is fluctuance, which is moveable and is usually surrounded by induration.) A mushy or boggy feeling suggests breakdown will follow. **Induration** is abnormal hardening or thickening of tissue. It is usually caused by underlying edema and signifies infection. As infection progresses, the skin gets red and induration develops if not present previously.

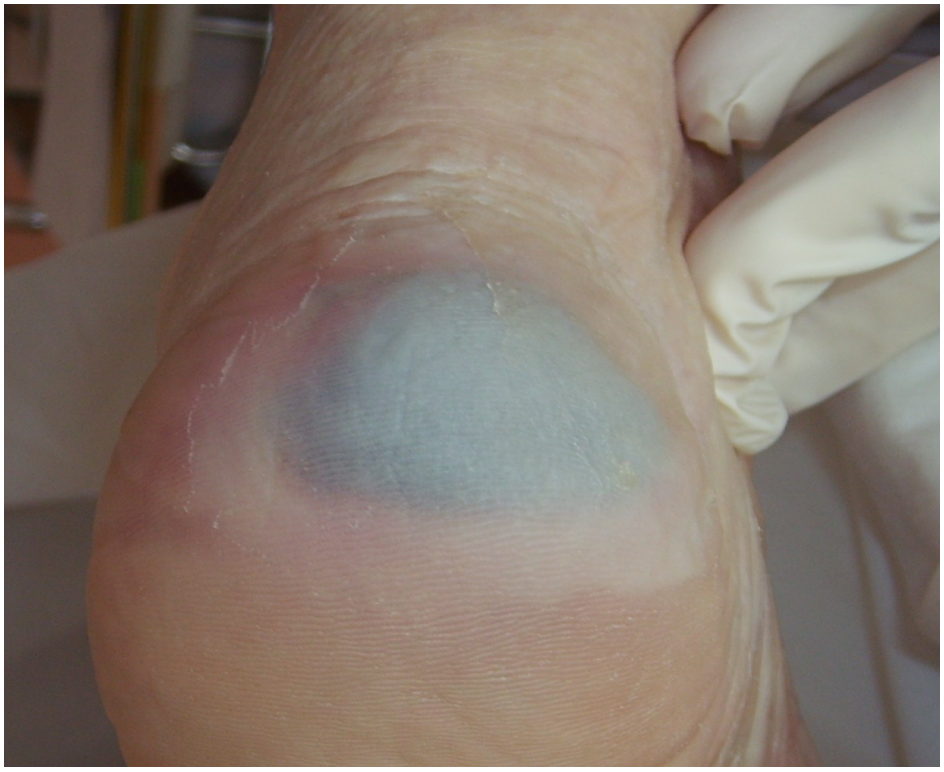
Suspected deep-tissue injury: A localized area of intact purple or maroon tissue or a lesion that looks similar to a blood blister. Pressure and shearing cause this tissue damage. The area may be painful before breakdown occurs, and other signs of tissue damage are evident. Upon palpation, it may feel firm, mushy, boggy, or warmer or cooler than the adjacent tissue.

- *Further description:* Deep-tissue injury may be difficult to identify in persons with dark skin. The injured area may have a dark appearance, and a thin blister may cover the wound bed. It may worsen rapidly, even with treatment. Alternatively, the wound may be covered with eschar. Deep-tissue injury poses a risk for rapid decline.

Stage I pressure ulcer: The skin is not broken, with a localized area of **nonblanchable erythema** (redness that persists when pressure is applied). This is a transient increase in blood flow that occurs following a brief episode of tissue ischemia. The pressure on the skin reduces the oxygen, and metabolic waste accumulates. The red color appears when blood rushes to the area when pressure is relieved. It should disappear after 30 to 60 minutes. The red, stage I area is usually over a bony prominence. In persons with dark skin, the color of the damaged area may differ from surrounding skin. Blanching may not be visible.

FIGURE 1.3

Fluctuance of the Heel



Source: Acello, Barbara, MS, RN. Pressure Ulcers: Long-Term Care Clinical Manual. *Danvers, MA: HCPro, 2010. CD.*

Check blanching 45 to 60 minutes after pressure is relieved to ensure the red color is not reactive hyperemia. Some experts believe reactive hyperemia results from Raynaud's phenomenon, in which vasospasms of blood vessels cause ischemia and tissue necrosis. A subsequent increase in blood flow (when pressure is relieved) removes accumulated waste products and cellular debris, at which point undamaged skin should blanch normally. The results of one study suggested that using nonblanchable erythema as a guide to initiating preventive care decreases the number of persons requiring preventive care without resulting in an increase in pressure ulcers.[7] This builds a case for tissue tolerance testing. For additional information, refer to the appendix of your book.

- *Further description:* The stage I area may be painful, firm, soft, or warmer or cooler than the adjacent tissue. It may be difficult to identify in dark-skinned persons. This category may be used to indicate persons at risk.[8] (The development of any area or history of a previous area suggests high-risk status.)

Stage II ulcer: A partial thickness of dermis is lost. The area looks like a shallow, open ulcer with a red-pink wound bed, without slough. It may also look like an intact serum-filled blister or open (ruptured) blister.

- *Further description:* Initially, the stage II area may appear shiny or as a dry, shallow ulcer without slough or bruising. (Bruising suggests deep-tissue injury may be present.) Avoid using this stage to describe skin tears, tape burns, perineal dermatitis, maceration, or excoriation.

Stage III ulcer: A full thickness of skin is lost, and subcutaneous tissue may be visible. Muscle, tendon, and bone are not exposed. Slough may or may not be present, but if present does not obscure the depth of tissue loss. Tunneling and/or undermining may be present.

- *Further description:* The depth of the stage III ulcer will vary with the location. For example, some areas have thin skin and no subcutaneous fat, such as the bridge of the nose, ear, occiput, and malleolus. Other areas, such as the bunion area of the foot over the first metatarsal and the heels, are also vulnerable. Because of the lack of subcutaneous fat, stage III ulcers are very shallow in these areas. Persons with significant subcutaneous tissue can develop very deep stage III ulcers, but bone and/or tendon are not visible or directly palpable because of the depth.

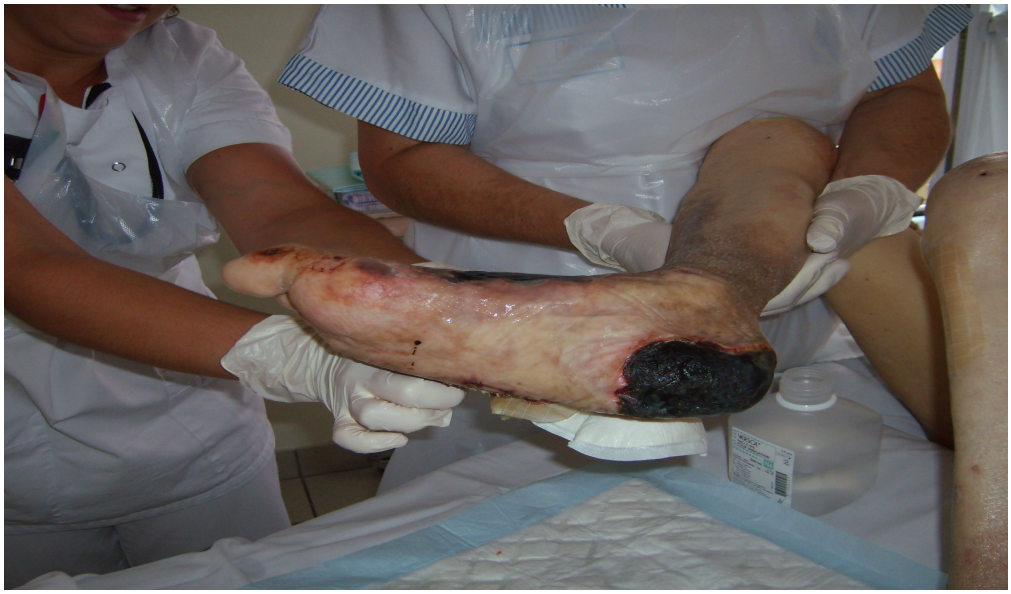
Stage IV ulcer: Full-thickness tissue loss with exposed bone, tendon, or muscle. Slough or eschar may be present. Tunneling and undermining are common. **Tunneling** is similar to sinus tracts, but the tunnels are larger and easily observed. Sinus tracts are small, with a narrower opening. Use the clock system to document location, e.g., tunnel at 3:00. Measure the depth of tunnels with a sterile cotton applicator, if possible. **Undermining** involves only the edges of the wound. In this situation, the upper edges appear to be detached from the lower margins, usually as a result of loss of subcutaneous tissue, infection (less common), or shearing.

- *Further description:* The depth of the stage IV ulcer also varies with anatomical location. As with the stage III ulcer, some areas have thin skin and no subcutaneous fat, so the ulcer can be very shallow. Exposed tendon or bone is visible or directly palpable. **Osteomyelitis** (see Chapter 2) is an infection of bone, usually as a result of bacteria. It is a risk because the ulcer can extend into muscle and/or underlying support structures (e.g., fascia, tendon, or joint capsule). Infections can contact bone by traveling through the bloodstream, by spreading from adjacent tissue, or when exposed bone is directly exposed to pathogens.

Unstageable ulcer: Full-thickness tissue loss in which the base of the ulcer is covered by slough and/or eschar in the wound bed.

- *Further description:* The stage cannot be identified until enough slough and/or eschar is removed so the wound bed can be examined and visualized. When this occurs, the stage is determined by the depth. Stable eschar on the heels serves as a protective mechanism and should not be removed. **Stable eschar** is dry and adherent and does not have edema, erythema, fluctuance, or drainage (see Figure 1.4).

FIGURE 1.4



This area is not stable and is separating around the edges. However, trying to help it along will worsen the condition.

Source: Barbara Acello, MS, RN.

Reverse Staging (Backstaging)

Healthcare professionals were formerly taught to stage healing wounds in reverse order. This is absolutely the wrong thing to do. Logically, one would think that wounds heal in reverse or from stage IV to III to II to I. However, a healed pressure ulcer contains scar tissue, and the skin is only about 75% as strong as it was originally. Because of this, the NPUAP and other experts do not support backstaging (reverse staging). Healing ulcers also do not replace the dermis, subcutaneous fat, or lost muscle during healing. In fact 13% to 56% of all ulcers will recur at the same location.

As an ulcer heals, it fills with granulation (scar) tissue. **Granulation tissue** is an accumulation of new connective tissue and capillaries that form on the surface of a healing wound. The lay term is *proud flesh*. Reverse staging does not accurately reflect the physiologic tissue changes. Aside from that, measurements can be misleading, because wounds may become larger as they heal, especially if they have been debrided. Loose skin can be pulled and stretched, causing very inconsistent measurements. A stage IV ulcer is always a stage IV. If it improves, it is a "healing stage IV." To support your findings, document wound assessment characteristics, such as length, width, depth, presence or absence of necrotic tissue or exudate, and presence of granulation tissue. Avoid subjective and uninformative statements with no basis in fact, such as "healing slowly" or "healing well." You may wish to use the PUSH tool on the NPUAP Web page.

Additional information

For more complete assessment information, refer to the appendix of your book.

The Kennedy Terminal Ulcer

A **Kennedy terminal ulcer** (**KTU**) is an ulcer some people get as they are dying. It was first identified by Karen Kennedy-Evans, RN, CS, FNP. Many experienced long-term care nurses have seen this ulcer but are unaware that it has a name. It is seen primarily in geriatrics and in those receiving hospice care. The skin is the largest organ of the body and, unlike other failing organs, changes are visible. A Kennedy terminal ulcer appears rapidly. Its usual location is the sacrum or coccyx, but it can appear elsewhere. It may be shaped like a pear, horseshoe, or butterfly.

The KTU begins with little black dots that look like BBs. They may initially appear to be fecal matter, specks of dirt on the skin, or dots drawn with a permanent marker. Some nurses have described the

lesions as blood blisters or abrasions. Caregivers may try to wash the spots off, only to find they cannot be removed because they are under the skin. The size of the BBs increases rapidly. They enlarge to the size of quarters within several hours. The KTU may look like a blister or stage II ulcer but rapidly progresses to a stage III or stage IV. The blister is very fragile, and the wound becomes large, even with gentle cleansing. The periwound tissue may be soft or loose beneath the skin surface. The ulcer starts to change color as it worsens and becomes deeper. Initially it is red, but it progresses to yellow and then black. The ulcers do not have much drainage.

The 2008 American Medical Directors Association (AMDA) Guidelines classify the KTU as an unavoidable ulcer. It falls within the NPUAP staging guidelines as a suspected deep-tissue injury. The Skin Changes At Life's End (SCALE) consensus statement may be downloaded from *http://tinyurl.com/EOL-consensus*.

For additional information, see:

- *www.kennedyterminalulcer.com*
- *http://tinyurl.com/KTUlcer*

Lower-Extremity Ulcers

Lower-extremity ulcers are a common, recurrent problem in about 1% of the general population and 3.5% of persons over age 65. This number increases to about 5% in those over age 80. Most are caused by venous disease, but about one-fifth of all persons have arterial disease, either alone or in conjunction with venous disease.

Ulcers of the lower legs and feet have many causes. Pressure causes some lower-extremity ulcers, such as when a person sits with the legs crossed for prolonged periods of time or wears tight, ill-fitting shoes. Other causes include diabetes, malignancies, infections, and underlying medical conditions in which ulcerations develop readily, spontaneously, or after a minor injury. As with any pressure ulcer, a complete assessment of the person is necessary.

Refer to the table in the appendix of your book for a side-by-side comparison of the characteristics of venous, arterial, and diabetic ulcers. Infection of these wounds is treated with the same products that you use for other wounds. However, remember that this person is at high risk for amputation.

Wound Healing

Wound healing occurs in three stages, although these stages may overlap. They are as follows:

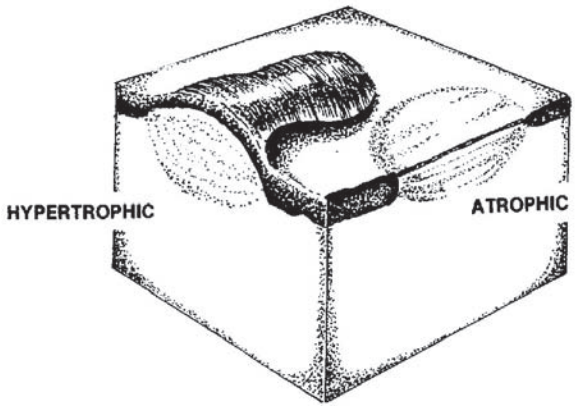
- The **inflammatory phase** occurs immediately after injury and lasts a brief time in partial-thickness wounds. The wound experiences an inflammatory response with heat, redness, pain, swelling, and impaired function. Vasoconstriction occurs within seconds after injury and lasts a few minutes. It is followed by vasodilation, which is caused by local stimulation of the nerve endings. The wound produces a serous exudate that forms a scab if allowed to dry. Inflammation usually lasts about three days.

- The **proliferative phase** overlaps the inflammatory phase slightly and continues until the wound heals. This phase involves regrowth of the epidermis. (Epithelialization is part of this stage but actually begins within hours of injury, during the inflammatory phase.) Small partial-thickness wounds that have been left open to air will heal in about six to seven days. Moist wounds will heal in about four days. Wounds involving loss of both epidermis and dermis repair both layers simultaneously. By the ninth day, collagen fibers emerge in the wound bed of a stage II ulcer. Collagen synthesis continues until about 10 or 15 days after the injury and continues to produce new connective tissue. Collagen synthesis requires vitamin C, amino acid, and adequate nutritional intake. Some experts theorize that cells surrounding hair follicles contribute considerably to dermal repair, accelerating healing in hairy areas of the body. In wounds with substantial tissue loss, granulation tissue contracts to close the area. This contracture does not occur in wounds with little tissue loss.

- The **maturation phase** begins about three weeks after injury and may continue for years in chronic wounds. In this stage, the collagen that has been deposited in the wound is remodeled and reorganized, which strengthens the wound and makes it more like adjacent tissue. New collagen is deposited, which compresses blood vessels and flattens and causes a scar (see Figure 1.5). The scar achieves maximum strength in about three months. Prior to this, it lacks tensile strength, and stress on the wound must be minimized. This area will never be as strong as it was prior to the injury. The scar will not sweat, grow hair, or get a sun tan. A wound is healed when the skin surface is continuous and strength is sufficient to support normal daily activities.

The presence of any wound warrants a complete assessment of the person, including nutrition, hydration, and underlying conditions.

FIGURE 1.5



Scar tissue is never as strong as it was originally and is always at risk for breakdown.

Source: United States Army.

Wound Infection

Infection occurs when the body is invaded by a pathogen that reproduces, damaging the tissue. Microorganisms are present in all wounds. They can enter the wound as soon as the skin is broken. However, the presence of microbes does not ensure an infection will develop. Other factors that contribute to the development of an infection are:

- Delayed treatment
- Decreased blood flow
- Shock
- Hematoma in the wound and surrounding tissues that restricts circulation
- Location of the wound: Areas with a good blood supply are less likely to develop an infection
- Presence of foreign body

- Inadequate cleansing or irrigation
- Inadequate removal of devitalized tissue
- Closing (suturing) a dirty wound
- Inadequate immobilization
- Contamination, breaches of sterile technique
- Presence of drug-resistant pathogens
- Secondary disease, such as obesity, immunocompromised state, diabetes, arteriosclerosis, poor circulation, decreased perfusion, and anemia
- Use of steroids
- Malnutrition
- Dehydration
- Cigarette smoking
- Alcohol abuse
- Advanced age
- Repeated trauma and injury to the area
- Edema
- Sickle cell disease

Chronic wound infection is often very subtle. It is usually characterized by initial healing followed by progressive deterioration. However, make sure to rule out other causes for nonhealing.

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CHRONIC WOUNDS

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